to serve as a marker. Sphincter pressure was found to be unchanged during saline infusion, and 34% of the solution refluxed into the stomach. In contrast, duodenal acidification with the HCL resulted in a greater than threefold increase in barrier pressure and only 1.8% reflux.[4] Relating these data to our case, we speculate that up to 2,500 ml saline irrigant could have refluxed into the stomach.

Our patient vomited more than a liter of irrigating saline during the postoperative period. A *p*H of less than 2.5 and a volume of greater than 0.4 ml/kg generally are associated with the syndrome of acid-aspiration pneumonitis.[5] While our *p*H was not measured, studies of elective, nonobese inpatients indicate that 80% have a *p*H of less than 2.5.[5,6] Using worst case estimates (three standard deviations from the means of Stoetling,[5] giving a *p*H of 1.36 and a volume of 74 ml) and a gastroduodenal reflux equal to the 1 l of vomited volume, *p*H of the vomitus would have been 2.53. The *p*H of the vomitus could have been much higher, since 70% of patients have preanesthetic volumes of 25 ml or less, and gastroduodenal reflux could have been as large as 2,500 ml. However, aspiration of large volumes of neutral saline produces a clinical picture with many features of acid-aspiration but without pulmonary necrosis.[7] Thus, while the vomitus of this patient was probably above *p*H 2.5, its large volume still presented a severe risk for aspiration pneumonitis.

In summary, we describe a case of vomiting on emergence from general anesthesia in a patient who had percutaneous lithotripsy for intrahepatic cholelithiasis. Its cause is presumed to be a large gastric volume of irrigating saline that refluxed retrograde through an "incompetent" pyloric sphincter. We believe that this procedure is associated with an increased risk of vomiting large volumes of gastric contents with the associated possibility of aspiration pneumonitis.

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Upper Airway Obstruction Following Cyst-to-peritoneal Shunt in a Child with a Dandy-Walker Cyst

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Bilateral vocal paralysis can occur in children with hydrocephalus, meningomyelocele, and the Arnold–Chiari malformation.[1–6] Although paralysis of the left vocal cord has been associated with cardiovascular and mediastinal malformations, it is rarely found as an isolated lesion.[7] We describe a case of unilateral vocal cord paralysis, resulting in severe upper airway obstruction following insertion of a cyst-to-peritoneal shunt in an infant with a Dandy-Walker cyst.

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Report of a Case

This patient was the term product of an uncomplicated gestation and vaginal delivery. Apgar Scores were 8 and 9 at 1 and 5 min, respectively. At 4 months of age, a prominent occiput and a head circumference above the 95th percentile was noted. A computerized tomography scan revealed hydrocephalus and a Dandy-Walker cyst.

He underwent uneventful insertion of a cyst-to-peritoneal shunt. Following extubation of the treachea, he experienced severe respiratory obstruction with retractions, which did not improve with administration of 100% oxygen and controlled ventilation. After administration of succinylcholine 8 mg iv, the trachea was reintubated without difficulty. Because of his irregular breathing pattern, ventilation was controlled for 16 h. At this point, since he had been weaned to an IMV of 4 and was tolerating this well, a decision to extubate the trachea was made. He again exhibited immediate severe upper airway obstruction that was unresponsive to 5 ml of 1:10 nebulized racemic epinephrine. Following the administration of succinylcholine 8 mg iv, the trachea was reintubated.

Over the next 36 h, he received six 4-mg doses of dexamethasone iv. Extubation of the trachea again was followed by the rapid onset of upper airway obstruction. Awake direct laryngoscopy revealed the right vocal cord to be paralyzed and resting in the median position; the left vocal cord crossed the midline superior to the right vocal cord. During inspiration, the glottis was closed almost completely because the flaccid right vocal cord was pulled medially over the midline toward the left vocal cord. The trachea was reintubated and a tracheostomy performed.

Ten days after the original surgery, he underwent a revision of the cyst-to-peritoneal shunt. Direct laryngoscopy at this time revealed normal position and movement of both vocal cords. At the time of this report he has not been decannulated because of his inability to handle his secretions. Repeat direct laryngoscopies reveal continued normal movement of the vocal cords.

Discussion

Laryngospasm,[8] vocal cord trauma,[8] edema of the tongue, uvula, and larynx[9,10] and dislocation of the cricoarytenoid cartilage[11] are the most common causes of upper airway obstruction following intubation and subsequent extubation. Isolated cases of vocal cord paralysis following endotracheal intubation[12] also have been reported. In these reports, the authors postulated that either pressure from the endotracheal tube cuff had caused damage to the internal branch of the recurrent laryngeal nerve[12] or that a toxic neuritis had developed from traces of cleaning solutions such as ethylene oxide remaining on the tube.[13] These particular patients complained of hoarseness rather than airway obstruction. Our report, however, describes a case of unilateral vocal cord paralysis, which we believe was associated with a cyst-to-peritoneal shunt.

Vocal cord dysfunction secondary to peripheral and central neurologic conditions is well recognized.[1-6] Possible mechanisms for vagal dysfunction associated with hydrocephalus and the Arnold-Chiari malformation include brain stem dysgenesis, ischemia, medial compression of the nucleus ambiguous and the vagus nerves, and traction of the cervical rootlets of the vagus.[3,14]

We postulate that with the decompression of the posterior fossa cyst, traction was placed on the vagus nerve, causing paralysis of the right vocal cord and paresis of the left vocal cord. When the cyst tubing was repositioned, releasing the traction, vocal cord function returned. Perhaps the shunt could have been positioned so that it caused direct pressure on the vagal nucleus, which was relieved with repositioning of the tubing.

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