

## Case Report

# Hoarseness after spinal anesthesia: a case report

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### ABSTRACT

Hoarseness is a common entity encountered post intubation in cases operatively managed under general anaesthesia and during regional anaesthesia mainly in cervical/brachial plexus block as a consequence of involvement of phrenic nerve. Limited reports of hoarseness, associated with spinal anaesthesia (sub arachnoid block) in obstetric and gynecology cases have been documented by various authors. Here we document a case of hoarseness in 24-year-old parturient lasting for 10-14 days, after spinal anaesthesia with bupivacaine (H) for lower segment caesarean section. The hoarseness in this case can be due to unilateral vocal cord palsy as a consequence of high-level sympathetic blockade by administration of sub arachnoid block or vagal neuropathy due to intracranial hypotension, as a consequence of spinal fluid egress from dural puncture site. However, in our case no evidence of post dural puncture headache (PDPH) due to egress of cerebrospinal fluid from puncture site was noted, interestingly pointing towards lower pressure change threshold for occurrence of vagal nerve and vocal cord palsy viz PDPH.

**Keywords:** Spinal anaesthesia, Hoarseness, Pregnancy, Vocal cord palsy, Post dural puncture headache, Sub arachnoid block

### INTRODUCTION

Spinal anaesthesia is most common and anaesthetic modality of choice for procedures involving lower extremities, perineum, lower abdomen and caesarean sections. 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.<sup>1,2</sup>

The causation of hoarseness and its incidence is tabulated in Table 1.<sup>1</sup>

Hoarseness is very rarely a manifestation of internal medical illness.<sup>1</sup> In anaesthesiology practice hoarseness is encountered primarily in patients undergoing operative management under general anaesthesia with endo

tracheal intubation and the rate for same is as high 37%.<sup>2</sup> In addition to direct trauma to vocal folds it can be attributed to palsies of recurrent laryngeal nerve.<sup>3</sup>

There has been very minimal data with respect to cranial nerve palsies due to spinal anaesthesia, the rate as per few published studies, varies from 1 in 200 to 1 in 1200.<sup>4</sup> The most common palsy noted is of abducent nerve accounting for 92-95% of cranial nerve (CN) palsies due to intra cranial hypotension.<sup>5</sup> Clinically abducent nerve (CN-VI) palsy manifests as diplopia between second to fifth post op day.<sup>5</sup> Other palsies encountered include oculomotor, facial, trochlear, trigeminal nerves with clinical presentation varying from transient hyperacusis to horners syndrome.<sup>6-10</sup> Cranial neuropathies/palsies have also been reported following myelography, lumbar puncture, idiopathic intracranial hypotension, and

accidental dural tear during operative intervention on lumbar spine.<sup>11-14</sup> Injuries leading to vocal fold palsy (VFP) mainly comprises of three main causes: trauma (thyroid surgeries, head and neck surgeries), pressure symptoms (pressure on the vagus, RLN) due to malignant growth, inflammatory manifestation (viral infections).<sup>15-17</sup>

**Table 1: Causes of hoarseness.**

Cause	Percentage
Acute laryngitis	42.1
Chronic laryngitis	9.7
Functional vocal disturbances	30
Benign tumors	10.7-31
Malignant tumors	2.2-3
Neurogenic causes (VFP)	2.8-8
Physiological ageing	2
Psychogenic	2-2.2

In this case report, we present one such case of hoarseness in our tertiary care hospital secondary to induction by spinal anaesthesia with bupivacaine (H) in a patient who underwent lower segment caesarean section. Patient complained of hoarseness intra op, which persisted for 10-14 days with spontaneous resolution.

