

ACTA

ORTHOPAEDICA ET TRAUMATOLOGICA HELLENICA

- Letter from the editor
- The art of diagnostic approach of a child
- Imaging and clinical approaches in the management of patients with spinal cord injury without radiographic abnormality (sciwora)
- Significance of quadrilateral plate in surgical treatment of complex bi-columnar acetabular fractures: descriptive analysis through three cases.
- Calcified tendonitis of the rotator cuff. A review of this common shoulder pathology
- Shoulder instability: a brief review

Young Scientists' Pages (53-83)



Official Journal of the
HELLENIC ASSOCIATION OF ORTHOPAEDIC SURGERY AND TRAUMATOLOGY
Athens Academy Award 2004

Haost Executive Board 2024

President	Eleftherios Tsiridis
Past President	Zoe Dailiana
First Vice President	Konstantinos Kateros
Second Vice President	Efstathios Chronopoulos
Secretary General	Vasileios Psychogios
Treasurer	Emmanuil Brilakis
Deputy General	Lazaros Poultsidis
Council Members	Nikolaos Zagoraios Konstantinos Tsivelekas

Chos Executive Committee 2024

President	Panos A. Efstathiou
Vice President (Ex Officio)	Konstantinos Kateros
Vice President	Stamatios A.N. Papadakis
Secretary	Anastasios V. Daras
Member	Ioannis Koulouris
Residents Delegate	Dimitra Melissaridou

Presidents Of Haost Sections 2024

Hip & Knee Reconstructive Surgery	Vassilios Nikolaou
Foot & Ankle	Dimitrios Hatziemmanouil
Spine Surgery	Nikolaos Sekouris
Shoulder & Elbow	Andreas Panagopoulos
Trauma	Mattheos Savvidis
Paediatric Orthopaedics	Rozalia Dimitriou
Research	Olga Savvidou
Musculoskeletal Oncology	Christos Zambakidis
Orthopaedic Infections	Sokratis Varitimidis
Primary Health Care	Konstantinos Saropoulos
Sport Injuries	Emanuil V. Brilakis





ZITA CONGRESS & EVENT
M A N A G E M E N T

εμείς τον ενθουσιασμό και την εμπειρία . . .

. . . εσείς την σιγουριά της επιτυχίας
του οργανωτικού και οικονομικού αποτελέσματος



συμβάλλουμε με διαφάνεια και εμπειρία **40** χρόνων
στους άμεσους στόχους και τα οράματά σας

www.zita-group.com

Ομήρου 29, Πέτα Σαρωνικού, 190 01, Αττική, Ελλάδα, Τηλ: +30 22994 40964
info@zitacongressevents.com

Editor in chief

Andreas F. Mavrogenis

Deputy Editors

George Babis (Greece)
 Efstathios Chronopoulos (Greece)
 Zoe Dailiana (Greece)
 Konstantinos Ditsios (Greece)
 George Drosos (Greece)
 Ioannis Gelalis (Greece)
 Panayiotis Givissis (Greece)
 Ioannis Gliatis (Greece)
 Michael Hantes (Greece)
 Theofilos Karachalios (Greece)

George Kontakis (Greece)
 Anastasios Korompilias (Greece)
 Panayiotis Megas (Greece)
 Panayiotis Papagelopoulos (Greece)
 Pericles Papadopoulos (Greece)
 Spyridon Pneumatikos (Greece)
 Eleftherios Tsiridis (Greece)
 Minos Tylianakis (Greece)
 Sokratis Varitimidis (Greece)
 Marios Vekris (Greece)

Associate Editors

Emmanuel Antonogiannakis (Greece)
 Athanasios Badekas (Greece)
 Emmanuel Brilakis (Greece)
 Rozalia Dimitriou (Greece)
 Dimitrios-Sergios Evaggelopoulos (Greece)
 Evanthia Galanis (USA)
 Panagiotis Giannoudis (UK)
 Ioannis Gkiatas (Greece)
 Efthymios Iliopoulos (Greece)
 Konstantinos Kateros (Greece)
 Efstathios Kenanidis (Greece)
 Zinon Kokkalis (Greece)
 Vasilios Kontogeorgakos (Greece)
 Dimitrios Koulalis (Greece)

Panayiotis Koulouvaris (Greece)
 Nikolaos Laliotis (Greece)
 Dimitrios Mastrokalos (Greece)
 Vasilios Nikolaou (Greece)
 Andreas Panagopoulos (Greece)
 Stamatios Papadakis (Greece)
 Lazaros Poultsides (Greece)
 Vasilios Psychoyios (Greece)
 Konstantinos Sountanis (Greece)
 Konstantinos Tilkeridis (Greece)
 Theodoros Tosounidis (Greece)
 Athanasios Ververidis (Greece)
 John Vlamis (Greece)
 Charalambos Zalavras (USA)

ΔΥΟ

ΔΙΑΤΡΟΦΗ ΥΓΕΙΑ ΟΜΟΡΦΙΑ

Η έγκριτη ενημέρωση
από Έλληνες Επιστήμονες,
σε ένα έντυπο και ηλεκτρονικό μέσο,
για το πολυτιμότερο αγαθό
του ανθρώπου

- 7.000.000 προσεγγίσεις
- 1.000.000 αλληλεπιδράσεις
- 30.000 followers
- 50.000 έντυπα



dyomagazine.gr

Zita Medical Management: Ομήρου 29, Πέτα Σαρωνικού, 190 01, Αττική, Ελλάδα, Τηλ: +30 22994 40962
g.kouloumpis@zita-management.com, info@zita-management.com



Instructions for Authors

1. Scope

“Acta Orthopaedica Et Traumatologica Hellenica” is the official journal of the Hellenic Association of Orthopaedic Surgery and Traumatology, first published in 1948. The current edition of Acta Orthopaedica Et Traumatologica Hellenica is published in English, online, without any article processing charges (APCs). It offers a compact forum of communication to orthopaedic surgeons and related science specialists. It publishes only peer reviewed articles. The peer review process is the established method for research validation in science whereby a work is critically assessed by expert referees demonstrating both the right level of knowledge in the field of the work, while being fully independent from it. Acta Orthopaedica Et Traumatologica Hellenica follows a blind peer review process mediated and ensured by the Editor-in-Chief and the Editorial Board members. Aiming for clinically pertinent, scientifically correct, ethical, original and review quality research, only scientifically sound articles, deemed of high enough interest and originality that will receive favorable reports from our Editors/Reviewers Board will be accepted for publication.

2. Types of papers

The journal accepts and publishes the following types of articles:

Original articles: Original articles are encouraged. They should provide novel insights and contribute to continuous medical education and transfer of knowledge. They should include a clear rationale, and the findings/conclusions need to be sound and supported by statistical analysis. When the accuracy of a diagnostic test is assessed, following the Standards for Reporting of Diagnostic Accuracy (STARD) flow diagram (<http://www.stard-statement.org>) is suggested. A structured

abstract of 250 words (divided into Background, Materials and Methods, Results and Conclusions), 3-5 keywords, text up to 4,500 words, figures up to five, tables up to six, and references up to 50 are recommended. (It is at the Editor’s discretion to allow differences in the above numbers).

Review Articles: All types are allowed including narrative reviews, systematic reviews, meta-analyses, literature reviews, mini reviews, monographs, and historical reviews on orthopaedic heritage. They should be extensive, educative, informative, adequately illustrated, and appropriately cited with up to date quality citations. An unstructured abstract of 150-250 words, 3-5 keywords, text up to 8,000 words, figures up to eight, tables up to six, references up to 100, and a maximum of six authors are recommended. (It is at the Editor’s discretion to allow differences in the above numbers).

Case Reports: Case reports should be didactic and educative, exceptional (or unique) and add to the current literature on an interesting topic, diagnostic criteria or therapeutic methods. An unstructured abstract of 150-250 words, 3-5 keywords, text up to 6,000 words, figures up to six, tables up to 4, references up to 80, and a maximum of six authors are recommended. (It is at the Editor’s discretion to allow differences in the above numbers).

Pictorial Essays (Images papers): The purpose of pictorial essays is to provide a teaching message through high quality images. A brief text (e.g., the history of the patient shown in the illustration) followed by a brief discussion are required to accompany the images. An unstructured abstract of 150-250 words, 3-5 keywords, text up to 4,000 words, figures up to four, tables up to two, references up to 20, and a maximum

of four authors are recommended. (It is at the Editor's discretion to allow differences in the above numbers).

Letters to the Editor: Letters to the Editor, Editorials, Communication to the editor are welcomed and will be published if they offer pertinent and constructive comment on articles previously published in *Acta Orthopaedica Et Traumatologica Hellenica*. These papers are usually solicited on a topical topic. No abstract, text up to 4,000 words, figures up to four, tables up to two, references up to 20, and a maximum of four authors are recommended. (It is at the Editor's discretion to allow differences in the above numbers).

3. Language

English is the official language of the journal. All submitted manuscripts should be written in English. The authors are encouraged to consult English speaking authors and services for correct English grammar and syntax.

4. Manuscript Submission

Submissions should be done online through the journal's website at <https://eexot-journal.com/index.php/index/login>. Submission should adhere to the journal's instructions with respect to the authorship, abstract, introduction (rationale of the paper), materials/methods, results, discussion, conclusions, references (format), illustrations and tables. Submissions not adhering to the journal's instructions will be send back to the authors for corrections that will delay the peer review process.

After submission, the Editorial office and the Editor-in-chief will check the submitted files and if appropriate will assign to section Editors or invite Reviewers. The time allocated for reviewers to assess the manuscript and submit their recommendation is 3 weeks. By that time the Editor-in-chief will make his final decision for publication.

5. Ethics and copyright

Submission of a manuscript implies that the work described has not been published before; that it is not under consideration for publication anywhere

else; that its publication has been approved by all co-authors, if any, as well as by the responsible authorities – tacitly or explicitly – at the institute where the work has been carried out. The Editors, the journal, and the Publisher will not be held legally responsible should there be any claims for compensation.

The journal follows the guidelines of the International Committee of Medical Journal Editors (www.icmje.org). For all original articles a statement in the text of approval from the local ethics committee, a statement that research was performed according to the ethical standards as described by the Declaration of Helsinki and a statement that informed consent for participation in the study was obtained from all subjects, are required. In case of study with animals the following statement needs to be added in the text: "All applicable international, national, and/ or institutional guidelines for the care and use of animals were followed".

All authors need to sign the copyright transfer form and must have made substantial contributions as established by the ICMJE (<http://www.icmje.org>).

6. Conflict of interest disclosure statement

Each author needs to disclose any type of financial interest that is related to the study and might create a potential conflict. Funding of the study, if any, needs to be disclosed. If there is no conflict of interest, this should be stated in the manuscript before the Reference section as follows: "The authors declared no conflicts of interest".

7. Permissions and plagiarism

Authors wishing to include figures, tables, or text passages that have already been published elsewhere are required to obtain permission from the copyright owner(s) for both the print and online format and to include evidence that such permission has been granted when submitting their papers. Plagiarism, as evidenced by appropriate dedicated software, will not be accepted. If excessive, the manuscripts with plagiarism will be returned to the corresponding author without consideration for peer review.

8. Submission checklist

A manuscript must contain the following files for submission:

Cover letter: Each manuscript should be accompanied by a cover letter signed by the corresponding author on behalf of the rest of the authors stating that the work submitted has not been published before; that it is not under consideration for publication anywhere else; that its publication has been approved by all co-authors, if any, as well as by the responsible authorities – tacitly or explicitly – at the institute where the work has been carried out. Any other information such as solicited paper, paper submitted for a special issue, letter to the Editor, etc should be communicated to the Editor in the Cover letter. In case of article resubmission a point-by-point answer to the reviewer's comments needs to be submitted with the cover letter.

Title page: It includes the title of the manuscript (concise, informative and capture of the message), the names of the authors, the affiliations of the authors, and the name, affiliation, address, e-mail address, and telephone number of the corresponding author.

Blinded manuscript: The manuscript should be blinded i.e. it should not include authors; names and affiliations.

Abstract and Keywords: An abstract and Keywords are required, as indicated above depending on the manuscripts types.

Text structure: the text of the Original Articles needs to be organized as follows: Introduction, Materials and Methods, Results and Discussion. Review Articles should include sections and subsections with appropriate headings depending on the topic; too many headings and subheadings should be avoided because they complicate reading. Case reports should include an Introduction, Case presentation, and Discussion. Pictorial Essays (Images papers) should include an Introduction and Discussion section only.

Abbreviations: Abbreviations should be used as minimum as possible, and should include only widely known and accepted abbreviations such as ORIF (open reduction and internal fixation), ICU (intensive care unit), etc. When used, they should be defined the first time they are used, followed by the acronym or

abbreviation in parenthesis.

Acknowledgements, sponsorships and grants: Acknowledgements should be added at the end of the manuscript before the References section. It should read as follows: "The authors thank... or acknowledge...".

Measurement Units: All measurements should be mentioned in international units (SI). The full stop should be used as a decimal (i.e. 3.5 cm). Spaces should be added around the plus/minus symbol (i.e. 13.6 ± 1.2). There should not be any spaces around range indicators (i.e. 15-20) or equality/inequality symbols (i.e. $r=0.37, p<0.005$).

Figure and Tables: Figures and tables should be cited in the text consecutively in the order in which they appear. They should be cited in parentheses at the end of the respected sentence, and not be referred to in the text. They should be counted in Arabic numbers: i.e. (Fig. 1) and (Table 1), and any Figure parts should be identified with lower case letters, i.e. (Fig. 1a).

Figures need to be of high quality (minimum resolution of 1,200 dpi) in TIFF or JPEG format.

Patient anonymity should be ensured and patient identifying images such as intraoperative or clinical photographs should be avoided. All identifying data (name, identification numbers, initials) must be removed from text, images and tables.

Figures and Tables legends should be explanatory and appropriate (what the figures and tables show). The legends should be listed at the end of the text, after the References section. The Figures and Tables should not be embedded in the text, but they should be uploaded in separate respective files, named respectively, i.e. (Fig. 1a).

Studies cited in the Tables should be cited according to the references list of the manuscript.

9. References

References section is not an afterthought but a continuum of the paper. They should be up to date and of acceptable quality. Their accuracy is the responsibility of the authors. They should be cited in the text in the order in which they appear. The numbering needs to be in Arabic numbers and formatted in su-

perscript in the respective areas of the text, after the punctuation (i.e. ¹).

All authors should be listed for all references of a manuscript.

When a book chapter is cited, the authors and title of the chapter, editors, book title, edition, city and country, publisher, year and specific chapter pages should be mentioned.

For Online Document, the following should be mentioned: authors (if any), title of page, name of institution or owner of Web site; URL; dates of publication, update, and access.

References examples:

Journal article:

Mavrogenis AF, Altsitzioglou P, Tsukamoto S, Er-rani C. Biopsy Techniques for Musculoskeletal Tumors: Basic Principles and Specialized Techniques. *Curr Oncol.* 2024;31(2):900-917. doi: 10.3390/curroncol31020067.

Sun J, Mavrogenis AF, Scarlat MM. The growth of scientific publications in 2020: a bibliometric analysis based on the number of publications, keywords, and citations in orthopaedic surgery. *Int Orthop.* 2021;45(8):1905-1910. doi: 10.1007/s00264-021-05171-6.

Kolovos S, Sioutis S, Polyzou M, Papakonstantinou ME, Karampikas V, Altsitzioglou P, Serenidis D, Koulalis D, Papagelopoulos PJ, Mavrogenis AF. The risk of DDH between breech and cephalic-delivered neonates using Graf ultrasonography. *Eur J Orthop Surg Traumatol.* 2024;34(2):1103-1109. doi: 10.1007/s00590-023-03770-0.

Book chapters:

Mavrogenis AF, Antoniadou T, Dimopoulos L, Filip-piadis D, Kelekis A. Metastasis (Chapter 26). In: Text-book of Musculoskeletal Disorders. Vincenzo Denaro, Umile Giuseppe Longo (Eds). © Springer Nature. 2023. ISBN 978-3-031-20986-4.

Online document:

Copyright Registration Guidance: Works Contain-ing Material Generated by Artificial Intelligence Available at: <https://www.federalregister.gov/documents/2023/03/16/2023-05321/copyright-reg-istration-guidance-works-containing-material-gen-erated-by-artificial-intelligence>. Published Feb 2023. Accessed on Jan 27, 2024.

10. Review and Proof reading of manuscripts

The Reviewers comments will be communicated to the Authors. The Authors should make proof corrections within 3 weeks. All comments should be addressed point-by-point in a cover letter with Authors' responses to Reviewers' comments. Upon acceptance, the authors will receive a proofs pdf document of their paper for proofs reading. Then, the Authors will be asked to check the integrity of the text (importantly the authors' names and af-filiations), accept any grammar or spelling chang-es and check if all the Tables and Figures are in-cluded and properly numbered. This should be done promptly, preferable within 72 hours. Once the publication is online, no further changes can be made. Further changes can only be published in form of Erratum.

**For new article submission visit
www.eexot-journal.com**

Writing for ACTA Orthopaedica et Traumatologica Hellenica

Andreas F. Mavrogenis

Editor-in-Chief, ACTA Orthopaedica et Traumatologica Hellenica

This article offers advices and tips on medical writing for the junior authors and the less experienced in medical writing on how to prepare a quality submission. These tips apply to any author and any journal, and it is the Editor's personal view and experience in medical writing. Before starting the paper, search the related literature; choose quality papers that are electronically available; provide appropriate correct citations for any material previously published to avoid plagiarism. Before writing the paper, read the authors' instructions. These instructions will need to be met in any case.

Authorship

The number and the order of the authors' names should be fair by reflecting their contribution and the order of their contribution to the manuscript. Those who authored should be listed as authors of the manuscript. Those who have contributed to the work, but not enough to merit their inclusion in the authorship, should be acknowledged in the acknowledgment section. Authorship is not a way to thank a colleague for support, access to resources, or mentorship. Scientific misconduct (fraud) in authorship includes a gift or complimentary authorship, ghost authorship, and coercion authorship.

Title

It should be short and concise; it should capture the message. Titles raising or answering questions will far be more appealing than titles merely pointing to the topic. Do not use run-on (long and busy) titles.

Abstract

It should include all the important information from each section that is the background, questions/purposes, materials/methods, results, and conclusions. The readers should be able to understand the total

paper by just reading the Abstract. Some read only the Abstract (e.g., because they do not have the time or access to the full text). Keywords are important for indexing and should be chosen carefully.

Introduction (approximately 500 words)

It is the most critical section. It should start with focus on the topic. General and irrelevant information should be avoided. The first paragraph should present the background. The second paragraph should present what is important on the topic. Appropriate citations (the related studies) should be added. These studies should be further discussed at the discussion section.

The section should end with a clear rationale. Questions to be asked when formulating the rationale are the following: (1) What is missing from the literature for this study to merit publication? (2) How does this study add to the related literature? (3) Does it confirm or reject previous reports? After the rationale, the purposes of the study (study questions or hypotheses) should be listed. The purposes may be primary (the most important) and secondary (the least important). Writing should be clear and concise.

Materials and Methods (approximately 1000-1500 words)

The section should start with the Materials in brevity and clarity. An example could read as follows: "We present patients admitted and treated at the authors' institution with from 2000 to 2024. There were ... men and ... women with a mean age of ... years (range, years)". These two sentences provide almost all basic demographic information of the materials of the study. Follow-up is materials and should be provided here; the same for loss to follow-up including the reasons for the loss. Clinical reports must state

inclusion and exclusion criteria and whether the series is consecutive or selected; if selected, criteria for selection should be stated. These should inform the readers for any sources of bias.

When reporting clinical studies, the authors must state informed consent (where appropriate) and approval of the institutional review board or ethics committees of their institution. These should be added at the first paragraph of the Materials and Methods sections as follows: *“All patients gave written informed consent for their data to be included in this study. This study was approved by the Institutional Review Board (IRB)-Ethics Committee of the authors’ institution”*. Alternatively, *“Informed consent was not necessary for review articles”* or *“IRB and Ethics Committee approval was not necessary at the authors’ institution for retrospective studies”*.

The Methods should contain adequate detail for another investigator to replicate the study. The authors should clearly present what they did and how they did it in the study and analysis. The Methods should be validated with appropriate citations such as for a used score, method, classification, etc.

If authors use statistical analysis, a paragraph should appear at the end of Materials and Methods stating all statistical tests used. When multiple tests are used, the authors should state which tests are used for which sets of data. The level of statistical significance is 0.05 in most cases.

Results (approximately 500 words)

It should be the answers to the study questions in the same order as formulated in the rationale at the last paragraph of the Introduction section. It is easier and more informative to format the study answers (results) in paragraphs. Each paragraph should start with a key statement of the most important result, and then the description and statistical analysis should follow.

The authors should provide which group/method/analysis is more significant compared to another and parenthetically state the p-value immediately after the comparative terms. Provide the actual p-values instead of p-values greater or lesser than 0.05. Parenthetic reference to all figures and tables

enables easier interpretation of the data. Avoid too many numeral data in tables because it complicates and fatigues reading.

Discussion (approximately 1500-3000 words)

The Discussion should start with a restatement of the problem or question in brief for emphasis, followed by the study findings and a synthesis of the comparison and the author’s new data to arrive at conclusions.

The second paragraph should be the limitations. I prefer the readers should be informed early for the limitations of the study. Failure to explore the limitations suggests the authors either do not know or choose to ignore them, potentially misleading the reader.

In the next paragraphs the authors should discuss their findings in comparison to the literature. They should synthesize their data with that in the literature. The text should be formatted in paragraphs respective to the study questions/answers. Appropriate and quality studies should be used. Generally, many of these reports will include those cited at the Introduction section. A Table that summarizes the results of the most important published related studies would be useful here (refer to papers with similar tables for the format).

The ultimate paragraph of the section should be the conclusions. The conclusions should be based solely on data that come out of the paper. Conclusions irrelevant of the study findings should not be used. General and philosophical statements should be avoided. Statements such as “need for further research” or “need for future studies” should be avoided because they underpower the study.

References

Choose quality references, and read the most important papers in full text; approximately 25% of the references used in the references list of a paper are actually read by the authors when writing the paper. References should be accurate and up-to-date. Electronically available citations should be preferred; abstracts and submitted articles (pend-

ing publication), newsletters, proceedings, and meetings syllabus should not be used because many in these categories ultimately do not pass peer review because it is not possible to be traced and cited. Use citations from the journal to submit your paper; this will gain the Editor that you are aware of the journal; it will increase the visibility of the paper and the impact of the journal.

Figures and Tables

Figures and tables should complement not duplicate material in the text. They present information that would be difficult to describe in text form. Well-written papers contain one or two tables or figures for every study question/purpose posed in the Introduction. The legends should be explanatory and concise; what the figure/table show.

References

1. Brand RA. Writing for clinical orthopaedics and related research. *Clin Orthop Relat Res.* 2008;466(1):239-47. doi: 10.1007/s11999-007-0038-x.
2. Mavrogenis AF, Auffret Babak I, Caton JH. Writing for SICOT-J. *SICOT J.* 2021;7:E1. doi: 10.1051/sicotj/2021042.
3. Mavrogenis AF, Scarlat MM. Writing for “International Orthopaedics”: authorship, fraud, and ethical concerns. *Int Orthop.* 2021 Oct;45(10):2461-2464. doi: 10.1007/s00264-021-05226-8.

Contents

■ Letter from the editor	1
<hr/>	
■ Historical Article	
The art of diagnostic approach of a child	
Nikolaos G. Markeas, Yvonne-Mary Papamerkouriou, Anastasios Daras	2-8
<hr/>	
■ Basic Science	
Imaging and clinical approaches in the management of patients with spinal cord injury without radiographic abnormality (sciwora)	
Nikolaos Siatos, Ioannis S. Benetos, Dimitrios-Sergios Evangelopoulos, John Vlamis, Maria-Eleftheria Evangelopoulos	9-23
<hr/>	
■ Original Article	
Significance of quadrilateral plate in surgical treatment of complex bi-columnar acetabular fractures: descriptive analysis through three cases.	
Fotios V. Nikolopoulos, Ioannis V. Papachristos, Konstantina Solou, Georgios Gourtzelidis, Dimitrios Samaras	24-31
<hr/>	
■ Reviews	
Calcified tendonitis of the rotator cuff. A review of this common shoulder pathology	
Chrissovalantis Tsirikas, Ioannis K. Triantafyllopoulos	32-40
<hr/>	
■ Shoulder instability: a brief review	
Spyridon Manthas, Ioannis Kotsalis, Lampros Oikonomou	41-52
<hr/>	
Young Scientists' Pages	
■ Psychological support for the patient with spinal cord injury	
Panagiota Efthimiou, Ioannis S. Benetos, Dimitrios-Sergios Evangelopoulos, John Vlamis	54-63
<hr/>	
■ The role of electrical stimulation in the management of lower urinary track dysfunction following spinal cord lesions	
Nikolaos Koutsogeorgis, Maria-Eleftheria Evangelopoulos	64-73
<hr/>	
■ Sexual and reproductive health of patients with spinal cord injuries - Orientation to female fertility and pregnancy	
Ariadni Petropoulou, Dimitrios-Sergios Evangelopoulos, John Vlamis, Maria-Eleftheria Evangelopoulos	74-83
<hr/>	

1+1... Εκσυγχρονισμός - Ανάπτυξη

Δύο μεγάλες Εταιρείες ενώνουν τις δυνάμεις τους και σας προσφέρουν Υπηρεσίες marketing, χρηματοοικονομικών και αναπτυξιακών ευκαιριών.

Στο όραμα σας για εκσυγχρονισμό του **ιατρείου** σας και αποτελεσματικότερη διεύθυνση στο κοινό σας, έχετε τώρα την πιο αξιόπιστη επιλογή!



ZITA  **BSS**
MEDICAL MANAGEMENT

Entering a new editorial era in ACTA Orthopaedica et Traumatologica Hellenica

Andreas F. Mavrogenis

Editor-in-Chief, ACTA Orthopaedica et Traumatologica Hellenica

Writing for ACTA Orthopaedica et Traumatologica Hellenica

Writing a scientific paper that will get published is one of the most rewarding achievements in a medical career. Once the privilege of few scholars, medical writing currently is feasible for anyone, and a must for career advancement and personal reputation. Writing a paper provides intellectual stimulation, generates discussion, advances discipline, and enhances authors' reputation. However, medical writing should include medical knowledge, expertise in writing, and special considerations and rules, including formal writing and easiness to understand.

Writing and publishing depends not only on the methodology used and quality of data analysis, but also on how the paper is written. A clear and concise language will help Editors and Reviewers concentrate on the scientific content of the paper and thus facilitate the peer review process. English language grammar and syntax is usually edited by the office and the assistant Editors. Occasionally, English language editing is extremely difficult, and substantial work may be required, mainly in cases where the authors' English proficiency is low. In those cases, we recommend manuscript editing by a professional or a native speaker prior to submission.

In the era of the internet, attractive scientific papers should be clear and well-illustrated; the readership enjoys illustrations and surgeons like graphic content, schemes, drawings and pictures. The illustrations should express important features of the methods and results. Importantly, illustrations should have adequate, meaningful and explanatory legends. Clinical photographs should be accurate and not allow the patient identification. Complex collections of data should be summarized in tables. The results of the most important published related studies are useful to allow the readers to make comparisons easily and should also be presented in tabular data. As Editor, I have the privilege to publish papers that I enjoy reading; an enjoyable article will help colleagues to learn and I am in favor of the old saying "a picture is worth a thousand words" for a surgical publication.

New Editorial Board

The backbone of any journal is an active, widely respected, diverse, and representative Editorial Board. Beginning from the present issue, the journal gains a new Editorial

Board. The Editorial Board is expected to assist in a variety of aspects of running the journal, mainly in providing scientific expertise, administering peer review and serving as a peer reviewer, helping the journal to attract high-quality manuscripts for example by promoting the journal at relevant conferences, and suggesting topics and authors for solicited reviews and commentaries. Importantly, the Editorial Board should bring the journal's attention to new audiences, particularly in new topics or countries, and should communicate the journal at conferences and talks.

A journal's Editorial Board is not static, but it is regularly reviewed in order to ensure the correct people are in place and the field is well represented. In this setting, new Editorial Board members will be frequently recruited; more active members will be receiving increased responsibility, and less active members will be given the chance to step down, creating vacancies for new members.

Quality online publishing

Today, the number of journals and papers published has increased dramatically, most of them with arguable clinical impact. The medical writing environment is becoming exhaustively competitive, yet no real standards have been established. Open access journals, predatory politics and effortless publishing often containing an almost satiric quality are major concerns that discourage writing. As the official journal of the Hellenic Association of Orthopaedic Surgery & Traumatology (HAOST) we publish articles mainly from Greek authors, mostly orthopaedic surgeons, as well as related disciplines and young scientists. The quality is not always the same but we presume that the authors will improve with each written paper. Our aim is to support the spirit of the HAOST where every surgeon could join to learn and improve. It is our enthusiastic wish to be indexed in PubMed and similar impact libraries. But for that purpose, a substantial number of yearly submissions of decent quality articles is necessary. Importantly, these articles should cite the journal aiming to increase the visibility of the journal and the impact of the published papers. Until then, our intention should be to improve the journal further; it is better to publish in a quality online journal than to not publish at all. We wish you to enjoy reading ACTA Orthopaedica et Traumatologica Hellenica and do hope that you will be publishing with us.

The art of diagnostic approach of a child

Nikolaos G. Markeas¹, Yvonne-Mary Papamerkouriou², Anastasios Daras¹

¹*Athens Children's Euroclinic*

²*Second Department of Orthopaedics, Children's Hospital of Athens "P. & A. Kyriakou"*

Abstract

While trying to approach a child diagnostically, the child has to be relieved from "white coat anxiety" as well as being in awe of the *doctor-ogre*. The clinicians need to establish a suitable environment in which the child's fears will be appeased and the parents will be reassured in order for them to be content and fulfilled. The diagnostic approach of the child is not always easy. Finding the true cause of a child's symptoms is not as simple as following a cookbook recipe as there are no specific ingredients or measurements that will lead to a safe result.

Keywords: child; diagnosis; medical history; clinical examination; radiographs

Introduction

The diagnostic approach of the sick child needs to be broken down into many components. In addition, the underlying analysis will take into account many factors in order to establish the true causes.

There are few publications dealing with the issue [1, 2]. As far as the clinician is concerned, experience may lead to hurried conclusions, which are subjective and not based on evidence. We live in an era characterized by inconsistency and pluralism therefore this should not be a surprise. Recanting is a part of human nature.

Medicine as both a science and an art needs to overcome these hurdles [3]. The achievements of technology can aid to this end. Artificial intelligence also offers solutions. Scientific breakthroughs have offered therapies for ailments formerly considered incurable. In addition, the mapping of the human genome has established the pathogenesis of several diseases thus opening the door to personalized treatment.

Human willpower is the backbone of resistance. Machines cannot and must not replace humans. The artificial intelligence of a robot is subject to programming and cannot evolve and feel in the same way as a human being. Even when the relationship between a doctor and a patient has been disrupted, it can always be mended. The art of the diagnostic approach to the sick child gives the clinician the opportunity to develop virtues that may have been in a state of hibernation.

Welcome

As the child enters the examination room, a new experience begins. Everything is relevant, ranging from the color of the walls, the pictures, the proper ventilation as well as the temperature, which all contribute to making a positive impression on the child's psyche. Overall, the physician's attitude is the cornerstone of this experience [4].

The doctor's smile and positive attitude will initial-

Corresponding
Author

Nikolaos G. Markeas MD, PhD
Former Senior Consultant of 2nd Pediatric Orthopaedic Department
General Children's Hospital of Athens "P. & A. Kyriakou"
42 Sikelianou St., 122 43 Egaleo, Greece E-mail: markeasn@otenet.gr

ize an optimistic beginning. Just by saying an honest “welcome”, the doctor will minimize the child’s anxiety and eliminate possible pre-existing stereotypes. In order to establish a positive climate there is no need for excessive compliments. The doctor should be brief and precise. He should be himself and avoid pretending. The child has intuition and is able to judge as well as compare (**Figure 1**). However, the doctors are good actors when they manage to hide their exhaustion and manage to appear sober and generous. This is required of the circumstances.

This can be achieved by drawing attention to an unimportant detail such as the child’s shoes, hair or bag. Phrases such as “What wonderful shoes you are wearing!” or “Those pigtails match your face” or “I’d like to know what you’re treasuring in that delightful bag of yours!” could prove calming for the child even though they may not be entirely true. It does not need too much effort to break the ice. The child will be occupied by trying to respond and will forget its fear regarding the consultation.

The archetype of a doctor is that of an individual in a white coat. White coats are connected to seriousness, expertise, authority and a protagonist role. This of course applies to the adult world but is not the case in the world of pediatrics. Children feel threatened by the white coat and this can be an obstacle in any attempt to approach the child [5, 6]. The alternative would be for the physicians to wear a badge with their credentials.

Children, however, cannot be fooled. If there is going to be unpleasantness during the examination the doctor should warn both the parents and the child beforehand. Otherwise, the child’s initial fears will be justified.

The first steps of approaching

The climate, which will be established in the following moments between the clinician and the parents, is the next step. Without trying to ignore the child, we need let the parents explain the reason of the consultation.

The clinician needs to show his concern for the child by asking questions which relate to the child’s life, his or her school, friends, schoolmates and outdoor activities. The clinician should seek the parents’ approval.

The child may promptly show unwillingness to be examined. Its mood should be taken into account, se-



Figure 1. The child has intuition and is able to judge as well as compare.



Figure 2. The child may promptly show unwillingness to be examined.

riously (**Figure 2**). However, the doctor may use tricks in order to distract the child. One way would be mention someone the child respects and say something like “What would grandpa say if he knew you didn’t let the doctor examine you?” for instance.

The pediatrician who initially examined the child is usually the doctor who has referred the child. In other cases, this could be a doctor of the same specialty who lacks experience with children. In every case, one needs to be careful and respect the reason for the referral. It is not advised to underestimate the view of the referring physician. It is always a good idea to be polite. The parents should be reassured that they are on the right track in order to find a solution for their child and have the impression that the doctors are in collaboration between them.

Even if the previous treatment did not work or the



Figure 3. The child may reveal the truth.

diagnostic approach was not right, it is useless to criticize colleagues. Being subtle and polite may not be mentioned in the Hippocratic Oath [7]; however, it is an essential part of good practice.

History, as the keystone of diagnosis

One must of course allow the child to speak for itself. Children as well as adolescents are often considered unreliable however; we need to listen to their point of view. We need to be resilient if both sides tell a different story while paying attention to the parents' comments. The child may reveal the truth (Figure 3). If, for instance, it tells us that during a fall on its outstretched hand it received a kick on the lateral side of its elbow from an obese schoolmate, we assume that the injury could be that of an avulsion fracture of the medial epicondyle. In conclusion, we need to weigh the information given by both the child and the parents against our clinical findings.

We also need to establish who is in closer contact with the child and therefore more likely to notice a problem [8]. Is it the parent, grandparent or baby siter? The grandma, for instance, may notice an intoeing gait; the teacher may sign a kyphosis or scoliosis of the spine. Establishing what the initial complaint is, leads us to the following questions:

- a) Was the child treated in the past, by whom, and what were the outcomes?
- b) Are there any other members of the family with similar problems?

The answers might help with the establishment of a diagnosis but also could reveal the caregiver who is closest to the child [9].



Figure 4. The clinical examination should not appear to be so.

Words that may frighten the child, such as "injection", "aspiration" or "operation", should be avoided especially if they do not bare any relevance to the situation other than to intimidate the child. In addition, certain phrases should be avoided such as telling the child "This will not hurt" because:

- We introduce the subject of pain into the conversation
- The child focuses on the potential pain, anyway
- There are fears of escalating painful activities

It is also good to avoid the presence of siblings or friends while examining the child. They can be told politely to stay in the waiting area.

The art of clinical examination

The embarrassment experienced by the child when seeing the clinician who is about to examine it can be paired to the awkwardness felt by the examiner. In this case, confusion and stress only make things worse. There is no need to be antagonizing. One should rather be gentle and patient. The doctors should use their ingenuity in order to overcome the dead ends [10, 11]. The following advice may prove helpful.

One should not touch the child from the very beginning. It is better to watch the child while recording the personal history. The child should be allowed to walk or run in the corridor. The physician can establish whether the child is walking in a coordinated manner or not. Are the hands hanging or is there spasticity? One can ask the child to walk on its toes and heels and hop on each leg.

When starting the physical examination, it is advised to start by the arm or leg, which is not painful.



Figure 5. Try to have a parent present always, especially when examining the spine in adolescents.

The physician's touch is therefore innocent and the child is encouraged. As long as it is not afraid, it tries to cooperate. If it still does not respond positively, one must change tactics. The parents may help by opening a mobile or tablet and trying to engage the child by playing videos with the child's favorite cartoon characters to calm it. Alternatively, they might try singing a familiar song and trying to get the child to participate (*"Donkey, donkey, old and gray/Open your mouth and gently bray/Lift your ears and blow your horn/to wake the world this sleepy morn"*).

The clinical examination has now taken off; however, it should not appear to be so (**Figure 4**). The clinician should not be in a hurry to examine both legs in their entirety. Rather, he should focus on the nails, bruises from previous injuries, bites from insects. A useful dialogue may evolve. In the meantime, muscle tone as well as range of motion of hips, knees and ankles are examined. Parents understand what is going on and contribute [12-15].

To many children the examining bed appears to be intimidating. In this situation, one must not insist that the child lies down, as this will probably end in a boxing match! It is best to ask the mother to take the child in her arms, in order to examine it. Always start with the normal limb. Do not be intimidated by the usual comment: *"but doctor, it's the other leg"*. Explain why you are doing this. While doing the Thomas test, hold the side that hurts, still. If nothing works, instruct



Figure 6. When testing for DDH by performing the Ortolani and Barlow tests, the baby must be calm and fed.

the parent on how to examine the child. Usually this works in cases of irritable hip [16, 17].

Try to have a parent present always, especially when examining the spine in adolescents (**Figure 5**). Otherwise, be sure at least a nurse is present. This is essential for both medical and legal reasons.

While trying to reach conclusions, try to get the parent involved as well. Parents usually have their own opinion based on observation of the child's gait while at home. Consider everything, finish your physical examination and base your conclusion on established criteria centered on the literature. Try to earn the parents' trust by using science instead of trying to generalize and simplify. For example, in the matter of flatfoot, do not get carried away by the parent's observation of the child's flat foot arch. Instead, do a proper clinical examination, perform the relevant tests and try to reassure the parents and rid them of the prejudice they may have.

Do not hesitate to repeat clinical tests if they are not performed correctly the first time. For instance, when testing for DDH by performing the Ortolani and Barlow tests, the baby must be calm and fed (**Figure 6**). Postpone the tests until the circumstances are right.

Try to be calm and do not hurry. Being in hurry leads to a disorganized process of thought. Parents can understand when you are not listening carefully to the clinical history and when you are not being thorough in your clinical examination. When you address the



Figure 7. When examining X-Rays, the doctor should always take into consideration normal variations so that there is no confusion.

parents, do it sitting down. Listen to their questions and try to answer them while being calm and patient.

The study of images

Often, the parents arrive at the consultation already holding blood test results, X-Rays, and CDs of CT scans and MRI scans. They have had previous consultations and are seeking a second opinion.

While studying the results one can understand the working diagnosis of the colleagues who have previously examined the child. When results are positive it could be helpful to let them know.

When examining X-Rays the doctor should always take into consideration normal variations so that there is no confusion (**Figure 7**). When discovering a fibrous cortical defect by chance, one should not hold it responsible for causing any symptoms provided it is limited and is not close to an articular surface [18]. When examining a scoliosis curve we need to be prudent. If the curve is considerable, both parents and child will not be prepared to accept treatment with a brace. We need to re assure them.

CT scans show the bones in detail and the doctor should be prepared to explain the results to the parents in detail. When examining images of a MRI scan, the doctor should be prepared to discuss common finding such as bone edema in a simple way to the



Figure 8. While facing a diagnostic challenge, the doctor will have to explain to the parents that he needs time to process the findings.

parents. The parents are often anxious when reading the results of tests and have already embarked on a journey of upsetting thoughts.

In the event, that additional imaging is needed for the portrayal of certain obscure fractures (radial head or coronoid process) we order a new referral and even speak to the radiologist ourselves. We may do the same if we notice something in the images, which is not referred to in the report. For example in the event of a calcaneonavicular coalition, which is fibrous, it may be hard for the radiologist to diagnose it. The pairing of imaging with clinical examination aids the diagnosis [16-18].

