Gastric outlet obstruction

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INTRODUCTION

Gastric outlet obstruction in the pediatric population, after the first few weeks of life, is an uncommon cause of persistent vomiting and most often requires surgical interventions. Our patient presented with a rare etiology of gastric outlet obstruction, with a possible etiology of drug-induced injury, which was amenable to successful nonsurgical management using advanced endoscopic procedures.

CASE

Our patient is a previously healthy, 3-year-old female who presented with three weeks of nausea, vomiting, abdominal distension and loss of appetite. During this period, she was admitted twice for IV fluids for dehydration. These symptoms started acutely within two to three days after she began taking azithromycin and ibuprofen to treat walking pneumonia and had been worsening since. Due to reduced oral intake of initially solids and then liquids, she developed significant weight loss and constipation.

She was admitted to another hospital for further workup. The hospital performed an abdominal ultrasound that showed hypertrophic pylorus, an UGI showing failure of contrast to exit the stomach

Ultrasound with evidence of hypertrophic pyloric muscle
esophagogram that showed no passage of contrast through the stomach, and an unsuccessful upper endoscopy with evidence of gastric outlet obstruction.

She was transferred to Children’s Hospital of Wisconsin for further gastroenterology care and management. Here, we started her on IV nutrition, placed her nasogastric tube to suction and corrected her metabolic acidosis with intravenous fluids. Once stabilized, she was taken to the operating room for a repeat upper endoscopy, during which we found some ulcers, erythema and erosions in the esophagus and stomach, with complete occlusion of her pylorus. We carefully passed a guide wire through the possible small opening of the pylorus and confirmed the position by fluoroscopy. We then threaded a balloon dilation catheter over the guide wire into the duodenum under fluoroscopic guidance. The balloon was gradually inflated with increasing pressures to dilate the pylorus successfully. After dilation, we injected Botox around the pylorus. When we then passed the endoscope into the duodenum with extensive manipulation, the area appeared normal.

The patient’s nasogastric tube was discontinued prior to discharge. She went home two days after the procedure on sucralfate and proton pump inhibitors with diet advanced to high-calorie oral liquids. She did well for a week or so until she was readmitted for recurrence of similar symptoms. She underwent a repeat upper endoscopy and dilation two weeks after the first procedure. The endoscopy showed a significantly improved esophagus and stomach. Next, we passed a guide wire under fluoroscopy, followed by the dilation catheter. We performed serial dilations at a pressure and diameter greater than the previous dilations. We noted a porous transparent vascular membrane with extensive surrounding granulation tissue beyond the dilated area. Despite extensive manipulation, we were unsuccessful in passing the scope into the duodenum. To assess the obstruction, we performed a contrast study, which showed dilated stomach and small amounts of contrast flowing into the duodenum.

The patient was discharged on proton pump inhibitors and a high-calorie liquid diet with instruction to return in one week for reassessment and possible repeat dilation. During this third dilation procedure we were able to enter the duodenum using a neonatal scope. Due to persistent narrowing of the pylorus, we repeated serial dilations under direct fluoroscopy in conjunction with the Interventional Radiology team. Biopsies taken from the pylorus were consistent with drug-induced injury to the strictured area.

We continued her on a high-calorie liquid diet and allowed gradual advancement as tolerated. Now, five months after her last dilation, she is completely asymptomatic, on a regular diet, off all medications and growing well.
DISCUSSION
In the pediatric population, gastric outlet obstruction can have two possible etiologies, mechanical and functional. Mechanical obstruction happens when the exit to the stomach is narrowed but the gastric nervous and muscular systems are intact. The causes of mechanical obstruction can be perinatal or postnatal. The perinatal causes mainly include anatomical abnormalities as antral webs, congenital gastric atresias, pyloric stenosis, annular pancreas or gastric duplication cysts or hypertrophic pyloric stenosis, though most of these present at a later age. On the other hand, postnatal causes range from more common peptic ulcer disease or drug-induced gastric ulcers and healing burns from caustic ingestion to more uncommon causes as eosinophilic gastroenteritis or Crohn’s disease and other granulomatous diseases, both infectious and noninfectious. The commonly implicated drugs include non-steroidal anti-inflammatory drugs, macrolides, fluoxetine in pregnancy, opium/opiates and Lipitor®. The common causes of luminal obstruction include gastric bezoar, percutaneous endoscopic gastrostomy tube migration or prolapse, malrotation or volvulus, gastric polyps, diaphragmatic hernia, acute or chronic pancreatitis causing external compression or adhesions, pancreatic pseudocyst, hematomas (traumatic or Henoch-Schönlein purpura), duodenal hamartoma, extrinsic band, lymphomas or malignancy, etc. Functional causes of gastroparesis include neurological causes that could be acute, secondary to viral infections, electrolyte abnormalities and surrounding tissue inflammation, or chronic as seen in uncontrolled diabetes; and intrinsic muscular diseases as seen in mitochondrial disorders and cerebral palsy.

Management of gastric outlet obstruction depends primarily on the identified etiology. A patient’s workup starts with an abdominal X-ray, which can identify an enlarged stomach and possible bezoar or migrated percutaneous endoscopic gastrostomy tube. Other investigations include esophago-gastro-duodenogram or upper gastrointestinal series, abdominal ultrasound, and CT or MRI as required. Most patients also need an endoscopy, which may be diagnostic and/or therapeutic. In a pediatric population, we attempt to manage most cases conservatively. The majority of these patients present with significant esophageal and gastric irritation due to obstruction of flow and acid reflux and hence require adjunct medical therapy with proton pump inhibitors, sucralfate or steroids. With an advanced endoscopic approach we can do pneumatic dilations, place stents, ablate antral webs or inject Botox, but most children ultimately require surgical interventions with antral web resection, partial gastrectomy, antrectomy or Billroth II operation.

CONCLUSION
With advanced endoscopic therapeutic procedures we may be able to completely avoid the need for surgical interventions and it should always be considered in appropriate cases prior to surgery.
To refer a patient, visit chw.org/refer or call (414) 266-2460 or toll-free (800) 266-0366.

To make an appointment, call Central Scheduling at (414) 607-5280 or toll-free (877) 607-5280.

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References: