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Addressing cervical intervertebral disc pathology and pain in Whiplash Associated Disorders: the role of epidural injections

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Purpose Statement

This paper describes the pathology, pain symptoms, and treatment of the cervical intervertebral disc in the context of whiplash associated disorder (WAD). Specifically, this paper will review the use of epidural injections as it pertains to the management of pain in the context of WAD. The intent of this paper is to:

1. Summarize the pathology of disc injury including the 4 types of herniations and the incidence in WAD
2. Review the prevalence of disc injury in motor vehicle accidents
3. Review the pathology of inflammation on nerves
4. Review the basic science of cortisone injections
5. Review the effects of steroids on neural inflammation
6. Review of evidence for epidural steroid injections
7. Review of evidence of early use of epidural steroid injections to prevent chronic pain

Executive Summary

Discogenic pathology often underlies more complex cases of WAD. Early intervention with epidural injections can reduce pain and neural inflammation, lower the likelihood of surgical intervention, and help prevent central sensitization and psychological complications linked to poor outcomes. Emerging evidence indicates disc injury may be more common in WAD than previously thought, and timely treatment by trained providers can enhance pain control and quality of life.

Key Findings

- Cervical disc pathology may appear on MRI as a bulge or herniation, though inflammation of the annulus fibrosus may not be visible.
- Injury forces in MVAs can produce direction-specific damage and exacerbate pre-existing, asymptomatic disc pathology.
- MRI findings should be interpreted alongside physical examination and clinical suspicion, as discogenic injuries may be missed on imaging alone.
- Post-traumatic inflammation can irritate nearby nerves, leading to discogenic pain and radiating symptoms.
- Persistent pain may sensitize the central nervous system, amplifying pain perception and contributing to chronicity.
- Epidural steroid injections can reduce inflammation, improve function and quality of life, and may help reverse central sensitization and related psychological symptoms.
- Early intervention in appropriately selected patients improves outcomes, reduces chronic pain risk, and may prevent surgical escalation.

Implications

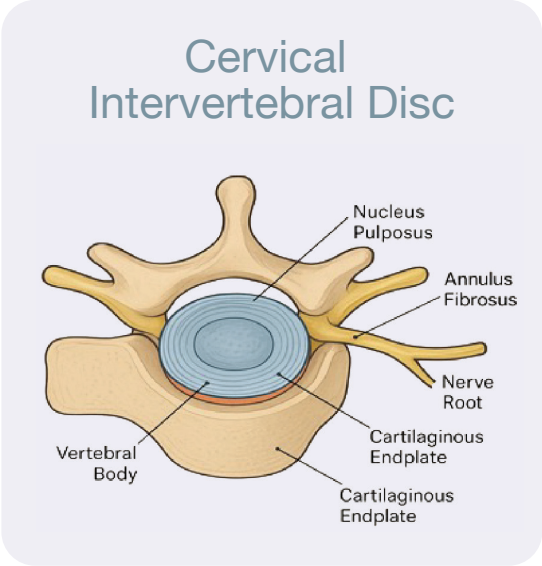
Epidural injections can significantly influence pain reduction and recovery outcomes in WAD by addressing underlying inflammation. Clinicians and legal professionals should recognize signs of discogenic pain and collaborate with experienced specialists to ensure optimal care for this complex patient population.

Introduction

Discogenic pain is a frequent source of spinal discomfort, reported in 16–41% of neck pain cases, most commonly affecting females in their 30s to 50s.[1-4] Disc injuries, including herniations and prolapses, may present variably and require tailored management.[5-7] Whiplash-associated disorder (WAD) is a complex condition involving cervical, cranial, and upper extremity systems, often resulting in pain, restricted motion, and both peripheral and central nervous system dysfunction.[8-11] Disc pathology in this context is commonly treated with corticosteroids delivered via intralaminar or transforaminal epidural injections, or systemically via oral or intravenous routes.[12-17] Given the traumatic nature of WAD, particularly following motor vehicle accidents, providers must be well-versed in its pathology, clinical presentation, and evidence-based interventions to guide optimal care. The following sections address these topics in detail.

