EDITORIAL

Basi- Vertebral Nerve (BVN) Ablation As an Emerging Treatment for Vertebrogenic Lower Back Pain

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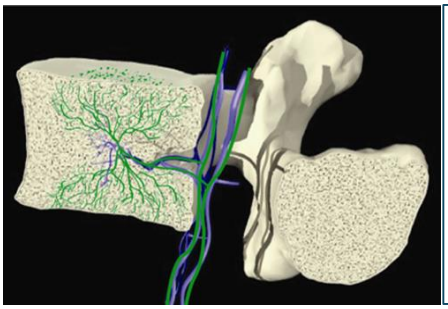
ABSTRACT:

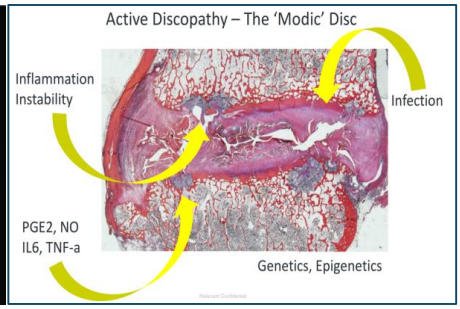
Chronic low back pain (CLBP) remains a leading global cause of pain and disability, with degenerative disc disease (DDD) historically implicated as the predominant aetiology. Recent research has introduced the concept of vertebrogenic back pain (VLBP), a condition where vertebral endplates contribute significantly to CLBP. The Basi-vertebral nerve (BVN), which innervates the vertebral endplates, has emerged as a key target for therapeutic intervention in VLBP. While BVN ablation (BVNA) has shown promise, its integration into clinical practice remains in the early stages. Supported by robust pre-clinical and clinical evidence, the American Society of Pain and Neuroscience (ASPN) has recognized the need for evidence-based guidelines to guide patient selection for BVNA. The ASPN's workgroup, which reviewed available literature, concluded that BVNA offers Level A evidence, confirming its substantial benefit in appropriately selected individuals.

INTRODUCTION:

Pathophysiology and Background of Vertebrogenic Pain

Degenerative disc disease (DDD) has long been considered a primary contributor to chronic low back pain (CLBP). However, recent histological, immunological, and radiological studies have illuminated a distinct pain generator: the vertebral endplates[1]. The endplates serve several critical functions, including dispersing intradiscal pressure and providing nutrients to the disc[2]. The Basi-vertebral nerve (BVN), which arises from the sinuvertebral nerves (SVN), innervates the vertebral body and its endplates. These nociceptive fibers are believed to be the primary pain generators in vertebrogenic back pain (VLBP)[3]. Histological evidence has shown that degeneration of the intervertebral disc leads to the proliferation of BVN fibers at the endplates, heightening sensitivity to pain from compressive forces.





The vertebral endplates, which are highly innervated by BVNs, are vulnerable to damage from physiological aging, trauma, inflammation, and other factors. Insults to the endplates trigger the release of proinflammatory and neurogenic factors, which exacerbate pain by promoting the proliferation of BVN fibers. This process increases sensitivity and pain perception, leading to a feedback loop of worsening vertebral degeneration and chronic pain[3].

Modic changes:

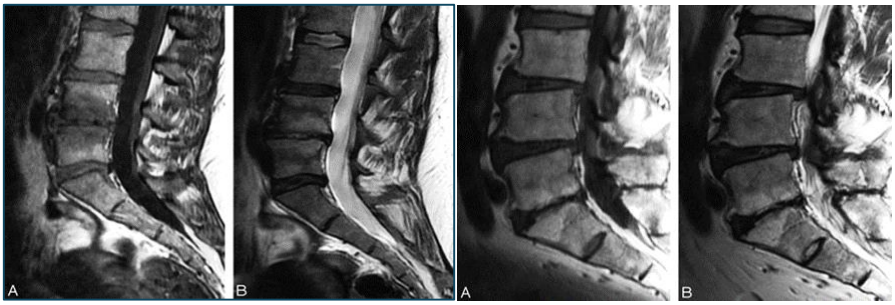
Dr. Modic, a radiologist, was the first physician to publish a classification of the degenerative changes of the endplate based on magnetic resonance imaging (MRI), with three types highlighting the evolutionary stages [4].

Type 1 denotes acute degeneration commonly associated with pain as fibrovascular changes occur within the subchondral bone marrow resulting in oedema and inflammation. On MRI, the endplates are hypointense on T1 and hyperintense on T2-weight images.

