

Hoarseness after Spinal Anaesthesia Persisting for Ten Days after Delivery: A Case Report

W-Q Sun, D-B Pan, A-G Zhou

ABSTRACT

Hoarseness is common in general anaesthesia after tracheal intubation and brachial or cervical plexus block. However, there have been no reports of hoarseness associated with spinal anaesthesia in obstetric practice. We present a case of hoarseness in a parturient lasting for 10 days after combined spinal epidural anaesthesia with bupivacaine for Caesarean section. The hoarseness in this case may be caused by oedema of the vocal folds, which was due to high-level sympathetic blockade induced by the spinal anaesthesia.

Keywords: Combined spinal epidural anaesthesia, hoarseness, pregnancy

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INTRODUCTION

Hoarseness refers to an abnormality in voice quality manifesting as breathy, strained, rough, raspy, tremulous, strangled, or weak voice or a voice that has a higher or lower pitch than normal. Hoarseness after tracheal intubation is reported at high incidence of 37.1% (1). In addition to direct trauma to the vocal cords, hoarseness can also be caused by vocal cord paralysis due to the recurrent laryngeal nerve block, such as brachial or cervical plexus block (2). However, to date there have been no reports of hoarseness associated with spinal anaesthesia in obstetric practice. In this report, we present one such case of hoarseness secondary to induction of spinal anaesthesia with bupivacaine in a patient who underwent Caesarean section, with the hoarseness persisting for a relatively long period.

CASE REPORT

The patient was a 41-year old woman, (weight 72.5 kg; height 155 cm), with an obstetric history of gravida 8 and one live birth, who was scheduled for elective Caesarean section. Ultrasonography revealed that placental function at gestational age of 39 weeks and 5 days was Class III, and this was the main indication for Caesarean section. The patient had a medical history of cephalosporin allergy. Laboratory tests at gestational age of 29 weeks and 6 days were positive for proteinuria (+), hypoalbuminaemia (34.6 g/L) and

gestational diabetes (oral glucose intolerance test). At admission, the patient's vital signs were normal: temperature 36.3 °C; pulse 88 beats/minute; respiratory rate 20 cycles/minute and blood pressure (BP) 122/80 mmHg. On physical examination, moderate pitting oedema was noted in both lower extremities. Before the start of the surgery, the patient's electrocardiogram was obtained and BP monitored noninvasively. Initially, the BP was 137/86 mmHg and heart rate, 110 beats/minute. After intravenous administration of 500 mL Ringer's lactate solution, the patient was placed in the right lateral position. Combined spinal-epidural anaesthesia was induced using the needle-through-needle technique. The epidural space was located using the loss of resistance to saline method by inserting a 17-gauge Tuohy needle at the L₂-L₃ lumbar interspace; then, 1.8 mL of 0.5% bupivacaine was administered intrathecally via a 25-gauge Whitacre spinal needle, after which an epidural catheter was inserted. The patient was then shifted to the left lateral position, with a tilt of 15°~20° to tip the head slightly low, and oxygen was administered at 2 L/min through the nasal catheter.

At this time (about three minutes after bupivacaine administration), the BP dropped dramatically to 76/42 mmHg and the heart rate reduced to 64 beats/min. At the same time, the level of sensory anaesthesia to pinprick was ascertained at T₂ dermatome. The patient was placed in the reverse Trendelenburg position immediately. The rate of intravenous fluid infusion was increased; oxygen administration was replaced with a face mask and 10 mg of ephedrine was administered twice. Within five minutes, the BP increased to 110/70 mmHg and the heart rate to 80 beats/min. During these changes, the patient experienced dizziness, tinnitus,

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loss of hearing, numbness in the hands and shortness of breath. While speaking, hoarseness was noted; her voice was low pitched, and her words were only audible from a very close distance. However, the patient remained conscious. She was administered another 10 mg of ephedrine, and the BP was 102/63 mmHg and heart rate 83 beats/min. During the delivery, the systolic and diastolic BP was maintained at 110–120 mmHg and 60–70 mmHg, respectively, and the heart rate, at 90–110 beats/min.

