● Case Reports

Rapid Onset of Massive Subdural Anesthesia

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Objective: This case report describes the accidental placement of a lumbar labor epidural catheter into the subdural space resulting in the rapid onset of massive subdural anesthesia.

Case Report: A single-orifice open-ended catheter was accidentally placed in the lumbar subdural space. After a test dose of 3 mL 1.5% lidocaine with 15 μg of epinephrine and 2 minutes after a bolus of 5 mL of 0.25% bupivacaine, there was a rapid onset of massive subdural anesthesia with a loss of consciousness, respiratory arrest, and absent pulses. The subdural catheter placement was confirmed by injection of a contrast agent under fluoroscopy.

Conclusion: Massive subdural anesthesia is a complication of epidural catheter placement and may have a rapid onset causing a loss of consciousness, respiratory arrest, and absent pulses. *Reg Anesth Pain Med 2005;30:299-302.*

Key Words: Anesthetic techniques, Epidural, Extradural, Complications, Subdural injection, Respiratory arrest, Hypotension.

This case report describes a patient who suffered from the rapid onset of massive subdural anesthesia. The case varies in presentation from previously described cases because of the speed of onset of the neuraxial blockade.

Case Report

A 25-year-old gravida 5 para 2 patient at 31 weeks' gestation by ultrasound requested labor epidural analgesia. Her prenatal course had been complicated by 4 weeks of premature rupture of membranes and a chronic placental abruption. She had been admitted to the hospital 2 days before because of uterine bleeding and contractions. The patient was 163-cm tall, weighed 104 kg, and was normotensive. Her cervix was 4 cm dilated. The fetus had a frank breech presentation. The patient was advised to have a cesarean delivery but declined and desired to proceed with a vaginal delivery.

The epidural catheter was placed with the patient in the sitting position. A bolus of Ringer's lactate solution was infused through a peripheral venous line. An 18-G Tuohy needle was inserted without difficulty using the loss-of-resistance-to-air technique. A 20-G single-orifice open-ended epidural catheter was inserted via the epidural needle at the lumbar (L) 2-3 interspace. Aspiration of the catheter was negative, and 3 mL 1.5% lidocaine with a 15 μg epinephrine test dose was administered to the patient. Four minutes after the test dose, sensory testing for an intrathecal injection was negative with no change in cold sensation or motor strength and stable vital signs. Then 5 mL 0.25% bupivacaine was administered rapidly to the patient, who was now sitting in the semireclined position.

One minute later, the patient whispered, "I can't breathe." Within the next minute, the patient became unresponsive, apneic, and pulseless and had dilated pupils. There were no drugs or monitoring available to proceed with advanced cardiac life support in the labor room. The patient was immediately intubated without muscle relaxants and ventilated with 100% oxygen; cardiac massage was administered, 5 mg ephedrine was given intravenously, and she commenced to receive fluid resuscitation. The patient was immediately transferred to the adjacent obstetric suite operating room for resuscitation, monitoring, and an emergency cesarean delivery.

On arrival at the operating room, the patient was administered an additional 10 mg ephedrine intravenously. The patient was rapidly prepared for sur-

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gery, and the obstetrician proceeded with performing a cesarean delivery. Initial electrocardiogram/EKG monitoring showed the patient to have sinus tachycardia. A noninvasive blood pressure of 110/45 was obtained after skin incision. A 1,950-g infant was delivered 6 minutes after the event with Apgar scores of 6 at 1 minute and 8 at 5 minutes. Anesthesia was maintained with 0.3% isoflurane in oxygen throughout the cesarian delivery. The operation lasted 105 minutes, and then anesthesia was discontinued. The patient was transferred to the intensive care unit. One hundred twenty minutes after the test dose was administered, the patient was following commands and had a fourth thoracic (T4) sensory level to pinprick. The block had receded completely by 450 minutes.

