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Unilateral Anterior Spinal Artery Syndrome following Spinal Anesthesia for Cesarian Section: A Case Report

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Keywords
Anterior spinal artery syndrome · Spinal cord infarction · Spinal anesthesia

Abstract
Introduction: Spinal cord infarction is a rare but serious neurologic complication of spinal anesthesia. Direct vessel injury, intra-arterial anesthetic injection, and anesthetic-induced local hypotension are potential mechanisms of infarction during this procedure. The proximity of the artery of Adamkiewicz to the spinal levels used for spinal anesthesia may also play a role. This case of unilateral anterior spinal artery syndrome highlights the potential for an atypical pattern of injury and deficits due to the complexity of the spinal cord's anterior circulation. ***Case Presentation:*** We present a 38-year-old female patient who presented with left lower extremity weakness, loss of temperature sensation, and urinary retention following spinal anesthesia for cesarian section. Magnetic resonance imaging of the spine demonstrated T2 hyperintensities in the left central spinal cord from T8 to the conus medullaris. A diagnosis of spinal cord infarction was made after lumbar puncture testing showed no evidence of inflammatory myelitis. The patient was treated with steroids empirically until lumbar puncture testing showed no inflammation. The patient was discharged on daily aspirin with persistent left lower extremity weakness and loss of temperature sensation. A plan for outpatient physical therapy was made for rehabilitation. ***Conclusion:*** Awareness of the potential for spinal cord infarction secondary to spinal anesthesia must increase among anesthesiologists, obstetricians, and neurologists. The risk of systemic hypotension during and after spinal anesthesia is important to recognize for both primary and secondary prevention of this complication. The hyperacute onset of myelopathic symptoms should point neurologists to investigate an ischemic etiology in the proper clinical context.

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Introduction

Anterior spinal artery (ASA) syndrome refers to dysfunction of the anterior 2/3 of the spinal cord due to hypoperfusion of the ASA. The syndrome is classically characterized by weakness or paralysis below the affected span of spinal cord as well as loss of pain and temperature sensation. Disrupted bowel, bladder, and sexual functions are also possible depending on the affected region of cord [1]. Any stimulus that decreases ASA perfusion can result in this syndrome, the most common being iatrogenic hypoperfusion during aortic aneurysm repair. Other causes include hypoperfusion secondary to aortic dissection, cardiac arrest, atherothrombotic disease, emboli, vasculitis, sickle cell disease, hypercoagulability, arteriovenous malformations, intervertebral disc herniation, and cocaine-induced vasospasm [2].

Lumbar epidural and spinal anesthesia are common methods of analgesia and anesthesia utilized during labor and delivery as well as cesarian section. Their overall complication risk is low, and their risk of persistent neurologic deficit has been shown to range from 0 to 0.10% of patients [3]. Despite their rarity, potential neurologic complications that have been associated with epidural anesthesia include epidural hematoma, anesthetic toxicity, spinal stenosis, epidural catheter trauma, epidural abscess, hypotension due to epinephrine injection, inadvertent subarachnoid injection, meningitis, spinal cord infarction, ASA thrombosis, or arachnoiditis [4, 5]. Thoracolumbar needle entry for these procedures can pose direct risk to the ASA due to the proximity of major aortic branches that feed the ASA [6]. Here, we present a case of unilateral ASA syndrome following single-shot spinal anesthesia prior to cesarian section.

Case Presentation

The patient was a 38-year-old G3P0020 with no significant past medical history who underwent epidural anesthesia for vaginal delivery at an outside hospital. An 18-gauge catheter was placed with a 17-gauge needle at the L3–4 level without complication. Due to arrest of descent after 3 h of pushing, cesarian section was performed with the addition of spinal anesthesia. Prior to receiving the spinal anesthesia, the patient was hemodynamically stable and reported no pain. A single-shot technique was performed at the L2–3 level, injecting bupivacaine, fentanyl, and morphine with a 25-gauge pencil-tip needle. The anesthesia report detailed positive birefringence with cerebrospinal fluid aspiration at the beginning, middle, and end of the injection. The patient reported an immediate electric shock-like pain traveling down both legs with the placement of the spinal needle. This intense pain was eventually replaced with a sensation of warmth followed by adequate anesthesia for the procedure. The cesarian delivery proceeded without complication.

On postoperative day 1, it was noted that the patient experienced left foot drop as well as numbness over the top of the foot and lateral leg. Neurology saw the patient and recommended a magnetic resonance imaging (MRI) of the head and spine while starting gabapentin as well as aspirin 81 mg daily. The MRI of the spine demonstrated increased T2 signal in the left central hemicord and gray mater extending from T8 to the conus medullaris suspicious for spinal artery infarction versus demyelination. The patient was also noted to have 944 mL of urinary retention with preserved sensation of fullness but lack of ability to void on postoperative day 3. The MRI findings prompted transfer to our institution for further workup on postoperative day 4.

