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A Rare Calcified Complication of Epidural Injections for Lumbar Spinal Stenosis: A Case Presentation and Literature Review

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Background: Neurogenic claudication secondary to degenerative lumbar stenosis is typically managed with nonsurgical options, such as epidural corticosteroid injections. As a standard and effective treatment for lumbar stenosis, clinicians must be aware of the corticosteroids choice when injecting in the epidural space.

Case Report: A 62-year-old man presenting with sciatic pain is treated with multiple neuroforaminal, facet, and caudal corticoid injections over the course of several months without any symptomatic resolution. A magnetic resonance imaging of his lumbar spine revealed focal bilateral central stenosis at the L4-L5 level. A computed tomography revealed hyperdense lesions at that level. The patient was referred for a surgical option. He underwent complete minimally invasive resection of the bilateral lesion with instrumented and interbody fusion. The final pathology report identified the mass as a calcified granuloma.

Conclusions: Following repetitive methylprednisolone acetate injections, one must be aware of all the potential complications arising from particulate corticosteroids.

Key words: Spinal stenosis, epidural injections, granulomas, corticosteroids

BACKGROUND

Degeneration of the lumbar spine can present with various pathologies of the spine, such as facet hypertrophy, disc bulging, and ligamentum hypertrophy (1,2). This leads to compression of the thecal sac and, as such, central canal and lateral recess stenosis. Generally, neuropathic pain is secondary to the irritation of one or multiple nerve roots, while somatic pain can be the consequence of discogenic or zygapophyseal degeneration. Possible etiologies for neuropathic pain presentations can be secondary to any stenosing process.

The pathophysiology behind symptomatic spinal stenosis (SS) is found within degenerative disc pathology (1). The zygapophyseal joints and the ligamentum flavum will consequently hypertrophy and gradually ankylose in response to the pathological stress. Remodeling of these structures will invade the canal and gradually reduce the intraspinal space.

Patients suffering from lumbar SS present with symptoms of progressive neurogenic claudication in both lower limbs that is brought about by walking or during active lumbar extension. The symptoms disappear rather rapidly when the patient stops walking or adopts positions to increase the central spinal canal (i.e., lumbar flexion). These symptoms can be debilitating for patients and have a functional negative impact in their activities of daily living (2). On physical examination, neurological evaluation might be initially unremarkable in early stages of the disease. As the disease progresses, objective sensorimotor deficits can be present (3,4).

Treatment of symptomatic lumbar SS follows a certain

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continuum of care. Initially, conservative treatment is employed. Physical therapy yields favorable results in milder cases of lumbar SS (5). Before resorting to surgical interventions, epidural injections with corticosteroids have also demonstrated their effectiveness. In a systematic review, Manchikanti et al (6) evaluated the effectiveness in addressing SS with interlaminar, transforaminal, or caudal injections. Relief could be present for up to 12 months based on their findings following the usage of corticosteroids combined with lidocaine. Lastly, surgical interventions are utilized when nonsurgical options have been unsuccessful.

Targeted injections of corticosteroids have become a mainstay treatment approach in recent years. Utilizing fluoroscopy-guided techniques, clinicians can precisely deposit corticoid injectates around nociceptive and neural structures. While every procedure exposes the patient to potential harmful effects, the use of corticosteroids yields relatively low-risk adverse effects. Adverse effects can present over a vast time frame: headaches, facial flushing, insomnia, decreased bone density, suppression of the hypothalamic-pituitary-adrenal axis, immunosuppression, mania or psychosis, uterine bleeding, and increase in blood pressure and glycemic levels (7-10). Multiple studies (11,12) have shown that particulate injectates will aggregate intravascularly and cause tissue infarction following distal vessel embolization. There also exists severe and possibly lethal complications, such as epidural abscess or hematomas, cerebral or cerebellar infarcts, ischemic myelopathy, and death (13-15).

