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Authors
Moheimani, Roya
Arastoo, Sara
Moheimani, A. Michael

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Narrative Review: Disc Herniation After Motor Vehicular Trauma

Roya S. Moheimani M.D.[1], Sara Arastoo B.S. [1], A. Michael Moheimani M.D. [2]
David Geffen School of Medicine at UCLA[1], Coast Spine & Sports Medicine[2]

Abstract:

Spinal pain that arises from motor vehicular trauma is challenging to the treating physician due to the wide spectrum of presenting symptoms and responses to treatment, including traumatic disc herniations. The severity of injuries varies from patient to patient, with imaging often not strictly correlating to symptomatology. However, with a systematic approach, including an understanding of the limitations of magnetic resonance imaging (MRI) and the role of cytokines and inflammatory mediators, the treatment and diagnosis from patients suffering from traumatic disc herniations can be improved[1,2]. Furthermore, evidence supports conservative management before escalation to more invasive procedures such as epidurals or surgery, specifically in uncomplicated spinal injury patients with no evidence of neural compromise warranting emergency surgery[3,4]. Should surgery be needed, mounting evidence supports intervention with minimally invasive discectomy for lumbar herniations and cervical disc arthroplasty for cervical herniations[5–8].

Introduction:

It is well-known that trauma, whether it be from a motor vehicle accident (MVA), a slip and fall incident, a sport injury, or a work injury, can result in pathologies associated with the onset of neck and back pain, requiring treatment and or intervention. Specifically, injury from MVAs is the second most common form of traumatic injury in older individuals (>65), with an estimated incidence of 250,000 emergency department (ED) visits per year.[9] Most of these individuals are discharged from the ED with moderate to severe pain, with an estimated 21% suffering from cervical pain and 13% suffering from lumbar pain, both contributing to the most common discharge diagnosesis.[10] In those suffering from whiplash or undifferentiated low back pain, the severity of the injury and the need for subsequent treatment varies greatly with individuals and with mechanism and extent of the injury.[11–13]

Disc herniation is often suspected when there is clinical findings that suggest a discogenic origins of pain, along with MRI findings that support the diagnosis.[14] Findings on history that suggest discogenic pain include a combination of midline pain, pain worse with sitting, and most importantly pain provocation when rising from sitting to standing.[13,15] Sacroilliac pain is more commonly associated with unilateral pain. Facet joint pain is less likely to be midline and worse with hyperextension, instead of flexion as seen with discogenic pain. Radicular pain due to nerve root compression or irritation is most likely described as paresthesias following dermatomal distributions.[16,17] MVAs are the leading causes of thoracic and lumbar injuries, which are

amplified in obese patients, putting this population at further risk for disc herniation.[18,19] Scientific data shows that the impact does not have to be significant to cause a spinal injury, especially with the increased use of seatbelts, increasing the risk of soft tissue injury and minor fractures while avoiding major spinal fractures.[12] Bannister et al. found that 90% of whiplash injuries occur with speeds less than 14 mph. The literature suggests that crash tests at as low as 2.5 miles per hour are sufficient to cause symptoms.[20,21] Conversely, it is noted that a speed of 8.7 mph is required to cause significant damage to the vehicle. This data shows that an axial load is transmitted from the seat onto the pelvis and spine, which can cause burst and compression fractures. One could infer that forces great enough to cause spinal fractures can potentially cause disc herniations.

An extreme example is demonstrated by reports of a 31-year-old woman, with prior history of prior disc pathology at L5-S1, presenting shortly after a motor vehicle accident to the emergency department with bowel and bladder incontinence, requiring emergency surgery.[22] MRI following the vehicular accident demonstrated that the lumbar disc herniation had extruded with significant compression of the neural elements, leading to acute loss of bowel and bladder function. In this scenario where preexisting MRI allows for comparison, little is left to dispute regarding the causation of the massive disc herniation following the vehicular accident and the need for subsequent spinal treatment.

