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Case Report

When Nerve Meets Needle: A Case Report of Bell's Palsy after Spinal Anesthesia

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ABSTRACT

Bell's palsy is an acute idiopathic nerve paralysis characterized by unilateral facial weakness. Common symptoms include blurry vision, eyebrow and mouth drooping, taste disturbances, and altered hearing. Treatment of Bell's palsy is supportive and usually resolves within 6 months. Although its etiology remains unclear, risk factors for developing the disease, such as diabetes, hypertension, pregnancy, stress, and immunosuppression, have been implicated. Although some reports of Bell's palsy are linked to an epidural, there is a paucity of studies linking spinal anesthesia to the development of Bell's palsy. This case highlights a rare occurrence of Bell's palsy occurring after spinal anesthesia. Pregnancy itself is a recognized risk factor, and most cases arise in the third trimester or immediately postpartum. Although a direct causal link is difficult to establish, it is important for clinicians to consider the possibility of Bell's palsy when a patient develops facial weakness after regional anesthesia. The timing of Bell's palsy development after spinal anesthesia could be coincidental. Alternatively, the administration of spinal anesthesia may induce a transient immune or inflammatory response, leading to nerve irritation or dysfunction. Regardless, early recognition and supportive management can aid in symptom resolution and patient reassurance.

Keywords: Bell's Palsy; Spinal Anesthesia; Duramorph

Introduction

Bell's palsy is a paralysis of the seventh cranial nerve that affects between 15 and 40 of every 100,000 people, with up to 80% of cases resolving spontaneously[1]. Patients typically present with hemifacial paralysis and prodromal pain of the ipsilateral ear as well as possible hyperacusis, eye dryness with tearing, and ipsilateral facial numbness[2]. Most cases of Bell's palsy in obstetric patients occur during the third trimester or within a week of delivery. Early treatment with corticosteroid therapy is key to mitigating poor long-term outcomes[3,4].

Cranial nerve dysfunction following neuraxial anesthesia is an uncommon but documented occurrence. Although Bell's palsy is an uncommon complication following an epidural administration, even fewer cases are reported of Bell's palsy following spinal anesthesia specifically. Abducens nerve (CN VI) palsy is the most frequently reported cranial nerve affected following neuraxial anesthesia, manifesting as diplopia and impaired lateral gaze[8]. The underlying pathophysiology is thought to involve dural puncture and local anesthetic administration that alters cerebrospinal fluid (CSF) pressures, leading to traction, inflammation, or compression of the nerve. Specifically, intracranial hypotension induced by CSF leakage may result in downward displacement of intracranial structures, leading to mechanical stretching of cranial nerves[5,8,9].

Although less common than CN VI involvement, there have been reported cases of facial nerve (CN VII) palsy following an epidural dural puncture, resulting in post dural puncture headache and CN VII nerve palsy[8,9,10]. The proposed mechanisms underlying this phenomenon are similar, involving shifts in intracranial pressure affecting the facial nerve anywhere along its anatomic course. One documented case reported the onset of facial nerve palsy within two hours of post dural puncture, underscoring the potential for rapid neurological sequelae[9]. The patient presented with classic signs including drooling, difficulty blinking, and blurring of the eyes.

This case report presents a previously healthy postpartum patient determined to have developed Bell's palsy several days after spinal anesthesia for cesarean delivery. The purpose of this report is to share a unique observation that may have associations with a commonly used anesthetic procedure. Our observations can contribute to medical knowledge for improving diagnosis and treatment strategies. Although the majority of cases of Bell's palsy following spinal anesthesia have complete resolution of symptoms, some cases may be permanent. There are not many reports of post-spinal Bell's palsy in the literature, so this case report may focus attention on a rare occurrence to further explore the etiology, prevention, identification, and management of such an event.

Case Presentation

A 28-year-old woman of Egyptian descent, G3P1011, with a past medical history of GERD, otherwise previously healthy and with no significant surgical history, was admitted for a planned cesarean section due to breech presentation. Her obstetric history included a previous section for fetal distress and one first trimester miscarriage. She had no previous history of facial or distal nerve palsy or dysfunction. She was afebrile, normotensive at 123/ 69, and reported active fetal movement.