The power of words

Several years ago, German classicist Bruno Snell had said, *“The man should listen to the echo of his own voice before knowing himself”*. The doctors need to be careful with the words they use during the diagnostic approach of a child, because the words reflect their personality, reveal the level of knowledge and create an impression on the parents [19, 20].

The physician need not hold back. All sentences need to be precise. Phrases follow one another in logical sequence. If there is a need for repetition, this must be done without complaint.

Parents are full of questions. Their mental

state of mind depends on the doctor's reaction. Everything may be relayed in different way. Even bad news can be delivered in a calm and controlled manner, leaving a door open to hope for a positive outcome.

A doctor should never swank. However, he should try to use medical terms when having to explain things. He should not try to use simplifications. When patients refer to a "break" this is actually a fracture. The doctor should make an effort to familiarize the parents with the proper terminology [21].

The use of key words leads the parents to a google search. This could prove dangerous. While surfing on the net, the algorithms will lead the parent to unpredictable findings. It is almost certain that they will not find the answers they seek. They will be overcome by the generalization. The doctor will have to step in.

The weakness of numbers

Man is weak when it comes to numbers. How much can he trust in statistics? The following example will portray this.

The incidence of DDH has both a racial and geographical parameter. The incidence is 1.7 in 1000 births in Sweden, 75 in former Yugoslavia and 188.5 in Manitoba Canada. The incidence in China and Africa is close to zero [17].

It is hard to make headway with statistics. Especially because of immigration, it is difficult to draw safe conclusions.

A question often asked by the parents is the percentage of positive outcome connected with the operative technique we are proposing. The dialogue between doctor and parent could go like this:

Parent: *What is the success rate, doctor?*

Doctor: *Between 17% and 67% depending on the authors.*

Parent: *Which percentage would you trust?*

Doctor: *I do not trust any of the percentages even if there are a product of meta-analysis. I only trust my own experience.*

Parent: *And what does your experience say, doctor?*

At this point, the conversation takes a hazardous turn. Experience is acquired after making mistakes

and is constructed on trying not to repeat them. The doctor knows of the possible complications and tries to avoid them. Nevertheless, this all exists in his conscience and cannot be published. It should not be the doctor's alibi since it produces confusion to the parent.

The doctor could answer: *"The success rate is 100%"*. This of course is bold and frivolous. However, it is not far from the truth. It creates optimism and acts in a dual way. It holds the doctor responsible for living up to his promises while giving the patient the trust he requires in order to proceed.

Some important points

While facing a diagnostic challenge the clinician will have to explain to the parents that he needs time to process the findings (Figure 8). The orthopedist should not think it shameful to have to reexamine the history and clinical findings as well as collaborate with the pediatrician. Reexamine the lab results and imaging. Parents usually like this [22-24].

If the problem is complicated, it might help if the doctor were to write a few things down for the parents including the treatment options and pros and cons of each choice.

Baron Munchhausen used to say, *"Luck often corrects our mistakes"* [25]. Even if this does occur, we should not rely on luck for the treatment of our patients. We should take responsibility in order to resolve the problem, however hard that may be. We should always be alert and communicate with our colleagues who can help.

Conclusions

The diagnostic approach of a child might seem hard since there are no specific rules; however, it is not impossible when it is based on knowledge and experience. Only when there is collaboration between humans the purpose of being is acknowledged. A wonderful mechanism of back-and-forth feedback prevails in the fight of humans for humanity. It is definitely worth the while and the prize is that of human dignity.

Conflict of interest

The authors declare no conflicts of interest.

References

1. Beebe AC, Kerpsack JM. *Pediatric musculoskeletal examination*. In Dormans JP (editor): *Pediatric Orthopedics: Core Knowledge in Orthopedics*. Philadelphia, Mosby 2005, pp 15-35.
2. Davis, Holly W, Zitelli, Basil J. Παιδιατρική φυσική εξέταση και διάγνωση, σε επιμέλεια Στυλιανού Μανταγού, Δημητρίου Καφετζή και Εμμανουήλ Καναβάκη (τόμοι 2). Εκδόσεις Π. Χ. Πασχαλίδης, Αθήνα, 2009.
3. Μουντοκαλάκης Θεόδωρος. Νέες έννοιες στην Ιατρική. Εκδόσεις Παρισιάνου, Αθήνα, 2021.
4. Hosalkar HS, Wells I. *Growth and Development*. In Kliegman, Behrman, Jenson, Shanton's (editors): *Nelson Textbook of Pediatrics*, 18th edition 2007, WB Saunders Company. pp 2771-2773.
5. Άντλερ Άλφρεντ. Η αγωγή του παιδιού, σε μετάφραση Σταύρου Καμπουρίδη. Εκδόσεις Γεράσιμου Αναγνωστίδη, Αθήνα 1978.
6. Μοντεσσόρι Μαρία. Η διαμόρφωση του ανθρώπου, σε μετάφραση Σόνιας Καλογεροπούλου. Εκδόσεις Γλάρος, Αθήνα 1979.
7. Ιπποκράτης. Ιατρική δεοντολογία. Νοσολογία, σε μετάφραση Δημητρίου Λυπουρλή. Εκδόσεις Ζήτηρος, Αθήνα, 2001.
8. Μαρκέας Νικόλαος. Γωνιώδεις παραμορφώσεις στο παιδικό γόνατο. Εκδόσεις ΕΕΧΟΤ, Αθήνα 1993.
9. Herring JA. *The Orthopedic examination: Clinical Application*. In Herring JA (editor): *Tachdjian's Pediatric Orthopedics*, 4th edition 2008, Philadelphia, WB Saunders, pp 67-78.
10. Ντράκορς Ρούντολφ. Το παιδί. Μια νέα αντιμετώπιση, σε μετάφραση Ιουλιέττας Καβαδά. Εκδόσεις Ερμής, Αθήνα 1964.
11. Ντινκμέγιερ Ντον, ΜακΚαίη Γκάρντ. Το υπεύθυνο παιδί και πώς να το μεγαλώσουμε, σε μετάφραση Εύης Νάντσου. Εκδόσεις Θυμάρη, Αθήνα 1980.
12. Πέτροβιτς-Ανδρουτσοπούλου. Μιλώντας για τα παιδικά βιβλία. Εκδόσεις Καστανιώτη, Αθήνα 1983.
13. Herring JA. *The Orthopedic examination: A comprehensive overview*. In Herring JA (editor): *Tachdjian's Pediatric Orthopedics*, 4th edition 2008, Philadelphia, WB Saunders, pp 27-66.
14. Hosalkar HS, Wells I. *Evaluation of the Child*. In Kliegman, Behrman, Jenson, Shanton's (editors): *Nelson Textbook of Pediatrics*, 18th edition 2007, WB Saunders Company. pp 2773-2774.
15. Ο' Brien Niall, Gill Denis. Κλινική εξέταση παιδιών, σε μετάφραση Ιωάννη Καβαλιώτη. Εκδόσεις Ροτόντα, Αθήνα, 2008.
16. Μαρκέας Νικόλαος. Πόνος στο παιδικό πόδι και χωλότητα. Εκδόσεις ΕΕΧΟΤ, Αθήνα 1999.
17. Μαρκέας Νικόλαος. Μυοσκελετικές παθήσεις κάτω άκρων σε βρέφη και παιδιά. Εκδόσεις University Studio Press. Θεσσαλονίκη, 2018.
18. Μαρκέας Νικόλαος. Ο ακτινογραφικός έλεγχος στον αναπτυσσόμενο σκελετό. Πόσο αξιόπιστος είναι; Εκδόσεις ΕΕΧΟΤ, Αθήνα 2006.
19. Snell Bruno. *The Discovery of the Mind*. Harper and Row, 1960
20. Φωκάς Αθανάσιος. Μονοπάτια κατανόησης. Εκδόσεις Broken Hill Publishers LTD, Λευκωσία Κύπρου, 2023.
21. Μαρκέας Νικόλαος. Η μάχη της διφθόγγου στην Ορθοπαιδική. Εκδόσεις Κωνσταντάρας, Αθήνα, 2014
22. Staheli LT. Normative data in Pediatric Orthopedics. *J Pediatr Orthop* 1996; 16:561.
23. Καρπάθιος Θεμιστοκλής, Ματοσιανιώτης Νικόλαος. Παιδιατρική (τόμοι 2). Εκδόσεις Λίτσας, Αθήνα, 1999.
24. Χρυσανθόπουλος Χρυσανθος. Παιδιατρική και εφηβική πρωτοβάθμια φροντίδα. Εκδόσεις Ροτόντα, Αθήνα, 2012.
25. Μπίργκερ Γκότφριντ Άουγκουστ. Οι περιπέτειες του βαρόνου Μινχάουζεν. Εκδόσεις Σμυρνιωτάκης, Αθήνα, 2021.

Cite this paper as



Markeas NG., Papamerkouriou YM, Daras A. The art of diagnostic approach of a child. *Acta Orthop Trauma Hell* 2024; 75(1): 2-8.

Imaging and clinical approaches in the management of patients with spinal cord injury without radiographic abnormality (SCIWORA)

Nikolaos Siatos¹, Ioannis S. Benetos^{1,2}, Dimitrios-Sergios Evangelopoulos^{1,2}, John Vlamis^{1,2}, Maria-Eleftheria Evangelopoulos^{1,3}

¹Postgraduate Training Program, KAT Hospital, National and Kapodistrian University of Athens School of Medicine, KAT Hospital, 2 Nikis str, 14561, Athens, Greece

²3rd Department of Orthopaedic Surgery, National and Kapodistrian University of Athens School of Medicine, KAT Hospital, 2 Nikis str, 14561, Athens, Greece

³1st Department of Neurology, National and Kapodistrian University of Athens School of Medicine, Eginition Hospital, 72-74 Vas. Sofias Av., 11528, Athens, Greece

Abstract

SCIWORA is a syndrome that defines posttraumatic SCI in patients with abnormal clinical neurological examination and apparently normal radiological findings in plain X-rays and CT. Under the suspicion of SCIWORA, early MRI is recommended for definitive diagnosis while prompt neuroprotective measures have to be taken to prevent secondary SCI that may cause further neurological deterioration.

Introduction. SCIWORA (Spinal Cord Injury Without Radiographic Abnormalities) is a syndrome that defines posttraumatic SCI in patients with abnormal clinical neurological examination and apparently normal radiological findings in plain X-rays and CT. This syndrome most commonly affects children, but can be also found in adults, with a predilection for the cervical spine. The aim of this study is to review the imaging and clinical approaches in the management of SCIWORA patients.

Materials & Methods. A literature review was conducted based on the Pubmed internet database, following the PRISMA Guidelines. Article titles were searched with the use of the keywords: "Spinal Cord Injury without Radiographic Abnormality" OR "SCIWORA". The search included only clinical studies evaluating SCIWORA in adults. Studies published in non-English language, animal studies, experimental studies, case reports, reviews, and commentary studies were excluded. Moreover, studies in children and adolescents were also excluded.

Results. Initially, 207 studies were identified after primary search on Pubmed electronic database. After screening of titles and abstracts, 15 articles were excluded. Among the remaining 192 studies, 167 were rejected for various reasons (figure 1). After checking the references lists of the included studies, 2 more studies were added, leaving 29 studies for final analysis. The total number of patients was 1418 (78.2%

Corresponding
Author

Dimitrios-Sergios Evangelopoulos,
ds.evangelopoulos@gmail.com

men) with a mean age of 53.5 years. The incidence of SCIWORA among all SCI cases varies from 1.3 – 12%. The most common cause of SCIWORA is fall from a height (53%), followed by motor vehicle accidents (33.6%), sports injuries (6.4%), occupational injuries (2.8%) and other injuries (4.6%). 94.4% of the SCIWORA occurred in the cervical spine, while the rest 5.2% occurred in the thoracic spine. The most common mechanism of injury is hyperflexion of the cervical spine especially in patients with preexisting cervical spondylosis. 12% of patients were AIS grade A, 20% AIS grade B, 35% AIS grade C and 33% AIS grade D. In 14.2% of patients, no MRI abnormalities were detected, while 85.8% of patients had abnormal MRI scan results. Among them, 57.0% had extraneural, 36.3% had intraneural and 6.6% of patients had combined extraneural and intraneural MRI abnormalities. Initial treatment is conservative. Indications for surgical management include MRI findings of cord compression and instability, along with deterioration of neurological symptoms. Prognosis of SCIWORA depends on the initial neurological deficit and magnitude of SCI on MRI; however, neurological improvement is expected in at least 75% of patients.

Conclusions. SCIWORA is an underestimated clinical condition in adult SCI patients. In the suspicion of SCIWORA, early MRI is recommended for definitive diagnosis and prompt neuroprotective measures have to be taken to prevent secondary SCI that may cause further neurological deterioration for a better prognosis. According to MRI findings, surgical treatment is indicated in patients with cord compression and instability and worsening neurological symptoms. More high quality studies are needed to fully elucidate the optimal imaging and clinical approaches in the management of SCIWORA patients.

Keywords: Spinal cord injury; SCIWORA.

Introduction

Spinal cord injury (SCI) is one of the most challenging medical conditions, strongly associated with high mortality rates, ongoing disability, significant deterioration of quality of life and a severe socio-economic burden to the patients and the society. SCI is usually caused by spinal trauma, usually due to motor vehicle accidents, falls, occupational injuries, gunshot or stabbing wounds. Non-traumatic SCIs may be caused by infections, tumors or degenerative conditions [1]. The degree of disability caused by the SCI depends on the site and the extent of the injury. Diagnosis is based on clinical and imaging findings, especially X-rays, computed tomography (CT) and magnetic resonance imaging (MRI), which may reveal spinal fractures, dislocations, spinal cord edema and hemorrhage. However, there are cases, especially in children, where the spinal cord is injured without any evident imaging findings.

Spinal cord injury without radiographic findings, also known as SCIWORA (Spinal Cord Injury Without Radiographic Abnormalities-SCIWORA) syndrome, was first described by Pang et al, as a

SCI in a child without radiologic abnormalities on plain x-ray film [2]. SCIWORA is a known entity in populations of children sustaining a cervical spine injury [2], but its incidence in adults varies considerably between different studies. In 1948, Barnes et al reported the first case series of five adults experiencing SCI presenting with a clinicoradiologic mismatch [3]. The concept of SCIWORA was extended to adult practice by Hirsh et al, who adopted the term to report the case of an adult with a thoracic SCI with apparently normal X-rays [4]. Thereafter, further reports provided more data to the existence of SCIWORA in adults. However, taking into consideration the differences in spinal anatomy, direct comparisons between children and adults with SCIWORA have usually been avoided [5, 6].

By definition, SCIWORA originally assumed negative radiographs only. With the evolution of more advanced imaging techniques, the absence of pathologic findings on CT was incorporated in the diagnostic criteria of SCIWORA. Subsequently some argued that the following terms should be used for adult patients: “Spinal Cord Injury Without Radio-

logical Evidence of Trauma" (SCIWORET) or "Spinal Cord Injury Without CT Evidence of Trauma" (SCIWOCTET), so as to exclude cases where plain radiographs and CT are normal, but cervical spondylosis is present [7, 8]. The introduction of CT and the imaging of very small lesions that may conceal hidden injuries have reportedly led to a reduction in the incidence of SCIWORET in adults from 14% to 5% [9, 10].

The increasing availability of MRI added another diagnostic examination for the identification of intraneural and extraneural abnormalities in patients experiencing SCI. However, the role of MRI in the diagnosis, prognosis, and management of SCIWORA patients has not been fully elucidated. There are rare cases of SCIWORA, where even MRI can be normal. These cases have been evaluated under the definition of "real SCIWORA" [11]. Furthermore, the lack of a reliable classification system based on the morphologic MRI abnormalities weakens the interpretability and comparability of relevant studies. Some authors referring to SCIWORA syndrome, have also included in their studies patients with an increased prevertebral intensity or a fracture in the anterior-superior angle of the vertebral body [12, 13], which, however, are well documented points suggestive of trauma, thus creating further confusion in the literature. In 2005, there has been a recommendation that the term SCIWORA in adults should also include negative MRI in addition to negative x-rays and CT [14]. However, typically, the radiological examination does not involve MRI and therefore it should not be considered a prerequisite for the definition of the syndrome [15].

The aim of this study was to review the imaging and clinical approaches in the management of patients with Spinal Cord Injury without Radiographic Abnormality (SCIWORA).

Materials And Methods

A literature review was conducted based on the online Pubmed database, following the PRISMA Guidelines, with the use of the EndNote X3 software (Thompson Reuters) [16]. Article titles were searched with the use of the keywords: "Spinal Cord Injury without Radiographic Abnormality" OR "SCIWO-

RA". The search included only clinical studies evaluating SCIWORA in adults. Studies published in non-English language, animal studies, experimental studies, case reports, reviews, and commentary studies were excluded. Moreover, studies in children and adolescents were also excluded.

Results

Initially, 207 studies were identified after primary search on the online Pubmed database. After screening of titles and abstracts, 15 articles were excluded. From the remaining 192 studies, 165 were rejected for various reasons (figure 1). After checking the references lists of the included studies, 2 more studies were added, leaving 29 studies for final analysis.

The 29 selected studies were published from 1999 to 2022. Table 1 depicts all the included studies in the present review. The total number of patients was 1418 (78.2% men) with a mean age of 53.5 years.

Incidence

According to the included studies in this review, the incidence of SCIWORA varied from 1.3% to 12% [5, 6, 15, 18, 20, 25]. The incidence of SCIWORA in the large NEXUS (National Emergency X-Radiography Utilization Study) study was 3%, but the authors stated that this low rate could be underestimated [22].

Mechanism of injury

Road traffic accidents and falls were the two most common causes of SCIWORA. According to the present review, the most common cause of SCIWORA was fall from a height (53%), followed by motor vehicle accidents (33.6%), sports injuries (6.4%), occupational injuries (2.8%) and other injuries (4.6%).

Site of injury

The most common site of SCIWORA was the cervical spine. According to the present review, 94.4% of the SCIWORA occurred in the cervical spine, while the rest 5.2% occurred in the thoracic spine. These findings come in agreement with the literature, as the thorax and abdomen protect the thoracic spine from excessive flexion or extension, providing inherent stability [42]. According to the epidemiological study by Guo et al, the C4 - C5 segment was the

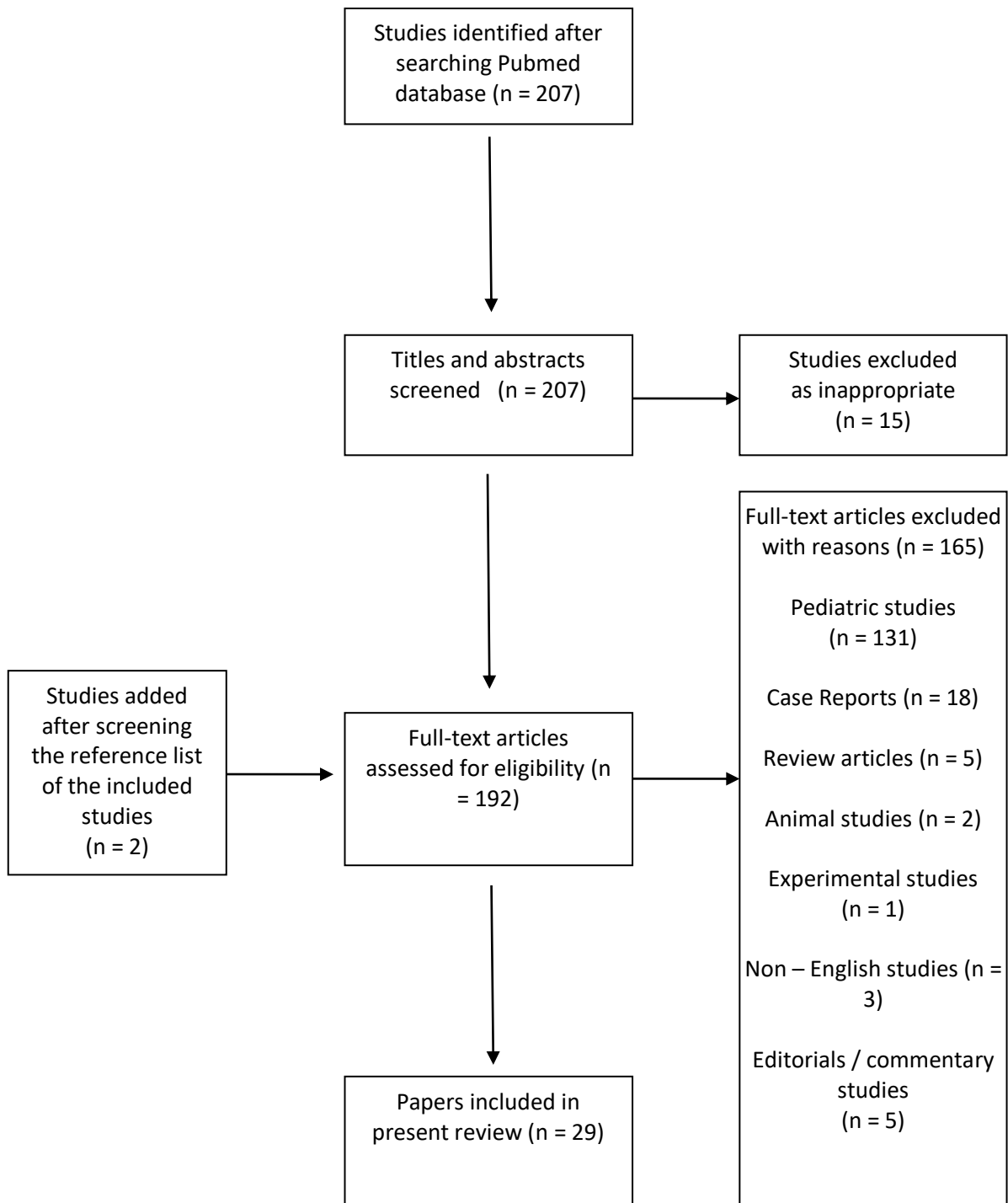


Figure 1. Study flowchart

most commonly affected level (48.7%) followed by C5 - C6 and C3 - C4 (30.5 and 12.8%, respectively) [21].

Pathophysiology

Guo et al, in the largest included case series, analyzed 203 cases of SCIWORA and concluded that combined hyperextension, flexion, and distraction of the head is the most frequent mechanism [21]. In most adult patients, SCIWORA are the result of hyperextension injury to the spine, as a result of rear end motor vehicle collisions or direct anterior craniofacial trauma [35]. In older patients, a syndrome of SCI without obvious skeletal injury was reported by the studies of Crooks, Birkett, Schneider et al [43, 44]. Many of these patients have pre-existing cervical spondylosis resulting in narrowing of the cervical canal and predisposing to SCI even after minor trauma [18]. Hyperextension injuries in these patients cause the spinal cord to be compressed between the posterior vertebral osteophytes and the inward bulging of the ligamentum flavum resulting in an acute central cord syndrome [15]. Moreover, a direct blow to the spine could produce shock-wave oscillations causing 'slapping damage' to the spinal cord against the bony spinal canal [45]. In case of hyperextension injuries, the upper extremities are more affected than the lower extremities and most patients have incomplete SCI, with a good prognosis [44].

Risk factors

The calculated mean age of patients in the present review is 53.5 years. 78.2% of patients with SCIWORA were men. Age is an important consideration when evaluating patients with SCIWORA, as the syndrome is very rarely seen between the ages of 16-35. Children under 8 years of age and adults over 60 are the patients who mostly experience it, but these two populations have different characteristics. As far as children are concerned, the relatively large size of the head together with the increased intrinsic mobility of the developing skeleton, as well as the concomitant ligamentous laxity, make spinal cord vulnerable to high energy damage. On the other hand, the elderly patient population has almost unique characteristics: these patients often

have posterior osteophytes and marginal bulging of the ligamentum flavum due to loss of intervertebral disc height, and are thus susceptible to developing central cord syndrome after a hyperextension injury of the cervical spine, as can happen after a fall from the same height.

Clinical Findings

SCIWORA patients may present with a variety of neurological signs, such as tetraplegia, motor and sensory dysfunction, and loss of bladder and bowel function, in addition to local pain, range of motion limitation, edema and bruising around the vertebral column. For the clinical evaluation of the SCIWORA patients, 3 clinical scales were used: the American Spinal Injury Association (ASIA) Impairment Scale (AIS), the Frankel scale and the Japanese Orthopedic Association (JOA) score [46, 47]. Fourteen of the included studies used the AIS scale. 12% of patients were AIS grade A, 20% AIS grade B, 35% AIS grade C and 33% AIS grade D. Six studies used the Frankel scale. The largest study of Guo et al included 66 AIS A patients, 77 AIS B, 36 AIS C and 24 AIS D [21].

Imaging findings

The timely and accurate diagnosis of SCI is of paramount importance in order to initiate appropriate therapeutic intervention [48]. X-rays along with CT are vital for the proper identification of the site and the severity of the injury [49]. Note that by definition, SCIWORA consists of a negative radiological screening (plain x-rays and CT), but not a negative MRI. Invasive imaging methods such as myelography have a limited use [50]. Dynamic imaging in SCIWORA patients following negative plain X-rays and CT images does not provide any diagnostic advantage [51].

Spinal MRI provides valuable additional data for the status of the spinal cord and spinal nerves [52, 53]. The rate of abnormal MRI findings after a normal CT result is 15%, but the majority of these findings had no clinical significance, and in only 0.3% of cases the additional information provided by the MRI led to a surgical intervention [52]. Taking these into consideration, abnormal findings on MRI may lead to overestimation of SCI, in comparison to intraoperative

Table 1. Case series included in the present review. N = Number of patients. NM: Non - mentioned				
AUTHOR	YEAR	N	MEAN AGE (years)	% MALE
Asan ^[17]	2018	11	55	36,4
Boese ^[18]	2013	21	35,5	57,1
Boese ^[19]	2016	26	52	65,4
Cao ^[20]	2022	164	56,3	74,4
Guo ^[21]	2011	203	55,9	88,2
Gupta ^[5]	1999	15	38,8	80,0
Hendey ^[22]	2001	27	42	81,5
Huang ^[23]	2013	5	48,8	80,0
Kasimatis ^[15]	2008	7	66,8	100,0
Kawano ^[24]	2012	54	62,4	81,5
Kothari ^[12]	2000	4	31	75,0
Liu ^[25]	2015	59	41,1	69,5
Machino ^[26]	2011	100	55	79,0
Machino ^[27]	2019	100	55	79,0
Maeda ^[28]	2012	88	64	89,8
Martinez-Perez ^[29]	2017	49	54	81,6
Na ^[30]	2021	11	63,8	100,0
Neva ^[31]	2011	32	46,9	90,6
Ouchida ^[32]	2016	68	62	76,5
Qi ^[33]	2020	57	49,2	77,2
Qi ^[34]	2022	106	50,5	NM
Sharma ^[35]	2009	12	38,7	83,3
Shen ^[36]	2006	5	27,8	60,0
Tan ^[37]	2022	86	51,7	57,0
Tewari ^[6]	2005	40	42,1	77,5
Wang ^[38]	2015	52	NM	NM
Yaqoob Hakim ^[39]	2021	11	46,5	90,9
Zhang ^[40]	2015	16	46,5	75,0
Zhu ^[41]	2019	16	47,5	81,3

Table 2. Classification of SCIWORA in adults by MRI imaging type [57].

MRI Imaging Type	MRI abnormalities
I	No detectable abnormalities
Ila	Extraneural abnormalities
Ilb	Intraneural abnormalities
Ilc	Extraneural + Intraneural abnormalities

Table 3. Classification of intramedullary abnormalities in adults by MRI imaging type [63].

Pattern	Cord Finding	MRI imaging
I	Hemorrhage	Large central area of hypointensity surrounded by a thin rim of hyperintensity on T2W images
II	Edema	Area of hyperintensity on T2W images
III	Contusion	Thin area of central hypointensity and thick rim of hyperintensity on T2W images

findings [54]. However, as a few SCIs may require immediate surgical management, MRI within 24 hours of injury has been recommended in patients, with clinical SCI findings and normal X-rays and CT [18]. In case of a normal early MRI, a second MRI in 72 hours post injury may reveal spinal cord changes [55, 56]. After all, MRI has been the gold standard in the diagnostic approach of suspected SCI patients.

MRI has the capacity to detect characteristic pathomorphological extraneural and intraneural abnormalities. Intraneural abnormalities include spinal cord edema, bleeding, contusion and transection (partial or complete). Extraneural findings concern lesions to the soft tissues surrounding the spinal canal including intervertebral disc herniation, ligamentum flavum folding, prevertebral soft tissue edema, or ligamentous abnormalities [57, 58]. Epidural hematomas are very rarely reported, especially in patients with ankylosing spondylitis or severe cervical spondylosis [59].

Boese et al described a classification system of SCIWORA based on MRI findings, where type I was defined as a normal MRI scan, while type II includes three abnormal subtypes (Table 2) [57]. The present review included 1175 adult SCIWORA patients with reported MRI findings. In 167 patients (14.2%), no MRI abnormalities were detected (Type I), while

1008 (85.8%) had abnormal MRI scan results (Type II). Of these, 575 patients (57.0%) had extraneural (Type Ila); 366 patients (36.3%) had intraneural (Type Ilb); and 67 patients (6.6%) had combined extra and intraneural MRI abnormalities (Type Ilc).

An intervertebral space abnormality is indicative of a possible damage of the intervertebral disc that can be clearly visualized by MRI as a disruption or herniation. In case of a disruption, the disc injury is best seen on sagittal T2WI as high signal intensity in the intervertebral space and low signal intensity on sagittal T1WI [60]. Disc abnormalities after injury are reported to range from 16% to 48% in patients with SCIWORA [22, 61, 62] and Tewari et al noted a 37.5% rate of disc herniation [6].

The anterior longitudinal ligament (ALL) is a crucial component of the anterior spinal column. ALL injury causes bleeding and edema in the prevertebral space, including ALL, the prevertebral muscle and the intervertebral disk. This change is visualized as high-signal intensity on T2W1 MR imaging. This prevertebral hyperintensity (PVH) suggests a hyperextension mechanism of the injury [32]. The rate of PVH in SCIWORA has been estimated at 76% to 90% [26-28, 32].

The posterior longitudinal ligament (PLL) is a

component of the posterior spinal column connecting the posterior surfaces of the vertebral bodies. It weakly prevents hyperflexion of the vertebral column. The cervical PLL is comparatively narrow and thin, and strongly adherent to the posterior annulus fibrosus. In patients with non-traumatic disc herniation, nucleus pulposus may break through the PLL and posterior annulus fibrosus. According to the large case series by Guo et al, PLL rupture is the most common type of injury in SCIWORA patients, accounting for 43.8%, followed by ALL rupture in 25.1% [21].

Significant changes in signal intensity of the spinal cord, because of hemorrhage, contusion, or edema, are best seen in T2WI. Increased high-signal intensity (ISI) is a finding, often seen in SCIWORA patients. According to the study by Kulkarni et al, published in 1988, there are 3 imaging patterns of intramedullary spinal cord abnormalities (cord haemorrhage, oedema and mixed type) (Table 3) [63], and their prognostic value has been the focus of a number of reports [52, 64]. The rate of ISI in SCIWORA has been estimated at 92% to 94%, according to the studies by Machino et al and Ouchida et al [26, 27, 32].

In contrast to the pediatric patient population, negative MRI in adult patients with SCIWORA is questionable. One could argue that the strength of the magnetic field or the acquisition technique is responsible for not imaging small intramedullary signal changes. In general, the disclosure of technical information on image acquisition was insufficient, although selection and interpretation of the appropriate imaging technique are critical for the diagnosis of SCIWORA in adults [65]. Various scanning methods are available, and novel approaches may provide additional information, particularly regarding intramedullary signal changes. In fact, a study by Shen et al reported that Diffusion-weighted MRI (DWI) can be used as a method to assess the spinal cord integrity [36]. Patients with absence of intramedullary signal changes on conventional MRI presented high-intensity signals on DWI. It is therefore possible that this technique will help to further investigate patients with SCIWORA who are considered to have a negative MRI.

Prognosis

According to this review, 75% of the SCIWORA patients were reported to exhibit neurological improvement. Prognosis of SCIWORA patients depends on neurological status and MRI findings. The association between the severity of initial neurologic impairment and subsequent improvement is similar to the outcome characteristics of SCIs with radiologic abnormalities [66]. According to Neva et al, in-hospital mortality of all SCIWORA patients was 0.9%, increasing to 3.7% in patients with complete tetraplegia or paraplegia (ASIA A) [67]. In contrast, ASIA B to D patients were associated with a significantly better overall survival (mortality rate 0.2%). Martinez-Perez et al observed that, at one-year after SCIWORA, complete neurological recovery was achieved only in patients with incomplete neurological injury at admission [29]. Most SCIWORA adult patients were unable to return to work; however, the extent of the entire socioeconomic impact has to be clarified [31]. Maeda et al confirmed that prognosis of SCIWORA patients was mainly related to the injury mechanism, the spinal canal diameter, patient age, the injury degree, the presence of disk-ligament injury and the severity of the neurological syndrome [28]. In contrast, according to a recent study by Cao et al, the prognosis is not affected by preoperative neurologic status, age, gender, comorbidities and cause of injury [20].

Although it has been suggested that MRI findings are important prognostic indicators in SCIWORA patients [5, 21, 68], this scenario has not yet been fully clarified. A systematic review published in 2008 showed an association between radiological findings and clinical improvement and observed a more favorable prognosis in patients without cord injury in MRI [69]. In 2016, Boeze et al reported an association between imaging type and neurological outcome in adult SCIWORA patients, presenting a superior prognosis of patients with no detectable neuroimaging abnormalities over patients presenting with type II lesions. Moreover, patients with combined extraneural and intraneural abnormalities had a higher risk of persistent neurological impairment in comparison to those with isolated ex-

traneural abnormalities, followed by patients with intraneural abnormalities [19].

On the contrary, Boese et al found that the absence of MRI abnormalities was not predictive of a good outcome. According to their study, spinal cord edema was correlated with only partial cord symptoms and patients experienced complete motor and sensory remission within 24 hours. 37.5% of patients without spinal cord damage on MRI and 40% of patients with isolated soft tissue bulging into the spinal canal showed incomplete recovery [18]. Asan et al observed 4 patients without SCI on MRI, who did not have a favorable course [17]. Moreover, there was no significant correlation between the severities of the clinical findings, the clinical progression and the MRI cord abnormalities [17].

In addition, MRI provides valuable information on intramedullary signal changes and their longitudinal extension, which are well-documented prognostic factors after SCI. The outcome is worst in patients with cord disruption, poor in patients with cord haemorrhage and good in patients with cord oedema or normal cord [41]. Edema of the spinal cord obviously has the best prognosis, while the presence of hemorrhage makes the outcome less good, especially if it occupies more than 50% of the transverse diameter of the spinal cord [6]. Occupancy ratio and spinal cord high signal changes in MRI T2WI were associated with poor prognosis, according to Cao et al [20]. Grabb and Pang reported a definite association between abnormal MRI findings and the severity of neurological impairment, and they presented their classification to prognosticate SCIWORA based on MRI findings [68]: (1) worse if there is intramedullary hematoma occupying more than 50% of the cross-sectional area of the spinal cord, (2) intermediate if the contusions are minor and associated with edema, (3) better in patients where there is cord edema, and (4) the best when there are no changes in the spinal cord. After edema subsides, improvement can be seen in patients [68, 70]. According to Na et al, intramedullary lesion length on T2 images and spinal cord compression rate on T1 images had the most powerful effect on neurological improvement [30]. Neva et al suggested that more severe maximum spinal cord compression

correlated well with worse Frankel grade [31]. It is also reported that the extension of the pathologic signal to multiple levels has a poor prognosis [71]. In 2006, Boldin et al quantified this parameter, defining that a hemorrhagic lesion ≤ 4 mm in length is an important factor for incomplete SCI damage and better prognosis [72]. Consequently patients with extensive intramedullary lesions, particularly with bleeding areas, should be expected to have a poor outcome.

According to Machino et al, the presence of ISI may be associated with the preoperative neurologic status of the patient. The range of ISI was larger in severely paralyzed cases. Increased ISI is associated with symptom severity and neurological prognosis [26]. Ouchida et al found that, when comparing acute and delayed MRI results, there were significant differences in the prevalence rates of ISI. Delayed MRI findings provide more accurate information about symptom severity in comparison with acute findings, giving useful information about the state of the spinal cord [32].

Extraneural findings are generally less severe than intraneural findings. According to Boese et al, intraneural MRI abnormalities (type II) were associated with an inferior outcome when compared with patients without intraneural abnormalities. The combination of extraneural and intraneural lesions (Type IIc) was associated with a high risk of poor neurological outcome [19]. According to the study by Gupta et al, the best neurological recovery occurred in patients with disc herniation. This is not surprising as these patients had no spinal cord damage and surgical removal of the herniated disk resulted in neurological recovery. In these patients, MRI therefore has a definite prognostic value [5].

According to Martinez-Perez et al, disruption of either the ALL or the ligamentum flavum and larger lesions in the MRI have been noted as predictors of lack of neurological improvement. Shorter lesions and integrity of the ligamentum flavum were significantly associated with neurological improvement [29]. According to Machino et al, the presence of PVH may indeed be associated with the preoperative neurologic status of the patient. The range of PVH was larger in severely paralyzed cases [26]. Ouchida

et al found that, when comparing acute and delayed MRI results, there were significant differences in the prevalence rates of PVH [32]. Maeda et al concluded that the area of PVH had a significant negative correlation with the ASIA motor score, indicating that patients who had larger PVH tended to show severe paralysis. All these findings suggest that the soft-tissue injury at the time of trauma strongly affects the patients' neurological status [28].

Cervical ossification of the PLL is another risk factor for SCIWORA. The prevalence of cervical ossification of the PLL among SCI patients was higher than the general prevalence rate. Two possible mechanisms have been proposed. At the time of traumatic injury, the spinal cord under static compression of ossified PLL could be abruptly pinched by ossification mass, resulting in secondary SCI. Moreover, patients with ossified PLL have a narrow cerebrospinal fluid zone, resulting in a decrease of the buffering and protective potential of cerebrospinal fluid. In traumatic injuries, the traumatic force will be directly conducted to the spinal cord, which may induce a concussion of the spinal cord [20]. Cao et al stated that when patients with cervical ossification of the PLL had traumatic injury, cervical SCI without effective protective potential of cerebrospinal fluid may lead to a poor prognosis [20].

Treatment

Prompt management of SCIWORA is mostly empirical and is not based on randomized controlled trials. Initial treatment of SCIWORA is conservative and includes immobilization and corticosteroid therapy. Immobilization is initiated immediately after the injury, and includes hard collars, cervical or cervical-thoracic braces or thoracolumbar orthosis, for at least 3 months. All SCIWORA patients should abstain from any physical activity for at least 6 months. Immobilization of the spine continues until clinical examination becomes normal, and no instability is evident on MRI. IV steroid therapy is routinely administered, within 8 hours after injury, before the implementation of MRI, for the prevention of secondary injury [51]. Asan et al administered only methylprednisone to the 11 patients of their study [17].

Surgical management of SCIWORA patients is

indicated in cases of clear MRI evidence of persistent ligamentous instability and cord compression, along with worsening neurological symptoms or lack of improvement [51]. Anterior cervical decompression with fusion (ACDF) is the main surgical method for surgical management of adult cervical SCIWORA, which can relieve the symptoms of cervical spinal cord compression promoting the recovery of cervical spinal cord function [20, 33]. Posterior surgery is recommended only in cases of severe total cervical spinal stenosis. Surgical decompression of the spinal cord can restore topical blood circulation and reduce edema, thus reducing or normalizing the high signal intensity in the intramedullary region. Patients with high signal intensity in the intramedullary region in MRI T2WI can have good prognosis after surgery when they are at early stage of edema and demyelination [20, 37]. In case of surgical management, ACDF with Fidji cervical cage is a safe option for these patients, leading to significant functional improvement [23].

Surgical treatment can significantly improve the prognosis of SCIWORA patients with type IIc abnormalities. In the case series by Boese et al, surgical procedures were performed in cases with mixed extraneural and intraneural lesions. All the other patients were treated conservatively [19]. Wang et al concluded that timing of surgery (less than 3 months) was not significantly associated with neurologic recovery [38]. On the contrary, according to Qi et al, the optimal schedule of surgical treatment was 3-7 days after injury, which can significantly improve the short and long-term follow-up effects. Longer the time to surgery from the time of injury, the worse was the prognosis [33].

