NEONATAL INTESTINAL OBSTRUCTION

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NEONATAL INTESTINAL OBSTRUCTION (NIO)

• Definition:

An intestinal obstruction occurring during the first month of life.

CAUSES OF NIO

- Esophageal:
 - Atresia (TOF)
- Gastric
- Duodenal:
 - Atresia
 - Stenosis
 - Diaphragm
 - Malrotation with bands or volvulus
 - Annular pancreas

CAUSES OF NIO

• Jeujunal & ileal obstruction:

- Obstructed inguinal hernia
- Intussusception
- Atresia
- Stenosis
- Meconium ileus
- Peritoneal bands or herniae
- Duplication cysts

CAUSES OF NIO

- Large bowl obstruction:
 - Hirschsprung's disease
 - Anorectal anomalies
 - Meconium plug syndrome
 - Atresia (rarest)
- Necrotizing enterocolitis
- Complicated Inguinal Hernias

GENERAL PRINCIPLES

Any baby presenting with persistent, bile stained vomiting should be considered to be surgical emergency until proven otherwise.

<u>Clinical picture:</u>

The cardinal signs are 2C, 2V, 2D

Colics, Constipation

Vomiting, Visible peristalsis

Distension, Dehydration

MANAGEMENT

First aid:

- Gastric decompression (naso-gastric tube)
- IV line (for fluid replacement)
 - Deficit therapy
 - Maintenance
- Urinary catheter (to monitor urine output)
- Fluids:
 - Lactated Ringer's solution 10-20ml/kg over an hour, to be repeated according to response
- Drugs:
 - To cover anaerobes as well as Gram stained bacteria



Definition:

The main pathology is atresia of the esophagus! Tracheoesophageal fistula is secondary to that. $\frac{323}{4}$

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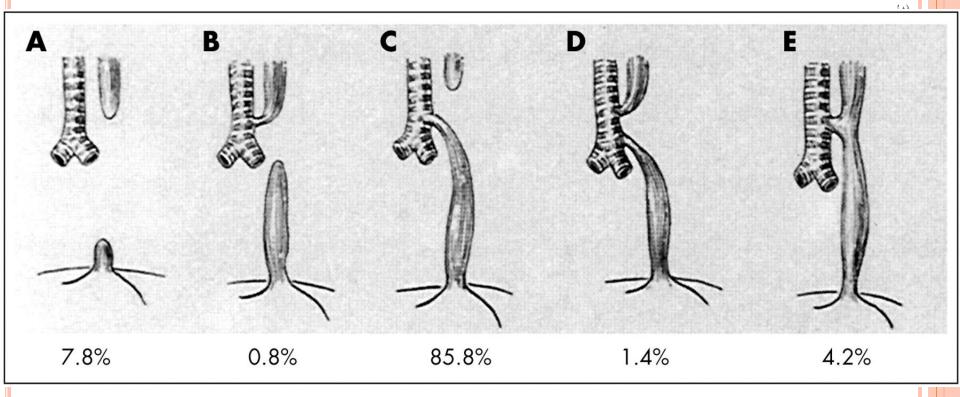
Incidence:

•1-2500 to 1-10000
•males slightly more affected than females
•the second sibling of an affected child has 0.2-0.5% chance of being also affected <u>Associations:</u>

Other congenital anomalies occur in 50-70% of infants affected with esophageal atresia. These are most common with esophageal atresia and distal fistula. Anomalies include cardiovascular (the majority), genitourinary, gastrointestinal and skeletal.

VACTERL association consists of Vertebral, Anorectal, Cradiac, Tracheo-Esophageal, and radial Limb deformities. It is not correlated with a known genetic abnormalitysor syndrome.

Types:



D.Diagnosis

<u>Antenatal:</u>

Maternal ultrasound

Postnatal:

Tracheoesophageal fistula present within the first few hours of life by:

- excessive salivation
- respiratory distress
- cyanosis
- inability to pass a nasogastric tube.
- Attempts at feeding cause choking, coughing, cyanosis, regurgitation.

