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Comprehensive Review for the Treatment of Multilevel Thoracic Compression Pain with a Case Report Demonstrating Use of Peripheral Stimulation

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Background: The pain, compromised spinal biomechanics, and limited mobility caused by thoracic vertebral compression fractures present complex clinical challenges. Conventional treatments for this condition have limitations, necessitating innovative solutions. Peripheral nerve stimulation (PNS), which interrupts pain-signaling pathways, offers a minimally invasive approach.

Case Report: We report a 20-year-old woman with T9 and T7 thoracic compression fractures resulting from a horse-riding accident. Although she had received the standard interventions, her pain persisted. PNS was introduced, resulting in an 85% pain reduction and improved quality of life for the patient. PNS utilizes neuromodulation principles to target peripheral nerves, intercepting nociceptive signals.

Conclusions: This case highlights PNS's potential as a transformative therapeutic strategy for thoracic compression fractures. PNS offers personalized pain relief with minimal invasiveness, making it a promising alternative to conventional treatments. As the prevalence of osteoporosis rises, PNS holds promise for better outcomes and enhanced quality of life in patients with vertebral compression fractures.

Key words: Peripheral nerve stimulation (PNS), thoracic compression fracture, vertebral integrity, neuromodulation, pain management, case report

BACKGROUND

Each year, many people suffer from vertebral compression fractures, which present complex clinical challenges. Located in a critical anatomical area, these fractures not only jeopardize the spine's structural integrity but also impact neural function (1). Beyond the intense pain they cause, vertebral compression fractures can lead to a range of issues, from compromised thoracic capacity that diminishes lung function to symptoms of neurogenic claudication (1,2). Vertebral compression fractures often result from conditions that weaken the bone, most commonly osteoporosis. Other causes can include trauma, certain types of cancers that have spread to the bone, and long-term use of certain medications, such as corticosteroids (3). Because of the aging global population and the increasing prevalence of osteoporosis, the incidence of vertebral compression fractures is anticipated to rise (1).

When the structural integrity of the thoracic spine is compromised, a series of biomechanical alterations

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may ensue, altering load distribution across the spinal column (4). This sequence of events can increase the risk of additional spinal injuries and contribute to a chronic cycle of pain and disability. Additionally, the proximity of thoracic vertebral fractures to vital organs means that such injuries can have far-reaching systemic effects, such as reduced pulmonary function due to compromised thoracic volume and diaphragmatic movement. Compromised neurovascular structures can be downstream effects of thoracic compression fractures and may lead to neurological deficits ranging from mild radiculopathies to severe myelopathies (4).

The thoracolumbar transition zone represents a pivotal area in spinal biomechanics, bridging the relatively immobile thoracic spine with the more mobile lumbar spine (4). This zone, traditionally defined anatomically at T12-L1, is subject to a unique distribution of mechanical stresses caused by the abrupt shift in the orientation of the facet joints and spinal curvature. The T12-L2 vertebrae situated at the critical junction of the transition zone bear the combined stresses of both the thoracic kyphotic curve and the lordotic curvature of the lumbar segment (1). This position subjects these vertebrae to heightened strain, increasing their vulnerability to fractures (1,5,6). Evidence suggests that the true functional thoracolumbar junction may be located in a higher area, around T10-11, where a transition from floating to false ribs occurs, introducing increased mobility and susceptibility to degenerative changes (4).

A retrospective MRI review and analysis of segmental loads from T8-9 to L1-2 indicate that the thoracolumbar transition zone's functional and mechanical demands are not confined solely to the anatomical junction of T12-L1 (4). The increased prevalence of disc degeneration and higher mean load gradients observed at T9-11 as compared to T11-L1 supports the hypothesis that the true biomechanical thoracolumbar junction is indeed at T10-11. This higher transition point aligns with the location of the floating ribs' transition to false ones, reflecting a shift in the rib cage's contribution to spinal stability. The ribs' articulation with the spine plays a significant role in this region, where the true ribs attach directly to the sternum, providing stability, whereas the floating ribs do not, resulting in a relative increase in spinal mobility and load stress. This finding has implications for clinical practice, suggesting that assessments and interventions for lower back pain and related spinal conditions should consider this higher thoracolumbar transition zone to better address the pathophysiology of disc degeneration and herniation observed in this region (4).

