PPS
PAIN PRACTICE SOLUTIONS

The Sacroiliac Joint and Motor Vehicle Accidents: The Role of Regenerative Medicine Interventions

How to cite this paper:
O'Connor, D. (2025). The Sacroiliac Joint and Motor Vehicle Accidents: The Role of Regenerative Medicine Interventions. Innovative Injury Solutions.

Purpose Statement

This white paper will examine the sacroiliac joint and its role as a pain generator in the context of motor vehicle accidents and whiplash associated disorder. Treatment of the pain and dysfunction associated with these conditions by platelet rich plasma and amniotic allograft products as well as the effectiveness based on research will be discussed in detail.

Executive Summary

The sacroiliac joint SIJ is a well-recognized source of lower back and pelvic pain particularly in the setting of Motor Vehicle Accidents MVA and Whiplash Associated Disorder WAD. Its unique anatomy and biomechanical role in load transfer make it susceptible to injury contributing to pain generation and functional impairment.

Orthobiologic interventions including platelet-rich plasma PRP and amniotic allograft AA products represent evolving regenerative therapies with growing evidence supporting improvements in pain and function in select patient populations. Clinical application of both conventional and regenerative approaches in our practice is consistent with outcomes reported in the literature.

Key Findings

The SIJ is a complex load-bearing structure and a common contributor to lower back and pelvic pain

- Stability is maintained through both structural form closure and dynamic force closure mechanisms involving bone ligaments and musculature
- MVA can generate multidirectional forces capable of disrupting SIJ integrity and associated soft tissues
- Physical examination maneuvers can aid diagnosis however image-guided diagnostic injections remain the gold standard for confirming SIJ-mediated pain
- PRP is an autologous regenerative therapy utilizing concentrated platelets from the patient's own blood to support tissue repair reduce pain and improve function
- AA products are biologic therapies derived from amniotic and umbilical tissues formulated to promote healing and modulate inflammation
- Both PRP and AA deliver growth factors and cytokines that enhance healing responses particularly in cases of trauma poor vascularity or compromised host factors
- These therapies may promote cellular signaling and recruitment of progenitor cells supporting tissue remodeling in ligamentous tendinous and facet-related pathology
- Clinical studies demonstrate improvements in pain and functional outcomes when orthobiologics are appropriately applied to SIJ-related conditions
- Early targeted intervention in appropriate patients may improve outcomes and reduce progression to chronic pain states.

Implications

PRP and AA therapies represent viable adjuncts for patients with SIJ-mediated pain who have plateaued with conventional care. When applied in appropriately selected patients and guided by objective findings these interventions may improve clinical outcomes and help mitigate long-term disability.

From a medico-legal perspective referral to providers experienced in both diagnostic confirmation and regenerative treatment of SIJ pathology is recommended to ensure appropriate care pathways and documentation.

Introduction

The sacroiliac joint SIJ is a biomechanically complex structure located inferior to the lumbar spine exhibiting both synovial and syndesmotic characteristics with hyaline cartilage fibrocartilage and a supporting fibrous capsule.[1-3] Anatomical variation within the SIJ may predispose certain regions to increased mechanical stress.[4-5]

Functionally the SIJ plays a critical role in transferring ground reaction forces from the lower extremities to the axial skeleton through an integrated system of ligamentous and muscular attachments.[3] This combination of structural complexity and biomechanical demand contributes to its recognized role as a generator of lower back pain.[1]

Pelvic Girdle Pain PGP as defined by European guidelines encompasses pain localized between the posterior iliac crest and gluteal fold often associated with SIJ dysfunction and functional limitations in standing walking and sitting.[11]

WAD represents a spectrum of injury following MVA encompassing not only spinal pain but also neurological vestibular and psychological symptoms.[12-27] These injuries frequently involve multiple regions including the SIJ and its supporting structures.

Epidemiologic data suggest that the SIJ contributes to approximately 10-30% of lower back pain cases with higher prevalence reported in post-traumatic populations.[1-28-29] DePalma et al. identified SIJ pathology in 26% of patients with MVA-related lower back pain.[30]

Biomechanically SIJ stability is achieved through two primary mechanisms.

Form closure defined as passive stability from joint congruency and ligamentous support. [34-35]

Force closure defined as dynamic stabilization via musculoligamentous systems. [34]

Key muscular systems include the longitudinal sling posterior oblique sling and anterior oblique sling which coordinate load transfer across the pelvis.[28] Additional contributions arise from the pelvic floor diaphragm and transverse abdominis enhancing stability and intra-abdominal pressure regulation. [34-36]

Ligamentous structures including the sacroiliac sacrotuberous sacrospinous interosseous and iliolumbar ligaments play a critical role in limiting excessive motion and maintaining joint integrity. [3-34-35-37]

A key functional movement sacral nutation optimizes load transfer and ligament tension during weight-bearing activities. [34-38-39] Disruption of these coordinated systems during trauma such as MVA can result in pain instability and functional impairment of the SIJ.

Mechanics of Sacroiliac Joint Injury in Motor Vehicle Accidents

Following an MVA the SIJ and associated structures are exposed to forces sufficient to produce injury.[30-32] These forces may be directionally specific. In frontal or rear-end collisions the lower extremities are often braced against the vehicle which transmits ground reaction forces proximally into the pelvis.[40-42] This loading can produce rapid shear or posterior tilt of the innominate and drive the sacrum into nutation while the lumbar spine transitions into extension. [43-44]

Driving posture typically places the lumbosacral spine in a relatively flexed position with increased tension on posterior structures and compression anteriorly. [45-46] This position correlates with sacral counternutation which places increased strain on the dorsal and interosseous SIJ ligaments and represents a mechanically less stable state. [3-34-36-42-47-48]

At impact the pelvis accelerates forward while the spine initially extends followed by a rebound flexion phase.[45] This sequence generates combined tensile compressive and shear forces across the lumbosacral region. The anterior spinal column undergoes a traction to compression transition while posterior soft tissues are subjected to tensile loading. [34-45-49-50] These combined forces are poorly tolerated by the SIJ and have been associated with injury to both intraarticular and extraarticular structures. [3-43-51-54] As described by Vleeming et al disturbed or excessive force transfer through the SIJ can result in abnormal compressive or torsional stresses with deleterious tissue effects.[34]

In lateral impact collisions additional side bending and rotational forces are introduced.[55-57] The body undergoes an initial inertial movement followed by lateral bending toward the impacted side which creates tension on contralateral tissues and compression and shear on ipsilateral structures.[55-58] These forces may be amplified by contact with the vehicle interior and further increase stress on the SIJ and surrounding tissues.

In real-world MVAs forces are often multidirectional rather than isolated to a single plane.[58] This results in complex loading patterns across the SIJ with combined vectors that can exceed physiologic tolerance and contribute to ligamentous and joint injury.[51-59-60]

Pathophysiology of Sacroiliac Joint Injury

Traumatic loading of the SIJ can result in injury to capsular ligamentous and periarticular structures as well as activation of an inflammatory response. Collagen fiber disruption within SIJ ligaments has been demonstrated in patients with SIJ-mediated pain.[2] Both intraarticular and extraarticular structures may serve as pain generators given the presence of nociceptive fibers within capsuloligamentous tissues.[3-61-65]

This distribution of pain-sensitive structures may contribute to variability in diagnostic block responses and supports the need to evaluate both intraarticular and extraarticular sources when SIJ pathology is suspected.[66-67]

The inflammatory response following injury is part of normal tissue repair however excessive or prolonged inflammation may contribute to persistent pain states.[68-71] Elevated cytokine activity has been associated with both pain signaling and degenerative changes within the axial skeleton.[72-73] Pathological changes at the SIJ may include inflammatory cell infiltration cartilage degeneration subchondral bone disruption synovitis and enthesitis.[10-74]