**CASE REPORT**

24 year old female, (weight 72.5 kg; height 155 cm), with an obstetric history of Primi with Breech POG 39 weeks, Booked case with all antenatal record, was scheduled for caesarean section in view of breech presentation. Ultrasonography revealed that placental function at gestational age of 39 weeks and 5 days and breech presentation, and this was the main indication for caesarean section. The patients preoperative evaluation clinic assessment was unremarkable and accepted in ASA-II (pregnancy). Lab and routine antenatal record pointed to uneventful progress of pregnancy. Laboratory parameters were all within normal limits.

At admission the patient was conscious oriented to time place and person, comprehension and phonation was normal and GCS -15/15. BP-110/80 mm Hg right arm

supine position, temperature 36.3°C, pulse 88 beats/minute, respiratory rate 20 cycles/minute and SPO<sup>2</sup> 100% on room air. 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, 100 beats/minute. After intravenous administration of 500 ml Ringer lactate solution, patient was administered spinal anaesthesia (under strict asepsis and LA, L3-4 subarachnoid space identification using palpation method was done and 2.6 ml of 0.5% bupivacaine heavy injected in sub arachnoid space in sitting position midline approach using 25 G Quincke’s spinal needle). The patient was then shifted to the supine position with a left lateral tilt of 15°~20° using Cardiff wedge under right hip. Oxygen administered @ 2l/min by nasal cannula.

Approximately three minutes after positioning and no significant drop in BP/ any hemodynamic parameter, level of sensory check was confirmed with operative team and go ahead to incision after due WHO safety checklist was given. Patient complained of sudden change in voice after approximately 10 mins and hoarseness was noted by attending anaesthesiologist. The patient was anxious and on being enquired about any other complaint, she denied. The vitals at that time were BP-110/60 mm Hg, pulse-100 per minute, respiratory rate -25 per minute with no abnormality, ECG- normal sinus rhythm with no ectopics. Patient denied any complaint of chest tightness, headache, dizziness, nausea or tinnitus. Gynaecology team was informed, successful and speedy delivery of baby was carried out. Healthy male child delivered and handed over to Pediatric nurse (for necessary resuscitation and documentation prior to showing to the mother). Patient continued to be administered oxygen @ 2l/min by nasal cannula, Inj midazolam 0.5 mg iv administered and airway management tray was kept ready for any respiratory or cardiovascular emergency. Patient showed no signs of any respiratory or cardiovascular compromise. BP remained stable with MAP >65 mm Hg, SPO<sup>2</sup> >94% on room air and 100% on supplemental oxygen.

**Table 2: Vocal cord evaluation over the period of convalescence.**

Post op day	Evaluation	Findings	Management
3	Phonation fiber optic	Hoarseness (+) vocal cord (rt): palsy persisted	Conservative, speech therapy
7	Phonation	Hoarseness (vowels and consonants evaluation): slight improvement	Speech therapy
10	Phonation fiber optic	Hoarseness: decreased Phonation: improved vocal cord (rt): slightly improved movements	Conservative, speech therapy
14	Phonation fiber optic	Hoarseness: improved with pre op phonation status vocal cord (rt): normal movements	

Patient continued having hoarseness and post LSCS, ENT consultation on table with fiber optic evaluation was carried out. On examination right vocal cord palsy was noted. Patient was advised conservative management incl observation and NPO for next 24 hours. Patient was stable during rounds except for persistence of hoarseness. Simultaneously other central causes were also ruled out and all investigations turned out to be in normal limits. No active intervention was suggested in view of isolated finding of right vocal cord palsy and patient managed conservatively.

Subsequent evaluation was done on post op 3<sup>rd</sup>, 7<sup>th</sup>, 10<sup>th</sup> day and fiber optic laryngoscopic examination after 2 weeks, concluded complete recovery and return of normal phonation with no residual complaints. Periodic vocal cord evaluation details during the course of two weeks are tabulated in Table 2.

