Journal of Anesthesia & Clinical Research

Guevara, J Anesth Clin Res 2016, 7:7
DOI: 10.4172/2155-6148.1000640

Case Report Open Access

Is Unilateral Vocal Fold Paralysis a Rare Complication of Spinal Anesthesia? A Case Report

John C Guevara, MD*

Department of Anesthesiology, Salem Hospital, Salem, Oregon, USA

***Corresponding author:** John C Guevara, MD., Oregon Anesthesiology Group, 707 SW Washington St., Suite 700, Portland, Oregon 97205, USA, Tel: 503-299-9906; Fax: 503-295-2232; E-mail: jguevara@oagpcgroups.com

Received date: May 31, 2016; **Accepted date:** Jun 30, 2016; **Published date:** July 06, 2016

Abstract

A 47 year old woman underwent an open reduction and internal fixation of a left ankle fracture under spinal anesthesia. In the recovery room, she noticed a sudden onset of dysphonia. Fiberoptic examination revealed an immobile right vocal fold. Her symptoms gradually improved over the next few weeks with conservative treatment. By the eighth week, her voice had returned to normal. Anesthesiologists should be aware that dysphonia that develops immediately or shortly after spinal anesthesia may represent a unilateral vocal fold paralysis (VFP).

Keywords: Vocal fold paralysis; Hoarseness; Dysphonia; Vagus nerve; Cranial neuropathy; Recurrent laryngeal nerve; Abducens nerve; Intracranial hypotension; Spinal anesthesia complications

Case Report

A 47 year old woman underwent an open reduction and internal fixation of a left posterior pilon (tibia) and fibula fracture. The patient refused general anesthesia and intubation because she had undergone a partial cricotracheal resection for subglottic stenosis seven years earlier. Her only symptom at that time was dyspnea. She had never been previously intubated; she had not experienced any changes in her vocal quality or strength. The cause of her subglottic stenosis was considered to be idiopathic. Her cricotracheal resection had been performed successfully and she had normal vocal fold function postoperatively. The patient was 58 kg and 173 cm in height. She was ASA Class II, not a diabetic, and a nonsmoker. She had no history of neuritis, neuropathies, inflammatory conditions, recent vaccinations, or any recent upper respiratory infections. Past surgical history included a cesarean section under epidural anesthesia and a breast augmentation under local anesthesia. Medications included vitamins, calcium, fish oil, and magnesium.

The patient was consented for a spinal anesthetic with peripheral nerve blocks for postoperative pain management. After intravenous sedation with three milligrams of midazolam, the patient was placed into a sitting position for the spinal anesthetic. A 25 gauge pencil-point needle was used to place the spinal block at the L3-4 interspace. On the first attempt, in an atraumatic fashion, the patient received twelve milligrams of hyperbaric bupivacaine. The patient was returned to a supine position and five milligrams of ephedrine was given when there was an initial decrease in mean arterial pressure from 85 to 70 mmHg. The spinal block produced a surgical level block to T4. An ultrasound-guided saphenous nerve block was performed utilizing 10 mL of 0.25% bupivacaine.

The patient was placed into the prone position. All pressure points were well-padded, avoiding hyperextension and hyperflexion of the neck and shoulders. During positioning the patient spoke in a normal manner and stated that she was comfortable. Intravenous sedation consisted of propofol at a rate of 50-75 mcg/kg/minute and a total of three milligrams of midazolam and 500 mcg of fentanyl were administered incrementally.

At the completion of the three hour surgery, the patient received an ultrasound-guided popliteal-sciatic nerve block utilizing 30 mL of 0.50% bupivacaine. The patient's head position was unchanged from the beginning of surgery. The propofol infusion was discontinued and the patient was returned to supine position. In the recovery room, the patient was alert and without pain or discomfort. She did, however, notice a mild to moderate hoarseness to her voice quality. The patient continued to be free of pain after her spinal had completely worn off; she was discharged to home four hours after surgery.

By the next morning, the patient's voice was breathy, raspy, and rough in quality. Her dysphonia did not improve with voice rest or with fluid intake. By the third postoperative day, she became concerned that she had developed laryngitis and made an appointment to see an Ear, Nose, and Throat (ENT) physician. On postoperative day seven, her ENT physician found an immobile right vocal fold on flexible laryngoscopy. No other abnormalities were seen. The patient did not complain of dysphagia, aspiration, or any symptoms which might be indicative of a post-dural puncture headache. She was given the option of further workup with computed tomography (CT) scan and/or a referral to a specialized laryngologist. She opted for a conservative approach of watchful waiting and agreed to follow-up in two months if no improvement was seen. She was not prescribed corticosteroids or any other medications.

