Correspondence

Post-Heller myotomy mucosal perforation because of missed intraluminal esophageal foreign body: a case report

To the Editor,

Achalasia is failure of relaxation in the distal part of esophagus and esophagogastric junction. It is relatively uncommon in children and even rarer in infants with about 300 cases reported in the literature [1]. Heller myotomy is one of the standard methods of treatment that is performed through either thorax or abdomen. Complications of Heller myotomy consist of bleeding, infection, esophageal perforation, incisional hernia, bowel obstruction, and recurrence of achalasia. Mucosal perforation is one of the most serious complications of Heller procedure that may occur during esophagomyotomy.

On the other hand incidental ingestion of foreign body is common in children [2]. Fortunately, most pass through gastrointestinal tract without difficulty; however, those that remain in the esophagus may cause mucosal ulceration and esophageal obstruction and can lead to morbidity and even mortality [3]. Therefore, removal of esophageal foreign bodies is highly recommended.

According to our knowledge, post-Heller myotomy mucosal perforation induced by missed esophageal foreign body has not yet been reported. The authors herein describe such a case in a boy with Down’s syndrome and achalasia.

1. Case report

A 2-year-old boy with Down’s syndrome was referred with a 6-month history of dysphasia, regurgitation, and weight loss. Symptoms became more severe in the last 2 months. He weighed 7 kg and was 75 cm tall. Barium swallow study showed a dilated esophagus with ineffectual peristalsis and a bird-beak deformity of distal esophagus typical for achalasia (Fig. 1). Esophagoscopy was performed 3 months earlier and showed a very large tortuous esophagus, which was narrowed distally but permitted passage of the flexible endoscope without any resistance. After preoperative preparation of the patient (including insertion of a nasoesophageal tube and esophageal washout), Heller myotomy plus modified Belsey-Mark fundoplication was performed through left thoracotomy. At the end of the procedure, the esophageal mucosa seemed intact. He became ill, was febrile, and had some dark fluid noted in the tube thoracostomy 4 days after the operation. At the sixth day, he became toxic, and esophageal leak was confirmed by methylene blue ingestion.

At thoracotomy, a 1.0-cm esophageal mucosal perforation at the site of myotomy was observed because of missed intraluminal esophageal foreign body (apricot pit) (Fig. 2). It was removed, and the mucosal tear was repaired and reinforced by a serosal patch from fundus of stomach. He recovered and was discharged in good condition 8 days after the second operation. Three weeks later, he was able to swallow a solid meal with no problem. Three months after the operation, he was well and without any complications.

2. Discussion

In many centers, the diagnosis of achalasia is made on clinical grounds, contrast study, and/or esophagoscopy.

Fig. 1 Barium swallow—large and dilated esophagus with classic appearance of achalasia.
Intraoperative mucosal perforation can occur during performance of a Heller myotomy, but in this case, mucosal perforation on the fourth postoperative day was likely related to a missed intraesophageal foreign body. The foreign body was an apricot pit, but there was no recent history of eating or incidental ingestion of apricot in this case. About the growing season for apricots, we guessed he may have eaten the apricot some months before presentation to our clinic. We recently had another patient with congenital esophageal stenosis who underwent esophagoscopy just before operation, and an apricot pit was observed and removed. Because mucosal injury and leakage is a serious complication of Heller myotomy and because preoperative esophageal washout cannot extrude such foreign bodies (as noted in this case), we suggest preoperative esophagoscopy by the surgeon just before operation in the pediatric age group to prevent such a complication. This is especially important in children as they are more likely to swallow foreign bodies.

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References

Multiple *Streptococcus milleri* cerebral abscesses after repeated esophageal caustic stricture dilation

To the Editor,

Brain abscesses have been reported as a rare complication of repeated esophageal dilations; however, routine periprocedural antibiotic prophylaxis is not currently recommended. Caustic injury remains the commonest cause of esophageal strictures in children with strictures developing in 5% to 17% [1-3]. Late presentation is not infrequent in developing countries with serial stricture dilation and bouginage is the preferred treatment of established strictures.

Esophageal dilation is associated with bacteremia in 22% to 72% of procedures [4-7] with *Streptococcus viridans* species predominating [5]. Cerebral abscesses after stricture dilation have been reported sporadically as a rare complication of the procedure [8-17]. Their onset is subtle and depends largely on clinical awareness of this potentially life-threatening complication.

We report a case of multiple cerebral abscesses developing insidiously during a course of serial stricture dilation, serving as a reminder to surgeons and clinicians of the long-term risks associated with bacteremia and discuss strategies for prevention.

A 2-year-old male presented with a 2-week history of dysphagia for solid food, preceded by accidental ingestion of a caustic substance (oven cleaner containing corrosive alkali) 2 months earlier. The patient swallowed fluids easily and regurgitated solid food but was otherwise fit and well, with an unremarkable physical examination. His nutritional status was satisfactory, and he had maintained a normal growth trend.

The patient underwent flexible upper endoscopy that revealed a tight stricture in the upper esophagus. Prograde dilation over a guidewire was attempted. A localized perforation of the esophagus by the guidewire was however suspected, and a small contained contrast extravasation was seen on contrast swallow. The injury was managed conservatively with intravenous antibiotics (amoxicillin/clavulanic acid) for 5 days, and the patient was discharged without further complications on a nutritionally balanced liquid enteral formula.

At follow-up, 14 days after discharge, the patient was asymptomatic, and a program of weekly dilations was recommenced. Before the fifth dilation, 64 days after the guidewire injury, the procedure was postponed because of complaints of malaise and loss of appetite. Physical examination was unremarkable, and dilation was rescheduled for the following week at which time the patient also experienced early morning nausea and vomiting with occasional fever but an absence of headache. Result of