PAIN MEDICINE CASE REPORTS

ISSN 2768-5152

Volume 9, Number 1, pp. 59-62

A Common Complaint With an Uncommon Etiology: A Case Report of Back Pain Caused by Paravertebral Compartment Syndrome

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Background: Patients presenting with acute low back pain (LBP) require prompt evaluation, diagnosis, and management to prevent permanent complications. Up to 10% to 15% of patients with LBP have been shown to display neurologic deficits that could lead to downstream complications, such as lower extremity paralysis, numbness, and neurogenic bowel and bladder.

Case Report: The authors present a 44-year-old man who arrived at our emergency room after a drug overdose. He endorsed severe left-sided back pain, a heavy sensation in his bilateral lower extremities, and complete loss of motor and sensory ability from the waist down. Imaging of his thoracic and lumbar spine revealed diffuse edema and expansion of the left paraspinal muscles with postcontrast enhancement, concerning for acute compartment syndrome.

Conclusions: Compartment syndrome of the paraspinal muscles is an extremely rare condition. However, paravertebral compartment syndrome must remain on the differential for practitioners who are presented with acute back pain.

Key words: Back pain, compartment syndrome, paravertebral, rhabdomyolysis, case report

BACKGROUND

Patients presenting with acute low back pain (LBP) require prompt evaluation, diagnosis, and management to prevent permanent complications. Up to 10% to 15% of patients with LBP have been shown to display neurologic deficits that could lead to downstream complications, such as lower extremity paralysis, numbness, and neurogenic bowel and bladder (1). While progressive degenerative spine disease and trauma account for most cases of acute LBP, it is important to identify and recognize other etiologies to determine the course and urgency of treatment. In this case report, the authors examine a case of severe LBP in the setting of paravertebral compartment syndrome, seemingly induced by rhabdomyolysis and opioid overdose.

Acute compartment syndrome (ACS) is defined as "a critical pressure increase within a confined compartmental space causing a decline in the perfusion pressure to the tissue" (2). The most common diagnosis to precede ACS is a tibial diaphyseal fracture, marking about 36% of reported cases (3). Within any kind of fracture, the incidence of ACS can range from 2% to 53%, depending on the location. Other common causes of ACS include crush injury, burns, infection, arterial damage, and any infiltration that can elevate the compartment pressure above the perfusion pressure (2-4). One such infiltration includes myoglobin and creatine kinase (CK), as seen in rhabdomyolysis. Rhabdomyolysis is severe myopathy resulting in muscle death and the release of breakdown products into the bloodstream and surronding structures. Although

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Disclaimer: There was no external funding in the preparation of this manuscript.
Conflict of interest: Each author certifies that he or she, or a member of his or her immediate family, has no commercial association (i.e., consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted manuscript.
Patient consent for publication: Consent obtained directly from patient(s).
This case report adheres to CARE Guidelines and the CARE Checklist has been provided to the journal editor.
Accepted: 2024-10-21, Published: 2025-02-28

classic complications include myalgias, acute kidney failure, and electrolyte abnormalities, other sequelae can produce permanent morbidity (5).

CASE PRESENTATION

The authors present a 44-year-old man with a significant past medical history of proximal common femoral artery thrombus, polysubstance use disorder, seizures, amputation of digits 2 through 5 of the right hand, and splenic rupture status post splenectomy who initially presented for drug overdose after snorting a bag of fentanyl at home. Since the patient was unconscious, he was down for an unknown amount of time but was eventually revived with intranasal naloxone by paramedics. In the emergency room, the patient endorsed severe left-sided back pain and a heavy sensation in his bilateral lower extremities along with complete loss of motor and sensory ability from the waist down. The patient was also unable to spontaneously void, prompting the placement of a Foley catheter that returned dark, brown urine. His labs displayed a CK of > 20,000 and an acute kidney injury with a creatinine phosphokinase (CPK) of 3.16. Magnetic resonance imaging (MRI) of the thoracic and lumbar spine revealed diffuse edema and expansion of the left paraspinal muscles with postcontrast enhancement and degenerative changes, indicating ACS (Fig.1).

The patient underwent an urgent left paraspinal decompressive fasciotomy with closure the following day. Postoperatively, the patient regained both function and sensation in his lower extremities. On manual motor testing, using the Visual Analog Scale, he was grossly 2/5 for his left lower extremity and 3/5 for his right lower extremity. His sensation to light touch remained impaired for his left lower extremity but was intact for his right lower extremity. He also endorsed improvement in the previously described heavy sensation of his bilateral lower extremities.

Over the next 3 days, the patient reported new and persistent paresthesias of his entire left upper extremity. This prompted a cervical spine MRI that revealed cord edema and patchy areas of cord enhancement extending from C4-C5 through C7-T1, with concerns for subacute spinal cord ischemia. Subsequently, the patient was taken for emergent C3-C7 laminectomies with posterior cervical disc fusions. Since then, the patient endorsed improvement in his paresthesias along with overall strength in his left upper extremity.

DISCUSSION

ACS is a well-known medical emergency, but there are still no established guidelines for immediate surgical intervention. Currently, the presumed diagnosis of ACS is equivocal for a patient requiring an urgent fasciotomy (6). If intracompartmental pressure is > 30 mm Hg or if the perfusion pressure is < 30 mm Hg in any muscle compartment, a fasciotomy is indicated. Regardless of the location of ACS, the diagnosis and management are the same (6).

