PEM GUIDE – MALROTATION / INTESTINAL OBSTRUCTION

INTRODUCTION
The surgical abdomen in the neonate is a rare event, but is associated with a very high morbidity and mortality. It can quickly lead to severe dehydration, hypoglycemia, electrolyte imbalance, and to irreversible ischemia to the intestine. Early recognition and early intervention are crucial.

In almost every case of an intestinal obstruction the neonate will present with vomiting. Vomiting is rare in the neonatal period or early infancy and should always raise concern of a serious underlying disease. Inexperienced parents may confuse spitting up secondary to food-regurgitation and overfeeding with vomiting. It is helpful to know the amount a newborn is normally feeding (Approx: 1/6th of its body weight a day = 1 oz / kg body weight / every 4 hrs)

Age and presenting symptoms may help in the differential diagnosis of the surgical abdomen in the newborn and early infancy: Bilious vomiting suggests a level of obstruction below the sphincter of Odi. Abdominal distension (as apposed to gastric distension) occurs due to more distal obstructions
• Congenital GI obstruction in a lethargic neonate with bilious vomiting
• Pyloric stenosis in a young infant if there is recurrent non-bilious projectile vomiting
• Intussusception in an infant beyond neonate period (peak 5-10 months of age) who’s vomiting occurs with bouts of pain, a change in mental status, or bloody stools.

INITIAL MANAGEMENT
• IV access, fluid resuscitation with colloidal fluids (NS, LR)
• CBC/diff, Electrolytes, Dextrostick
• Blood Culture, Catheterized UA/Urine Culture – LP if rectal temp > 38 (100.4)
• XRAY - single abdomen supine AP and upright with chest AP
• Consider Orogastric or nasogastric tube (Fr 8-10)
  • Avoid in pyloric stenosis
• Surgical consult, Radiology consult
## INTESTINAL OBSTRUCTION IN THE NEONATE

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### MALROTATION

During embryonic life, the colon and small bowel grow rapidly rotating in a counter clockwise direction, with the cecum passing anterior the superior mesenteric artery and coming to rest in the right lower quadrant. In patients with malrotation, the rotation ceases after the first 90 degrees, and the duodenum and ascending colon are juxtaposed around the superior mesenteric vessels, with the entire midgut suspended from a narrow mesenteric pedicle.

The malrotated bowel does not in itself cause any significant problem. However, two distinct clinical situations may complicate malrotation. The first and most severe is midgut volvulus. The midgut can at any time twist around it’s narrow mesenteric pedicle resulting in a midgut volvulus. Progressive bowel strangulation then results in an ischemic loss of extensive bowel. The second complication is duodenal obstruction due to Ladd’s band.
**MIDGUT VOLVULUS**
Most patients with malrotation develop midgut volvulus within the first weeks of life. But volvulus can also happen later. Bilious vomiting is the initial symptom. The abdomen may or may not be distended. The clinical course is acute with rapid progression to midgut ischemia, unstable hemodynamics, intractable metabolic acidosis and necrosis with perforation. Depending on the progression of the strangulation the xray will show the picture of small bowel obstruction or lack of any gas in the abdomen. Obstruction at the second portion of the duodenum and “corkscrew” appearance of the small bowel on the upper gastrointestinal (GI) contrast study are diagnostic. Urgent surgical treatment should be considered once the diagnosis is established.

**DUODENAL OBSTRUCTION DUE TO LADD’S BANDS**
With normal rotation the cecum migrates to the right lower quadrant and the cecal mesentery attaches to the abdominal wall in that location. In malrotation, the cecum remains in the right upper quadrant. The cecal mesentery (Ladd’s Bands) crosses the duodenum to attach to the liver. These bands can result in duodenal obstruction. Infants present early in life with bilious vomiting.

**DUODENAL ATRESIA AND JEJUNOILEAL ATRESIA**
Duodenal atresia is a congenital obstruction of the second portion of the duodenum. Its etiology is believed to be failure of canalization of this bowel segment during the early gestational stage. Jejunoileal atresia is caused by a mesenteric vascular accident during fetal life. In such intestinal obstructions pregnancy is associated with polyhydramnios.

The neonate with duodenal atresia presents with feeding problems from day 1 of life on. In 80 percent of these patients, the papilla of Vater opens into the proximal duodenum and the vomiting is bilious. Abdominal plain film shows a characteristic "double-bubble" sign, demonstrating the bubbles in the stomach and the dilated proximal duodenum; this confirms the diagnosis.
Surgery is required but is not urgent. A 24- to 48-hour delay may be allowed before operation for transport, further evaluation and fluid resuscitation.

In jejunoileal atresia abdominal distention with bilious vomiting is observed within the first 24 hours after birth.

Abdominal films show air-fluid levels proximal to the lesion, confirming the diagnosis of bowel obstruction.

Preoperatively, stomach decompression, intravenous hydration, and correction of any electrolyte disturbance must be achieved. An interval of 12 to 24 hours is allowed for preoperative preparation.