Regardless of the etiology of the pain, MVAs often leave patients with persistent pain and functional deficits that is difficult to diagnose, manage and treat.[23] Effects of the injuries often persist as long as 6 months after the accident. And with medical legal factors that are often at play in MVAs, management is even more complicated due to conflict of interest.[24,25] We seek to provide a narrative review to this complex yet common problem of traumatic disc injuries in the absence of spinal cord damage, spinal fractures, or spinal instability, defined as hypermobility between two vertebral segments.

Clinical Presentation

Patients who present after MVAs may present with a variety of symptoms, not limited to neck and back pain. Shoulder pain should not quickly be attributed to cervical pathology, as approximately 5% to 9% of post MVAs patients go on to develop subacromial impingement syndrome.[26] Of those with persistent pain who undergo subacromial decompression after a whiplash injury, 50% described benefit. Motor vehicular trauma may manifest in a multitude of pathologies beyond conventional neck and back pain, that may contribute to the patient's overall perception of their neck or back pain, including depression and anxiety.[27] Alarming findings, including neurological compromise to the upper and/or lower extremities,

bowel/bladder incontinence, would warrant prompt and immediate surgical attention.[4,28,29]

Diagnosis and Assessment

In patients recovering from MVAs, after the physical exam and plain films (X-rays), Magnetic Resonance Imaging (MRI) is often the main stay of diagnostic work up for moderate to severe cervical or lumbar pain.[30] However, it is well-known that individuals may exhibit radiological evidence of disc herniations but maybe asymptomatic.[31] Estimates suggest that of the adult population, who do not have disabling lower back pain, the incidence of disc herniation and/or disc degeneration is between 28% to 85%, a staggering wide range.[32] The percentage of abnormality naturally increases with age. This puts into question the utility of imaging; however, patients with symptoms have more severe or significant herniations.[31] Additionally, neural compromise was present, in a study by Boos et al., in only 4% of asymptomatic subjects compared to 54% of symptomatic subjects.[31] Therefore, although MRI may be abnormal in asymptomatic individuals, it is much more likely to be highly abnormal in individuals having neurologic symptoms. Neural compromise is much more commonly seen in individuals requiring interventional treatment such as epidural injections or surgery.

In patients with no evidence of spinal fracture or instability, disc herniation is often attributed as the causative agent to pain and disability.[30] It is well-known that discs herniate with axial compression and or torsion.[33] This is the same mechanism that occurs in spinal fractures following MVAs. Evidence suggests that with the increased use of seatbelts and new airbag technologies, there is a decrease in severe spinal fractures, but an actual overall increase in the total numbers of spinal fractures and soft tissue injury.[20]

Interestingly disc herniations do not necessarily precede disc degeneration.[34] Histiologic examination of herniated disc tissue versus non-herniated disc tissue suggests that there is no evidence that the disc must be degenerative before herniation. In addition, it is noted that not all individuals react the same way following a disc injury.[23] An individual with a 2 to 3 mm disc herniation may complain of severe back and radicular pain and yet someone with a 6 mm disc herniation may not have significant pain.[31] Similarly, the presence of an annular tear of the disc on MRI is not predictive necessarily of clinical outcomes.[35] Despite this, MRI remains the gold standard for diagnosing disc herniation, with a sensitivity and specificity of greater than 90%, making it essential for planning more invasive treatment options.[1] However, the most reliable finding on MRI remains the presence of neural canal and foraminal compromise.[36] The percentage of degree of compromise is most important, as larger canals can tolerate larger sized herniations.

Pathophysiology:

Besides the traditional understanding of traumatic disc herniation pain arising from mechanical irritation of the spinal nerve roots, increasing evidence suggest that cytokines and immuno-regulatory cascades also play a role in the etiology of neck and back pain. Specifically, patients with symptomatic disc herniations are noted to have increased levels of nitric oxide, prostaglandin E2, and interleukin-6.[37,38] Therefore, there are chemical mediators of pain, which are not detected by imaging studies. These cytokines have been implicated in causing radiculopathy, thus perhaps mechanically compressive disc protrusions are not the only causes of radicular pain.[2] For this reason, chemical markers from the disc have been implicated in the inflammatory response. They point to higher concentrations of prostaglandin E2 being found in individuals with positive straight leg raise tests. They also support matrix metalloproteinases, nitric oxide, IL-6, and prostaglandin E2 elevations in lumbar herniations.[37,38,2]