Over the next few weeks, the patient's symptoms of dysphonia began to improve. By the end of the eighth week, her voice quality had completely returned to normal. Because her symptoms had resolved, no further clinic visits or tests were performed. During telephone interviews with the patient at four and five months postoperatively, she reported that she had no further complaints of hoarseness.

Discussion

Vocal fold movements result from the coordinated contraction of a complicated system of laryngeal muscles. An injury to the recurrent

laryngeal nerve (RLN) is necessary to cause a gross VFP. This may occur alone or in combination with an injury to the superior laryngeal nerve (SLN). Laryngeal electromyography (EMG) is a clinically useful tool in the management of laryngeal nerve injuries; it has prognostic value if it is used at least two months after the onset of symptoms [1]. Most of the nerve injuries that lead to VFP arise from three main causes: mechanical trauma from surgery of the thyroid, head, and neck; malignant growth causing pressure on the vagus nerve or RLN; or inflammatory processes, usually viral infections [2-4].

Without an intact RLN, the laryngeal muscles will not be able to open the vocal folds (to breathe or to cough), to close the vocal folds (to allow vibration for speech), or to close them completely (to protect the airway when swallowing). Hence, patients with a unilateral VFP can have symptoms of dyspnea, dysphonia, and aspiration. Patients may develop anxiety and extreme frustration as these symptoms exact a physical, emotional, and financial toll on their lives. Many patients require speech and swallowing therapy during their recovery. Patients may become less productive at work due to difficulties with speech and voice fatigue. It is not unusual for these patients to experience a significant decline in their overall quality of life scores [5]. Occasionally, depending on the severity of the symptoms, it is necessary to medialize the affected vocal cord by injection or by surgery.

When a patient develops dysphonia after a spinal anesthetic, there is not an obvious causal relationship. Transient upper cranial neuropathies (CN I-VIII) are known to have resulted from spinal anesthesia with an incidence of between 1:200 and 1:1200 [6]. The mechanism of injury is thought to begin with a puncture of the dura and a subsequent leak of cerebrospinal fluid (CSF). Intracranial hypotension can then develop and result in a caudal displacement of the brain and brainstem which places traction on the cranial nerves [7,8]. This traction is thought to cause stretching, compression, or ischemia of the nerve [7]. Clinically, we see this as the development of a palsy, with a complete or partial muscle paralysis. A slow leak of CSF over time may lead to a delayed presentation of this palsy, sometimes up to one or more weeks postoperatively. Typically, these palsies are of a transient nature and usually resolve completely within a few weeks to months [9].

Palsies of the oculomotor, trochlear, trigeminal, abducens and facial nerve have occurred after both spinal and spinal-epidural anesthesia [6-8,10-12]. The most commonly affected cranial nerve is the abducens (CN VI). It may represent up to 92-95% of all cranial nerve injures associated with intracranial hypotension [8]. An affected patient might typically present with diplopia between two to ten days after a spinal anesthetic. In addition, these patients usually present with an associated post-dural puncture headache.

While upper cranial neuropathies after spinal anesthesia are well-recognized, lower cranial neuropathies (CN IX-XII) are not. Guardiani & Sulica suggested that traction on the vagus nerve after spinal anesthesia was causing unilateral VFP [9]. In their case series, they identified three women who developed VFP immediately or within seven days after receiving a spinal or spinal-epidural anesthetic. After three months, their first patient had evidence on laryngeal EMG of denervation of both the RLN and the SLN. This implies a more cephalad lesion in the vagus nerve, occurring before it gives off the SLN and RLN branches. This also supports the hypothesis that intracranial hypotension and a downward displacement of the brainstem may be causing traction or compression on the vagus nerve leading to VFP [9].

The third patient in Guardiani & Sulica's case series is the most suggestive of a causative link between VFP and spinal anesthesia. This patient had spinal-epidural anesthesia for two vaginal deliveries occurring two years apart. Within a few days after each delivery, she developed dysphonia, had choking episodes, and had difficulties with voice production. Flexible laryngoscopy revealed a right VFP after her first delivery and a left VFP after her second delivery. After each episode, she received a tapering dose of corticosteroids and her voice returned to normal within six months. It is remarkable that she experienced unilateral VFP on alternating sides after each spinal-epidural anesthetic.

