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Case Report

Esophageal Diverticulum as a Cause of Dysphagia after Surgery for Esophageal Atresia

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Abstract

Esophageal diverticulum is an evagination of the mucous projecting from the lumen of the esophagus. It can be characterized according to their pathogenesis as either traction or pulsion or congenital. It is uncommon sequelae of esophageal atresia repaired. We report a childhood esophageal diverticulum case, which presented as recurrent food impaction and difficulty of swallowing. Upper barium swallow and esophagogastroduodenoscopy reveal esophageal diverticulum because of esophageal dysmotility due to esophageal atresia.

Keywords: *Esophageal Diverticulum; Esophageal Atresia*

Abbreviation

EA: Esophageal Atresia

Introduction

Esophageal diverticulum is an evagination of the mucous projecting from the lumen of the esophagus. The congenital or acquired variety of esophageal diverticulum is a rare childhood disease. However, congenital esophageal diverticulum is commonly associated with additional esophageal anatomical deformities or iatrogenic injury in the early post-natal period [1]. Esophageal diverticulum as a cause of dysphagia after surgery for esophageal atresia (EA) at birth is extremely rare. The present paper describes a 12-year-old boy presented with recurrent food impaction and difficulty of swallowing. Upper GI barium swallow and esophagogastroduodenoscopy reveal esophageal diverticulum. A review of the literature of esophageal diverticulum is also provided.

Case Report

A 12-year-old boy presented with recurrent food impaction and difficulty of swallowing since of the age of 5 year. This dysphagia is intermittent and not aggressive. His past medical history revealed that he had esophageal atresia at birth repaired by thoracotomy (multi stage extra-thoracic procedure for elongation of proximal pouch. He was kept in neonatal intensive care unit for 2 months then discharged with regular follow up with pediatric surgery for esophageal dilatation three times in the first year, then he lost follow up. His parents reported that he had poor weight gain but no vomiting or abdominal pain. No history of atopy or food allergy. The family history revealed no

history of gastrointestinal diseases. The patient had normal developmental milestones. Physical examination showed growth retardation (weight and height below 3rd percentile). He had no dysmorphic features. Abdominal examination revealed no hepatosplenomegaly or ascites. Other systemic examination was otherwise unremarkable.

His laboratory results were as following; hemoglobin: 12.9 (normal range: 12.2 - 15.3 gm/dL), White Blood Cell: 7.8 (normal range: 6 - 16 × 10^9/L), Platelet: 372 (normal range: 150 - 450 × 10^9/L). Serum alanine transaminase, aspartate aminotransferase, alkaline phosphatase, gammaglutamyl transferase, albumin and bilirubin were normal. Upper GI barium swallow revealed there is a pouch arising from the midline of the posterior wall of the proximal esophagus with filling defect (Figure 1). Esophagogastroduodenoscopy was performed. Esophageal diverticulum was discovered (Figure 2). Multiple biopsies from the esophagus demonstrated no eosinophil infiltrations or dysplasia.

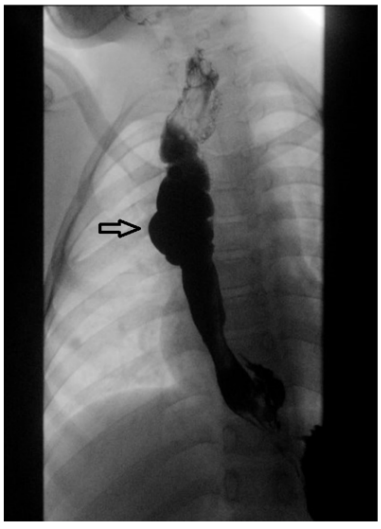

Figure 1: *Upper barium swallow revealed there is a pouch in the middle of esophagus (arrow) with diverticulum but no strictures are apparent.*