Persistent inflammatory signaling may also contribute to peripheral and central sensitization leading to amplification of pain responses.[75-76] This mechanism is well supported in the literature with neuroimmune processes playing a central role in chronic pain development.[77]

Certain populations may be at increased risk for SIJ pathology. Patients with prior lumbar fusion may experience altered force distribution resulting in increased stress transfer to the SIJ.[78-79] Additionally females have been shown to have a higher prevalence of SIJ-related pain which may be related to anatomical and biomechanical differences.[80-82]

Diagnosis of Sacroiliac Joint Pathology

Accurate diagnosis of SIJ-mediated pain requires correlation of symptom location physical examination and confirmatory testing. Pain localized to the region inferomedial to the posterior superior iliac spine identified during provocation testing Fortin Finger Test has been described as characteristic of SIJ pathology. 33-83-84

Provocative testing clusters including thigh thrust Patrick test distraction compression sacral thrust and Gaenslen tests have demonstrated clinical utility when at least three tests reproduce the patient's familiar pain.[85-87] Variability in examination technique and training may influence diagnostic accuracy.[86-91]

Due to these limitations diagnostic SIJ blocks remain the most reliable method for confirming intraarticular SIJ pain.[92] Image-guided injections using fluoroscopy or CT are recommended to ensure accuracy as non-guided injections have demonstrated poor reliability.[96-98]

Evidence supports the use of single or dual diagnostic blocks to confirm SIJ as a pain generator.[29-92-95] Systematic reviews have demonstrated good evidence for controlled anesthetic blocks in diagnosing SIJ-mediated pain.[66]

In cases of diagnostic uncertainty advanced imaging such as MRI may be utilized to identify structural pathology not visible on standard radiographs.[99]

The Basic Science of Platelet-Rich Plasma PRP and Amniotic Allografts

Platelet-Rich Plasma PRP is an autologous concentration of platelets derived from the patient and processed to supraphysiologic levels to support tissue repair reduce pain and improve function[100-102] PRP has demonstrated efficacy across multiple musculoskeletal conditions including knee osteoarthritis carpal tunnel syndrome lateral epicondylitis rotator cuff pathology and lower back pain.[102-107]

PRP is produced by drawing a defined volume of venous blood and centrifuging it to isolate a plasma fraction with elevated platelet concentration.[101] Normal platelet counts range from 150000 to 450000 per microliter whereas PRP is typically defined as 3-5 times baseline concentration.[101-108] Several formulations exist including leukocyte-rich PRP leukocyte-poor PRP platelet-poor plasma platelet-rich fibrin and platelet lysate each with condition-specific applications.[109-112]

The therapeutic effects of PRP are driven by the release of growth factors and bioactive proteins that initiate and support the healing cascade.[113] These include vascular endothelial growth factor epidermal growth factor hepatocyte growth factor platelet-derived growth factor tumor necrosis factor alpha and transforming growth factor beta 1 which collectively promote angiogenesis cellular proliferation and tissue regeneration.[113-117]

Platelet activation also facilitates recruitment and signaling of progenitor cells which migrate to sites of injury and contribute to tissue remodeling.[113-116] These cells may differentiate into tendon cartilage muscle nerve and bone tissue supporting structural and functional recovery.[118-122]

Amniotic Allografts AA are regenerative biologic therapies derived from human amniotic membrane fluid umbilical cord tissue and or blood and are used in regenerative orthopedics.[123] These products may be processed into injectable formulations or surgical grafts while preserving growth factors cytokines extracellular matrix components and antimicrobial peptides.[123-124]

AA products function through similar biologic pathways as PRP including modulation of inflammation enhancement of tissue repair and promotion of cellular signaling.[123] Research supports their role in recruiting progenitor cells and facilitating healing responses in injured tissues.[125-128]

AA products demonstrate low immunogenicity due to reduced expression of human leukocyte antigens which minimizes immune recognition while also modulating local immune responses at the treatment site.[129-130] These properties allow for safe application across a range of conditions including osteoarthritis tendinopathies degenerative spinal conditions cartilage defects and radiculopathy.[131-139]

Both PRP and AA have demonstrated clinical utility in patients who have not responded to conventional conservative care. Their use represents a logical progression in treatment when standard therapies such as physical therapy medications or corticosteroid injections fail to provide adequate relief.

From a practical standpoint treatment selection should consider patient-specific factors. PRP requires venipuncture and on-site processing which may take 30-60 minutes and may be influenced by patient health factors that affect platelet quality.[101-140-142] In contrast AA products are readily available without the need for blood draw or processing and may be preferable in patients with poor biologic substrate procedural limitations or time constraints.

Both therapies offer meaningful potential to improve pain and function however optimal outcomes depend on appropriate patient selection clinical judgment and alignment of treatment approach with patient-specific needs.

Evidence for PRP or AA Use in the Sacroiliac Joint Complex

Current literature consistently demonstrates that orthobiologic interventions such as PRP provide more durable improvements in pain and function compared to corticosteroid injections. Corticosteroids remain effective for short-term symptom relief but are not recommended as a stand-alone long-term solution due to potential adverse effects and risk of tissue degeneration.[143]

Multiple case series and clinical studies involving complex SIJ pain populations including trauma MVA postpartum conditions and prior lumbar fusion have demonstrated meaningful improvements in pain and function following PRP treatment.[144-149] These improvements are typically observed within 3 to 6 months and are often sustained at follow-up intervals ranging from 6 months to 4 years. Image guidance using ultrasound or fluoroscopy was consistently utilized to optimize accuracy and safety.[146]

Prospective and single-arm trials further support these findings. One study evaluating ultrasound-guided PRP injections into the SIJ demonstrated progressive improvement in pain and function sustained at 6 months.[150] Similar improvements were reported at 1 and 3 months in a more recent trial.[151]

Comparative studies have consistently shown superior long-term outcomes with PRP relative to corticosteroids. In a 2017 study by Singla et al outcomes at 6 weeks and 3 months favored PRP with reported efficacy of 90 percent in the PRP group compared to 25 percent in the corticosteroid group.[152] Another trial comparing fluoroscopy-guided injections found corticosteroids provided greater short-term benefit at 4 weeks whereas PRP demonstrated superior outcomes by 8 weeks including improvements in imaging findings.[153] Additional comparative trials involving a combined total of 187 patients have demonstrated similar trends with greater long-term improvements in pain and disability favoring PRP.[154-156]

A randomized controlled trial in 2022 evaluated PRP combined with prolotherapy targeting ligamentous structures around the SIJ.[157] This study demonstrated improved outcomes at 6 months and highlighted the importance of addressing both intraarticular and extraarticular pain generators.[157-158] Additional studies have supported the role of extraarticular structures in SIJ pain and demonstrated positive outcomes when these tissues are targeted with PRP.[159-161]

Systematic reviews further reinforce these findings. Reviews published in 2020 and 2021 reported clinically meaningful improvements in SIJ-related pain with PRP including up to 90 percent of patients achieving at least 50 percent symptom reduction at 12 months.[162-163] More recent analyses in 2024 have confirmed a positive association between PRP use and improvements in pain and disability with evidence suggesting superiority over corticosteroid injections in long-term outcomes.[164]

These consistent findings have led to inclusion of PRP in current best practice guidelines which recommend consideration of PRP when conservative treatments fail or are contraindicated.[95]

With respect to AA products emerging evidence supports their use in SIJ-related pain. A multicenter retrospective study evaluating 38 patients with persistent SIJ dysfunction who failed conservative care demonstrated meaningful improvements in pain and function at 90 days following treatment.[165] Reported outcomes included a 42 percent reduction in pain scores and a 22 percent improvement in functional measures.[165]

Although the volume of evidence for AA is currently less robust than for PRP the underlying biologic mechanisms are similar including modulation of inflammation and support of tissue repair. When patient-specific factors such as overall health tolerance for blood draw or time constraints are considered AA may represent an appropriate and effective alternative.