Rupture of the ALL is definite evidence of cervical disc injury. Intraoperative disc contrast injection during anterior cervical surgery can detect cervical disc rupture and determine the segment responsible for SCIWORA [40]. In comparison to percutaneous disc injection, intraoperative disc injection avoids the risk of iatrogenic injury during needle puncture. However, intraoperative disc contrast injection may prolong surgical time and is associated with increased X-ray exposure of the patients [40].

According to Martinez-Perez et al, type of treat-

ment (conservative vs surgical) does not have a significant impact on neurological outcome [29]. Na et al stated that adequate surgical decompression may have limited contribution to the recovery of neurological function [30]. Kawano et al observed that surgical management was not beneficial in comparison to conservative treatment, in SCIWORA patients [24]. Mazaki et al treated conservatively the SCIWORA patients with incomplete injury and had a good prognosis [73]. However, long-term follow-up showed that some patients were at enormous risk of secondary injury, with more severe neurological damage and poor prognosis [74]. Therefore, blind conservative treatment is also not recommended.

Conclusions

SCIWORA is a syndrome that defines posttraumatic SCI in patients with abnormal clinical neurological examination and apparently normal radiological findings in plain X-rays and CT. This syndrome most commonly affects children, but can also be found in adults, with a predilection for the cervical spine. In adults, most common causes are falls from height and motor vehicle accidents. The most common mechanism of injury is hyperflexion, of the cervical spine especially in patients with preexisting cervical spondylosis. In the present review, the total number of patients was 1418 (78.2% men) with a mean age of 53.5 years. According to the included studies of this review, the incidence of SCIWORA varies from 1.3% to 12%. 94.4% of the SCIWORA occurred in the cervical spine, while the rest 5.2% occurred in the thoracic spine. Diagnosis of SCIWORA includes histo-

ry and clinical examination, followed by plain X-rays and CT. Taking into consideration that plain radiological examination is normal, MRI is the gold standard in the diagnostic evaluation of these patients, not only for its ability to depict the injured spinal cord, but also its capacity to predict the outcome. In 14.2% of patients, no MRI abnormalities were detected, while 85.8% of patients had abnormal MRI scan results. Among them, 57% had extraneural, 36.3% had intraneural and 6.6% of patients had combined extraneural and intraneural MRI abnormalities. Initial treatment is conservative including immobilization for at least 3 months and steroid administration within 8 hours following injury. Surgical treatment, mostly ACDF, is indicated for patients with clear MRI evidence of spinal cord compression, ligamentous injury and instability, along with worsening neurological condition. However, there are studies which do not find any benefit of the surgical treatment over the conservative management. Prognosis of SCIWORA depends on the initial neurological deficit and magnitude of SCI on MRI; however, neurological improvement is expected in at least 75% of patients. Under the suspicion of SCIWORA, early MRI is recommended for definitive diagnosis while prompt neuroprotective measures have to be taken to prevent secondary SCI that may cause further neurological deterioration. More high-quality studies are needed to fully elucidate the optimal imaging and clinical approaches in the management of SCIWORA patients.

Conflict of interest

The authors declare no conflicts of interest.

References

1. El Masri WS, Kumar N. Traumatic spinal cord injuries. *Lancet*. 2011 Mar 19;377(9770):972-4.
2. Pang D, Wilberger JE, Jr. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg*. 1982 Jul;57(1):114-29.
3. Barnes R. Paraplegia in cervical spine injuries. *J Bone Joint Surg Br*. 1948 May;30B(2):234-44.
4. Hirsh LF, Duarte L, Wolfson EH. Thoracic spinal cord injury without spine fracture in an adult: case report and literature review. *Surg Neurol*. 1993 Jul;40(1):35-8.
5. Gupta SK, Rajeev K, Khosla VK, Sharma BS, Paramjit, Mathuriya SN, et al. Spinal cord injury without radiographic abnormality in adults. *Spinal Cord*. 1999 Oct;37(10):726-9.
6. Tewari MK, Gifti DS, Singh P, Khosla VK, Mathuriya SN, Gupta SK, et al. Diagnosis and prognostication of adult spinal cord injury without radiographic abnormality using magnetic resonance imaging: analysis of 40 patients. *Surg Neurol*. 2005 Mar;63(3):204-9; discussion 9.
7. Tator CH, Duncan EG, Edmonds VE, Lapczak LI, Andrews DF. Changes in epidemiology of acute spinal cord injury from 1947 to 1981. *Surg Neurol*. 1993 Sep;40(3):207-15.
8. Como JJ, Samia H, Nemunaitis GA, Jain V, Anderson JS, Malangoni MA, et al. The misapplication of the term spinal cord injury without radiographic abnormality (SCIWORA) in adults. *J Trauma Acute Care Surg*. 2012 Nov;73(5):1261-6.
9. Tator CH. Spine-spinal cord relationships in spinal cord trauma. *Clin Neurosurg*. 1983;30:479-94.
10. Saruhashi Y, Hukuda S, Katsuura A, Asajima S, Omura K. Clinical outcomes of cervical spinal cord injuries without radiographic evidence of trauma. *Spinal Cord*. 1998 Aug;36(8):567-73.
11. Dreizin D, Kim W, Kim JS, Boscak AR, Bodanapally UK, Munera F, et al. Will the Real SCIWORA Please Stand Up? Exploring Clinicoradiologic Mismatch in Closed Spinal Cord Injuries. *AJR Am J Roentgenol*. 2015 Oct;205(4):853-60.
12. Kothari P, Freeman B, Grevitt M, Kerslake R. Injury to the spinal cord without radiological abnormality (SCIWORA) in adults. *J Bone Joint Surg Br*. 2000 Sep;82(7):1034-7.
13. Bhatoe HS. Cervical spinal cord injury without radiological abnormality in adults. *Neurol India*. 2000 Sep;48(3):243-8.
14. Diaz JJ, Jr., Aulino JM, Collier B, Roman C, May AK, Miller RS, et al. The early work-up for isolated ligamentous injury of the cervical spine: does computed tomography scan have a role? *J Trauma*. 2005 Oct;59(4):897-903; discussion -4.
15. Kasimatis GB, Panagiotopoulos E, Megas P, Matzaroglou C, Gliatis J, Tyllianakis M, et al. The adult spinal cord injury without radiographic abnormalities syndrome: magnetic resonance imaging and clinical findings in adults with spinal cord injuries having normal radiographs and computed tomography studies. *J Trauma*. 2008 Jul;65(1):86-93.
16. Barnett I, Malik N, Kuijjer ML, Mucha PJ, Onnela JP. EndNote: Feature-based classification of networks. *Netw Sci (Camb Univ Press)*. 2019 Sep;7(3):438-44.
17. Asan Z. Spinal Cord Injury without Radiological Abnormality in Adults: Clinical and Radiological Discordance. *World Neurosurg*. 2018 Jun;114:e1147-e51.
18. Boese CK, Nerlich M, Klein SM, Wirries A, Ruchholtz S, Lechler P. Early magnetic resonance imaging in spinal cord injury without radiological abnormality in adults: a retrospective study. *J Trauma Acute Care Surg*. 2013 Mar;74(3):845-8.
19. Boese CK, Müller D, Bröer R, Eysel P, Krischek B, Lehmann HC, et al. Spinal cord injury without radiographic abnormality (SCIWORA) in adults: MRI type predicts early neurologic outcome. *Spinal Cord*. 2016 Oct;54(10):878-83.
20. Cao B, Li F, Tang Y, Jia L, Chen X. Risk Factors for Poor Prognosis of Spinal Cord Injury without Radiographic Abnormality Associated with Cervical Ossification of the Posterior Longitudinal Ligament. *Biomed Res Int*. 2022;2022:1572341.
21. Guo H, Liu J, Qi X, Ning G, Zhang H, Li X, et al. Epidemiological characteristics of adult SCIWORA in Tianjin, China: a preliminary study. *Eur Spine J*. 2012 Jan;21(1):165-71.
22. Hendey GW, Wolfson AB, Mower WR, Hoffman JR.

- Spinal cord injury without radiographic abnormality: results of the National Emergency X-Radiography Utilization Study in blunt cervical trauma. *J Trauma*. 2002 Jul;53(1):1-4.
23. Huang SL, Yan HW, Wang KZ. Use of Fidji cervical cage in the treatment of cervical spinal cord injury without radiographic abnormality. *Biomed Res Int*. 2013;2013:810172.
 24. Kawano O, Ueta T, Shiba K, Iwamoto Y. Outcome of decompression surgery for cervical spinal cord injury without bone and disc injury in patients with spinal cord compression: a multicenter prospective study. *Spinal Cord*. 2010 Jul;48(7):548-53.
 25. Liu Q, Zhao J, Yu H, Ma X, Wang L. Early MRI finding in adult spinal cord injury without radiologic abnormalities does not correlate with the neurological outcome: a retrospective study. *Spinal Cord*. 2015 Oct;53(10):750-3.
 26. Machino M, Yukawa Y, Ito K, Nakashima H, Kanbara S, Morita D, et al. Can magnetic resonance imaging reflect the prognosis in patients of cervical spinal cord injury without radiographic abnormality? *Spine (Phila Pa 1976)*. 2011 Nov 15;36(24):E1568-72.
 27. Machino M, Ando K, Kobayashi K, Ota K, Morozumi M, Tanaka S, et al. MR T2 image classification in adult patients of cervical spinal cord injury without radiographic abnormality: A predictor of surgical outcome. *Clin Neurol Neurosurg*. 2019 Feb;177:1-5.
 28. Maeda T, Ueta T, Mori E, Yugue I, Kawano O, Takao T, et al. Soft-tissue damage and segmental instability in adult patients with cervical spinal cord injury without major bone injury. *Spine (Phila Pa 1976)*. 2012 Dec 1;37(25):E1560-6.
 29. Martinez-Perez R, Munarriz PM, Paredes I, Cotrina J, Lagares A. Cervical Spinal Cord Injury without Computed Tomography Evidence of Trauma in Adults: Magnetic Resonance Imaging Prognostic Factors. *World Neurosurg*. 2017 Mar;99:192-9.
 30. Na BR, Seo HY. Adult Spinal Cord Injury without Major Bone Injury: Effects of Surgical Decompression and Predictors of Neurological Outcomes in American Spinal Injury Association Impairment Scale A, B, or C. *J Clin Med*. 2021 Mar 6;10(5).
 31. Neva MH, Roeder CP, Felder U, Kiener B, Meier W, Perler M, et al. Neurological outcome, working capacity and prognostic factors of patients with SCIWORA. *Spinal Cord*. 2012 Jan;50(1):78-80.
 32. Ouchida J, Yukawa Y, Ito K, Katayama Y, Matsumoto T, Machino M, et al. Delayed Magnetic Resonance Imaging in Patients With Cervical Spinal Cord Injury Without Radiographic Abnormality. *Spine (Phila Pa 1976)*. 2016 Aug 15;41(16):E981-E6.
 33. Qi C, Xia H, Miao D, Wang X, Li Z. The influence of timing of surgery in the outcome of spinal cord injury without radiographic abnormality (SCIWORA). *J Orthop Surg Res*. 2020 Jun 16;15(1):223.
 34. Qi C, Cao J, Xia H, Miao D, Liu Y, Guo J, et al. Does cervical curvature affect neurological outcome after incomplete spinal cord injury without radiographic abnormality (SCIWORA): 1-year follow-up. *J Orthop Surg Res*. 2022 Jul 26;17(1):361.
 35. Sharma S, Singh M, Wani IH, Sharma N, Singh D. Adult Spinal Cord Injury without Radiographic Abnormalities (SCIWORA): Clinical and Radiological Correlations. *J Clin Med Res*. 2009 Aug;1(3):165-72.
 36. Shen H, Tang Y, Huang L, Yang R, Wu Y, Wang P, et al. Applications of diffusion-weighted MRI in thoracic spinal cord injury without radiographic abnormality. *Int Orthop*. 2007 Jun;31(3):375-83.
 37. Tan J, Hu F, Ou J, Su X, Liu J. Analysis of the Curative Effect and Prognostic Factors of Anterior Cervical Surgery for Spinal Cord Injury without Radiographic Abnormalities. *Evid Based Complement Alternat Med*. 2022;2022:6836966.
 38. Wang Y, Xue Y, Zong Y, Ding H, Li Z, He D, et al. Treatment of Atypical Central Cord Injury Without Fracture or Dislocation. *Orthopedics*. 2015 Jun;38(6):e524-8.
 39. Yaqoob Hakim S, Gamal Altawil L, Faidh Ramzee A, Asim M, Ahmed K, Awwad M, et al. Diagnosis, management and outcome of Spinal Cord Injury without Radiographic Abnormalities (SCIWORA) in adult patients with trauma: a case series. *Qatar Med J*. 2021;2021(3):67.
 40. Zhang JD, Xia Q. Role of Intraoperative Disc Contrast Injection in Determining the Segment Responsible

- for Cervical Spinal Cord Injury without Radiographic Abnormalities. *Orthop Surg.* 2015 Aug;7(3):239-43.
41. Zhu F, Yao S, Ren Z, Telemacque D, Qu Y, Chen K, et al. Early durotomy with duroplasty for severe adult spinal cord injury without radiographic abnormality: a novel concept and method of surgical decompression. *Eur Spine J.* 2019 Oct;28(10):2275-82.
 42. Pang D, Pollack IF. Spinal cord injury without radiographic abnormality in children—the SCIWORA syndrome. *J Trauma.* 1989 May;29(5):654-64.
 43. Crooks F, Birkett AN. Fractures and dislocations of the cervical spine*. *British Journal of Surgery.* 2005;31(123):252-65.
 44. Schneider RC, Cherry G, Pantek H. The syndrome of acute central cervical spinal cord injury; with special reference to the mechanisms involved in hyperextension injuries of cervical spine. *J Neurosurg.* 1954 Nov;11(6):546-77.
 45. Holmes G. The Goulstonian Lectures ON SPINAL INJURIES OF WARFARE: Delivered before the Royal College of Physicians of London. *Br Med J.* 1915 Nov 27;2(2865):769-74.
 46. Roberts TT, Leonard GR, Cepela DJ. Classifications In Brief: American Spinal Injury Association (ASIA) Impairment Scale. *Clin Orthop Relat Res.* 2017 May;475(5):1499-504.
 47. Yamazaki M, Mochizuki M, Ikeda Y, Sodeyama T, Okawa A, Koda M, et al. Clinical results of surgery for thoracic myelopathy caused by ossification of the posterior longitudinal ligament: operative indication of posterior decompression with instrumented fusion. *Spine (Phila Pa 1976).* 2006 Jun 1;31(13):1452-60.
 48. Fredrickson MD. Acute spinal cord injury management. *J Trauma.* 2007 Jun;62(6 Suppl):S9.
 49. Parizel PM, van der Zijden T, Gaudino S, Spaepen M, Voormolen MH, Venstermans C, et al. Trauma of the spine and spinal cord: imaging strategies. *Eur Spine J.* 2010 Mar;19 Suppl 1(Suppl 1):S8-17.
 50. Djang WT. Radiology of acute spinal trauma. *Crit Care Clin.* 1987 Jul;3(3):495-518.
 51. Atesok K, Tanaka N, O'Brien A, Robinson Y, Pang D, Deinlein D, et al. Posttraumatic Spinal Cord Injury without Radiographic Abnormality. *Adv Orthop.* 2018;2018:7060654.
 52. Bozzo A, Marcoux J, Radhakrishna M, Pelletier J, Goulet B. The role of magnetic resonance imaging in the management of acute spinal cord injury. *J Neurotrauma.* 2011 Aug;28(8):1401-11.
 53. Lammertse D, Dungan D, Dreisbach J, Falci S, Flanders A, Marino R, et al. Neuroimaging in traumatic spinal cord injury: an evidence-based review for clinical practice and research. *J Spinal Cord Med.* 2007;30(3):205-14.
 54. Goradia D, Linnau KF, Cohen WA, Mirza S, Hallam DK, Blackmore CC. Correlation of MR imaging findings with intraoperative findings after cervical spine trauma. *AJNR Am J Neuroradiol.* 2007 Feb;28(2):209-15.
 55. Hayashi K, Yone K, Ito H, Yanase M, Sakou T. MRI findings in patients with a cervical spinal cord injury who do not show radiographic evidence of a fracture or dislocation. *Paraplegia.* 1995 Apr;33(4):212-5.
 56. Shimada K, Tokioka T. Sequential MRI studies in patients with cervical cord injury but without bony injury. *Paraplegia.* 1995 Oct;33(10):573-8.
 57. Boese CK, Lechler P. Spinal cord injury without radiologic abnormalities in adults: a systematic review. *J Trauma Acute Care Surg.* 2013 Aug;75(2):320-30.
 58. Mhuircheartaigh NN, Kerr JM, Murray JG. MR imaging of traumatic spinal injuries. *Semin Musculoskelet Radiol.* 2006 Dec;10(4):293-307.
 59. Pérez-López C, Isla A, Gómez Sierra A, Budke M. Cervical epidural hematoma without fracture in a patient with ankylosing spondylitis. A case report. *J Neurosurg Sci.* 2004 Jun;48(2):91-4; discussion 4.
 60. Grant GA, Mirza SK, Chapman JR, Winn HR, Newell DW, Jones DT, et al. Risk of early closed reduction in cervical spine subluxation injuries. *J Neurosurg.* 1999 Jan;90(1 Suppl):13-8.
 61. Benzel EC, Hart BL, Ball PA, Baldwin NG, Orrison WW, Espinosa MC. Magnetic resonance imaging for the evaluation of patients with occult cervical spine injury. *J Neurosurg.* 1996 Nov;85(5):824-9.
 62. Pang D. Spinal cord injury without radiographic abnormality in children, 2 decades later. *Neurosurgery.* 2004 Dec;55(6):1325-42; discussion 42-3.

63. Kulkarni MV, Bondurant FJ, Rose SL, Narayana PA. 1.5 tesla magnetic resonance imaging of acute spinal trauma. *Radiographics*. 1988 Nov;8(6):1059-82.
64. Ramón S, Domínguez R, Ramírez L, Paraira M, Olona M, Castelló T, et al. Clinical and magnetic resonance imaging correlation in acute spinal cord injury. *Spinal Cord*. 1997 Oct;35(10):664-73.
65. Ehara S, Shimamura T. Cervical spine injury in the elderly: imaging features. *Skeletal Radiol*. 2001 Jan;30(1):1-7.
66. Wilson JR, Cadotte DW, Fehlings MG. Clinical predictors of neurological outcome, functional status, and survival after traumatic spinal cord injury: a systematic review. *J Neurosurg Spine*. 2012 Sep;17(1 Suppl):11-26.
67. Varma A, Hill EG, Nicholas J, Selassie A. Predictors of early mortality after traumatic spinal cord injury: a population-based study. *Spine (Phila Pa 1976)*. 2010 Apr 1;35(7):778-83.
68. Grabb PA, Pang D. Magnetic resonance imaging in the evaluation of spinal cord injury without radiographic abnormality in children. *Neurosurgery*. 1994 Sep;35(3):406-14; discussion 14.
69. Yucesoy K, Yuksel KZ. SCIWORA in MRI era. *Clin Neurol Neurosurg*. 2008 May;110(5):429-33.
70. Dare AO, Dias MS, Li V. Magnetic resonance imaging correlation in pediatric spinal cord injury without radiographic abnormality. *J Neurosurg*. 2002 Jul;97(1 Suppl):33-9.
71. Schaefer DM, Flanders AE, Osterholm JL, Northrup BE. Prognostic significance of magnetic resonance imaging in the acute phase of cervical spine injury. *J Neurosurg*. 1992 Feb;76(2):218-23.
72. Boldin C, Raith J, Fankhauser F, Haunschmid C, Schwantzer G, Schweighofer F. Predicting neurologic recovery in cervical spinal cord injury with postoperative MR imaging. *Spine (Phila Pa 1976)*. 2006 Mar 1;31(5):554-9.
73. Mazaki T, Ito Y, Sugimoto Y, Koshimune K, Tanaka M, Ozaki T. Does laminoplasty really improve neurological status in patients with cervical spinal cord injury without bone and disc injury? A prospective study about neurological recovery and early complications. *Arch Orthop Trauma Surg*. 2013 Oct;133(10):1401-5.
74. Bosch PP, Vogt MT, Ward WT. Pediatric spinal cord injury without radiographic abnormality (SCIWORA): the absence of occult instability and lack of indication for bracing. *Spine (Phila Pa 1976)*. 2002 Dec 15;27(24):2788-800.

Cite this
Paper as



Siatos N, Benetos IS, Evengelopoulos DS, Vlamis J, Evangelopoulou M. Imaging and clinical approaches in the management of patients with spinal cord injury without radiographic abnormality (SCIWORA). *Acta Orthop Trauma Hell* 2024; 75(1): 9-23.

Significance of quadrilateral plate in surgical treatment of complex bi-columnar acetabular fractures: descriptive analysis through three cases.

Fotios V. Nikolopoulos, Ioannis V. Papachristos, Konstantina Solou, Georgios Gourtzelidis, Dimitrios Samaras

General Hospital of Piraeus "Tzaneio", Hellenic NHS, Piraeus, Greece

Abstract

Complex acetabular fractures affecting both columns remain one of the hardest challenges to trauma orthopaedic surgeons with high morbidity and often poor outcomes. Treatment dilemmas arise especially in both column fractures where the fracture line dissociates acetabulum in different directions and sizes resulting in quadrilateral plate (Quad) fragments of various sizes. This plate serves as a vertical girder-wall structure for the acetabulum. Order of fixation along with selection of its associated surgical approach are the cornerstones of operative strategy in such injuries and are driven by various factors one of the most important being quadrilateral plate involvement. A traditional rule of thumb dictates to first fix the column that can be addressed easier and more directly. Then the second column can be addressed. As the quad is the connection girder between the two columns, the fragmental condition of this plate, is one of the most decisive factors influencing decision making. The fracture line which crosses the quad plate is crucial and if there is a multifragmentary component it dictates the chosen plate. We will try to illustrate, discuss and explore this topic exemplifying three difficult cases dealt with three approaches: Ilioinguinal (ILO), Stoppa-Anterior Intrapelvic (AIP)-ILO modification and Kocher-Langenbeck (K-L) either in isolation or combined. Pearls, pitfalls and lessons learned are offered in a vivid illustrative way.

Keywords: quadrilateral, plate, column, acetabulum, approach

Introduction

Both column acetabular fractures can be classified according to Young and Burgess classification as CM type (Combined Mechanism)¹, according to Tile classification as C2 or C3² and according to AO/OTA modification of the Letournel-Judet classifica-

tion as C2.2, or C2.3, or C3.2, or C3.3³ Typical mechanisms causing pelvic disruption are: traffic injuries (60%), falls from height (30%) and crush injury under heavy weights (10%).⁴ Armamentarium of surgical approaches utilized in surgical fixation of such fractures includes: Stoppa or Anterior Intrapelvic

Corresponding
Author

Ioannis V. Papachristos Consultant in Trauma and Orthopaedics, M.D., M.Sc., MRCS, FEBOT General Hospital of Piraeus "Tzaneio", Hellenic NHS, Piraeus, Greece Zani & Afentouli Avenues 18536 Piraeus, Greece
Email: ioannispapachristos@gmail.com Tel: +306975852104

Approach (AIP), Ilioinguinal (ILO), AIP-ILO modification (the AIP in conjunction with the first or the two lateral windows of ilioinguinal) and Kocher-Langenbeck (K-L) approaches. Commonly presenting problem in such cases is how to efficiently reduce the posterior column along with the quadrilateral plate, which both relate directly to the proper sequence of approaches and selection of the component to be reduced first. The role of quadrilateral plate has recently started to be an area of interest.⁵ However its significance had been recognized since 1997 when Perry et al. stated that it functions as a third column rather than a connection between anterior and posterior columns.⁶ We have realised that the key role of the Quad gives partially or sometimes entirely, the answer from where to start and how (which exposure). Apart from that, it is well known that pelvic stability depends on the ability of osseoligamentous structures to withstand physiologic stress without abnormal deformation. Posterior Sacroiliac ligaments are a key vertical stabilizer maintaining the sacrum in its normal position in the pelvic ring.⁷

In this short case series we illustrate the reduction and fixation sequence of complex bi-columnar acetabular fractures striving to extract useful conclusions, lessons and recommendations about this difficult topic.

Case 1

A seventy-two-year-old man fell from height and presented with a both column-fracture (Figure 1). Further workup revealed a complex bi-column acetabular fracture with posterior wall involvement and more specifically a transtectal fracture because the fracture line crossed the superior weight bearing surface into the acetabulum, comprising the fracture with the worst prognosis (Figures 2 and 3).⁸ Ideally complex pelvic and acetabular fractures should be addressed through one single exposure; therefore as the major lesion is posteriorly we tried to rectify the problem utilizing Kocher-Langenbeck (K-L) approach. We planned to fix the posterior column firstly and secondly the Quad. In figure 4a is shown how with an asymmetric clamp we reduced from posteriorly the Quad holding in place with a Schanz

screw the posterior column. Notice how the two long screws in Fig 5, (red arrows) keep in place the anterior column from posteriorly. Trochanteric osteotomy was very helpful in order to provide access to placing two screws in the upper portion of the acetabulum wall.

Case 2

A forty-five-year-old man fell from height sustaining a two-column acetabular fracture with posterior wall divided in three major pieces (Figure 6). CT scan showed that quad was detached and that we had to deal with a transverse transtectal fracture, high anterior column fracture with multifragmentary iliac wing and that quad fracture line did not extend into major sciatic foramen (Figures 7,8).

Initially, the Anterior Intrapelvic Approach (AIP)-Ilioinguinal modification (AIP-ILM), more specifically the Stoppa approach with the two lateral windows of the IL approach, were used. There was great difficulty, reducing the quad from anterior because the posterior column (with the quad on it) was displaced far posteriorly, too deep into the true pelvis and neither the colinear, nor other clamps could pull the posterior column back to its original position to the pelvic brim. We decided not to make any more attempts to reduce the quad with the posterior column to its initial place from anterior due to fear of uncontrollable bleeding. We succeeded only at an almost 2 cm reduction of the posterior column, with the quad on, and compromised accepting a 2 cm gap. A suprapectineal plate was applied and a screw from anterior to posterior was used to stabilize the posterior column to its new position (red arrows in Figure 9). Also, from the 1st window of the IL approach, a big plate applied to the iliac wing. The distance from the pelvic brim to the quad was left almost 2 cm "open" and fresh frozen allograft was applied. Then, the patient was switched to a lateral decubitus position and via K-L approach we attempted to address posterior wall and column. Now even though the posterior wall was fixed easily with two spring plates the posterior column was found "locked" due to the anterior fixation and did not permit the any posterior column movement at all. The gap of the unreduced column



Figure 1: Case 1 Xray AP view depicting a left both column acetabular fracture



Figure 3: Case 1 3D Recon. **a)** Transverse transtectal acetabular fracture, **b)** Fracture line posteriorly through Quad up to major sciatic foramen

(with the quad on it) could be palpated also from posteriorly and fresh frozen allograft was interposed. This combined approach procedure took approximately 6 hours surgical time. Despite our initial reservations radiographic and clinical follow up six months post-op showed a good outcome with a happy and fully functional patient (Figure 10).

Case 3

A 27-year-old woman was involved as passenger in a motorcycle accident when their motorbike crushed on the concrete fringe of the pavement.

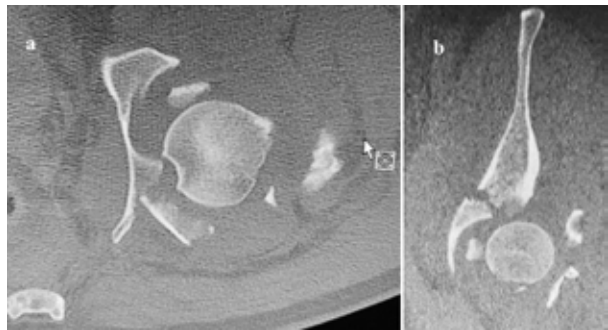


Figure 2: Case 1 CT scan. **a)** axial view showing bi-columnar fracture with significant comminution and head posterior dislocation, **b)** Sagittal view showing transverse fracture

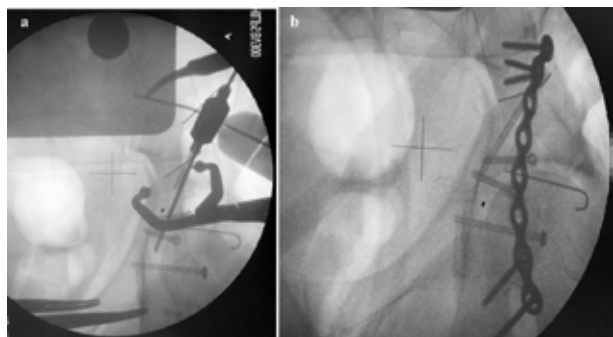


Figure 4: Case 1 Intraoperative c-arm views. **a)** asymmetric clamp reducing Quad from posteriorly plus Schanz screw holding it in place, **b)** satisfactory reduction of Quad and fixation with posterior column plate. Very small piece of cartilage fixed in situ with a needle

Contrary to case 2 now we had to deal with a transverse juxtatectal fracture (fracture line crossed the top of cotyloid fossa) along with a high anterior column fracture and comminuted iliac wing (Figure 11). Quadrilateral plate was disrupted in two pieces but in continuity with iliac bone near major sciatic foramen as the fracture juxtatectal. Sacroiliac joint was indeed affected but with intact posterior elements hence we decided not to apply any treatment to the sacrum. Quad surface was totally separated from anterior and the posterior columns and was lying as an independent piece in the middle of the



Figure 5: Case 1 Post-op AP Xray. Red arrows showing independent screws from posterior to anterior column. Notice that trochanteric flip osteotomy was restored with 3 cannulated screws



Figure 6: Case 2 Xray AP view. Comminuted both column acetabular fracture

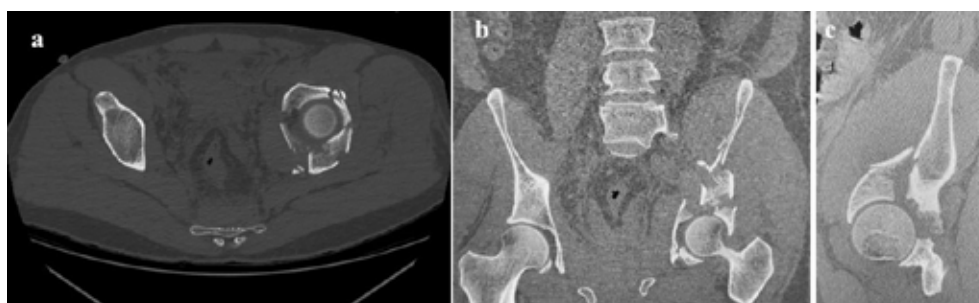


Figure 7: Case 2 CT scan. a) axial view-quad is detached, b) coronal view-transtectal transverse fracture. iliac wing is also fractured, c) sagittal view

upper and lower pelvis. In Fig.11 b-c, CT/scan reveals that the anterior ligamentous complex is torn but the posterior one is stable. Nevertheless, the gap of the SIJ isn't more than 2 mm anteriorly. The multifragmentary of the sacral makes the SIJ plating inappropriate and on the other hand SIJ screws would have yielded a small advantage to the overall patient condition.

We opted to fix the posterior column via K-L approach first, contrary to previous case number 2. The fracture seemed to be Juxtatectal which means that the fracture line crosses the top of the cotyloid fossa of the acetabulum. In this way, it cut the quad almost in the middle in a transverse plane. So it appeared to be easier to reduce the posterior column from behind. Two plates applied at the posterior column (Fig 12a) via K-L approach for better

stability. When we realized that quad and anterior column were displaced (red arrows in Fig. 12a) we turned the patient supine to fix them through ilioinguinal approach (ILA) and the iliac wing from its 1st window. In post-operative x-rays the femoral head did not appear to be concentrically located in the acetabulum and further investigation with CT scan revealed that although metalwork was appropriately applied, part of the quad was malreduced resulting in a spur entering the cotyloid fossa and preventing the femoral head from sitting normally (Figure 13). Six weeks later, the patient was brought again to theatres for further exploration of the acetabulum. Using modified Hardinge approach, (anterolateral) we performed hip dislocation and smoothed the residual quad bone from the acetabulum. In one year follow up (Figure 14) x-rays in multiple views

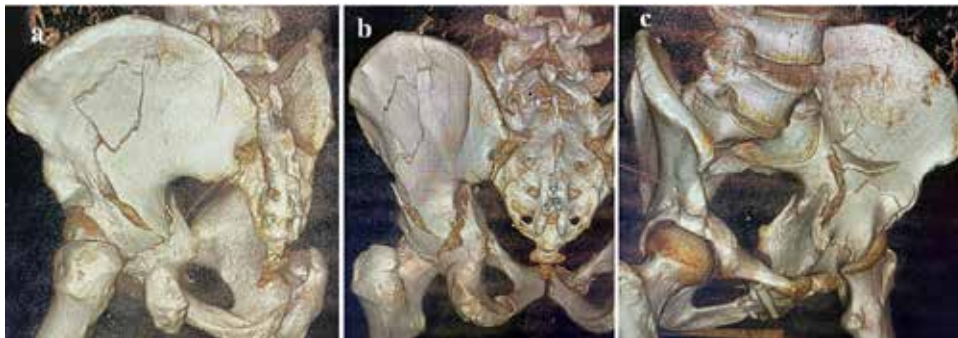


Figure 8: Case 2 3D Recon. *a) High anterior column fracture. Iliac wing multifragmentary starting from acetabular roof to iliac crest, b) quad fracture line does not extend into major sciatic foramen, c) posterior column completely separated from ilium with part of Quad*



Figure 9: Case 2 Post-op Xray. *a) AP view. 2 spring plates for posterior wall, a plate in ilium and suprapectineal plate in quad from Stoppa approach, b) Obturator oblique view. Red arrow shows screw from anterior to posterior column, c) Iliac oblique view, d) clinical photo showing both approaches K-L and AIP-ILM*

prove that the fracture is healed without osteoarthritis and that the patient enjoys a fully pain-free sporting lifestyle.

Discussion

Increased controversy regarding the optimal way of treatment of complex associated bi-column acetabular fractures exists. Traditionally as an effective way it is suggested to deal with the posterior column first via a posterior approach and use a clamp from the back to the front to reduce and hold in position the anterior column before the final fixation with screws.⁹ That is what we have done in case number 1. Summarising our approaches we have used K-L approach alone in case 1, AIP-ILA modification and then K-L approach in case 2 and K-L and then ILA alone in case number 3. In all three cases we treated transverse fractures (Juxtatectal or transtectal) and fractures of posterior and anterior column. Quadrilateral surface has been highlighted as an important factor

in such fractures and some authors attempted to understand these fractures by classifying quad fractures into in two categories relating to two areas formed by an imaginary line from iliopubic eminence to ischial tuberosity.¹⁰ Again this does not assist too much when we have to deal with such horrific injuries. In case 2 we also had fracture of the posterior wall of the acetabulum and we had the expectation that if we could reduce the quad from anteriorly first, then the posterior column would come closer to acetabulum and especially the posterior wall pieces would follow so that the pieces of the wall and the posterior column could be fixed in a more accurate manner. Unfortunately this hypothesis was not proven true. Even though the anterior approach is the approach of choice for the quad (and the anterior column), it was not possible to grasp the quad because it had fallen very deep to the minor pelvis, almost near to the retroperitoneal area, very deep beside the bladder. All attempts with the collin-



Figure 10: Case 2 6-months post-op. *a) AP view, b) Outlet view c) Obturator oblique view, d) Iliac oblique view, e) patient walking independently without crutches, f, g) good hip ROM*

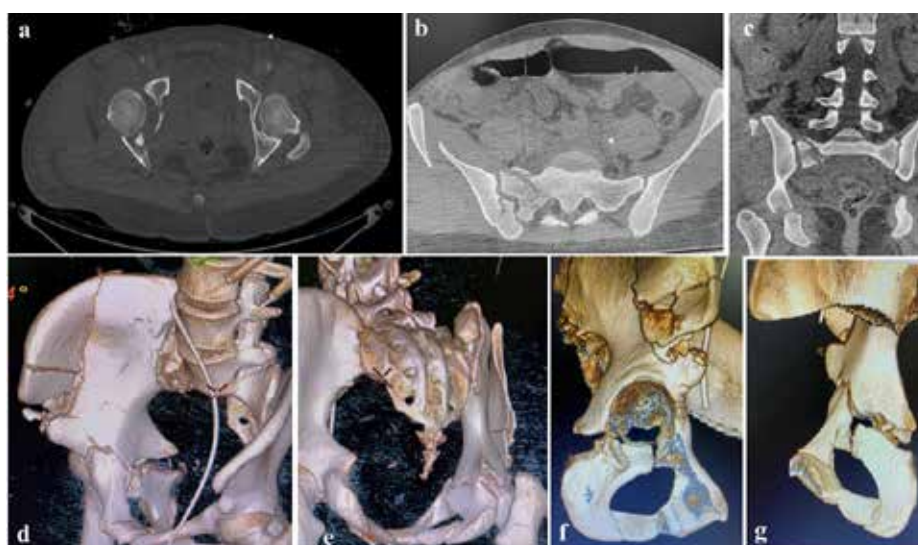


Figure 11: Case 3 CT scan. *a) axial view- Quad is floating, b) comminuted SIJ with intact posterior elements and fractured ilium, c) coronal view showing SIJ disruption and posterior column displacement, d) 3D Recon quad in 2 major pieces, e) posterior column with part Quad, f) Juxtatectal fracture-quad near major sciatic foramen stable in continuity with comminuted iliac bone, g) quad in a large piece but has lost its continuity with iliac bone, acetabular brim in front and ischial tuberosity-posterior column behind*

ear clamp, asymmetric forceps etc. were ineffective. Only a small reduction was achieved. Then, at the posterior exposure it was difficult to reduce the posterior column because the osteosynthesis which proceeded from the front prevented any

movements. Only the posterior wall was able to be fixed easily. In cases 1 and 3 starting from posteriorly with K-L approach the overall obstacle of the unreduced posterior column was surpassed. In case 3 especially where an anterior approach



Figure 12: Case 3. *a)* Intra-op c-arm views. With K-L posterior column was fixed but red arrow shows unreduced anterior column & quad, *b)* Post-op AP view, *c)* Post-op obturator oblique view, *d)* Post-op iliac oblique view. Multiple plates through both (K-L + ILA). Femoral head seems non-congruent inside acetabulum

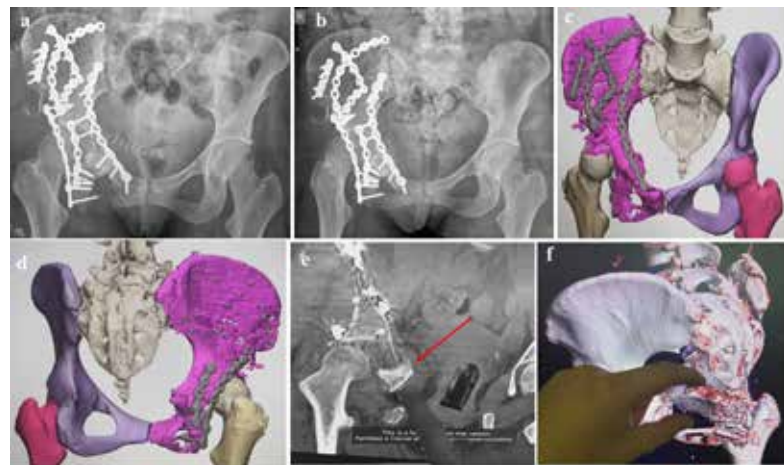


Figure 13: Case 3. *a, b)* malreduced hip was confirmed in serial views and xrays, *c, d)* metalwork found in good position, *e)* CT scan coronal view. Finger and red arrow showing spur of malreduced rotated Quad invading cotyloid fossa and narrowing joint forcing head to sit abnormally, *f)* 3D Recon finger showing quad malrotation

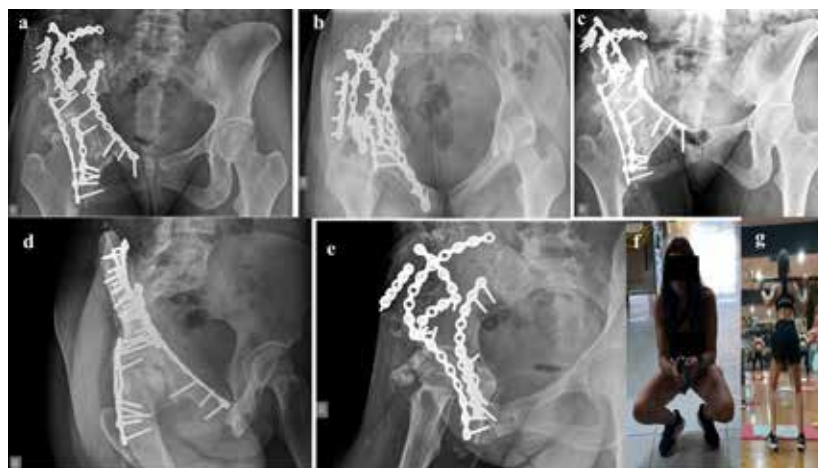


Figure 14: Case 3. 1 year clinical + radiological follow up. No signs of OA and head sits well *a)* AP Xray, *b)* Inlet, *c)* Outlet, *d)* Obturator oblique, *e)* Iliac oblique, *f, g)* fully functional patient doing sports

was also needed, the quad was mobile and reduced more easily but it was not restored ideally. Therefore, it seems that we cannot safely offer a recipe “one size fits all” but rather a relevant recommendation. Our perceived algorithm that we want to share is: Operate as soon as possible, fix the posterior column first using your most famil-

iar approach but especially when you have low or middle posterior column fractures use K-L approach without trochanteric osteotomy. Perform trochanteric osteotomy only if you have high posterior column fractures or the fracture line bisects the upper part of the acetabulum. Then, close and fix the quad and the anterior column using AIP-

ILO modification or ILO approach alone. With combined approaches restrict initial fixation from posterior to a minimum to avoid any potential conflict when you go from the front. In case when posterior column is impossible to be reduced via posterior approach, then abandon it and switch to a fixation from anterior solely. Due to increased morbidity and risks, we do not advocate switching from posterior to anterior and then back to posterior to finalize the posterior column. Apart from infection risk, this approach does not seem

to be cost-effective, is troublesome and only pelvic experts could potentially consider it in exceptional circumstances. In our hospital, the latest five years we perform two complex pelvic cases on a monthly basis utilizing a great variety of modern approaches and fixation methods and needless is to say that such complex injuries should only be dealt by specialized referral centers.