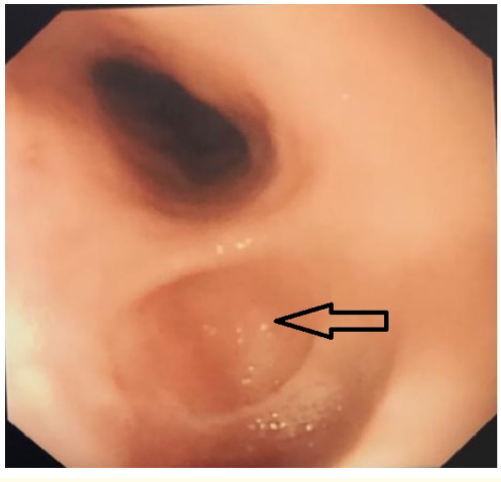

Figure 2: *Endoscopic view of diverticulum of the esophagus that developed on the anterior right wall in the middle of esophagus (arrow).*

As the patient had episodic dysphagia, surgical diverticulectomy was planned but the family refused the surgery. Omeprazole was prescribed and there was no progression of esophageal diverticulum during the 12-month follow-up period.

Discussion

Esophageal atresia is a congenital anomaly defined by an interruption of the continuity of the esophagus with or without a communication with the trachea. Even if the continuity of the esophagus is anatomically replaced, the children are prone to several gastrointestinal problems including dysphagia and gastroesophageal reflux. Dysphagia is observed in more than one-half of children who had surgery for esophageal atresia at birth [2].

Esophageal diverticulum is an evagination of the mucous projecting from the lumen of the esophagus, with a prevalence of approximately 0.06% to 4%, and it primarily affects the elderly [3]. Esophageal diverticulum can be characterized according to their pathogenesis as either traction or pulsion or congenital [4].

There are several types of esophageal diverticula, each of different origin. Zenker (pharyngeal) diverticula are posterior outpouchings of mucosa and submucosa through the cricopharyngeal muscle, probably resulting from an incoordination between pharyngeal propulsion and cricopharyngeal relaxation. Mid-esophageal (traction) diverticula are caused by traction from mediastinal inflammatory lesions or, secondarily, by esophageal motility disorders. Epiphrenic diverticula occur just above the diaphragm and usually accompany a motility disorder such as achalasia and diffuse esophageal spasm [5].

Esophageal diverticula comprise a rare condition that causes dysphagia, regurgitation and chest pain as they progress [5]. Our patient has midesophageal (traction) diverticulum. He progresses to dysphagia secondarily, by esophageal motility disorder because of esophageal atresia. Unfortunately, esophageal manometry is not available in our institution.

Approximately 65% of esophageal diverticulum cases are asymptomatic and are found by endoscopic examination [6]. It has been gradually accepted that esophageal diverticula result from esophageal motor disorders rather than from primary anatomic abnormalities. Twenty-seven patients with these diverticula were evaluated with respect to pathogenesis, clinical aspects, diagnostic tests, therapy, and natural history for a mean of 27 months of follow-up. Thirteen diverticula were midesophageal, 11 were situated in the distal third of the esophagus and 3 were in both regions. Esophageal dysmotility was observed in 85% of patients [6]. Chapuy., *et al*. [7], reports 3 patents with mucosal bridge as a cause of dysphagia after surgery for esophageal atresia. These mucosal bridges may also be a consequence of inflammation caused by chronic food stasis due to esophageal dysmotility. The resection of esophageal mucosal bridges appears to be efficacious in improving dysphagia

In a retrospective analysis by Porcaro [8], 105 children affected by congenital esophageal atresia and tracheoesophageal fistula surgically repaired. Twenty-nine patients have undergone to chest CT with contrast enhancement detecting esophageal diverticulum (14%). Huynh Trudeau., *et al.* [9] demonstrated dysphagia among 41 adult patients who underwent surgery for esophageal atresia at birth. Esophagogastroduodenoscopy was performed in 32 patients in which esophageal diverticulum was present in 13% (n = 4).

Specific treatment of esophageal diverticulum is usually not required, although resection is occasionally necessary for large or symptomatic diverticula. Diverticula associated with motility disorders require treatment of the primary disorder. As the patient had episodic dysphagia, surgical diverticulectomy was planned but the family refused the surgery. Omeprazole was prescribed and there was no progression of esophageal diverticulum during the 6-month follow-up period.

Conclusion

Childhood esophageal diverticulum is exceedingly rare but should be included in the differential diagnosis of dysphagia. Esophageal diverticulum is uncommon sequelae of esophageal dysmotility due to esophageal atresia.

Conflicts of Interest

The authors have no conflicts of interest to disclose.

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