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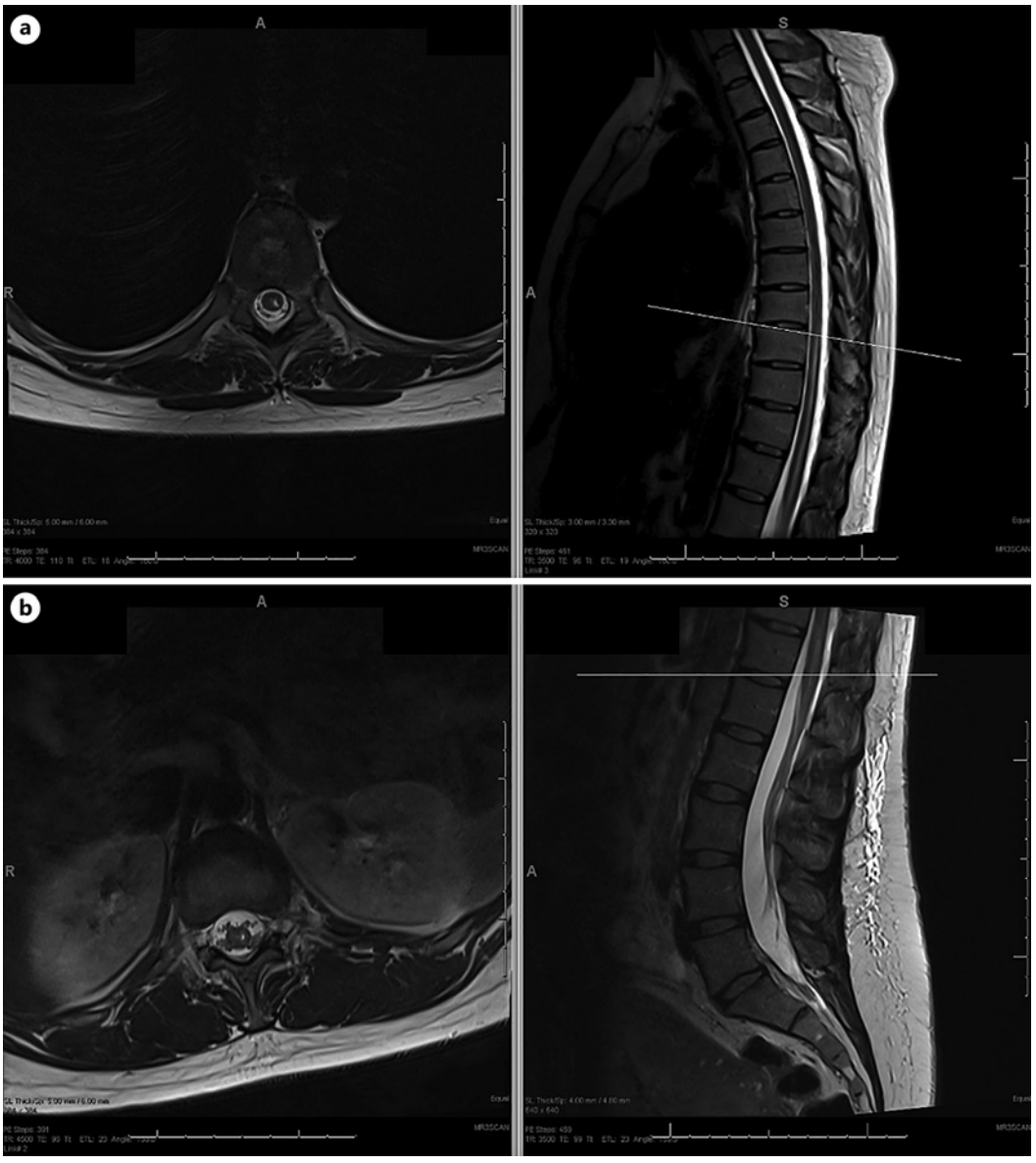

Fig. 1. T2 MRI of the thoracic spine (**a**) and T2 MRI of the lumbar spine (**b**) demonstrating T2 hyperintensities within the left hemicord extending from T8 to the conus medullaris.

In addition to foot drop, the patient was noted to have more diffuse weakness of the left lower extremity as well as loss of temperature sensation up to the hip. Figure 1 displays the results of a repeat MRI that was performed with and without contrast due to movement artifact on the prior study. Similar findings were noted, as well as contrast enhancement of the inferior aspect of the lesion spanning from T12 to the conus medullaris and left proximal cauda equina roots concerning for ischemia versus acute inflammatory myelitis.

A lumbar puncture was performed under fluoroscopy to rule out inflammatory myelitis. While awaiting results, 1 g of daily IV methylprednisolone was begun empirically. A magnetic resonance angiography of the thoracic and lumbar spine was also performed to rule out the presence of a dural fistula which could put the cord at continued risk of ischemia. There was no fistula noted in this study. Lumbar puncture results eventually

Table 1. Lower extremity neurological exam on discharge

Power	Hip flexion	Knee extension	Plantar flexion	Dorsiflexion	Hallux extension
RLE	5	5	5	5	5
LLE	4	4	4	2	1
Sensory	LLE light touch intact but diminished compared to RLE up to inguinal ligament, LLE loss of temperature up to inguinal ligament, LLE vibration intact in MTP joints, RLE normal sensation				
Gait	Marked LLE foot drop with walker needed for ambulation				