Conflict of interest

The authors declare no conflicts of interest.

References

1. AOTRAUMA, "Fractures of the Pelvis and Acetabulum, Principles and Methods of Management-Fourth Edition" Marvin Tile, David L Helfet, et al. Vol 1-Pelvis, 2015, Table 1.3-1, p.49
2. "Rockwood and Greens' Fractures in Adults" Paul Tornetta III, et al, Ninth Edition, 2020 Wolters Kluwer, Vol 2, Section three "Axial Skeleton, Pelvis and Acetabulum", Ch.49 Pelvic Ring Injuries, Table: Young and Burgess Classification, P.1976
3. AOTRAUMA, "Fractures of the Pelvis and Acetabulum, Principles and Methods of Management-Fourth Edition" Marvin Tile, David L Helfet, et al. Vol 2-Acetabulum, 2015,
4. Fig 2.3-28a-c & Fig 2.3-29a-c p.469
5. Schmal H, Markmiller M, Mehlhorn AT, Sudkamp NP. Epidemiology and outcome of complex pelvic injury. *Acta Orthop Belg* 2005;71(1):41-47.
6. Butler BA, Stover MD, Sims SH. The Quadrilateral Plate in Acetabular Fracture Surgery: What Is It and When Should It Be Addressed? *J Am Acad Orthop Surg.* 2021;29: e109-e115.
7. Perry DC, DeLong W. Acetabular fractures. *Orthop Clin North Am.* 1997; 28:405-417.
8. Kellam JF, Mayo KA. Pelvic ring disruption. In: *Skeletal trauma.* 3rd ed. Philadelphia, Pa: Saunders; 1052-1108.
9. AOTRAUMA, "Fractures of the Pelvis and Acetabulum, Principles and Methods of Management-Fourth Edition" Marvin Tile, David L Helfet, et al. Vol 2-Acetabulum, 2015, Fig 2.3-16a-c, p.460-461
10. Mitchell PM, Shaath MK, Routt MC. Clamp assisted reduction of the transverse acetabular fracture with anterior column screw fixation and posterior plating: A technical trick and case series. *J Orthop Trauma.* 2021;35(12): e521-4
11. Yang Y, Yi M, Zou C, Yan Z, Yan X, Fang Y. Mapping of 238 quadrilateral plate fractures with three-dimensional computed tomography. *Injury.* 2018; 49:1307-1312.

Cite this paper as



Nikolopoulos FV, Papachristos IV, Solou K, Gourtzelidis G, Samaras D. Significance of quadrilateral plate in surgical treatment of complex bi-columnar acetabular fractures: descriptive analysis through three cases. *Acta Orthop Trauma Hell* 2024; 75(1): 24-31.

Calcified tendonitis of the rotator cuff. A review of this common shoulder pathology

Chrissovalantis Tsikrikas,¹ Ioannis K. Triantafyllopoulos²

¹Postgraduate Program “Metabolic Bone Diseases”,
National and Kapodistrian University of Athens, School of Medicine
²Fifth Department of Orthopaedics, HYGEIA Hospital, Athens, Greece

Abstract

Calcified tendonitis is an acute or chronic painful condition due to the presence of calcium phosphate crystals within or around the tendons that form the rotator cuff.

The precise mechanism leading to the deposition of calcium crystals remains unclear. Factors that appear to contribute to this pathology are metabolic diseases (diabetes, thyroid hormone abnormalities), medication factors (e.g., corticosteroids), genetic predisposition of the individual, and overuse injuries.

Clinically, calcified tendonitis is usually asymptomatic. However, in some cases it is characterized by severe pain, which is more addressed in the morning. Many times, the intensity of the pain is so high that it inhibits movements, resulting in stiff shoulder joints. A simple x-ray is the most appropriate imaging method to diagnose the disease. As far as concerns the course of the disease, in many cases, an automatic resorption of calcium is observed while the symptoms recede. The precise mechanisms associated with absorption are unknown.

Numerous treatment options have been reported in the literature that are applied on a case-by-case basis and vary in efficacy.

Keywords: Calcified Tendonitis, Shoulder Stiffness, Shoulder Treatment, Rotator Cuff, Shoulder Pain.

Introduction

In 1872, Duplay was the first who described calcified tendonitis as a “painful shoulder periarthritis” [1]. In 1934, Codman was the first time who found that deposits were growing either inside or around the tendon of the rotator cuff, contrary to the earlier theory that the deposits had been developing in the subacromial bursa. [2] (**Figure 1**)

Shoulder tendonitis is the most frequently occur-

ring painful disorder, characterized by the presence of calcified deposits in the rotor cuff tendons. The tendon of the supraspinatus muscle is the one mainly affected; rarely we find these problems in the tendon of the infraspinatus and the subscapularis muscle (**Table 1**) [3,4].

The prevalence of the disease is 2.7% in people without symptoms. It mainly attacks people aged from 30 to 60 years. Women appear to have the dis-

Corresponding
Author

Chrissovalantis Tsikrikas,
tsikrikasval@yahoo.gr
tel. +30-6932704449

ease at a higher rate than men. Indeed, contrary to what is expected, the disease mainly attacks people who do sedentary work [5,6].

Calcified tendonitis is treated with the use of non-steroidal anti-inflammatory (NSAIDs), ultrasound-guided needling (UGN), physiotherapy, subacromial outburst, and extra-corporeal shock wave therapy (ESWT) [7,8]. In cases where conservative treatment does not work, surgical removal of calcium deposits is recommended [9,10].

Histopathology

According to Uthoff, the evolutionary course of calcified tendonitis is divided into three stages: the pre-calcific, the calcifying, and the post-calcific [11].

In the pre-calcification stage, numerous factors stimulate the transformation of tendon cells into chondrocytes. The one that follows, the calcification stage, is subdivided into three different phases: the formation, the phase of rest and the absorption one. In general, the disease progresses with a pattern. Initially, amorphous calcium phosphate is deposited. Then, angiogenesis is developed and the calcium crystals are absorbed and the collagenosis of fibroblast damage takes place during the post-calcification stage [11].

Samples collected from the affected area either had the form of sandy hard mass or liquid-like toothpaste, or were an amorphous mass consisting of many small round or oval particles [12]. The material of these deposits, it is recognized as calcium carbonate (apatite) [13]. Carbonate apatite has been further classified as type A and B. Classification is based on positions occupied by carbonate ions in the molecule [14]. Studies on the chemical composition of calcification have found that, in different proportions, both types of apatite carbonate coexisted during the formation, resting and absorption phases [15].

Histochemical studies have shown the presence of extracellular vesicles, also known as microvesicles (MVs), in the area of the rotator cuff calcification. MVs vesicles are normally required for the formation of cartilage, bone and dentin. The attempt to correlate the specific finding with the pathogenesis of calcifying tendonitis has led to

discovering that the deposition of calcium crystals is the result of an active and complex process involving the participation of alkaline phosphatase, proteins and enzymes, [11,16,17]. However, the expression of alkaline phosphatase and the presence of MVs are controversial in the international bibliography. On the other hand, it has been shown that despite osteopontin (OPN) is found in the formation phase its role has not been yet clarified. However, IL-1 and IL-18, as well as the inflammatory factor NLRP3, appear to play a critical role in absorption [16, 17]. The exact mechanism of the specific pathology has not yet revealed its data and thus all the information has not been clarified in its whole.

Clinical symptoms

The main clinical symptom is pain. Pain, in this condition, is considered to be a complication, as the latter remains primarily asymptomatic in most cases [18]. When calcified tendonitis becomes symptomatic, pain is acute and usually does not reflect below the middle part of the arm [19]. In the acute phase, the disease is so painful that it restricts shoulder's mobility. In the chronic or subacute phase, the pain may be acute but generally allows movement [20].

The occurrence of pain is due either to the inflammatory response to local chemical pathology or to direct mechanical irritation [21]. Four mechanisms that stimulate the onset of pain have been described: the chemical irritation of the tissue by calcium, the pressure of the tissues due to swelling, the impact on the subacromial bursa due to deposition and the chronic stiffness of the glenohumeral joint in a patient's effort to avoid severe pain [22].

Peptide P (neurotransmitter) is involved in the transmission of pain caused by $A\delta$ / C fiber stimulation. It is expressed in the small aesthetic neurons of the tissues of the affected area. It

Is released from sensory neurons and plays an important role in mediating neurogenic inflammation [23]. Researchers who studied the relation between the amount of P in the subacromial bursa and the pain in patients with calcified tendonitis, have discovered an increase in the number of immunoreac-

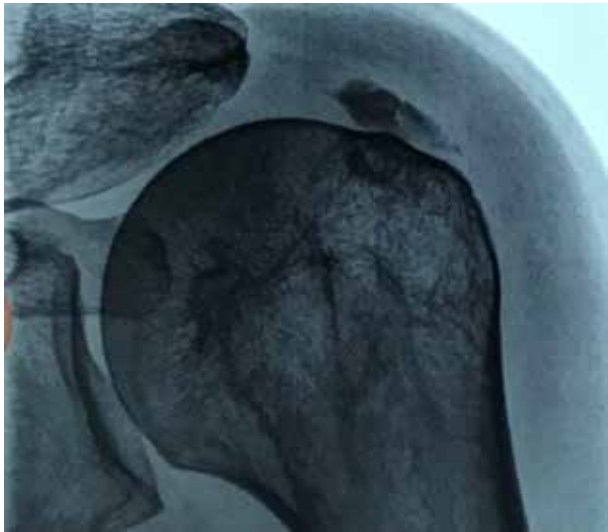


Figure 1. Shoulder plain x-ray showing calcification of the distal end of supraspinatus tendon.

tive nerve fibers in the tissues around calcification. These fibers were mainly located around the blood vessels, suggesting an active role of the peptide in regulating subsequent inflammation. They also hypothesize that both the mechanical (subacromial impingement) and the chemical agents (subacromial bursitis) could be a source of harmful stimuli, which cause increased amounts of substance P in the affected nerves. Therefore, it is found that inflammation of the subacromial bursa is the complication associated with pain of the shoulder girdle in patients with calcified tendinitis [23].

Imaging

The preferred imaging methods for the identification of calcified tendinitis is plain radiography (Xray) and ultrasound (US), mainly because calcium deposits are easily recognizable in both of the processes. In X-rays, calcium deposits appear to be homogeneous, amorphous, without rough distribution, allowing the differentiation from ectopic osteogenesis [24]. Ultrasound (US) is particularly effective for the diagnosis of calcified tendonitis, as it detects other pathological conditions that co-exist in the area (e.g. tendon injuries) [25]. High resolution ultrasound in combination with color Doppler allows the distinction between the state of formation and the absorption of calcium [26].



Figure 2. Needle aspiration of soft calcification of infraspinatus tendon during the initial inflammatory phase. The location of the calcification is identified easily above the skin by palpation of a bulky painful inflammatory area or by U/S guidance. (I.K.T. personal archive)

On the other hand, computed tomography offers excellent resolution for detecting calcium deposition as a compact or amorphous high-density hob; however, its cost and exposure limit its use. Finally, magnetic tomography should not be used as a first imaging form because the deposits appear as vague low signal regions at T1 and T2 and may not be recognized. In general, CT and MRI should only be selected for controversial cases [27].

Treatment

Conservative management of patients with calcified tendonitis of the shoulder is always the first treatment option. According to the literature, it utilizes the following means: non-steroidal anti-inflammatory (NSAIDs) to relieve pain, physiotherapy to maintain shoulder joint range of motion,



Figure 3. Arthroscopic identification of the calcified tissue (a). Removal of the degenerated tendon (b). Repair of the tendon with re-attachment to its footprint (c) (I.K.T. personal archive)

extracorporeal shock waves therapy (ESWT), PRP (Platelet Rich Plasma), ultrasound-guided needling (UGN) and Surgical Treatment [28].

NSAIDs

Non-Steroidal Anti-Inflammatory Drugs are commonly used as the primary choice in the acute phase of calcified tendonitis of the rotator cuff both due to analgesia they offer and for their anti-inflammatory properties. Reducing pain is a key factor for maintaining the shoulder's motion range. As far as the Non-Steroidal Anti-Inflammatory Drugs concern, there is some thought give on the fact that they may adversely affect healing of the tendon-bone adhesion [29,30].

Determining the adverse effects of non-steroidal anti-inflammatory drugs, in terms of healing proves to be rather complicated; this is a mixture of interconnections between the mechanisms found in cellular level, including inhibitors of cyclooxygenase-1 (COX-1), the cyclooxygenase-2 (COX-2) and ways they are affected by the time of administration of the drugs [31].

The existing bibliography does not fully clarify whether the use of non-steroidal anti-inflammatory drugs adversely affects healing or not.

Corticosteroid injections

Cortisone injections may be applied mainly sub-acromially and rarely intra-articularly. It should be noted that steroid infusion during the acute phase is often chosen. However, the works on this sub-

ject reflect the ambiguity of scientific community. Some studies showed that this method has positive results [32], other works label it as ineffective [33] while it has also been mentioned that it negative consequences because absorption of deposits is inhibited [34].

Finally, surgical removal is selected only in cases where conservative treatment will not produce the desired results.

Physiotherapy

The main role of physiotherapy in calcified tendonitis of the rotator cuff is to reduce pain in the affected and peripheral area of the lesion, as well as to maintain the range of motion of the shoulder joint.

Shock waves in the treatment of the disease are described below so no reference will be made to this chapter.

Passive mobilization by a physiotherapist or a continuous passive movement machine (CPM) device during the acute phase is an important tool for maintaining range of motion.

Myofascial therapy and trigger point therapy play an important role in reducing pain. [35] Natural remedies such as LASER, microwave diathermy, and TENS significantly help reduce pain and expand range of motion. In the second year, it is really important to strengthen the muscles of the shoulder girdle to maintain shoulder stability. [36]

It is significant to train the patient to avoid movements that cause subachromial friction, as well as to learn exercises that maintain the range of motion.

Table 1. The frequency of calcification in each tendon of the rotator cuff	
Prevalence rates of calcification in the anatomical structures of the rotator cuff [4]	
Supraspinatus tendon	63%
Supraspinatus Subscapularis tendon	20%
Subscapularis tendon	3%
Infraspinatus tendon	7%
Subacromial Bursa	7%

Ultrasound-Guided Needling and Decompression

Ultrasound-guided needling was first performed using a radiograph by Comfort et al. [37]. Farin et al. first attempted to use ultrasound for saline wash [38]. (Figure 2)

Ultrasound-guided needling (UGN) is a treatment that can be performed easily with local anesthesia and at a low cost. In 2014, a study was published that involved 121 patients who applied the UGN technique. It was found that within three months of applying the method the results were satisfactory [39]. In a 2013 study, patients were divided into two groups, the first of which used UGN wash and corticosteroid infusion, and the second only corticosteroid infusion. Both groups showed improvement, but patients in the first group performed better [40]. However, a more recent comparative study concluded that the two groups had no significant statistically variance [41].

A systematic review of the bibliography on the efficacy of the UGN method in calcified tendonitis of the shoulder shows that studies do not provide strong evidence for the efficacy of the method. In addition, there is an extensive variation in the results, indicating that more reliable studies is a necessity [41]. In another systematic review, the UGN method is suggested as an alternative treatment when the initial conservative treatment has failed [42]

Extracorporeal Shock Waves Therapy

Extracorporeal shock waves therapy (ESWT) has been used to treat calcified tendinitis since the 1990s. This method is becoming increasingly popular and new research is being published on its dosing, duration and the number of sessions. Shock waves therapy can be generated by electromagnetic or piezoelectric devices. The categorization of shock

waves in relation to energy appears as follows:

- (a) low energy (below 0.08 mJ / mm²)
- (b) medium energy (0.08-0.28 mJ / mm²)
- (c) high energy (0.28-0.60 mJ / mm²) [43].

In a research study in which the control group received placebo treatment, it was found that the results were better in the shock wave treatment group [44]. Various doses of ESWT energy have been indicated occasionally for the treatment of calcified tendonitis, and most authors have described positive clinical results with low and medium energy waves [43,47,48]. In a study comparing a dose of 0.3 mJ / mm² versus two doses of 0.2 mJ / mm², the former proved to be more effective [44]. Another study revealed that a dosage of 0.20 mJ / mm² is more effective than 0.10 mJ / mm² [45]. Albert et al. found positive results with high doses of ESWT. However, in this case the follow-up of patients lasted only three months and no significant decrease in the size of the deposition was observed on radiographs [46].

A comparative study between the UGN and ESWT methods showed that the UGN technique had better radiological and clinical results, although both groups showed improvement compared to the original findings [49]. In a 2018 research study, 66 patients with LCT were given a needle-guided xylocaine infusion followed by a five-session ESWT session over a month. The results showed clinical improvement in patients and disappearance of deposition at the end of the fifth treatment [50].

PRP (Platelet Rich Plasma)

The use of PRP in the treatment of calcified tendonitis of the rotator cuff, based on the existing literature, does not appear to have positive results [51] although it should be noted that current research so far is not as extended as need.

Surgical Treatment

Arthroscopic treatment of rotator cuff calcified tendonitis has been described by many researchers as a treatment with positive results [52,53,54,55] (**Figure 3**) However, it is a fact that many heterogeneous techniques have been used as well as short-term comparative studies that have not convinced the scientific community which is the best technique that can effectively tackle the problem.

In the international bibliography there is a controversy as far as concerns the need for complete removal of calcium. Several researchers appear to associate successful outcomes of surgery with complete absence of calcareous tendons [57,58,59]. However, the view that total removal of calcium deposits is not necessary is strongly supported, since cellular absorption begins from the surgical incision site of the affected tendon. Several studies even confirm that there was no difference in functional outcome between patients with calcified residues and patients who had been fully calcified [54,55,56].

The issue of surgical repair of the tendon also causes controversy. Researchers argue that the tendon should not be repaired in all cases, as it results in natural healing [57]. However, researchers who studied a group of patients who underwent complete removal of calcium deposits without tendon

repair after 24 months of ultrasound monitoring [56] found that 31% of those patients developed defective healing in the rotator cuff.

The necessity of acromioplasty is another controversial issue regarding the surgical treatment of shoulder calcified tendonitis. Many studies have considered acromioplasty as a routine treatment for rotator cuff calcifications [60,61]. However, other studies have not confirmed the benefit of additional subacromial decompression [52]. Marder et al. performed a comparative study between a group of 25 patients who underwent arthroscopic calcification, and a group of 25 patients who underwent calcification and simultaneous acromioplasty. Finally, acromioplasty was found beneficial because it delayed both the elimination of pain and ultimately the recovery [63].

Conclusion

Calcified tendonitis of the rotator cuff, although commonly found in clinical practice, has many areas yet to be discovered in terms of the factors that play a significant role in its formation. At the same time, there is great controversy about the most appropriate therapeutic method to achieve full functional recovery of the patient.

Conflict of interest

The authors declare no conflicts of interest.

References

1. Duplay S. De la peri-arthritis scapulo-humerale et des raideurs de l'épaule qui en sont la consequence. Arch Gen Med. 1872 ;20:513-542.
2. Codman EA. The Shoulder: Rupture of the Supraspinatus Tendon and Other Lesions in or about the Subacromial Bursa. Boston: Thomas Todd Co; 1934.
3. Speed CA, Hazleman BL. Calcific tendinitis of the shoulder. N Engl J Med. 1999; 340:1582-1584.
4. Loew M, Jurgowski W, Mau HC, et al. Treatment of calcifying tendinitis of rotator cuff by extracorporeal shock waves: A preliminary report. J Shoulder Elbow Surg. 1995;4:101-106.
5. Rathbun JB, Macnab I. The microvascular pattern of the rotator cuff. J Bone Joint Surg Br. 1970; 52:540-553.
6. Booth RE, Jr, Marvel JP., Jr Differential diagnosis of shoulder pain. Orthop Clin North Am. 1975; 6:353-379.
7. Gosens T, Hofstee DJ. Calcifying tendinitis of the shoulder: advances in imaging and management. Curr Rheum Rep. 2009; 11:129-134.
8. Farin PU, Räsänen H, Jaroma H, et al. Rotator cuff calcifications: treatment with ultrasound-guided percutaneous needle aspiration and lavage. Skeletal Radiol. 1996; 25:551-554.
9. Serafini G, Sconfienza LM, Lacelli F, et al. Rotator cuff calcific tendinitis: short-term and 10-year outcomes

- after two-needle US-guided percutaneous treatment. Non randomized controlled trial. *Radiology*. 2009; 252:157-164.
10. del Cura JL, Torre I, Zabala R, et al. Sonographically guided percutaneous needle lavage in calcific tendinitis of the shoulder: short- and long-term results. *AJR Am J Roentgenol*. 2007;189:W128-134.
 11. Uthoff HK, Sarkar K, Maynard JA. Calcifying tendinitis: a new concept of its pathogenesis. *Clin Orthop Relat Res*. 1976; 118:164-168.
 12. Oliva F, Via AG, Maffulli N. Physiopathology of intratendinous calcific deposition. *BMC Med*. 2012; 10:95.
 13. Hamada J, Tamai K, Ono W, Saotome K. Does the nature of deposited basic calcium phosphate crystals determine clinical course in calcific periarthritis of the shoulder? *J Rheumatol*. 2006; 33:326- 332.
 14. Penel G, Leroy G, Rey C, Bres E. MicroRaman spectral study of the PO₄ and CO₃ vibrational modes in synthetic and biological apatites. *Calcif Tissue Int*. 1998; 63:475-481.
 15. Chiou HJ, Hung SC, Lin SY, Wei YS, Li MJ. Correlations among mineral components, progressive calcification process and clinical symptoms of calcific tendonitis. *Rheumatology*. 2010;49:548-555.
 16. Archer R, Bayley J, Archer C, Ali S. Cell and matrix changes associated with pathological calcification of the human rotator cuff tendons. *J Anat*. 1993;182:1-12.
 17. Gohr CM, Fahey M, Rosenthal AK. Calcific tendonitis: a model. *Connect Tissue Res*. 2007;48:286-291.
 18. Merolla G, Dave AC, Paladini P, Campi F, Porcellini G (2014) Ossifying tendinitis of the rotator cuff after arthroscopic excision of calcium deposits: two case reports and literature review. *J Orthop Traumatol* 15 [Epub ahead of print]
 19. Bayam L, Ahmad MA, Naqui SZ, Chouhan A, Funk L. Pain mapping for common shoulder disorders. *Am J Orthop*. 2011;40(7):353-358.
 20. Spivey JL, Carrell TM. Severe pain in the shoulder with no history of trauma. *Calcific tendinitis*. *JAAPA*. 2009;22(4):59-60.
 21. Hurt G, Baker CL., Jr Calcific tendinitis of the shoulder. *Orthop Clin North Am*. 2003;34:567-575.
 22. Neer CS., II. Less frequent procedures. In: Neer CS II, editor. *Shoulder reconstruction*. Philadelphia: WB Saunders; 1990. pp. 421-485.
 23. Gotoh M, Hamada K, Yamakawa H, Inoue A, Fukuda H. Increased substance P in subacromial bursa and shoulder pain in rotator cuff diseases. *J Orthop Res*. 1998;16(5):618-621.
 24. Gosens T, Hofstee DJ. Calcifying tendinitis of the shoulder: advances in imaging and management. *Curr Rheumatol Rep*. 2009;11(2):129-134.
 25. Le Goff B, Berthelot JM, Guillot P, Glémarec J, Maugars Y. Assessment of calcific tendonitis of rotator cuff by ultrasonography: comparison between symptomatic and asymptomatic shoulders. *Joint Bone Spine*. 2010;77:258-263.
 26. Martinoli C, Bianchi S, Prato N, Pugliese F, Zamorani MP, Valle M, Derchi LE. US of the shoulder: non-rotator cuff disorders. *RadioGraphics*. 2003;23:381-401.
 27. Flemming DJ, Murphey MD, Shekitka KM, Temple HT, Jelinek JJ, Kransdorf MJ. Osseous involvement in calcific tendinitis: a retrospective review of 50 cases. *AJR Am J Roentgenol*. 2003;181:965-972.
 28. Ogon P, Suedkamp NP, Jaeger M, Izadpanah K, Koesler W, Maier D. Prognostic factors in nonoperative therapy for chronic symptomatic calcific tendinitis of the shoulder. *Arthritis Rheum*. 2009;60(10):2978-2984. doi: 10.1002/art.24845. [PubMed] [CrossRef]
 29. Brown KM, Saunders MM, Kirsch T, Donahue HJ, Reid JS. Effect of COX-2-specific inhibition on fracture-healing in the rat femur. *J Bone Joint Surg Am*. 2004;86-a:116-123.
 30. Connizzo BK, Yannascoli SM, Tucker JJ, et al. The detrimental effects of systemic Ibuprofen delivery on tendon healing are time-dependent. *Clin Orthop Relat Res*. 2014;472:2433-2439.
 31. Gerstenfeld LC, Al-Ghawas M, Alkhiary YM, et al. Selective and nonselective cyclooxygenase-2 inhibitors and experimental fracture-healing. Reversibility of effects after short-term treatment. *J Bone Joint Surg Am*. 2007;89:114-125. [PubMed] [Google Scholar]
 32. Uthoff H, Sarkar K, Maynard J. Calcifying tendinitis: a new concept of its pathogenesis. *Clin Orthop Relat Res*. 1976;118:164-168.

33. Noel E, Carillon Y, Gaillard T, Bouvier M. Needle aspiration irrigation in calcifying tendinitis of rotator cuff. In: Gazielly DF, Gleyze PT, editors. *The cuff*. Paris: Elsevier; 1997. pp. 152-157.
34. Tillander B, Franzen LE, Karlsson MH, Norlin R. Effect of steroid injections on the rotator cuff: an experimental study in rats. *J Shoulder Elbow Surg*. 1999;8(3):271-274. doi: 10.1016/S1058-2746(99)90141-6.
35. G. Fryer, L. Hodgson. The effect of manual pressure release on myofascial trigger points in the upper trapezius muscle. *J. Bodyw. Mov. Ther.*, 9 (4) (2005), pp. 248-255
36. Haik, M.N.; Alburquerque-Sendin, F.; Moreira, R.F.; Pires, E.D.; Camargo, P.R. Effectiveness of physical therapy treatment of clearly defined subacromial pain: a systematic review of randomised controlled trials. *Br. J. Sports Med*. 2016, 50, 1124-1134,
37. Comfort TH, Arafles R. Barbotage of the shoulder with image-intensified fluoroscopic control of needle placement for calcified tendinitis. *Clin Orthop Relat Res*. 1978;135:171-178.
38. Farin PU, Jaroma H, Soimakallio S. Rotator cuff calcifications: treatment with US-guided technique. *Radiology*. 1995;195(3):841-843. doi: 10.1148/radiology.195.3.7754018.
39. Castillo-González FD, Ramos-Álvarez JJ, Rodríguez-Fabián G, González-Pérez J, Calderón- Montero J. Treatment of the calcific tendinopathy of the rotator cuff by ultrasound-guided percutaneous needle lavage. Two years prospective study. *Muscles Ligaments Tendons J*. 2014;4(2):220-225.
40. de Witte PB, Selten JW, Navas A, Nagels J, Visser CP, Nelissen RG, Reijnen M. Calcific tendinitis of the rotator cuff: a randomized controlled trial of ultrasound-guided needling and lavage versus subacromial corticosteroids. *Am J Sports Med*. 2013;41(7):1665-1673.
41. De Witte, P. B., Kolk, A., Overes, F., Nelissen, R. G. H. H., & Reijnen, M. (2017). Rotator Cuff Calcific Tendinitis: Ultrasound-Guided Needling and Lavage Versus Subacromial Corticosteroids: Five-Year Outcomes of a Randomized Controlled Trial. *The American Journal of Sports Medicine*, 45(14), 3305-3314.
42. Vignesh KN, McDowall A, Simunovic N, Bhandari M, Choudur HN. Efficacy of ultrasound-guided percutaneous needle treatment of calcific tendinitis. *AJR Am J Roentgenol*. 2015;204(1):148-152.
43. Yi-Cheng Wu, Wen-Chung Tsai, Yu-Kung Tu, Tung-Yang Yu. Comparative Effectiveness of Nonoperative Treatments for Chronic Calcific Tendinitis of the Shoulder: A Systematic Review and Network Meta-Analysis of Randomized Controlled Trials. *Archives of Physical Medicine and Rehabilitation* 2017;98:1678-92.
44. Rompe JD, Kirkpatrick CJ, Kullmer K, Schwitalle M, Kirschek O. Dose-related effects of shock waves on rabbit tendo Achillis: a sonographic and histological study. *J Bone Jt Surg Br*. 1998;80(3):546-552.
45. Farr S, Sevelde F, Mader P, Graf A, Petje G, Sabeti-Aschraf M. Extracorporeal shockwave therapy in calcifying tendinitis of the shoulder. *Knee Surg Sports Traumatol Arthrosc*. 2011;19(12):2085-2089.
46. Ioppolo F, Tattoli M, Di Sante L, Attanasi C, Venditto T, Servidio M, Cacchio A, Santilli V. Extracorporeal shock-wave therapy for supraspinatus calcifying tendinitis: a randomized clinical trial comparing two different energy levels. *Phys Ther*. 2012;92(11):1376-1385.
47. Albert JD, Meadeb J, Guggenbuhl P, Marin F, Benkalfate T, Thomazeau H, Chalès G. High-energy extracorporeal shock-wave therapy for calcifying tendinitis of the rotator cuff: a randomised trial. *J Bone Jt Surg Br*. 2007;89(3):335-341.
48. Hsu CJ, Wang DY, Tseng KF, Fong YC, Hsu HC, Jim YF. Extracorporeal shock wave therapy for calcifying tendinitis of the shoulder. *J Shoulder Elbow Surg Br*. 2008;17(1):55-59.
49. Daecke W, Kusnierczak D, Loew M. Long-term effects of extracorporeal shockwave therapy in chronic calcific tendinitis of the shoulder. *J Shoulder Elbow Surg*. 2002;11(5):476-480
50. Kim YS, Lee HJ, Kim YV, Kong CG. Which method is more effective in treatment of calcific tendinitis in the shoulder? Prospective randomized comparison between ultrasound-guided needling and extracorporeal shock wave therapy. *J Shoulder Elbow Surg*. 2014;23(11):1640-1646.

51. Verhaegen, F., Brys, P., & Debeer, P. (2016). Rotator cuff healing after needling of a calcific deposit using platelet-rich plasma augmentation: a randomized, prospective clinical trial. *Journal of Shoulder and Elbow Surgery*, 25(2), 169-173.
52. Pakos, E., Gkiatas, I., Rakkas, G., Papadopoulos, D., Gelalis, I., Vekris, M., & Korompilias, A. (2018). Calcific deposit needling in combination with extracorporeal shock wave therapy (ESWT): A proposed treatment for supraspinatus calcified tendinopathy. *SICOT-J*, 4, 45.
53. El Shewy MT. Arthroscopic removal of calcium deposits of the rotator cuff: a 7-year follow-up. *Am J Sports Med*. 2011; 39:1302-1305.
54. Jacobs R, Debeer P. Calcifying tendinitis of the rotator cuff: functional outcome after arthroscopic treatment. *Acta Orthop Belg*. 2006; 72:276-281.
55. Oliva F, Via AG, Maffulli N. Physiopathology of intratendinous calcific deposition. *BMC Med*. 2012; 10:95.
56. Seil R, Litzenburger H, Kohn D, Rupp S. Arthroscopic treatment of chronically painful calcifying tendinitis of the supraspinatus tendon. *Arthroscopy*. 2006; 22:521-527. [PubMed]Hurt G, Baker CL. Calcific tendinitis of the shoulder. *Orthop Clin North Am*. 2003; 34:567-575.
57. Jerosch J, Strauss JM, Schmiel S. Arthroscopic treatment of calcific tendinitis of the shoulder. *J Shoulder Elbow Surg*. 1998; 7:30-37.
58. Rizzello G, Franceschi F, Longo UG, et al. Arthroscopic management of calcific tendinopathy of the shoulder—do we need to remove all the deposit? *Bull NYU Hosp Joint Dis*. 2009; 67:330-333.
59. DE Witte PB, Selten JW, Navas A, et al. Calcific tendinitis of the rotator cuff: a randomized controlled trial of ultrasound-guided needling and lavage versus subacromial corticosteroids. *Am J Sports Med*. 2013;41:1665-1673.
60. Seyahi A, Demirhan M. Arthroscopic removal of intraosseous and intratendinous deposits in calcifying tendinitis of the rotator cuff. *Arthroscopy*. 2009;25:590-596.
61. Maier D, Jaeger M, Izadpanah K, Bornebusch L, Suedkamp NP, Ogon P. Rotator cuff preservation in arthroscopic treatment of calcific tendinitis. *Arthroscopy*. 2013; 29:824-831.
62. Marder RA, Heiden EA, Kim S. Calcific tendonitis of the shoulder: is subacromial decompression in combination with removal of the calcific deposit beneficial? *J Shoulder Elbow Surg*. 2011; 20:955- 960.

Cite this
paper as



Tsikrikas C, Triantafyllopoulos IK. Calcified tendonitis of the rotator cuff. A review of this common shoulder pathology. *Acta Orthop Trauma Hell* 2024; 75(1): 32-40.

Shoulder instability: a brief review

Spyridon Manthas¹, Ioannis Kotsalis², Lampros Oikonomou²

¹ Department of Orthopaedics, "Hygeias Melathron" TYPET's General Clinic, Mutual Health Fund of National Bank of Greece Personnel (TYPET), Greece

² First Department of Orthopaedics, General Hospital of Athens G. Gennimatas, Greece

Abstract

Shoulder instability is a condition in which the humeral head partially or completely dislocates from the glenoid fossa. This can occur due to a traumatic injury (traumatic instability), repetitive strain on the joint, or generalized ligamentous laxity (atraumatic instability). In the majority of cases, glenohumeral instability is associated by a labral tear which prevents the humeral head from fitting properly into the glenoid fossa, causing it to shift out of place. Anterior shoulder dislocations comprise the majority of cases of glenohumeral instability.

Symptoms of shoulder instability may include a feeling of looseness or instability in the joint, pain or discomfort in the shoulder, weakness or loss of strength, and a sensation of the shoulder "popping out" or "slipping." In severe cases, the humeral head may completely dislocate from the glenoid fossa, causing intense pain and disability. Diagnosis of shoulder instability usually involves a thorough physical exam and imaging studies such as x-rays, computed tomography (CT) and magnetic resonance imaging (MRI).

Treatment of shoulder instability depends on the severity of the instability and the underlying cause. Conservative treatment for shoulder instability may include rest, ice, physical therapy, and anti-inflammatory medication. Physical therapy is especially important in cases of multidirectional instability, as strengthening exercises can help improve stability in the joint. If conservative treatment fails to relieve symptoms, surgery may be necessary. Absolute indications of surgical management are contradictory. Operative treatment is reserved after failed conservative management, recurrent dislocation at a young age, irreducible dislocation, open dislocation, post-reduction instability of the shoulder and first-time dislocation in young elite athletes. Surgical options for shoulder instability include arthroscopic procedures and open surgeries. Arthroscopic procedures are less invasive and may have fewer complications, but may not be appropriate for all types of instability. The choice of surgery depends on the underlying cause of the instability, the patient's age and activity level, and other factors. For traumatic instability, the most common surgical procedure is an arthroscopic Bankart repair, where the torn labrum is reattached to the glenoid fossa using sutures or anchors. In cases of atraumatic instability, surgery may involve tightening the capsule and ligaments around the joint, or transferring a portion of the coracoid process to the anterior aspect of the glenoid (Latarjet procedure).

Recovery from shoulder stabilization may take several months, and may involve a period of immobilization, followed by physical therapy to regain strength and range of motion in the joint. Patients should avoid activities that put stress on the shoulder, such as lifting heavy weights or participating in contact sports. Overall, shoulder instability can be a debilitating condition that affects many people, especially those involved in sports or other activities.

Keywords: Shoulder, glenohumeral, instability.

Corresponding
Author

Manthas S. Department of Orthopaedics, "Hygeias Melathron" TYPET's General Clinic, Mutual Health Fund of National Bank of Greece Personnel (TYPET), Greece.
Address: Therianou 4-6, Athens, 11473. E-mail: spyros.manthas@gmail.com

Normal glenohumeral stability

The glenohumeral joint, also known as the shoulder joint, is a ball-and-socket joint that is formed between the humeral head and the glenoid fossa of the scapula. The glenoid fossa is a shallow depression on the lateral side of the scapula, surrounded by a rim of fibrocartilage called the glenoid labrum, which helps to deepen the socket and stabilize the joint. Joint capsule is a thick, fibrous sheath that surrounds the joint. The humeral head is a rounded structure that fits into the glenoid fossa. It is held in place by a group of muscles and tendons, known as the rotator cuff, which attach to the humerus and scapula. Together, the articular surfaces of the head of the humerus and the glenoid fossa, along with the surrounding ligaments and muscles, form the glenohumeral joint [1,2].

The glenohumeral joint is a highly mobile joint that provides the upper extremity with a wide range of motion. This flexibility enables the shoulder to move the upper limb at multiple positions in space, acting as a stable fulcrum. However, this mobility also makes the glenohumeral joint inherently unstable, and its stability relies heavily on the surrounding muscles, tendons, ligaments, and bones. The normal glenohumeral stability is maintained through a combination of factors that work together to provide both static and dynamic stability to the joint [3].

Static stability is provided by glenoid labrum, joint capsule, glenohumeral ligaments, articular congruity and version and negative intraarticular pressure. The joint capsule and labrum help to hold the humeral head in place within the glenoid fossa. The labrum adds more than 50% to the glenoid depth. Superior, middle and inferior glenohumeral ligament provide static restraint. The bony anatomy of the glenohumeral joint, including the shape of the glenoid and the size and orientation of the humeral head, provides some stability to the joint. The shallow glenoid fossa is deepened by the glenoid labrum, which helps to create a more stable socket for the humeral head [4].

Dynamic stability is provided by rotator cuff muscles, rotator interval, long head of the biceps and periscapular muscles. The rotator cuff muscles help to control the position of the humeral head within the glenoid fossa during movement and provide a compressive force that stabilizes the joint, by compressing

the humeral head into the glenoid fossa during shoulder movement. Long head of the biceps acts as humeral head depressor. Periscapular muscles that control scapular movement, such as the serratus anterior and trapezius, help to position the glenoid fossa for optimal contact with the humeral head. Neuromuscular control is important in maintaining the stability of the glenohumeral joint. The proprioceptive feedback from the joint and surrounding muscles allows for fine-tuning of the muscle activity around the joint [5,6].