## DISCUSSION

Spinal anaesthesia as a probable cause of vocal cord palsy and associated hoarseness is an unusual and rare finding. At first glance, these cases may be classified as idiopathic despite the coincidence of onset because spinal anaesthesia is generally not considered primarily in the differential diagnosis of vocal cord immobility. However, a causal relationship may be accepted in light of the documented occurrence of upper cranial neuropathy as a complication of spinal anaesthesia, especially in the absence of important confounding factors. Emergence and recognition of symptoms in close approximation to institution of spinal anaesthesia in our patient, is consistent with previously reported cases of cranial neuropathy following spinal anaesthesia. Along with it is the transient nature of these vocal fold palsies. While upper cranial neuropathies after spinal anaesthesia are well recognised, lower cranial neuropathies (CN IX-XII) don't hold the same causation. Guardiani & Sulica suggested that traction on the vagus nerve after spinal anaesthesia caused unilateral VFP.<sup>13</sup> The authors concluded the downward displacement of brain stem and traction on vagus nerve, as a consequence of intracranial hypotension, due to csf egress through needle puncture site. Electromyography (EMG) evaluation studies performed on these patients, subjected to spinal Anaesthesia and developed hoarseness, demonstrated denervation of both RLN and SLN, implying cephalad lesion in Vagus nerve.<sup>13</sup>

Laryngeal EMG is a clinically useful tool for carrying out and ascertaining extent of laryngeal nerve injuries, it has prognostic value if it is used at least two months post onset of symptoms.<sup>14</sup>

Cranial neuropathy following dural puncture is thought to be due to reduction of cerebral spinal fluid volume and consequent decreased intracranial pressure. This is hypothesised to cause traction on the nerve at the level of the brainstem or possibly neural compression by

surrounding structures (e.g., brain parenchyma, blood vessels).<sup>1,10,13</sup> This idea is well accepted with observational finding of transient cranial neuropathies in spontaneous intracranial hypotension.<sup>8</sup>

In our patient who underwent cesarean section, this clinical entity i.e. hoarseness was suggestive of a vagal lesion, rather than an isolated neuropathy of the recurrent laryngeal nerve. In our patient its presumed that post spinal anaesthesia continuous egress of CSF from puncture site caused intracranial hypotension consequently pressure symptoms and traction on vagus nerve at cephalad level. Holes in the dura can continue to leak following puncture, and continued egress of spinal fluid may result in a varied time interval presentation of the neuropathy, combined with the lack of recognition of spinal anaesthesia as a potential cause, these neuropathies may be overlooked or attributed to other factors or considered idiopathic.

The patient presented with episode of hoarseness after spinal anaesthesia administration and had fiber optic proven new onset vocal cord palsy. The same was managed conservatively in ward with simultaneous carrying out of neurological and radiological evaluation. The findings were within normal limits. Patient improved over a period of 2 weeks and reevaluation and assessment yielded complete recovery and normal phonation.

Since the patient's symptoms resolved during the second postoperative week, she did not receive a laryngeal EMG. Therefore, there was not an opportunity to definitively to prove a vagal origin for her VFP. Interestingly the patient didn't complain of any headache, which could be correlated with post dural puncture headache (PDPH), which is proven to be caused due to continuous egress of CSF from dural puncture site and traction on cerebral structures.<sup>18</sup>

## CONCLUSION

Anesthesiologists and treating clinicians must be vigilant and cognizant of the causal relationship of cranial neuropathies with spinal anaesthesia. A greater attention must be paid towards any patient who develops hoarseness shortly after administration of spinal anaesthesia. Patients may have an unrecognised unilateral VFP. Patient with a vagal neuropathy may not show the classic symptoms of an associated post dural puncture headache. In the absence of PDPH, most Anaesthesiologists would not recognize that this complication could be directly related to spinal anaesthesia and might go unevaluated. With the review of the current literature and this case report, it appears that spinal anaesthesia can cause vocal fold paralysis (VFP) as a consequence of spinal hypotension. Fortunately, unilateral VFP does occur, a full recovery or near-complete resolution of symptoms usually occurs within a few weeks to months

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