CASE PRESENTATION

A healthy 62-year-old man with no prior past medical history presents to the ambulatory neurosurgery clinic with symptoms of predominant right-sided neurogenic claudication for the past 2 years. He had marked standing and walking limitations of only a few minutes, which brought on numbness, as well as heaviness, in both of his lower extremities. Furthermore, these symptoms were relieved by lumbar flexion, such as leaning on a grocery cart. He had undergone conservative treatment, which included physiotherapy, as well as various repeated epidural injections of methylprednisolone acetate. On exam, his gait was normal. He was able to walk on his toes and heel without any difficulties. The Trendelenburg test was negative. There were no positive nerve root tension signs, nor any weakness or any paresthesia were noted. His reflexes were within normal limits. An electromyogram study did not reveal any acute nor chronic denervation.

Prior to the first epidural injection, the patient had a lumbar magnetic resonance imaging (MRI) done in February 2020 (Fig. 1). A moderate-to-severe SS at L4-L5 secondary to degenerative disc disease, ligamentum flavum, and bilateral articular facet hypertrophy was identified. In March 2020, he underwent an L5 and S1 right-sided epidural foraminal injection with 1 mL of dexamethasone 10 mg/mL and 1 mL of xylocaine 1% per level, as well as bilateral L4-L5 and L5-S1 intraarticular facet joint injection with 0.5 mL of triamcinolone acetonide 40 mg/mL and 0.5 mL of xylocaine 1% per facet. There was minimal improvement in his low back pain and claudication symptoms. In April 2020, he received an epidural caudal injection with 2 mL of methylprednisolone acetate 40 mg/mL, 2 mL of xylocaine 2% and 10 mL of sodium chloride 0.9% with no relief either. In June 2020, a repeat right-sided epidural foraminal injection of L5 and S1, as well as L4-L5 and L5-S1 intraarticular facet joint injections, were performed with the same posology on March 2020 and with temporary relief of his symptoms.

Following a subjective decrease of his walking endurance, an MRI of his lumbar spine was performed in September 2020. The report described a focal severe SS at the L4-L5 level due to facet and ligamentum flavum hypertrophy, as well as a degenerative bulging disc. A narrowed congenital canal with short pedicles was also noted. There is minimal cerebrospinal fluid noted (T2 signal) within the thecal sac at that level (Fig. 2). Moreover, a computed tomography (CT) scan performed, in November 2020, revealed short pedicles, in the context of a congenitally narrowed canal. However, there was a bilateral hyperdense lesion causing severe SS at that level (Fig. 3). This intraspinal mass described on the CT scan was not noted on the initial MRI.

In the context of a congenital narrowed canal, short pedicles, bilateral calcified lesions, and facet hypertrophy, the patient underwent a minimally invasive bilateral decompression and transforaminal interbody instrumented fusion. There were no intraoperative complications. At 6 months follow-up, there was complete resolution of his claudication, as well as back pain symptoms. He had no limitation of walking or standing tolerance. Postoperative radiograph showed that there was fusion at the instrumented level, with no hardware complications. Finally, the macroscopic pathology evaluation revealed the lesions to be dystrophic calcifications.