Early Intervention to Prevent Chronic Pain

In patients presenting with acute pain following MVA and WAD early management should prioritize prevention of progression to chronic pain. Secondary prevention strategies focused on early identification and targeted treatment of acute or subacute pain can significantly improve outcomes and reduce long-term disability.[166] The primary objective is to prevent central sensitization even in the presence of peripheral sensitization.[166-168]

Central sensitization refers to amplification of nociceptive signaling within the central nervous system resulting in heightened pain perception and reduced thresholds for pain.[169] Peripheral sensitization involves increased responsiveness of nociceptive pathways at the site of injury leading to amplified signaling to the central nervous system.[170] Both processes contribute to the development and persistence of chronic pain and are commonly observed in WAD populations where they are associated with reduced quality of life.[171-174]

Importantly these processes may be mitigated or reversed when peripheral pain generators are effectively treated. Studies have demonstrated that normalization of nociceptive input from injured tissues can reduce central sensitization and improve functional outcomes.[175-179] Early intervention targeting the inflammatory response associated with WAD has been shown to reduce progression to chronic pain and decrease the likelihood of more invasive treatments.[180-183]

In this context early use of targeted interventions including injections should be considered to control inflammation reduce pain and improve function. These approaches may provide short-term symptom relief while facilitating progression to longer-term regenerative strategies such as PRP or AA which have demonstrated sustained benefits.

Psychological factors also play a significant role in chronic pain development. Variables such as kinesiophobia anxiety depression post-traumatic stress disorder and negative recovery expectations are associated with poorer outcomes following MVA.[184-191] Early psychological response within the first 3 weeks has been shown to predict long-term pain severity.[192] Effective pain control has been associated with improvement in these psychological factors further supporting early intervention.[193-194]

Early identification of high-risk patients combined with targeted medical management is essential to prevent transition to chronic pain and should be a central component of clinical decision making in WAD-related conditions.

Real World Application

Clinical evidence supports a multimodal approach to managing SIJ-related pain following MVA. Patients frequently present in a pro-inflammatory state with high levels of pain and functional limitation.[195-197] While active rehabilitation is critical for recovery pain often limits participation in physical therapy and delays return to normal function.[198-200]

Strategic use of corticosteroid injections in the acute to early subacute phase can reduce pain sufficiently to allow participation in rehabilitation and provide insight into treatment response. As noted in the literature early effective pain management is critical in reducing progression to chronicity.[201]

However many patients continue to experience persistent symptoms despite corticosteroid use.[201-202] To improve long-term outcomes evidence supports transitioning from corticosteroid-based management to regenerative approaches. An early corticosteroid injection followed by PRP or AA intervention within an appropriate time frame may optimize the healing environment and improve long-term recovery.[182]

This approach aligns with evidence demonstrating superior long-term outcomes with orthobiologic therapies and the potential detrimental effects of repeated corticosteroid exposure on joint structures.[143-203-204]

PRP and AA therapies provide concentrated biologic signaling that supports tissue repair and reduces pain allowing greater participation in rehabilitation and improved functional outcomes. When integrated into a structured treatment pathway these interventions offer a practical and evidence-based strategy to improve recovery and reduce chronic disability in patients with SIJ-related pain following WAD.

Clinical Implications and Future Directions

Management of SIJ pain associated with WAD remains challenging with standard care alone often yielding suboptimal long-term outcomes. Evidence indicates that approximately 50 percent of patients recover within 3 to 6 months while a substantial proportion continue to experience symptoms for 1 to 2 years or longer. Among those with persistent symptoms 30 to 40 percent report ongoing mild to moderate pain and 10 to 20 percent develop moderate to severe chronic pain syndromes.[201]

Given these outcomes there is a clear need for more effective treatment strategies. Emerging evidence supports the integration of orthobiologic therapies such as PRP and AA into a multimodal care model. When combined with appropriate pain management and structured physical therapy these interventions may improve pain control enhance functional recovery and alter the trajectory toward chronic disability.

Clinical experience within this organization has demonstrated consistent improvements in pain and function when early targeted interventions are followed by regenerative treatments directed at the SIJ and associated soft tissues. This approach emphasizes early symptom control restoration of mobility and progressive strengthening to return patients to baseline function.

Future research including retrospective analyses and randomized controlled trials is warranted to further validate these findings and support broader adoption of this treatment paradigm. Ongoing efforts are focused on publishing outcomes associated with this multimodal approach to contribute to the evolving standard of care.

Conclusion

SIJ pathology in the setting of WAD presents a complex clinical challenge with a high risk of persistent pain and disability. Traditional care pathways alone may not sufficiently address the underlying mechanisms driving chronicity.

Evidence and clinical experience support the use of orthobiologic interventions such as PRP and AA following initial pain management to improve long-term outcomes. These therapies offer a pathway toward enhanced recovery by addressing both the biologic and functional components of injury.

Adoption of a structured multimodal approach that integrates early intervention regenerative therapies and rehabilitation may significantly improve patient outcomes and reduce long-term disability in this population.

Next Steps

For attorneys and healthcare teams managing patients with WAD and SIJ-related pain a clear understanding of evidence-based treatment pathways is essential. The principles outlined in this document support a structured approach to care that emphasizes early intervention accurate diagnosis and progression to regenerative therapies when appropriate.

PRP and AA interventions have demonstrated the potential to improve pain and function and to reduce the likelihood of chronic pain when incorporated into a comprehensive treatment plan. Referral to providers experienced in these techniques may improve both clinical outcomes and the overall strength of medico-legal documentation.

References:

1. Poilliot AJ, Zwirner J, Doyle T, et al. A systematic review of the normal sacroiliac joint anatomy and adjacent tissues for pain physicians. Pain Physician 2019;22:E247.
2. Toyohara R, Ohashi T. A literature review of biomechanical studies on physiological and pathological sacroiliac joints: Articular surface structure, joint motion, dysfunction and treatments. Clinical Biomechanics 2024;114:106233.
3. Kiapour A, Joukar A, Elgafy H, et al. Biomechanics of the sacroiliac joint: anatomy, function, biomechanics, sexual dimorphism, and causes of pain. International journal of spine surgery 2020;14:S3-S13.
4. Jurik AG, Herregods N. The sacroiliac joint across ages–what is normal? Therapeutic advances in musculoskeletal disease 2024;16:1759720X241241126.
5. Trentadue TP, Anderson TL, Wenger DE, et al. Prevalence of accessory sacroiliac joint anatomy and associated clinical features. Skeletal radiology 2023;52:1359-1368.
6. Goldthwait JE, Osgood RB. A consideration of the pelvic articulations from an anatomical, pathological and clinical standpoint. The Boston Medical and Surgical Journal 1905;152:593-601.
7. De Salvatore S, Russo F, Vadala G, et al. Interventional treatments for low back pain due to sacroiliac joint dysfunction: a systematic review of the literature. European Spine Journal 2024;33:1407-1423.
8. Yao Z, Li L, Zhang P, et al. Biomechanics evaluation of sacroiliac joint pain after lumbosacral fusion: a finite element analysis. Journal of Orthopaedic Research® 2023;41:875-883.
9. Whitney KE, Sugimoto D, d'Hemecourt CA, et al. Running gait biomechanics in female runners with sacroiliac joint pain. Journal of Physical Therapy Science 2022;34:327-334.
10. Aranke M, McCrudy G, Rooney K, et al. Minimally invasive and conservative interventions for the treatment of sacroiliac joint pain: a review of recent literature. Orthopedic reviews 2022;14:31915.
11. Vleeming A, Albert HB, Östgaard HC, et al. European guidelines for the diagnosis and treatment of pelvic girdle pain. European Spine Journal 2008;17:794-819.
12. Ghorayeb JH. The nosological classification of whiplash-associated disorder: a narrative review. The Journal of the Canadian Chiropractic Association 2021;65:76.
13. Vállez García D, Dierckx RAJO, Otte A, et al. Whiplash, Real or Not Real? A Review and New Concept. PET and SPECT in Neurology, 2014;947-963.
14. Bannister G, Amirfeyz R, Kelley S, et al. Whiplash injury. The Journal of Bone & Joint Surgery British Volume 2009;91:845-850.
15. Fundaun J, Kolski M, Baskozos G, et al. Nerve pathology and neuropathic pain after whiplash injury: a systematic review and meta-analysis. Pain 2022;163:e789-e811.
16. Särkilahti N, Leino S, Takatalo J, et al. The symptom profile of people with whiplash-associated disorder–a mixed-method systematic review. Journal of Bodywork and Movement Therapies 2024.
17. Alderink GJ, Ashby BM. Cervical Spine: Lateral Whiplash, Neck Pain, and Nerve Palsy. Clinical Kinesiology and Biomechanics: A Problem-Based Learning Approach: Springer, 2023;145-177.
18. Urano F, Ono S, Hara R, et al. SAT0400 Is whiplash injury a triggering or exacerbating factor for axial spondyloarthritis? Annals of the Rheumatic Diseases 2017;76:923.
19. Cernovsky ZZ, Mann SC, Velamoor VR, et al. The Need for Three Separate Parallel WAD Ratings of Whiplash Injuries to Cervical, Lumbosacral, and Thoracic Spine in Clinical Assessments of Injured Motorists. European Journal of Medical and Health Sciences 2021;3:154-159.
20. Al-Khazali HM, Ashina H, Iljazi A, et al. Psychiatric sequelae following whiplash injury: a systematic review. Frontiers in psychiatry 2022;13:814079.
21. Kuperman P, Granovsky Y, Fadel S, et al. Head- and neck-related symptoms post-motor vehicle collision (MVC): Separate entities or two-sides of the same coin? Injury 2021;52:1227-1233.
22. Sterling M, Jull G, Vicenzino B, et al. Physical and psychological factors predict outcome following whiplash injury. Pain 2005;114:141-148.
23. Rebbeck T, Evans K, Elliott JM. Concussion in Combination With Whiplash-Associated Disorder May Be Missed in Primary Care: Key Recommendations for Assessment and Management. J Orthop Sports Phys Ther 2019;49:819-828.

