



**Society of Radiologists in Ultrasound
2012 Toshiba Residents Program**

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Clinical History:

This is a white male neonate with feeding intolerance and non-bilious vomiting. Prenatal course was uneventful; he was born at term without complications and discharged home on hospital day 2. On day 3, he was re-admitted with persistent non-bilious vomiting.

Figures:

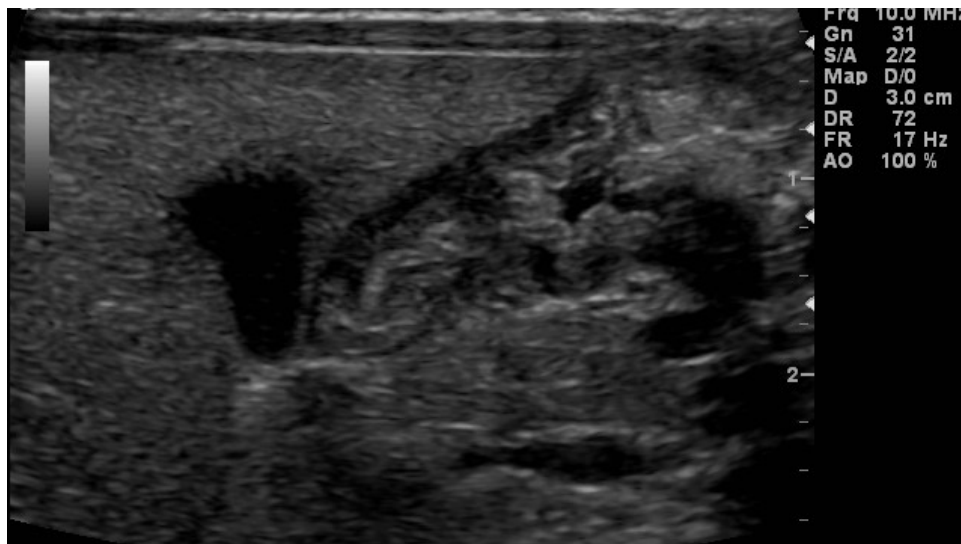


Figure 1. Initial abdominal ultrasound. No pathology was identified on this study. The image above shows a collapsed gastric antrum.



Figure 2. Upper GI with barium contrast injected into nasogastric tube. This image shows total obstruction at the gastric outlet.



Figure 3. 2 hour delayed AP radiograph following upper GI study shows paucity of bowel gas with no movement of oral contrast beyond the gastric antrum.



Figure 4. Abdominal ultrasound repeated three days after initial workup revealing a 2.6mm thick structure traversing the gastric antrum.

Final Diagnosis:

Congenital Gastric Atresia

Discussion:

Congenital gastric atresia is a rare cause of vomiting in early infancy, accounting for less than 1% of all congenital gastrointestinal obstructions.¹ Gastric atresia is important to recognize because it can be misdiagnosed if clinical suspicion is low. The case above is unique because this neonate was initially worked up for pyloric stenosis with ultrasound. The study was negative and he subsequently underwent an upper GI which showed total obstruction at the gastric outlet. A second ultrasound was performed which was diagnostic for congenital gastric atresia with a membranous diaphragm.

Congenital gastric atresia is usually limited to the gastric antrum or pylorus. There are 3 types of gastric atresia. The first type involves no connection between the lumen of the stomach and the duodenum. The second type involves a fibrous band connecting the stomach and the duodenum. The third type is most common and involves a membranous diaphragm between the stomach and duodenum. This membrane is thought to be due to localized vascular occlusion during fetal life, rather than failure of recanalization. Hereditary link is poorly understood, however, this anomaly has been associated with epidermolysis bullosa. This association is

thought to be due to inflammatory stricturing at the gastric antrum. Gastrointestinal atresias can also be associated with VACTERL syndrome, microgastria and polysplenia/asplenia syndromes.²

Congenital gastric atresia usually presents as non-bilious vomiting, and may present at birth or later in childhood depending on the size of the defect. Incomplete diaphragms present later due to the ability for some gastric contents to pass through the membrane. It has been proposed that some diaphragms may rupture spontaneously, making the diagnosis more prevalent than the literature would suggest. Thicker membranes require surgical resection to relieve the obstruction and are typically diagnosed in the first few days of life.³

A plain radiograph of a patient with gastric atresia shows a dilated stomach with paucity of distal bowel gas. An upper GI would then reveal complete obstruction at the gastric outlet. The ultrasound above is diagnostic in that it demonstrates the membranous diaphragm. In figure 4 above, note the band-like structure in the pre-pyloric region which extends across the stomach. This represents a hypoechoic submucosal layer covered by echogenic mucosa on either side. Sonographic findings typically show a distended stomach with a collapsed duodenum distal to the gastric outlet obstruction.⁴ The upper GI study above (fig. 2) shows an enlarged stomach with total obstruction at the gastric antrum and no distal bowel gas. The upper GI may also show a thin, linear filling defect in gastric antrum.³ Further imaging would not be necessary for diagnosis in this case.

The differential diagnosis for upper GI obstruction in the first few weeks of life is broad, but can easily be narrowed once the level of obstruction is determined. Esophageal anomalies include esophageal atresia and tracheoesophageal fistula, where there is narrowing of the esophagus and/or tracts between the esophagus and the trachea. Esophageal duplications are less common and involve a cystic mass which compresses the esophagus. Vascular rings can also cause compression of the esophagus, resulting in vomiting and failure to thrive. The primary modality for diagnosing esophageal anomalies is the esophagram.¹

The stomach is less likely to have congenital malformations causing obstruction because it undergoes less alteration in form during development. In addition to gastric atresia, other anomalies affecting the stomach are pyloric stenosis and ectopic pancreatic tissue. Hypertrophic pyloric stenosis is a more common type of gastric obstruction which is not caused during embryonic development, and typically presents in the second week of life. Gastric duplication is another etiology of gastric outlet obstruction which accounts for 7% of all gastrointestinal duplications.¹

Obstructive anomalies involving the duodenum are much more common than those of the stomach or esophagus. Obstructions occurring distal to the ampulla of Vater typically cause bilious vomiting. More common duodenal obstructions include duodenal atresia, annular pancreas and midgut volvulus. Ladd bands, preduodenal portal vein and duodenal webs are less common causes of duodenal obstruction. Duodenal duplications can also cause duodenal obstruction and are rare.

Radiological investigation is essential for the workup of these anomalies. Ultrasound is one of the more important diagnostic tools due to the lack of radiation and speed of diagnosis. It is helpful for differentiating congenital gastric outlet obstruction from hypertrophic pyloric stenosis. It is also helpful for diagnosis of duplication cysts. It can also be useful in diagnosing malrotation and midgut volvulus.¹ With increasing healthcare costs and concerns for radiation safety, ultrasound will continue to have an increasing number of applications in diagnosing anomalies of the upper gastrointestinal tract.

References:

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