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Basivertebral Nerve Radiofrequency Ablation for Vertebral Osteomyelitis: A Technical Report

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Background: Vertebral osteomyelitis (VO) frequently results in persistent vertebrogenic back pain (VBP) due to ongoing endplate inflammation and structural damage despite infection-directed therapy. The application of basivertebral nerve radiofrequency ablation (BVNRFA) for VBP resulting from vertebral infection has not been explored in the current literature.

Case Report: A 53-year-old man with poorly controlled diabetes and recurrent L3-L4 osteomyelitis underwent multiple hospitalizations for sepsis and progressive vertebral body destruction. Serial magnetic resonance imaging demonstrated chronic L3-L4 osteomyelitis with marrow edema, and 2 persistent enhancing fluid pockets, but no surgically drainable epidural abscess. Despite prolonged intravenous antibiotics and the absence of operative indications, severe axial lumbar pain continued with significant functional limitation. BVNRFA was performed to target nociceptive signaling originating from chronically diseased vertebral endplates. The intervention was tolerated without complications and resulted in meaningful improvement in axial pain and mobility.

Conclusion: Our case illustrates a novel application of BVNRFA for VBP associated with chronic VO.

Key words: Osteomyelitis, vertebral body, basivertebral nerve, radiofrequency ablation, case report

BACKGROUND

Vertebral osteomyelitis (VO) is an infection of the vertebral body that is most commonly caused by hematogenous spread of bacteria from an infection elsewhere in the body, direct introduction of bacteria during spinal surgery, or spinal trauma (1). Staphylococcus aureus is the most common causative organism; however, gram-negative bacteria, such as Escherichia coli, are also common (1). Diagnostic criteria for the diagnosis of VO are variable, especially when blood cultures and biopsy results are negative; however, imaging modalities, most commonly magnetic resonance imaging (MRI), are used (1). The most frequent presenting symptom of VO is severe localized back pain, present in 86% to 99% of cases, accompanied by elevated erythrocyte sedimentation rate or C-reactive protein, fever, and possible neurological deficits (2).

Treatment includes antibiotic therapy, with surgical intervention reserved for severe cases or failure of medical management (2). Multimodal pain management is often needed, such as anti-inflammatory pain medication, short-term opioids, and spinal immobilization (1,3). This is especially significant in cases where the back pain persists even after the infection has been resolved. A retrospective study (4) found that 18.6% of patients who had hematogenous VO had persistent pain 12 months after being treated for the initial condition.

More specifically, chronic pain can manifest from

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vertebral endplate destruction, vertebral body collapse, and spinal cord deformity (5). The basivertebral nerve (BVN) innervates the vertebral endplates and contains nociceptors that transmit pain signals when the area is damaged (5). BVN radiofrequency ablation (BVNRFA) is used to reduce vertebrogenic back pain (VBP) by using thermal energy to interrupt the nociceptive signaling from the damaged endplates (6). BVNRFA is an effective procedure that can help provide patients with significant relief, especially if there is radiographic evidence of Modic changes (MCs) to the vertebral endplates (6).

There have not been any cases reported in the literature of using BVNRFA to treat chronic back pain from VO. We report a case of BVNRFA used to manage chronic, severe VBP originating from VO. The patient provided informed consent to be included in our case report.

CASE PRESENTATION

A 53-year-old male patient with a past medical history of type 2 diabetes, prior stroke, hypertension, enlarged prostate, acid reflux, anxiety, and recurrent L3-L4 osteomyelitis (January 2025 to June 2025) with drainage of an epidural abscess (January 2025) presented in June 2025 with 2 days of chills and one day of nausea, vomiting, and diarrhea. The patient was last hospitalized from May 2025 through mid-June 2025 for severe sepsis with lactic acidosis secondary to L3-L4 discitis complicated by epidural abscesses. He was treated with intravenous daptomycin during that admission and discharged with instructions to complete a course of oral antibiotics that included moxifloxacin. However, he was unable to obtain the medications and had not taken them for 4 days before this presentation. The patient reported increased swelling in the lower spine with tenderness to palpation at the L3-L4 location and continued nausea and vomiting.