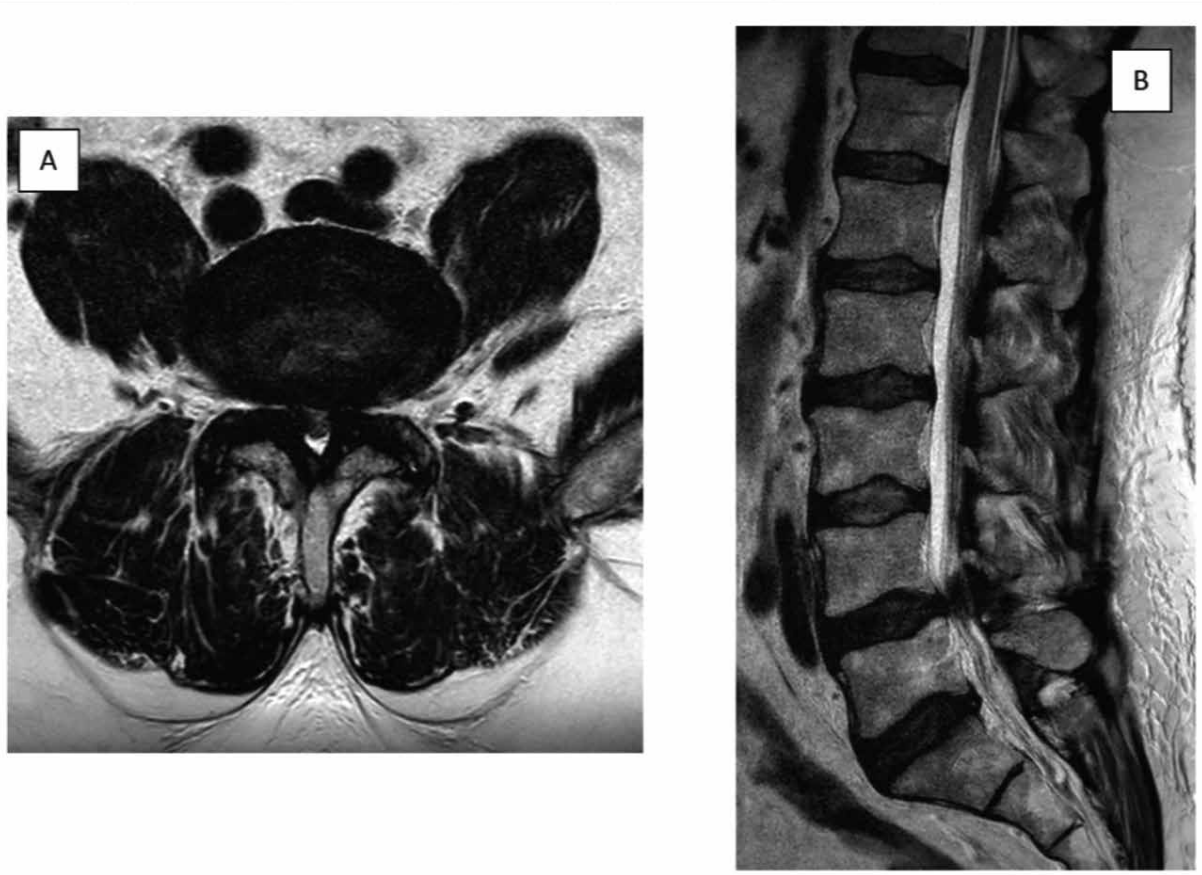

Fig. 1. A. Axial T2 MRI demonstrating bilateral central stenosis at the L4-L5 level (February 2020). B. Sagittal T2 MRI demonstrating the well-defined lesion at the L4-L5 disc space (February 2020).
MRI, magnetic resonance imaging.

DISCUSSION

Here we present a classic case of lumbar stenosis where a patient presents with neurogenic claudication. This study conforms to all CARE guidelines and reports the required information accordingly. As per the North American Spine Society recommendations, the patient was treated with a regiment of conservative treatment, including physical therapy as well as epidural injections. However, there was a failure of the latter. As such, he underwent an MRI that revealed a single level L4-L5 bilateral central stenosis, which on a CT scan revealed a hyperdense lesion. A pathology report confirmed this to be a granuloma. To the best of our knowledge, this is the first report of the formation of calcified granuloma following epidural injection for the treatment of lumbar stenosis.

Commonly reported etiologies for epidural granulomas include postcatheter placement, inflammatory pseudotumor, sarcoidosis, and infectious etiology (16-20). Iatrogenic epidural hematomas are potential complications following epidural injections, and while the calcifications of hematomas can occur, this phenomena remains a rare entity (21). Lastly, calcifications following intradiscal injections of triamcinolone with symptomatic intraspinal expansions have been described previously in multiple case reports (22,23). However, the formation of the calcifications does not resemble the one described in the case report; the pathology report described a necrotic granulomatous lesion. In that report, the calcifications originated from the intervertebral discs and continued their course in the neural canal (23).

Presently, there is one case in the literature describing calcifications following epidural injection of triamcinolone acetonide. However, there are no case reports describing similar complications following epidural methylprednisolone acetate injections.

