



Postoperative Acute Parotitis After Cesarean Delivery Under Spinal Anaesthesia

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Abstract

Postoperative acute painless parotid gland swelling, which is a rare complication has been reported after caesarian section (CS) under neuraxial anaesthesia. Hereby, we aimed to present a parturient suffering from acute parotitis complication for her elective CS under spinal anaesthesia who had a previous history of acute parotitis after epidural anaesthesia.

Keywords: Cesarean section, parotitis, spinal anaesthesia

Introduction

Acute painless swelling of the parotid gland in the postoperative period is an extremely rare clinical situation. However, limited number of cases have been reported that are identified as acute parotitis developing after obstetrics and plastic surgery performed under neuraxial anaesthesia in the literature (1, 2). This asymptomatic sudden growth in the parotid gland is usually temporary, and the treatment was provided with intravenous (iv) hydration, dexamethasone, anti-inflammatory, and/or anti-histaminic drugs (1, 2). Even though this complication is considered to be self-limiting up to 48 hours, theoretically, urgent tracheostomy as a result of full airway obstruction may be required. Hereby, we aimed to draw attention to the repetitive nature of this complication by presenting our parturient with previous medical history of acute parotitis after caesarian section (CS) under epidural anaesthesia who suffered from acute parotitis complication again after her elective CS under spinal anaesthesia.

Case Presentation

A 31-year-old multiparous parturient without any known history of systemic disease and allergy was admitted because of repeated CS indication after obtaining her informed written consent. After aspiration prophylaxis and providing standard anaesthesia monitoring, spinal anaesthesia was performed using 12-mg hyperbaric bupivacaine, 100-mcg morphine, and 10-mcg fentanyl between L3-L4 intervertebral spaces with a 27-gauge atraumatic spinal needle in the sitting position. Onset of the operation was allowed when the sensory block reached to the thoracic (Th) 4 dermatome. To maintain perioperative uteroplacental circulation and to prevent spinal-induced hypotension, 5 mg of ephedrine iv bolus was administered four times in total. Approximately 3.5 minutes after skin incision, a male baby (3,345-gram, 48-cm) with vertex presentation was born. Apgar scores of the newborn at the 1st and 5th minutes were 9 and 10, respectively. After the umbilical cord was clamped, iv oxytocin infusion was initiated, and iv 4 mg of dexamethasone bolus was given as part of our antiemetic protocol. At the postoperative 5th hour visit, the patient had painless swelling in both parotid glands of our case who is an otorhinolaryngologist. She had a history of bilateral painless parotitis complication after her previous CS under epidural anaesthesia in another hospital 3 years ago, which recovered spontaneously in approximately 12 hours. For differential diagnosis, when the patient was questioned, she had been given a mumps vaccine during childhood. Postoperative whole blood and biochemical laboratory parameters were

within normal limits. Besides, the motor block disappeared after 3 hours in our patient who had a normal neurological examination with vital signs and did not require any respiratory support. According to our postoperative analgesia protocol, 1 gram of iv paracetamol was administered 6 hours after spinal anaesthesia. Considering the risk of an allergic and/or inflammatory etiology due to the parotid swelling, 45.5 mg of pheniramine maleate and 20 mg of tenoxicam were administered intravenously. The patient received isotonic fluid infusion of $5 \text{ mL kg}^{-1} \text{ h}^{-1}$ for the first 6 hours postoperatively and was allowed to take oral intake at the 6th postoperative hour. In our evaluation at the 9th postoperative hour, $20 \text{ mL kg}^{-1} \text{ h}^{-1}$ of oral fluid was given in the last 3 hours, and bilateral reduction in swelling was observed, which was first prominent in the left parotid gland (Figures 1 and 2).

Discussion

However, we believe that only a total dose of 20 mg ephedrine for the treatment of spinal induced hypotension would not have affected the drainage of the parotid gland in the present case.