A computed tomography of the thoracic and lumbar spine demonstrated multilevel degenerative joint disease and recurrence of L3-L4 osteomyelitis with thickening of the anterior epidural fat at L3-L4 and showing involvement of the right psoas muscle (Figs. 1 and 2).

After consultation with neurosurgery, the patient received cefepime and vancomycin to treat the osteomyelitis, as well as ondansetron and morphine for the nausea and pain.

The next visit occurred in mid-October 2025 when the patient presented with back pain and vomiting. He notes that this was a continuation of the chronic lumbar pain present since January 2025, but that it has progressed in the preceding days and now radiates to the right leg, making it hard to walk, and he now uses a wheelchair. He denied fever or chills, but reported vomiting once daily and 3 episodes of diarrhea. The patient notes that he has been out of his medication for a prolonged period and was only taking short-acting insulin and tamsulosin. An MRI without contrast demonstrated stable residual osteomyelitis/discitis at L3-L4 with improved bone marrow edema and inflammatory changes (Fig. 3).

The contrast-enhanced MRI showed abnormal soft tissue enhancement and residual discitis at L3-L4. Two areas of residual fluid measured 3 cm and 1.3 cm. Two lytic lesions in L4, measuring 1.4 cm and 1 cm, were concerning for a possible abscess. Possible recurrent osteomyelitis was discussed with the neurosurgeon on call. The patient was discharged, but returned the following day with continued back pain. After conferring with 2 additional surgeons, he was given fentanyl, vancomycin, and piperacillin-tazobactam and discharged.

A few days later, the patient presented again with lower back pain without radiation to the lower extremity and generalized weakness without numbness/tingling in the saddle area. MRI demonstrated abnormal osseous and soft tissue enhancement at L3-L4 consistent with residual osteomyelitis, as well as a stable lytic lesion in the L4 vertebral body that was possibly indicative of an abscess. When compared with the prior MRI, there was no significant interval improvement. The patient was told he would be an appropriate candidate for BVNRFA, although he expressed a preference to continue with medication rather than undergo the procedure. He was treated with baclofen, pregabalin, hydrocodone/acetaminophen, and hydromorphone and was advised to participate in physical therapy. However, during acute rehabilitation, the patient decided to undergo the procedure.

The procedure was performed under sterile conditions and general anesthesia. Preprocedure laboratory values and vital signs showed no evidence of active infection. Using fluoroscopic guidance, a transpedicular approach was used to gain access to the L3 and L4 vertebral bodies. An introducer cannula was advanced through the pedicle into the vertebral body, targeting the BVN. An RF probe was then inserted through the cannula, and ablation was performed at 85°C for 15 minutes. The patient tolerated the procedure well with no periprocedural complications observed. At one-month follow-up,

