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# BASIVERTEBRAL NERVE ABLATION FOR VERTEBROGENIC CHRONIC LOW BACK PAIN: CURRENT EVIDENCE AND CLINICAL IMPLICATIONS

**Sazdov D.**

[1] *Department of Anesthesia and Intensive Care Medicine, Acibadem Sistina Hospital Skopje*

[2] *Department of Education and Training, Acibadem Sistina Hospital Skopje*

## Abstract

Chronic low back pain (CLBP) is one of the leading causes of disability worldwide and represents a major clinical challenge in anesthesiology and pain medicine. While discogenic and facetogenic mechanisms have traditionally dominated diagnostic paradigms, increasing evidence supports vertebrogenic pain as a distinct entity mediated by the basivertebral nerve (BVN), particularly in patients with vertebral endplate degeneration and Modic type I or II changes on magnetic resonance imaging.

The aim of this review is to summarize current knowledge on vertebrogenic pain pathophysiology, the anatomical and biological role of the BVN, and the clinical evidence supporting basivertebral nerve radiofrequency ablation (BVN RFA) as a treatment option for chronic low back pain.

A narrative review of randomized controlled trials, prospective cohort studies, and systematic reviews was performed, focusing on clinical efficacy, durability of outcomes, safety profile, and patient selection criteria. Available evidence demonstrates clinically meaningful and sustained reductions in pain intensity and disability following BVN ablation in appropriately selected patients, with benefits persisting for several years and a low incidence of serious adverse events.

Basivertebral nerve ablation represents a promising minimally invasive intervention for vertebrogenic CLBP and may expand therapeutic options available to anesthesiologists and pain specialists. Further independent studies are needed to refine patient selection and confirm long-term outcomes.

***Key words:*** *basivertebral nerve; chronic low back pain; Modic changes; radiofrequency ablation; vertebrogenic pain.*

## Introduction Epidemiology of Low Back Pain

Low back pain (LBP) is a major global health problem affecting all age groups, with lifetime prevalence estimates exceeding 60% in many populations (1,2). It is the leading global cause of years lived with disability and it imposes substantial socioeconomic burden due to healthcare utilization, work absenteeism, and functional impairment (1,3). CLBP — usually defined as pain persisting beyond 12 weeks — contributes disproportionately to chronic disability (1,3). The

heterogeneous etiology of CLBP has historically challenged clinicians. In many cases, definitive pain generators could not be established, leading to a diagnosis of non-specific low back pain in up to 85% of patients (4). Disc degeneration has long been considered a principal cause ("discogenic pain"), with nociceptors within the degenerated annulus fibrosus implicated in symptom generation (5-7). However, accumulating evidence suggests that nociceptive input from vertebral endplates mediated by the BVN also plays a significant role in a subset of patients (4,8,9).

## Shift from Discogenic to Vertebrogenic Pain Paradigm

For decades, the dominant model for CLBP centered on disc degenerative changes and associated annular nociception ("discogenic pain") (5). However, advances in spinal imaging and histologic studies have revealed a robust population of nociceptive fibers within vertebral endplates and along the BVN (10). Endplate damage and associated inflammatory processes correlate with specific MRI findings termed Modic changes (type I and II), which are strongly associated with persistent CLBP and poorer response to conventional conservative treatments (11-13). Anatomically, the BVN originates within the vertebral body, entering through the basivertebral foramen and innervating adjacent endplates. Damaged or degenerated endplates show increased density of nociceptive fibers expressing substance P and related neuropeptides, supporting a distinct vertebrogenic pain mechanism transmitted via the BVN (10,14-16). In recognition of this mechanistic subset, vertebrogenic pain has been conceptualized as a phenotype distinct from discogenic, facetogenic, or neuropathic pain (14).

## Other Pain Generators in Low Back Pain

CLBP is multifactorial, with several well-recognized pain generators:

- Discogenic pain: Nociceptor activation in annular fissures of degenerated intervertebral discs (5,6).
- Facetogenic pain: Facet joint arthropathy with nociception from medial branch nerves (17).
- Sacroiliac joint pain: Inflammation or dysfunction of the sacroiliac joints (18).
- Radiculopathy: Nerve root compression or irritation producing radiating symptoms (19).
- Musculoligamentous pain: Soft-tissue strain or dysfunction contributing to pain without a clear structural lesion (20).

Emerging evidence recognizes vertebrogenic pain — where endplate pathology and BVN-mediated nociception drive axial low back pain — as an important and potentially treatable component of CLBP (4,10).

## Basivertebral Nerve Ablation: Mechanism and Technique

BVN ablation aims to disrupt nociceptive signal transmission from sensitized vertebral endplates to the central nervous system (21). Radiofrequency energy is delivered intraosseously to thermally ablate the BVN via a transpedicular approach under fluoroscopic guidance (21,22).

Patient selection typically includes axial LBP of at least 6 months' duration, failure of conservative therapies, and the presence of Modic type I or II changes on MRI, which serve as imaging biomarkers for vertebrogenic pain (11,23-25).

## Clinical Evidence for BVN Ablation

Multiple clinical studies, including randomized controlled trials (RCTs), cohort studies, and meta-analyses, support the efficacy of BVN ablation in appropriately selected patients. An early sham-controlled multi-center RCT demonstrated significantly greater improvements in Oswestry Disability Index (ODI) scores and responder rates in the BVN ablation arm at 3 months compared with sham controls (26). Other prospective trials have shown sustained improvements in pain and function at 12- and 24-month follow-up (9,27).