<u>Radiography:</u>

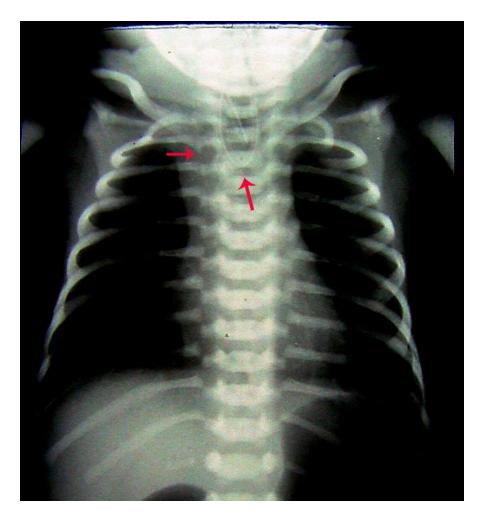
•Plain X-ray

Contrast study(pouchogram)

Plain X-ray With Ryle's tube

Note:

Coiling loop denotes approximate length of upper pouch of esophagus.
Presence of air in the abdomen denotes a distal fistula.



MANAGEMENT

The treatment is ultimately surgical but management strategy include also preoperative, operative and postoperative measures

Preoperative evaluation includes a an Echo and Bronchoscopy: why?

Surgical treatment:

Division and closure of the fisula and primary anastomosis of the two esophageal segments.

If the "Gap" between the two segments is too long, the infant is managed temporarily with an esophagostomy and gastrostomy. Later in life an procedure to replace the esophagus is considered e.g. colon interposition bypass.

CONGENITAL HYPERTROPHIC PYLORIC STENOSIS (CHPS)

Any neonate presenting with projectile, non bilious vomiting, associated with hunger and constipation should be considered CHPS

• Incidence:

- 8:1000
- M/F ratio=4:1
- More in first born babies
- More in infants born to a mother who had suffered from CHPS
- More during spring and autumn!

• Etiology:

- Unknown, but appears to be polygenic (environmental, and genetic)
- Drugs?

• Pathology:

- Progressive hypertrophy of circular pyloric muscles
- Persistent vomiting leads to development of hypochloremic hyponatremic alkalosis and dehydration

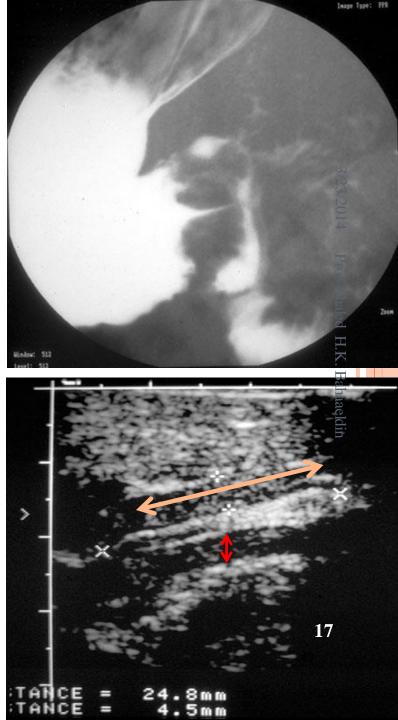
• Clinical picture:

• Symptoms:

- projectile, progressive, non bilious vomiting after which the baby is hungry and ready to suckle again. Classically the symptoms between 3-12 weeks after birth
- Signs:
 - Signs of dehydration which may be severe and life threatening.
 - Visible peristalsis in the upper abdomen can usually be seen after the baby is given a test feed followed by projectile vomiting.