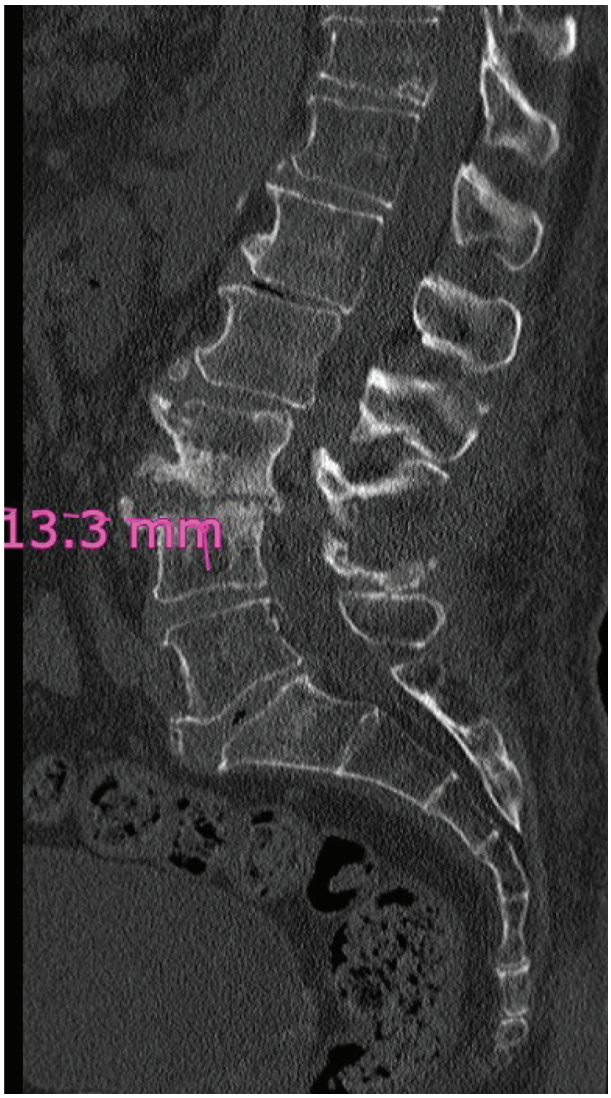

Fig. 1. CT showing evidence of L3-L4 osteomyelitis. CT, computed tomography.

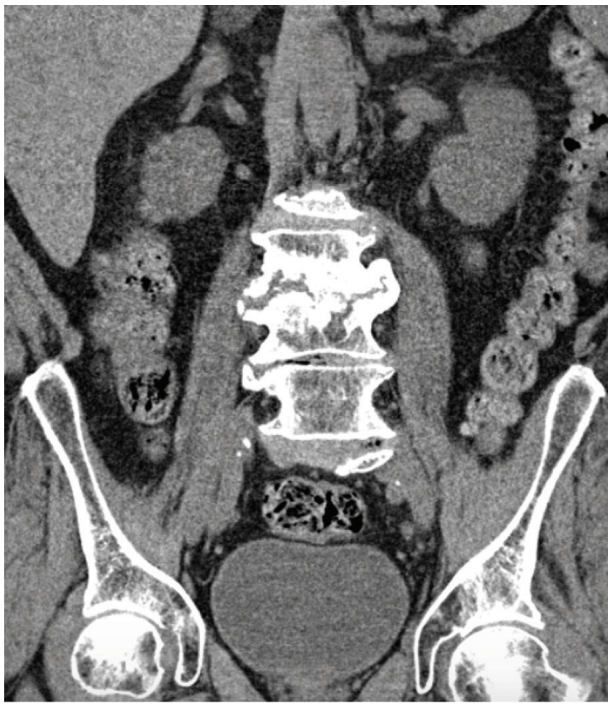

Fig. 2. CT showing involvement of the right psoas muscle. CT, computed tomography.

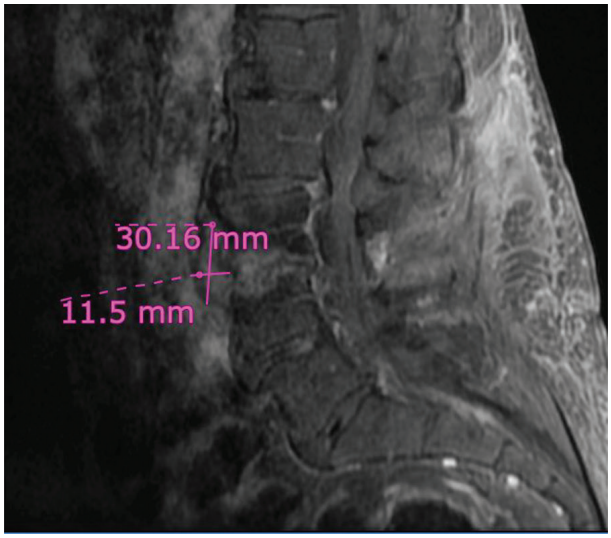

Fig. 3. MRI showing persistent endplate changes. MRI, magnetic resonance imaging.

the patient reported 100% pain relief, and antibiotics will be continued for a total of 6 months.