Detailed morphological assessments categorize compression fractures based on the predominantly affected segment of the vertebrae (6). The "vertebral plana" or colloquially termed "pancake" fracture represents a distinct variant of vertebral compression fractures, characterized by a reduction of > 70% in anterior vertebral height relative to its posterior counterpart. Such distinctions present specific challenges in therapeutic approaches to vertebral compression fracture treatments (7). Contemporary therapeutic paradigms encompass a spectrum of interventions. The noninvasive spectrum ranges from pharmacological agents that modulate pain pathways to physical therapeutic modalities that optimize spinal biomechanics. Primary interventions for thoracic compression fractures aim to alleviate pain, stabilize fractures, and thwart further complications (1,8). Common approaches encompass rest periods for natural healing, analgesics for pain management, back braces for support, and physical therapy for muscle strength and mobility enhancement (9).

Nevertheless, in instances of persistent pain or increased risk of injury, more invasive procedures like vertebroplasty or kyphoplasty might be considered. Both are centered on using bone cement to reinforce the compromised vertebra (7). In vertebroplasty, the bone cement known as polymethylmethacrylate is administered directly into the broken vertebral body by way of a needle, which is usually introduced percutaneously through the spinal pedicle (10). Kyphoplasty, meanwhile, uses a specialized balloon that is inserted and inflated within the vertebral body to restore its height and create space for the cement prior to its injection (7,10). Vertebroplasty, however, is not typically the preferred treatment due to potential complications associated with transpedicular access to the vertebral body (1,7). A notable concern is the unintended migration of bone cement into the spinal canal, which can lead to spinal cord compression (7). Additionally, vertebroplasty introduces a heightened risk of fractures in the adjacent vertebrae, both above and below the augmented site. Moreover, current literature presents conflicting evidence regarding the long-term efficacy of vertebral augmentation, with some studies indicating pain relief durations ranging from a month to up to 3 years (1,7).

In the interest of meeting the inherent therapeutic challenges associated with treating vertebral compression fractures, especially of the plana subtype, this case

study introduces the innovative approach of peripheral nerve stimulation (PNS) as a viable alternative to more conventional methods. Rooted in the principles of neuromodulation, PNS operates based on the foundational concepts of the gate control theory (11). This theory posits that nonpainful input can effectively "close the gates" to painful input, preventing pain sensations from traveling to the central nervous system (CNS) (11,12). PNS's mode of action in this context is multifaceted and posited to work through what is termed "peripherally induced reconditioning of the [CNS]" (13), suggesting that PNS not only blocks or modulates pain signals at the spinal level but may also induce lasting changes in the central processing of pain, which can sustain pain relief well after the stimulation period.

One of the key objectives of PNS is to induce paresthesia in painful areas. Paresthesia refers to abnormal sensations, such as tingling, numbness, or "pins and needles," often associated with nerve-related conditions (14). By stimulating specific peripheral nerves to generate paresthesia, PNS can effectively override or mask the sensation of pain, providing substantial relief. In the context of PNS, regular tonic stimulation refers to the consistent and continuous delivery of electrical pulses to peripheral nerves (15).

PNS is described as engaging selective large-diameter afferent fibers, which are associated with non-nociceptive sensory input. By activating these fibers, PNS may counteract the changes associated with chronic pain at both the peripheral and central levels (16). In the periphery, PNS may help to calm the hyperexcitability of nociceptive afferents, thus reducing the spontaneous discharge that contributes to pain. Centrally, persistent PNS has the potential to induce plastic changes within the spinal cord and brain (17). By increasing the input from large-diameter fibers, PNS may assist in recalibrating the sensitization of neurons in the spinal cord's dorsal horn. This recalibration can normalize the heightened excitability and reduce the excessive signaling that characterizes chronic pain states (13).