Common causes of acute parotitis are anatomical anomaly, mass, stone, viral or bacterial infection, radiation, sarcoidosis, and inflammatory pathologies or conditions secondary to inflammatory pathologies such as amyloidosis, tuberculosis, Wegener's granulomatosis, and Sjogren's syndrome. In addition, medical treatments such as L-asparaginase, phenylbutazone, and clozapine, which cause ductal stenosis, may result in acute parotitis. However, postoperative acute parotitis was first encountered in 1960 (3). The acute, transient, noninfectious parotid or submandibular gland swelling associated with general anaesthesia has been defined as anaesthesia mumps (4, 5). The possible mechanism in postoperative acute parotitis cases after general anaesthesia is thought to be obstruction of the parotid duct as a result of gland enlargement. Factors such as increased positive airway pressure, prone position, obesity, prolonged operation, and anti-cholinergic drug use during mask ventilation have been considered. The symptoms of the patients can vary

from asymptomatic swelling to complete blocking of the airway, which may lead to severe respiratory distress. Enlarged



Figure 1. Front view of the patient's parotid swelling



Figure 2. Side view of the patient's parotid swelling

Main Points:

- Anaesthesia-related acute parotitis is a very rare complication.
- There are a limited number of cases in the literature where acute parotitis is associated with neuraxial anaesthesia.
- Acute parotitis case report that developed after neuraxial anaesthesia with the same patient several years apart has not been made yet. We think that this is important in terms of emphasizing on the repetitive nature of this complication.

bilateral parotid gland resulted in postoperative upper airway obstruction after carotid endarterectomy, which required emergency intubation and even tracheostomy (6-8). The parotid gland swelling can also be observed as a result of arterial obstruction-induced glandular ischemia in the prone or sitting position and hyperextension of the head, especially in obese patients who underwent general anaesthesia (9, 10). Although prolonged operation under general anaesthesia is one of the contributing factors potentially because of venous stasis or mechanical obstruction in the parotid duct, the situation would be expected to be different after a short operation such as CS generally preferred in supine position under spinal or epidural anaesthesia. There are limited number of publications related to postoperative parotid enlargement because of neuraxial (spinal, epidural, and combined spinal-epidural) anaesthesia (1, 2, 11). Parotitis that developed with mild respiratory distress 18 hours after spinal anaesthesia for CS was reported in a pregnant patient after *in vitro* fertilization (1). In our case, swelling of the parotid gland without respiratory distress lasted approximately 5 hours after spinal anaesthesia; it was noticed by the patient herself, who is an otorhinolaryngologist. Then, she was referred to us. The other article in the literature, presenting three cases of parotitis following plastic surgery under epidural anaesthesia (2). Similarly, our patient had a history of acute parotitis after epidural anaesthesia for CS after her first delivery. In another case report, bilateral parotid gland enlargement is published after combined spinal-epidural anaesthesia, which is one of the neuraxial methods for CS; in this case, the beta stimulant effect of ephedrine (48–52 mg) used intraoperatively was held responsible (11). However, in our case, only a dose of iv 20 mg of ephedrine (in 5-mg fractionated doses) was administered in the treatment of spinal-induced hypotension that we believe will not affect the drainage of the parotid gland.

Today, postoperative parotitis is quite rare because of the widespread use of antibiotics, good oral hygiene, and sufficient hydration, which provide adequate electrolyte balance perioperatively. This rare complication can generally regress spontaneously, and supportive treatment can be performed with elevation of the head, hydration, and anti-inflammatory drugs. Close follow-up of postoperative good hydration might be required in this sensitive pregnant patient because this complication limited itself within 3 hours when compared to the previously reported cases by adjusting hydration and prescribing anti-inflammatory agents.

Conclusion

The mechanism of postoperative acute painless parotitis complication, which is rarely seen after CS anaesthesia in

pregnant patient population, has not been clearly understood yet. This complication can cause the risk of airway obstruction that may lead to respiratory distress as much as in cases with medical support treatment and close follow-up reported to date as a result of self-limitation. We should also keep in mind that this complication can be observed again under the same conditions.

Informed Consent: Written informed consent was obtained from the patient presented in the case.

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