Neonatal Intestinal obstruction – Dr. Nian

Congenital intestinal obstruction occurs in approximately 1:2000 live births and is one of the most common causes of admission to a neonatal surgical unit, accounting for up to one third of all admissions. Morphologically, congenital defects related to continuity constitute one of the most common causes of neonatal intestinal obstruction.

**Duodenal Obstruction**

**Classification:**
1/ Stenosis or incomplete obstruction may be due to a diaphragm or web with a small opening.
2/ Atresia or complete obstruction; May be seen with duodenal muscular continuity or with a gap that is usually filled in with pancreatic tissue.

**Diagnosis:**
1/ Polyhydramnios resulting from high intestinal obstruction with dilated stomach and proximal duodenum are seen on antenatal ultrasonography.
2/ the new born presented with bile stained vomiting within hours of birth, abdominal distension may not be evident owing to vomiting.
3/ Up right abdominal radiograph.
   Shows double bubble appearance in case of complete obstruction

**Treatment:**
1/ Gastric decompression and fluid and electrolyte correction.
2/ Operative correction, the operation preferred by most is a diamond –shaped or side to side duodeno – duodenostomy.

**Jejunoileal Artesia and Stenosis**

**Etiology**
The most favored theory is that a localized intrauterine vascular accident with ischemic necrosis and subsequent reabsorption of the affected segment or segments.

**Pathology**
The morphologic classification of jejunoileal atresia has been of significant prognostic and therapeutic value:
1/ stenosis;
   Defined as a localized narrowing of the intestinal lumen without disruption of the continuity or defect in the mesentery.
2/ Atresia type I:
   The obstruction is caused by a membrane or web formed by mucosa and sub mucosa without mesenteric affect.
3/ Artesia type II:
   In this type, blind bowel ends joined by a fibrous cord along the edges of an intact mesentery.
4/ Atresia type III (a):
   There are disconnected blind ends, with a v-shaped mesenteric defect of varying size.
5/ Atresia type III (b):
   Apple peel or Christmas tree deformity consist of a proximal jejunal atresia, large mesenteric defect, the distal small bowel assumes a helix configuration around a single perfusing vessel.
6/ Atresia type IV:
   There is multiple segments atresia of a combination of type I to III.

**Diagnosis:**
The diagnosis of jejunoileal atresia can usually be made by radiographic examination of the abdomen wit. Swallowed air reaches the proximal bowel by 1 hour and the distal small bowel by 3 hours in a normal infant. Jejunal atresia patients have a few gas-filled and fluidfilled loops of small bowel, but the remainder of the abdomen is gasless. Because haustral markings are rarely seen in neonates,distal ileal atresia may be difficult to differentiate from colonic atresia
   A limited-contras meal may be useful if intestinal stenosis is suspected.
Surgical consideration
The operative procedure depends on the pathologic finding; resection of the proximal dilated and hypertrophied bowel with primary end-to-end anastomosis is most common. Intraoperative injection of saline into the distal bowel to confirm distal bowel patency is necessary to exclude multiple atresia.

Malrotation
Rotational anomalies create a spectrum of anatomic conditions with critical importance to the pediatric surgeon. Rotational anomalies may be isolated or occur as an intrinsic component of gastrochisis, omphalocele, or congenital diaphragmatic hernia. In children with malrotation, the bowel is not fixed adequately and is thus held by a precariously narrow-based mesentery.

The most common forms of rotational disorders include nonrotation, incomplete rotation, and reversed rotation. In nonrotation, there is failure of the normal intestinal 270-degree counter-clockwise rotation around the superior mesenteric artery. Midgut volvulus due to a narrow mesenteric pedicle and extrinsic duodenal obstruction secondary to abnormally positioned cecal attachments are significant risks.

In cases of incomplete rotation, normal rotation has been arrested at or near 180 degrees. The cecum will usually reside in the right upper abdomen. Obstructing peritoneal bands are present.

With reversed rotation, an errant 90-degree clockwise rotation occurs. Reverse rotation with volvulus may occur with obstruction of the transverse colon.

Stages of midgut development
1/ Herniation of the primary midgut loop which occur into the base of the umbilical cord, at 5th gestational week.
2/ Rotation
3/ Retraction of the extracoelemic intestine; between 10-12 gestational weeks.
4/ positioning process is the final step, where the fixations of the intestine to the posterior body wall occur. The duodenojejunal junction becomes fixed in the left upper abdomen while the cecum is anchored in the right lower quadrant.

Malrotation results from interruption of the aforementioned embryological events.

