Vocal Cord Paralysis Secondary to Impacted Esophageal Foreign Bodies in Young Children

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ABSTRACT. Impacted foreign bodies in the esophagus can result in respiratory symptoms including stridor and aphonia. Several mechanisms have been proposed to explain these symptoms, but the possibility of vocal cord paralysis and its cause has not been adequately emphasized.

Two cases of young children with esophageal foreign body are described; both presented with respiratory symptoms, 1 with aphonia and the other with stridor. In both cases, the symptoms were secondary to vocal cord paralysis. A possible mechanism of recurrent nerve injury is proposed. Pediatrics 2001;107(6). URL: http://www.pediatrics.org/cgi/content/full/107/6/e101; esophageal foreign body, vocal cord paralysis, recurrent nerve injury, respiratory symptoms.

Esophageal foreign bodies are a common and potentially serious cause of morbidity and mortality in children. The common presenting symptoms of esophageal foreign bodies are excessive drooling, poor feeding, dysphagia, and vomiting. Occasionally, esophageal foreign bodies, particularly those with a long duration in the esophagus, may cause respiratory symptoms, such as cough, stridor, and wheezing.

In this report we describe 2 cases of infants who had ingested foreign bodies that were impacted in the esophagus and whose presenting symptoms were primarily respiratory. In both cases, fiberoptic bronchoscopy revealed posterior budging of the trachea with vocal cord paralysis.

CASE REPORTS

Case 1

A 7-month-old girl with an uneventful history was first admitted to another hospital with fever, stridor, barking cough, and dyspnea. She was diagnosed as having laryngotracheobronchitis and was treated with nebulized salbutamol and oral prednisone with improvement in her clinical condition. She was discharged from the hospital after 10 days. At home, she continued using nebulized salbutamol.

She was brought to our institution 4 weeks later because of intermittent dyspnea, stridor, drooling, and fever. Twenty-four hours before his admission, the child had ingested a foreign body that was removed by his caregiver. On presentation, he looked ill, with moderate respiratory distress and stridor. Arterial oxygen saturation was 93% in room air. Lateral soft tissue neck film revealed air within the esophagus (Fig 2). Because of the clinical findings of respiratory distress and stridor, the child was taken to the operating room and the cords and trachea were examined with

Case 2

A previously healthy 22-month-old boy presented with an acute onset of dyspnea, stridor, drooling, and fever. Twenty-four hours before his admission, the child had ingested a foreign body that was removed by his caregiver. On presentation, he looked ill, with moderate respiratory distress and stridor. Arterial oxygen saturation was 93% in room air. Lateral soft tissue neck film revealed air within the esophagus (Fig 2). Because of the clinical findings of respiratory distress and stridor, the child was taken to the operating room and the cords and trachea were examined with

Fig 1. Esophagogram showing dilatation of the upper third of the esophagus with an occult foreign body and circumferential narrowing of the airway at that level (arrow).
the flexible bronchoscope (using fentanyl and midazolam for sedation and with the patient breathing spontaneously). The cords were found to be in the median position without any lateral movements on inspiration. Mild extrinsic compression of the upper trachea was noted. General anesthesia with intubation and rigid esophagoscopy were then performed, and a plastic candle-holder was found lodged in the esophageal inlet below the cricopharyngeus. After removal of the foreign body, there was no immediate improvement in the vocal cord mobility. Serial flexible bronchoscopies showed slow but steady return of function, more gradual in the right cord. By 6 months postremoval, laryngeal mobility had returned to normal.

DISCUSSION

Bilateral vocal cord paralysis secondary to an esophageal foreign body, to the best of our knowledge, has not been described previously as a cause of respiratory symptoms. Approximately 80% of all foreign body ingestions occur in children, with a median age between 1 and 3 years. Foreign body presentations are varied and often mimic other pathologic conditions. The most frequent presenting symptoms of esophageal foreign bodies are gastrointestinal, including dysphagia, drooling, and vomiting. Approximately half of the incidents are unwitnessed. In addition, there is often a relatively asymptomatic period after ingestion, before significant signs and symptoms appear. The longer the foreign body remains in the esophagus, the greater the incidence of respiratory symptoms, cough, stridor, fever, congestion, wheezing, apnea, and pneumonia. Cough, fever, and congestion are often interpreted as upper respiratory infections, and stridor mimics croup.

