A 15-Year-Old Girl with Dysphagia, Failure to Thrive

Christina Ryan, MD, PhD; Muhammad Khan, MD; Eva Delgado, MD; William Berquist, MD; and Christopher Longhurst, MD

EDUCATIONAL OBJECTIVES

1. Define failure to thrive and review the multiple mechanisms that cause growth failure.
2. Review the pathophysiology of esophageal stricture.
3. Describe the diagnosis and treatment of peptic esophageal stricture, including initial workup, medical and surgical therapy, and prognosis.

A 15-year-old girl presented with feeding intolerance and failure to thrive. One year prior to presentation, she developed difficulty consuming solids, with painful choking episodes upon swallowing, followed by emesis. The mother reported she had performed the Heimlich maneuver during some of the patient’s more severe choking episodes. She transitioned her daughter to pureed foods, which she tolerated for 1 month. At the time of presentation, the patient was able to tolerate only liquids, including 16 oz each of Pedialyte, PediaSure, and papaya nectar daily. The patient’s pediatrician had prescribed both ranitidine and omeprazole for potential reflux without significant improvement.

The patient carried a diagnosis of chromosomal translocation trisomy with associated developmental delay, hypotonicity, hypopituitarism, and epilepsy. She experienced recurrent pneumonias, allergic rhinitis, and asthma. She was full-term, via uncomplicated vaginal delivery. She was able to crawl, but had not walked for 5 years because of worsening seizures. The...
patient’s current medications included budesonide, montelukast, levetiracetam, iron, lamotrigine, hydrocortisone, levothyroxine, and omeprazole. She had no allergies, and immunizations were up-to-date.

Already below the growth curve, she had lost an additional 4 kg over the past year, equivalent to 15% of her body weight (Figure 1A). She had no URI symptoms, fevers, diarrhea, or rashes; urine output was decreased; last bowel movement was 4 days before presentation. Tonic seizures were daily. She was playful with her two siblings, who have the same chromosomal disorder.

The patient’s height and weight were less than the third percentile. Temperature was 97.3°F, pulse 76, respiratory rate 27, blood pressure 117/54, saturating 97% on room air. She was alert, cooperative, appeared younger than stated age, and seemed in no acute distress. She had dysmorphic facial features — prominent ears, small mandible, large incisors, and micrognathia. Neck was supple without lymphadenopathy or thyromegaly. Chest, abdomen, and extremity exam was normal. Complete blood count (CBC) showed white count 3.8, hematocrit 31, platelets 200, normal differential. Electrolytes and free T4 were normal. A modified barium swallow performed 1 month earlier demonstrated no aspiration with thin liquids or purees. Her chest X-ray revealed a radiopaque foreign body below the clavicles in the distal esophagus without obstruction.

EGD revealed a retained penny in the distal esophagus, loosely adherent to the esophageal epithelium at 15 cm from the teeth; it was easily removed. There were no adhesions or strictures at the site of the foreign body, but a tight stricture was seen in the distal one-third of the esophagus, 25 cm from the teeth, spanning 1.5 cm in length, with an opening of 1 mm (Figure 1B). There was no inflammation, ulceration, active bleeding, polyps or other strictures.

Biopsies revealed increased eosinophils at the site of the stricture, suggestive of reflux esophagitis. An esophagram demonstrated normal swallowing with moderate dilation of the upper-to-mid esophagus with to-and-fro peristalsis (Figure 1C). The distal esophagus below the stricture appeared normal. A small amount of contrast intermittently passed through the stricture into the distal esophagus and stomach.

She was subsequently treated with serial mechanical balloon dilation, started on a clear liquid diet, which was slowly advanced, and reflux prophylaxis was re-initiated. On follow-up, she is currently tolerating oral intake, has partially regained her baseline weight, and will undergo a laparoscopic Nissen fundoplication.
DIAGNOSIS
Retained penny in distal esophagus

DISCUSSION
Failure to thrive, progressive feeding intolerance, and significant weight loss are symptoms consistent with dysphagia caused by gastroesophageal reflux, eosinophilic esophagitis, or foreign body; achalasia; structural lesion; celiac disease; or other malabsorptive etiology.1,2

The finding of the retained coin initiated further studies that revealed the reason for her dysphagia — a tight distal esophageal stricture. An esophageal foreign body present for more than 24 hours poses significant risk for perforation and fistulization with mediastinal structures, necessitating prompt removal. While dilatation of the stricture was important for management of the anatomic issue, identification of the factors precipitating the stricture was equally critical. Two likely etiologies included eosinophilic esophagitis and chronic reflux, especially given her classic reflux symptoms — asthma, feeding intolerance, and weight loss.3 However, neither H-2 blockade nor proton pump inhibition yielded symptom resolution. Her dysphagia for solids more than liquids suggested an esophageal-specific diagnosis, including esophageal motility disorder (achalasia, muscular dystrophy, scleroderma) or anatomic esophageal abnormality (foreign body, stricture, web, vascular ring, hiatal hernia).

