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A REVIEW OF THE SINUVERTEBRAL NERVE IN DISCOGENIC PAIN: ADVANCES IN DIAGNOSIS AND MANAGEMENT

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ABSTRACT

Chronic low back pain (CLBP) is a leading cause of disability globally, significantly affecting patients' quality of life, and posing a substantial socioeconomic burden. As a major contributor to CLBP, discogenic low back pain (DLBP) is caused by degenerative changes in the intervertebral discs. This review explores the role of the sinuvertebral nerve (SVN) in the transmission of pain associated with DLBP. The complex anatomy of the SVN, with its sympathetic components and multiple levels of origin, contributes to the diffuse and poorly localized nature of pain, thereby complicating the diagnosis and management of DLBP. Imaging techniques like magnetic resonance imaging have limitations in detecting endplate pathologies, whereas more specific approaches such as SVN block and discography offer promise for both diagnosis and pain relief. This review summarizes existing knowledge regarding the role of the SVN in transmitting pain from intervertebral discs and related structures, while also emphasizing the contribution of intervertebral discs to the etiology of discogenic pain.

Keywords: Sinuvertebral nerve, chronic low back pain, discogenic pain, sinuvertebral nerve blocks

INTRODUCTION

For decades, chronic low back pain (CLBP) has been recognized as a major global health concern due to its profound impact on patients' social lives and its widespread disruption[1]. CLBP consistently ranks as the leading cause of years lived with disability and has held this position for many years[1,2]. Given its substantial socioeconomic burden, CLBP remains a critical issue that demands continued attention.

Low back pain (LBP) is a multifactorial condition, with pain originating from various structures such as facet joints, ligaments, spinal muscles, intervertebral discs, and vertebral endplates (Table 1). As a result, diagnosing LBP can be highly challenging, requiring both clinical expertise and the ability to address complex cases, grounded in a solid theoretical understanding.The primary challenge with LBP is the absence of reliable early diagnostic criteria, which can result in central sensitization, ultimately leading to chronic pain and hyperalgesia[3,4]. At this point, central sensitization can have significant consequences. The presence of central sensitization increases the likelihood of treatment-resistant. Furthermore, treatment of the underlying spinal condition may not fully resolve the central sensitization and the associated pain. The pain may persist despite conventional treatments for LBP and often leads to poor surgical outcomes. Prolonged LBP can unfortunately lead to extended opioid use[5]. Therefore, we believe that gaining a deeper understanding of discogenic low back pain (DLBP) is crucial for improving diagnosis and developing more effective treatments.

DLBP constitutes one of the most prevalent causes of CLBP, accounting for approximately 26-42% of cases[6,7]. It may occur with or without referred pain and arises from degenerative changes within the disc. Typically, this involves distruption of the internal disc, with fissures observed in the annulus fibrosus[7,8]. Additionally, disc space narrowing at two or more levels is strongly linked to CLBP[9].

Current understanding suggests that the pathomechanisms of DLBP are complex, involving sensory innervation, inflammation, and mechanical hypermobility[10]. Despite extensive research in both humans and animal models, these mechanisms remain

Table 1. Multifactorial pain generators can mainly be discussed in four groups

Myofascial structures
Spinal canal and foramina (stenosis)
Posterior column structures: Facet joint and sacroiliac joint (SIJ) (arthropathy)
Anterior column structures: Disc, Vertebra (Herniated discs, discogenic pain, vertebrogenic pain and compression fractures)

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only partially understood. Discogenic pain is driven by factors such as inflammation, modic changes (MC), and the ingrowth of blood vessels and nerve fibers, with neurotrophins like tumor necrosis factor-α and interleukins promoting nerve growth[10,11]. Additionally, disc degeneration increases collagenase activity, leading to hypermobility and pain. Chronic pain can alter central nervous system (CNS) function, resulting in central sensitization, which manifests as hyperalgesia and allodynia. This central sensitization complicates pain management, requiring targeted treatments beyond addressing spinal problems alone. Managing DLBP effectively requires preventing nerve sensitization, reducing cytokine levels, and controlling disc hypermobility.

Disc aging, disc injury, decreased cellularity, and impaired healing play significant roles in the progression of disc degeneration, which is most commonly observed at the lumbar levels. Notable risk factors include prolonged mechanical loading, trauma, infection, smoking, and genetic predisposition. A classic twin study highlighted the substantial influence of genetic inheritance, estimating heritability at 74%[12].