A systematic review with single-arm meta-analysis reported that approximately 65% of patients achieved ≥50% pain relief at 6 and 12 months post-BVN RFA, with similar proportions showing clinically meaningful functional gains (28). Likewise, comparative analyses indicate significant improvements in visual analog scale (VAS) and ODI scores at various follow-up intervals versus baseline (7,29). Importantly, real-world pooled analyses confirm these findings across multiple study designs (24,30).

Safety profiles are favorable, with low rates of serious adverse events reported when appropriate patient selection and technique are applied (27,29). Nevertheless, the literature underscores the need for more high-quality, non-industry-funded studies to validate these outcomes across broader patient populations (28.29).

## Meta-Analysis and Comparative Effectiveness

While early meta-analyses historically included a limited number of studies, recent systematic reviews strengthen the evidence base. A single-arm meta-analysis of six unique BVN RFA study populations (414 treated patients) demonstrated that approximately 65% of patients achieved ≥50% pain reduction at both 6 and 12 months, and ~75% experienced ≥15-point ODI improvement at these time points (1).

Another meta-analysis combining randomized and prospective non-randomized studies (total n = 429 participants) reported a significant pooled mean ODI difference of –28.08 points and VAS difference of –3.16 cm favoring BVN RFA versus control groups, underscoring consistent and clinically relevant improvements in pain and disability across heterogeneous study designs (2).

A broader systematic review and meta-analysis comparing BVN ablation with other minimally invasive interventions found that BVN ablation produced statistically significant improvements in pain and function at 6-, 12-, and 24-month follow-ups relative to other modalities such as annular disc RFA, facet joint procedures, or injections. BVN ablation, biological therapy, and multifidus stimulation were among the only interventions showing durable benefits beyond short-term relief, with no significant differences in serious adverse events (SAEs) across treatment modalities (13).

## Discussion

The growing body of evidence investigating BVN ablation highlights its promise as a targeted, minimally invasive intervention for vertebrogenic CLBP, particularly in patients with Modic changes on MRI that reflect endplate degeneration and vertebral body inflammation. This shift in conceptualizing pain — from predominantly discogenic or nonspecific mechanisms to a vertebrogenic phenotype — has been substantiated by both experimental and clinical trial data demonstrating that nociceptive fibers within the endplates and BVN carry significant pain signals, making them viable therapeutic targets (10,14,15).

## Clinical Trial Evidence

The first large randomized, double-blind, sham-controlled multicenter trial provided rigorous evidence supporting the efficacy of BVN ablation. In this study (n = 225), patients randomized to BVN radiofrequency ablation (RFA) had significantly greater improvements in Oswestry Disability Index (ODI) and Visual Analog Scale (VAS) pain scores at 3 months compared with sham controls. In the per-protocol analysis, mean ODI reduction was greater by ~20 points in the treatment arm ($p < 0.001$), and responder rates (≥10-point ODI improvement) were higher (75.6% vs 55.3%) (12).

Significantly, these benefits were sustained at 12 months with continued large effect sizes — average ODI decreases ~25.7 points and VAS reductions ~3.8 cm from baseline, with ~64% achieving ≥50% pain relief and ~29% reporting being pain-free. [7] Longer term follow-up further strengthens the durability of BVN ablation outcomes. In the same cohort, the five-year treatment arm data showed sustained improvement in both pain and function (ODI and VAS) and reduced reliance on injection or opioid therapies compared with the baseline, corroborating that the clinical benefit is not merely short-lived (15).

Real-world prospective cohort data align with these RCT results. In a 60-participant real-world study, over half of the participants achieved clinically meaningful improvements (≥30% ODI improvement and ≥50% NRS/VAS reduction) at 12 months, and more than half judged themselves "much improved" on global impression measures (3). Similarly, community practice cohorts have reported statistically and clinically significant reductions in ODI and VAS at multiple early and late follow-ups, demonstrating that outcomes observed in controlled research are generalizable to routine clinical settings (8).

## Clinical Interpretation and Patient Selection

The consistency of the positive results — across RCTs, meta-analyses, and real-world cohorts — supports the notion that BVN ablation as a true biological effect rather than a transient placebo response. The magnitude of ODI and VAS changes frequently exceeds established minimal clinically important differences (MCIDs), suggesting real functional and quality-of-life improvements for patients (1,3,7,8).

Accurate phenotyping appears critical to achieving optimal results. Most trials required either Modic type I or II changes or evidence of vertebrogenic pain syndrome — a subset distinct from

patients with primarily discogenic, facetogenic, or sacroiliac joint–related pain (4,7,12).

## Limitations and Future Directions

Despite compelling data, the literature contains certain limitations that must be acknowledged:

- Many studies are industry-sponsored and may have inherent biases (1,8,24).
- The number of high-quality, large-scale independent trials remains limited, and more non-industry–funded research is needed (1,8).
- Longer-term comparative data with standard surgical or multimodal strategies are still evolving.

Future research would benefit from direct head-to-head comparisons with alternative interventions, detailed cost-effectiveness analyses, and further biomarker refinement (e.g., quantifying Modic changes) to enhance patient stratification (14).

## Conclusion

The advent of BVN ablation reflects a significant evolution in understanding CLBP pathophysiology and offers a promising therapeutic option for vertebrogenic pain. Current evidence demonstrates meaningful pain and disability reduction in selected patients, with acceptable safety. Continued research, particularly large-scale pragmatic trials, is needed to refine selection criteria, long-term outcomes, and comparative effectiveness relative to other established interventions.

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