24. Gil C, Decq P. How similar are whiplash and mild traumatic brain injury? A systematic review. Neurochirurgie 2021;67:238-243.
25. Alexander MP. The evidence for brain injury in whiplash injuries. Pain Research and Management 2003;8:19-23.
26. He Y, Gao J, Liu Y, et al. Global trends and hotspots related to whiplash injury: A visualization study. Medicine 2024;103:e38777.
27. Stone D, Ward E, Knijnik S, et al. Whiplash-associated dysphagia and dysphonia: A scoping review. Dysphagia 2021;36:303-315.
28. Enix DE, Mayer JM. Sacroiliac joint hypermobility biomechanics and what it means for health care providers and patients. Pm&r 2019;11:S32-S39.
29. Thawrani DP, Agabegi SS, Asghar F. Diagnosing sacroiliac joint pain. JAAOS-Journal of the American Academy of Orthopaedic Surgeons 2019;27:85-93.
30. DePalma M, Ketchum J, Saullo T, et al. Structural etiology of chronic low back pain due to motor vehicle collision. Pain medicine 2011;12:1622-1627.
31. Cohen SP, Chen Y, Neufeld NJ. Sacroiliac joint pain: a comprehensive review of epidemiology, diagnosis and treatment. Expert review of neurotherapeutics 2013;13:99-116.
32. Chou LH, Slipman CW, Bhagia SM, et al. Inciting events initiating injection-proven sacroiliac joint syndrome. Pain Medicine 2004;5:26-32.
33. van der Wurff P, Buijs EJ, Groen GJ. Intensity mapping of pain referral areas in sacroiliac joint pain patients. Journal of manipulative and physiological therapeutics 2006;29:190-195.
34. Vleeming A, Schuenke M, Masi A, et al. The sacroiliac joint: an overview of its anatomy, function and potential clinical implications. Journal of anatomy 2012;221:537-567.
35. Vleeming A, Schuenke M. Form and force closure of the sacroiliac joints. PM&R 2019;11:S24-S31.
36. Pel J, Spoor C, Pool-Goudzwaard A, et al. Biomechanical analysis of reducing sacroiliac joint shear load by optimization of pelvic muscle and ligament forces. Annals of biomedical engineering 2008;36:415-424.
37. Eichenseer PH, Sybert DR, Cotton JR. A finite element analysis of sacroiliac joint ligaments in response to different loading conditions. Spine 2011;36:E1446-E1452.
38. Yoo W-g. Effects of individual strengthening exercises for the stabilization muscles on the nutation torque of the sacroiliac joint in a sedentary worker with nonspecific sacroiliac joint pain. Journal of Physical Therapy Science 2015;27:313-314.
39. van Wingerden J-P, Vleeming A, Buyruk H, et al. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. European Spine Journal 2004;13:199-205.
40. Kibsgård TJ, Røise O, Sturesson B, et al. Radiosteriometric analysis of movement in the sacroiliac joint during a single-leg stance in patients with long-lasting pelvic girdle pain. Clinical Biomechanics 2014;29:406-411.
41. Lindsey DP, Parrish R, Gundanna M, et al. Biomechanics of unilateral and bilateral sacroiliac joint stabilization. Journal of Neurosurgery: Spine 2018;28:326-332.
42. Toyohara R, Kurosawa D, Hammer N, et al. Finite element analysis of load transition on sacroiliac joint during bipedal walking. Scientific reports 2020;10:13683.
43. Zhang S, Chen Y, Ren R, et al. Quantitative study on the biomechanical mechanism of sacroiliac joint subluxation: A finite element study. Journal of Orthopaedic Research® 2022;40:1223-1235.
44. Vleeming AA, Snijders C, Stoeckart R, et al. The role of the sacroiliac joints in coupling between spine, pelvis, legs and arms. Movement, stability & low back pain: Churchill Livingstone, 1997;53-71.
45. Gates D, Bridges A, Welch TD, et al. Lumbar loads in low to moderate speed rear impacts: SAE Technical Paper, 2010.
46. Fewster KM, Zehr JD, Gooyers CE, et al. Characterizing Lumbar Spine Kinematics and Kinetics During Simulated Low-Speed Rear Impact Collisions. Journal of Applied Biomechanics 2022;38:155-163.
47. McParland B, Sina RE, DeLuca A. Osteopathic Manipulative Treatment: Muscle Energy Procedure-Sacral Dysfunctions. StatPearls [Internet]: StatPearls Publishing, 2024.
48. Li J, Li Y, Ping R, et al. Biomechanical analysis of sacroiliac joint motion following oblique-pulling manipulation with or without pubic symphysis injury. Frontiers in Bioengineering and Biotechnology 2022;10:960090.
49. Siegmund GP. The biomechanics of whiplash injury. British Columbia Medical Journal 2002;44:243-247.
50. Fast A, Sosner J, Begeman P, et al. Lumbar spinal strains associated with whiplash injury: a cadaveric study. American journal of physical medicine & rehabilitation 2002;81:645-650.
51. Miller JA, Schultz AB, Andersson GB. Load-displacement behavior of sacroiliac joints. Journal of orthopaedic research 1987;5:92-101.
52. Fortin JD. Sacroiliac joint dysfunction. Journal of back and musculoskeletal rehabilitation 1993;3:31-43.
53. Kondratiev A, Smetankina N, Staude V. Biomechanical Analysis of Stress–Strain Distribution in the Lumbar Spine–Sacrum–Pelvis System with Emphasis on Sacroiliac Joint Dysfunction. Prosthesis 2024;7:4.
54. Raj MA, Ampat G, Varacallo MA. Sacroiliac joint pain. StatPearls [Internet]: StatPearls Publishing, 2023.
55. Furbish C, Welcher J, Brink J, et al. Occupant kinematics and loading in low speed lateral impacts. SAE International Journal of Advances and Current Practices in Mobility 2019;1:1470-1490.