The sinuvertebral nerve (SVN) plays a crucial role in transmitting pain from axial structures to the CNS. In this review, we explore the pain associated with intervertebral disc disruption and examine the role of the SVN in CLBP. Overall, the study provides a comprehensive overview of how the SVN plays a role in CLBP, emphasizing its diagnostic and therapeutic significance.

MATERIALS AND METHODS

This study aims to provide a comprehensive literature review on discogenic pain to enhance physicians' understanding of the significant factors contributing to CLBP. We systematically researched the published literature for studies focused on patients experiencing CLBP related to issues with the SVN. Data for this review were retrieved from Pubmed, a comprehensive resource that includes peer-reviewed journals, clinical trials, and relevant scientific studies. A systematic search, mostly focusing on articles published between 2000 and 2024, was conducted using relevant keywords, such as "SVN" and "discogenic pain".

Initially, we discuss the clinical presentation of CLBP associated with discogenic pain and the role of imaging in these cases. In this review, we categorize published studies addressing CLBP with a focus on the roles of the SVN.

Additionally, we discuss existing studies on the following topics:

- Clinical Presentation of Patients with LBP Related to Discogenic Pain
- Is Imaging Useful for Patients with LBP Related to Discogenic Pain?
- The Role of SVN in CLBP
- Studies Investigating the Origin of the SVN
- Studies Investigating the Effects of SVN Blocks in DLBP Diagnosis and Management

DISCUSSION

Clinical Presentation of Patients with LBP Related to Discogenic Pain

Since the diagnostic process begins with a suspicion of underlying pathology, clinicians need relevant background information to effectively approach CLBP. The clinical presentation of the patient can significantly aid in the diagnostic process (Table 2). Discogenic pain originating from the anterior column of spine is typically characterized by deep, aching, and burning pain located in the midline of the lower back. Patients often report that their pain intensifies with activities such as sitting, bending forward, and changing position from sitting to standing[13]. Generally, these patients tend to prefer walking over sitting, as they find it challenging to tolerate prolonged periods in a seated position.

As flexion-based movements exert significant amount of stress on the anterior column of spine, a key expectation during physical examination is the presence of pain during such movements. This finding is particularly significant for patients, as it is widely acknowledged that extension-based movements are generally associated with posterior column structures, including the facet joints. Additionally, tenderness during palpation serves as a crucial indicator for clinicians in their diagnostic approach.

Is Imaging Useful for Patients with LBP Related to Discogenic Pain?

While magnetic resonance imaging (MRI) is an essential diagnostic tool for spinal-related pain issues, its utility in CLBP can be limited due to its insufficient diagnostic value. The correlation between MRI findings and patients' symptoms can often be unclear, and internal disc disruptions are not well visualized using this imaging technique. In contrast, discography with contrast material presents a more effective method for diagnosing internal disc disruption.

It is known that innervation in endplates is more extensive in symptomatic patients. Increased innervation is particularly expected in painful discs exhibiting annular fissures and radial tears. Current evidence indicates that endplates with pathologies have significantly higher nerve densities than those without pathologies. Vertebral endplate signal changes have been identified as a potential MRI finding in patients with non-specific LBP, with a median prevalence of 43%[14].

Table 2. Important clinical presentation of anterior column pain

Midline low back pain (deep, aching and burning)
Pain worsens with sitting, bending forward, changing position
Patient prefers walking around rather than sitting in a position long time
Tenderness on palpation and percussion
Pain worsens with flexion

Unfortunately, endplate pathologies can be undetectable on MRI in the majority of the cases[15]. Therefore, MRI findings have limited utility in CLBP patients with endplate pathologies.

In contrast to the limited diagnostic value of MRI for endplate pathologies, vertebral bone marrow lesions can be identified as MC on MRI. MC exhibit high specificity for DLBP[16]. Since MC prevalence is high in CLBP patients and back pain severity can correlate with MC lesion size, the high specifity of MRI findings may have importance in the clinical practice[16]. Therefore, the presence of high-intensity zones and MC on MRI serves as an indicator of discogenic pathology associated with discogenic pain.

In a study analyzing lumbar radiographs of 2,819 participants, de Schepper et al.[9] found a significant association between disc space narrowing at two or more levels and LBP. Their findings indicated that disc space narrowing was more closely linked to LBP than osteophytes and other radiographic features, particularly after excluding the L5-S1 level.