This report describes 2 infants with an impacted esophageal foreign body who presented with respiratory symptoms. One had a 4-week history of cough, stridor, and subsequently aphony without stridor—the aphony was caused by the paramedian position of the vocal cords and this allowed adequate air flow without stridor. There was no dyspnea, despite a 90% extrinsic compression of the trachea. The other patient presented with acute onset of fever, dyspnea, and stridor (secondary to the median position of the vocal cords). The minor posterior tracheal compression was not thought to be a major contributory factor to the dyspnea. In both patients, the vocal cord dysfunction was established by flexible endoscopy under sedation with the patients breathing spontaneously.

Because of anatomic and functional narrowing, the majority of esophageal foreign bodies become impacted in the cricopharyngeal area, where they are likely to cause airway symptoms because of the proximity of the larynx and trachea. Cough or stridor results from direct pressure on the membranous posterior tracheal wall (which is particularly soft in infants and young children) by the foreign body itself or by secondary esophageal dilatation, resulting in narrowing of the trachea. With prolonged retention, the foreign body may produce a periesophagitis or imbed in the wall of the esophagus producing a foreign body granuloma resulting in compression of the trachea and stridor. Close proximity of the airway to the partially obstructed esophagus predisposes to tracheal aspiration and symptoms of pneumonia. The foreign body may erode the wall of the esophagus and create a tracheoesophageal fistula. The foreign body may even pass through the acquired tracheoesophageal fistula and obstruct the airway.

Poncz and Schwartz described a case in whom an intense inflammatory reaction caused by a nutshell had penetrated the esophagus in the area of the aortic arch and caused unilateral left recurrent laryngeal nerve paralysis and subsequently an aortoesophageal fistula. Similar cases of paralysis of the recurrent nerves because of foreign body impaction are described in the literature because of endotracheal intubations. Even short-term nontraumatic intubations have been rarely reported to cause unilateral or even bilateral paralysis.

It is well-known that endotracheal intubation can
cause vocal cord paralysis. The proposed mechanism for intubation-related cord paralysis is entrapment of the anterior (adductor) ramus of the recurrent nerve 6 to 10 mm below the level of the cords among the thyroid lamina, the superiorly located arytenoid, and the inflated cuff of the endotracheal tube.9 The effect of nitrous oxide diffusion into the cuff raising its pressure is also considered to be important. No pediatric cases (intubations sans cuffs) have ever been reported. The difficulty with this mechanism is that one would expect an adductor rather than an abductor paralysis and that hoarseness rather than airway obstruction should predominate. The explanation for the abductor paralysis (the median position of the cords) is that either there is variable innervation (occasionally the anterior-adductor-ramus has been found to innervate all the intrinsic muscles of the larynx10) or the uninhibited action of the intact cricothyroid innervation via the superior laryngeal nerves results in the bilateral adduction.9 The point is made that when the cuff of the endotracheal tube is located in the cricoid region, the circumferential ring of cartilage confers protection from compression injury to the nerves from the tube/cuff.

In contrast to the proposed mechanism for intubation-related paralysis (described above), the bilateral abductor paralysis in case 2 was attributable to an esophageal foreign body just below the cricopharyngeus. Because there is no surrounding protective framework in the esophagus, compression phenomena of the recurrent nerves would be expected when an appropriately sized foreign body becomes lodged. Furthermore, the foreign body could conceivably also produce traction on the main trunk of the recurrent nerve as it courses posterior to the cricothyroid joint (which would tend to tether the nerve). One would then expect both an adductor and abductor paralysis of the recurrent nerve but preserved adducting function of the cricothyroid muscles—causing a median cord position and airway difficulty. It has been shown that the time course of the axon reaction after stretch injury is longer than that obtained after crush injuries.11 Therefore, a more prolonged recovery would be expected from a stretch-compression type of injury to the nerve trunk. Furthermore, it has been shown that the right recurrent nerve is more prone to stretch injuries than its counterpart on the left.12 This phenomenon would explain the different recovery times of the 2 nerves in case 2. In contrast to case 2, case 1 illustrates a simple compression neuropraxia of both recurrent nerve trunks that was relieved with removal of the foreign body and rapid return of function.

CONCLUSION

Both of these cases illustrate the importance of the consideration of an esophageal foreign body when there is associated respiratory symptoms and vocal cord immobility. In these situations, a rigid esophagoscopy can be life saving and should be regarded as an adjunct to the evaluation of a compromised airway.

REFERENCES

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