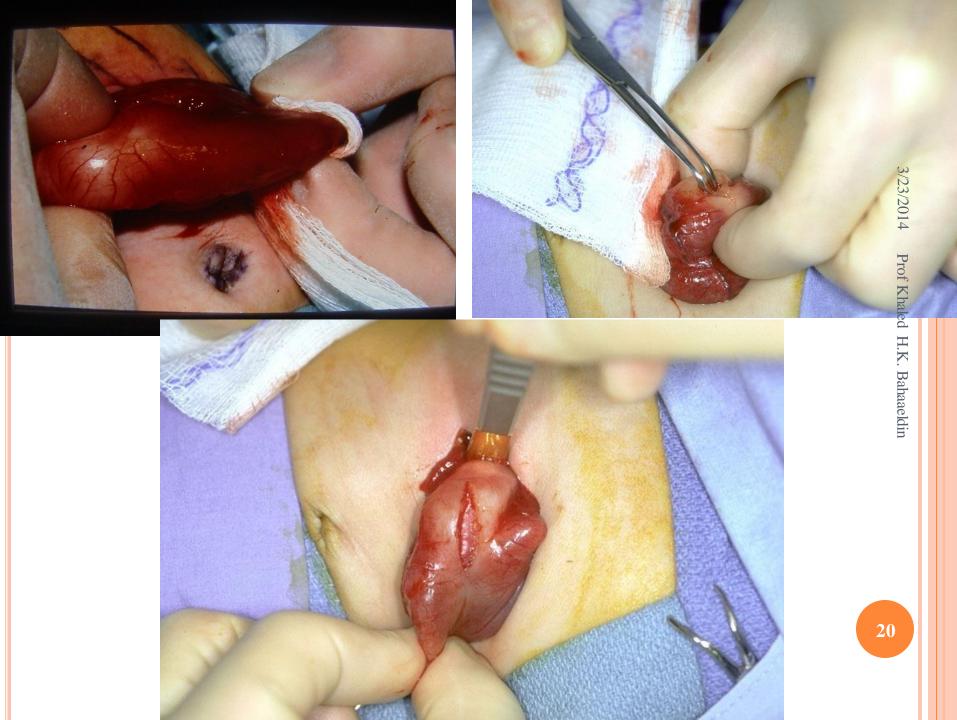
o Differential diagnosis:

- Gastroenteritis
- Gastro-esophageal reflux
- Other obstructive lesion of the gut
- Increased Intracranial tension

• Investigations:

- Imaging: **sonography** and contrast X ray
 - pyloric channel length (normal 11 mm, pyloric stenosis > 15-18 mm),
 - pyloric muscle length (normal 13-17 mm, pyloric stenosis > 19-21 mm),
 - pyloric muscular wall thickness (normal < 2 mm, pyloric stenosis > 3-5 mm),
 - pyloric diameter (normal < 10-15 mm, pyloric stenosis > 10-15 mm)

- Laboratory: to detect and correct electrolyte and metabolic disturbances
- Treatment:
 - First aid treatment to correct dehydration and metabolic disturbances
 - Definitive surgery, the Ramstedt's pyloromyotomy



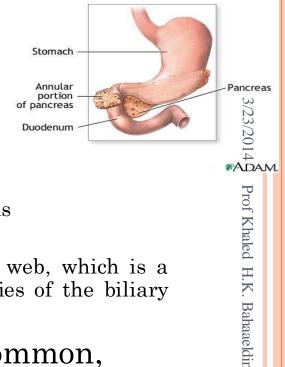
DUODENAL OBSTRUCTION

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DUODENAL ATRESIA

- Due to failure to recanalize the lumen after the solid phase of intestinal development in the 4th and 5th wk of gestation.
- The incidence of duodenal atresia is 1/10,000 births
- Accounts for 25–40% of all intestinal atresias.
- Half the patients are born prematurely.
- Down syndrome occurs in 20–30% of patients with duodenal atresia.
- Other congenital anomalies that are associated with duodenal atresia include malrotation (20%), esophageal atresia (10–20%), congenital heart disease (10–15%), and anorectal and renal anomalies (5%).