Diagnosis
1/ clinically: bilious emesis is a cardinal manifestation, but abdominal distension not present in absence of volvulus, or presented with septic shock from a catastrophic midgut volvulus, abdominal wall erythema and hematemesis or melena from progressive mucosal ischemia.

Malrotation may present as an incidental, subtle finding discovered during the radiographic evaluation of another diagnosis, or present less dramatically as failure to thrive, gastroesophageal reflux, and early satiety.

The diagnosis becomes more challenging with the older child or teenager because the symptoms are often very vague.

2/ Abd-X-R: shows distended stomach and proximal duodenum, (“double bubble” sign) may be observed with paucity of air in the distal small bowel.

3/ Contrast study: shows incomplete obstruction of distal duodenum with appearance of extrinsic compression described as a“ bird’s beak”, positioning of the duodenojejunal flexure to the right of the spine.

Treatment:
Malrotation require operative management even in older asymptomatic patients because there is no upper limit to the age at which one is at risk for volvulus and bowel ischemia.

If present, mid gut volvulus is relieved by rotating the affected small intestine opposite direction of torsion, generally in a counterclockwise direction. The recurrence of the volvulus is prevented by broadening the base of the mesenteric vascular pedicle by dividing the peritoneal bands that tether the cecum, small bowel mesentery, mesocolon, and duodenum around the base of the superior mesenteric artery.

Ladd’s bands from the cecum to the right abdominal wall must be divided to relieve any extrinsic obstruction of the duodenum.

Then the additional need to demonstrate complete duodenal luminal patency. This can be done operatively by injecting air or saline and demonstrating adequate distal progression.

Appendectomy is considered standard because the malposition of the cecum and the attached appendix can make acute appendicitis a difficult diagnosis to make if it were to develop in the future.

At the conclusion of the procedure, the intestine is replaced into the abdomen without mesenteric torsion, generally, the small intestine on the right, the cecum and colon on the left.
MECONIUM DISEASE
The genetically determined disease cystic fibrosis is the predominant cause of meconium disease in newborns. Meconium ileus in the newborn is the earliest manifestation of CF and occurs in approximately 15% of CF patients. The CF gene is autosomal recessive.

Presentation;
The clinical presentation of meconium ileus results from obstruction of the distended meconium-filled small bowel, which narrows to an unused distal ileum impacted with inspissated pellets of colorless mucus. Approximately one half of these neonates present with a simple uncomplicated obstruction. The remaining patients present with complications of meconium ileus, including; volvulus, gangrene, atresia, and perforation, which may result in meconium peritonitis and giant cystic meconium peritonitis.

Diagnosis;
Clinically
Newborns with uncomplicated meconium ileus can appear healthy immediately after birth. However, within 1 to 2 days, they develop abdominal distention and bilious vomiting. Normal meconium will not be passed; dilated loops of bowel become visible on examination and have a “doughy” character that indents on palpation.
- Complicated meconium ileus may present dramatically and immediately after birth, with signs of peritonitis (abdominal distention, tenderness, abdominal wall erythema), as well as clinical evidence of sepsis. Abdominal distention can be so severe as to cause immediate respiratory distress. A palpable mass suggests pseudocyst formation, which results from in utero bowel perforation. Often the neonate is in extremis and needs urgent resuscitation and surgical exploration.

Abdominal XR show more variation in the bowel loop caliber, fewer air fluid levels, and more meconium mottling (ground glass appearance).

- Contrast colon enema, is most diagnostic study, which may also therapeutic.

- The only reliable and definitive test for CF is a pilocarpin iontophoresis sweat test.

Treatment:
Many newborns with simple meconium ileus can be managed nonoperatively, by repeated contrast enema with hyperosmolar enema to loosen the meconium if it does not appear to be complicated by perforation and peritonitis, pseudosyct, or atresia.
Under fluoroscopic control, a 25% to 50% dilution of the hyperosmolar enema is slowly infused at low hydrostatic pressure Gastrograffin are commonly used contrast materials for Meconium Ileus Patients. If the enema fails to promote passage of meconium within 24 to 48 hours, or two attempts at washout are unsuccessful, an operative approach is indicated.

Operative treatment;
Resecting the dilated ileum and forming an ileostomy with the advent of total parenteral nutrition, and the use of N-acetylcysteine. Care must be taken to replenish fluids, electrolytes, and nutrients in accordance with the stoma output, Stomas placed in the course of surgical management should be closed as soon as possible (4 to 6 weeks) to help avoid prolonged problems with fluid, electrolyte, nutritional losses, and cholestatic jaundice.