Beyond the spinal cord, the robust stimulation provided by PNS is thought to influence cortical plasticity (13). By selectively activating many afferent fibers in a targeted manner, PNS may drive activity-dependent cortical remapping. This process could potentially reverse maladaptive plastic changes that chronic pain has made to the somatosensory cortex, thereby restoring a more normal balance of sensory processing and alleviating pain in the long term (18). Furthermore, PNS is hypothesized to engage descending inhibitory pathways, which can further modulate pain processing at the spinal level. In this way, PNS may enhance natural inhibitory control over pain signals, contributing to a sustained analgesic effect (16). PNS operates not just by modulating signals at the gate level but also by actively reconditioning the CNS to reverse the central features of chronic pain. The reconditioning involves a reduction of peripheral sensitization, modulation of spinal cord neurons, induction of plasticity in the brain, and engagement of descending pain control pathways, leading to a comprehensive, sustained decrease in pain (13). This targeted approach not only offers potential pain relief but does so in a manner that can minimize the side effects and risks associated with more invasive treatments (19).

The multifaceted nature of thoracic vertebral compression fractures necessitates a multidisciplinary approach to treatment, integrating pain management, physical rehabilitation, and sometimes surgical intervention. This complexity underscores the need for innovative therapies, such as PNS, which offer a noninvasive yet effective modality to address the intricate challenges posed by these fractures (7). By targeting the peripheral nerves and modulating the pain signals before they escalate within the central nervous system, PNS presents a promising frontier in the management of vertebral compression fractures and their extensive clinical sequelae. Our case examines a 20-year-old woman who had thoracic compression fractures at T9 and T7 and was successfully treated with PNS. Emphasizing the transformative impact of PNS, this case offers a nuanced perspective into the potential of neuromodulation as a revolutionary therapeutic strategy for treating vertebral compression fractures. Informed consent was obtained from the patient.

CASE PRESENTATION

A 20-year-old woman with a medical history of generalized anxiety disorder, obsessive-compulsive disorder, post-laparoscopic cholecystectomy, and an ovarian cyst presented with thoracic pain radiating around her rib cage. This pain resulted from a horse-riding incident in which her horse halted abruptly at an obstacle, causing her to twist to the side without falling. On physical examination, the patient appeared to be a healthy-looking woman who was not in distress and was alert and oriented to time, place, and person. Her vitals remained stable throughout examination, and the patient noted

she was a nondrinker and nonsmoker. Labs revealed that her levels of parathyroid hormone (PTH) and calcium remained intact. However, her 25-hydroxy vitamin D level was low, so she was given a vitamin D supplement.

The patient's upper-to-mid-thoracic region experienced tenderness upon palpation. Her range of motion was unaffected, however, and her bilateral straight leg raise (SLR) test result was negative. Muscle strength, pulses, and reflexes were intact in both her upper and lower extremities. The patient underwent a thoracic MRI, which identified recent compression fractures at the T9 and T11 vertebral bodies, resulting in approximately 20% vertebral body height reduction. The most pronounced changes were observed at T9. Lateral thoracic spine x-ray imaging further highlighted multilevel spondylosis and degenerative disc changes, with no evidence of tumors, fractures, or masses. Notably, there was no sign of retropulsion or spinal canal stenosis. Rib fractures were also excluded from the findings.

The patient had been receiving the Depo-Provera shot for approximately 5 years, leading to progressive bone density loss, eventual osteoporosis, and early bone degeneration. This degeneration was implicated in the T9 and T11 compression fractures. Initial treatment for the fractures included a CASH brace, calcium and vitamin D supplements, and physical therapy. Her pain was managed using over-the-counter NSAIDs, methocarbamol (Robaxin), and gabapentin. From a pain management viewpoint, she initially benefited from bilateral T9-T11 thoracic facet blocks, both diagnostic and confirmatory, which led to a left-side thoracic rhizotomy from T9 to T11. However, her pain persisted, and a subsequent right-side thoracic rhizotomy from T9 to T11 provided relief for about 2 months.