- Atresia
 - Types:
 - Type I: mucosal diaphragmatic (web) membrane
 - Type II: short fibrous cord connects two atretic ends
 - Type III: complete separation of two atretic ends
 - An unusual cause of obstruction is a "windsock" web, which is a distensible flap of tissue associated with anomalies of the biliary tract.
- membranous form of atresia is most common, with obstruction occurring distal to the ampulla of Vater in the majority of patients.
- Duodenal obstruction can also be a result of an extrinsic compression such as an annular pancreas or from Ladd bands in patients with malrotation.

• Annular pancreas

- Incidence is around 1:7000
- Failure of normal fusion of the two
- pancreatic buds which may obstruct the duodenum from without

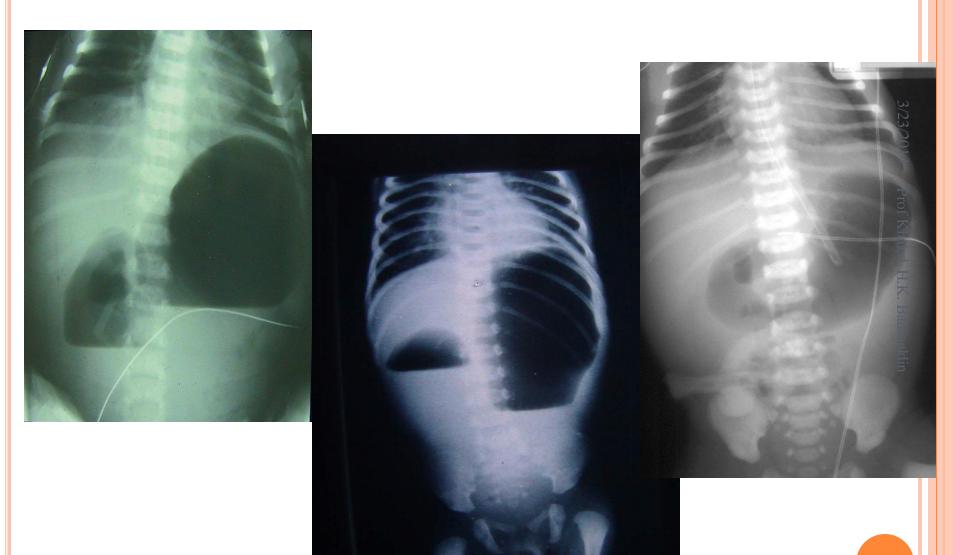
CLINICAL MANIFESTATIONS

- The hallmark is bilious vomiting without abdominal distention, which is usually noted on the 1st day of life.
- Peristaltic waves may be visualized early in the disease process.
- A history of polyhydramnios is present in half the pregnancies and is caused by a failure of absorption of amniotic fluid in the distal intestine.
- Jaundice is present in one third of the infants.



- The diagnosis is suggested by the presence of a "double-bubble sign" on plain abdominal radiographs.
- The appearance is caused by a distended and gasfilled stomach and proximal duodenum.
- Contrast studies may occasionally be needed to exclude malrotation and volvulus because intestinal infarction may occur within 6–12 hr if the volvulus is not relieved.
- Prenatal diagnosis of duodenal atresia is readily made by fetal ultrasonography





TREATMENT

- Initial Nasogastric or Orogastric decompression and intravenous fluid replacement.
- Echocardiogram and radiology of the chest and spine should be performed to evaluate for associated anomalies.
- Definitive correction of the atresia is usually postponed to evaluate and treat these life-threatening anomalies.
- Surgery
 - duodenoduodenostomy.
 - Duodenal diaphragmatic obstruction is managed by duodenoplasty