Overall, the normal glenohumeral stability is a complex interaction between several factors that work together to provide adequate stability while allowing for the mobility required for shoulder function. The maintenance of normal glenohumeral stability requires a complex interplay between these various factors. Any disruption to this balance can result in instability and potentially lead to injury or dysfunction of the shoulder joint.

Shoulder Instability

Definition and Classification

Shoulder instability refers to the condition where humerus and scapula lose their normal relationship, resulting in excessive translation of the humeral head within the glenohumeral joint. Glenohumeral subluxation is a partial dislocation, defined as "translation of the humeral head against the glenoid fossa without a complete loss of contact between the articular surfaces" [7]. Generally, glenohumeral instability may be categorized according to mechanism, duration, occurrence, and direction.

According to the mechanism, shoulder instability can be classified as either traumatic or atraumatic. Traumatic shoulder instability typically occurs as a result of a sudden injury or dislocation of the shoulder joint. Atraumatic shoulder instability, on the other hand, can develop gradually over time due to repetitive overhead motions or general wear and tear on the shoulder joint. According to duration, acute glenohumeral instability is defined in case the dislocation has taken place within 24 to 36 hours after trauma. After 4 weeks, glenohumeral instability is classified as chronic. According to the direction of dislocation, instability may be defined as anterior, posterior or inferior. In anterior dislocations, the humeral head moves forwardly in relation to the anterior glenoid rim and is usually found below the coracoid process. Multiligamentous laxity leads to multidirectional instability which is as-

sociated with generalized instability of the glenohumeral joint in at least 2 planes of motion (anterior, posterior, or inferior). Multidirectional instability is also referred as atraumatic multidirectional bilateral rehabilitation inferior capsular shift (AMBRI) [8]. Recurrent or habitual dislocation is a condition where the shoulder is vulnerable to repeated dislocations by slight force, offering a subnormal resistance to redislocation [9].

Epidemiology

Anterior dislocation is one of the most common shoulder injuries, with a reported 2% annual rate in general population. Military and contact athlete patients have a higher incidence for anterior traumatic instability [10]. Posterior shoulder dislocations are rare and comprise up to 10% of unstable shoulders [11]. Multidirectional shoulder instability is more often in 2nd and 3rd decade of life.

Pathogenesis

The most common mechanism of shoulder instability includes the application of direct or indirect force to the shoulder, in a susceptible position. A sudden load to the arm with the shoulder in extension, abduction and external rotation may result in anterior shoulder dislocation. Axial load with the shoulder in flexion, adduction, and internal rotation may cause posterior dislocation [11]. Acute glenohumeral instability or dislocation may be caused by other less usual mechanisms, such as electrocution and epileptic seizures. In cases with co-existing generalized ligamentous laxity, glenohumeral instability may be associated with minor or overuse trauma, [6].

Shoulder instability can be combined with bone or soft tissue injuries, usually occurring at the initial episode of dislocation, including fractures of the humeral head, ligamentous injuries, rotator cuff injuries, fractures of the greater or lesser tuberosity, fractures of the glenoid, vascular damage, and nerve injuries. These concomitant injuries are rare in cases with nontraumatic instability. The majority of patients with traumatic anterior instability suffer from an avulsion of the anterior labrum and the inferior glenohumeral ligament from the anteroinferior glenoid (Bankart lesion). At the time of dislocation, the humeral head may impact upon the glenoid rim, causing a compression fracture. In most anterior shoulder dislocations, this compression fracture may be created on the posterosuperior aspect of the humeral head (Hill-Sachs le-

sion) [12]. The co-existence of rotator cuff injuries along with shoulder dislocation increases with age, reaching 80% in patients older than 60 [13]. Other concomitant injuries include humeral avulsion of the glenohumeral ligament (HAGL), glenoid labral articular defects (GLAD), and anterior labral periosteal sleeve avulsion (ALPSA). In posterior shoulder dislocations, compression fractures may be created on the anteromedial aspect of the humeral head (reverse Hill-Sachs lesions). Other co-existing disorders combined with posterior instability include posterior Bankart lesions, posterior labral cysts and posterior glenoid fractures. The rate of axillary nerve injury at the time of dislocation has been reported at 5% - 25%, increasing with age [14]. Identification of these injuries is of vital importance, as they can define direction of instability affecting patient management [15].

Clinical presentation

The diagnosis of acute shoulder instability is primarily based on history and clinical examination. The patient usually recounts shoulder trauma and the mechanism of the applied force. Sometimes, there is a history of previous shoulder trauma or dislocation. Posterior inspection of the shoulder in a sitting position reveals gross deformity. Following a shoulder dislocation, the head of the humerus may be easily palpated beneath the skin. Pain and muscle contraction limits dramatically the shoulder motion. Anterior dislocation reduces abduction and internal rotation while posterior dislocation reduces external rotation. A rare form of shoulder dislocation is luxatio erecta (inferior glenohumeral dislocation), where the arm is locked in full abduction [16]. It is important to perform a complete neurovascular examination of the upper limb, as anterior shoulder dislocation is associated with axillary nerve injury [14].

In case of non-acute shoulder instability, patients may complain for a recent exacerbation of a recurrent instability, or a chronic vague pain without any previous diagnosis of instability. Sometimes, they mention avoiding the placement of their shoulder in a position prone to dislocations. Inspection may reveal shoulder asymmetry, muscular atrophy, localized edema, or ecchymoses. Palpation may reveal positions of localized tenderness and bony defects. Active and passive

range of motion and muscular strength should be compared with the contralateral shoulder [17]. Further clinical examination may reveal signs of generalized ligamentous laxity, which is assessed by Beighton's criteria, where a score more than 4 suggests joint hypermobility [18]. Special clinical tests of shoulder instability are usually performed at the end of the clinical examination. These include the drawer test, the sulcus test, the jerk test, the "load and shift" test, the apprehension test and its variations and the relocation sign [19]. If the diagnosis of instability is unclear in some patients, clinical examination under general anesthesia could be considered.

Radiographic Studies

Anteroposterior (AP) shoulder x-ray will easily reveal acute anterior shoulder dislocation. Axillary views display the location of the humeral head in relation to the glenoid. In case of inability of arm abduction, modified views (trauma axillary lateral view, Velpeau axillary lateral view) may be performed. A scapular lateral view x-ray is taken at an oblique angle, with the patient's arm raised and the X-ray machine positioned at an angle to visualize the scapula and the humeral head. Transthoracic view is not useful for the evaluation of shoulder instability. The West Point axillary view and the apical oblique view reveal glenoid bony defects [20].

Computed tomography (CT) may sufficiently depict glenoid defects. CT with 3D reconstructions can describe in detail the osseous anatomy and calculate glenoid bone loss [21, 22]. Magnetic resonance imaging (MRI) is the gold standard for the evaluation of soft tissue injuries, especially rotator cuff injuries and labral defects, with a sensitivity exceeding 90%. MRI arthrogram has an increased sensitivity and specificity in the diagnosis of soft tissue injuries [23].

Management

The management of glenohumeral instability depends on a multitude of factors, depending on the type and severity of the instability. Generally, the principles of management can be divided into non-operative and operative approaches. The decision is based on the equilibrium between failure of conservative treatment and risk of complications of surgical treatment [24].

The risk of recurrent instability is a major factor for

the choice of management. After the first episode of glenohumeral dislocation, the incidence of recurrent instability has been calculated to be 14% to 100% [25]. Factors affecting the possibility of the development of recurrent instability include age, gender, the number of previous dislocations, participation in sports, structural glenohumeral abnormalities and associated injuries. The younger the patient's age at first shoulder dislocation, the greater the likelihood of recurrent instability. Anterior dislocation in a patient aged less than 20 years results in a 90% rate of recurrent dislocation. Risk of recurrent instability is significantly higher in athletes compared to non-athletic patients [26].

The Instability Severity Score (ISS) is a scoring system used to assess the severity of shoulder instability. It takes into account various factors, including the patient's age, degree of sports participation, participation in contact or overhead sports, shoulder hyperlaxity and the presence of associated injuries, such as Hill-Sachs lesion and loss of glenoid contour. The ISS is scored on a scale of 0 - 10, with higher scores indicating greater severity of instability. A score less than 6 suggests an acceptable risk of recurrent instability, below 10%. The ISS can be used to guide treatment decisions, with more severe cases often requiring surgical intervention to restore stability to the shoulder joint [27].

Acute Management

After an acute shoulder dislocation, the glenohumeral joint should be reduced as soon as possible. The reduction may be facilitated with the use of muscle relaxants and analgesics. The faster a closed reduction is attempted after initial trauma, the greater the chances of success. The most widely used techniques for closed reduction of the glenohumeral joint include the Hippocrates technique, the Stimson method, the Kocher method and the Milch method [28]. If closed reduction at the emergency department is not successful, general anesthesia should be applied. After the radiographic confirmation of a successful reduction of anterior dislocation, the arm should be immobilized in a sling for 1 week. There is no benefit for a more prolonged immobilization. In case of posterior dislocation, after reduction, the shoulder should be immobilized in external rotation for 4 - 6 weeks [29].

Conservative Treatment

Conservative management includes physical therapy,

rest, and activity modification and it is generally indicated for patients with mild instability, who can respond successfully to rehabilitation and are less prone to develop recurrent instability. After a first episode of dislocation, initial conservative management may be attempted for patients regardless of age with minimal damage on x-rays, low demand patients who are less likely to engage in high-risk activities, and patients with atraumatic multidirectional glenohumeral instability, without trauma history and with signs of general ligamentous laxity [8,30].

Initial immobilization in a sling for a brief period allows for the recovery of the static stabilizers. By limiting range of motion and avoiding positions of increased vulnerability for dislocation, the shoulder is protected from recurrence of instability. Rehabilitation includes strengthening of dynamic stabilizers, especially rotator cuff muscles, deltoid and peri-scapular muscles, to provide additional stability for the injured shoulder. Return to sport may be allowed after 3 weeks [30].

Surgical Treatment

After the first episode of anterior or posterior shoulder dislocation, indications for surgical treatment include failed conservative management, multiple dislocations in young patients, irreducible dislocations, open dislocations, post-reduction instability of the shoulder and first-time dislocation in young elite athletes. American Shoulder and Elbow Surgeons suggest that the first episode of dislocation should be treated surgically in athletes aged 14 to 30 at the end of their competitive season if they have positive apprehension testing and bone loss [31]. Plenty of studies suggest that young and active patients with a first episode of shoulder dislocation should be operated as the rate of recurrent instability is relatively high. Moreover, surgical treatment is indicated for patients suffering from Traumatic Unilateral dislocations with a Bankart lesion requiring Surgery (TUBS) [32].

Operative management typically involves surgical repair of the injured structures. The specific surgical approach depends on the type and severity of the instability, but may include open or arthroscopic techniques, as well as various types of surgical anchors or sutures. Regardless of the approach, the ultimate goal of management is to restore stability to the shoulder joint, while minimizing the risk of complications and optimizing the patient's functional outcome. Patients may also benefit from post-operative rehabilitation to restore range of motion, strength,

and function [33].

Surgical options include arthroscopic procedures, open procedures with soft tissue repair or augmentation, and open procedures with bony augmentation. While, in the past, open anterior shoulder stabilization methods were the gold standard, after the invention of modern instrumentation and surgical methods, the outcome of arthroscopic procedures are nearly equivalent if not superior to those after an open stabilization [34]. If studies published after 2000 are taken into consideration, arthroscopic shoulder stabilization has better results than open repair [35]. Moreover, arthroscopic procedures are associated with less postoperative pain, preservation of shoulder motion, shorter hospitalization and decreased morbidity and rate of complications [36]. However, there are few situations where open techniques are preferred, especially in cases of large bony fragment fixation in either the glenoid or the humeral head.

In case of a Hill-Sachs defect, its position may affect the choice of type of surgical treatment. An "on-track" Hill-Sachs lesion is located in a more central or superior position on the humeral head, and it does not extend beyond the glenoid. This means that the humeral head stays "on-track" with the glenoid during shoulder movement, and there is less risk of instability. In this case, the primary focus of surgical management is usually the repair of the labrum and the supportive ligaments. This can typically be achieved using arthroscopic Bankart repair or open Latarjet procedure. On the contrary, an "off-track" Hill-Sachs lesion is located more laterally or inferiorly on the humeral head, and it extends beyond the usual confines of the glenoid. This means that the humeral head can engage with the glenoid during shoulder movement, leading to instability and a higher risk of recurrent dislocations. In these cases, treatment includes the remplissage procedure or the Latarjet procedure [37,38].

Arthroscopic Procedures

Arthroscopic procedures usually start with an inspection of the glenohumeral joint to identify the injuries of the glenoid, the humeral head, the rotator interval and the labrum. If a Bankart lesion is identified after a first episode of dislocation in athletes younger than 25 years, it should be repaired, through an arthroscopic Bankart repair, as it results in a 7-fold lower rate of

recurrence^[39,40]. The procedure is relatively indicated in elite athletes, in recurrent dislocations, after a failed course of physical therapy, provided there is less than 25% glenoid bone loss. After the full mobilization of labrum, it is fixed on the anterior glenoid rim, with the use of 3 or more suture anchors. The reported results of this technique are excellent and the rate of recurrent instability is below 7%. In case of large Hill-Sachs lesions, the method can be combined with remplissage, where posterior capsule and infraspinatus tendon are sutured into the humeral head defect. In these patients, combined Bankart repair with remplissage has better outcome than isolated Bankart repair^[41].

If there is a posterior capsulolabral detachment, it should be mobilized with a periosteal elevator and stabilized on the posterior glenoid rim. The reattachment of the labrum was initially attempted with the use of metallic staples or transglenoid sutures with a more than 10% incidence of complications and a 33% rate of recurrent instability^[42-44]. Nowadays, suture anchors are used for the capsulolabral repair in arthroscopic posterior stabilization and capsular plication. In the past, thermal capsulorrhaphy has been applied with the aim to increase glenohumeral stability by contracting capsular tissue, with a high rate of chondrolysis, leading to the abandonment of the method^[45,46].

In case of multidirectional instability, anterior or posterior arthroscopic capsulorrhaphy with suture-tying techniques has good to excellent short-term results in 95% of cases, with a 2 – 5% rate of recurrent instability^[47]. Arthroscopic thermal capsulorrhaphy has produced mixed results, with increased rate of complications such as axillary nerve neuropraxias and glenoid chondrolysis^[48].

Open Procedures

Open procedures usually start with a deltopectoral approach and include soft tissue or bony techniques. The most widely performed soft tissue technique is the open Bankart procedure, where the labrum is mobilized with a periosteal elevator and then stabilized to the anterior inferior glenoid rim, using screws or suture anchors^[49]. The method is indicated for Bankart lesions with less than 25% glenoid bone loss, for co-existing glenoid fractures and after failure of arthroscopic Bankart repair. The results of the technique are ex-

cellent; however equivalent to arthroscopic repair, with a 8 – 12% rate of recurrent dislocation^[50,51]. More than 80% of athletes may return to the previous level of sport; however, the rate of osteoarthritis reaches 70%^[51,52]. Another used technique is the capsulolabral reconstruction, which reattaches the torn or damaged labrum and capsule to the shoulder socket, typically using sutures or anchors. The reported results are excellent in 95% of patients with an up to 4% incidence of recurrent dislocation^[53].

At the beginning of the 20th century, the shortening of subscapularis tendon (Putti-Platt procedure) was used to treat anterior instability with significantly inferior results^[54,55]. In the early 1940s, the Magnuson-Stack procedure was popularized. During the procedure, a portion of the subscapularis tendon was taken and attached to the glenoid using sutures. Then the tendon was passed through a drill hole in the humerus and attached to the bone using sutures. Even though early results were excellent, the method fell into disfavor, as it modified the normal biomechanics of the glenohumeral joint^[9].

Posterior instability may be addressed through a posterior or a deltoid-splitting approach. In patients with posterior instability, surgical release and anterior advancement of the capsule through an anterior approach, has been described with satisfactory results^[56]. In case of a reverse Hill-Sachs lesion less than 40%, through the McLaughlin procedure, damaged bone is removed and the remaining humeral head is reshaped to create a smooth surface. Then through a drilled hole, a button-like implant is inserted, acting as a new “ball” for the glenohumeral joint, preventing the humeral head to engage the posterior glenoid rim^[57]. Posterior capsulorrhaphy may be conducted in a manner similar to anterior capsulorrhaphy. Good to excellent results have been reported in about 90% of patients, with an up to 23% rate of recurrent instability^[58]. However, only 68% of athletes return to the preinjury level of sport^[59].

Additionally, anterior glenohumeral instability may be managed with several bony techniques. These bony procedures are indicated in chronic bony deficiencies with a more than 25% bone loss of glenoid. In these cases, where part of glenoid bone is absent, excessive stress is transferred to anterior labrum, increasing the risk of failure of isolated labral repair. Relative indications also include recurrent anterior instability with subcritical (>13.5%) glenoid bone loss. The basic concept of the methods is the

placement of a bony fragment on the glenoid neck, providing the native glenoid with additional surface, preventing anterior dislocation. Two common procedures have used the adjacent coracoids process: the Bristow procedure and the Latarjet procedure. In the Bristow procedure, the tip of the coracoid process is osteotomized, with the attachment of the conjoined tendons and the coracoacromial ligament and stabilized onto the anterior inferior glenoid rim ^[60]. Even though the technique has been associated with increased patient satisfaction, and a low rate of recurrent dislocations, severe complications have been reported including loss of external rotation, residual pain, and graft nonunion ^[61-66]. In the more commonly performed Latarjet procedure, a larger fragment of the coracoid process is osteotomized and stabilized to the neck of the glenoid with screws. The results of the technique are excellent and the reported rate of recurrent instability is less than 10%. However, the method has been associated with a 30 – 70% rate of glenohumeral arthritis ^[67-69]. A recent meta-analysis reported an overall complication rate of 16% and a 2.6% risk of re-operation ^[70]. Few recent studies have shown that Latarjet procedure yields a superior outcome in comparison to Bankart repairs, with reduced risk of recurrence and redislocation ^[71, 72]. In patients older than 40 years, pain relief and satisfaction is similar with either arthroscopic Bankart repair or Latarjet procedure ^[73]. For the aforementioned reasons, there are surgeons, especially in Europe, who prefer a direct Latarjet procedure after a first anterior dislocation, instead of a Bankart repair.

In the 1970s, the subscapularis tendon was repositioned to the back of the humeral head, increasing the distance of dislocation (Eden-Hybbinette procedure). The outcome was good or excellent in 80% of patients with a 4 – 33% rate of recurrent dislocation. Long-term incidence of glenohumeral osteoarthritis reached 90% leading to the abandonment of the method ^[74-77].

In patients with bone loss in glenoid more than 25% and after failure of Bristow/Latarjet procedures, the glenoid defect may be filled with autografts or allografts, usually tricortical iliac crest or distal clavicle autograft. Grafts are placed at the site of glenoid defect after the capsulolabral reconstruction, and are fixed with multiple cortical screws or buttons. A 90% healing rate has been reported after a 1.5 years follow-up ^[78-81]. Mid-term results are satisfactory ^[82]. According to a recent meta-analysis, free bone block procedures yield equivalent results with Latarjet

procedures, for anterior instability ^[83]. In case of large Hill-Sachs lesion (>40%), shoulder arthroplasty or rotational osteotomies may be applied ^[84].

In case of recurrent posterior dislocations, excessive glenoid retroversion may be the cause of posterior instability ^[85]. A posterior glenoid osteotomy can restore glenoid version with good to excellent results in 82% of the patients and a 12 – 17% rate of recurrent dislocation. However, the method has a high rate of complications, such as anterior instability, glenoid fracture, coracoid impingement and shoulder osteoarthritis. For the aforementioned reasons, glenoid osteotomy is reserved for cases of glenoid retroversion more than 30 degrees and after failed posterior capsulorrhaphy ^[86-88]. Posterior bone block augmentation does not yield improved patient-reported outcomes and is associated with more than 13% rate of complications ^[89, 90]. If posterior dislocation is more than 6 months or there is a reverse Hill-Sachs defect more than 40%, hemiarthroplasty or total shoulder arthroplasty may be considered ^[91, 92].

Patients with multidirectional instability may be managed with an anterior capsulolabral reconstruction, focusing on the inferior aspect of the shoulder capsule, which should be released and advanced anteriorly and superiorly. Good to excellent outcome has been reported to 90% of cases and the rate of recurrent instability is more than 26% ^[93]. In case, patients complain mostly for posterior instability, a posterior capsular shift procedure may be performed.

Postoperative Rehabilitation

The goal of rehabilitation after arthroscopic or open shoulder stabilization is the protection of the surgical repair and the progressive reestablishment of full range of motion. After a Bankart or Latarjet procedure, the operated shoulder is immobilized in a sling for approximately 4 to 6 weeks. Passive range of motion exercises in the supine position may be initiated 1 to 2 weeks after surgery. Careful active assisted external rotation is recommended for the first 4 weeks. Resistive strengthening exercises can be initiated 3 months postoperatively once full, painless, active forward flexion has been recovered; however external rotation should be limited to half the range of motion of the contralateral shoulder. Full use of the shoulder and

return to contact sports is typically allowed 6 months after surgery [41]. The return to overhead sports after an arthroscopic Bankart repair may be delayed until 13 months postoperatively [94].

Complications

Infection is a rare complication after a shoulder stabilization procedure with an incidence less than 0.25%. In case of a diagnosis of a postoperative deep infection, immediate open or arthroscopic irrigation and surgical debridement is indicated, followed by intravenous antibiotics [95]. The incidence of nerve injuries is significantly decreased in arthroscopic procedures compared to open surgeries [96]. The musculocutaneous nerve and the axillary nerve are mostly susceptible to injury. Risk factors for nerve injuries include lateral traction, compression because of fluid extravasation, and a tourniquet effect after an over tightened wrapping

of the upper extremity. Most axillary nerve injuries are transient neuropraxias that progress to full recovery [97].

Osteolysis, chondrolysis and synovitis are established complications of arthroscopic Bankart repair [98]. Stiffness is another established complication after shoulder stabilization, especially after Latarjet procedure. Risk factors include excessive capsular tightening, non-anatomic techniques of reconstruction, prolonged postoperative immobilization and low compliance with the rehabilitation regime [70]. Treatment of stiffness includes manipulation under anesthesia and arthroscopic debridement of scar tissue [99]. Persistent postoperative shoulder pain may be attributed to overtightening during labral repair.

Conflict of interest

The authors declare no conflicts of interest.

References

- Bakhsh W, Nicandri G. Anatomy and Physical Examination of the Shoulder. *Sports Med Arthrosc Rev.* 2018 Sep;26(3):e10-e22.
- Chang LR, Anand P, Varacallo M. Anatomy, Shoulder and Upper Limb, Glenohumeral Joint. 2022 Jan.
- Kadi R, Milants A, Shahabpour M. Shoulder Anatomy and Normal Variants. *J Belg Soc Radiol.* 2017 Dec 16;101(Suppl 2):3.
- Curl LA, Warren RF. Glenohumeral joint stability. Selective cutting studies on the static capsular restraints. *Clin Orthop Relat Res.* 1996 Sep(330):54-65.
- Klungsoyr JA, Vagstad T, Klungsoyr PJ, Hellevik AI, Drogset JO. Dynamic and Static Stabilization of Anterior Shoulder Instability With the Subscapular Sling Procedure. *Arthrosc Tech.* 2021 Jul;10(7):e1773-e81.
- Omoumi P, Teixeira P, Lecouvet F, Chung CB. Glenohumeral joint instability. *J Magn Reson Imaging.* 2011 Jan;33(1):2-16.
- Eljabu W, Klinger HM, von Knoch M. The natural course of shoulder instability and treatment trends: a systematic review. *J Orthop Traumatol.* 2017 Mar;18(1):1-8.
- Neer CS, 2nd. Involuntary inferior and multidirectional instability of the shoulder: etiology, recognition, and treatment. *Instr Course Lect.* 1985;34:232-8.
- Karadimas JE. Recurrent traumatic anterior dislocation of the shoulder. 218 consecutive cases treated by a modified Magnuson-Stack procedure and follow for 2-18 years. *Acta Orthop Scand Suppl.* 1997 Oct;275:69-71.
- Krøner K, Lind T, Jensen J. The epidemiology of shoulder dislocations. *Arch Orthop Trauma Surg.* 1989;108(5):288-90.
- Brelin A, Dickens JF. Posterior Shoulder Instability. *Sports Med Arthrosc Rev.* 2017 Sep;25(3):136-43.
- Hill HA, Sachs MD. The Grooved Defect of the Humeral Head. *Radiology.* 1940;35(6):690-700.
- Itoi E, Tabata S. Rotator cuff tears in anterior dislocation of the shoulder. *Int Orthop.* 1992;16(3):240-4.
- Hardie CM, Jordan R, Forker O, Fort-Schaale A, Wade RG, Jones J, et al. Prevalence and risk factors for nerve injury following shoulder dislocation. *Musculoskelet Surg.* 2022 Nov 29.
- Tokish JM. Shoulder Instability. *Sports Med Arthrosc Rev.* 2020 Dec;28(4):121.
- Yamamoto T, Yoshiya S, Kurosaka M, Nagira K, Nabeshima Y. Luxatio erecta (inferior dislocation of the shoulder): a report of 5 cases and a review of the literature. *Am*

- J Orthop (Belle Mead NJ). 2003 Dec;32(12):601-3.
17. Callanan M, Tzannes A, Hayes K, Paxinos A, Walton J, Murrell GA. Shoulder instability. Diagnosis and management. *Aust Fam Physician*. 2001 Jul;30(7):655-61.
 18. Malek S, Reinhold EJ, Pearce GS. The Beighton Score as a measure of generalised joint hypermobility. *Rheumatol Int*. 2021 Oct;41(10):1707-16.
 19. Lizzio VA, Meta F, Fidai M, Makhni EC. Clinical Evaluation and Physical Exam Findings in Patients with Anterior Shoulder Instability. *Curr Rev Musculoskelet Med*. 2017 Dec;10(4):434-41.
 20. Goud A, Segal D, Hedayati P, Pan JJ, Weissman BN. Radiographic evaluation of the shoulder. *Eur J Radiol*. 2008 Oct;68(1):2-15.
 21. Ruiz Santiago F, Martínez Martínez A, Tomás Muñoz P, Pozo Sánchez J, Zarza Pérez A. Imaging of shoulder instability. *Quant Imaging Med Surg*. 2017 Aug;7(4):422-33.
 22. Makhni EC, Tramer JS, Anderson MJ, Levine WN. Evaluating Bone Loss in Anterior Shoulder Instability. *J Am Acad Orthop Surg*. 2022 Jun 15;30(12):563-72.
 23. Steinbach LS. MRI of shoulder instability. *Eur J Radiol*. 2008 Oct;68(1):57-71.
 24. Provencher MT, Midtgaard KS, Owens BD, Tokish JM. Diagnosis and Management of Traumatic Anterior Shoulder Instability. *J Am Acad Orthop Surg*. 2021 Jan 15;29(2):e51-e61.
 25. Polyzois I, Dattani R, Gupta R, Levy O, Narvani AA. Traumatic First Time Shoulder Dislocation: Surgery vs Non-Operative Treatment. *Arch Bone Jt Surg*. 2016 Apr;4(2):104-8.
 26. Olds M, Ellis R, Donaldson K, Parmar P, Kersten P. Risk factors which predispose first-time traumatic anterior shoulder dislocations to recurrent instability in adults: a systematic review and meta-analysis. *British Journal of Sports Medicine*. 2015;49(14):913-22.
 27. Balg F, Boileau P. The instability severity index score. A simple pre-operative score to select patients for arthroscopic or open shoulder stabilisation. *J Bone Joint Surg Br*. 2007 Nov;89(11):1470-7.
 28. Hussein MK. Kocher's method is 3,000 years old. *J Bone Joint Surg Br*. 1968 Aug;50(3):669-71.
 29. Paterson WH, Throckmorton TW, Koester M, Azar FM, Kuhn JE. Position and duration of immobilization after primary anterior shoulder dislocation: a systematic review and meta-analysis of the literature. *J Bone Joint Surg Am*. 2010 Dec 15;92(18):2924-33.
 30. Dang V. The nonoperative management of shoulder instability. *JAAPA*. 2007 Mar;20(3):32-8.
 31. Lemme NJ, Kuczmarski AS, Goodman AD, Ready LV, Dickens JF, Owens BD. Management and Outcomes of In-Season Anterior Shoulder Instability in Athletes. *JBJS Reviews*. 2019;7(11):e2.
 32. Farrar NG, Malal JJ, Fischer J, Waseem M. An overview of shoulder instability and its management. *Open Orthop J*. 2013;7:338-46.
 33. Rossy WH, Cieslak K, Uquillas CA, Rokito A. Current trends in the management of recurrent anterior shoulder instability. *Bull Hosp Jt Dis* (2013). 2014;72(3):210-6.
 34. Rashid MS, Arner JW, Millett PJ, Sugaya H, Emery R. The Bankart repair: past, present, and future. *J Shoulder Elbow Surg*. 2020 Dec;29(12):e491-e8.
 35. Gao B, DeFroda S, Bokshan S, Ready LV, Sullivan K, Etzel C, et al. Arthroscopic Versus Open Bankart Repairs in Recurrent Anterior Shoulder Instability: A Systematic Review of the Association Between Publication Date and Postoperative Recurrent Instability in Systematic Reviews. *Arthroscopy*. 2020 Mar;36(3):862-71.
 36. Fabbriani C, Milano G, Demontis A, Fadda S, Zirano F, Mulas PD. Arthroscopic versus open treatment of Bankart lesion of the shoulder: a prospective randomized study. *Arthroscopy*. 2004 May;20(5):456-62.
 37. Calvo E, Delgado C. Management of off-track Hill-Sachs lesions in anterior glenohumeral instability. *J Exp Orthop*. 2023 Mar 21;10(1):30.
 38. Hatta T, Yamamoto N, Shinagawa K, Kawakami J, Itoi E. Surgical decision making based on the on-track/off-track concept for anterior shoulder instability: a case-control study. *JSES Open Access*. 2019 Mar;3(1):25-8.
 39. Murphy AI, Hurley ET, Hurley DJ, Pauzenberger L, Mullett H. Long-term outcomes of the arthroscopic Bankart repair: a systematic review of studies at 10-year follow-up. *J Shoulder Elbow Surg*. 2019 Nov;28(11):2084-9.
 40. Hurley ET, Manjunath AK, Bloom DA, Pauzenberger L, Mullett H, Alaia MJ, et al. Arthroscopic Bankart Repair Versus Conservative Management for First-Time Traumatic Anterior Shoulder Instability: A Systematic Review and Meta-analysis. *Arthroscopy*. 2020 Sep;36(9):2526-32.
 41. DeFroda SF, Mehta N, Owens BD. Physical therapy

- protocols for arthroscopic Bankart repair. *Sports health*. 2018;10(3):250-8.
42. Detrisac DA, Johnson LL. Arthroscopic shoulder capsulorrhaphy using metal staples. *Orthop Clin North Am*. 1993 Jan;24(1):71-88.
 43. McIntyre LF, Caspari RB. The rationale and technique for arthroscopic reconstruction of anterior shoulder instability using multiple sutures. *Orthop Clin North Am*. 1993 Jan;24(1):55-8.
 44. Morgan CD, Bodenstab AB. Arthroscopic Bankart suture repair: technique and early results. *Arthroscopy*. 1987;3(2):111-22.
 45. Hawkins RJ, Krishnan SG, Karas SG, Noonan TJ, Horan MP. Electrothermal arthroscopic shoulder capsulorrhaphy: a minimum 2-year follow-up. *Am J Sports Med*. 2007 Sep;35(9):1484-8.
 46. Mohtadi NG, Kirkley A, Hollinshead RM, McCormack R, MacDonald PB, Chan DS, et al. Electrothermal arthroscopic capsulorrhaphy: old technology, new evidence. A multicenter randomized clinical trial. *J Shoulder Elbow Surg*. 2014 Aug;23(8):1171-80.
 47. Chen D, Goldberg J, Herald J, Critchley I, Barmare A. Effects of surgical management on multidirectional instability of the shoulder: a meta-analysis. *Knee Surg Sports Traumatol Arthrosc*. 2016 Feb;24(2):630-9.
 48. Miniaci A, McBirnie J. Thermal capsular shrinkage for treatment of multidirectional instability of the shoulder. *J Bone Joint Surg Am*. 2003 Dec;85(12):2283-7.
 49. Bankart ASB. The pathology and treatment of recurrent dislocation of the shoulder-joint. *British Journal of Surgery*. 2005;26(101):23-9.
 50. Jolles BM, Pelet S, Farron A. Traumatic recurrent anterior dislocation of the shoulder: two- to four-year follow-up of an anatomic open procedure. *J Shoulder Elbow Surg*. 2004 Jan-Feb;13(1):30-4.
 51. Fabre T, Abi-Chahla ML, Billaud A, Geneste M, Durandea A. Long-term results with Bankart procedure: a 26-year follow-up study of 50 cases. *J Shoulder Elbow Surg*. 2010 Mar;19(2):318-23.
 52. Monk AP, Crua E, Gatenby GC, Walsh AJ, Stanley JC, Rosenfeldt MP, et al. Clinical outcomes following open anterior shoulder stabilization for glenohumeral instability in the young collision athlete. *J Shoulder Elbow Surg*. 2022 Jul;31(7):1474-8.
 53. Jobe FW, Giangarra CE, Kvitne RS, Glousman RE. Anterior capsulolabral reconstruction of the shoulder in athletes in overhand sports. *Am J Sports Med*. 1991 Sep-Oct;19(5):428-34.
 54. Fredriksson AS, Tegner Y. Results of the Putti-Platt operation for recurrent anterior dislocation of the shoulder. *Int Orthop*. 1991;15(3):185-8.
 55. Inui H, Nobuhara K. Modified Putti-Platt procedure for recurrent anterior shoulder instability. *Int Orthop*. 2020 Jun;44(6):1123-9.
 56. Wirth MA, Groh GI, Rockwood CA, Jr. Capsulorrhaphy through an anterior approach for the treatment of atraumatic posterior glenohumeral instability with multidirectional laxity of the shoulder. *J Bone Joint Surg Am*. 1998 Nov;80(11):1570-8.
 57. Besnard M, Audebert S, Godenèche A. Arthroscopic McLaughlin Procedure for Treatment of Posterior Instability of the Shoulder With an Engaging Reverse Hill-Sachs Lesion. *Arthrosc Tech*. 2019 Dec;8(12):e1491-e4.
 58. Fuchs B, Jost B, Gerber C. Posterior-inferior capsular shift for the treatment of recurrent, voluntary posterior subluxation of the shoulder. *J Bone Joint Surg Am*. 2000 Jan;82(1):16-25.
 59. Gouveia K, Kay J, Memon M, Simunovic N, Bedi A, Ayeni OR. Return to Sport After Surgical Management of Posterior Shoulder Instability: A Systematic Review and Meta-analysis. *Am J Sports Med*. 2022 Mar;50(3):845-57.
 60. Cowling PD, Akhtar MA, Liow RY. What is a Bristow-Latarjet procedure? A review of the described operative techniques and outcomes. *Bone Joint J*. 2016 Sep;98-B(9):1208-14.
 61. Khawaja K, Mohib Y, Khan Durrani MY, Juman NM, Habib AA, Hashmi P. Functional outcomes of modified Bristow procedure in recurrent shoulder dislocation. *J Pak Med Assoc*. 2021 Oct;71(10):2448-50.
 62. Shao Z, Song Q, Cheng X, Luo H, Lin L, Zhao Y, et al. An Arthroscopic "Inlay" Bristow Procedure With Suture Button Fixation for the Treatment of Recurrent Anterior Glenohumeral Instability: 3-Year Follow-up. *Am J Sports Med*. 2020 Sep;48(11):2638-49.
 63. Zarezade A, Dehghani M, Rozati AR, Banadaki HS, Shekarchizade N. Comparison of Bristow procedure and Bankart arthroscopic method as the treatment of recurrent shoulder instability. *Adv Biomed Res*. 2014;3:256.
 64. Banas MP, Dalldorf PG, Sebastianelli WJ, DeHaven KE. Long-term followup of the modified Bristow procedure.