56. Yoganandan N, Pintar FA, Stemper BD, et al. Biomechanics of side impact: injury criteria, aging occupants, and airbag technology. Journal of biomechanics 2007;40:227-243.
57. Kumaresan S, Sances A, Carlin F, et al. Biomechanics of side impact injuries: evaluation of seat belt restraint system, occupant kinematics and injury potential. 2006 International Conference of the IEEE Engineering in Medicine and Biology Society 2006;87-90.
58. Ivancic PC. Mechanisms and mitigation of head and spinal injuries due to motor vehicle crashes. journal of orthopaedic & sports physical therapy 2016;46:826-833.
59. BOWEN V, CASSIDY JD. Macroscopic and microscopic anatomy of the sacroiliac joint from embryonic life until the eighth decade. Spine 1981;6:620-628.
60. Leone E, Garipoli A, Ripani U, et al. Imaging review of pelvic ring fractures and its complications in high-energy trauma. Diagnostics 2022;12:384.
61. Buchanan P, Vodapally S, Lee DW, et al. Successful diagnosis of sacroiliac joint dysfunction. Journal of pain research 2021:3135-3143.
62. Murakami E, Tanaka Y, Aizawa T, et al. Effect of periarticular and intraarticular lidocaine injections for sacroiliac joint pain: prospective comparative study. Journal of Orthopaedic Science 2007;12:274-280.
63. Le Goff B, Berthelot J-M, Maugars Y. Ultrasound assessment of the posterior sacroiliac ligaments. Clinical and Experimental Rheumatology-Incl Supplements 2011;29:1014.
64. Wyke B. The neurology of joints: a review of general principles. Clinics in Rheumatic Diseases 1981;7:223-239.
65. Kurosawa D, Murakami E, Ozawa H, et al. A diagnostic scoring system for sacroiliac joint pain originating from the posterior ligament. Pain Medicine 2017;18:228-238.
66. Simopoulos TT, Manchikanti L, Singh V, et al. A systematic evaluation of prevalence and diagnostic accuracy of sacroiliac joint interventions. Pain physician 2012;15:E305.
67. Hansen HC, Brown AM, Cohen SP, et al. Sacroiliac joint interventions: a systematic review. Database of Abstracts of Reviews of Effects (DARE): Quality-assessed Reviews [Internet] 2007.
68. Chen L, Deng H, Cui H, et al. Inflammatory responses and inflammation-associated diseases in organs. Oncotarget 2017;9:7204.
69. Molinos M, Almeida CR, Caldeira J, et al. Inflammation in intervertebral disc degeneration and regeneration. Journal of the Royal Society Interface 2015;12:20141191.
70. Gerum M, Simonin F. Behavioral characterization, potential clinical relevance and mechanisms of latent pain sensitization. Pharmacology & Therapeutics 2022;233:108032.
71. Rotem G, Herman A, Lidar M, et al. Post-traumatic arthritis of the sacroiliac joints mimicking inflammatory sacroiliitis: analysis of consecutive computed tomography examinations. Clinical Radiology 2020;75:433-440.
72. Palada V, Ahmed AS, Finn A, et al. Characterization of neuroinflammation and periphery-to-CNS inflammatory cross-talk in patients with disc herniation and degenerative disc disease. Brain, behavior, and immunity 2019;75:60-71.
73. Buchanan BK, Varacallo M. Sacroiliitis. 2017.
74. Cui Y, Zhang X, Zhao Z, et al. The relationship between histopathological and imaging features of sacroiliitis. International Journal of Clinical and Experimental Medicine 2015;8:5904.
75. Peng B, DePalma MJ. Cervical disc degeneration and neck pain. Journal of pain research 2018:2853-2857.
76. Öz N, Özer A, Duruöz MT. Central Sensitization and Its Role in Persistent Pain Among Spondyloarthritis Patients on Biological Treatments. Medicina 2025;61:319.
77. Lutke Schipholt IJ, Scholten-Peeters GG, Koop MA, et al. Systemic neuroimmune responses in people with non-specific neck pain and cervical radiculopathy, and associations with clinical, psychological, and lifestyle factors. Frontiers in Molecular Neuroscience 2022;15:1003821.
78. Ivanov AA, Kiapour A, Ebraheim NA, et al. Lumbar fusion leads to increases in angular motion and stress across sacroiliac joint: a finite element study. Spine 2009;34:E162-E169.
79. Ha K-Y, Lee J-S, Kim K-W. Degeneration of sacroiliac joint after instrumented lumbar or lumbosacral fusion: a prospective cohort study over five-year follow-up. Spine 2008;33:1192-1198.
80. O'shea F, Boyle E, Salonen D, et al. Inflammatory and degenerative sacroiliac joint disease in a primary back pain cohort. Arthritis care & research 2010;62:447-454.
81. Ebraheim NA, Biyani A. Percutaneous computed tomographic stabilization of the pathologic sacroiliac joint. Clinical Orthopaedics and Related Research (1976-2007) 2003;408:252-255.
82. Joukar A, Shah A, Kiapour A, et al. Sex specific sacroiliac joint biomechanics during standing upright: a finite element study. Spine 2018;43:E1053-E1060.
83. Forst SL, Wheeler M, Fortin JD, et al. The sacroiliac joint: anatomy, physiology and clinical significance. Pain physician 2006;9:61.
84. Fortin JD, Falco F. The Fortin finger test: an indicator of sacroiliac pain. American journal of orthopedics (Belle Mead, NJ) 1997;26:477-480.
85. Laslett M, Aprill CN, McDonald B, et al. Diagnosis of sacroiliac joint pain: validity of individual provocation tests and composites of tests. Manual therapy 2005;10:207-218.

