

Five Reasons Why Some Herniated Intervertebral Discs are Painless

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Abstract: Herniated intervertebral discs (HIVDs) can cause lower back pain and leg pain, but not all cases are painful. We suggest five potential reasons for painless HIVDs. First, when inflammation resolves or is insufficient, pain may not occur. Second, rare cases exhibit motor or sensory deficits without pain, suggesting a mechanical rather than inflammatory component. Third, changes in gene expression within the dorsal root ganglia may prevent persistent inflammation and pain. Fourth, reorganization of neural circuits in the spinal dorsal horn can reduce nociceptive signals and lead to painlessness. Fifth, effective pain coping strategies and higher pain tolerance may mask any discomfort. Understanding these factors can improve clinicians' knowledge and aid in pain management.

Keywords: herniated intervertebral disc, painless, inflammation, reorganization

Introduction

A herniated intervertebral disc (HIVD) refers to the rupture of the annulus fibrosus of the intervertebral disc, leading to the herniation of the nucleus pulposus beyond the intervertebral disc space.¹ It is caused by a sudden back injury or by gradual wear and tear of the disc (ie disc degeneration).¹ HIVDs induce lower back and lumbosacral radicular pain in the lower extremities and are a common cause of musculoskeletal pain.¹ The prevalence of symptomatic HIVDs in the general population is known to be 1–3%.¹ A diagnosis of an HIVD is confirmed using magnetic resonance imaging (MRI) and computed tomography (CT). However, despite clear findings of an HIVD on MRI or CT, many HIVDs do not cause pain.² Jensen et al reported that only 36% of 98 asymptomatic patients had normal discs at all levels on MRI, with 52% showing a bulging disc at least at one level and 27% with a disc herniation. Here, we propose five reasons why some HIVDs do not induce pain.²

Five Reasons Why Some HIVDs Do Not Induce Pain

First, patients may not experience pain when inflammation around an HIVD has resolved following an acute phase, or when there is insufficient inflammation to cause pain. Chemical inflammation around the sinuvertebral nerve and nerve root is the main mechanism of pain in an HIVD, together with direct mechanical compression of these nociceptive nerves.³ An HIVD can be painless if compression and inflammation are insufficient to cause pain.

Second, although rare, motor weakness or sensory deficits without pain may develop following HIVDs.⁴ Painless weakness or sensory deficits following HIVDs suggest less of a transient, inflammatory component and more of an established mechanical component.⁴ Compression of nerve tissue in the absence of inflammation results in painless loss of motor or sensory function.

Third, the secondary change in gene expression within the dorsal root ganglia (DRG) after HIVDs might be attributed to painless HIVDs. Gene expression within the DRG dynamically changes after an HIVD has developed, which alters the level of protein production of certain factors related to the inflammation process and nociception (ie pro-inflammatory, pro-apoptotic, and pro-nociceptive factors).⁵ Acute inflammation plays a crucial role in clearing cellular debris and

creating a favourable environment for nerve repair. However, if acute inflammation progresses to chronic inflammation, it can lead to cell death and neuropathic pain, ultimately impeding the recovery process.⁵ If gene expression within the DRG changes in a way that prevents persistent inflammation through natural adaptation processes or treatments, there may be no pain even in the presence of an HIVD.

Fourth, reorganisation of neural circuits within laminae I–III of the spinal dorsal horn could be the cause of a painless HIVD. Inhibitory and excitatory interneurons play an important role in the pain signalling pathway through modulating the activity of wide dynamic range neurons and nociceptive-specific neurons in the spinal dorsal horn.⁶ Laminae I–III of the spinal dorsal horn are populated with a plexus of excitatory and inhibitory interneurons.⁶ In situations without pain, a balance between excitatory and inhibitory interneurons is well maintained. However, if neuroinflammation occurs due to an HIVD, neural circuits within I–III of the spinal dorsal horn are reorganised. During reorganisation, when inhibitory synapses are greatly increased compared with excitatory synapses, the secretion of neurotransmitters involved in the transfer of nociceptive signals is significantly reduced. In such situations, patients may not experience pain, even in the presence of an HIVD.

Lastly, an HIVD can be painless in patients with an effective pain coping strategy. Pain perception is a complex sensory and emotional experience that involves both physiological and psychological factors.⁷ Coping strategies can influence pain perception and the level of pain experienced by patients.⁷ Additionally, people with effective coping strategies may have a higher tolerance for pain, and their bodies may release pain-relieving chemicals that mask any discomfort from pain.

Conclusion

We have proposed five reasons why some HIVDs are painless. Our discussion on this topic can enhance clinicians' understanding of HIVDs, which could also be applied in the management of pain related to HIVDs. Future research is needed to further validate these five proposed reasons.

Abbreviations

CT, computed tomography; DRG, dorsal root ganglia; HIVD, herniated intervertebral disc; MRI, magnetic resonance imaging.

Author Contributions

Both authors (DAS & MCC) made a significant contribution to the work reported, in the conception, study design, execution, acquisition of data, analysis and interpretation; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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