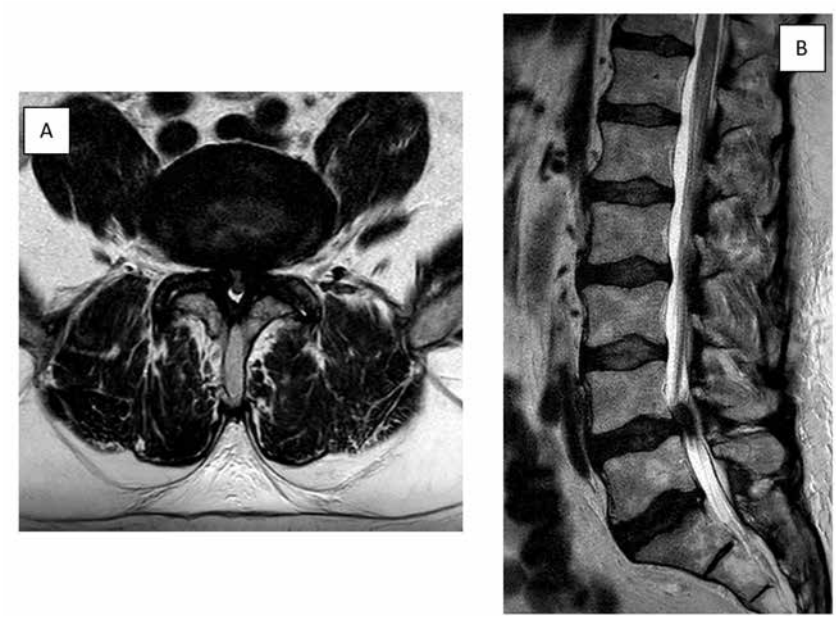

Fig. 2. A. Axial T2 MRI demonstrating a progression in central stenosis at the L4-L5 level (September 2020). B. Sagittal T2 MRI demonstrating the well-defined lesion at the L4-L5 disc space (September 2020).
MRI, magnetic resonance imaging.

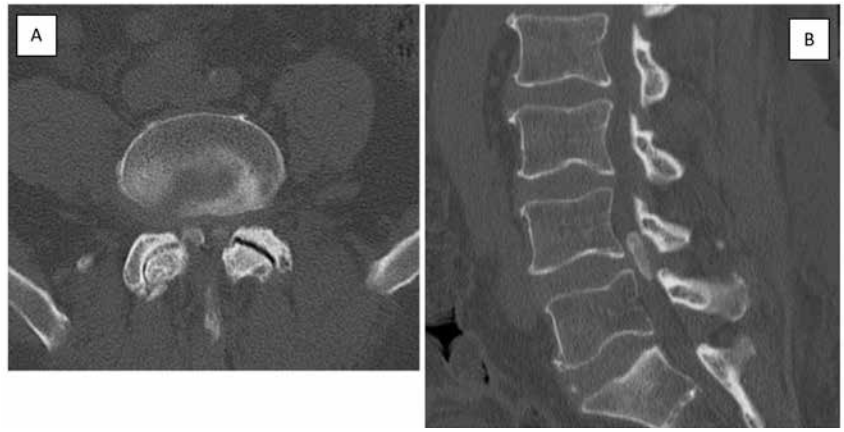

Fig. 3. A. Axial CT scan showing the hyperdense lesion at the L4-L5 level with facet arthropathy (November 2020). B. Parasagittal CT scan demonstrating the hyperdense lesion (November 2020).
CT, computed tomography.

The sole case, published in 2011 by Jin et al (23), goes over the formation of a dystrophic epidural calcification in a 66-year-old patient having received 10 epidural injections of triamcinolone acetonide in the span of 6 months at the level of L4-L5. The patient demonstrated bilateral motor weakness in her lower limbs. An MRI and CT scan revealed severe central SS at the level of L4-L5. A decompressive laminectomy was performed, and salt-like particles were observed at the level of the lesion. Similar findings were found within the left L4-L5 foramina where the patient had received the epidural injections. The pathological report demonstrated degenerated cartilage and bone with dystrophic calcification and the absence of an inflammatory reaction surrounding the calcified deposit (23).