In our case of paravertebral compartment syndrome, it is important to know the anatomy of the thoracolumbar fascia (TLF) to understand the mechanism of the resulting injury. The TLF attaches to thoracolumbar transverse processes medially and lumbosacral spinous processes posteriorly. Relevant muscles of the TLF include the multifidi, longissimus, iliocostalis, and semispinalis. The muscles within this fascia are innervated by both the ventral and dorsal rami of the lumbar spinal nerves (7). It is important to note this compartment's proximity to the exiting nerve roots that are medial to it, a possible contributing cause of the weakness seen in our case. As the pressure from myoglobin building within the compartment jeopardizes the integrity of the fascia, surrounding structures find themselves at risk.

While fentanyl was the direct cause of the patient's overdose, it likely contributed to his rhabdomyolysis. The pathophysiology involves the excessive release of calcium ions into the intracellular space. Actin and myosin have disastrous reactions with the surplus of calcium, resulting in muscle destruction and fiber necrosis. There is a plethora of drugs that can cause rhabdomyolysis, such as antidepressants, barbiturates, diuretics, corticosteroids, amphetamines, and drugs of abuse like fentanyl (8,9). Multiple case reports (10-12) have suggested that rhabdomyolysis can either induce or exacerbate ACS. Babak et al (13) reported a similar association between opioid toxicity and rhabdomyolysis in the intensive care unit of a poisoning center.

While rhabdomyolysis with acute renal failure/injury has been documented in opioid overdoses and subsequent use of naloxone, the described atypical presentation of unilateral paravertebral compartment syndrome causing bilateral neuromuscular symptoms concurrent with genitourinary deficits remains a mystery (13-15). Possible contributing factors could be the left paraspinal muscles' expansion causing compression on the spinal canal, resulting in functional transverse myelitis. It might be hypothesized that the instigating

agent was some component of the opioid mixture that the patient injected, the exact combination of substances remaining unknown as is the case with most recreational drug use.

As an extension of the concept that substance abuse can lead to presentations of transverse myelitis, Hussain et al (16) reported a case of a chronic heroin user developing progressive sensorimotor deficits after heroin insufflation. In this case, cervical MRI revealed abnormal T2 signaling in C2-C7 with cord expansion further supporting the picture of transient myelitis (16). Another case reported by Sahni et al (17) describes a young man who experienced absent sensation, strength, and paralysis below C3 with urinary retention and CPK of 38,905 postheroin insufflation. This patient's cervical MRI also revealed abnormal patchy increased intramedullary signal intensity from C2-T1 (17). Concern for rhabdomyolysis and ACS in his left leg led to next-day emergent fasciotomy (17).

Our case report adds to a growing body of evidence of the effects that opioid insufflation can have on inducing transverse myelitis and ACS, with a unique presentation of unilateral paravertebral compartment syndrome not previously described in the literature. In each case, patients have been found down postopioid insufflation with sensorimotor deficits and concerns for compartmental edema demanding fasciotomies for resolution. A hypothetical mechanism that can contribute to these cases includes the concept of an immune-mediated hypersensitivity reaction, which could be triggered by differential potency or dosing of the substance used (17). As transverse myelitis is known to occur from a myriad of triggers, including infectious and immunological sources, this proposed pathophysiology would fit within the realm of current scientific understanding.

Based on our literature search, there are fewer than 30 reported cases of paravertebral compartment syndrome. Within this small number, there have only been 2 cases that were drug induced, further highlighting the rarity of this case (18). Our case also supports current literature that states surgical fasciotomy leads to improved outcomes as compared to conservative measures (19). Finally, as our patient is a 44-year-old man, he had the risk factors for acute lumbar paraspinal compartment syndrome as described by Nathan et al (20).

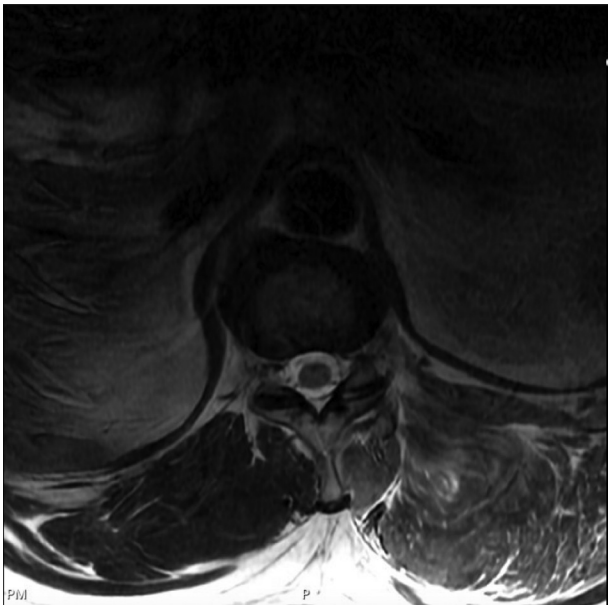

Fig. 1. Axial T2 image with hyperintense signal within the left paraspinal muscle that is asymmetrically enlarged as compared to the right with postcontrast enhancement.

CONCLUSIONS

ACS of the paraspinal muscles is an extremely rare condition. This case is particularly unique as it is in the setting of opioid overdose and reversal. However, paravertebral compartment syndrome must remain on the differential for practitioners who are presented with acute back pain in the setting of drug-induced rhabdomyolysis.

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