Discussion led to the decision to administer spinal anesthesia for the procedure. She received a single-shot spinal anesthesia with bupivacaine 1.8ml of 8.25% bupivacaine and 200 mcg of morphine for her cesarean delivery. She tolerated the spinal with no complications, and her cesarean delivery was unremarkable. She received 2 liters of lactate ringer, her urine output was 500ml, and she had an estimated blood loss of 600ml. Postoperative recovery was routine, and the patient was discharged on postoperative Day 2. On post-op Day 4 she presented to the emergency room with acute left-sided facial weakness and numbness that had begun 3 hours prior when she woke up. There was no pain or any other neurological deficits. She denied systemic symptoms such as fever, chills, trauma, or gastrointestinal disturbances. Her vital signs were stable, and she had no signs of infection or trauma.

We performed an MRI of the brain to rule out other causes of facial nerve palsy, such as a stroke or mass lesion. Imaging was normal. Both the obstetric and anesthesia services were consulted because of the absence of other neurological signs and the rapid onset of symptoms. The conclusion was Bell's palsy. After discussion with the patient, we decided to start the patient on a 10-day course of corticosteroids (prednisone 60 mg daily). The patient was also advised to use artificial tears and eye protection due to the difficulty of closing her eyelid. By her two-week follow-up, symptoms had significantly improved, and resolution was documented at 1 month.

Discussion

Bell's palsy is an acute idiopathic facial nerve paralysis characterized by unilateral facial weakness. Common symptoms include blurry vision, eyebrow and mouth drooping, taste disturbances, and altered hearing. Although its etiology remains unclear, risk factors such as diabetes, hypertension, pregnancy, stress, and immunosuppression have been implicated. Most cases resolve spontaneously within 6 months, often with supportive care.

The annual incidence of Bell's palsy during pregnancy is 25 per 100,000[6]. Some studies suggest a two-to-fourfold increase in Bell's palsy cases during the third trimester[7]. The most widely accepted cause of Bell's palsy is viral reactivation within the geniculate ganglion, leading to inflammation of the facial nerve within the bony canal of the temporal bone and subsequent ischemic insult. However, other factors may also contribute to nerve inflammation or compression during pregnancy. Significant increases in total blood volume, plasma volume, and red blood cell mass can lead to perineural edema and compression, and hormonal shifts causing immunosuppression may increase the likelihood of latent viral reactivation-both of which can independently elevate the risk of developing facial nerve palsy.

As mentioned above, cranial nerve dysfunction following neuraxial anesthesia is a documented occurrence. Along with cranial nerve dysfunction, media nerve palsy that leads to carpal tunnel syndrome is a common nerve compression in pregnancy. Likely due to volume expansion and perineural edema, a similar mechanism is believed to underlie Bell's palsy[8]. In Bell's palsy perineural edema within the facial canal of the temporal bone leads to facial nerve compression. Hypercortisolemia and altered cytokine production favoring an anti-inflammatory state also increase the risk of Bell's palsy because reduced immune response increases the likelihood of latent viral reactivation.

Although Bell's palsy can present after pregnancy, sources and data are inconclusive about the cut-off time[7]. Spinal anesthesia, a widely utilized neuraxial technique, inherently involves dural puncture and the introduction of local anesthetic into the subarachnoid space. Although effective in providing profound sensory and motor blockade, this procedure carries the risk of several complications, including CSF leakage, which may subsequently lead to changes in intracranial pressure. This change in pressure can possibly lead to the development of Bell's palsy. With our patient above, the nerve palsy occurred soon after neuraxial anesthesia, which begs the question: what caused the nerve palsy? Was it the risk factors associated with the third trimester and immediate postpartum, or was it the spinal anesthesia? Although some reports of Bell's palsy are linked to an epidural, there are few to no reports of spinal anesthesia being linked to the development of Bell's palsy.

Given the close temporal relationship between the administration of spinal anesthesia and the onset of Bell's palsy in this patient (four days post procedure), spinal anesthesia appears to be the most likely contributing factor. However, it is crucial to consider all possible causes of Bell's palsy in a pregnant individual following a C-section, including the physiological changes resulting from pregnancy and postpartum recovery.

Conclusion

This case highlights the rare occurrence of Bell's palsy after spinal anesthesia, suggesting that this complication, although uncommon, should be considered in patients who develop facial weakness following regional anesthesia. Further studies are needed to understand the potential link between spinal anesthesia and the development of Bell's palsy.

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