Case Report

Transient unilateral brachial plexopathy and partial Horner's syndrome following spinal anesthesia for cesarean section

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Abstract

A healthy 21-year-old primigravida presented for elective cesarean section. At 45 min after intrathecal (IT) injection of bupivacaine, morphine and fentanyl she developed dysphagia, right sided facial droop, ptosis and ulnar nerve weakness. This constellation of signs and symptoms resolved 2 h later. Based on the time course and laterality of her symptoms, as well as the pharmacologic properties of spinal opioids, we believe her symptoms can be attributed to the IT administration of fentanyl.

Key words: Fentanyl, Horner's syndrome, intrathecal opioids

Introduction

A variety of neurologic symptoms have been reported following neuraxial administration of opioids. These symptoms, including dysphagia and cranial nerve palsy, are perplexing for the anesthesiologist and frightening to patients. We encountered such a case with a transient partial Horner's syndrome and brachial plexopathy following subarachnoid block for cesarean section. Previous case reports have only described the symptoms of neurologic deficits. We are reporting a constellation of symptoms not previously reported and attempt to construct a neurophysiologic explanation for the deficits.

Case Report

A healthy 21-year-old, 69 kg, 170 cm primigravida presented at 38 weeks gestation for elective cesarean section due to fetal anomalies. A spinal anesthetic was performed in the sitting position with 10.5 mg of 0.75% hyperbaric bupivacaine, 10 mcg fentanyl and 200 mcg of morphine. After an uncomplicated intrathecal (IT) injection, she was positioned supine with left uterine displacement. A sensory block was present to T3 bilaterally.

The surgery proceeded uneventfully and a viable infant was delivered. Approximately 40 min after the IT injection, the uterus was externalized for hysterotomy repair. Shortly thereafter, the patient complained of right sided peri-oral numbness, dysphagia and dysarthria. Five minutes later she developed a constellation of neurologic symptoms including: Horizontal nystagmus of the right eye, right sided ptosis with facial droop and right upper extremity weakness with a "claw hand" (hand strength 2/5). Vital signs and mentation remained normal. She denied shortness of breath or chest pain. Differential diagnosis at that time included: High spinal, stroke or transient ischemic attack, air embolus, amniotic fluid embolus and seizure.

At the conclusion of surgery, the neurologic symptoms were unchanged. A non-contrast computed tomography of the head showed no intracranial pathology. Approximately, 90 min after the IT injection, her facial droop and ptosis began to resolve but the right upper extremity weakness persisted. All neurologic symptoms resolved within 120 min. A brain magnetic resonance imaging and a magnetic resonance angiography of the neck vessels were negative. An echocardiogram with agitated saline bubble study showed no right-to-left shunt, ruling out paradoxical embolus.

Discussion

Neurologic complications attributed to spinal anesthesia for cesarean section are rare, with reported incidence of

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0%-0.35%.[1] Neurologic complications are most commonly caused by hematoma, infection, traumatic placement or drug toxicity.[1] Despite extensive testing, no obvious diagnosis was discovered in this patient. The absence of intracranial or cardiac abnormalities narrows the differential diagnosis and increases the likelihood that her presentation could be attributed to spinal anesthesia.

Thus, each component of the spinal anesthesia was scrutinized as a potential cause: All symptoms were right sided despite significant left uterine displacement. Her positioning combined with the absence of hypotension makes it unlikely that her symptoms were caused by cephalad spread of hyperbaric bupivacaine. IT fentanyl and morphine are both hypobaric relative to cerebrospinal fluid (CSF) of a full term pregnant patient.[2] However, the density difference between fentanyl and CSF is more likely to influence the movement within the spinal canal than the density difference between morphine and CSF.[2] Thus, of the two opioids, fentanyl is more likely to exhibit cephalad spread.

The time course of our patient's symptoms is consistent with previous reports. Eisenach *et al*.[3] studied the cephalad movement of IT morphine and fentanyl after injection at a low lumbar interspace and sampling of CSF at a higher interspace with a second needle. They demonstrated both fentanyl and morphine concentrations peaked at similar times at the cephalad sampling site (41 ± 13 min for fentanyl, 57 ± 12 min for morphine).[3] Fentanyl was cleared more rapidly from CSF and the ratio of morphine to fentanyl was 4:1 at 103 min.[3] Our patient's symptoms first appeared 45 min after the spinal, which reflects the cephalization time for both drugs. By approximately 120 min, all symptoms had resolved, which is based on a study by Eisenach *et al*.[3] which correlates with the relative clearance of fentanyl from CSF. Based on the literature demonstrating IT fentanyl to be hypobaric with pharmacokinetic properties that mirror the time course of this patient's symptoms, it was concluded that fentanyl was the most likely cause of her symptoms.

We believe this to be a unique report of unilateral sensory, sympathetic and motor symptoms attributable to IT fentanyl. Transient dysphagia and sensory changes including perioral tingling have been reported following IT fentanyl administration in two patients receiving a combined spinal-epidural technique for labor analgesia.[4] However, these symptoms were bilateral and lacked an upper extremity component. Similar sensory changes (facial numbness and dysphagia) have been reported in a series of six laboring patients receiving IT sufentanil.[5] Cephalad spread of epidural fentanyl has been reported to cause nystagmus.[6] The authors confirmed their hypothesis by successfully "reversing" the nystagmus with naloxone.[6]

Horner's syndrome has been reported following epidural anesthesia for labor.[7-9] Our patient's presentation partially fits the classic Horner's triad of ptosis, meiosis and anhydrosis. Horner's Syndrome following neuraxial anesthesia has been attributed to drug effects on second order neurons as they exit the spinal cord between C8 and T1.[7] Cephalad spread of fentanyl to the level of C8-T1 in our patient would explain both her facial symptoms and her brachial plexus weakness. A "claw hand" is caused by the ulnar nerve dysfunction. The ulnar nerve originates from the C8-T1 nerve roots; therefore, cephalad spread of fentanyl to this level could explain all of her neurologic symptoms. It is anatomically possible to have a concurrent Horner's syndrome with ipsilateral brachial plexus block. This combination has been reported following lumbar epidural anesthesia for labor, but never with spinal anesthesia.[9]

An unanswered question in all previous reports is the mechanism by which fentanyl causes these effects. Fentanyl has been shown to exhibit a local anesthetic-like effect on peripheral nerves.[10] This effect is amplified in desheathed nerve preparations, suggesting IT injection of fentanyl may cause a degree of conduction blockade similar to local anesthetics.[10] A potential mechanism for the local anesthetic-like effects of opioids has been described in a study showing high concentrations of fentanyl, sufentanil and tramadol reversibly block voltage-gated sodium channels *in vitro*.[11]

Although our patient's presentation does not fall into one conclusive diagnosis, her constellation of symptoms could be explained by the cephalad spread of IT fentanyl. This case report aims to remind clinicians that although neurologic complications from IT fentanyl are rare they can cause a wide range of distressing symptoms. If diagnosed early, IT fentanyl induced neurologic changes can potentially be reversed with naloxone, although further studies are warranted. At a minimum, patients can be reassured that the symptoms are transient and perhaps resolve completely within a few hours.

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