- Am J Sports Med. 1993 Sep-Oct;21(5):666-71.
65. Ferlic DC, DiGiovine NM. A long-term retrospective study of the modified Bristow procedure. *Am J Sports Med.* 1988 Sep-Oct;16(5):469-74.
 66. Spoor AB, de Waal Malefijt J. Long-term results and arthropathy following the modified Bristow-Latarjet procedure. *Int Orthop.* 2005 Oct;29(5):265-7.
 67. Chillemi C, Guerrisi M, Paglialunga C, Salate Santone F, Osimani M. Latarjet procedure for anterior shoulder instability: a 24-year follow-up study. *Arch Orthop Trauma Surg.* 2021 Feb;141(2):189-96.
 68. Ernstbrunner L, Wartmann L, Zimmermann SM, Schenk P, Gerber C, Wieser K. Long-term Results of the Open Latarjet Procedure for Recurrent Anterior Shoulder Instability in Patients Older Than 40 Years. *Am J Sports Med.* 2019 Nov;47(13):3057-64.
 69. Hurley ET, Jamal MS, Ali ZS, Montgomery C, Pauzenberger L, Mullett H. Long-term outcomes of the Latarjet procedure for anterior shoulder instability: a systematic review of studies at 10-year follow-up. *J Shoulder Elbow Surg.* 2019 Feb;28(2):e33-e9.
 70. Cho CH, Na SS, Choi BC, Kim DH. Complications Related to Latarjet Shoulder Stabilization: A Systematic Review. *Am J Sports Med.* 2023 Jan;51(1):263-70.
 71. Bliven KCH, Parr GP. Outcomes of the Latarjet procedure compared with Bankart repair for recurrent traumatic anterior shoulder instability. *Journal of athletic training.* 2018;53(2):181-3.
 72. Imam MA, Shehata MSA, Martin A, Attia H, Sinokrot M, Bahbah EI, et al. Bankart Repair Versus Latarjet Procedure for Recurrent Anterior Shoulder Instability: A Systematic Review and Meta-analysis of 3275 Shoulders. *Am J Sports Med.* 2021 Jun;49(7):1945-53.
 73. Ernstbrunner L, De Nard B, Olthof M, Beeler S, Bouaicha S, Gerber C, et al. Long-term Results of the Arthroscopic Bankart Repair for Recurrent Anterior Shoulder Instability in Patients Older Than 40 Years: A Comparison With the Open Latarjet Procedure. *Am J Sports Med.* 2020 Jul;48(9):2090-6.
 74. Rachbauer F, Ogon M, Wimmer C, Sterzinger W, Huter B. Glenohumeral osteoarthritis after the Eden-Hybbinette procedure. *Clin Orthop Relat Res.* 2000 Apr(373):135-40.
 75. Rahme H, Wikblad L, Nowak J, Larsson S. Long-term clinical and radiologic results after Eden-Hybbinette operation for anterior instability of the shoulder. *J Shoulder Elbow Surg.* 2003 Jan-Feb;12(1):15-9.
 76. Villatte G, Spurr S, Broden C, Martins A, Emery R, Reilly P. The Eden-Hybbinette procedure is one hundred years old! A historical view of the concept and its evolutions. *Int Orthop.* 2018 Oct;42(10):2491-5.
 77. Wildner M, Wimmer B, Reichelt A. Osteoarthritis after the Eden-Hybbinette-Lange procedure for anterior dislocation of the shoulder. A 15 year follow up. *Int Orthop.* 1994 Oct;18(5):280-3.
 78. Longo UG, Loppini M, Rizzello G, Ciuffreda M, Berton A, Maffulli N, et al. Remplissage, humeral osteochondral grafts, weber osteotomy, and shoulder arthroplasty for the management of humeral bone defects in shoulder instability: systematic review and quantitative synthesis of the literature. *Arthroscopy.* 2014 Dec;30(12):1650-66.
 79. Boehm E, Gerhardt C, Kraus N, Scheibel M. Arthroscopic Glenoid Reconstruction for Chronic Anteroinferior Shoulder Instability Using a Tricortical Iliac Crest Bone Graft. *JBJS Essent Surg Tech.* 2016 Dec 28;6(4):e39.
 80. Ranalletta M, Tanoira I, Bertona A, Maignon G, Bongiovanni S, Rossi LA. Autologous Tricortical Iliac Bone Graft for Failed Latarjet Procedures. *Arthrosc Tech.* 2019 Mar;8(3):e283-e9.
 81. Warner JJ, Gill TJ, O'Hollerhan J D, Pathare N, Millett PJ. Anatomical glenoid reconstruction for recurrent anterior glenohumeral instability with glenoid deficiency using an autogenous tricortical iliac crest bone graft. *Am J Sports Med.* 2006 Feb;34(2):205-12.
 82. Ueda Y, Sugaya H, Takahashi N, Matsuki K, Tokai M, Morioka T, et al. Arthroscopic Iliac Bone Grafting for Traumatic Anterior Shoulder Instability With Significant Glenoid Bone Loss Yields Low Recurrence and Good Outcome at a Minimum of Five-Year Follow-Up. *Arthroscopy.* 2021 Aug;37(8):2399-408.
 83. Gilat R, Haunschild ED, Lavoie-Gagne OZ, Tauro TM, Knapik DM, Fu MC, et al. Outcomes of the Latarjet Procedure Versus Free Bone Block Procedures for Anterior Shoulder Instability: A Systematic Review and Meta-analysis. *Am J Sports Med.* 2021 Mar;49(3):805-16.
 84. Hasler A, Fornaciari P, Jungwirth-Weinberger A, Jentzsch T, Wieser K, Gerber C. Reverse shoulder arthroplasty in the treatment of glenohumeral instability. *J Shoulder Elbow Surg.* 2019 Aug;28(8):1587-94.
 85. Brewer BJ, Wubben RC, Carrera GF. Excessive retroversion of the glenoid cavity. A cause of non-traumatic pos-

- terior instability of the shoulder. *J Bone Joint Surg Am*. 1986 Jun;68(5):724-31.
86. Lacheta L, Singh TSP, Hovsepian JM, Braun S, Imhoff AB, Pogorzelski J. Posterior open wedge glenoid osteotomy provides reliable results in young patients with increased glenoid retroversion and posterior shoulder instability. *Knee Surg Sports Traumatol Arthrosc*. 2019 Jan;27(1):299-304.
87. Malik SS, Jordan RW, Tahir M, MacDonald PB. Does the posterior glenoid osteotomy reduce the rate of recurrence in patients with posterior shoulder instability - A systematic review. *Orthop Traumatol Surg Res*. 2021 Feb;107(1):102760.
88. Waltenspül M, Häller T, Ernstbrunner L, Wyss S, Wieser K, Gerber C. Long-term results after posterior open glenoid wedge osteotomy for posterior shoulder instability associated with excessive glenoid retroversion. *J Shoulder Elbow Surg*. 2022 Jan;31(1):81-9.
89. Cognetti DJ, Hughes JD, Kay J, Chasteen J, Fox MA, Hartzler RU, et al. Bone Block Augmentation of the Posterior Glenoid for Recurrent Posterior Shoulder Instability Is Associated With High Rates of Clinical Failure: A Systematic Review. *Arthroscopy*. 2022 Feb;38(2):551-63 e5.
90. Mojica ES, Schwartz LB, Hurley ET, Gonzalez-Lomas G, Campbell KA, Jazrawi LM. Posterior glenoid bone block transfer for posterior shoulder instability: a systematic review. *J Shoulder Elbow Surg*. 2021 Dec;30(12):2904-9.
91. Aydin N, Enes Kayaalp M, Asansu M, Karaismailoglu B. Treatment options for locked posterior shoulder dislocations and clinical outcomes. *EFORT Open Rev*. 2019 May;4(5):194-200.
92. Seppel G, Braun S, Imhoff AB. Neglected Posterior Dislocations and Treatment Modalities. In: Doral MN, Karlsson J, editors. *Sports Injuries: Prevention, Diagnosis, Treatment and Rehabilitation*. Berlin, Heidelberg: Springer Berlin Heidelberg; 2015. p. 193-204.
93. Mitchell BC, Siow MY, Carroll AN, Pennock AT, Edmonds EW. Clinical Outcomes, Survivorship, and Return to Sport After Arthroscopic Capsular Repair With Suture Anchors for Adolescent Multidirectional Shoulder Instability: Results at 6-Year Follow-up. *Orthop J Sports Med*. 2021 Feb;9(2):2325967121993879.
94. Harada Y, Iwahori Y, Kajita Y, Takahashi R, Yokoya S, Sumimoto Y, et al. Return to sports after arthroscopic bankart repair on the dominant shoulder in overhead athletes. *J Orthop Sci*. 2022 Nov;27(6):1240-5.
95. Williams HLM, Evans JP, Furness ND, Smith CD. It's Not All About Redislocation: A Systematic Review of Complications After Anterior Shoulder Stabilization Surgery. *Am J Sports Med*. 2019 Nov;47(13):3277-83.
96. Hamada H, Sugaya H, Takahashi N, Matsuki K, Tokai M, Ueda Y, et al. Incidence of Axillary Nerve Injury After Arthroscopic Shoulder Stabilization. *Arthroscopy*. 2020 Jun;36(6):1555-64.
97. Rodeo SA, Forster RA, Weiland AJ. Neurological complications due to arthroscopy. *J Bone Joint Surg Am*. 1993 Jun;75(6):917-26.
98. Owens BD, Harrast JJ, Hurwitz SR, Thompson TL, Wolf JM. Surgical trends in Bankart repair: an analysis of data from the American Board of Orthopaedic Surgery certification examination. *The American journal of sports medicine*. 2011;39(9):1865-9.
99. Griesser MJ, Harris JD, McCoy BW, Hussain WM, Jones MH, Bishop JY, et al. Complications and re-operations after Bristow-Latarjet shoulder stabilization: a systematic review. *J Shoulder Elbow Surg*. 2013 Feb;22(2):286-92.

Cite this
paper as



Manthas S, Kotsalis I, Oikonomou L. Shoulder instability: a brief review. *Acta Orthop Trauma Hell* 2024; 75(1): 48-52.

ACTA

Young
Scientists'
Pages

Psychological support for the patient with spinal cord injury

Panagiota Efthimiou¹, Ioannis S. Benetos², Dimitrios-Sergios Evangelopoulos³, John Vlamis⁴

¹Postgraduate Training Program, Third Department of Orthopaedic Surgery, National and Kapodistrian University of Athens, KAT Hospital, Athens, Greece

²Third Department of Orthopaedic Surgery, NKUA, KAT Hospital, Athens, Greece

³Third Department of Orthopaedic Surgery, NKUA, KAT Hospital, Athens, Greece

⁴Third Department of Orthopaedic Surgery, NKUA, KAT Hospital, Athens, Greece

Abstract

Reality after spinal cord injury (SCI) is a life-long adjustment. Although much of the effort is focused on the motor, sensory, and autonomic dysfunctions, SCI can have very serious psychosocial consequences on affected individuals. Research shows that many with sudden onset SCI will exhibit extreme negative emotions which impair psychological as well as social integration after injury. Also, people with SCI are at a higher risk of developing anxiety, depression, and post-traumatic stress disorder among others, with psychological and social factors playing a major role in incidence and progression. The purpose of this study was to review the common psychosocial consequences for people after SCI and the current psychosocial challenges within the SCI population, as well as to provide considerations that promote quality of life from a biopsychosocial perspective. A review of the current literature was performed using the online Pubmed and Google Scholar databases and the PRISMA guidelines. Relevant analyses helped to derive conclusions that may benefit the psychological rehabilitation of SCI patients. Patients with robust psychological support seem to have better physical rehabilitation rates, less emotional and behavioral problems, less alcohol/drug abuse, higher rates of reintegration and engaging in meaningful activities and less suicide attempts.

Keywords: spinal cord injury, psychological support, rehabilitation, mental health

Introduction

A spinal cord injury (SCI) damages more than the spinal cord; the lives of people with SCI as well as their support system are never the same (1, 2). Depending upon the height and severity of SCI, medical comorbidities include not only paralysis but also bradycardia, neuro-

genic orthostatic hypotension, circulatory hypokinesia, adaptive cardiomyopathy, neurogenic restrictive lung disease, neurogenic obstructive lung disease, obstructive sleep apnea, neuropathic pain, spasticity, reflex neurogenic bladder, reflex neurogenic bowel, neurogenic erectile dysfunction, neurogenic infertility, neuro-

Corresponding
Author

Panagiota Efthimiou

Postgraduate Training Program, 3rd Department of Orthopaedic Surgery, NKUA
KAT Hospital, Athens, Greece

Email: efpanagiota064@gmail.com

Tel: +30-6980960162

genic skin, neurogenic obesity, heterotopic ossification, osteopenia/osteoporosis, and the metabolic syndrome including diabetes mellitus, dyslipidemia and hypertension (3). George Engel proposed a “biopsychosocial” model, which introduced a philosophical and practical approach to clinical care (4), and, from a practical standpoint, expanded the standard medical model enabling multiple disciplines to approach the multifaceted components of the “person” and “social environment” (5) that directly affect subjective wellbeing and overall outcomes in the context of medical problems. For instance, problems with mood, relationships, or personal sense of meaning can result in maladaptive behaviors (6) (i.e. alcohol misuse) that can escalate into physical, social, and functional impairments, compounding challenges in an already difficult circumstance (7). Emotional psychosomatic complications are already under robust research, such as pain, that has already been proposed to ameliorate by psychological interventions (8).

Although bio-psycho-social components cannot be fully separated, the purpose of this study was to review the common psychosocial consequences for people after SCI and the current psychosocial challenges within the SCI population, as well as to provide considerations that promote quality of life from a biopsychosocial perspective. A review of the current literature was performed using the online Pubmed and Google Scholar databases and the PRISMA guidelines and by using the following key words: “spinal cord injury rehabilitation”, “psychological support spinal cord injury”, “psychological rehabilitation spinal cord injury”, “support group spinal cord injury”, “depression spinal cord injury”, “suicide spinal cord injury”, “mental hospitalization spinal cord injury”, “spinal cord injury mental health”. The primary search revealed 3190 titles, which were screened for relevance, eligibility and diversity, and 63 remained. Exclusion criteria included non-human subjects (6), non-English language (5) and non-full text availability (15). Finally, 37 original research articles were included in this review (Figure 1).

Discussion

Research has shown that individuals who have sustained a spinal cord injury can experience strong and abrupt variations in their emotional state (9). With regard to psychological problems, reported prevalence

rates are 14% for post-traumatic stress disorder (PTSD) (10), 20–25% for clinically significant anxiety and 30–40% for depression (11), with increased suicidal ideation in many; furthermore, these can persist over significant periods of time (12). Compared to the general population, people with SCI experience higher levels of distress and lower levels of life satisfaction on average (13). Although many patients with SCI adapt to the new realities given time, roughly 10% experience increased psychological symptoms which can possibly be mollified by early interventions emphasizing coping strategies (14). Such early adjustments have been shown to increase life satisfaction in some patients (15).

On the negative side of the spectrum, inability to adjust has been linked to mortality. Suicide is at least 3 times more common in individuals with SCI than in individuals without SCI, and anxiety and PTSD are at least twice as prevalent (16). Substance use and abuse is also significantly more prevalent (17). However, even though individuals with SCI usually have contact with health care professionals, these mental health disorders are often not recognized and therefore often not addressed optimally, perhaps because of the other more obvious physical health impairments caused by SCI and the stigma that surrounds mental health disorders (18). An important cohort of people with SCI suffering from emotional distress, mental health problems and substance abuse is the United States of America war veterans. Studies show that 40% of veterans received at least one mental disorder diagnosis, most commonly depressive disorder (19%), posttraumatic stress disorder (12%), and substance or alcohol use disorders (11%) (19). Several patient characteristics predicted mental disorders, including age, racial minority identity, non-traumatic SCI etiology, and incomplete vs. complete injury. Mental disorders were associated with greater impairment from health and mental health-related problems and less satisfaction with life (19). A 2007 study showed that among the veterans with SCI and depression diagnoses (20), 70% were also diagnosed with another psychiatric illness, with post-traumatic stress disorder and other anxiety disorders being the most common. Veterans with SCI and depression had more healthcare visits and received more pharmaceutical prescriptions than their counterparts without depression (20).

Social interactions

But the question of perception of self after SCI is not only subjective to a mirror. Social interactions, stigma and prejudice contribute to the aforementioned psychological issues. Ableism is the disability-based discrimination that results from conceptualizing able-bodied people as “normal” and superior to people with disabilities. Despite its presence, stigma and bias towards people with disabilities are studied much less frequently than other attitude-relevant domains such as age, race, or gender. Although bias can influence judgment and actions in either a positive or negative direction (21, 22), studies revealed a general negativity towards people with disabilities (23, 24)—a negative attitude that people with disabilities expressed themselves (23). Misconceptions about the lived experiences for people with SCI are regrettably common and can inadvertently influence an emotional bias (e.g., prejudice), cognitive bias (e.g., stereotype), or behavioral bias (e.g., discrimination). These can result in self-stigma, public-stigma, or professional or institutional stigma. Self-stigma is when a person sees themselves in a stigmatized way. Research shows that self-stigma appears to change over the course of injury (25). Self-stigma is most prevalent during the first two to three years of SCI (26), although the experience of stigma often persists over the course of one’s lifetime (27). Early models of disability argued that disability was a static experience, and concerns waned after one “adapted to disability” (2). Although the majority of psychosocial consequences are most intense immediately after SCI (26, 28), and they qualitatively differ between immediate and long-term effects of SCI, many psychosocial consequences remain consistent throughout one’s life. A static view of living with disability neglects the reality of dynamic and continual psychosocial challenges over time (29). SCI is a permanent condition requiring lifelong, daily adaptations for both the person with SCI and those caring for them (30).

Support system interactions

Living with SCI often forces individuals to re-evaluate and re-construct their personal and social goals and identities in their family and social systems as a result of transitioning to more dependent-functioning and changes in emotional, psychological, economic, envi-

ronmental, and social stressors (5, 30, 31, 32, 33, 34). Such physical limitations of SCI may initially disrupt the original way in which spouses or family members interact or meet traditional expectations (2, 5, 35). Despite these new and challenging stressors, individuals with SCI and their family members can better adapt to life with SCI by learning to accept the disability, staying solution focused (36, 37), accentuate abilities and values [24], and utilize socialization and supportive communication (30, 33, 34). Depression is a common problem for women with SCI, and many do not receive treatment, particularly psychological treatment (38), which can have an impact on the underlying support system. Families and friends of SCI survivors often have to work with a depressed individual.

Importantly, people with spinal cord injury (SCI) have to fight with their own and societal attitudes and stereotypes that limit sexuality to the physiological functions of genitalia, phallogocentric primacy of sexual pleasure, and sexual attractiveness of perfect bodies, thus affecting spousal connection. Research has provided with psychoeducational initiatives that try to meet the sexual needs of people with SCI and their partners, whilst providing adequate education and psychological support, involving partners, and creating a space to talk among peers (39). Results suggested that women with SCI experience greater symptom bother in certain areas, but that patterns of symptom bother across menopause and transition through menopause and age at final menstrual period (FMP) is similar to their peers. Larger studies are needed to examine menopause outcomes with respect to level of injury and completeness of injury. These findings provide a framework that women with SCI and their health care providers can use to address the menopause transition and highlights the importance of multidisciplinary involvement to maximize health and wellbeing during this transition (40).

Compared with the general population, low serum total testosterone concentration occurs earlier in life in men with SCI, at a higher prevalence by decade of life, and their age-related decline in circulating total testosterone concentration is greater (41).

Understandably, not all partners cope with SCI. Studies show a divorce rate of 17% [95% CI: 13%–20.9%] after SCI in a sample of Iranian population. The protective influence of age in maintenance of marriage

was only detected in men, which proposes existence of a sexual polymorphism in the role of age. Divorce rate was similar between two genders and injury characteristics were not related to divorce rate as well (42).

Employment

Studies have shown that life satisfaction is positively correlated with employment, regardless of income, as vocational outcomes predict life satisfaction (29) and longevity (43, 44). Employment is especially impactful on the quality of life for people with SCI (33), notably to enhance self-esteem, foster positive role model experiences, promote optimism, positive coping, and increase motivation. Moreover, people with SCI who are employed seem to advance in psychological adjustment compared to those who are unemployed (43, 45). Psychosocial problems for persons with SCI are mainly associated with financial hardship due to unemployment and the high cost of living, followed by difficulties with transportation, house modification, education, marriage, social communication, sports, and entertainment. Psychological problems include sadness, depression, irritability/anger, suicidal thoughts, and a lack of self-confidence. The levels of the aforementioned problems differ with respect to sex (46).

COVID-19

People living with SCI during the COVID-19 pandemic experienced a variety of personal physical, psychological, and social challenges, each of which could disrupt daily functioning and quality of life. Increased utilization of telehealth is recommended to support continued engagement in rehabilitation, and foster connection and community amongst others with SCI and health professionals (47). A survey of people with SCI who were in active in-patient rehabilitation from two SCI Rehabilitation Centers in Bangladesh showed that participants reported high levels of knowledge, adoption of positive attitudes, and the practice of positive health advisory behaviors related to COVID-19 prevention procedures. However, high levels of depression, anxiety, and stress were also reported, while women and younger participants were more likely to have high Knowledge, Attitude and Behavioral practices (KAP), whereas those living in rural areas and

with literacy challenges were less likely to report high knowledge scores (48). A study exploring the impacts of COVID-19 on aspects of the lives of individuals living with SCI in Nepal reported also that the pandemic has tremendously impacted the physical, mental, social, and economic aspects of the lives of individuals with SCI (49). These, in turn, could impede the functioning and well-being of this population. The utilization of telehealth to provide education, psychosocial support, social awareness programs, and the provision of essential medical supplies appears necessary to maintain and improve the well-being of individuals with SCI during this pandemic. Future studies using an in-depth interview approach and psychosocial interventions are recommended (49).

Treating Psychological Issues in SCI- where are we now?

People with SCI first come in contact with hospital personnel - and their journey in the muddy waters of SCI reality starts there. Research suggests that even small adjustments in hospital routine, such as Nursing Assessment of Psychological Status Questionnaires, can be valid and reliable, while training nurses to use this instrument may help to enhance good emotional care of patients (50). Also, rehabilitation programs should focus on defining critical terms such as self-management and intentionally describe the components included in the self-management programs, especially those informed by theory, because this is rarely done in the current scientific literature (51). Comparative studies of different self-management programs are needed to identify those best suited to certain patient characteristics (i.e., sociodemographic, clinical), while contextual research in the form of qualitative designs will help clinicians in identifying how best to tailor self-management programs to unique clusters of individuals with SCI using effective messaging, educational, and behavioral strategies (52). Educational support should be a part of a comprehensive rehabilitation program and geared towards addressing the patients' personal and family needs, while educating the community about SCI in order to allow for reintegration into society (53). Of note, reviewed literature suggests that existing guidelines concerning mental health following SCI neglect positive processes of adjustment and sug-

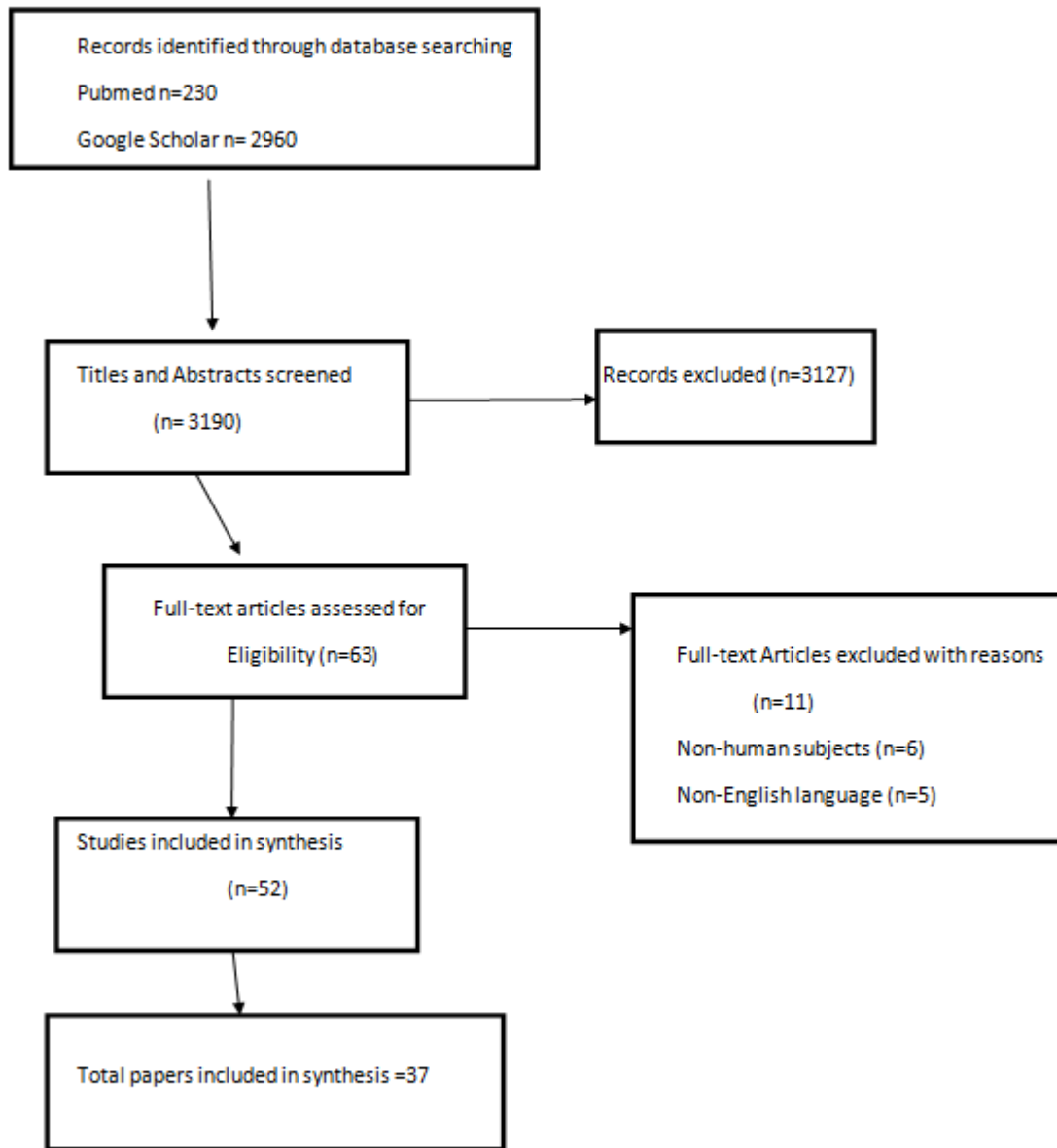


Figure 1. Flowchart of the study

gest this neglect contributes to a deficits-based view of mental health following SCI. Research into “positive” or adjustment-enhancing processes is mostly cross-sectional, heterogenous, and poorly positioned to inform future guideline-development. Researchers should achieve consensus over the operationalization of essential processes and overcome a fixation with “outcomes” to better inform management of mental

health after SCI (54). Also, patients with SCI who present with PTSD symptoms in the acute trauma care setting should be provided with specific cognitive behavioral interventions (55). Interventions for depression in SCI, including a self-management program, should target factors such as self-efficacy and mastery, which could improve secondary medical complications and overall quality of life (56). It is imperative to remain

vigilant in the health profession and acknowledge the need for possible education and training (e.g., coping strategies, communication skills training) as well as counseling prior to discharge to assist individuals with SCI and family caregivers with adaptation to a new life post-injury (34).

Psychological support is imperative in people with SCI. Mental health providers are the cornerstone of such approaches, and research demonstrates the practicality of cognitive behavioral therapy, which consists of educating patients about their pain, focusing in techniques on how to cope with their pain, such as relaxation training, and how to implement these cognitive coping techniques in real-life situations (57). This form of psychological therapy has been shown to successfully reduce depression and anxiety symptoms in individuals with SCI (9, 58, 59, 60). Several studies have examined if cognitive behavioral therapy is a useful treatment for reducing pain after SCI (58, 59, 61, 62). Heutink *et al.*, carried out a multicenter randomized control trial on the use of cognitive behavioral therapy for 10 weeks in individuals with SCI-induced neuropathic pain (62). They assessed pain intensity and pain-related disability as well as anxiety, depression, and life satisfaction before treatment and 3- and 6-months after the intervention started. They found that the intervention group, but not the control group, displayed a significant decrease in pain intensity and pain-related disability at the 3-month timepoint. The intervention group also displayed a decrease in anxiety and an increase in participation in activities at the 3- and 6-month timepoints compared to baseline. Importantly, the participants stated they would recommend this type of treatment program to others but suggested it be offered earlier following SCI. Burns *et al.*, measured the influence of 10-weeks of cognitive behavioral therapy paired with either group exercise or guided relaxation in individuals with SCI-induced neuropathic pain (61). They found that although this treatment program did not reduce pain severity, it did help individuals cope with their pain, lessened pain interference with daily activities, and improved their sense of control. Many of the studies using cognitive behavioral therapy as a treatment for SCI-induced pain, anxiety, and/or depression suggest that “refresher” courses to reinforce skills learned during the treatment program

are important to maintain efficacy of this treatment.

On a different note, mindfulness-based interventions are different from cognitive behavioral therapy as these interventions aim to facilitate present-moment awareness and acceptance, rather than attempting to change behavioral and psychological responses (63). A systemic review by Streijger *et al.*, on the use of mindfulness-based interventions in individuals with SCI pain found a variation in results on this therapy’s efficacy in the 5 studies reviewed (63). One study reported a significant reduction in SCI pain, while the others reported no change. Additionally, 4/5 studies reported a significant reduction in symptoms of depression while 3/5 reported reductions in anxiety. Authors suggest that while mindfulness might not improve SCI pain, it could be used to reduce incidence of depression and anxiety in individuals with SCI-induced co-morbid pain and mood disorders. However, two recent studies using different forms of mindfulness interventions [meditation (64) and yoga (65)] in individuals with SCI, demonstrated positive outcomes on pain, anxiety, and depression. Zanca *et al.*, used a 4-week clinical meditation and imagery program, which included mindfulness, meditation, and guided imagery in individuals with chronic nociceptive and/or neuropathic SCI pain (64). They measured pain outcomes as well as depressive symptomology and perceived stress. Their results indicated that the intervention group showed a greater decrease in depressive symptomology and worst pain intensity over the last week and a greater increase in perceived control over pain. Although their results did not reach statistical significance, the authors note that it was a pilot study and that their sample size was small. Chalageri *et al.*, used 1 month of *raja yoga*, which is a meditation technique, and found a significant decrease in numeric pain rating, anxiety and depression, and perceived stress scale in individuals with SCI that received the *raja yoga* treatment compared to those that received conventional rehabilitation (65). Additionally, they saw a significant increase in quality-of-life scores in the intervention group. Importantly, SCI patients that have developed neurogenic bowel and/or neurogenic bladder report psychological distress, anxiety, and embarrassment about their abdominal pain, constipation, incontinence, or need for catheterization. Prescribing cognitive behavioral therapy, meditation,

or yoga/exercise may improve the mental strain of neurogenic bowel and neurogenic bladder inhibiting the hyperactive autonomic nervous system as suggested by functional gastrointestinal disorders models, such as irritable bowel syndrome (57). Interestingly, in an era of microbiome and epigenetics, studies show the importance of understanding the mechanisms underlying the relationship between SCIs, gut dysbiosis and psychological stress, which could contribute to the development of novel therapeutic strategies to improve SCI patients' quality of life (66).

Although age and education do not seem to significantly influence psychological empowerment, self-esteem can have a direct effect on psychological empowerment, whereas physical impairment duration, functional ability, and social support had indirect effects through self-esteem. These four predictors explained 64% of the total variance in a model of psychological empowerment for people with spinal cord injury (67), and these findings can be used as a guideline for developing appropriate interventions to promote psychological empowerment among patients with spinal cord injury (67).

In conclusion, rehabilitation is a lifelong journey for people with SCI. Psychological support and mental health is a balance that should never be lost. Research has shown that rehabilitation professionals have a central role in promoting purpose in life as a means of increasing longevity, since by assessing personality

factors predictive of specific causes of mortality, those at risk may be targeted for cause-specific prevention strategies (68). Specific targets and socioeconomic groups, such as attempts to improve the outcomes of Veterans with SCI, should focus on a tailored approach that emphasizes patients' demographic, medical, and psychosocial assets (e.g., building their sense of self-esteem or increasing their feelings of mastery), while providing services targeted to their specific limitations (e.g., reducing depression and anxiety) (69). Also, disability-sensitive and affordable depression treatment must be made available to everyone, especially women with SCI (38). Studies of testosterone replacement therapy in men with SCI should assist in determining the possible functional and clinical benefits from reversing low serum total testosterone concentration (41). As far as the COVID-19 pandemic is concerned, future studies using an in-depth interview approach and psychosocial interventions are recommended (49). SCI deeply affects multiple dimensions of a person's psychological and social well-being, with vast consequences. Health care providers are first contacts of newly acquired SCI people; so, the accountability to mindfully consider the individuality and associated psychosocial traits remains imperative.

Conflict of interest

The authors declare no conflicts of interest.

References

- Jenkins HT, Cosco TD. Spinal cord injury and aging: an exploration of the interrelatedness between key psychosocial factors contributing to the process of resilience. *Health Psychol Behav Med.* 2021;9(1):315-21.
- Jeppsson Grassman E, Holme L, Taghizadeh Larsson A, Whitaker A. A long life with a particular signature: life course and aging for people with disabilities. *J Gerontol Soc Work.* 2012;55(2):95-111.
- Gater DR. Neurogenic bowel and bladder evaluation strategies in spinal cord injury: New directions. *J Spinal Cord Med.* 2020;43(2):139-40.
- Engel GL. The need for a new medical model: a challenge for biomedicine. *Science.* 1977;196(4286):129-36.
- Weber L, Voldsgaard NH, Holm NJ, Schou LH, Biering-Sorensen F, Moller T. Exploring the contextual transition from spinal cord injury rehabilitation to the home environment: a qualitative study. *Spinal Cord.* 2021;59(3):336-46.
- Craig A, Tran Y, Guest R, Middleton J. Trajectories of Self-Efficacy and Depressed Mood and Their Relationship in the First 12 Months Following Spinal Cord Injury. *Arch Phys Med Rehabil.* 2019;100(3):441-7.

7. Budd MA, Gater DR, Jr., Channell I. Psychosocial Consequences of Spinal Cord Injury: A Narrative Review. *J Pers Med.* 2022;12(7).
8. Eller OC, Willits AB, Young EE, Baumbauer KM. Pharmacological and non-pharmacological therapeutic interventions for the treatment of spinal cord injury-induced pain. *Front Pain Res (Lausanne).* 2022;3:991736.
9. Kennedy P, Duff J, Evans M, Beedie A. Coping effectiveness training reduces depression and anxiety following traumatic spinal cord injuries. *Br J Clin Psychol.* 2003;42(Pt 1):41-52.
10. Kennedy P, Evans MJ. Evaluation of post traumatic distress in the first 6 months following SCI. *Spinal Cord.* 2001;39(7):381-6.
11. Galvin LR, Godfrey HP. The impact of coping on emotional adjustment to spinal cord injury (SCI): review of the literature and application of a stress appraisal and coping formulation. *Spinal Cord.* 2001;39(12):615-27.
12. Kennedy P, Rogers BA. Anxiety and depression after spinal cord injury: a longitudinal analysis. *Arch Phys Med Rehabil.* 2000;81(7):932-7.
13. Post MW, van Leeuwen CM. Psychosocial issues in spinal cord injury: a review. *Spinal Cord.* 2012;50(5):382-9.
14. Bonanno GA, Kennedy P, Galatzer-Levy IR, Lude P, Elfstrom ML. Trajectories of resilience, depression, and anxiety following spinal cord injury. *Rehabil Psychol.* 2012;57(3):236-47.
15. van Leeuwen CM, Post MW, van der Woude LH, de Groot S, Smit C, van Kuppevelt D, et al. Changes in life satisfaction in persons with spinal cord injury during and after inpatient rehabilitation: adaptation or measurement bias? *Qual Life Res.* 2012;21(9):1499-508.
16. Giannini MJ, Bergmark B, Kreshover S, Elias E, Plummer C, O'Keefe E. Understanding suicide and disability through three major disabling conditions: Intellectual disability, spinal cord injury, and multiple sclerosis. *Disabil Health J.* 2010;3(2):74-8.
17. Lusilla-Palacios P, Castellano-Tejedor C. Spinal cord injury and substance use: a systematic review. *Adicciones.* 2015;27(4):294-310.
18. Bombardier CH, Azuero CB, Fann JR, Kautz DD, Richards JS, Sabharwal S. Management of Mental Health Disorders, Substance Use Disorders, and Suicide in Adults with Spinal Cord Injury: Clinical Practice Guideline for Healthcare Providers. *Top Spinal Cord Inj Rehabil.* 2021;27(2):152-224.
19. McDonald SD, Mickens MN, Goldberg-Looney LD, Mutchler BJ, Ellwood MS, Castillo TA. Mental disorder prevalence among U.S. Department of Veterans Affairs outpatients with spinal cord injuries. *J Spinal Cord Med.* 2018;41(6):691-702.
20. Ullrich PM, Smith BM, Blow FC, Valenstein M, Weaver FM. Depression, healthcare utilization, and comorbid psychiatric disorders after spinal cord injury. *J Spinal Cord Med.* 2014;37(1):40-5.
21. *The Oxford Handbook of Rehabilitation Psychology.* Kennedy P, editor: Oxford University Press; 2012 21 Nov 2012.
22. Budd MA, Haque OS, Stein MA. Biases in the evaluation of self-harm in patients with disability due to spinal cord injury. *Spinal Cord Ser Cases.* 2020;6(1):43.
23. Nosek BA, Smyth FL, Hansen JJ, Devos T, Lindner NM, Ranganath KA, et al. Pervasiveness and correlates of implicit attitudes and stereotypes. *European Review of Social Psychology.* 2007;18(1):36-88.
24. Tyrrell AC, Hetz SP, Barg CJ, Latimer AE. Exercise as stigma management for individuals with onset-controllable and onset-uncontrollable spinal cord injury. *Rehabil Psychol.* 2010;55(4):383-90.
25. Monden KR, Philippus A, MacIntyre B, Welch A, Sevigny M, Draganich C, et al. The impact of stigma on psychosocial outcomes following spinal cord injury: A cross-sectional analysis of stigma-mediated relationships. *Rehabil Psychol.* 2021;66(2):202-12.
26. Manns PJ, Chad KE. Components of quality of life for persons with a quadriplegic and paraplegic spinal cord injury. *Qual Health Res.* 2001;11(6):795-811.
27. Link B, Phelan JC. Conceptualizing Stigma. *Annual Review of Sociology.* 2001;27:363-85.
28. Link BG, Phelan JC. Conceptualizing Stigma. *Annual Review of Sociology.* 2001;27(1):363-85.
29. Jørgensen S, Hedgren L, Sundelin A, Lexell J. Global and domain-specific life satisfaction among older adults with long-term spinal cord injury. *J Spinal Cord Med.* 2021;44(2):322-30.
30. Charlifue SB, Botticello A, Kolakowsky-Hayner SA, Richards JS, Tulsy DS. Family caregivers of individuals with spinal cord injury: exploring the stresses and bene-

- fits. *Spinal Cord*. 2016;54(9):732-6.
31. Engblom-Deglmann ML, Hamilton J. The Impact of Spinal Cord Injury on the Couple Relationship: A Grounded Theory Exploration of the Adjustment Process. *Journal of Couple & Relationship Therapy*. 2020;19(3):250-75.
 32. Tough H, Gross-Hemmi M, Eriks-Hoogland I, Fekete C. Pathways to loneliness: a mediation analysis investigating the social gradient of loneliness in persons with disabilities in Switzerland. *Int J Equity Health*. 2021;20(1):261.
 33. DeSanto-Madeya S. Adaptation to spinal cord injury for families post-injury. *Nurs Sci Q*. 2009;22(1):57-66.
 34. Jeyathevan G, Cameron JI, Craven BC, Munce SEP, Jaglal SB. Re-building relationships after a spinal cord injury: experiences of family caregivers and care recipients. *BMC Neurol*. 2019;19(1):117.
 35. Chan RC. How does spinal cord injury affect marital relationship? A story from both sides of the couple. *Disabil Rehabil*. 2000;22(17):764-75.
 36. Burkhart L, Kale IO, LaVela SL. Grief and Loss Among Veterans With Spinal Cord Injury: A Qualitative Study. *Rehabil Nurs*. 2021;46(5):270-8.
 37. Pollard C, Kennedy P. A longitudinal analysis of emotional impact, coping strategies and post-traumatic psychological growth following spinal cord injury: a 10-year review. *Br J Health Psychol*. 2007;12(Pt 3):347-62.
 38. Robinson-Whelen S, Taylor HB, Hughes RB, Wenzel L, Nosek MA. Depression and depression treatment in women with spinal cord injury. *Top Spinal Cord Inj Rehabil*. 2014;20(1):23-31.
 39. Federici S, Artegiani F, Pigliautile M, Antonelli P, Diotallevi D, Ritacco I, et al. Enhancing Psychological Sexual Health of People With Spinal Cord Injury and Their Partners in an Italian Unipolar Spinal Unit: A Pilot Data Study. *Front Psychol*. 2019;10:754.
 40. Kalpakjian CZ, Quint EH, Bushnik T, Rodriguez GM, Terrill MS. Menopause characteristics and subjective symptoms in women with and without spinal cord injury. *Arch Phys Med Rehabil*. 2010;91(4):562-9.
 41. Bauman WA, La Fountaine MF, Spungen AM. Age-related prevalence of low testosterone in men with spinal cord injury. *J Spinal Cord Med*. 2014;37(1):32-9.
 42. Merghati Khoi E, Latifi S, Rahdari F, Shakeri H, Arman F, Koushki D, et al. The Effect of Injury-Related Characteristics on Changes in Marital Status after Spinal Cord Injury. *Iran J Public Health*. 2015;44(10):1395-402.
 43. Ottomanelli L, Lind L. Review of critical factors related to employment after spinal cord injury: implications for research and vocational services. *J Spinal Cord Med*. 2009;32(5):503-31.
 44. Wilbanks SR, Ivankova NV. Exploring factors facilitating adults with spinal cord injury rejoining the workforce: a pilot study. *Disabil Rehabil*. 2015;37(9):739-49.
 45. Goetz LL, Ottomanelli L, Barnett SD, Sutton B, Njoh E. Relationship Between Comorbidities and Employment Among Veterans with Spinal Cord Injury. *Top Spinal Cord Inj Rehabil*. 2018;24(1):44-53.
 46. Khazaeipour Z, Norouzi-Javidan A, Kaveh M, Khanzadeh Mehrabani F, Kazazi E, Emami-Razavi SH. Psychosocial outcomes following spinal cord injury in Iran. *J Spinal Cord Med*. 2014;37(3):338-45.
 47. Hearn JH, Rohn EJ, Monden KR. Isolated and anxious: A qualitative exploration of the impact of the COVID-19 pandemic on individuals living with spinal cord injury in the UK. *J Spinal Cord Med*. 2022;45(5):691-9.
 48. Hossain MA, Hossain KMA, Sakel M, Kabir MF, Saunders K, Faruqui R, et al. Knowledge, Attitudes, Behavioural Practises, and Psychological Impact Relating to COVID-19 Among People Living With Spinal Cord Injury During In-Patient Rehabilitation in Bangladesh. *Front Neurol*. 2021;12:739354.
 49. Bhattarai M, Limbu S, Sherpa PD. Living with spinal cord injury during COVID-19: a qualitative study of impacts of the pandemic in Nepal. *Spinal Cord*. 2022;60(11):984-9.
 50. Smyth C, Spada MM, Coultry-Keane K, Ikkos G. The Stanmore Nursing Assessment of Psychological Status: Understanding the emotions of patients with spinal cord injury. *J Spinal Cord Med*. 2016;39(5):519-26.
 51. Modi AC, Pai AL, Hommel KA, Hood KK, Cortina S, Hilliard ME, et al. Pediatric self-management: a framework for research, practice, and policy. *Pediatrics*. 2012;129(2):e473-85.
 52. McIntyre A, Marrocco SL, McRae SA, Sleeth L, Hitzig S, Jaglal S, et al. A Scoping Review of Self-Management Interventions Following Spinal Cord Injury. *Top Spinal Cord Inj Rehabil*. 2020;26(1):36-63.
 53. Khazaeipour Z, Abouie A, Zarei F, Mirzaaghaie H,

- Abd-Mousavi A, Salehi-Nejad A, et al. Personal, family and societal educational needs assessment of individuals with spinal cord injury in Iran. *Neurosciences (Riyadh)*. 2018;23(3):216-22.
54. Sandalic D, Arora M, Pozzato I, Simpson G, Middleton J, Craig A. A Narrative Review of Research on Adjustment to Spinal Cord Injury and Mental Health: Gaps, Future Directions, and Practice Recommendations. *Psychol Res Behav Manag*. 2022;15:1997-2010.
 55. Warren AM, Reynolds M, Driver S, Bennett M, Sikka S. Posttraumatic Stress Symptoms Among Spinal Cord Injury Patients in Trauma: A Brief Report. *Top Spinal Cord Inj Rehabil*. 2016;22(3):203-8.
 56. Munce SE, Straus SE, Fehlings MG, Voth J, Nugaeva N, Jang E, et al. Impact of psychological characteristics in self-management in individuals with traumatic spinal cord injury. *Spinal Cord*. 2016;54(1):29-33.
 57. Waters SJ, McKee DC, Keefe FJ. Cognitive behavioral approaches to the treatment of pain. *Psychopharmacol Bull*. 2007;40(4):74-88.
 58. Perry KN, Nicholas MK, Middleton JW. Comparison of a pain management program with usual care in a pain management center for people with spinal cord injury-related chronic pain. *Clin J Pain*. 2010;26(3):206-16.
 59. Norrbrink Budh C, Kowalski J, Lundeberg T. A comprehensive pain management programme comprising educational, cognitive and behavioural interventions for neuropathic pain following spinal cord injury. *J Rehabil Med*. 2006;38(3):172-80.
 60. Craig AR, Hancock K, Chang E, Dickson H. Immunizing against depression and anxiety after spinal cord injury. *Arch Phys Med Rehabil*. 1998;79(4):375-7.
 61. Burns AS, Delparte JJ, Ballantyne EC, Boschen KA. Evaluation of an interdisciplinary program for chronic pain after spinal cord injury. *PM R*. 2013;5(10):832-8.
 62. Heutink M, Post MWM, Bongers-Janssen HMH, Dijkstra CA, Snoek GJ, Spijkerman DCM, et al. The CONECISI trial: results of a randomized controlled trial of a multidisciplinary cognitive behavioral program for coping with chronic neuropathic pain after spinal cord injury. *Pain*. 2012;153(1):120-8.
 63. Hearn JH, Cross A. Mindfulness for pain, depression, anxiety, and quality of life in people with spinal cord injury: a systematic review. *BMC Neurol*. 2020;20(1):32.
 64. Zanca JM, Gilchrist C, Ortiz CE, Dyson-Hudson TA. Pilot clinical trial of a clinical meditation and imagery intervention for chronic pain after spinal cord injury. *J Spinal Cord Med*. 2022;45(3):339-53.
 65. Chalageri E, Vishwakarma G, Ranjan RL, Govindaraj R, Chhabra HS. Effect of Raja yoga Meditation on Psychological and Functional Outcomes in Spinal Cord Injury Patients. *Int J Yoga*. 2021;14(1):36-42.
 66. Musleh-Vega S, Ojeda J, Vidal PM. Gut Microbiota-Brain Axis as a Potential Modulator of Psychological Stress after Spinal Cord Injury. *Biomedicines*. 2022;10(4).
 67. Rattanasuk D, Khuwatsamrit K. Causal Model of Psychological Empowerment Among People With Spinal Cord Injury in Thailand. *Orthop Nurs*. 2021;40(3):136-43.
 68. Krause JS, Cao Y, DiPiro N. Psychological factors and risk of mortality after spinal cord injury. *J Spinal Cord Med*. 2020;43(5):667-75.
 69. Myaskovsky L, Gao S, Hausmann LRM, Bornemann KR, Burkitt KH, Switzer GE, et al. How Are Race, Cultural, and Psychosocial Factors Associated With Outcomes in Veterans With Spinal Cord Injury? *Arch Phys Med Rehabil*. 2017;98(9):1812-20 e3.