86. van der Wurff P, Buijs EJ, Groen GJ. A multitest regimen of pain provocation tests as an aid to reduce unnecessary minimally invasive sacroiliac joint procedures. Archives of physical medicine and rehabilitation 2006;87:10-14.
87. Szadek KM, van der Wurff P, van Tulder MW, et al. Diagnostic validity of criteria for sacroiliac joint pain: a systematic review. The Journal of pain 2009;10:354-368.
88. Laslett M. Evidence-based diagnosis and treatment of the painful sacroiliac joint. Journal of Manual & Manipulative Therapy 2008;16:142-152.
89. Werneke MW, Deutscher D, Hart DL, et al. McKenzie lumbar classification: inter-rater agreement by physical therapists with different levels of formal McKenzie postgraduate training. Spine 2014;39:E182-E190.
90. Lam OT, Strenger DM, Chan-Fee M, et al. Effectiveness of the McKenzie method of mechanical diagnosis and therapy for treating low back pain: literature review with meta-analysis. Journal of orthopaedic & sports physical therapy 2018;48:476-490.
91. van Helvoirt H, Tempelman H, van der Vet P, et al. Reliability of the McKenzie Method of Mechanical Diagnosis and Therapy in the examination of spinal pain, including the OTHER classifications: Reliability of the McKenzie Method in spinal pain. Brazilian Journal of Physical Therapy 2025;29:101154.
92. Chuang C-W, Hung S-K, Pan P-T, et al. Diagnosis and interventional pain management options for sacroiliac joint pain. Tzu Chi Medical Journal 2019;31:207-210.
93. Slipman CW, Chow WSWDW, Chou L, et al. Sacroiliac joint syndrome. Pain Physician 2001;4:143.
94. Jung MW, Schellhas K, Johnson B. Use of diagnostic injections to evaluate sacroiliac joint pain. International Journal of Spine Surgery 2020;14:S30-S34.
95. Sayed D, Deer TR, Tieppo Francio V, et al. American Society of Pain and Neuroscience Best Practice (ASPN) guideline for the treatment of sacroiliac disorders. Journal of Pain Research 2024:1601-1638.
96. Rosenberg JM, Quint DJ, de Rosayro AM. Computerized tomographic localization of clinically-guided sacroiliac joint injections. The Clinical journal of pain 2000;16:18-21.
97. Nacey NC, Patrie JT, Fox MG. Fluoroscopically guided sacroiliac joint injections: comparison of the effects of intraarticular and periarticular injections on immediate and short-term pain relief. American Journal of Roentgenology 2016;207:1055-1061.
98. Bessar AA, Arnaout MM, Basha MAA, et al. Computed tomography versus fluoroscopic guided-sacroiliac joint injection: a prospective comparative study. Insights into Imaging 2021;12:38.
99. Diekhoff T, Sieper J, Greese J, et al. Comparison of Radiography and MRI to Detect Structural Lesions of the Sacroiliac Joints: The SIMACT Study. Seminars in Musculoskeletal Radiology 2017;A008.
100. Middleton KK, Barro V, Muller B, et al. Evaluation of the effects of platelet-rich plasma (PRP) therapy involved in the healing of sports-related soft tissue injuries. The Iowa orthopaedic journal 2012;32:150.
101. Arnoczky SP, Shebani-Rad S. The basic science of platelet-rich plasma (PRP): what clinicians need to know. Sports medicine and arthroscopy review 2013;21:180-185.
102. Shen L, Yuan T, Chen S, et al. The temporal effect of platelet-rich plasma on pain and physical function in the treatment of knee osteoarthritis: systematic review and meta-analysis of randomized controlled trials. Journal of orthopaedic surgery and research 2017;12:16.
103. Senna MK, Shaat RM, Ali AAA. Platelet-rich plasma in treatment of patients with idiopathic carpal tunnel syndrome. Clinical rheumatology 2019;38:3643-3654.
104. Li S, Yang G, Zhang H, et al. A systematic review on the efficacy of different types of platelet-rich plasma in the management of lateral epicondylitis. Journal of Shoulder and Elbow Surgery 2022;31:1533-1544.
105. A. Hamid MS, Sazlina SG. Platelet-rich plasma for rotator cuff tendinopathy: A systematic review and meta-analysis. Plos one 2021;16:e0251111.
106. Xuan Z, Yu W, Dou Y, et al. Efficacy of platelet-rich plasma for low back pain: a systematic review and meta-analysis. Journal of Neurological Surgery Part A: Central European Neurosurgery 2020;81:529-534.
107. Wongjarupong A, Pairuchvej S, Laohapornsvan P, et al. "Platelet-Rich Plasma" epidural injection an emerging strategy in lumbar disc herniation: a Randomized Controlled Trial. BMC Musculoskeletal Disorders 2023;24:335.
108. Straum OK. The optimal platelet concentration in platelet-rich plasma for proliferation of human cells in vitro—diversity, biases, and possible basic experimental principles for further research in the field: A review. PeerJ 2020;8:e10303.
109. Kim J-H, Park Y-B, Ha C-W, et al. Adverse reactions and clinical outcomes for leukocyte-poor versus leukocyte-rich platelet-rich plasma in knee osteoarthritis: a systematic review and meta-analysis. Orthopaedic Journal of Sports Medicine 2021;9:23259671211011948.
110. Alves R, Grimalt R. A review of platelet-rich plasma: history, biology, mechanism of action, and classification. Skin appendage disorders 2018;4:18-24.
111. Collins T, Alexander D, Barkatali B. Platelet-rich plasma: a narrative review. EFORT open reviews 2021;6:225-235.
112. Ehrenfest DMD, Rasmusson L, Albrektsson T. Classification of platelet concentrates: from pure platelet-rich plasma (P-PRP) to leucocyte-and platelet-rich fibrin (L-PRF). Trends in biotechnology 2009;27:158-167.
113. Dos Santos RG, Santos GS, Alkass N, et al. The regenerative mechanisms of platelet-rich plasma: A review. Cytokine 2021;144:155560.

114. Duchesne E, Dufresne SS, Dumont NA. Impact of inflammation and anti-inflammatory modalities on skeletal muscle healing: from fundamental research to the clinic. Physical therapy 2017;97:807-817.
115. Järvinen TA, Järvinen M, Kalimo H. Regeneration of injured skeletal muscle after the injury. Muscles, ligaments and tendons journal 2014;3:337.
116. Etulain J. Platelets in wound healing and regenerative medicine. Platelets 2018;29:556-568.
117. Pineda-Cortel MR, Suarez C, Cabrera J-T, et al. Complexity of Platelet-Rich Plasma: Mechanism of Action, Growth Factor Utilization and Variation in Preparation. Plasmatology 2024;18:26348535241277625.
118. Li H, Usas A, Poddar M, et al. Platelet-rich plasma promotes the proliferation of human muscle derived progenitor cells and maintains their stemness. PloS one 2013;8:e64923.
119. Imai S, Kumagai K, Yamaguchi Y, et al. Platelet-rich plasma promotes migration, proliferation, and the gene expression of scleraxis and vascular endothelial growth factor in paratenon-derived cells in vitro. Sports Health 2019;11:142-148.
120. Xie X, Zhang C, Tuan RS. Biology of platelet-rich plasma and its clinical application in cartilage repair. Arthritis research & therapy 2014;16:204.
121. Fernandes G, Yang S. Application of platelet-rich plasma with stem cells in bone and periodontal tissue engineering. Bone Research 2016;4:1-21.
122. Dou X-Y, An M. Advances in the application of platelet-rich plasma in peripheral nerve injuries. Anesthesiology and Perioperative Science 2025;3:1-9.
123. Nakagawa H, Panero AJ, Qin X, et al. Basic science and preclinical evidence for amniotic membrane– derived allografts in orthobiologics. OrthoBiologics: Elsevier, 2025;111-120.
124. Bedewy N, Elbordiny M, Swelem M, et al. Biochemical assessment of amniotic membrane extract for optimal application in regenerative medicine. Alexandria Journal of Medicine 2024;60:215-221.
125. Maan ZN, Rennert RC, Koob TJ, et al. Cell recruitment by amnion chorion grafts promotes neovascularization. Journal of Surgical Research 2015;193:953-962.
126. Koob TJ, Rennert R, Zabek N, et al. Biological properties of dehydrated human amnion/chorion composite graft: implications for chronic wound healing. International wound journal 2013;10:493-500.
127. Massee M, Chinn K, Lei J, et al. Dehydrated human amnion/chorion membrane regulates stem cell activity in vitro. Journal of Biomedical Materials Research Part B: Applied Biomaterials 2016;104:1495-1503.
128. Moreno SE, Massee M, Koob TJ. Dehydrated human amniotic membrane inhibits myofibroblast contraction through the regulation of the tgfβ-SMAD pathway in vitro. JID Innovations 2021;1:100020.
129. Magatti M, Vertua E, Cargnoni A, et al. The immunomodulatory properties of amniotic cells: the two sides of the coin. Cell transplantation 2018;27:31-44.
130. Wassmer C-H, Berishvili E. Immunomodulatory properties of amniotic membrane derivatives and their potential in regenerative medicine. Current diabetes reports 2020;20:31.
131. Natali S, Farinelli L, Screpis D, et al. Human amniotic suspension allograft improves pain and function in knee osteoarthritis: a prospective not randomized clinical pilot study. Journal of Clinical Medicine 2022;11:3295.
132. McIntyre JA, Jones IA, Danilkovich A, et al. The placenta: applications in orthopaedic sports medicine. The American journal of sports medicine 2018;46:234-247.
133. Hou W-H, Lin P-C, Tung K-M, et al. Effectiveness of amnion allograph injections in patients with degenerative musculoskeletal diseases: a systematic review and meta-analysis. American Journal of Physical Medicine & Rehabilitation 2024:10.1097.
134. Aratikatla A, Maffulli N, Rodriguez HC, et al. Allogenic perinatal tissue for musculoskeletal regenerative medicine applications: a systematic review. Biomedicines 2022;10:3173.
135. Ackley JF, Kolosky M, Gurin D, et al. Cryopreserved amniotic membrane and umbilical cord particulate matrix for partial rotator cuff tears: A case series. Medicine 2019;98:e16569.
136. Bennett DS. Cryopreserved amniotic membrane and umbilical cord particulate for managing pain caused by facet joint syndrome: a case series. Medicine 2019;98:e14745.
137. Castellanos R. Injectable amniotic membrane/umbilical cord particulate for facet joint syndrome: A retrospective, single-center study. Journal of Back and Musculoskeletal Rehabilitation 2022;35:559-564.
138. Miedema M, Anderson A. Outcomes of transforaminal epidural injection of amniotic membrane/umbilical cord particulate for lumbar radiculopathy: a case series. Frontiers in Pain Research 2024;5:1322848.
139. Gomoll AH, Farr J, Cole BJ, et al. Safety and efficacy of an amniotic suspension allograft injection over 12 months in a single-blinded, randomized controlled trial for symptomatic osteoarthritis of the knee. Arthroscopy: The Journal of Arthroscopic & Related Surgery 2021;37:2246-2257.
140. Argut SK, Celik D, Ergin ON, et al. Factors affecting the features of platelet-rich plasma in patients with knee osteoarthritis. Acta Orthopaedica et Traumatologica Turcica 2023;57:148.
141. Rossi L, Ranalletta M, Pasqualini I, et al. Substantial variability in platelet-rich plasma composition is based on patient age and baseline platelet count. Arthroscopy, Sports Medicine, and Rehabilitation 2023;5:e853-e858.
142. Chowdhary K, Sahu A, Iijima H, et al. Aging affects the efficacy of platelet-rich plasma treatment for osteoarthritis. American journal of physical medicine & rehabilitation 2023;102:597-604.