Four months after a right-side rhizotomy, which provided limited relief after facet joint injections, the patient reported an 85% pain reduction from a PNS trial. We proceeded to implant Abbott's Eterna™ (32400) rechargeable pulse generator, an advanced spinal cord stimulator, which the FDA approved for chronic pain treatment in 2022. For thoracic compression fractures at T9 and T11, the nerves chosen for peripheral stimulation were the corresponding thoracic spinal nerves, particularly the intercostal nerves at these levels. During her trial period with this pulse generator, our patient reported a marked improvement in her daily activities.

To optimize the effectiveness of PNS, several parameters are crucial. In PNS, a frequency of approximately 30 Hz is often used, meaning that 30 electrical pulses are administered to the nerves every second. Pulse width, or pulse duration, defines the duration of each electrical pulse. A pulse width of 250 microseconds was used, and the amplitude (mA) was adjusted based on the patient's tolerance and the desired therapeutic effect. Leads one and 2 were positioned on the left side, in alignment with the patient's pain site (Fig. 1). Lead one is situated midline on the left, while lead 2 is located approximately 10 centimeters apart to the left.

CONCLUSIONS

Vertebral compression fractures, particularly those that occur in the thoracic region, present well-documented complex clinical challenges that demand innovative therapeutic interventions (2,3). While the array of available treatments for the condition ranges from pharmaceutical drugs to invasive procedures, there persists a pressing need for solutions that are both effective and minimally invasive (2,3). This case serves as a testament to the potential of PNS in bridging this therapeutic gap.

The patient, who had experienced thoracic compression fractures because of a horse-riding incident, underwent a range of standard interventions, including pharmaceutical analgesics and thoracic rhizotomies, all of which provided temporary relief. Significantly, the patient's prolonged use of the Depo-Provera shot was found to have contributed to osteoporosis, thus increasing her susceptibility to fractures. This phenomenon has been well-documented in the literature (20-22). Her underlying condition, coupled with the traumatic incident, ultimately led to the development of thoracic compression fractures. Since receiving PNS, the patient has transitioned to a contraceptive option sold under the brand name Yaz, which combines an estrogen component (ethinyl estradiol) with drospirenone.

Nociceptive pain arises from actual or threatened damage to non-neural tissue and involves the activation of nociceptors. The pain is typically acute, often described as sharp, aching, or throbbing, and is associated with tissue injury and inflammation, such as from fractures or sprains (23). Neuropathic pain, by contrast, is chronic and arises from direct injury to nerves. This type of pain is often described as burning, shooting, or similar to an electric shock. Understanding the underlying mechanisms of these pain types is crucial for effective treatment (24). In this patient's case, the nociceptive pain is due to the compression fractures and the associated tissue injury.