In the present case, there was no reason to expect anything other than a routine spinal anesthetic. Great care was taken to avoid any intraoperative positioning injuries. The placement of her spinal anesthetic was unremarkable. The patient's blood pressure was maintained within a normal range throughout the case with intravenous fluids. A tourniquet was used during surgery for 120 minutes and her blood loss was minimal. Her sudden development of dysphonia in the recovery room was unexpected. Guardiani & Sulica's first patient had a similar presentation after her spinal anesthetic for a total knee arthroplasty.

Because the patient's symptoms resolved during the eighth postoperative week, she did not receive a laryngeal EMG. Therefore, there was not an opportunity to definitively prove a vagal origin for her VFP. Nonetheless, all of the patients discussed here have similarities: all are women, all had spinal or combined spinal-epidural anesthetics, all developed dysphonia immediately or within a few days after dural puncture, all had proven unilateral VFP, and all fully recovered within a few weeks to months after onset. Interestingly, none had symptoms of post-dural puncture headache.

Conclusion

Anesthesiologists should be cognizant of the fact that cranial neuropathies can occur as a complication of spinal anesthesia. A greater attention should be paid towards any patient who develops dysphonia shortly after spinal anesthesia. They may, in fact, have an unrecognized unilateral VFP. In addition, a patient with a vagal neuropathy may not show the classic symptoms of an associated post-dural puncture headache as they would with an abducens neuropathy. In the absence of a persistent headache, most anesthesiologists would not recognize that this complication could be directly related to their spinal anesthetic. With the review of the current literature and this case report, it appears that spinal anesthesia may cause vocal fold paralysis (VFP). Fortunately, when unilateral VFP does occur, a full recovery or near-complete resolution of symptoms usually occurs within a few weeks to months.

Patient Consent

The patient has provided written informed consent for publication of this case report.

Conflicts of Interest

There are no conflicts of interest with the writing of this case report.

References

1. Wang CC, Chang MH, Wang CP, Liu SA (2008) Prognostic Indicators of Unilateral Vocal Fold Paralysis. Arch Otolaryngol Head Neck Surg 134: 380-388.
2. Toutounchi SJS, Eydi M, Golzari SEJ, Ghaffari MR, Parvizian N (2014) Vocal Cord Paralysis and its Etiologies: A Prospective Study. J Cardiovasc Thorac Res 6: 47-50.
3. Chen HC, Jen YM, Wang CH, Lee JC, Lin YS (2007) Etiology of vocal cord paralysis. ORL J Otorhinolaryngol Relat Spec 69: 167-171.
4. Al-Khtoum N, Shawakfeh N, Al-Safadi E, Al-Momani O, Hamasha K (2013) Acquired Unilateral Vocal Fold Paralysis: Retrospective Analysis of a Single Institution Experience. N Am J Med Sci 5: 699-702.
5. Daniero JJ, Garrerr CG, Francis DO (2014) Framework Surgery for Treatment of Unilateral Vocal Fold Paralysis. Curr Otorhinolaryngol Rep 2: 119-130.
6. Robles R (1968) Cranial nerve paralysis after spinal anesthesia. Northwest Med 67: 845-847.
7. Zada G, Solomon TC, Giannotta SL (2007) A review of ocular manifestations in intracranial hypotension. Neurosurgical Focus 23: E8.
8. Hofer JE, Scavone BM (2015) Cranial Nerve VI Palsy After Dural-Arachnoid Puncture. Anesth Anal 120: 644-646.
9. Guardiani E, Sulica L (2014) Vocal Fold Paralysis following Spinal Anesthesia. JAMA Otolaryngol Head Neck Surg 140: 662-663.
10. Whiting AS, Johnson LN, Martin DE (1990) Cranial nerve paresis following epidural and spinal anesthesia. Trans Pa Acad Ophthalmol Otolaryngol 42: 972-973.
11. Fang JY, Lin JW, Li Q, Jiang N Gao Y (2010) Trigeminal nerve and facial nerve palsy after combined spinal-epidural anesthesia for cesarean section. J Clin Anesth 22: 56-58.
12. Day CJE, Shutt LE (1996) Auditory, ocular, and facial complications of central neural block: a review of possible mechanisms. Reg Anesth 21: 197-201.