PARADOXICAL VOCAL CORD MOTION: A POSTOPERATIVE DILEMMA

- A Case Report -

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Abstract

Paradoxical vocal cord motion (PVCM) is a dysfunction more often seen by otolaryngologists, but of which the anesthesiologist must also be aware of in order to prevent inappropriate invasive airway interventions. For the anesthesiologist, PVCM is most often seen as inspiratory stridor during the postoperative recovery period. Unfortunately, inspiratory stridor can also be a sentinel of impending respiratory failure, and so it is crucial that the serious etiologies be efficiently ruled out. Presented is a case of postoperative PVCM, diagnosed by direct fiberoptic examination, in which timely recognition of this benign, psychogenic postoperative complication resulted in effective and appropriate noninvasive management.

Case Report

A 25-year old, 91 kg, 73 inch tall male with right shoulder instability secondary to motor vehicle accident, presented for shoulder arthroscopy. Past surgical history was significant for one prior shoulder arthroscopy under general anesthesia without complication. He had no known

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medication allergies and was otherwise healthy.

Interscalene block was performed in the preoperative holding area for postoperative pain management. The brachial plexus was located using a peripheral nerve stimulator, and 45 ml of 0.5% bupivacaine with 1:200,000 epinephrine was injected. No cardiovascular or central nervous system effects were observed upon administration of the block.

During transfer to the operating room, the patient complained of difficulty breathing and was noted to be tachypneic and anxious. Pulse oximetry was 100% on room air at the time of arrival in the OR. General anesthesia was induced intravenously with 180 mg of propofol followed by 100 mg of succinycholine. Intubation was accomplishedatraumatically following direct laryngoscopy. Anesthesia was maintained with sevoflurane and muscle relaxation was achieved with rocuronium. Intraoperative anesthetic course was uneventful. The trachea was extubated immediately upon arrival to the post anesthesia care unit (PACU).

While in the PACU, the patient’s vital signs remained within normal limits; he denied pain. His breathing remained spontaneous and unlabored. Approximately one and one-half hours after tracheal extubation, the patient was transferred to stage II recovery. Soon after arrival, the patient became anxious, indicating to the nurse that he was having difficulty breathing and complaining of a sore throat. Inspiratory stridor was first noted at this time. Vital signs were blood pressure 145/80 mmHg respiratory rate 18 breaths per minute, pulse 95 beats per minute, and pulse oximeter 100% on room air. Aerosolized racemic epinephrine was administered. A chest radiograph demonstrated an elevated right hemidiaphragm and was otherwise unremarkable. Upon treatment with midazolam, and subsequently with alfentantil, the patient’s inspiratory stridor resolved completely.

As the sedation began to wear off, the patient again became distressed, stridorous, and anxious, using accessory muscles to breathe. His respiratory rate ranged from 18-32 breaths per minute, blood pressure 143-158/80-103 mmHg, and heart rate 95-109 beats per minute. Pulse
oximeter reading was consistently 99-100% on either room air or 2 liters of oxygen via nasal cannula.

An otolaryngology consult was requested. Direct visualization of the airway with a flexible fiberoptic laryngoscope demonstrated paradoxical vocal cord motion and otherwise normal anatomy. The patient was admitted for overnight observation. He was evaluated by a speech therapist, whose physical exam noted phonation on inspiration. He was discharged the following day with follow up planned with speech therapy. After discharge, the patient’s symptoms spontaneously cleared over the ensuing two weeks.

Discussion

Paradoxical vocal cord motion is a functional abnormality causing bilateral adduction of the vocal folds on inspiration, resulting in the classic inspiratory stridor\(^1\). Other synonyms for this condition include hysterical stridor, psychogenic stridor, Munchausen’s stridor, spasmodic croup, atypical asthma, and benign vocal fold dysfunction. Paradoxical vocal cord motion is considered a type of conversion disorder. A conversion disorder, as defined by the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), is a subtype of somatoform disorder characterized by one or more neurologic complaints that cannot be explained by any known neurologic disorder, in addition, the onset or exacerbation of symptoms must be associated with psychologic factors\(^2\). Risk factors for paradoxical vocal cord motion include female gender, working in the health care professions, middle age, a diagnosis of asthma, and having an ongoing upper respiratory infection. Patients with other psychiatric diagnoses, including depression or anxiety disorders, are also at increased risk\(^3\). An awareness of paradoxical vocal cord motion as one of the etiologies of postoperative stridor is necessary to avoid unnecessary airway intervention in these patients. Pervious reports have described reintubation and even tracheostomy in patients eventually diagnosed with paradoxical vocal cord motion\(^4\).
The differential diagnosis for postoperative inspiratory stridor includes laryngospasm, allergic airway edema, laryngeal stridor (commonly from residual relaxant effect), intubation injury, and mass lesion or foreign body. Given the potentially serious nature of these etiologies, immediate management should be undertaken with the urgency appropriate to the possibility of impending respiratory failure. Medical interventions may include nebulized racemic epinephrine, antihistamines, corticosteroids, intravenous or topical lidocaine, and confirmation of reversal of neuromuscular blockade. If the index of suspicion for paradoxical vocal cord motion is high, a trial of anxiolytic therapy is appropriate. Resolution of stridor suggests the diagnosis.

The diagnosis of paradoxical vocal cord motion is made by a combination of relevant history and physical examination including direct fiberoptic examination. If fiberoptic examination of the airway is not performed during an episode of the stridor, it can be considered a diagnosis of exclusion after other, more serious etiologies have been ruled out. Our patient did not complain of shortness of breath nor did he exhibit stridor until after admission to stage II recovery. It is possible that once the sedative effects of general anesthesia had dissipated, the awareness of decreased pulmonary function from ipsilateral phrenic nerve block caused the anxiety that triggered the paradoxical vocal cord motion. Treatment of paradoxical vocal cord motion includes reassurance and short term anxiolytic therapy, as well as education. A good prognosis is associated with the presence of an identifiable stressor, a sudden onset of symptoms, a good premorbid adjustment, and a lack of accompanying medical disorder.

Perioperative anesthetic management of patients with a history of paradoxical vocal cord motion should include measures to reduce the number of possible triggers. Preoperative psychological consultation is advisable. Anesthetic techniques which avoid tracheal intubation should be considered as mechanical stimulation of the airway has been suggested as a trigger. Adequate anxiolysis in the perioperative period may be preventative.
References