DISCUSSION

In our case report, we present a novel use of BVNRFA to manage VO. Although BVNRFA is typically used for VBP, its role in other VBP etiologies has been unexplored. To our knowledge, this is the first report detailing the use of BVNRFA for VO, which may help contribute to a greater understanding of VBP pathways. The decision to utilize BVNRFA was driven by the chronic nature of this disease course that has been refractory to medical and antibiotic management. Imaging also showed persistent endplate changes, and the patient wishes to avoid additional pain medication manage-

ment. Given these variables, BVNRFA was chosen as the procedure ablates the nociceptive transmission within the vertebral body, with the impression that it would reduce pain regardless of pathologic etiology, whether degenerative or infectious.

An important safety concern for this procedure with prior osteomyelitis is the risk of reinfection and spinal destabilization of a structurally weak bone. However, our case was performed only after the completion of a full course of antibiotics, stable imaging findings, and no signs of an active infection. No peri- or postprocedural complications were observed, such as increased pain, vertebral body structural collapse, and reinfection. Overall, BVNRFA is associated with low rates of complications. However, reported complications include motor/sensory neuropraxia, radiculopathy, incisional pain, and urinary retention (7). Other general contraindications are active infection, previous spinal surgery at the targeted site, type 3 MCs, pregnancy, incomplete skeletal maturity, preexisting implanted pulse generators, vertebral compression fracture, and retroperitoneal hemorrhage (7).

BVNRFA effectiveness for VBP caused by degenerative endplate pathology with MCs has moderate evidence for reducing pain and improving functional disability (8). Additionally, in a pooled cohort analysis of 3 clinical trials (9) at a 5-year follow-up, there was a clinically and statistically significant reduction in low back pain health care utilization, such as conservative care, opioid usage, lumbar injections, and lumbar RFA. The BVN has also been implicated in endplate pathology aside from degeneration, but also encompasses inflammatory conditions, autoimmune, trauma/microfractures, and low-grade infections (10).

Our case adds to the literature by possibly expanding the indications for BVNRFA for a postinfectious VBP etiology. By targeting the underlying pain generator rather than systemic analgesics, BVNRFA can potentially reduce pain, opioid burden, and improve function. Most importantly, careful patient selection, as for other use cases of BVNRFA, is paramount to successful outcomes. For example, it is important to highlight that BVNRFA treatment should not be utilized for early/active osteomyelitis, in the absence of antimicrobial treatment, or active infections, such as an epidural abscess. While this procedure was successful and safe for the management of VO in this case, a single case report's results must be interpreted with caution. Repeat imaging was not performed due to sustained clinical improvement, but future studies would benefit from interval imaging to directly assess vertebral stability. Furthermore, although BVNRFA creates a localized thermal lesion with a low risk of affecting structural integrity, there is a theoretical concern that the ablated tissue could provide a nidus for recurrent infection. However, successfully treated osteomyelitis is typically associated with remodeling and a sclerotic phase, which would lead to improved bone density. Imaging follow-up would help validate this safety concern in a postinfectious setting. Larger studies and longitudinal data are required to clarify the duration of pain reduction, safety profile, and impact on vertebral biomechanics in an infectious rather than degenerative setting. Additionally, it is unclear which patients with VO are likely to benefit from BVNRFA or if different patterns of endplate changes are contraindications.

CONCLUSIONS

BVNRFA may be a promising strategy for the management of VO when persistent endplate changes are seen on MRI and without signs of an acute infection. Our case adds to the growing literature on VBP and the need for further investigation of the usage of BVNRFA outside of traditional VBP degenerative etiologies with MCs.

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