These inflammatory factors which irritate nerves and cause pain, could be released post trauma. Interestingly, in vitro and in vivo studies using rats modeling human degenerative discs reveal that the use of anti-oxidants suppresses nitric oxide release, preventing further disc degeneration.[39] This coincides with increasing amount of evidence that oxidative damage induces apoptosis and calcification of cartilage in the intervertebral disc.[40] When tissue injury occurs, as it does with trauma, polymorphonucleated leukocytes (PMNLs) migrate and accumulate at the site of tissue injury, which results in the release of reactive oxygen species, causing an inflammatory burden[41]. This explains why epidural injections, which are anti-inflammatory, can provide pain relief.[42] Epidurals do not reduce the size of disc herniation but can result in resolution of pain.

Treatment:

To our knowledge, there is a limited amount of literature focusing on the recovery of traumatic lumbar herniations that do not involve spinal fractures, spinal instability, or spinal cord compromise. What is known includes the first step in recovery from most spinal injuries, with no alarming neurological findings, is rehabilitation by initiating physical therapy and or chiropractic care with conservative medical management in the first 6 weeks of injury, as most acute pain resolves within the first 6 weeks.[43,44] Unfortunately, in the older aged population (>65), spinal pain can persist beyond the 6 month period causing disability and functional decline.[23]

For conservative management, evidence supports recommending manipulative therapy (chiropractic) and physical therapy versus recommending a soft collar and administering basic initial medical advice, including Nonsteroidal anti-inflammatory drugs (Nsaids), in patients suffering

from neck whiplash.[45] The same trend is observed in patients with acute low back pain.[45,46] If there is still pain beyond this 6 week period with conservative therapies, pain management options, such as epidurals and facet blocks should be considered, but there is limited studies on these modalities specifically targeting persistent facet or discogenic pain stemming from MVAs. Risk factors for persistent pain beyond the conventional time window include psychological factors and evidence of neurologic compromise on exam.[47]

Should all non-operative measures fail, then considering surgical options to alleviate pain and disability is warranted. In cervical herniations, evidence supports either cervical arthroplasty or anterior cervical discectomy and fusion for trauma patients with traumatic cervical herniation with no evidence of spinal cord injury, spinal fracture or spinal instability.[7,8] However, from a cost analysis perspective, for single level disease, cervical disc arthroplasty is superior.[3]

For lumbar herniations, newer surgical options, which are less invasive and allow outpatient surgical management including endoscopic discectomy, allowing more rapid recovery and restoration of pre-injury function, giving patients greater hope and satisfaction.[48,5,6,49] Reviews of various randomized control trials reveal that minimally invasive discectomies hold promise in both minimizing the time for recovery with similar or better outcomes compared to conventional open discectomies.

Discussion/Conclusion:

In summary, the severity of neck and back pain, along with disability, following vehicular accidents is often difficult for the clinician to quantify. However, evidence suggests there is a much higher level of MRI abnormalities seen in symptomatic individuals than asymptomatic individuals.[1] MVAs can cause severe injuries, including fractures, dislocations, and paralysis. However, it is also known that even impacts at much lower speeds can cause onset of neck and lower back pain that stem from traumatic disc herniations.[18] The extent of vehicular damage does not always explain the severity of spinal pain.

As evidence by our review there is a growing understanding of the multi-factorial contributors of spinal pain, as cytokines and other inflammatory makers that are released from disc injuries.[2] The understanding of the chemical mediators of pain has led to the development of new technologies, advancing from epidural steroid injections to platelet rich plasma injections and stem cell therapy and warrants more clinical research into these topics.[50–53] For those with traumatic cervical and lumbar herniations, refractory to non-operative interventions, promise may lie in cervical disc arthroplasty and minimally invasive lumbar discectomies.

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