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Lumbar Sympathetic Block Causing Transient Paralysis in a Patient with Complex Regional Pain Syndrome: A Case Report

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Background: Complex regional pain syndrome (CRPS) is a condition characterized by the development of spontaneous pain with features of allodynia; hyperalgesia; vasomotor, sudomotor, and trophic changes; as well as motor dysfunction. For lower extremity symptoms, the primary sympathetic intervention is the lumbar sympathetic block (LSB). There are several complications associated with the procedure including paraplegia, especially in the setting of neurolysis.

Case Report: In this case, we describe a patient who underwent a successful LSB with local anesthetic resulting in 4 days of transient lower extremity paraplegia and subsequent complete resolution.

Conclusion: It is essential to understand that this is a potential complication of LSBs in patients with CRPS once all other explanations have been ruled out and that the symptoms will resolve with supportive care.

Key words: Complex regional pain syndrome, lumbar sympathetic block, sympathetic mediated pain, transient paraplegia, case report

BACKGROUND

Complex regional pain syndrome (CRPS) is a condition characterized by the development of spontaneous pain with features of allodynia; hyperalgesia; and vasomotor, sudomotor, and trophic changes. It is also associated with motor dysfunction leading to a decrease in range of motion, weakness, tremor, and dystonia (1-4). Based on the International Association for the Study of Pain and the development of the Budapest Criteria, CRPS is classified as Type 1 (symptoms following injuries without any obvious or specific peripheral nerve involvement), and Type 2 (symptoms following a specific peripheral nerve injury).

The underlying pathophysiology is not well understood but is hypothesized to be based on neurochemical changes induced by catecholamine surges leading to peripheral neurovascular sensitization and subsequent central sensitization (1,2,5-7). It has also been hypothesized that CRPS develops due to a cascade of events related to dendritic cell activation and a subsequent adaptive immune response leading to the alteration of signaling within the dorsal root ganglion and basal ganglia (8). It is further subdivided into sympathetically mediated pain (SMP) and sympathetically independent pain (SIP) groups, which illustrates the varying level of sympathetic nervous system involvement in the syndrome (1-4).

Since the 1940s, the sympathetic block has been a mainstay of diagnosis and treatment for CRPS (9). It is particularly useful in the SMP subgroup of patients with CRPS where there is evidence of sympathetic nervous system coupling to the peripheral and central nervous system (1,10). For lower extremity symptoms, the primary sympathetic intervention is the lumbar

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sympathetic block (LSB). The technique targets the lumbar sympathetic chain that is commonly located in the retroperitoneal space, anterior to the psoas muscle, and on the anterolateral aspect of the lumbar vertebral bodies. There are 5 paired lumbar sympathetic ganglia, but cadaveric dissections have shown significant variability in number and location (11,12). The vast majority of sympathetic ganglia have been found at the L2 and L3 vertebral levels (11-13).

Complications with the procedure are segmental nerve and lumbar plexus injury, genitofemoral neuralgia, renal and ureteral trauma, infection, and discitis, as well as inadvertent intravascular, subarachnoid, and intrathecal injection. There are few reports of paraplegia as a complication from an LSB; the majority of them being permanent neurologic deficits (14,15) as a result of possible Artery of Adamkiewicz injury or subarachnoid/intrathecal injection. There are even fewer reported cases of transient paraplegia, with most of them illustrating cases of weakness lasting weeks to months (16,17). In this case, we describe a patient who underwent a successful LSB resulting in 4 days of transient lower extremity paraplegia and subsequent complete resolution.

CASE PRESENTATION

A 42-year-old woman with a past medical history of CRPS Type I (of the left facial and upper extremity and bilateral lower extremities), which started after a coccyx fracture surgery complicated by an infection in 2007. She underwent multiple sympathetic blocks (including stellate ganglion and lumbar sympathetic blocks), multiple courses of intravenous ketamine infusions, and status-post cervical and thoracolumbar dorsal column stimulator (many years prior to presentation). A month earlier to presentation, the patient had a 3-day course of intravenous ketamine infusions. Additionally, in the same month, she had both internal pulse generators replaced in the dorsal column stimulators of her cervical and thoracolumbar spine. While the dorsal column stimulators provided 60%-70% relief of her upper extremity symptoms, she was still experiencing a severe amount of bilateral lower extremity pain that was deemed to be related to CRPS based on the Budapest Criteria. It was decided to undergo a bilateral LSB. The patient noted in the past that she had developed transient paraplegia for a few days after a prior LSB a few years ago. At that time, a work-up that included a computed tomography (CT) angiogram and neurologic evaluation was negative and spontaneously resolved over a one-week duration. The patient had relief of her lower extremity symptoms for the following 2 years.

The LSB was performed at the anterolateral aspects of the bilateral L3 vertebral body with a total of 10 mL of 0.5% lidocaine on each side with prior confirmation of successful contrast medium spread patterns (Figs. 1A -1C). Given that the patient had experienced prolonged motor weakness using 10 mL of bupivacaine 0.5% on both sides in the previous LSB performed 2 years ago, we elected to proceed with a lower concentration of local anesthetic using 10 mL of lidocaine 0.5%. Immediately after the procedure, the patient subsequently developed bilateral lower extremity weakness and numbness, which required admission to the hospital.

