Common Pediatric Surgical Conditions – Dr. Nian

1- *Neonatal intestinal obstruction*
   a. Intrinsic e.g. cystic fibrosis, which may present with intestinal obstruction from inspissated meconium.
   b. From the wall .e.g. Intestinal Artesia and stenosis, where the bowel obstructed by a web.Hirshsprung’s disease the bowel obstructed by agangionosis of the bowel.
   c. Extrinsic: as in malrotation by Ladd’s band, or by a duplication cyst.

2- *Esophageal abnormalities*
The most common abnormalities are: gastro-esophageal reflux and tracheoesophageal malformations.

3- *Congenital and acquired ano-rectal diseases.*

4- *Appendicitis, meckel’s diverticulum, intussusceptions.*

5- *Inguinoscrotal disorders and penis congenital malformations.* E.g. Inguinal hernia, hydrocele, acute scrotum, undescended testis, hypospadias, phymosis and paraphymosis.

6- Other less common congenital malformations which need surgical correction like congenital diaphragmatic hernia, umbilical hernia, Ranula,Torticollis, and Thyroglossal Cysts and Fistula.

The Lesions of the Stomach
The stomach forms from the foregut and is recognizable by the fifth week of gestation. It then elongates, descends, and dilates to form its familiar structure by the seventh week of gestation. The vascular supply to the stomach is very robust, and ischemia of the stomach is rare. The stomach is supplied by the right and left gastric arteries along the lesser curvature, the right and left gastroepiploic arteries along the greater curvature, and the short gastric vessels from the spleen. There is also contribution from the posterior gastric artery, which is a branch of the splenic artery, as well as the phrenic arteries.

HYPERTROPHIC PYLORIC STENOSIS

Incidence
- PS occur in 1-3 of every 1000 live births in the United States, the incidence is increasing
- 4:1 male to female ratio remains constant.
- The etiology of PS remains unknown, but may have genetic contribution.

Presentation
The infant with PS is usually a full-term infant, with the onset of non-bilious projectile vomiting between 3-6 weeks of age, and can present with significant weight loss and failure to thrive.

Physical examination
The diagnostic finding is a mobile, avoid mass in epigastrium or right upper quadrant, upper abdominal distension with visible gastric peristaltic waves also may be seen.

Investigation:
- Sometimes needed to confirm the diagnosis.
- U/S: shows increase muscle thickness and pyloric channel length.
- Upper GIT contrast study: shows string sign and shoulder sign.
Differential Diagnosis
Are the differential diagnosis for non bilious vomiting includes over feeding, gastroesophageal reflux, elevated intracranial pressure, or anatomic abnormalities causing gastric outlet obstruction.

Treatment
1- Fluid and electrolyte resuscitation
Prolonged vomiting produces losses of H⁺ and Cl⁻ ions, with lesser losses of Na⁺ and K⁺ ions causing hypochloremic, hypokalemic alkalosis
-initial fluid resuscitation should be with normal saline in boluses of 10-20 ml/kg. A continuous infusion of 5% or 10% dextrose in 0.45% saline should then be started at 1.5 times the calculated maintenance rate, potassium supplementation added to the infusion when the fluid volume is properly restored.

2- The definitive treatment is surgical (Pyloromyotomy).

3-balloon dilatation may represent therapeutic option when direct surgical repair is not possible.

PYLORIC ATRESIA
- Pyloric atresia is a rare disease (1:100,000 live births). PA may occur as a web, a cord, or a gap between the antrum of the stomach and the first portion of the duodenum.
- These infants may have similar electrolyte abnormalities to infants with hypertrophic pyloric stenosis and presents as symptoms of gastric outlet obstruction.
- The disease is difficult to characterize because it is so rare. PA may be associated with epidermolysis bullosa and other gastrointestinal anomalies, such as duplications.
- PA is diagnosed with a “single bubble” on the abdominal radiograph. The diagnosis may be confirmed with a contrast study. Repair is performed after resuscitation. Repair is usually with an anastomosis (gastroduodenostomy).

GASTRIC PERFORATION
- The causes of gastric perforation are spontaneous perforation in the newborn, iatrogenic perforation from instrumentation, peptic ulcer disease (very rare), and trauma (About half of neonatal perforations are spontaneous, and the other half are iatrogenic). Neonatal gastric perforations most commonly occur in premature infants with an increased mortality.
- Gastric perforation usually presents as abdominal distention and signs of sepsis or shock related to the perforation. The diagnosis is suspected when a large amount of extraluminal gas is seen on an abdominal radiograph.
- The perforations are usually managed with laparotomy or laparoscopy. The perforation can usually be closed primarily with or without an omental patch.

Congenital microgastria
- Congenital microgastria is a rare disorder that usually occurs in conjunction with other congenital anomalies or, more rarely, alone.
- Associated anomalies include the VACTERL association (Vertebral anomalies, Anorectal atresia, Cardiac anomalies, TracheoEsophageal fistula and esophageal atresia, Renal and Limb anomalies), tracheoesophageal cleft, malrotation, and asplenia.
- Operative intervention consists of jejunal feedingtubes and gastric augmentation.
ANTRAL WEB

- The etiology is unknown and is generally thought to be congenital or the result of an inflammatory process. In adults there has been a case reports that strongly suggested peptic ulcer disease can lead to antral web.
- The patient presents with a typical gastric outlet obstruction. In the infant, antral web may be confused with hypertrophic pyloric stenosis. The abdominal examination may be normal. The patient with antral web may have a normal abdominal sonogram. However, an upper gastrointestinal series will show the lesion.
- Treatment of an antral web consists of resuscitation and operative correction. The procedure can be completed with laparotomy or laparoscopically.

GASTRIC VOLVULUS

- Gastric volvulus can occur from primary or secondary causes. Primary gastric volvulus is thought to be due to laxity of the gastric ligaments. Secondary disease may occur due to a paraesophageal hernia or other diaphragmatic hernia.
- The average age at presentation is 2.5 years. Equal numbers of males and females are affected.
- The presenting symptoms can be intermittent or complete gastric obstruction, pain, intractable retching, and/or bleeding. The most common signs of gastric volvulus in children include acute abdominal pain, and the inability to pass a nasogastric tube into the stomach lumen.
- Gastric volvulus is classified into categories based on the axis of gastric rotation. Mesenteroaxial gastric volvulus is rotation about the gastric short axis.
- Organoaxial gastric volvulus is rotation around the long axis of the stomach.
- Treatment consists of patient resuscitation, nasogastric decompression, and surgical correction. The volvulus is reduced and any diaphragmatic defects are repaired in secondary gastric volvulus. A gastropexy is then performed. This has traditionally been accomplished with a gastrostomy tube or button. However, there have been several recent reports of successful laparoscopic gastropexy.