Journal of Clinical Anesthesia (2015) **27**, 68–72

ELSEVIER

Journal of Clinical Anesthesia

Case Report

Persistent paralysis after spinal anesthesia for cesarean delivery

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Received 12 July 2013; revised 14 August 2014; accepted 22 August 2014

Keywords:
Cesarean delivery;
Spinal anesthesia;
Neuraxial anesthesia;
Neurologic complication;
Paralysis/paraplegia;
Adverse outcome;
Anterior spinal artery syndrome

Abstract Anterior spinal artery syndrome has rarely been reported as a cause of permanent neurologic complications after neuraxial anesthesia in obstetric patients. A parturient developed anterior spinal artery syndrome after spinal anesthesia for cesarean delivery. A healthy 32-year-old parturient presented at $41^{2/7}$ weeks for primary elective caesarean delivery for breech presentation. Spinal anesthesia was easily performed with clear cerebrospinal fluid, and block height was T4 at 5 minutes. Intraoperative course was uneventful except for symptomatic bradycardia (37-40 beats per minute) and hypotension (88/44 mm Hg) 4 minutes postspinal anesthesia, treated with ephedrine and atropine. Dense motor block persisted 9 hours after spinal anesthesia, and magnetic resonance imaging of the lumbosacral region was normal, finding no spinal cord compression or lesion. Physical examination revealed deficits consistent with a spinal cord lesion at T6, impacting the anterior spinal cord while sparing the posterior tracts.

1. Introduction

The decrease in anesthesia-related maternal mortality over the second half of the 20th century is greatly attributed to the increase in neuraxial anesthesia for cesarean delivery (CD) and remains the anesthetic of choice for elective CDs [1,2]. Fortunately, serious and permanent neurologic complications related to the use of neuraxial anesthesia in the obstetric population are rare events [3-6]. The exact incidence of permanent neurologic complications is difficult to determine but has been quoted to be between 0.3 and 1.2 per 100,000 [4]. The etiology of injury includes direct trauma, injection of toxic substances, injection into unintended places, epidural abscess or hematoma, transient neurologic syndrome, and vascular phenomenon [3-5].

The complication of anterior spinal artery syndrome after neuraxial anesthesia in the obstetrical population is especially rare [3-5]. The exact incidence is difficult to determine, although in a retrospective series of more than 500,000 obstetric patients in the United Kingdom, 1 patient was found to have had anterior spinal artery syndrome after neuraxial anesthesia; and it is uncertain whether epidural anesthesia was the direct cause [6]. Anterior spinal artery syndrome presents as a predominant motor deficit, with or without loss of pain and temperature sensation, and with intact vibration sense and proprioception [4]. Anatomically,

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http://dx.doi.org/10.1016/j.jclinane.2014.08.003
0952-8180/

a single anterior artery and 2 posterior arteries supply the spinal cord. The anterior spinal artery begins superiorly at the fusion of the vertebral arteries, and radicular arteries support its blood supply distally. The largest and most important of these is the great radicular artery of Adamkiewicz, which arises from the aorta between T9 and L2. Multiple levels of the spinal cord do not receive radicular branches, thus leaving watershed areas that are susceptible to ischemic injury. Often, the etiology is related to severe, prolonged hypotension, arteriosclerosis, or mechanical interference with the aortic blood flow, caused by emboli or vasospasm. Certain types of surgical procedures can predispose or increase the risk of anterior spinal artery syndrome such as surgery to the thoracolumbar aorta or instrumented spine surgery [3,5,7].

A 32-year-old woman developed persistent neurologic findings consistent with anterior spinal artery syndrome after spinal anesthesia for a primary elective CD.

2. Case report

The patient provided written informed consent for publication of this report. A 32-year-old woman of Ethiopian origin, gravida 4 para 2 abortus 1, presented at $41^{2/7}$ weeks for primary elective CD for breech presentation. Her current pregnancy had an unremarkable prenatal course. Obstetrical history revealed 2 previous children both delivered via assisted vaginal delivery (forceps) with epidural analgesia. She had no previous medical or anesthetic history. Her preoperative blood pressure (BP) was 98/70 mm Hg, and hemoglobin (Hb) level was 10.8 g/dL.