The Role of SVN in CLBP

A thorough understanding of the SVN's anatomy is crucial for understanding its role in CLBP (Figure 1). The initial understanding of this topic began to take shape after the research conducted by Bogduk[17] and Bogduk et al. [18], which included microdissection and histological studies in the early 1980s. In these studies, they proposed the possibility of dual innervation of the intervertebral disc by both somatic and sympathetic systems and provided detailed anatomical description of the rami communicans. Over time, the concept of dual innervation by the somatic nervous system and the sympathetic nervous system has gained wide acceptance[19]. Then, this understanding helps explain how diffuse LBP can trigger sympathetic pain. The SVN has a multilevel origin, the primary branch composed by from the subjacent intervertebral level, the smaller branches composed from the level below and above, allowing it to extend over three segments. This complex structure of SVN, combined with its sympathetic component and multi-level origin, likely contributes to the diffuse and poorly localized nature of discogenic pain associated with the SVN.

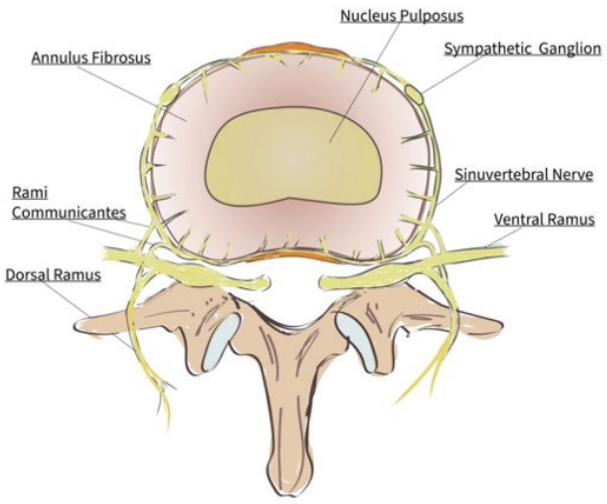

Figure 1. Illustrates the anatomy of the sinuvertebral nerve and the innervation of the intervertebral disc

Similarly, understanding the innervation of the annulus is essential for comprehending the SVN 's role in discogenic pain. Nociceptive signals from the anterior and lateral annuli are clearly transmitted via the sympathetic pathway[20]. However, the pathways for nociceptive signals from the posterior annuli are still debated. These nociceptive pathways may involve both the somatic and sympathetic systems or could rely entirely on the sympathetic pathway via rami communicans fibers[20].

The sympathetic components are the SVNs and the rami communicantes. These nerves provide innervation to many of the key anatomical structures associated with diffuse CLBP, such as the dorsal longitudinal ligament, intervertebral discs, and the ventral portion of the dura mater[19]. The notable feature of this nerve is that the SVN cannot directly reach a somatic element at each lumbar spine level. Instead, it transmits pain impulses via the rami communicantes, which are sympathetic fibers, and connects to the L2 spinal ganglion[19]. As a result, pain originating from the L3, L4, and L5 levels is transmitted by the SVN s, which relay signals to the CNS through the L2 spinal ganglion. This raises discussions regarding the potential advantages of infiltrating the L2 spinal ganglia as a treatment option for patients with CLBP.

Top of FormBottom of FormStudies Investigated Origin of SVN

While the precise origin of the SVN remains a topic of debate, recent studies suggest that it consists of branches from both somatic and autonomic roots. Specifically, the somatic roots originate from the ventral ramus, while the autonomic roots arise from the gray ramus, collectively forming the SVN[21].

A recent anatomical study provides a detailed anatomical understanding of the SVNs. In this study, Zhao et al.[21] examined 10 embalmed human cadavers, identifying a total of 450 SVNs across 100 lumbar intervertebral foramina. Their findings categorized the SVN s into two groups: SVN accessory (or deputy) branches and SVN main (or trunk) branches. The SVN main trunks were mainly (97.00%) present in the intervertebral foramina. The initial segment of the SVN was located along the posterior-lateral edge of the disc, and the main trunks originated from two primary sources: 44.2% from the gray ramus communicans and 55.8% from the anterior surface of the spinal ganglia.

In an animal study, Nakamura et al.[22] examined the intervertebral discs following the resection of sympathetic trunks, both unilaterally and bilaterally, at various levels in forty-five rats. Their primary focus was on the posterior aspect of the lumbar intervertebral discs, as disk lesions typically occur in this region. The results revealed distinct differences between unilateral and bilateral resections. In cases of total bilateral resection of the sympathetic trunks, the neural network in the

posterior portion of the intervertebral discs was found to be absent. Conversely, a slight decrease in innervation was noted in instances of bilateral single-level resection or unilateral multisegmental resection. Thus, the researchers concluded that the innervation of the posterior lumbar intervertebral discs is supplied by multi-segmental and bilateral sympathetic nerves.