Pathological inflammation, amongst other processes, is a root cause of symptomatic SS (24). It has been shown that various enzymes and mediators, such as prostaglandins and phospholipids, sustain proinflammatory activity in epidural spaces. Corticosteroids are utilized to treat the root causes of the presenting pains due to their potent anti-inflammatory properties (25). There are 2 types of injectable corticosteroids: particulate and nonparticulate (29). Particulate, or insoluble preparations, contain esters and have a delayed effect as the active particles are gradually released following hydrolysis by cellular esterase. The soluble nonparticulate corticosteroids are readily available and act instantly. Studies were conducted to assess the magnitude of particulate corticosteroid aggregation (26,28). An observational study (14) evaluated the size of particulate aggregates relative to red blood cells in the context of arterial embolization. For example, methylprednisolone acetate particles are smaller than erythrocytes, but have a greater propensity to be densely packed.

To our knowledge, no studies currently have investigated what constitutes these aggregates, nor what could arise from them in the epidural space. However, an article by Conti et al (26) hypothesized that the particulate corticosteroids within soft tissue can induce a calcified granuloma as the insoluble injectate can act as a foreign body. This hypothesis, coupled with the notion that insoluble particles remain present at the site of the injectate for multiple weeks before being completely absorbed, might explain why the patient developed such pathology (22,23,26). This notion has been addressed by Aldrete (27) where the triamcinolone injectate could potentially remain in the epidural compartment for up to 6 weeks if there are no breach and leakage in the dural sac. We hypothesize that the granuloma formation could be secondary to a type IV hypersensitivity reaction in the epidural space. Combined with an irritant, such as the trauma caused by the needle during the injection, and the high dose of methylprednisolone acetate injected, a local immune reaction might occur. Moreover, Hwang et al (28) have demonstrated the increase crystal precipitation following mixes between local anesthetics and corticosteroids. Considering the large size of methylprednisolone acetate particles, its mixture with lidocaine and saline in the caudal preparation might have increased the propensity for granular formation (11,28). The epidural caudal approach demonstrated a spread covering the epidural space bilaterally, offering a potential explanation behind the localization of the lesion (Fig. 4).

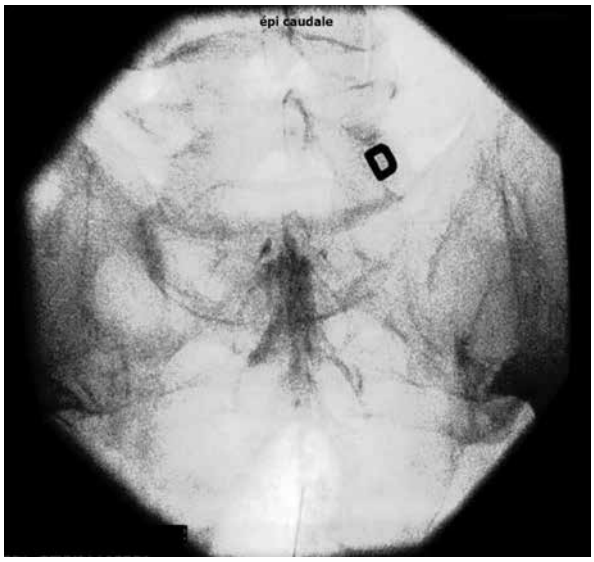

Fig. 4. Fluoroscopic AP view of the contrast spread following a caudal epidural injection.
AP, anteroposterior.

Considering this rare complication, clinicians must review their choice of corticosteroids in epidural injections. Ultimately, it would be judicious to favor nonparticulate corticosteroids as both types of corticosteroids are equally efficacious in reducing lumbar radicular pain and offer fewer fatal complications (7,11,29). Guidelines have stressed the potential dangers in utilizing particulate corticosteroids in epidural spaces. The risk of catastrophic outcomes is significantly greater in cervical and lumbar levels with the use of particulate corticosteroids. However, the Spine Intervention Society states that no evidence of superior safety between either corticosteroids has been demonstrated in the literature in caudal epidural approaches (30).

CONCLUSIONS

To the best of our knowledge, this present case is the only one described in the literature wherein an epidural injection of methylprednisolone acetate led to the formation of a symptomatic calcified granuloma. An argument can be made against using particulate steroids in the epidural space considering its lack of superiority of nonparticulate steroids in the efficacy of pain relief; however, this goes beyond the scope of this article. Clinicians must be aware of the rare complication of this injectate in the epidural space. Further clinical trials and long-term follow-up may help in determining the exact cause and clinical outcomes of both types of infiltrations.

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