Alternatively, food intolerance and weight loss may be caused by cystic fibrosis, celiac disease, hyperthyroidism, renal disease, and metabolic disorders. Ultimately, upper endoscopy revealed the foreign body and the associated lesion, likely a peptic stricture from chronic esophageal reflux.4

DIFFERENTIAL DIAGNOSIS
Growth failure has been defined as:
• Height or weight less than the third to fifth percentiles for age on more than one occasion;
• Height or weight falling 2 major percentile lines using the standard growth charts of the National Center for Health Statistics; or
• Presence of documented malnutrition (weight <80% of ideal body weight for age).5

Failure to thrive in children may be classified as nonorganic, organic, or a combination of both.1 Nonorganic failure to thrive usually results from environmental and psychosocial factors and is frequently associated with abnormal interactions between caregiver and child. Etiologies include poor feeding; lack of support; weak parenting preparation; family dysfunction; child neglect; emotional deprivation syndrome; and eating disorders.1,6

Prenatal organic explanations for growth failure include prematurity with complications, maternal malnutrition, toxic exposure in utero, alcohol, smoking, medications, infection, intrauterine growth retardation, and chromosomal abnormalities.7 Postnatal organic causes include inadequate intake, poor absorption and/or nutrient use, or increased metabolic demand.1,6 Inadequate intake may stem from lack of appetite (iron deficiency anemia, chronic infection); poor suck or swallow (CNS or muscular); vomiting (CNS, metabolic, obstruction, renal); or gastroesophageal reflux and esophagitis.2

Poor absorption or nutrient use may result from a gastrointestinal disorder (cystic fibrosis, celiac, chronic diarrhea), renal disease, hypothyroidism, diabetes, growth hormone deficiency, inborn error of metabolism, or chronic infection. Etiologies of increased metabolic demand include hyperthyroidism, chronic disease (heart failure, bronchopulmonary dysplasia), chronic inflammatory conditions (inflammatory bowel disease, systemic lupus erythematosus), renal failure, or malignancy.2,6

Precipitating causes of esophageal stricture include peptic acid, autoimmune, infectious, caustic, congenital, iatrogenic, medication- or radiation-induced and malignant or idiopathic processes.7,8 Conditions that cause distal esophageal strictures include gastroesophageal reflux disease (peptic stricture), Zolliger-Ellison syndrome, adenocarcinoma, collagen vascular disease (scleroderma, systemic lupus erythematosus, rheumatoid arthritis), extrinsic compression, alkaline reflux from gastric resection, Crohn’s disease, sclerotherapy, and prolonged nasogastric intubation. Peptic strictures account for 70% to 80% of esophageal stricture cases, and result from dysfunctional lower esophageal sphincter, disordered motility, or hiatal hernia.

CLINICAL MANIFESTATIONS
Patients may present with heartburn, dysphagia, odynophagia, food impaction, weight loss, and chest pain. Progressive dysphagia for solids with progression to liquids is the most common presenting symptom. The obstruction is usually perceived at a point above or at the level of the lesion. Benign esophageal strictures produce dysphagia with insidious progression (months to years) and minimal weight loss. Malignant esophageal strictures display rapid progression (weeks to months), frequently associated with significant weight loss.3,4,7,8

In patients with esophageal stricture, while CBC results are within reference range, anemia may develop because of chronic bleeding from esophagitis or carcinoma. A complete metabolic panel can assess nutritional status in conjunction with weight loss.4 The etiology of esophageal stricture may be identified using radiologic and endoscopic modalities and confirmed by endoscopic visualization and tissue biopsy.
Esophagogastroduodenoscopy is used to confirm diagnosis of esophageal stricture, seek evidence of esophagitis, exclude malignancy, obtain biopsy specimens, and implement dilation therapy. Histologic findings include edema, cellular infiltration, basal cell hyperplasia, and vascular changes with an increase in type III collagen deposition. Barium esophagram complements endoscopic findings and provides information about stricture location, length, and diameter and contour of the esophageal wall. It identifies diverticula and paraesophageal hernias, and may detect subtle esophageal narrowing caused by rings and peptic strictures greater than 10 mm in diameter. Chest radiography should be obtained if extrinsic compression is a concern. Twenty-four-hour esophageal pH monitoring may assess adequacy of therapy in patients who remain symptomatic despite medical treatment. Endoscopy and manometry evaluate severe esophageal dysmotility. Manometry can be diagnostic when dysmotility is suspected as the primary process.

**TREATMENT**

Most benign esophageal strictures respond to endoscopic, pharmacologic, and/or surgical interventions. Acute treatment often involves serial endoscopic mechanical dilatation. Aggressive therapy for coexistent esophagitis with acid suppression is beneficial for long-term management. Some patients may be candidates for definitive surgical treatment with Nissen fundoplication and G-tube.

**CONCLUSION**

This case depicts the importance of entertaining a complete differential diagnosis in the assessment of a child who presents with growth failure, especially a patient with a condition that has historically limited growth—in this case, the triad of cerebral palsy, developmental delay, and epilepsy related to underlying chromosomal abnormality. It might be tempting to attribute the patient’s symptoms to progression of a previously established condition. However, while her genetic disorder was responsible for baseline suboptimal growth, a new condition exacerbated her growth failure. The patient was admitted to our institution for a reflux work-up, but progressive dysphagia with weight loss clearly indicated that her condition had progressed beyond the need for simple prophylaxis. As the severity of her reflux was sufficient to cause significant esophageal stricture and foreign body retention.

**REFERENCES**