JEJUNOILEAL ATRESIA

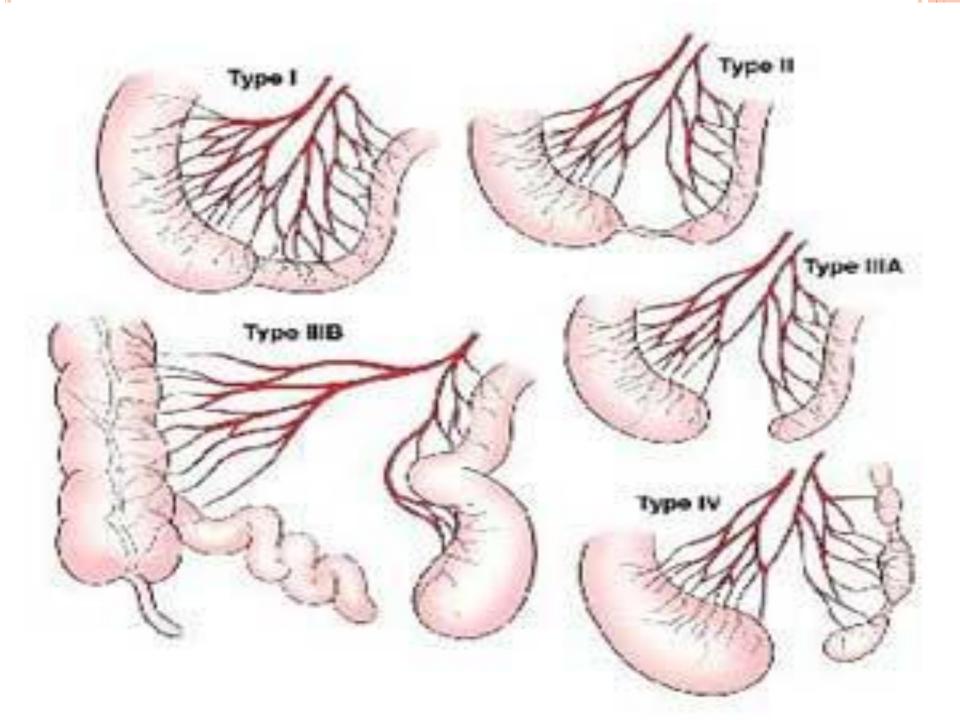
- Jejunoileal atresia involves an obstruction of the jejunum or ileum of the small intestine.
- The segment of intestine just before the obstruction becomes massively dilated, thus hindering its ability to absorb nutrients and peristalsis through the digestive tract.

- Jejunoileal atresia have been attributed to intrauterine vascular accidents leading to ischemic necrosis of the sterile bowel and resorption of the affected segments.
- Polyhydramnios occurs in 25% of affected patients
- Monozygotic twins are at higher risk
- Premature birth occurs in 30% of infants.
- Atresia has also been associated with low birthweight, multiple births, and maternal cocaine use and cigarette smoking.

• <u>Cystic fibrosis</u> is also an associated disorder and may seriously complicate the management of jejunoileal atresia. Infants with jejunoileal atresia should be screened for cystic fibrosis.

TYPES

- **Type I atresia (23%)** is a transluminal septum with proximal dilated bowel in continuity with collapsed distal bowel. The bowel is usually of normal length.
- **Type II atresia (10%)** involves two blind-ending atretic ends separated by a fibrous cord along the edge of the mesentery with mesentery intact.



• **Type IIIa atresia (15%)** is similar to type II, but there is a mesenteric defect and the bowel length may be foreshortened.

• **Type IIIb atresia (19%)** ("apple peel") consists of a proximal jejunal atresia, often with malrotation with absence of most of the mesentery and a varying length of ileum surviving on perfusion from retrograde flow along a single artery of supply. • **Type IV atresia** is a multiple atresia of types I, II, and III, like a string of sausages. Bowel length is always reduced. The terminal ileum, as in type III, is usually spared.

PRESENTATION

- Infants with jejunoileal atresia, regardless of the subtype, usually vomit green bile within the first 24 hours of life.
- However, those with obstructions farther down in the intestine may not vomit until two to three days later.