In a study that investigates the anatomy of the SVN to enhance understanding of its potential role in lumbar discogenic diffuse pain[23]. Quinones et al.[23] conducted on six lumbar blocks from donors, the dissection revealed the SVN's origin from somatic and sympathetic branches of the rami communicantes. Out of 48 intervertebral canals examined, 43 SVNs were evaluable, with some levels exhibiting two SVNs. The SVN displayed various patterns of course in the vertebral canal, primarily an ascending branch, and had connections with adjacent SVNs in several cases. The findings suggest that a thorough understanding of SVN anatomy could lead to improved treatments for DLBP, with recommendations to block the SVN at the inferior vertebral notch of adjacent segments.

Studies Investigated Effects of SVN Blocks in DLBP Diagnosis and Management

Wang et al.[24] conducted a study that aimed to assess the effectiveness of SVN blocks in diagnosing DLBP, data from 48 patients with suspected discogenic pain at L4/5 were analyzed. Twenty-four patients received discoblocks (intradiscal injection of 1 mL 0.5% lidocaine), while another 24 received SVN blocks. Patients who responded positively underwent percutaneous endoscopic radiofrequency thermal annuloplasty. Both groups showed similar improvements in visual analogue scale (VAS) and Oswestry Disability Index (ODI) scores at all time points, with significant improvements post-surgery. The study concluded that SVN block is as effective as discoblock for diagnosis and warrants further research.

In a study aimed at assessing the sensitivity and target specificity of SVN block (SVNB) for diagnosing lumbar discogenic pain, and comparing it to the gold standard of discography[25], Schliessbach et al.[25] concluded that SVNB cannot yet replace discography. However, the results suggest potential for future improvements in target specificity. Success of SVNB was defined by Schliessbach et al.[25] as at least 80% pain reduction or significant relief of physical limitations. They conducted the study with fifteen patients who had positive discography results and underwent SVNB, finding that the sensitivity of SVNB was 73.3%, while its target specificity was lower, at 40%.

In a retrospective study, Liu et al.[26] investigated the diagnostic and clinical efficacy of SVNB for the management of DLBP. Their research involved 32 patients with DLBP and tracked their outcomes over time. The improvement rates in VAS scores were 56.52% at 3 days, 54.34% at 7 days, 38.61% at 1 month, and 34.26% at 3 months following SVNB. This study demonstrated that SVNB is a rapid and cost-effective minimally invasive treatment. ODI scores were also improved in the study patients. These findings indicated that SVNB not only assists in diagnosis but also provides short-term pain relief and improves physical function in patients with DLBP.

CONCLUSION

The review emphasizes that understanding the anatomy and role of the SVN is critical for diagnosing and managing CLBP related to discogenic pathology. The involvement of sympathetic components like the SVN and rami communicantes, and their role in transmitting diffuse, poorly localized pain, underlines the potential of SVNB as a diagnostic tool. Studies indicate that while the effectiveness of SVN blocks is comparable to other methods such as discoblock (intradiscal injections), their specificity is still limited, warranting further investigation and refinement for better clinical application in diagnosing DLBP. By focusing on the distinct roles of the SVN nerves, novel treatment strategies such as nerve blocks may offer potential improvements in pain management and patient outcomes.

Footnote

Authorship Contributions

Concept: B.C., A.C., Design: B.C., A.C., Analysis or Interpretation: B.C., Literature Search: B.C., Writing: B.C.,