The subdural placement of the catheter was confirmed under fluoroscopy by injecting 5 mL contrast dye through the catheter. A neuroradiologist confirmed spread of the contrast into the subdural space with the tip of the single-orifice open-ended epidural catheter sited at the L2 level (Fig 1).

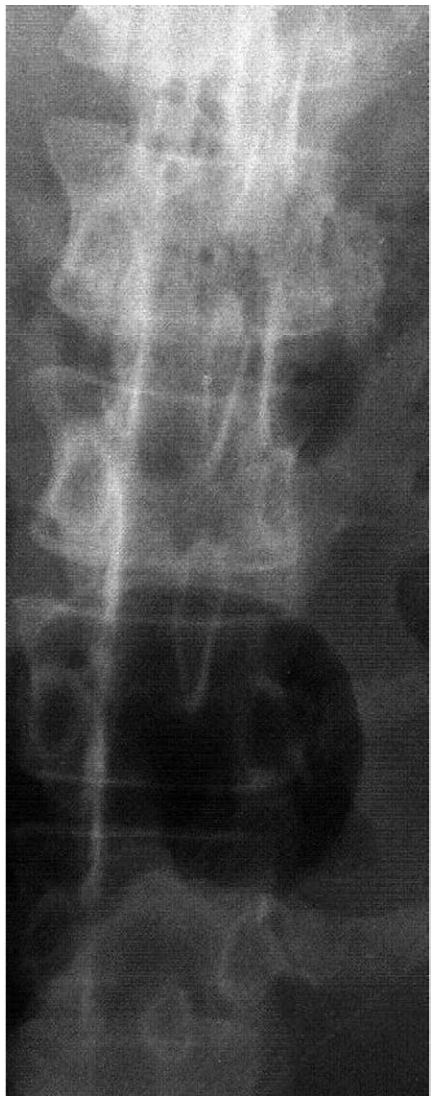

Fig 1. Anteroposterior view of the thoracolumbar spine with contrast agent visible in the subdural space.

Discussion

Accidental subdural analgesia is a known infrequent complication of epidural blockade. De Saram[1] first postulated inadvertent subdural injection was the cause of brief lived respiratory paralysis, pupillary dilatation, and unconsciousness without hypotension during attempted epidural anesthesia. Dawkins[2] coined the term "massive subdural" for atypical signs presenting after attempted subarachnoid or extradural injections. These atypical signs were extensive neural blockade up to the cranial nerves, respiratory failure, pupillary dilatation, and unconsciousness. These blocks had a slow onset of up to 20 minutes, a short duration of effect, and a rapid recovery of complete neurologic function that was not consistent with recovery of subarachnoid injections. Boys and Norman[3] were the first clinicians to confirm with radiographic contrast studies that atypical signs associated with epidural catheter placement were caused by subdural blockade.

The mechanism of subdural space entry is believed to be caused by needle penetration because modern soft-tipped epidural catheters are not thought to be capable of penetrating the dura mater.[4,5] Radiologists report a 5% to 13% incidence of subdural injection of contrast agents during myelograms.[6,7] Mehta and Salmon[8] used radiographic contrast to show the sites of Tuohy needle orifices with intentional epidural placement. They studied 100 planned epidural injections and found that the Tuohy needle bevel was sited partly in the subdural space and partly in the epidural space 7% of the time.[8]

The characteristic signs of an extensive subdural block have been described as (1) the presence of only moderate hypotension (a minimum systolic pressure of 80 mm Hg is an almost constant feature of all reported cases), (2) the slow onset of symptoms after 15 to 20 minutes, with gradual progression over the following 20 minutes, (3) progressive respiratory depression and uncoordination, and (4) complete recovery after almost 2 hours.

Our case did not present with any of these classical signs. First, the patient was pulseless. Second, the massive subdural block had an onset at only 6 minutes. Third, sudden apnea occurred without progressive respiratory depression. Fourth, the block took over 450 minutes to recede completely.