On manual motor testing, the patient's strength was a one out of 5 in the entire lower extremities, except for quadriceps strength which was a 4 out of 5 bilaterally. Additionally, there was a noted decreased sensation to light touch and pinprick in the entire lower extremities bilaterally following the S1 dermatomes. CT of the lumbar spine with contrast medium was performed and showed no explanation for the lower extremity weakness. Due to the presence of the dorsal column stimulators, magnetic resonance imaging was contraindicated. A neurology consult was also unable to explain the cause for weakness. A day later, the patient developed urinary continence, which resolved after discontinuation of a scopolamine patch that was being used for nausea prophylaxis. Over the next couple of days, with physical therapy, the patient demonstrated progressive improvement in her lower extremity weakness and numbness, with functional status returning to baseline after 4 days.

The patient has provided consent for this case to be published in accordance with the Health Insurance Portability and Accountability Act privacy regulations, and all personal identifiers were removed from this case report.

DISCUSSION

Paraplegia is a known complication of LSBs and has been described before in the setting of neurolytic procedures (18). However, transient paraplegia that lasts significantly beyond the usual duration of local anesthetic block, in this case over 4 days, has not been described in the literature to our knowledge. Additionally, the patient experienced this phenomenon on multiple occasions without any long-term neurologic deficits

with subsequent long-term persistent pain relief from the sympathetic blocks.

The pathophysiology of CRPS is not fully understood and has been subdivided into many types and groups. The sympathetic nervous system has been theorized to be involved in the SMP subgroup of patients with CRPS (1). The concept of SMP is when there is persistent abnormal activation of the sympathetic nervous system leading to peripheral and central sensitization, along with the associated formation of sympathetic nerve coupling to sensory afferent nerves (19). Some patients with CRPS also demonstrate motor dysfunction symptoms as a part of their syndrome (1), which could indicate a contribution of neuronal fibers traveling through the sympathetic chain in the SMP subgroup. Although the mechanisms are not fully explained, there is likely central reorganization of the motor and nociceptive pathways in CRPS (20), with adaptive motor changes (21) and possible compensatory involvement of the sympathetic system in the maintenance of motor function. Thus, patients with CRPS who undergo an LSB could experience short-term transient motor weakness due to transient loss of compensatory sympathetic mechanisms following the sympathetic blockade.

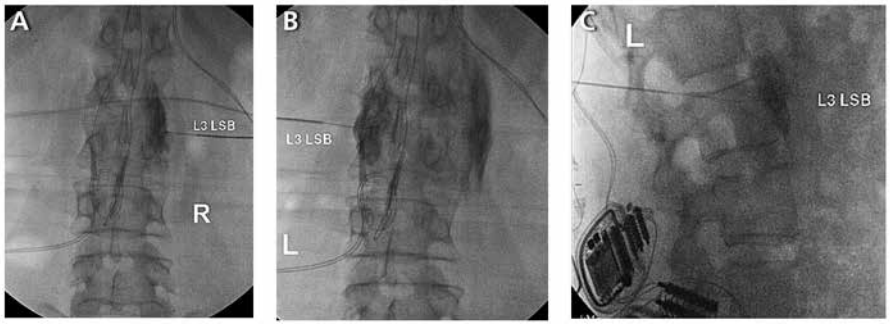

Fig.1. L3 lumbar sympathetic contrast medium spread pattern under fluoroscopy. (A) right-sided approach anteroposterior view; (B) left-sided approach anteroposterior view; (C) lateral view.

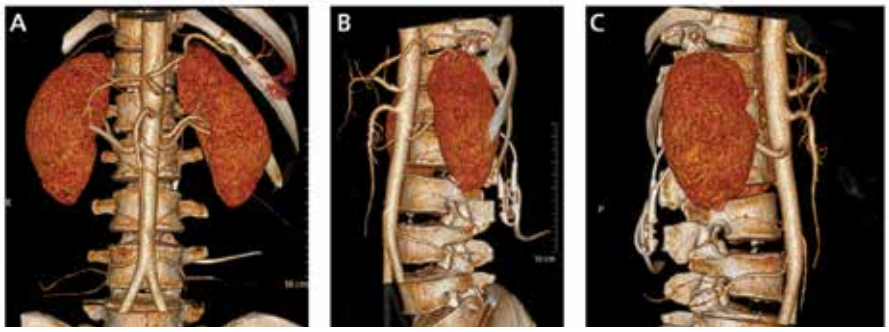

Fig. 2. Three-dimensional rendering of computed tomography angiogram revealing no evidence of vascular ischemic/occlusion. (A) anteroposterior view; (B) left lateral view; (C) right lateral view.

Given the needle placement and contrast medium spread, it is unlikely, but paraplegia can result from blockade of the lumbar plexus. The lumbar plexus lies superficial to the psoas muscle, which is necessary to pass through anteriorly to reach the lumbar sympathetic chain. Additionally, vascular injury and absorption have been postulated to cause spinal cord infarction and weakness (14-17). However, a CT of the lumbar spine with intravenous contrast medium done within 24 hours of the procedure revealed no significant vascular abnormalities indicative of ischemic events (Figs. 2A- 2C). Another possibility is that the needle passed through an unidentified small arterial vessel leading to subsequent vasospasms causing transient paraplegia. The patient experienced the same symptoms during the LSB she had a few years prior, making it less likely that a needle would hit an artery with the same resultant symptoms.

CONCLUSION

The treatment of CRPS demands a multifaceted approach and often requires the use of sympathetic blocks. In a subset of patients with CRPS, disruption of the sympathetic chain may alter the normal functioning pathway; that could lead to short-term transient paralysis or paraplegia. It is essential to understand that this is a potential complication of sympathetic blocks in patients with CRPS once all other explanations have been ruled out and that the symptoms will resolve with supportive care.

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