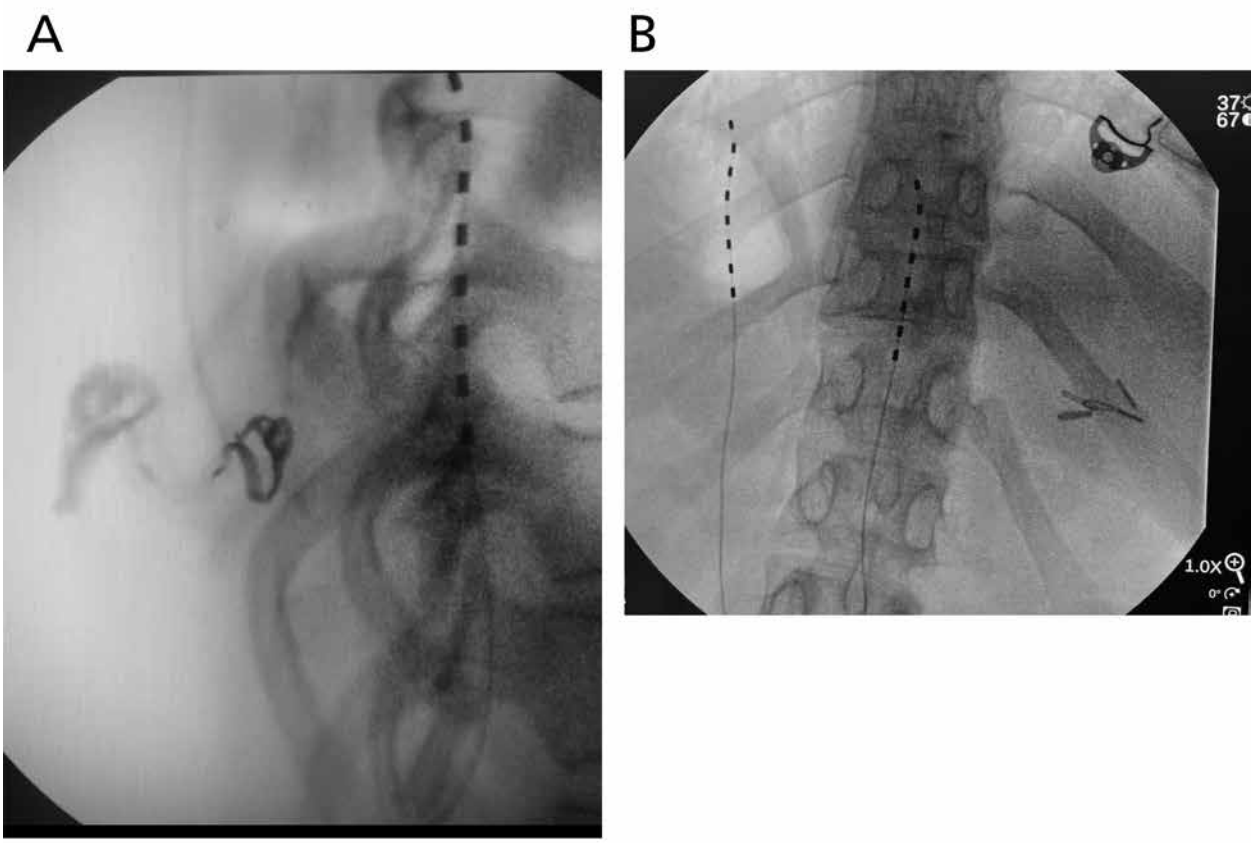

Fig. 1. A: Lateral fluoroscopic view of percutaneous peripheral nerve stimulator implants. B: AP view of percutaneous peripheral nerve stimulator implants.

The patient presented with thoracic pain caused by compression fractures that occurred during a horse-riding incident. The pain was likely nociceptive initially, due to the mechanical trauma and subsequent inflammation. The initial success in managing the patient's pain with NSAIDs and muscle relaxants supports the notion that the pain was primarily nociceptive. The use of gabapentin, which is typically used for neuropathic pain, may be indicative of a mixed pain type. The patient's response to thoracic facet blocks and rhizotomies also suggests an inflammatory component of nociceptive pain that was well managed by interventions that targeted the sensory pathways associated with the vertebral injuries. The use of a CASH brace also aligns with the management of nociceptive pain, since the brace stabilizes the physical injury. However, the patient's continued pain and partial response to rhizotomies suggest a complex pain syndrome that may have both nociceptive and neuropathic elements. Nociceptive pain can develop into a chronic condition and be accompanied by neuropathic pain, especially when nerve damage or a significant alteration in pain-signaling pathways is involved (24).