Within five minutes of the start of the operation, a healthy girl was delivered. The weight and height of the infant was 3.7 kg and 52 cm, respectively, and Apgar score at both one and 10 minutes was 10. Immediately after the delivery, the patient was administered 40 U of oxytocin in 500 mL Ringer's lactate solution by intravenous drip infusion with a rate of 0.02–0.04 U/min. Additionally, 2 mg of morphine was administered through the epidural tube; for postoperative analgesia, the patient was advised to use a patient-controlled analgesia pump with morphine (6 mg), ropivacaine (150 mg) diluted with saline to 100 mL, with background infusion of 2 mL/h; the administered dose was 0.5 mL and lock time was 15 minutes. The intraoperative infusion volume, blood loss, and urine volume were 1200 mL, 150 mL and 150 mL, respectively. Intraoperative blood glucose was 5.2 mmol/L. All the newly appearing symptoms were relieved within about 30 minutes of onset, except for the hoarseness. About four to six hours after the operation, the nerve block had completely worn off and sensorimotor function in the lower extremities had been restored; however, there was no improvement in the hoarseness. An ear, nose, throat specialist was then consulted, who suggested that the hoarseness was possibly due to paralysis or oedema of the vocal cords, but this could not be confirmed because laryngoscopy was not advisable on the first postoperative day. Aerosol inhalation with gentamicin or dexamethasone was recommended, but the patient refused this.

The next day, the patient reported an improvement in the hoarseness, and the motor power and sensation in the lower extremities had been restored to normal. The patient controlled analgesia was then discontinued. The patient was discharged on the third postoperative day. Post-discharge follow-up revealed that she continued to improve significantly after three days and hoarseness had completely disappeared on the tenth postoperative day.

DISCUSSION

To date, no reports have been published about hoarseness associated with spinal anaesthesia in obstetric practice, although cases of Horner's syndrome have been recorded. In one such case of a 27-year old parturient, the onset of Horner's syndrome occurred 20 minutes after puncturing the L₃–L₄ space of the spinal cord (10 mg/mL ropivacaine), with sensory anaesthesia to the T₃–T₄ level; the symptoms were relieved four to six hours after onset (3). In another case of a 28-year old parturient, the onset of Horner's syndrome was

delayed, occurring one hour after spinal anaesthesia with bupivacaine and fentanyl [anaesthesia level to T₄] (4). The complications observed in that case were nasal discharge, streaming from the eyes and conjunctival congestion, all of which are signs of parasympathetic hyperfunction, which regressed spontaneously within eight hours of their onset. Thus, the complications were consistent with Horner's syndrome since the symptoms persisted only until the effect of the sympathetic blockade and were, therefore, caused by a deficiency of sympathetic activity.

In the present case, the hoarseness persisted for more than one week after onset, whereby it could be attributed merely to the local anaesthetic. Unfortunately, since the patient refused direct laryngoscopy, we were not able to identify the exact cause of the hoarseness. We speculate that the hoarseness in this patient could be congestion and oedema of the larynx due to vasodilation of the laryngeal vessels. Anatomically, the mucosa of the larynx up to the vocal cords is supplied by a branch of the superior laryngeal artery and the cricothyroid artery of the superior thyroid artery that originates from the external carotid artery. These mucosal vessels are innervated by postganglionic sympathetic fibres arising from the superior cervical sympathetic ganglion. The functions of the superior cervical sympathetic ganglion cells are then controlled by the preganglionic fibres of the lateral horn of the T₁–T₂ spinal cord. Thus, disruption of the sympathetic innervation of the laryngeal vessels caused by the local anaesthetic could lead to vasodilation, congestion and oedema of the laryngeal mucosa, and thereby result in a hoarse voice. This overstimulation of the parasympathetic reflexes after spinal anaesthesia for Caesarean delivery has been documented in a previous report (4). Spinal block is known to depress sympathetic responses at levels well above those of the sensory block [two to six spinal segments higher] (5). In this case, the level of the block to sensation rose to T₂, resulting in blockade of the preganglionic fibres supplying the laryngeal vessels and subsequent oedema of the vocal cords, as explained above. The lymphatic vessels draining the vocal folds are few in number, and therefore, the reabsorption of oedema would be gradual and the recovery of the vocal folds would be prolonged.

In addition, this patient had some risk factors for oedema of the vocal folds, including pregnancy at advanced age (6), hypoalbuminaemia (7) and gestational diabetes mellitus. The persistence of the hoarseness for 10 days could be partly attributed to the general anasarca, since the spontaneous regression of the oedema coincided with the disappearance of the hoarseness.

Thus, we presented a case of persistent hoarseness secondary to spinal-epidural anaesthesia for elective Caesarean delivery. The hoarseness in this case was diagnosed to be caused by oedema of the vocal folds, which was due to high-level sympathetic blockade induced by the spinal anaesthesia.

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