Cite this
paper as



Efthimiou P, Benetos IS, Evangelopoulos DS. Psychological support for the patient with spinal cord injury. *Acta Orthop Trauma Hell* 2024; 75(1): 54-63.

The role of electrical stimulation in the management of lower urinary track dysfunction following spinal cord lesions

Nikolaos Koutsogeorgis¹, Maria-Eleftheria Evangelopoulos²

¹Postgraduate training program: "Rehabilitation following spinal cord lesions. Spinal pain management", National & Kapodistrian University of Athens, KAT General Hospital of Attica, 2 Nikis Street, Kifisia, 14561, Greece.

²First Department of Neurology, National & Kapodistrian University of Athens, Aiginition Hospital, Athens, Greece. Email: evangelopoulos@yahoo.com

Abstract

Spinal cord lesions are traumatic or non-traumatic. Spinal cord injuries (SCI) may be complete or incomplete and lead to lower urinary track dysfunction (LUTD) in 95%. Multiple sclerosis is the most frequent cause of non-traumatic spinal cord lesions and leads to LUTD in more than 90% of patients 10 years after diagnosis. LUTD usually presents as neurogenic detrusor overactivity and/or detrusor-sphincter dyssynergia where oral medication is considered to be the first line of treatment and intravesical onabotulinum toxin injections the second, but there are side-effects and refractory cases. In addition, LUTD may present as detrusor underactivity where the above treatment options are not effective.

Clearly there is a need for a third line of treatment. In this review, we discuss the feasibility, safety and efficacy of electrical stimulation for the management of neurogenic LUTD, spanning from historic clinical to recent pre-clinical approaches. Neurostimulation methods are used on complete SCI patients, while neuromodulation methods are mostly used on incomplete spinal lesion patients and can be invasive or non-invasive. There is evidence that neuromodulation inhibits the development of neurogenic LUTD when applied shortly after acute spinal cord lesions. More high-quality studies are needed to prove efficacy of neuromodulation on neurogenic LUTD.

Keywords: Urinary track dysfunction, spinal cord injuries

Introduction

Spinal cord (SC) lesions, traumatic or non-traumatic, bring disastrous consequences upon the patients, who, in addition to motor/sensory impairments, deal

with autonomic, sexual, bowel and urinary, disorders. Of the autonomic disorders, the lower urinary track dysfunction (LUTD) poses the most serious threat on health, because of the risk of developing renal failure.

Corresponding
Author

Koutsogeorgis N, MD. MSc Student, Postgraduate training program: "Rehabilitation following spinal cord lesions. Spinal pain management", National & Kapodistrian University of Athens, KAT General Hospital of Attica, 2 Nikis Street, Kifisia, 14561, Greece. E-mail: nikkoutsog@gmail.com

This risk is considerably lower in patients with slowly progressive nontraumatic neurological disorders, such as multiple sclerosis (MS), compared to spinal cord injury (SCI) or spina bifida¹. Mortality due to LUTD has decreased in recent decades (3% of SCI deaths), but LUTD still results in symptoms that significantly impact quality of life² and patients sometimes even prioritize recovery of LUT function above walking^{3,4}.

LUTD is reported in an estimated 95% of suprasacral SCI patients and in more than 90% of children with spina bifida. Over 90% of patients suffering from MS for more than 10 years also report LUTD symptoms and urinary incontinence is considered to be one of the worst aspects of the disease⁵. Other causes for SC lesions include SC compression, due to spondylosis, developmental abnormalities or tumors, and ischemia. In this review, we discuss the pathophysiology of LUTD following SC lesions and the applications of electrical stimulation for bladder control, spanning from historic clinical to recent pre-clinical approaches.

There are two functional (and anatomical) units in the LUT: 1) a reservoir (the urinary bladder) and (2) an outlet (bladder neck/internal urethra smooth muscles and external urethra sphincter striated muscle)⁶. A complex neural network controls a reciprocal relationship between the bladder and sphincter function.

During bladder filling, the sympathetic hypogastric nerves (T11-L2) mediate contraction of the smooth (internal) urethral sphincter and inhibition of the detrusor, while the somatic pudendal nerves (S2-S4) mediate contraction of the striated (external) urethral sphincter. This results in low filling and continence pressure^{6,7}, under the control of the Pontine Continence Center (PCC)¹. The voiding phase is initiated by a conscious decision, when bladder fullness is perceived, given it is socially appropriate⁸. The Pontine Micturition Center (PMC) is then released from the tonic inhibition of higher centers and the parasympathetic pelvic nerves (S2-S4) mediate detrusor contraction accompanied by relaxation of the pelvic floor and of the outlet, resulting in effective bladder emptying, with no post-void residual (PVR) volume⁸. In health individuals, thinly myelinated A δ -fibers are responsible for conveying sensations of bladder filling and normally initiate the micturition reflex, triggered by bladder distension, whereas unmyelinated C-fibers, have a

greater threshold for activation and are thought to normally remain "silent"^{3,6,7}. Spinal cord lesions at cervical or thoracic levels disrupt voluntary control of voiding as well as the normal reflexes that coordinate bladder and sphincter function. Following SCI the bladder is initially areflexic but later becomes hyper-reflexic due to the emergence of a spinal micturition reflex. Studies on animals indicate that LUTD after SCI depends on plasticity of bladder afferent pathways and reorganization of synaptic connections in the SC⁶. Following SC damage, C fibers become mechanosensitive at lower bladder volumes. A segmental spinal reflex then emerges that is mediated by C fiber afferent nerves and results in neurogenic detrusor overactivity (DO).

However, the bladder does not empty efficiently because the detrusor and urethral sphincters contract simultaneously, in a condition termed detrusor-sphincter dyssynergia (DSD), seen in up to 85% of SCI and up to 50% of MS patients⁹. Thus, pressures within the bladder may rise considerably, increasing the risk for vesicoureteral reflux and upper urinary tract damage. These high pressures may even trigger life-threatening dysreflexia episodes on patients suffering from complete SCI above the T7 level^{9,10}. Furthermore, DSD results in bladder wall hypertrophy that causes the course of the distal ureter to become progressively perpendicular to the inner surface of the bladder. The vesicoureteral junction consequently becomes incompetent, permitting reflux of urine^{3,8}. Injury to the conus medullaris, cauda equina, or peripheral nerves results in poor detrusor contractions, termed neurogenic detrusor underactivity (DU) leading to bladder distension, to the point of overflow incontinence.

Neurogenic LUTD can be managed by intermittent catheterization (IC) if proper bladder emptying is impaired. In case of neurogenic DO and/or DSD, oral medications, mostly anticholinergics, are considered to be the first line of treatment, but, because of side effects, only 30% of the patients continue to take the drug one year after initiation, although they have seen some benefit⁹. Intravesical onabotulinum toxin injections is considered to be the second line of treatment, but there are complications, mostly due to the invasive nature of the procedure. Finally, there are some surgical procedures for carefully selected neurological patients that have high complication and morbidity rates¹¹. Clearly

there is a need for a third line of treatment for refractory neurogenic LUTD. Electrical stimulation could be an option. Safety and efficacy of some methods of electrical stimulation have already been proved on non-neurological LUTD patients and could be used to manage different kinds of neurogenic LUTD as well, even though most of them remain off-label, due to the lack of high-quality studies.

After searching Pubmed and Scopus for relevant articles written in English and checking for duplicates, all identified abstracts were imported into Mendeley bibliography management software. The reference lists of included studies and relevant review articles were additionally searched. (Figure 1: Bibliography flowchart)

Discussion

There are different methods of managing neurogenic LUTD by electrical stimulation. Electrical neurostimulation is the typical, direct stimulation of a neuron with an immediate activating effect on the aimed organ and is usually used on complete SCI patients. On the other hand, electrical neuromodulation (NM) is the indirect stimulation that influences, in other words modulates, pre-existing activity in neural pathways. We therefore stimulate a neuron which affects the function of subsequent neurons to inhibit or activate the aimed organ. NM is usually used on non-traumatic spinal cord lesions or on incomplete SCI patients. Mechanisms of action are debatable. Finally, the direct electrical stimulation of neurons with high-frequency currents is used in order to block them from propagating an undesired action potential. Some of these methods are clinically used on patients already for many years, but others have been used experimentally on humans or animals only.

Electrical Neurostimulation

The idea of direct electrical neurostimulation of the LUT came from Functional Electric Stimulation (FES) of denervated skeletal muscles.

Sacral anterior root stimulation (SARS): SARS was developed by Brindley, 40 years ago, to restore urinary and bowel functions of SCI individuals. Stimulation electrodes were surgically disposed on S2 to S5 sacral anterior roots, following laminectomy, in order

to induce detrusor contraction and promote effective (on demand) micturition¹². SARS at different stimulation settings also may enable defecation and erections. Electrical stimuli are evoked by radiofrequency waves from an external stimulator¹¹. Mostly used to restore LUT function, SARS implantation is coupled with sacral differentiation (sectioning of the S2-S5 dorsal roots, a procedure called rhizotomy), to prevent high-pressure DO, and, consequently, promote bladder compliance and prevent incontinence^{12,13}. Posterior S2-S5 rhizotomy also reduced the, LUT mediated, autonomic dysreflexia episodes¹¹ from 43% to 3%¹². Unfortunately, rhizotomy also results in the potentially irreversible loss of spared perineal sensation and function, thus SARS is not performed on patients with MS or incomplete SCI¹².

SARS simultaneously evokes contraction of the urethral sphincter, resulting in emptying the bladder inadequately¹¹. Smooth muscles of the bladder relax more slowly than striated muscles of the urethral sphincter. If intermittent stimulation periods are applied, the urethral muscles will relax but the bladder smooth muscles will keep on contracting, which results in post-stimulus voiding with an intermittent flow pattern of micturition during the stimulation-free intervals^{11,12}. Long term usage of SARS results in increased bladder capacity and compliance, decreased intravesical pressure and quality of life improvement. The implants have been successfully tested with 1.5 Tesla MRI¹². Despite promising results, a decline in implantations was observed. Apart from the required expertise to do this surgery, this decline can be linked to the complication rate (mostly device failures), as well as to the development of mini-invasive alternatives, such as botulinum toxin injections¹¹. Furthermore, some patients prefer to wait for a new solution (spinal cord stimulation, stem-cell therapy, neuroprosthesis etc), while others ideologically reject the implantation of electronic devices¹².

Intravesical electrical stimulation (IVES): Katona et al, on 1975¹⁴ was the first to treat 420 patients with transurethral, intravesical electrotherapy in order to improve the function of their “paralyzed” urinary bladders (“2nd-neuron damage”), by reactivation of the intramural bladder receptors. Normal conscious micturition control was obtained by 314 patients. Eb-

ner et al, on 1992¹⁵ experimentally confirmed on animals that IVES involved a direct activation of bladder mechanoreceptor A δ afferents, while optimal stimulation frequency was 20 Hz.

Direct bladder wall stimulation (DBWS): Starting in 1958, DBWS was developed, again, for complete SC lesions resulting in a decentralized areflexic bladder, by direct stimulation of viable post-ganglionic nerves located in the bladder wall. The electrodes are placed invasively and optimal stimulation frequency is 40 Hz¹⁶. When performed on incomplete sacral SC lesion animals it results in pain and strong skeletal muscle contractions¹⁶. Since Merrill first used the "Mentor" stimulator on 1975, the increasing use of intermittent catheterization for the management of neurogenic DU, made DBWS, as well as IVES, less interesting for clinicians. Nevertheless, efforts are still made for non-migrating electrodes that induce adequate detrusor contractions without any concomitant abdominal wall, leg or anal sphincter contractions¹⁶.

Pelvic nerve stimulation: Pelvic nerve stimulation produced bladder contractions in dogs, but also resulted in co-activation of urethral sphincters. Although it requires lower amplitudes of stimulation than DBWS, application in humans is limited due to the difficulty of electrode placement¹⁰.

Spinal cord stimulation (SCS): It was observed that SCS improved neurogenic DO. A sensory rhizotomy is not required. Most of the studies involved incomplete SCI subjects. Stimulation parameters that have been configured for storage may not be effective for voiding². SCS primarily involved lead placement over the epidural space to manage refractory neuropathic pain. There are commercially available systems that employ epidural SCS to treat chronic neuropathic pain (Abbott, Boston Scientific, Medtronic, Nevro) that are FDA-approved. Permanent electrodes are placed surgically via a laminotomy, only after a successful test period of percutaneous stimulation¹⁷.

Herrity et al¹⁸ found that epidural SCS, with a stimulation frequency of 30Hz, on 5 complete SCI patients, increased efficiency of reflexive voiding from 0-5% to 10-70%. Nevertheless, improvement of LUTD after epidural SCS could be the result of the accompanying step training, since there is an interaction of spinal networks that control bladder and hind limb locomotor

function². Havton et al¹⁹ experimentally demonstrated that non-invasive Transcutaneous Spinal Cord Stimulation (TSCS) over the thoracolumbar spine of neurologically intact rhesus macaques can activate the bladder detrusor muscle, the urethral sphincter and pelvic floor muscles. Havton et al suggested that TSCS could augment LUT function if applied on SCI humans as well. The placement of the transcutaneous electrodes is not fixed, since vertebral level of the tip of the conus medullaris varies extensively between humans. This provides an obvious advantage of the non-invasive transcutaneous approach in comparison to the epidural approach. Intraspinal and trans-spinal SCS approaches have also been examined in animal models of SCI². SCS, in general, activates both afferent and efferent pathways. Thus, apart from the direct effect of SCS resulting in effective micturition, there seems to be a neuromodulation effect as well.

Interferential medium frequency current electrical stimulation (IMFC-ES): IMFC ES is a non-invasive approach for the treatment of LUTD first reported in 1985 by Dougall. The interaction of the medium frequencies, inside the body, produces a low frequency field which stimulates the urinary structures, without any significant adverse reactions. IMFC-ES is applied on pubic and abdominal areas immediately after IC, to prevent voiding during stimulation. Daia et al²⁰ used IMFC-ES on 332 patients shortly after SCI diagnosed, with neurogenic LUTD. IMFC-ES was effective in patients with AIS B/C SCI, since it significantly decreased PVR and incontinence compared with standard care. Patients that exhibit preserved bladder sensitivity were the best beneficiaries. Intentional control of voiding was completely regained by 37 patients after IMFC-ES and only by 13 patients from the control group. This certain study did not allow discriminating between the spontaneous recovery from the spinal shock and the IMFC-ES effect. At 0-5Hz, the IMFC-ES causes innervated skeletal muscles to contract and 5-10Hz may further cause contraction of denervated skeletal muscles. At 11-35Hz, smooth muscles are stimulated and furthermore, 3680Hz can also activate denervated smooth muscles. At 80-100Hz, it relaxes both smooth and skeletal muscles. Thus, IMFC-ES may improve the neural muscular control in neurogenic LUTD in various ways²⁰.

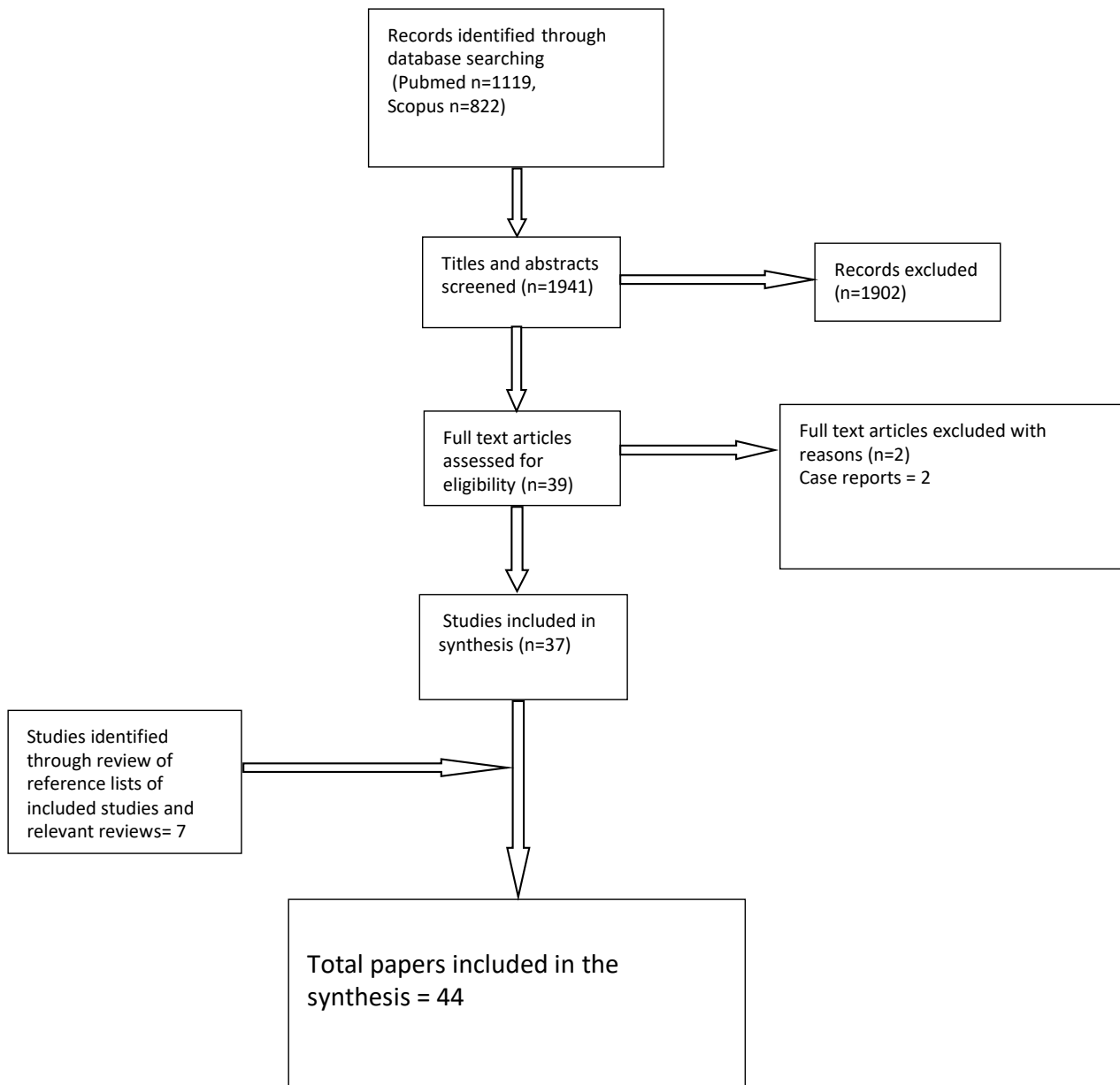


Figure 1. Flowchart of the study.

Electrical Neuromodulation (NM)

Neuromodulation is mostly applied on incomplete SC lesions, non-invasively or invasively. Permanently implanted neuromodulation devices are not commonly used on MS patients because of the need for frequent MRIs, since heating of hardware may occur. Most systems are compatible with 1.5Tesla closed magnetic

field brain MRIs of specific parameters for no longer than 30 minutes, while the stimulator is turned off²¹.

NM was initially used for the treatment of already existing LUT symptoms on non-neurogenic and then on neurogenic patients. We will discuss electrical NM on patients with SC lesions, but since the quality of studies on NM treatment of neurogenic LUTD is not

satisfactory, it is necessary to take some of the RCTs investigating NM of non-neurogenic LUTD patients under consideration. The main outcome may be indicated by different parameters in different studies, such as urinary frequency, number of leakage episodes, or the score of a questionnaire. The most used definition of success after a certain method of therapy is a >50% reduction in the most important symptom²². It was later suggested that application of electrical neuromodulation during the acute phase of SC lesions, mostly SCI, could prevent the development of pathologic reflexes before they lead to OD/DSD. Researchers believe that stimulation of the peripheral sensory afferent fibers blocks the abnormal C-fiber afferent signals from the bladder or inhibits the sensitization of normally “silent” C-fibers²³.

Someone might hypothesize that early NM could also affect the pathological deep tendon reflexes, clonus and spasticity, but this is not the case. It seems that NM protocols do not affect the somatic motoneurons, but only affect bladder innervation²³ and probably other autonomic innervation, such as of the bowel.

NM BY electrical sacral neuromodulation (SNM): SNM involves stimulating the sacral nerves to inhibit DO and was first applied by Tanagho and Schmidt in the 80s^{24,25}. Low frequency (10-20Hz) stimulation of S3, with pulse widths between 180 and 210 μ s, has been associated with therapeutic success in 43-85%, by modulation of micturition reflexes through stimulation of somatic afferents of the LUT^{21,22,25-27}. If a trial stimulation is effective, then a permanent stimulator is implanted (InterStim by Metronic). SNM is an option for symptom control in patients with neurogenic LUTD, who are at low risk of upper urinary tract deterioration. Most of the evidence is focused on incomplete SCI and MS. ASIA D/E SCI patients with preserved bladder filling sensation had higher success rates²¹. Relative contraindications include severe or rapidly progressive neurologic disease, complete SCI, abnormal sacral anatomy or anticipated frequent need for MRI below the head²¹. SNM is not recommended for kids, pregnant women or MS patients with detrusor underactivity¹⁷. Side effects include stimulation-related pain and hardware infection or malfunction.

Amundsen et al²⁸ investigated whether botulinum toxin injections are superior to SNM in controlling

refractory episodes of urgency/urinary incontinence among 381 non-neurogenic women. Women intravesically injected with 200 μ of Botox showed small but statistically significant superiority in incontinence episodes reduction (-3.9 vs -3.3 episodes per day), but also showed a higher risk of urinary tract infections (35% vs 11%) probably due to a higher need for transient self-catheterizations. New improved devices are developed, but studies still have a short follow-up. The Axonics® r-SNM System is a novel miniaturized rechargeable SNM device that can deliver therapy for at least 15 years²². Sievert et al²⁹ showed that early implantation of bilateral sacral nerve modulators (SNMs) in 10 complete SCI patients, during the acute bladder-areflexia phase, prevented the development of neurogenic DO. After a mean follow-up of 26 months there was normal bladder capacity, no urinary incontinence, reduced UTI rates, and improved bowel and erectile functionality without nerve damage. They suggested that even earlier SNM could result in more benefits and proposed future fMRI studies in order to prove whether neuronal information is passed through the sympathetic trunk ganglion to the brain even with complete SCI.

NM BY electrical tibial nerve stimulation (TNS): The (posterior) tibial nerve (L5-S3) is a mixed branch of the sciatic nerve that runs superficially behind the lower shin bone medially, making it an easy target for electrical NM. McGuire et al³⁰, in 1983, were the first to apply TNS on 15 SCI patients with DO, who showed a symptom improvement of 87%. Pulse width is usually 200 μ s and frequency is low (5-20Hz)³¹ and can be delivered by any Transcutaneous Electrical Nerve Stimulation (T.E.N.S.) device. Sessions usually last 30min each and current intensity is as high as the patient feels comfortable with, but not higher than the intensity that results in flexion of the big toe or fanning of the toes. There are no commonly accepted parameters³².

Transcutaneous TNS (TTNS) is delivered by adhesive skin electrodes, non-invasively, at home, even by the patient or a caregiver, for at least 3 times every week. The active (red) electrode is placed behind the (right) medial malleolus and the ground (black) approximately 10cm higher.³¹ Chen et al³³ performed a RCT involving 100 SCI patients with NDO and proved that 4 weeks of TTNS is as effective as solifenacin. Fur-

thermore, TTNS had no adverse effects. De Seze et al³⁴ found that 20 minutes of TTNS every day for 3 months reduced urinary urgency, frequency and incontinence on a sample of 70 MS patients. The results lasted for the entire day. Frequency of TTNS usually is 10Hz and intensity is high due to high electrical resistance of the skin.

In the 90s, Stoller was the first to apply Percutaneous TNS (PTNS), that is minimally invasive but more efficient than TTNS.^{24,25,32,35} PTNS is delivered by the insertion of a 34G needle active electrode near the tibial nerve (4-5cm cephalad to the medial malleolus) and the ground adhesive electrode is usually placed on the plantar arch. Usually, frequency of PTNS is 20Hz and intensity is only up to 10mA.^{24,31} 12 weekly sessions of PTNS seems to be the most effective, while practical as well, protocol.^{24,25} Finazzi et al³⁶ showed that PTNS results in neuroplastic changes of the brain cortex. The efficacy of PTNS at the management of non-neurogenic DO is well established and comparable to anticholinergics.^{25,37,38} Tudor et al. conducted a study to compare the results of PTNS in 25 idiopathic and 49 neurogenic (mostly MS:19) patients with DO. No significant differences in outcomes were found. MS patients had a higher probability to require maintenance treatment.²² Kabay et al³⁹ found that 12 weeks of PTNS (once every week) on 19 MS patients with NDO resulted in increased bladder capacity and decreased detrusor contractions. More studies are needed to prove efficacy of PTNS on neurogenic LUTD. PTNS is not recommended for MS patients with detrusor underactivity¹⁷.

To overcome the disadvantages of TTNS and PTNS, permanently implanted devices have been developed. "Urgent-SQ" was the first such stimulator used in 2006 and nowadays remotely controlled and charged devices (RENOVA iStim) are implanted, showing good efficacy on idiopathic DO³¹. Stampas et al²³ suggested that neuromodulation via the tibial nerve could also prevent the development of LUTD on acute phase SCI patients. In a randomized control pilot study they showed similar results after 2 weeks of TTNS on complete or incomplete SCI patients with a neurologic level of injury above T10 in order to avoid the possibility of coexisting damage of the detrusor's lower motor neuron. Improved efficacy was found when TTNS achieved toe flexion²³. TASCi is a RCT that started on

2019 to investigate the same potential of acute phase TTNS on 114 SCI patients. It is planned to be completed on 2024 and also aims to clarify the mechanism behind early NM⁴.

NM BY electrical pudental nerve stimulation (PNS): Animal studies have shown that neurogenic DO is inhibited even with complete SCI, by PNS but not by TNS. Thus, it is believed that PNS modulates sacral reflexes while TNS modulates higher suprasacral reflexes that may even be cortically integrated²⁵.

Peters et al⁴⁰ compared PNS to SNM for non-neurogenic LUTD and found symptoms improved in 63% versus 46% respectively. Even surgical procedure is easier for PNS. Nevertheless, it is still not preferred clinically, probably due to lack of good studies to confirm PNS superiority²⁵. Liao et al⁴¹ implanted electrodes in order to stimulate the pudental nerves of 3 dogs the next day after completely dissecting their SC, while 3 more SCI dogs served as the control group. Stimulation frequency was low (5Hz) and resulted in preservation of high bladder capacity and compliance with no contractions after 1 and 3 months. Furthermore, histological examination of their bladders showed that there was no fibrosis, which is thought to be responsible for irreversible deterioration of bladder capacity and compliance and for pressure elevation. This offers an explanation why early neuromodulation has better results than chronic-phase neuromodulation does.

NM BY electrical dorsal genital nerve stimulation (DGNS): The dorsal genital nerve (penile or clitoral) is a sensory branch of the pudental nerve that is easily stimulated, transcutaneously or percutaneously in order to inhibit DO. Efficient DGNS is confirmed by concomitant reflex contraction of the external anal sphincter²⁵. Danish researchers showed that DGNS can acutely suppress unwanted detrusor contractions on SCI⁴² and MS⁴³ patients with DO. DGNS was delivered every time intravesical pressure raised by 10cm-H₂O. Unfortunately, the method used to detect this rise could not be clinically applied.

NM BY electrical spinal cord stimulation (SCS): There are studies suggesting that SCS is a viable method for modulating the function of the LUT in human SCI participants, through unclear mechanisms². It is hypothesized that SCS increases the excitability of the

spinal reflexes necessary for proper LUT function¹⁸.

NM BY electrical perineal nerve stimulation (PeNS): Krhut et al³⁵ recently proposed a new method of low voltage transcutaneous stimulation of the common peroneal nerve (L4-S2), delivered by the "URIS" device, while a biofeedback foot sensor detects the optimal point for stimulation behind the head of the fibula³⁵.

Other electrical NM methods: Perineal electrical stimulation (mainly of S3 dermatome) improves DO, but has limited clinical use because of the difficulty in applying the electrodes²³.

In the past, NM methods involving intravesical, anal and vaginal electrical stimulation proved to be inconvenient and ineffective¹⁷.

High-frequency electrical stimulation (HF BLOCK)

High-frequency (10KHz) stimulation can block neurons that would convey a signal with unwanted results. However, although kilohertz frequency nerve block does not produce acute nerve damage, safety and durability of chronic high frequency nerve block remain to be determined.¹⁰

High-frequency spinal cord stimulation (HF-SCS): Epidural HF-SCS of the dorsal columns was initially

used for refractory neuropathic pain management.¹¹ Schieferdecker et al⁴⁴ used HF-SCS on five patients with SCI or MS, resulting in improvement of LUTD and of quality of life; however larger studies are needed for safer conclusions.

High-frequency pudental nerve stimulation (HF-PNS): HF block of the pudental nerve can inhibit the external sphincter from contracting, in an effort towards efficient low-pressure micturition with minimum PVR. The procedure is invasive and experimental.¹¹

In conclusion, it appears that it is only a matter of time before research and technological advances lead to safe, feasible and efficient electrical stimulation methods for managing, or even avoiding the development of, the disfunction of the LUT on neurogenic patients.

New stimulation strategies are currently studied experimentally, mostly on animals and are mainly based on direct spinal cord stimulation or on a combination of spinal root and pudental nerve stimulation¹².

Conflict of interest

The authors declare no conflicts of interest.

References

1. Benarroch EE. Neural control of the bladder: Recent advances and neurologic implications. *Neurology*. 2010;75(20). doi:10.1212/WNL.0b013e3181fdabba
2. Steadman CJ, Grill WM. Spinal cord stimulation for the restoration of bladder function after spinal cord injury. *Healthc Technol Lett*. 2020;7(3). doi:10.1049/htl.2020.0026
3. Kirshblum, Steven; Donovan, Jayne; Nieves, Jeremiah; Gonzalez, Priscila; Cuccurullo, Sara; Luciano L. Spinal cord injuries: Bladder dysfunction. In: *Physical Medicine and Rehabilitation Board Review*. Fourth edi. ; 2019:566-577.
4. Birkhäuser V, Liechti MD, Anderson CE, et al. TASC-transcutaneous tibial nerve stimulation in patients with acute spinal cord injury to prevent neurogenic detrusor overactivity: Protocol for a nationwide, randomised, sham-controlled, double-blind clinical trial. *BMJ Open*. 2020;10(8). doi:10.1136/bmjopen-2020-039164
5. de Sèze M, Ruffion A, Denys P, Joseph PA, Perrouin-Verbe B. The neurogenic bladder in multiple sclerosis: Review of the literature and proposal of management guidelines. *Mult Scler*. 2007;13(7). doi:10.1177/1352458506075651
6. de Groat WC, Yoshimura N. Plasticity in reflex pathways to the lower urinary tract following spinal cord injury. *Exp Neurol*. 2012;235(1):123-132. doi:10.1016/J.EXPNEUROL.2011.05.003
7. de Groat WC, Griffiths D, Yoshimura N. Neural control of the lower urinary tract. *Compr Physiol*. 2015;5(1). doi:10.1002/cphy.c130056
8. Panicker JN. Neurogenic Bladder: Epidemiology, Diagnosis, and Management. *Semin Neurol*. 2020;40(5):569-579. doi:10.1055/s-0040-1713876

9. Sivan, Manoj; Phillips, Margaret; Baguley, Ian; Nott M. Neurogenic Bladder and Bowel. In: *Oxford Handbook of Rehabilitation Medicine*. third. ; 2019:168-178.
10. McGee MJ, Amundsen CL, Grill WM. Electrical stimulation for the treatment of lower urinary tract dysfunction after spinal cord injury. *J Spinal Cord Med*. 2015;38(2). doi:10.1179/2045772314Y.0000000299
11. Martens FMJ, Sievert KD. Neurostimulation in neurogenic patients. *Curr Opin Urol*. 2020;30(4). doi:10.1097/MOU.0000000000000773
12. Guiho T, Azevedo-Coste C, Bauchet L, et al. Sacral Anterior Root Stimulation and Visceral Function Outcomes in Spinal Cord Injury—A Systematic Review of the Literature Over Four Decades. *World Neurosurg*. 2022;157. doi:10.1016/j.wneu.2021.09.041
13. Martens FMJ, Den Hollander PP, Snoek GJ, Koldewijn EL, Van Kerrebroeck PEVA, Heesakkers JPFA. Quality of life in complete spinal cord injury patients with a Brindley bladder stimulator compared to a matched control group. *NeuroUrol Urodyn*. 2011;30(4). doi:10.1002/nau.21012
14. Katona F. Stages of vegetative afferentation in reorganization of bladder control during intravesical electrotherapy. *Urol Int*. 1975;30(3). doi:10.1159/000279979
15. Ebner A, Jiang C, Lindstrom S. Intravesical electrical stimulation - An experimental analysis of the mechanism of action. *J Urol*. 1992;148(3 I). doi:10.1016/S0022-5347(17)36778-2
16. James Walter 1, 2 IH 3, 4 BT, 5 AAC, et al. A New Electrode Design for Direct Bladder Wall Stimulation: A Pilot Minipig Study with Chronic Testing. *Appl Sci*. 2022;12(1149):1-10.
17. Abboud H, Hill E, Siddiqui J, Serra A, Walter B. Neuromodulation in multiple sclerosis. *Mult Scler*. 2017;23(13). doi:10.1177/1352458517736150
18. Herrity AN, Williams CS, Angeli CA, Harkema SJ, Hubscher CH. Lumbosacral spinal cord epidural stimulation improves voiding function after human spinal cord injury. *Sci Rep*. 2018;8(1). doi:10.1038/s41598-018-26602-2
19. Havton LA, Christe KL, Edgerton VR, Gad PN. Non-invasive spinal neuromodulation to map and augment lower urinary tract function in rhesus macaques. *Exp Neurol*. 2019;322. doi:10.1016/j.expneurol.2019.113033
20. Daia C, Bumbea AM, Dumitru Badiu C, Ciobotaru C, Onose G. Interferential electrical stimulation for improved bladder management following spinal cord injury. *Biomed Reports*. 2019;11(3):115122. doi:10.3892/br.2019.1227
21. Goldman HB, Lloyd JC, Noblett KL, et al. International Continence Society best practice statement for use of sacral neuromodulation. *NeuroUrol Urodyn*. 2018;37(5):1823-1848. doi:10.1002/nau.23515
22. Ammirati E, Giammò A, Manassero A, Carone R. Neuromodulation in urology, state of the art. *Urol J*. 2019;86(4). doi:10.1177/0391560319866075
23. Stampas A, Korupolu R, Zhu L, Smith CP, Gustafson K. Safety, Feasibility, and Efficacy of Transcutaneous Tibial Nerve Stimulation in Acute Spinal Cord Injury Neurogenic Bladder: A Randomized Control Pilot Trial. *Neuromodulation Technol Neural Interface*. 2019;22(6):716-722. doi:10.1111/NER.12855
24. de Wall LL, Heesakkers JPFA. Effectiveness of percutaneous tibial nerve stimulation in the treatment of overactive bladder syndrome. *Res Reports Urol*. 2017;9. doi:10.2147/RRU.S124981
25. Janssen DAW, Martens FMJ, de Wall LL, van Breda HMK, Heesakkers JPFA. Clinical utility of neurostimulation devices in the treatment of overactive bladder: Current perspectives. *Med Devices Evid Res*. 2017;10. doi:10.2147/nder.s115678
26. Girtner F, Burger M, Mayr R. Sacral neuromodulation in under- and overactive detrusor – quo vadis?: Principles and developments. *Urol*. 2019;58(6). doi:10.1007/s00120-019-0949-7
27. Assmann R, Douven P, Kleijnen J, et al. Stimulation Parameters for Sacral Neuromodulation on Lower Urinary Tract and Bowel Dysfunction–Related Clinical Outcome: A Systematic Review. *Neuromodulation*. 2020;23(8):1082-1093. doi:10.1111/ner.13255
28. Amundsen CL, Richter HE, Menefee SA, et al. Onabotulinumtoxin a vs sacral neuromodulation on refractory urgency urinary incontinence in women: A randomized clinical trial. *JAMA - J Am Med Assoc*. 2016;316(13). doi:10.1001/jama.2016.14617
29. Sievert, Karl-Dietrich; Amend, Bastian; Gakis G et al. Early sacral neuromodulation prevents urinary incontinence after complete spinal cord injury. *Ann Neurol*.