The patient was educated on her treatment options, which included interlaminar epidural steroid injections (ITESIs), kyphoplasty, and PNS. ITESIs are a treatment modality commonly used for radicular pain, which is often associated with inflammation and nerve root irritation due to conditions such as herniated discs and spinal stenosis (25). Kyphoplasty is a minimally invasive procedure that aims to achieve pain relief and spinal stabilization by inserting a balloon into the compressed vertebra and then filling the cavity with bone cement (26). The decision to pursue PNS over ITESIs or kyphoplasty was multifactorial and patient centered.

Because of the patient's age, the goal was to minimize long-term risks and prioritize interventions with the lowest potential for adverse effects. ITESIs, while effec-

tive for radicular pain, were deemed inappropriate due to the absence of radiculopathy or nerve root compression. Moreover, the potential side effects of ITESIs, such as steroid-related systemic effects and rare but serious complications like infection or dural puncture, were considered significant, especially given the patient's otherwise stable condition (27).

Kyphoplasty, although useful in certain cases of vertebral compression fractures for alleviating pain and restoring vertebral height, also carries risks, particularly in young patients with osteoporosis. The possibility of post-kyphoplasty adjacent segment fracture and concerns about the long-term durability of the procedure in osteoporotic vertebrae influenced the decision-making process in this case (28,29). Additionally, the patient's vertebral fractures, with only a 20% reduction in height and absence of spinal instability, did not reach the threshold at which the benefits of kyphoplasty would clearly outweigh the risks (30).

PNS presented a minimally invasive, reversible option with a low side effect profile. The treatment method also offered the advantage of targeting the pain pathway directly without the systemic effects of steroids or the structural risks associated with kyphoplasty. The patient's active participation in choosing PNS, informed by a thorough discussion of the risks and benefits of all options, exemplified patient-centered care. Her choice reflects a shared decision-making process that prioritizes personal values and preferences, particularly the patient's desire for a nonpharmacological, minimally invasive approach to pain management.

The substantial pain relief achieved suggests that modulating the peripheral nociceptive signals was effective, despite the pain's nociceptive origin. This result underscores the importance of a multimodal approach to pain management, particularly in complex cases in which the pain may not fit neatly into one category. The transition to PNS therapy, which resulted in an 85% reduction in pain, indicates that while the patient's pain was nociceptive initially, the chronicity and severity of her condition necessitated a broader approach that addressed both nociceptive and potential neuropathic elements.

PNS represents a transformative step in pain management by leveraging neuromodulation principles that extend beyond the traditional gate control theory (7,11-13,19). By targeting peripheral nerves precisely, PNS modulates nociceptive signals, potentially offering targeted pain relief that persists even after the stimulation period has ended. PNS has been shown to induce plastic changes within the spinal cord and brain, altering the central processing of pain to achieve long-term relief. The capacity of PNS to activate large-diameter afferent fibers selectively may counteract the changes associated with chronic pain at both the peripheral and central levels, reducing hyperexcitability and recalibrating the sensitization of neurons (7,11-13,19). This approach is highly customizable and adaptable to the unique needs and tolerance levels of individual patients, optimizing pain relief while minimizing discomfort and the risk of side effects commonly associated with more invasive procedures.

In the context of this patient, the trial of PNS yielded transformative results. The 85% reduction in pain meant that the patient experienced a substantial enhancement in her quality of life, underscoring the profound impact of PNS. It should be noted that while this patient's distinct medical history and the series of events leading to the fractures she received created a unique case, the overarching efficacy of PNS holds promise for a broader range of patients dealing with vertebral compression fractures.

This case study illustrates the complexities of treating thoracic vertebral compression fractures, especially in patients with osteoporosis and traumatic injuries. Although traditional treatments offer limited relief, PNS emerges as a promising alternative. In the case described above, PNS significantly reduced a patient's pain and improved her quality of life. The personalized and minimally invasive nature of PNS makes it a compelling option for vertebral compression fracture patients. While this case is unique, PNS holds broad potential for pain management and functional improvement in similar cases. In view of the rising prevalence of osteoporosis and the challenges posed by vertebral compression fractures, PNS represents an innovative and customizable strategy, offering hope for better outcomes and improved quality of life for future patients.

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