After placing standard monitoring and a 16-gauge intravenous (IV) catheter, spinal anesthesia was performed (T0) in the sitting position with an aseptic technique at level L3-L4 with a 25-gauge Whitacre needle (BD Medical, Franklin Lakes, NJ). Clear cerebrospinal fluid was obtained on the first attempt without trauma or paresthesia and was followed by painless injection of hyperbaric bupivacaine 0.75% 12 mg, fentanyl 20 μg, and preservative-free morphine 150 μg. The patient was placed supine with left lateral tilt, and a phenylephrine infusion (routinely used) was begun at 50 μg/min. Block height progressed as expected attaining a T4 level at 5 minutes after spinal anesthesia. Four minutes after spinal anesthesia, the patient presented symptomatic bradycardia (37-40 beats per minute) with ventricular escape beats and hypotension (BP, 88/44 mm Hg). The phenylephrine infusion was initially reduced by half (25 μg/min), and a bolus of ephedrine 10 mg IV and atropine 0.6 mg was given. Surgery began 8 minutes after this hemodynamic, event and the remainder of the CD proceeded uneventfully. Blood loss was 750 mL, and the lowest intraoperative BP was 78/33 mm Hg at the end of surgery, which was promptly treated with ephedrine 10 mg IV (Fig. 1).

One hour after spinal anesthesia (T0 + 0:57), the patient was transferred to the postanesthesia care unit (PACU) and was stable (BP, 107/65 mm Hg; heart rate, 82 beats per minute; oxygen saturation as measured by pulse oximetry, 97%). Over an hour after arrival at PACU (T0 + 2:10), the patient had an episode of mild hypotension (BP, 90/49 mm Hg). She had a soft abdomen, no excessive vaginal bleeding, denied pain or nausea but still had a complete motor block. A bolus of 500 mL of crystalloid was given, and the head of the bed was lowered. Two more boluses of 500 mL of crystalloid and colloid (Voluven; RxList, San Clemente, CA) were given within the next 90 minutes to treat mild hypotension (systolic BP, 85-90 mm Hg). Less than 3 hours after spinal anesthesia, the patient started feeling abdominal pain and received oral acetaminophen 1 g and naproxen 500 mg. Four hours after spinal anesthesia, the patient's abdominal incision pain increased (verbal rating score, 7/10) and she was given 10 mg of oxycodone. Hemoglobin level was 8.2 g/dL 5 hours postpartum.

With no further hypotension and return of pain sensation, PACU discharge was still not possible due to slow motor block regression. The on-call staff anesthesiologist performed a neurologic assessment (T0 + 6:25). She had little hip flexor movement, was able to move her toes bilaterally, decreased pinprick sensation to T4 but able to feel touch over her abdomen. Initial clinical impression was slow spinal block regression, with the plan for continued observation with hourly neurologic examinations. The patient was reassessed 1 hour later, and the neurologic examination remained essentially unchanged. After telephone consult with neurosurgery, a compressive spinal lesion, such as a hematoma, seemed clinically unlikely due to the high sensory block, sensation of pain, and little improvement in the serial motor assessments. A diffusion-weighted unenhanced magnetic resonance imaging (MRI) of the lumbosacral spine demonstrated no spinal cord compression, and the spinal cord and conus medullaris were normal (T0 + 9:40) (Fig. 2).

Serial neurologic examinations overnight continued to have some evidence of slow motor block regression with increased movement in her feet but minimal hip flexion. She remained afebrile, and her BP was stable. The next morning, 16 hours after spinal anesthesia, neurology was consulted and found proximal hip/knee strength 2+ to 3 per 5 bilaterally, ankle strength 4+ per 5, and decreased sensation to soft touch and pinprick to T6 but intact vibration sense and proprioception. Neurology suggested that her examination was suggestive of an anterior spinal cord lesion at T6 with a probable ischemic etiology due to hypotension, vasospasm, or emboli or, less likely, an inflammatory etiology. A second diffusion-weighted MRI (Fig. 2) was requested to image the whole spine, which concluded no spinal cord compression, no syrinx, and no filum lipoma; however, ischemia or inflammation of the spinal cord could not be ruled out. During the course of post-operative day 1, motor and sensory function improved.

During the next week, the patient's motor and sensory function continued to improve with the left side now more affected than the right. Investigations included a normal transesophageal echocardiogram, a negative sickle cell screen, and a negative Hb electrophoresis. The patient's