142. Chowdhary K, Sahu A, Iijima H, et al. Aging affects the efficacy of platelet-rich plasma treatment for osteoarthritis. American journal of physical medicine & rehabilitation 2023;102:597-604.
143. Stanbury RM, Graham EM. Systemic corticosteroid therapy—side effects and their management. British Journal of Ophthalmology 1998;82:704-708.
144. Ko GD, Mindra S, Lawson GE, et al. Case series of ultrasound-guided platelet-rich plasma injections for sacroiliac joint dysfunction. Journal of Back and Musculoskeletal Rehabilitation 2017;30:363-370.
145. De Sire A, Lippi L, Mezian K, et al. Ultrasound-guided platelet-rich-plasma injections for reducing sacroiliac joint pain: A paradigmatic case report and literature review. Journal of Back and Musculoskeletal Rehabilitation 2022;35:977-982.
146. Siahaan Y, Tiffani P, Hartoyo V. Long-term pain free effect of platelet-rich plasma injection in a highly frequent case of sacroiliac joint pain. Journal of the Neurological Sciences 2021;429.
147. Aufiero D, Vincent H, Sampson S, et al. Regenerative injection treatment in the spine: review and case series with platelet rich plasma. J Stem Cells Res. Rev & Rep 2015;2:1019.
148. Navani A, Gupta D. Role of intra-articular platelet-rich plasma in sacroiliac joint pain. Techniques in Regional Anesthesia and Pain Management 2015;19:54-59.
149. Flores S, Peres R, Nunes R, et al. CLINICAL OUTCOMES OF PLATELET-RICH PLASMA IN SACROILIAC JOINT DYSFUNCTION: A CASE SERIES. Fizikalna i rehabilitacijska medicina 2025;39:372-372.
150. Wallace P, Wallace LB, Tamura S, et al. Effectiveness of ultrasound-guided platelet-rich plasma injections in relieving sacroiliac joint dysfunction. American journal of physical medicine & rehabilitation 2020;99:689-693.
151. Khosromanesh M, Alikarami S, Espahbodi E, et al. Efficacy of Ultrasound-Guided Platelet-Rich Plasma (PRP) Injection in Patients with Sacroiliac Joint Pain: A Single-Arm, Before and After, Open-Label Clinical Trial. Archives of Neuroscience 2024;12.
152. Singla V, Batra YK, Bharti N, et al. Steroid vs. platelet-rich plasma in ultrasound-guided sacroiliac joint injection for chronic low back pain. Pain Practice 2017;17:782-791.
153. Kumar A, Kumar R, Shankar A, et al. Platelet-rich Plasma Versus Steroid Injection under C-Arm Guidance in the Management of Sacroiliitis. Journal of Orthopaedic Diseases and Traumatology 2024;7:17-20.
154. Ulusoy I, Yilmaz M, Kivrak A. Assessing the outcomes of Prp and corticosteroid injections for sacroiliac joint pain. Medicine Science 2023;12.
155. Ali MNA, Nazim WM, Ahmed MAH, et al. Steroid vs. Platelet-Rich Plasma in Fluoroscopy-Guided Sacroiliac Joint Injection for Chronic Low Back Pain. Egyptian Journal of Medical Research 2025;6:132-141.
156. Dev P, Dey S, Bhattacharyya P, et al. Comparison of the Efficacy of Intra-articular Platelet-rich Plasma with Intra-articular Steroid in the Management of Pain due to Sacroiliac Joint Dysfunction. Indian Journal of Pain 2023;37:173-177.
157. Won SJ, Kim D-y, Kim JM. Effect of platelet-rich plasma injections for chronic nonspecific low back pain: a randomized controlled study. Medicine 2022;101:e28935.
158. Kim WM, Lee HG, Won Jeong C, et al. A randomized controlled trial of intra-articular prolotherapy versus steroid injection for sacroiliac joint pain. The journal of alternative and complementary medicine 2010;16:1285-1290.
159. Ko GD. Platelet-Rich Plasma Prolotherapy for Low Back Pain Caused by Sacroiliac Joint Laxity. human studies 2010;13:15.
160. Saunders J, Cusi M, Hackett L, et al. An exploration of ultrasound-guided therapeutic injection of the dorsal interosseous ligaments of the sacroiliac joint for mechanical dysfunction of the joint. JSM Pain Manag 2016;1:1003-1006.
161. Saunders J, Cusi M, Hackett L, et al. A comparison of ultrasound guided PRP injection and prolotherapy for mechanical dysfunction of the sacroiliac joint. J Prolotherapy 2018;10:e992-999.
162. Ling JF, Wininger AE, Hirase T, et al. Platelet-rich plasma versus corticosteroid injection for lumbar spondylosis and sacroiliac arthropathy: a systematic review of comparative studies. Cureus 2021;13.
163. Burnham T, Sampson J, Speckman RA, et al. The effectiveness of platelet-rich plasma injection for the treatment of suspected sacroiliac joint complex pain; a systematic review. Pain Medicine 2020;21:2518-2528.
164. Zhang X, Zhang A, Guan H, et al. The clinical efficacy of platelet-rich plasma injection therapy versus different control groups for chronic low back pain: A Network Meta-Analysis of Randomized Controlled Trials. Journal of Pain Research 2024:1077-1089.
165. Lai A, Shou J, Traina SA, et al. The Durability and Efficacy of Cryopreserved Human Umbilical Cord Tissue Allograft for the Supplementation of Cartilage Defects Associated with the Sacroiliac Joint: A Case Series. Reports 2023;6:12.
166. McGreevy K, Bottros MM, Raja SN. Preventing chronic pain following acute pain: risk factors, preventive strategies, and their efficacy. European journal of pain supplements 2011;5:365-376.
167. Herren-Gerber R, Weiss S, Arendt-Nielsen L, et al. Modulation of central hypersensitivity by nociceptive input in chronic pain after whiplash injury. Pain Medicine 2004;5:366-376.
168. Radresa O, Chauny J-M, Lavigne G, et al. Current views on acute to chronic pain transition in post-traumatic patients: risk factors and potential for pre-emptive treatments. Journal of Trauma and Acute Care Surgery 2014;76:1142-1150.
169. Latremoliere A, Woolf CJ. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. The journal of pain 2009;10:895-926.
170. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. pain 2011;152:S2-S15.

170. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. pain 2011;152:S2-S15.
171. Van Oosterwijck J, Nijs J, Meeus M, et al. Evidence for central sensitization in chronic whiplash: a systematic literature review. European Journal of Pain 2013;17:299-312.
172. Coppieters I, Ickmans K, Cagnie B, et al. Cognitive performance is related to central sensitization and health-related quality of life in patients with chronic whiplash-associated disorders and fibromyalgia. Pain physician 2015;18:E389-E401.
173. Kosek E, Januszewska A. Mechanisms of pain referral in patients with whiplash associated disorder. European journal of pain 2008;12:650-660.
174. Banic B, Petersen-Felix S, Andersen OK, et al. Evidence for spinal cord hypersensitivity in chronic pain after whiplash injury and in fibromyalgia. Pain 2004;107:7-15.
175. Kosek E, Ordeberg G. Lack of pressure pain modulation by heterotopic noxious conditioning stimulation in patients with painful osteoarthritis before, but not following, surgical pain relief. Pain 2000;88:69-78.
176. Kosek E, Ordeberg G. Abnormalities of somatosensory perception in patients with painful osteoarthritis normalize following successful treatment. European journal of pain 2000;4:229-238.
177. Aranda-Villalobos P, Fernández-de-las-Peñas C, Navarro-Espigares JL, et al. Normalization of widespread pressure pain hypersensitivity after total hip replacement in patients with hip osteoarthritis is associated with clinical and functional improvements. Arthritis & Rheumatism 2013;65:1262-1270.
178. Graven-Nielsen T, Wodehouse T, Langford R, et al. Normalization of widespread hyperesthesia and facilitated spatial summation of deep-tissue pain in knee osteoarthritis patients after knee replacement. Arthritis & Rheumatism 2012;64:2907-2916.
179. Nystrom NA, Freeman MD. Central sensitization is modulated following trigger point anesthetization in patients with chronic pain from whiplash trauma. A double-blind, placebo-controlled, crossover study. Pain Medicine 2018;19:124-129.
180. Walter BA, Purmessur D, Likhitpanichkul M, et al. Inflammatory kinetics and efficacy of anti-inflammatory treatments on human nucleus pulposus cells. Spine 2015;40:955-963.
181. Kleimeyer JP, Koltsov JC, Smuck MW, et al. Cervical epidural steroid injections: incidence and determinants of subsequent surgery. The Spine Journal 2020;20:1729-1736.
182. Dickson RR, Reid JM, Nicholson WT, et al. Corticosteroid and cortisol serum levels following intra-articular triamcinolone acetonide lumbar facet joint injections. Pain Practice 2018;18:864-870.
183. Sterner RC, Sterner RM. Immune response following traumatic spinal cord injury: Pathophysiology and therapies. Frontiers in immunology 2023;13:1084101.
184. Anarte-Lazo E, Liew BXW, Devecchi V, et al. Network analyses reveal the interaction between physical features, fear of movement and neck pain and disability in people with acute and chronic whiplash-associated disorders. European Journal of Pain 2023;28:322-334.
185. Ravn SL, Karstoft KI, Sterling M, et al. Trajectories of posttraumatic stress symptoms after whiplash: A prospective cohort study. European Journal of Pain 2019;23:515-525.
186. Buitenhuis J, de Jong PJ, Jaspers JP, et al. Relationship between posttraumatic stress disorder symptoms and the course of whiplash complaints. Journal of psychosomatic research 2006;61:681-689.
187. Campbell L, Smith A, McGregor L, et al. Psychological factors and the development of chronic whiplash–associated disorder (s): a systematic review. The Clinical journal of pain 2018;34:755-768.
188. Carroll LJ, Holm LW, Hogg-Johnson S, et al. Course and prognostic factors for neck pain in whiplash-associated disorders (WAD): results of the Bone and Joint Decade 2000–2010 Task Force on Neck Pain and Its Associated Disorders. Spine 2008;33:S83-S92.
189. Pedrero-Martin Y, Falla D, Rodriguez-Brazzarola P, et al. Prognostic factors of perceived disability and perceived recovery after whiplash: a longitudinal, prospective study with one-year follow-up. The Clinical Journal of Pain 2024;40:165-173.
190. Szucs G. Emotionally concussed: The impact of pre-existing anxiety on concussion recovery. 2023.
191. Elliott J, Walton D, Albin S, et al. Biopsychosocial sequelae and recovery trajectories from whiplash injury following a motor vehicle collision. The Spine Journal 2023;23:1028-1036.
192. Modarresi S, MacDermid JC, Suh N, et al. How is the probability of reporting various levels of pain 12 months after noncatastrophic injuries associated with the level of peritraumatic distress? Clinical Orthopaedics and Related Research® 2022;480:226-234.
193. Wallis BJ, Lord SM, Bogduk N. Resolution of psychological distress of whiplash patients following treatment by radiofrequency neurotomy: a randomised, double-blind, placebo-controlled trial. Pain 1997;73:15-22.
194. Smith AD, Jull G, Schneider GM, et al. Modulation of cervical facet joint nociception and pain attenuates physical and psychological features of chronic whiplash: a prospective study. PM&R 2015;7:913-921.
195. Stenneberg MS, Scholten-Peeters GG, den Uil CS, et al. Clinical characteristics differ between patients with non-traumatic neck pain, patients with whiplash-associated disorders, and pain-free individuals. Physiotherapy theory and practice 2022;38:2592-2602.

196. Stenneberg MS, Rood M, de Bie R, et al. To what degree does active cervical range of motion differ between patients with neck pain, patients with whiplash, and those without neck pain? A systematic review and meta-analysis. Archives of physical medicine and rehabilitation 2017;98:1407-1434.
197. Anstey R, Kongsted A, Kamper S, et al. Are people with whiplash-associated neck pain different from people with nonspecific neck pain? journal of orthopaedic & sports physical therapy 2016;46:894-901.
198. Vassiliou T, Kaluza G, Putzke C, et al. Physical therapy and active exercises–an adequate treatment for prevention of late whiplash syndrome?: Randomized controlled trial in 200 patients. Pain 2006;124:69-76.
199. Scholten-Peeters GG, Bekkering GE, Verhagen AP, et al. Clinical practice guideline for the physiotherapy of patients with whiplash-associated disorders. Spine 2002;27:412-422.
200. Blanpied PR, Gross AR, Elliott JM, et al. Neck pain: revision 2017: clinical practice guidelines linked to the international classification of functioning, disability and health from the orthopaedic section of the American Physical Therapy Association. Journal of Orthopaedic & Sports Physical Therapy 2017;47:A1-A83.
201. Jull GA, Söderlund A, Stemper BD, et al. Toward Optimal Early Management After Whiplash Injury to Lessen the Rate of Transition to Chronicity. Spine 2011;36:S335-S342.
202. Park D. The Pathophysiology and Mechanism of Acute and Chronic Whiplash Injury: A Narrative Review. International journal of Pain 2022;13:20-24.
203. Bharadwaj UU, Lynch JA, Joseph GB, et al. Intra-articular Knee Injections and Progression of Knee Osteoarthritis: Data from the Osteoarthritis Initiative. Radiology 2025;315:e233081.
204. Ayub S, Kaur J, Hui M, et al. Efficacy and safety of multiple intra-articular corticosteroid injections for osteoarthritis—a systematic review and meta-analysis of randomized controlled trials and observational studies. Rheumatology 2021;60:1629-1639.