Pathology of Disc Injury

The intervertebral disc is part of a functional spinal unit that includes two vertebrae, the disc, facet joints, and associated soft tissues, allowing segmental spinal motion. [18], [19] The disc consists of an inner nucleus pulposus and an outer annulus fibrosus made of concentric lamellae, bordered by cartilaginous endplates attaching to adjacent vertebral bodies. [20-22]



Trauma or repetitive stress can lead to disc degeneration and herniation. [20], [22]

Types of herniation include:

- **Protrusion**: Nuclear material extends outward but annular lamellae remain intact.
- **Bulge**: Generalized circumferential disc extension beyond vertebral margins.
- **Extrusion**: Annular lamellae are stretched or torn beyond normal boundaries.
- **Sequestration**: Herniated material is fully detached from the disc body.

When the annulus is breached, either through macro or micro-disruption, the nucleus becomes exposed to the immune system, triggering inflammation. [23], [24] This response degrades collagen and releases extracellular matrix fragments and crystals, perpetuating inflammation and potentially leading to degenerative disc disease. [23],[24]

Vascular ingrowth into damaged discs also brings nociceptive nerve fibers, sensitizing the region to inflammation. [20], [25], [26] Cervical spine motion can then provoke pain via mechanical stress on the annulus. [27]

Secondary inflammation can affect adjacent nerve roots, causing radicular symptoms through cytokine-mediated mechanisms, including intraneural swelling, ischemia, and chemical irritation. [28-30] These mechanisms often interact, compounding the pain experience. [28], [29]

Additionally, nerve roots may be compressed by herniated disc material, annular bulging, or osteophytes at the neural foramen or spinal cord exit. [31] Such compression can stretch dorsal/ventral rootlets and provoke pain on spinal or limb movement. [21], [28], [29],[31]

Upper extremity motion may traction the brachial plexus, worsening pain or causing abnormal sensations across its innervation zones, including the neck, head, shoulder, arms, and hands. [22], [31-33] These mechanisms underlie conditions such as discogenic pain, cervicogenic headache, radiculopathy, radiculitis, and brachial plexopathy.

Disc Injuries in Motor Vehicle Accidents

The cervical spine is highly vulnerable during motor vehicle accidents (MVAs), and intervertebral discs are commonly affected. Rear-end collisions produce an “S”-shaped curve in the cervical spine, involving anterior distraction and posterior compression. This motion causes annular tears, nucleus damage, and vertebral endplate avulsions, most often at C5 - T1. [34],[35],[36],[37],[38],[39],[40]

Frontal impacts cause abrupt cervical flexion and protraction, commonly injuring C5/6 and C2/3 discs. [41], [42] Lateral impacts introduce complex rotational forces and frequently affect C6/7, with strain observed from C4, T1. [36],[43],[44] For more on injury mechanics, see this organization's whitepaper, Whiplash: An In-depth Review of Pathology and Biomechanics of Injury.

Low-speed impacts (<10 mph delta-V) are generally insufficient to cause frank disc herniation. [45],[46] However, Eckardt et al. documented symptomatic herniations with radiologic trauma signs even in low-impact MVAs.[47] Brinjikji's population study showed widespread asymptomatic disc pathology,[48] suggesting that pre-existing degeneration reduces the force threshold for symptomatic injury. For details, see the whitepaper The Impact of Pre-Existing Conditions on the Risk and Severity of Whiplash-Associated Disorders.

Although asymptomatic disc pathology is common, posterior disc protrusion is more frequent in WAD patients.[49],[50] As Taylor and Twomey noted, standard MRI may detect traumatic herniations but often misses annular avulsions and facet joint lesions.[51] Disc herniation typically co-occurs with other injuries, endplate fractures, joint lesions, and worsens with flexion or greater impact severity.[52],[53],[54] Among 531 patients reviewed, 270 had symptomatic single-or two-level disc herniations.[55] Pre-injured but previously stable discs may also become symptomatic post-MVA due to impaired load tolerance.[56],[57],[58],[59]

Diagnostic challenges arise from reliance on supine MRI. Upright MRI has demonstrated greater disc bulging and foraminal narrowing in symptomatic individuals. [60] Tawa's review found supine MRI only 25% sensitive for neural encroachment. [61] Jinkins concluded recumbent MRI underestimates dynamic pathology. [62] Upright imaging has revealed cervical instability and cord compression missed in supine views. [63] Botchu emphasized that conventional MRI does not replicate physiological upright forces. [64]

Omar et al. (2025) highlighted the limitations of conventional imaging, noting a disconnect between structural findings and functional changes. Their study using diffusion tensor imaging (DTI) found significant spinal tract abnormalities in chronic neck pain patients despite normal MRIs. These microstructural changes correlated with pain and psychological distress. [65]