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REFERENCES

1. Hoy D, March L, Brooks P, Blyth F, Woolf A, Bain C, et al. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. Ann Rheum Dis. 2014;73:968-74.
2. Wu A, March L, Zheng X, Huang J, Wang X, Zhao J, et al. Global low back pain prevalence and years lived with disability from 1990 to 2017: estimates from the Global Burden of Disease Study 2017. Ann Transl Med. 2020;8:299.
3. O'Neill S, Manniche C, Graven-Nielsen T, Arendt-Nielsen L. Generalized deep-tissue hyperalgesia in patients with chronic low-back pain. Eur J Pain. 2007;11:415-20.
4. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152(Suppl 3):2-15.
5. Deyo RA, Von Korff M, Duhrkoop D. Opioids for low back pain. BMJ. 2015;350:6380.
6. Zhang YG, Guo TM, Guo X, Wu SX. Clinical diagnosis for discogenic low back pain. Int J Biol Sci. 2009;5:647-58. Erratum in: Int J Biol Sci. 2010;6:613.
7. Peng BG. Pathophysiology, diagnosis, and treatment of discogenic low back pain. World J Orthop. 2013;4:42-52.
8. Urban JP, Roberts S. Degeneration of the intervertebral disc. Arthritis Res Ther. 2003;5:120-30.
9. de Schepper EI, Damen J, van Meurs JB, Ginai AZ, Popham M, Hofman A, et al. The association between lumbar disc degeneration and low back pain: the influence of age, gender, and individual radiographic features. Spine (Phila Pa 1976). 2010;35:531-36.
10. Ohtori S, Inoue G, Miyagi M, Takahashi K. Pathomechanisms of

discogenic low back pain in humans and animal models. Spine J. 2015;15:1347-55.

11. Jha R, Bernstock JD, Chalif JI, Hoffman SE, Gupta S, Guo H, et al. Updates on pathophysiology of discogenic back pain. J Clin Med. 2023;12:6907.
12. Sambrook PN, MacGregor AJ, Spector TD. Genetic influences on cervical and lumbar disc degeneration: a magnetic resonance imaging study in twins. Arthritis Rheum. 1999;42:366-72.
13. McCormick ZL, Sperry BP, Boody BS, Hirsch JA, Conger A, Harper K, et al. Pain location and exacerbating activities associated with treatment success following basivertebral nerve ablation: an aggregated cohort study of multicenter prospective clinical trial data. Pain Med. 2022;23(Suppl 2):14-33.
14. Jensen TS, Karppinen J, Sorensen JS, Niinimäki J, Leboeuf-Yde C. Vertebral endplate signal changes (Modic change): a systematic literature review of prevalence and association with non-specific low back pain. Eur Spine J. 2008;17:1407-22.
15. Fields AJ, Liebenberg EC, Lotz JC. Innervation of pathologies in the lumbar vertebral end plate and intervertebral disc. Spine J. 2014;14:513-21.
16. Dudli S, Sing DC, Hu SS, Berven SH, Burch S, Deviren V, et al. Issls prize in basic science 2017: Intervertebral disc/bone marrow cross-talk with Modic changes. Eur Spine J. 2017;26:1362-73.
17. Bogduk N. The innervation of the lumbar spine. Spine (Phila Pa 1976). 1983;8:286-93.
18. Bogduk N, Tynan W, Wilson AS. The nerve supply to the human lumbar intervertebral discs. J Anat. 1981;132:39-56.
19. Raoul S, Faure A, Robert R, Rogez JM, Hamel O, Cuillère P, et al. Role of the sinu-vertebral nerve in low back pain and anatomical basis of therapeutic implications. Surg Radiol Anat. 2003;24:366-71.
20. Das G, Roy C. Rami communicans fibers in discogenic low back pain: The controversies. Indian J Pain. 2018;32:60-2.
21. Zhao Q, Cheng L, Yan H, Deng S, Zhao J, Liu Z, et al. The anatomical study and clinical significance of the sinuvertebral nerves at the lumbar levels. Spine (Phila Pa 1976). 2020;45:61-6.
22. Nakamura S, Takahashi K, Takahashi Y, Morinaga T, Shimada Y, Moriya H. Origin of nerves supplying the posterior portion of lumbar intervertebral discs in rats. Spine (Phila Pa 1976). 1996;21:917-24.
23. Quinones S, Konschake M, Aguilar LL, Simon C, Aragones P, Hernández LM, et al. Correction to clinical anatomy of the lumbar sinuvertebral nerve with regard to discogenic low back pain and review of literature. Eur Spine J. 2021;30:2409.
24. Wang G, Zhang Z, Liu J, Cheng L. Comparison of the effect of diagnosing discogenic low back pain by sinuvertebral nerve block versus discoblock a retrospective cohort study. Eur Spine J. 2023;32:2093-100.
25. Schliessbach J, Siegenthaler A, Heini P, Bogduk N, Curatolo M. Blockade of the sinuvertebral nerve for the diagnosis of lumbar diskogenic pain: an exploratory study. Anesth Analg. 2010;111:204-6.
26. Liu Z, Ma R, Fan C, Chen J, Zhang R, Zheng Z, et al. Sinuvertebral nerve block treats discogenic low back pain: a retrospective cohort study. Ann Transl Med. 2022;10:1219.