RLE, right lower extremity; LLE, left lower extremity.

demonstrated normal protein, <1 nucleated cell, no oligoclonal bands, and a completely negative myelopathy immunology panel. Unfortunately, after 3 days of steroid treatment, melena and a drop in hemoglobin were noted, prompting endoscopic investigation revealing 3 duodenal ulcers without signs of ongoing bleeding. At this point, aspirin was held and steroids were discontinued given the presumptive diagnosis of spinal cord infarction based on the non-inflammatory lumbar puncture results. The patient was discharged the following day on gabapentin, aspirin, and omeprazole with a plan for outpatient physical therapy and rehabilitation. The patient's foot drop mildly improved over the course of the hospital stay, but the other lower extremity weakness and loss of temperature sensation remained constant. Lower extremity physical exam on discharge is displayed in Table 1. A CARE checklist has been completed by the authors for this case report and is attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000539405).

Discussion

The anterior 2/3 of the spinal cord is supplied by the ASA which originates from branches of the vertebral arteries superiorly and is reinforced by segmental aortic branches more inferiorly. It descends along the ventral aspect of the spinal cord, running in the anterior median fissure along with its corresponding vein [6]. The artery of Adamkiewicz is the largest reinforcing blood supply and supplies a significant distribution of the thoracolumbar spinal cord. Its origin is variable by individual, but 78% originate from the left and most originate from a level between T8-L3 with most between T9–T12 [6]. It has been shown to be susceptible to injury during thoracotomies, prolonged aortic clamping, left-sided nephrectomy, splenectomy, pneumonectomy, and adrenalectomy [6].

Several case reports and series have demonstrated spinal cord infarctions secondary to epidural or spinal anesthesia, as well as other epidural injections. One report demonstrated a case of epidural anesthesia with bupivacaine and epinephrine causing ASA syndrome with ischemic spinal cord changes from T4-L1 [7]. It was theorized that the mechanism for this injury was the vasoconstrictive effect of epinephrine in conjunction with possible hypotensive episodes. Another report detailed the case of 2 patients with persistent lower extremity weakness following either epidural or spinal anesthesia during delivery [8]. One case report explains a case of ASA syndrome with delayed onset following single-shot spinal anesthesia [6]. In this case, the injury was thought to be caused by postdelivery hypotension to an area of cord made more susceptible due to the spinal anesthesia.

Despite the rarity of neurologic injury, ischemia to the spinal cord remains a possible complication of epidural or spinal anesthesia due to the proximity of elements of the cord's

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blood supply relative to the areas being targeted by these procedures. There are several possible mechanisms for ischemia secondary to these procedures. These include direct vessel trauma, intra-arterial injection of anesthetic, or hypotension secondary to the properties of local anesthetics. It has been shown that epidural administration of epinephrine can decrease spinal cord blood flow by up to 50% [9]. This propensity toward hypoperfusion is only worsened by periods of intraoperative or postoperative hypotension. Brief periods of hypotension can lead to deleterious effects, with only 2–3 min of spinal artery ischemia being shown to cause permanent spinal cord injury [10].

A prior study showed that 2 of 28 patients with acute ASA stroke presented with unilateral limb motor deficits [11]. Accordingly, only 2 of the 28 patients had unilateral lesions in the peripheral distribution of the ASA with a majority of MRIs demonstrating infarction of the central territory of the ASA. This case highlights a unique unilateral manifestation of ASA syndrome. There are prior reports of unilateral symptoms following ASA stroke, but no case reports demonstrating MRI evidence of a unilateral infarct following spinal anesthesia. The unilateral nature of the injury and deficits highlight the complexity of the anterior blood supply of the spinal cord. Since the ASA supplies the cord bilaterally, unilateral injury suggests local hypotension to a susceptible region of cord. This is likely due to the hypotensive properties of the opioids used in the spinal anesthesia in combination with periods of transient systemic hypotension. Spinal cord infarction is a rare but possible adverse outcome of spinal anesthesia whose awareness must increase among anesthesiologists, obstetricians, and neurologists. For the neurologist, this case highlights that the hyperacute onset of myelopathy symptoms is the largest clue pointing toward an etiology of infarction [12]. It also demonstrates that unilateral symptoms can potentially locate an ischemic lesion to the spinal cord rather than the brain. While the exact mechanism of injury in this case is unknown, the proposed mechanism highlights the importance of strict blood pressure monitoring in both the prevention and management of hypotensive infarction of the spinal cord following spinal anesthesia.

Acknowledgment

We would like to graciously thank the patient for their willingness to share this case with the scientific community.

Statement of Ethics

This retrospective review of patient data did not require ethical approval in accordance with local/national guidelines. Verbal and written informed consent were obtained from the patient for the details of their medical case and accompanying images to be included in this case report.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

M.K. performed a literature review, synthesized the key findings of the case, and was a major contributor to manuscript writing. J.N. provided clinical expertise and oversight of the project while contributing to and thoroughly reviewing the manuscript.

Data Availability Statement

No data or statistics were used in this report. Further inquiries regarding details of the case can be directed to the corresponding author.

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