Gornet (2019) showed magnetic resonance spectroscopy (MRS) may improve identification of symptomatic discs, offering up to 93% accuracy in non-herniated discs and a 97% success rate in guiding treatment. [66]

Inflammation and Neuropathology

Following trauma such as a motor vehicle accident (MVA), the body initiates an inflammatory repair response involving chemical messengers associated with pain and long-term sensitization.[23],[67] Persistent pain promotes the expression of pro-inflammatory cytokines, which also contribute to disc degeneration.[30],[68] These mediators enhance peripheral nerve signaling, leading to peripheral sensitization and amplified pain.[69] As Lutke Schipholt et al. state: "There is overwhelming evidence from preclinical studies that neuroimmune responses are central to the initiation, progression and resolution of persistent pain.[70]

These chemical messengers diffuse across the cervical spine, affecting structures including nerve roots, the sinuvertebral nerve, and rami communicans. [71], [72], [73],[74] This can cause local neck pain, radicular pain, and multilevel symptoms due to overlapping innervation. Even in the absence of disc herniation, damaged disc tissue can trigger inflammation and neural irritation. [23], [24] Radicular pain is usually detectable on exam if the nerve root is involved,[73] but pain from sinuvertebral nerve irritation may be more elusive. This nerve innervates the intervertebral disc, posterior longitudinal ligament, vertebral periosteum, and dura mater, and connects across multiple levels. [72],[75]

Importantly, the sinuvertebral nerve carries autonomic fibers responsive to stress, which can intensify the pain experience. [72], [76], [77], [78] This inflammation may produce mechanical discogenic pain even without imaging evidence of herniation. Additionally, the inflammatory healing response may induce nerve ingrowth into the annulus fibrosus, contributing to pain despite unremarkable imaging. [23], [65], [79], [80],[81]

Chronic inflammation can result in widespread pain through neural injury, microvascular dysfunction, increased metabolic demands, breakdown of the blood-nerve barrier, and hypersensitivity in joints, nerves, and soft tissue. [82], [83], [84], [85],[86],[87],[88] For further detail, refer to this organization's paper: Whiplash: An In-depth Review of Pathology and the Biomechanics of Injury.

Another mechanism involves immune priming, where inflamed tissues become hypersensitive to future minor trauma.[89],[90] This increases susceptibility to pain flare-ups and mechanical dysfunction long after the initial injury.[26],[91],[92] This has been demonstrated in tendon and muscle injuries, and in the proliferation of fibroblasts and growth factors near damaged discs.[26],[30],[93],[94],[95],[96],[97]

Basic Science and Mechanism of Corticosteroid Treatments

Corticosteroids used in WAD treatment are synthetic analogs of natural hormones with immunosuppressive, anti-inflammatory, and vasoconstrictive properties. [98] They act by binding to intracellular glucocorticoid receptors, altering gene transcription and suppressing the production of inflammatory mediators. [98] Delivery can be systemic or localized, such as via cervical epidural injection. Steroids used in injection form are classified as particulate (longer-lasting, suspended solids) or non-particulate (faster-acting, fully dissolved solutions). [99], [100]

In healthy tissues, corticosteroids mildly suppress baseline inflammatory cytokines (e.g., IL-1β, IL-6, TNF-α) and reduce matrix metalloproteinases (MMPs) involved in cartilage breakdown. [101], [102] In pathological states, their effects are more pronounced, including:

- Marked suppression of cytokines (IL-1β, IL-6, TNF-α), and inhibition of macrophage and T-cell activity[98], [103]
- Reduction of MMPs (e.g., MMP-1, MMP-3, MMP-13) and increased TIMPs, decreasing cartilage degradation[104]
- Inhibition of synovial hyperplasia, VEGF production, and vascular permeability[105]
- Modulation of nociceptive ion channels, reducing inflammatory sensitization of nerve pathways[106], [107]
- Prevention of pathological neovascularization through VEGF pathway inhibition[108]

These mechanisms make corticosteroids a valuable therapeutic option for patients with WAD, offering both symptom relief and functional improvement.