- Infants often develop a distended abdomen and may not have a bowel movement during the first day of life.
- Given the age of the patient and the symptoms, an abdominal X-ray is usually sufficient to establish a diagnosis.

SURGERY

- The type of surgery depends on the type of atresia, the amount of intestine present and the degree of intestinal dilation.
- The most common operation involves removal of the blind intestinal segments, and the remaining ends are anastomosed. Similarly, a narrowed (stenosed) segment of the intestine can be removed and the bowel sutured together, thus establishing anastomosis.

COLONIC ATRESIA

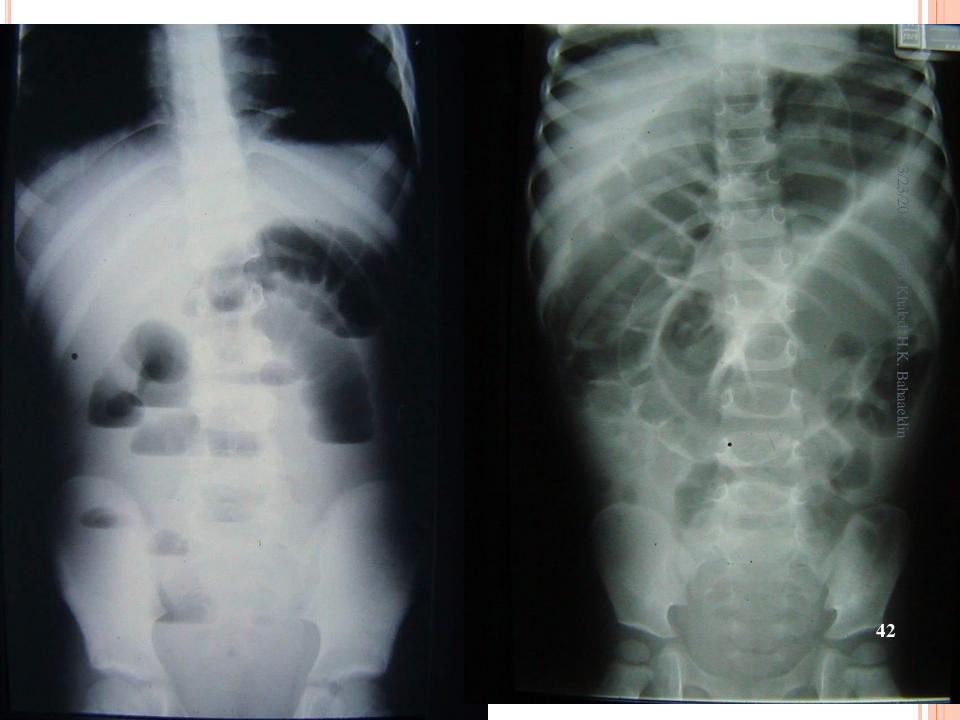
- This rare form of intestinal atresia accounts for less than 15% of all intestinal atresias. The bowel becomes massively enlarged (dilated), and patients develop signs and symptoms similar to those associated with jejunoileal atresia. Colonic atresia may occur in conjunction with small bowel atresia, <u>Hirschsprung's</u> <u>disease</u> or <u>gastroschisis</u>.
- The diagnosis is confirmed by an abdominal X-ray along with an X-ray contrast enema.

SURGERY

• Colonic Atresia: Babies with colonic atresia may undergo removal of the dilated colon in addition to a temporary colostomy. Less frequently, the ETE or STE anastomosis.

oPrenatal ultrasounds.

• Intestinal obstructions are increasingly being identified through prenatal ultrasounds. This imaging technique may indicate excess amniotic fluid (polyhydramnios), which is caused by the failure of the intestine to properly absorb amniotic fluid.