Type 2 classifies subacute and chronic changes as fatty bone marrow infiltration occurs within the vertebral body with the MRI demonstrating hyperintense signals on both T1 and T2 weighted images.

The progression of chronic changes may lead to bony sclerosis at the endplates, observed within the Type 3 Modic classification. These changes are visible on MRI as hypointense on T1/T2 [(4)].

A recently proposed scoring system ("Mo-di-disc") including Modic changes, fatty infiltration in the paraspinal muscles and disc degeneration was found to be the most significant predictor for patients with more intense low back pain[(5)]. Yet, although Modic type 1 and 2 changes are highly specific for low back pain, MRI findings should always be correlated with clinical findings [(2)].



Ongoing scientific evidence supports that pathological neurotization of the BVN may lead to significant disability by causing axial low back pain that is worse with sitting and forward flexion.[1] Therapeutic modalities aimed at ablation of the BVN have been shown to be very effective for the treatment of chronic low back pain (LBP)[(1)] .

Evidence Supporting BVN Ablation

Basi vertebral nerve ablation (BVNA) represents a novel intervention targeting vertebrogenic low back pain (VLBP), typically identified by vertebral endplate degeneration and the presence of Modic changes on MRI[(6)].These Modic changes, characterized by disruption, fissuring, and inflammation of the endplates, are often accompanied by nociceptive input carried by the BVN, supporting the notion that VLBP is a distinct subset of chronic axial low back pain. This pathology is often refractory to conservative treatments, and BVNA has emerged as a potential solution for such patients. [(2)]

Currently, BVNA is FDA-cleared for use in patients with chronic axial low back pain (duration >6 months) who have failed conservative treatments and exhibit Modic changes on MRI [(7,8)]. Clinical evidence supporting BVNA is robust, with several Level I randomized controlled trials (RCTs) and Level II studies demonstrating significant improvements in pain, disability, and function, with effects sustained up to five years.

One of the most compelling studies, conducted by Koreckij et al. (2021) [(1)], demonstrated that BVNA led to sustained improvements in pain, function, and quality of life for up to 24 months, with over 50% pain reduction in 72% of patients, 31% of whom were pain-free at follow-up[(1)] . Similarly, Smuck et al. (2021) reported significant pain and functional improvements in patients undergoing BVNA compared to a standard of care treatment group, with these effects lasting for at least 12 months [(9)].

Opioid use reduction is another key benefit of BVNA. Multiple studies have shown that BVNA can lead to a significant decrease in opioid consumption. For example, the prospective study by Truumees et al. (2019) reported that 50% of participants discontinued opioids at three months following BVNA[(10)] .Other studies, including those by Fischgrund et al. (2019) and Koreckij et al. (2021), also highlighted significant reductions in opioid use, with some patients completely eliminating their need for opioid therapy[(12)].

In terms of functional outcomes, BVNA consistently results in significant improvement, with most studies reporting a clinically meaningful reduction in the Oswestry Disability Index (ODI) score, indicating a marked improvement in patient mobility and daily function. Improvements in quality of life have also been noted, although the evidence is less robust, with some studies showing positive results at the 24-month follow-up [(12)].

Safety Profile and Adverse Events

BVNA is associated with a favourable safety profile, with adverse events being rare and generally minor [(13)]. Reported side effects include

transient motor or sensory deficits, incisional pain, and minor procedural complications such as radiculitis or vertebral compression fractures in osteoporotic patients. Serious adverse events, such as spinal cord injury or severe complications related to the procedure, have not been reported 21. Long-term follow-up imaging has shown no significant adverse changes in disc degeneration or vertebral bone health, further supporting the safety of BVNA as a therapeutic option for VLBP [(12)].

CONCLUSION:

Cumulative evidence strongly supports the use of BVNA in the treatment of vertebrogenic low back pain, demonstrating substantial improvements in pain, function, and quality of life, as well as reductions in opioid use. BVNA has shown superiority over standard care treatments for VLBP, and the procedure is associated with a favourable safety profile. The available literature provides moderate-to-high quality evidence supporting the integration of BVNA into clinical practice, with the American Society of Pain and Neuroscience (ASPN) assigning it a Level A recommendation based on high certainty of benefit for appropriately selected patients. Ongoing studies will likely continue to refine patient selection criteria and expand our understanding of BVNA's long-term efficacy and safety.

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