- Published online 2010.
30. McGuire EJ, Shi Chun Z, Horwinski ER, Lytton B. Treatment of motor and sensory detrusor instability by electrical stimulation. *J Urol.* 1983;129(1). doi:10.1016/S0022-5347(17)51928-X
 31. Bhide AA, Tailor V, Fernando R, Khullar V, Digesu GA. Posterior tibial nerve stimulation for overactive bladder—techniques and efficacy. *Int Urogynecol J.* 2020;31(5). doi:10.1007/s00192-01904186-3
 32. Gross T, Schneider MP, Bachmann LM, Blok BF, Groen J H, LA et al. Transcutaneous electrical nerve stimulation for treating neurogenic lower urinary tract dysfunction: a systematic review. *Eur Urol.* 2016;69((6)):1102-1111.
 33. Chen G, Liao L, Li Y. The possible role of percutaneous tibial nerve stimulation using adhesive skin surface electrodes in patients with neurogenic detrusor overactivity secondary to spinal cord injury. *Int Urol Nephrol.* 2015;47(3). doi:10.1007/s11255-015-0911-6
 34. De Sèze M, Raibaut P, Gallien P, et al. Transcutaneous posterior tibial nerve stimulation for treatment of the overactive bladder syndrome in multiple sclerosis: Results of a multicenter prospective study. *Neurourol Urodyn.* 2011;30(3). doi:10.1002/nau.20958
 35. Krhut J, Peter L, Rejchrt M, Slovak M, Skugarevska B, Zvara P. Peroneal Electric Transcutaneous NeuroModulation (eTNM®): A Novel Method for the Treatment of the Overactive Bladder. *J Healthc Eng.* 2021;2021. doi:10.1155/2021/4016346
 36. Finazzi-Agrè E, Rocchi C, Pachatz C, et al. Percutaneous tibial nerve stimulation produces effects on brain activity: Study on the modifications of the long latency somatosensory evoked potentials. *Neurourol Urodyn.* 2009;28(4). doi:10.1002/nau.20651
 37. Finazzi-Agrè E, Petta F, Sciobica F, Pasqualetti P, Musco S, Bove P. Percutaneous tibial nerve stimulation effects on detrusor overactivity incontinence are not due to a placebo effect: A randomized, double-blind, placebo controlled trial. *J Urol.* 2010;184(5). doi:10.1016/j.juro.2010.06.113
 38. Peters KM, Carrico DJ, Perez-Marrero RA, et al. Randomized Trial of Percutaneous Tibial Nerve Stimulation Versus Sham Efficacy in the Treatment of Overactive Bladder Syndrome: Results From the SUmIT Trial. *J Urol.* 2010;183(4). doi:10.1016/j.juro.2009.12.036
 39. Kabay S, Kabay SC, Yucel M, et al. The clinical and urodynamic results of a 3-month percutaneous posterior tibial nerve stimulation treatment in patients with multiple sclerosis-related neurogenic bladder dysfunction. *Neurourol Urodyn.* 2009;28(8). doi:10.1002/nau.20733
 40. Peters KM, Feber KM, Bennett RC. Sacral versus pudendal nerve stimulation for voiding dysfunction: A prospective, single-blinded, randomized, crossover trial. *Neurourol Urodyn.* 2005;24(7). doi:10.1002/nau.20174
 41. Li P, Liao L, Chen G, Zhang F, Tian Y. Early low-frequency stimulation of the pudendal nerve can inhibit detrusor overactivity and delay progress of bladder fibrosis in dogs with spinal cord injuries. *Spinal Cord.* 2013;51(9). doi:10.1038/sc.2013.60
 42. Dalmose AL, Rijkhoff NJM, Kirkeby HJ, Nohr M, Sinkjaer T, Djurhuus JC. Conditional stimulation of the dorsal penile/clitoral nerve may increase cystometric capacity in patients with spinal cord injury. *Neurourol Urodyn.* 2003;22(2). doi:10.1002/nau.10031
 43. Fjorback MV, Rijkhoff N, Petersen T, Nohr M, Sinkjaer T. Event driven electrical stimulation of the dorsal penile/clitoral nerve for management of neurogenic detrusor overactivity in multiple sclerosis. *Neurourol Urodyn.* 2006;25(4). doi:10.1002/nau.20170
 44. Schieferdecker S, Neudorfer C, El Majdoub F, Maarouf M. A Retrospective Case Series of High-Frequency Spinal Cord Stimulation (HF10-SCS) in Neurogenic Bladder Incontinence. *Oper Neurosurg.* 2019;17(1). doi:10.1093/ons/opy236

Cite this paper as



Koutsogeorgis N, Evangelopoulos ME. Koutsogeorgis and Evangelopoulos. The role of electrical stimulation in the management of lower urinary track dysfunction following spinal cord lesions. *Acta Orthop Trauma Hell* 2024; 75(1): 64-73.

Sexual and reproductive health of patients with spinal cord injuries - Orientation to female fertility and pregnancy

Ariadni Petropoulou¹, Dimitrios-Sergios Evangelopoulos^{1,2,3}, John Vlamis^{1,2}, Maria-Eleftheria Evangelopoulos⁴

¹ Postgraduate Training Program, KAT Hospital, National and Kapodistrian University of Athens School of Medicine, KAT Hospital, 2 Nikis str, 14561, Athens, Greece

² Third Department of Orthopaedic Surgery, National and Kapodistrian University of Athens School of Medicine, KAT Hospital, 2 Nikis str, 14561, Athens, Greece

³ Laboratory for Research of the Musculoskeletal System "Th. Garofalidis", National and Kapodistrian University of Athens School of Medicine, KAT Hospital, 2 Nikis str, 14561, Athens, Greece

⁴ First Department of Neurology, National and Kapodistrian University of Athens School of Medicine, Eginition Hospital, 72-74 Vas. Sofias Av., 11528, Athens, Greece

Abstract

This exploratory review focus on the sexual and reproductive health of female spinal cord injury (SCI) patients. This investigation seeks to determine whether or not women with spinal cord impairments can become pregnant. Women with SCI are unable to procreate, contrary to the alternative theory. We searched PubMed and Scopus for relevant articles published between 2003 and 2023. Consideration was given to studies, with an emphasis on the sexual and reproductive health, including reproduction, of women with SCI. Animal studies, studies published in languages other than English, male-focused research, studies on neurological diseases other than SCI, and non-relative studies were all disqualified. The findings of this review shed light on the likelihood of conception among individuals with SCI, while the findings of individual studies contribute to our understanding of female fertility and pregnancy outcomes in this population.

Keywords: spinal cord injury, female fertility, pregnancy outcomes, sexual and reproductive health, women with disabilities

Introduction

Worldwide, spinal cord injuries (SCIs) are the most common cause of catastrophic disability. In addition to sexual and reproductive health, these traumas may have far-reaching consequences in other areas as well. Neurogenic dysfunction after SCI has been linked to significant changes in sexual function, fertility, and

pregnancy outcomes. Several studies have assessed the effects of SCI on men's sexual and reproductive health, but few have examined the effects of SCI on women's fertility and pregnancy outcomes [1, 2].

To provide comprehensive treatment and support, tailored to the specific needs of women with SCI, sexual and reproductive health knowledge is necessary. For women

Corresponding
Author

Petropoulou A., Student of the Postgraduate Training Program, KAT Hospital, National and Kapodistrian University of Athens School of Medicine, Athens, Greece. Address: KAT Hospital, 2 Nikis Street, Kifisia, 14561. E-mail: ar.petropoulou@gmail.com

with SCI, reproductive health presents unique concerns and problems [3, 4]. Changes in sensation, decreased sexual response, changes in vaginal lubrication, and the inability to attain orgasm are all examples of these problems. Additionally, a SCI may have a negative impact on the autonomic nervous system, which affects ovulation, implantation, and uterine contractility [5, 6].

Despite its necessity, research on the sexual and reproductive health of women with SCI is limited. Male sexual function and reproductive health outcomes have been the primary focus of research, whereas women's particular concerns and experiences have been largely ignored. Therefore, it is essential to fill this informational void by analyzing the existing literature on female fertility and pregnancy outcomes in SCI patients [7].

The purpose of this analysis is to provide a comprehensive overview of the sexual and reproductive health of women with SCI. The question at issue is "Can women with SCIs get pregnant and have healthy babies?" A comprehensive literature review was conducted using rigorous inclusion and exclusion criteria to identify relevant papers addressing our research question. This systematic approach aimed to thoroughly examine the available studies on the sexual and reproductive health of women with SCI, specifically focusing on their capacity to conceive and carry a child to term. Through this selection process, the results of these studies aim to bridge knowledge gaps and provide guidance for future research, ultimately ensuring that women with SCIs receive appropriate and effective treatment.

A thorough examination of the PubMed and Scopus databases was conducted to locate relevant publications published between 2003 and 2023. In addition to terms related to SCI, the search terms included terms related to sexual and reproductive health, fertility, and pregnancy. Inclusion criteria prioritized human clinical trials and observational studies investigating sexual and reproductive health, including pregnancy outcomes, among women with SCI. Studies that were not published in English, utilized animals as subjects, focused primarily on male populations, or dealt with neurological diseases other than SCI were excluded (Figure 1).

Epidemiology

SCI may have a significant impact on a person's fertility, and an understanding of epidemiology is required

to comprehend this impact. The fact that 50% of SCI occurs in patients aged between 15 and 25 years, with 15–20% being women, can have a detrimental impact on their desire to start a family. [8] In the United States, approximately 78% of newly diagnosed SCI patients are male, with an average injury age of 43[9]. According to national estimates, approximately 40,000 to 45,000 women with SCI are living in the United States [10]. It is estimated that there are currently 20,000 women between the ages of 16 and 30 years with SCI in the USA [11]. Traumatic SCI affects 16–54 new individuals per million each year in Western countries with a four times higher incidence in males, while non-traumatic SCI, estimated incidence ranges from 6 to 76 cases per million individuals/year [12].

Traumatic SCI occurs when an external physical impact acutely damages the spinal cord, whereas non-traumatic SCI occurs when an acute or chronic disease process, such as a tumor, infection or degenerative disc disease, generates the primary injury [13]. The leading causes of traumatic SCI are motor vehicle accidents, falls, violence (including firearm wounds), and sports-related injuries [14]. The commonest cause of non-traumatic SCI in developed countries is represented by degenerative myelopathy [12].

Sexual function and reproductive health are frequently affected as a consequence of SCI. Patients with SCI experience sexual dysfunction involving different domains. It is reported that 50% of women and 29% of men report impaired sexual desire, 70–81% of men have erectile dysfunction, 50–80% of women experience reduced arousal or vaginal dryness [15]. Approximately 95% of males with SCI have ejaculatory difficulties, such as an inability to ejaculate or diminished ejaculatory function [11]. In addition, men with SCI commonly have low sperm concentration and quality, making it difficult for them to conceive without medical intervention, such as assisted reproductive technologies [16].

It has been shown that the fertility outcomes for women with SCI differ from those for men. According to studies, women with SCI have an equal probability of conceiving and giving birth. Injuries to the spinal cord may affect a person's quality of life, but they have little effect on fertility. This differentiation highlights the specific sexual and reproductive health needs of

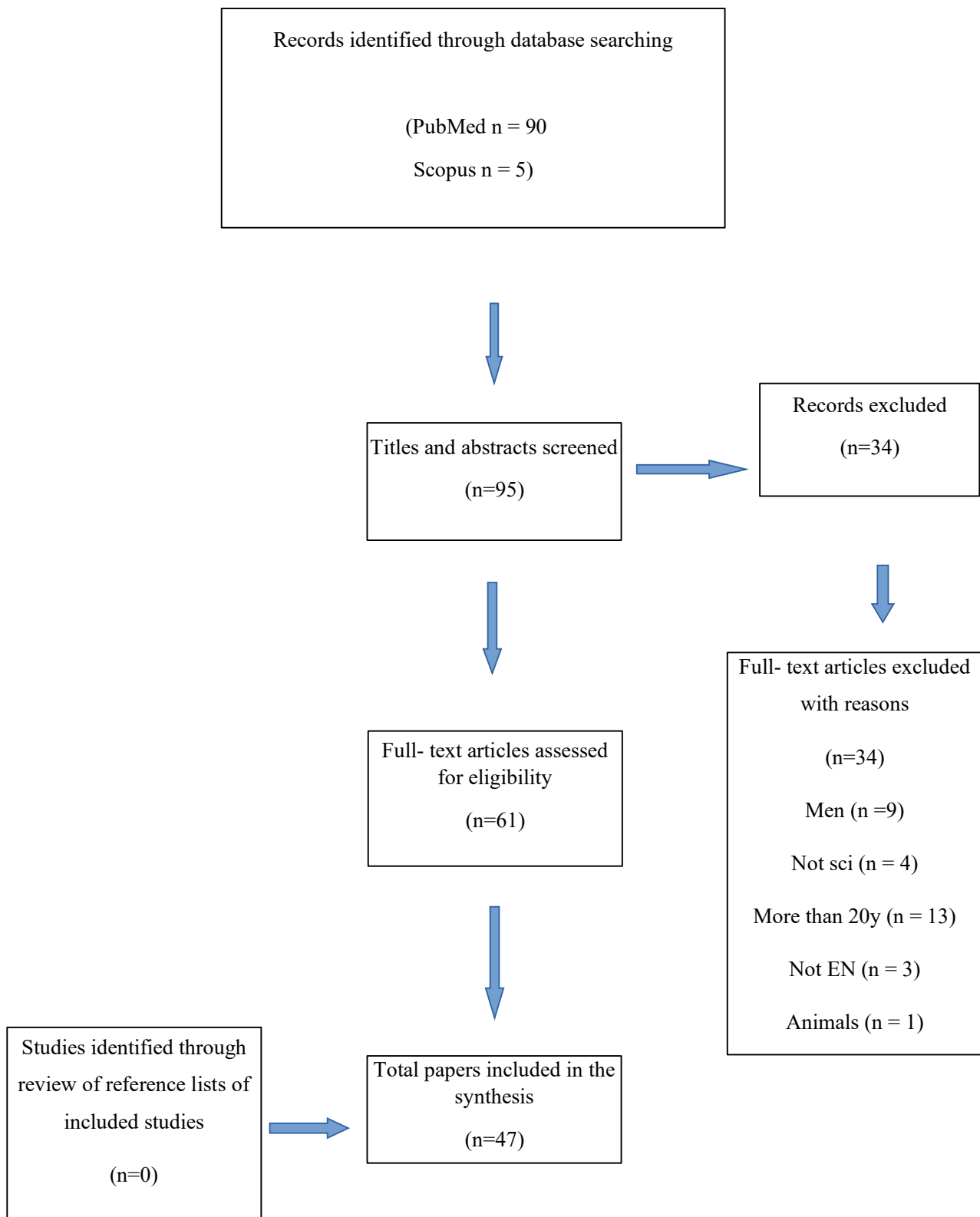


Figure 1. Flowchart of the study.

women with SCI, particularly during pregnancy and delivery [17]. When queried [18], people with SCI indicate that their ability to engage in sexual and reproductive activities has a significant impact on their quality of life. These results demonstrate the detrimental effects of SCI on the sexual and reproductive health of men and women. Healthcare professionals are obligated to address these challenges and provide patients with a comprehensive treatment plan that addresses sexual dysfunction, fertility issues, and a lack of access to essential reproductive health treatments.

Notably, sexual dysfunction may also result from non-traumatic injuries to the spinal cord, such as a herniated disc. Although SCI is the primary focus of this document, it is essential to keep in mind that other spinal cord diseases may also affect reproductive health [19].

Sexual function and satisfaction in women with spinal cord injuries

When a person suffers from SCI, their sexual health and contentment become crucial to their overall well-being and quality of life. The World Health Organization recognizes sexual health as a fundamental human right for all individuals. However, people living with disabilities frequently encounter exclusion from active sexuality and incorrect assumptions of asexuality [20]. The significance of sexuality for individuals experiencing a SCI is underscored by the fact that enhancing sexuality ranks as the primary priority for paraplegic patients. Among tetraplegics, it is second only to the recovery of arm function, highlighting the importance placed on improving sexual well-being. It's noteworthy that this priority is applicable to both male and female sexuality [21].

To provide women with SCI with the necessary care and support, it is necessary to comprehend how SCI impacts their intimate experiences [22]. The level and completeness of SCI play a significant role in determining sexual functioning. In the immediate post-injury period, women experience a loss of the ability to have reflexive sexual responses. As reflexes gradually return, reflexive arousal – vaginal lubrication – can be achieved through genital stimulation, provided that the sacral spinal segments and peripheral pathways conveying sensations (cauda equina) remain intact [23].

While sexual desire and arousal may be maintained in women with SCI [24], the researchers observed that neurological disturbances may make it more difficult to have an orgasmic experience. The presence of sensation in the genital area suggests the preservation of the sacral reflex arc, enhancing the likelihood of achieving orgasm. According to literature, women with SCI at all levels and degrees were found to be significantly less likely than able-bodied control subjects to achieve orgasm. Specifically, 55% of women with SCI reported the ability to achieve orgasm, in contrast to 100% of able-bodied control subjects [25]. It's important to inform all women with SCIs that they may require longer or more intense genital stimulation to achieve orgasm and that the experience might be less intense than before [21].

While fundamental motives persist after SCI, there are notable physiological changes affecting sensation, voluntary motor function, and autonomic function. These alterations impact sexual arousal, orgasmic potential, ejaculation in men, and sexual positioning for both sexes [26]. The impact of SCI on sensory perception and motor function can hinder the ability to achieve climax. Conditions related to SCI, such as pain, weakness, pressure ulcers, sensory loss, fatigue, and lack of bowel and bladder control, contribute to dissatisfaction with sexual intimacy [27]. Additionally, depression and pharmacological management further complicate sexual self-esteem, expression, and satisfaction [26]. Women have been found to experience depression or other psychological disorders more often than men after the injury [28]

Research suggests that individuals with SCI may have comparable levels of sexual desire to those without such injuries. Both groups can participate in positive sexual relationships, with some reporting varying degrees of sexual satisfaction. Factors like personal choice, cultural and religious beliefs, the severity and timing of the injury, opportunities for intimacy, privacy, and access to meet others can influence the expression of intimacy and desire after a SCI [29].

Nevertheless, sexual satisfaction is still attainable through imaginative planning and open communication between partners. By experimenting with new erogenous zones, new sexual approaches, and assistive technologies, women with SCI can enhance

their sexual satisfaction [27]. However sexual function and satisfaction is often overlooked in the rehabilitation process [20]. Assessing patients with SCI using the International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) is vital. This evaluation helps tailor education about sexual potential and improve responses. By documenting the level of injury, clinicians can determine the necessary assistance for mobility and daily activities, crucial factors affecting the individual's ability to self-manage during sexual activity [30].

A female oriented approach to sexuality rehabilitation should commence early preparing women for psychological impact, altered sexual sensation and disclosure of altered sexual function to partners [31]. In addition, emotional or psychological difficulties that may arise as a result of a change in sexual function may be best treated with psychological assistance and therapy. By diagnosing the effects of the condition on sexual function and promoting an open dialogue about sexuality, healthcare professionals can help women with SCI regain control of their sexual lives [24].

It is crucial for individuals with SCI to receive accessible support and psychoeducational resources regarding post-injury sexuality. This is essential to reduce reluctance and discomfort in initiating conversations about their sexual lives. [20] It is important to identify both barriers and facilitators in rehabilitative practices and create an inclusive environment that encourages open discussions. The goal is to ensure optimal care for sexual health following SCI [26].

Fertility in women with SCIs

Over the past half-century, notable advancements in health outcomes for individuals with SCI and progress in medicine and assisted reproductive techniques have made the prospect of becoming pregnant increasingly successful for women with SCI [9].

Literature data support that pregnancy rates among reproductive-aged women with SCI in the United States was comparable to rates of women in the general population [9]. Immediately after the injury, there is an episode of acute amenorrhea that lasts for approximately 6 months, and then menstruation returns to normal levels [8]. This phenomenon happens due to a temporary rise in prolactin concentration, caused by

stress from the trauma [32]. There was no correlation between the level of injury, injury classification (complete versus incomplete) and the disruption of menstrual cycle [8]. Regarding women with SCI, a period of amenorrhea is followed by a resumption of fertility comparable to that of unaffected women [33].

Age, general health, and the underlying causes of infertility are just a few of the numerous factors that can influence fertility and pregnancy outcomes. Factors associated with age are commonly seen as influential in fertility and pregnancy rates, a consideration that applies to both the general population and individuals with SCI [1].

Due to advances in ART, individuals with SCI now have access to a greater variety of reproductive options. When determining the most suitable reproductive strategy for their circumstances, individuals may benefit from a comprehensive examination and discussion of the available options. [34].

Intravaginal insemination (IVI), also known as "at-home insemination," is the least invasive and least expensive of assisted reproductive technology (ART) techniques. IVI is the placement of sperm in the vagina so that a normal pregnancy can develop. Semen is collected in a specimen cup, is drawn up into an appropriately-sized syringe barrel and then introduced into the vagina. [29] There are no specific recommendations for establishing the minimum acceptable quantity of motile sperm for couples in which one partner has SCI, and this may impact the efficacy of IVI. While there are no definitive guidelines on the subject, it's typically recommended to consider self-insemination and/or Intrauterine Insemination (IUI) when there are at least four million motile sperm present in the ejaculate. If the sperm count is lower than four million, the options may involve in Vitro Fertilization (IVF) either with or without Intracytoplasmic Sperm Injection (ICSI) [35].

IUI should be the next option if multiple IVI attempts have failed or if it is not indicated [34]. This procedure involves gathering sperm from men with spinal cord injuries using penile vibratory stimulation (PVS) or electro-ejaculation (EEJ) in accordance with standard safety protocols. The sperm are separated from the sample of semen in the laboratory. Sperm is collected and inserted directly into a woman's uterus. IUI can be administered without stimulating the reproductive

system of the female partner (unstimulated cycles), or fertility medications can be prescribed to stimulate egg production and ovulation [29].

ICSI is a more advanced ART option when IVI or IUI fail [10]. In conventional IVF, sperm is extracted from the testis or epididymis of males with SCI via PVS, EEJ, or surgical extraction. The female partner is subjected to ovarian stimulation, and the retrieved eggs and sperm are combined in a laboratory container. The highest-quality embryos are transferred into the female partner's uterus after being incubated in an incubator until they reach the blastocyst stage. IVF-ICSI is frequently used when there are insufficient embryos for conventional IVF [29].

Recent studies conclude that successful pregnancies and live births have been reported in partners of men with spinal cord injuries through the use of ART [35-37]. Couples with an SCI male partner who have poor semen parameters can benefit from ART. The pregnancy and delivery rates for these couples appear to be similar to those of other couples with infertility but without an SCI situation [36].

Women with SCI choose specific methods of reproduction for several significant reasons. Firstly, despite the challenges they face, they are informed that their endocrine function returns to normal after a brief period of amenorrhea following acute SCI, enhancing their ability for natural conception. Additionally, methods such as IUI and artificial insemination have been successfully performed without increasing the risk of thrombosis, providing viable options. This approach minimizes the risk of multiple pregnancies, allowing women to choose the number of embryos transferred [38].

However, it is essential for couples to consult fertility specialists with experience treating SCI patients. Clinicians typically adopt a graduated approach, initiating treatment with natural and less invasive methods before progressing to more invasive and costly options within a fertility clinic [34].

Given that SCI does not seem to significantly impact female fertility, most research focuses on male infertility and how it can be better addressed through ART. While significant health challenges exist for women with SCI, there is an overall lack of rigorous studies addressing many of these issues. Population-based stud-

ies on the reproductive experiences of women with disabilities, including SCI, are nearly nonexistent [39].

Pregnancy outcomes and maternal health

Women with SCI encounter distinct challenges that require specialized care to improve pregnancy outcomes and maternal health. They may face obstetrical challenges such as preterm labor, unattended delivery, and an increased risk of prematurity [40]. To ensure the well-being of both the pregnant mother and her unborn child, it is crucial to recognize and address these obstacles. Women with SCIs confront unique pregnancy-related risks that demand close medical monitoring and, in certain instances, medical intervention [38].

According to the research, the outcomes of pregnancy and delivery in women with SCIs generally show favorable results, with only minor differences in adverse outcomes when compared to the general population. [32,42] Neonatal mortality rates of congenital malformations were not observed to be higher than those in the general population, but high rates of maternal and infant complications were reported. [43] Our study revealed that infants born to women with these conditions did not face a higher risk of long-term adverse outcomes, such as rehospitalization and death, despite being more prone to preterm birth or being small for gestational age. However, their mothers exhibited elevated risks of various complications compared to healthy women [38]

The level of injury plays a significant role in preventing complications during pregnancy, so it should be taken seriously into consideration. Injuries above the T10 level may prevent the patient from perceiving pain during childbirth, and above the T6 level, there is a risk of autonomous dysreflexia (AD) [44]. In lesions above T10, labor may not be perceived at all or perception of labor may be by concurrent sympathetic symptoms, such as AD, scalp tingling, an increase in the frequency of spasms, abdominal self-palpation. [3]

The most common complication observed in pregnant women with SCI was urinary tract infection (UTI). Managing urological issues proved to be more challenging, as a quarter of the women reported the necessity to alter their standard bladder management method during pregnancy. Additionally, between 27% and 70% of these women had to increase the frequency

of intermittent catheterizations per day [41]. The main goal of bladder management is to achieve low-pressure urine storage and effective bladder emptying. Most individuals with SCI require regular urologic assessments and treatment for their bladder dysfunction. If left untreated, UTI can cause significant pain and pose a risk to the kidneys. Preventing and treating UTIs in pregnant women with SCI requires careful monitoring of urinary health and proactive management of bladder function, including practices such as intermittent catheterization [45]. UTIs have been observed to be associated with low birth weight and preterm delivery, and the increased frequency of UTIs in these women is a parameter of concern regarding these complications [40]

Pregnancy introduces an additional risk factor for women with SCI, as AD has been reported to occur in 60% of cases [8]. AD, a potentially life-threatening complication of SCI, predominantly affects individuals with an injury level at T6 or above. This condition results from dysregulation of the autonomic system and is characterized by a sudden increase in systolic blood pressure (≥ 20 mmHg), triggered by a non-specific stimulus below the level of injury [12]. Women with spinal lesions at or above the sixth thoracic vertebra may experience potentially life-threatening AD during vaginal birth due to painful stimuli below the injury level. The risk of this complication diminishes with the use of epidural analgesia. The absence of epidural analgesia potentially heightens the risk of AD during vaginal birth, increasing the likelihood of acute cesarean section and potential harm for both mother and infant [32].

Pregnancy-related respiratory complications are more likely to occur in women with SCI. Changes in lung capacity, particularly in women with severe injuries, may have an effect on respiratory function. Depending on the level of injury, varying degrees of pulmonary dysfunction occur following SCI. As the gravid uterus enlarges, its pressure on the diaphragm may further compromise lung vital capacity, resulting in poor oxygenation, increased fatigue, and limitations in mobility [9]. Women with spinal cord lesions above T4 may experience partial or complete paralysis of respiratory muscles, posing challenges in breathing as pregnancy progresses. If respiratory function is com-

promised, interventions such as chest physiotherapy, continuous positive airway pressure, and mechanical ventilation may be necessary [3].

Special consideration should be given to alterations in the cushion-skin interface, as the risk of decubitus ulcers increases in this population. Factors contributing to this heightened risk include weight gain, changes in body habitus, increased spasms, pain, immobility, and even anemia [8]. It is advisable to undergo an assessment by a physiatrist and a physical therapist to ensure appropriate adjustments in seating and assistive devices are made to accommodate the woman's evolving body [39].

Spasticity, a symptom of upper motor neuron disorders, results from intact spinal reflexes persisting below the level of injury. It can be severe enough to lead to falls from wheelchairs, with a reported 12% incidence of worsening spasms during pregnancy [44]. Currently, there are no specific treatment guidelines for managing spasticity in this population [45]. Managing spasticity poses challenges as most medications can have potentially harmful effects on the fetus during pregnancy and lactation [38]. Preconception counseling, regular follow-up, reviewing and modifying bowel, bladder, and exercise programs, educating about the effects of pregnancy on SCI and maintaining overall health are crucial for optimizing spasticity management during this period [45].

Literature findings suggest that women with SCI can successfully and safely achieved vaginal delivery, regardless of the level of injury [46]. To make informed decisions, women with SCI should engage in discussions with obstetric physicians who specialize in high-risk pregnancies, exploring the risks associated with spontaneous vaginal delivery versus planned cesarean delivery [8].

People with spinal cord injuries (SCIs) often encounter challenges when planning a family or during pregnancy, stemming from issues like inaccessible healthcare providers and societal misconceptions. Both men and women face barriers, with women struggling to find wheelchair, accessible healthcare provider offices, as well as offices equipped with accessible exam tables [47].

Specialized treatment and multidisciplinary team involving specialists of neurological disability and preg-

nancy are required to improve pregnancy outcomes and maternal health in women with SCI [41]. Women should be mindful of changes in mobility, making adjustments with the assistance of physiotherapy. Issues such as accessibility and social support are frequently lacking and deserve more attention [40].

Conclusions

The findings of a literature review on the sexual and reproductive health of women with SCIs have been enlightening. To begin, it is indisputable that sexual function and pleasure are drastically altered in women with SCI. Spinal cord injury-related neurological deficits can alter genital sensation, lubrication, and the orgasmic response, all of which make it more difficult to attain orgasm. Nonetheless, sexual fulfillment is still attainable through imaginative planning and open communication between partners.

New research dispels long-held beliefs about the infertility of women with spinal cord injuries, demonstrating that such women can conceive and deliver healthy children. Researchers discovered that SCI did not significantly influence fertility rates in women and that pregnancy rates were comparable to those of the general population regardless of injury severity, degree of incompleteness, or duration since injury. Nevertheless, impaired sexual function and altered uterine contractility may necessitate the use of ART.

For women with SCI, maternal and fetal health are of the utmost importance. Obstacles and problems unique to pregnancy require specialized care and the collaboration of professionals from other fields. UTIs, AD, pressure lesions, and respiratory issues are all conditions that require close monitoring and treatment. Preconception counseling, prenatal care, and access to appropriate rehabilitative treatments are essential for optimal pregnancy outcomes and the health of the mother and expectant child.

Epidemiological data indicate that the preponderance of SCIs occurs in young adult males. SCI may be the result of a car accident, a fall, an act of violence, or even sports participation. A large proportion of males with SCI (90%) are sterile and unable to naturally conceive a child due to erectile dysfunction and infertility. However, the pregnancy success rate for women with SCI is comparable to that of the general population. The ability to engage in sexual and reproductive activities is regarded as a crucial factor in determining the quality of life for individuals with SCI.

Assisted reproductive procedures offer SCI patients, particularly men, a viable reproductive option. In addition to ART, males with SCI who cannot produce ejaculate during sexual interaction require assisted ejaculation treatments. It is simple and affordable to perform IVI at home. Consultation with reproductive professionals is necessary for couples to select the most suitable technique, as success rates vary depending on the method used.

This literature review uncovered a bounty of new information regarding the sexual and reproductive health of women with spinal cord injuries. Despite the difficulties brought on by the neurological abnormalities caused by SCI, women with the condition can still find erotic fulfillment through creative problem-solving and open communication. It has been demonstrated that women with SCI are able to conceive and deliver healthy infants at rates comparable to the general population, disproving long-held beliefs about their incapacity to do so. To conceive a child when sexual function is diminished, IVF or another ART may be required. Women with SCI must have access to specialized care and multidisciplinary collaboration in order to enhance pregnancy outcomes and maternal health.

Conflict of interest

The authors declare no conflicts of interest.

References

1. Courtois F, Alexander M, McLain ABJ. Women's Sexual Health and Reproductive Function After SCI. *Top Spinal Cord Inj Rehabil.* 2017; 23:20-30.
2. Lysberg K, Severinsson E. Spinal cord injured women's views of sexuality: A Norwegian survey. *Rehabil Nurs* 2003; 28:23-26
3. Biering-Sørensen I, Hansen RB, Biering-Sørensen F. Sexual function in a traumatic spinal cord injured population 10-45 years after injury. *J Rehabil Med.* 2012; 44:926-31.
4. Ferreiro-Velasco ME, Barca-Buyo A, de la Barrera SS et al. Sexual issues in a sample of women with spinal cord injury. *Spinal Cord* 2005; 43:51-55.
5. Sipski ML, Rosen RC, Alexander CJ et al. Sexual responsiveness in women with spinal cord injuries: differential effect of anxiety-eliciting stimulation. *Arch Sex Behav* 2004; 33:295-302.
6. Komisaruk BR, Whipple B, Crawford A et al. Brain activation during vaginocervical self-stimulation and orgasm in women with complete spinal cord injury: fMRI evidence of mediation by the vagus nerves. *Brain Res* 2004; 1024:77-88.
7. Alexander MS, Rosen RC, Steinberg S et al. Sildenafil in women with sexual arousal disorder following spinal cord injury. *Spinal Cord* 2011; 49:273-279.
8. Stoffel JT, Van der Aa F, Wittmann D et al. Fertility and sexuality in the spinal cord injury patient. *World J Urol.* 2018; 36:1577-1585.
9. Iezzoni LI, Chen Y, Jackson McLain AB. Current pregnancy among women with spinal cord injury: findings from the US national spinal cord injury database. *Spinal Cord.* 2015; 53:821- 826.
10. Kathiresan ASQ, Ibrahim E, Aballa TC et al. Pregnancy outcomes by intravaginal and intrauterine insemination in 82 couples with male factor infertility due to spinal cord injuries. *Fertility and Sterility* 2011; 96:328-331.
11. Anderson D, Borisoff JF, Johnson RD et al. Spinal cord injury influences psychogenic as well as physical components of female sexual ability. *Spinal Cord* 2007; 349-359.
12. Pavese C, Kessler TM. Prediction of Lower Urinary Tract, Sexual, and Bowel Function, and Autonomic Dysreflexia after Spinal Cord Injury. *Biomedicines.* 2023; 11:1644.
13. Ahuja CS, Wilson JR, Nori S et al. Traumatic spinal cord injury. *Nat Rev Dis Prim.* 2017; 3:17018.
14. Salomon J, Schnitzler A, Ville Y et al Prevention of urinary tract infection in six spinal cord-injured pregnant women who gave birth to seven children under a weekly oral cyclic antibiotic program. *Int J Infect Dis.* 2009; 13:399-402.
15. Hentzen C, Musco S, Amarenco G et al. Approach and management to patients with neurological disorders reporting sexual dysfunction. *Lancet Neurol.* 2022; 21:551-562.
16. Kreuter M, Taft C, Siösteen A et al. Women's sexual functioning and sex life after spinal cord injury. *Spinal Cord.* 2011; 49:154-60.
17. Deforge D, Blackmer J, Moher D et al. Sexuality and reproductive health following spinal cord injury. *Evid Rep Technol Assess (Summ)* 2004; 109:1-8.
18. Maasoumi R, Zarei F, Merghati-Khoei E et al. Development of a Sexual Needs Rehabilitation Framework in Women Post-Spinal Cord Injury: A Study From Iran. *Arch Phys Med Rehabil.* 2018; 99:548-554.
19. Merghati-Khoei E, Emami-Razavi SH, Bakhtiyari M et al. Spinal cord injury and women's sexual life: case-control study. *Spinal Cord* 2017; 55:269-273.
20. Barrett OEC, Mattacola E, Finlay KA. "You feel a bit unsexy sometimes": The psychosocial impact of a spinal cord injury on sexual function and sexual satisfaction. *Spinal Cord.* 2023; 61:51-56.
21. Otero-Villaverde S, Ferreiro-Velasco ME, Montoto-Marqués A et al. Sexual satisfaction in women with spinal cord injuries. *Spinal Cord* 2015; 53:557-560.
22. Andrei Krassioukov, Stacy Elliott. *Neural Control and Physiology of Sexual Function: Effect of Spinal Cord Injury.* *Top Spinal Cord Inj Rehabil* 2017; 23: 1-10.
23. Hess MJ, Hough S. Impact of spinal cord injury on sexuality: broad-based clinical practice intervention and practical application. *J Spinal Cord Med.* 2012; 35:211-218.
24. Parker, M.G., Yau, M.K. Sexuality, Identity and Women with Spinal Cord Injury. *Sex Disabil* 2012; 30:15-27.
25. Sipski ML, Arenas A. Female sexual function after spinal cord injury. *Prog Brain Res* 2006; 152:441-447.
26. Stacy Elliott, Gaya Jeyathevan, Shea Hocaloski et al. Conception and development of Sexual Health indicators to advance the quality of spinal cord injury rehabilitation: SCI-High Project, *J Spinal Cord Med.* 2019; 42:68-84

27. Fritz HA, Dillaway H, Lysack C. "Don't think paralysis takes away your womanhood": Sexual intimacy after spinal cord injury. 2015; 69:1-10.
28. Lombardi G, Del Popolo G, Macchiarella A et al. Sexual rehabilitation in women with spinal cord injury: a critical review of the literature. *Spinal Cord* 2010; 48:842-849.
29. Zizzo J, Gater DR, Hough S et al. Sexuality, Intimacy, and Reproductive Health after Spinal Cord Injury. *Journal of Personalized Medicine*. 2022; 12:1985.
30. Marcalee Alexander, Frédérique Courtois, Stacy Elliott et al. Improving Sexual Satisfaction in Persons with Spinal Cord Injuries: Collective Wisdom. *Top Spinal Cord Inj Rehabil* 2017; 23: 57-70.
31. Thrussell H, Coggrave M, Graham A et al. Women's experiences of sexuality after spinal cord injury: a UK perspective. *Spinal Cord* 2018; 56:1084-1094
32. Khalili M, Berlin M, Pettersson K et al. Pregnancy, delivery, and neonatal outcomes among women with spinal cord injury in Sweden 1997-2015: A population-based cohort study. *Acta Obstet Gynecol Scand*. 2022; 101: 1282-1290.
33. Van den Borne K, Brands I, Spijkerman D. et al. Prevalence of parenthood in wheelchair-dependent persons with long-term spinal cord injury in the Netherlands. *Spinal Cord* 2018; 56:607-613
34. Deforge D, Blackmer J, Garritty C et al. Fertility following spinal cord injury: A systematic review. *Spinal Cord* 2005; 43:693-703.
35. Leduc BE. Treatment of infertility in 31 men with spinal cord injury. *Can J Urol*. 2012; 19:6432-6.
36. Sonksen J, Fode M, Lochner-Ernst D et al. Vibratory ejaculation in 140 spinal cord injured men and home insemination of their partners. *Spinal Cord* 2012;50:63-66.
37. Kathiresan AS, Ibrahim E, Aballa TC et al. Comparison of in vitro fertilization/intracytoplasmic sperm injection outcomes in male factor infertility patients with and without spinal cord injuries. *Fertil Steril*. 2011; 96:562-566.
38. Bertschy S, Schmidt M, Fiebag K. et al. Guideline for the management of pre-, intra-, and postpartum care of women with a spinal cord injury. *Spinal Cord* 2020; 58: 449-458
39. Smeltzer, Suzanne & Wetzel-Effinger, Lisa. Pregnancy in Women with Spinal Cord Injury. *Topics in Spinal Cord Injury Rehabilitation* 2009;15:29-42.
40. Sterling L, Keunen J, Wigdor E et al. Pregnancy outcomes in women with spinal cord lesions. *J Obstet Gynaecol Can*. 2013; 35:39-43.
41. Le Liepvre H, Dinh A, Idiard-Chamois B. et al. Pregnancy in spinal cord-injured women, a cohort study of 37 pregnancies in 25 women. *Spinal Cord* 2017; 55:167-171
42. Ghidini A, Healey A, Antreany M. and Simonson M.R. Pregnancy and women with spinal cord injuries. *Acta Obstetrica et Gynecologica Scandinavica* 2008; 87: 1006-1010
43. Pannek J., Bertschy S. Mission impossible? Urological management of patients with spinal cord injury during pregnancy: a systematic review. *Spinal Cord* 2011; 49: 1028-1032.
44. Robertson K, Ashworth F. Spinal cord injury and pregnancy. *Obstet Med*. 2022; 15:99-103.
45. Cabahug, P.G. Managing Spasticity in a Pregnant Woman with Spinal Cord Injury: a Review. *Curr Phys Med Rehabil*, 2018; 6:245-256.
46. Robertson K, Dawood R, Ashworth F. Vaginal delivery is safely achieved in pregnancies complicated by spinal cord injury: a retrospective 25-year observational study of pregnancy outcomes in a national spinal injuries centre. *BMC Pregnancy Childbirth*. 2020 29; 20:56.
47. Pebdani RN, Johnson KL, Amtmann D. Personal experiences of pregnancy and fertility in individuals with spinal cord injury. *Sexuality and Disability* 2014; 32:65-74.

Cite this paper as



Petropoulou A, Evangelopoulos DS, Vlamis J, Evangelopoulos ME. Sexual and reproductive health of patients with spinal cord injuries - Orientation to female fertility and pregnancy. *Acta Orthop Trauma Hell* 2024; 75(1): 74-83.