Evidence for Epidural Injections

Epidural steroid injections (ESIs) are supported by substantial evidence for managing cervical discogenic pain in WAD patients. Pampati et al. (2013) conducted a randomized controlled trial (RCT) showing 72% of patients with disc herniation experienced ≥50% pain relief and functional improvement at two years with epidural steroid injections. [109] A 2014 follow-up study in patients without herniation demonstrated similar outcomes with ESIs, with 75% and 78% achieving significant functional and pain improvements.[110]

Lin (2006) reported that cervical ESIs provided pain relief sufficient to avoid surgery in most patients with herniated discs, especially when administered within 100 days of diagnosis and in patients over 50.[111] Similarly, Kwon et al. (2007) found better outcomes in patients with disc herniation compared to stenosis and in those treated within six months of symptom onset.[112] Choi (2016) confirmed that patients with discogenic pain responded better to ESIs than those with cervical stenosis, with acute pain showing the most robust response. [113]

A 2025 systematic review concluded that cervical ESIs offer benefits in short- and long-term disability and short-term pain reduction. [114] Manchikanti (2015) provided Level II evidence supporting cervical interlaminar ESIs for long-term relief of neck and upper extremity pain.[115] Another study noted that improvements in neck pain and disability scores following ESI were independent of initial symptom severity and MRI findings.[17]

A 2006 trial involving surgical candidates with cervical disc herniation or protrusion found that 94% avoided surgery after ESI, 82% would choose the treatment again, and most experienced significant reductions in pain, disability, and analgesic use. [116]

The journal Medicine (2016) reported significant pain and function improvements at 2 and 8 weeks with both interlaminar and transforaminal ESI approaches in patients with cervical disc herniation. [117] McCormick (2020) found interlaminar injections more effective than transforaminal, although both provided benefits. [118]

Kesikburun et al. found higher success rates among patients with initially higher pain scores, indicating those with more severe symptoms may derive the greatest benefit from ESIs. [119]

Early Intervention to Prevent Chronic Pain

In patients with acute pain following a motor vehicle accident (e.g., WAD), early intervention is essential to prevent progression to chronic pain. Secondary prevention, defined as the early recognition and treatment of acute or subacute pain, aims to halt this transition. [120] A central goal is to prevent central sensitization, even if peripheral sensitization has already occurred. [120], [121], [122]

Central sensitization involves altered neuronal processing of nociceptive input, resulting in amplified pain signaling from the periphery. [123] It underlies many features of chronic pain syndromes and is frequently seen in patients with chronic WAD, contributing to significant quality of life impairments. [124], [125], [126], [127], [128]

Fortunately, this process can be modulated or reversed if nociceptive signals from peripheral pain generators, such as nerves, discs, facets, trigger points, and fascia, are normalized.[129],[130],[131],[132],[133] Evidence suggests early anti-inflammatory treatment, including epidural injections, is most effective when delivered early in the inflammatory cascade and may reduce the need for surgical intervention.[134],[135]

The psychological dimension of pain is another critical factor influencing prognosis. Conditions such as kinesiophobia, low recovery expectations, anxiety, depression, and PTSD are associated with poorer outcomes after MVAs.[136],[137],[138],[139],[140],[141],[142],[143] Early psychological trauma, particularly within the first 3 weeks, can predict higher pain levels at 12 months.[144] Fortunately, effective pain control can also positively impact psychological well-being, making comprehensive pain management crucial.[145],[146]

Clinicians should prioritize early identification and treatment of at-risk patients to prevent chronicity and optimize outcomes in WAD.

Attorney Guidance of Client Healthcare

For attorneys navigating the medicolegal process on behalf of clients recovering from MVAs, the volume of clinical information can be overwhelming. Early referral to an interventional team specializing in WAD, particularly those experienced in diagnosing and managing discogenic pain, can significantly reduce a client's pain and long-term disability. Timely involvement of the right medical team may be the key to preventing a lifelong condition.

This whitepaper provides a synthesized review of the current evidence and offers clear guidance on appropriate medical management for patients suffering from WAD with discogenic involvement.

Conclusion

Following a motor vehicle accident (MVA), discogenic pathology is often present in more complex cases at risk for chronic pain and may be more common than previously recognized. Persistent inflammation can drive discogenic and neural pain symptoms associated with WAD. Epidural injections play a key role in reducing inflammation and supporting recovery as part of a multimodal treatment approach. Awareness of these mechanisms enables better aftercare planning and patient education.

Next Steps

For attorneys wanting the best for their clients, the healthcare team associated with client care needs to have a thorough understanding of the elements of this white paper. Epidural injections can have a strong effect on discogenic and neural pain conditions for clients suffering from WAD. Referral to providers with this knowledge can help to prevent transition to chronic pain through appropriate management of these conditions.

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