MECONIUM ILEUS

- Obstruction of Ilieum due to thick inspissated meconium
- Approximately 10% of infants with cystic fibrosis develop meconium ileus;
- 80–90% of infants presenting with meconium ileus have cystic fibrosis.
- simple meconium ileus
 - the last 20–30 cm of ileum is collapsed and filled with pellets of pale-colored stool, above which a dilated loop of varying length appears obstructed by meconium of the consistency of thick syrup or glue.
- complicated meconium ileus
 - Accompained by Volvulus, atresia, or perforation of the bowel



- Perforation in utero produces meconium peritonitis. Intraperitoneal meconium can cause dense adhesions, leading postnatally to adhesive intestinal obstruction, and can rapidly become calcified.
- Most symptomatic in the 1st day of life with abdominal distention and bile-stained emesis or gastric aspirate.
- 60 to 75% of the infants fail to pass meconium.
- Jaundice has been found in 20–30% of the patients.



- Plain radiographs demonstrate many air-fluid levels or peritoneal calcification associated with meconium peritonitis.
- Contrast studies of the upper and lower bowel can delineate the level of obstruction and differentiate atresia from meconium ileus, meconium plug, and Hirschsprung disease.
- Abdominal ultrasound can help distinguish meconium ileus from ileal atresia and identify intestinal malrotation.



- In meconium ileus-
 - typical hazy or ground-glass appearance in the right lower quadrant. Small bubbles of gas trapped in meconium are dispersed within this area.
 - Because of their viscid contents, moderately dilated loops of bowel do not have the air-fluid levels usually seen radiographically on the erect projection.
- If there is meconium peritonitis, patchy calcification may be noted, usually in the flanks.
- Pneumoperitoneum as free air between the liver and the diaphragm on an upright radiograph of the abdomen; if there is a large amount of free air, the entire abdomen may look like a football from distention with air; the ligamentum teres is sometimes clearly visible in the midline.

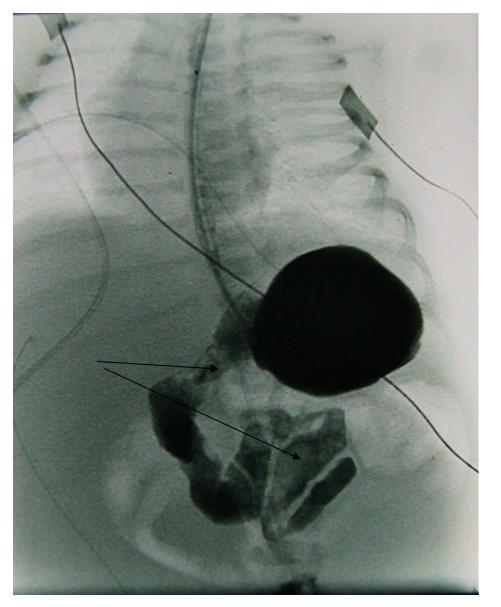
TREATMENT

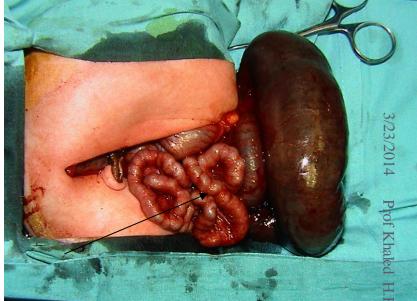
- Initial -fluid and electrolyte balance
- Prophylactic antibiotics
- In uncomplicated meconium ileus,
 - Gastrografin enemas will diagnose the obstruction and wash out the inspissated material. Gastrografin is hypertonic and care must be taken to avoid dehydration, shock, and bowel perforation, may have to be repeated after 8–12 hr



- 50% of patients with simple meconium ileus do not adequately respond to water-soluble enemas and need laparotomy.
- Operative management is indicated when the obstruction cannot be relieved by repeated attempts at nonoperative management and for infants with complicated meconium ileus.
- In simple meconium ileus, the plug can be relieved by manipulation or direct enteral irrigation with *N*-acetylcysteine following an enterotomy.
- In complicated cases, bowel resection, peritoneal lavage, abdominal drainage, and stoma formation may be necessary.
- Total parenteral is usually required.







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