Notable Grand Rounds

of the

Michael & Marian Ilitch
Department of Surgery

Wayne State University
School of Medicine

Detroit, Michigan, USA

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Michelle Veenstra, MD

VOMITING BABY: GASTROINTESTINAL OBSTRUCTION IN THE NEWBORN

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October 15, 2025

About Notable Grand Rounds

These assembled papers are edited transcripts of didactic lectures given by mainly senior residents, but also some distinguished attending and guests, at the Grand Rounds of the Michael and Marian Ilitch Department of Surgery at the Wayne State University School of Medicine.

Every week, approximately 50 faculty attending surgeons and surgical residents meet to conduct postmortems on cases that did not go well. That “Mortality and Morbidity” conference is followed immediately by Grand Rounds.

This collection is not intended as a scholarly journal, but in a significant way it is a peer reviewed publication by virtue of the fact that every presentation is examined in great detail by those 50 or so surgeons.

It serves to honor the presenters for their effort, to potentially serve as first draft for an article for submission to a medical journal, to let residents and potential residents see the high standard achieved by their peers and expected of them, and by no means least, to contribute to better patient care.

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Vomiting Baby: Gastrointestinal Obstruction in the Newborn

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October 15, 2025

Editor's Note: This paper is derived from the authors' presentation at Surgical Grand Rounds.

Introduction

Vomiting in the immediate neonatal period spans a spectrum from physiologic reflux to time-critical surgical emergencies. This paper presents a pragmatic, algorithm-driven approach to the vomiting neonate that emphasizes rapid triage, clear imaging pathways, and decisive management for the dominant obstructive entities encountered by pediatric surgeons.

The framework reflects Grand Rounds teaching points and board-relevant "can't miss" conditions (e.g., malrotation with midgut volvulus) and is calibrated to the practical realities of neonatal intensive care and inter-hospital transfers. The overarching aim is to minimize morbidity from delayed diagnosis, preserve bowel length, and optimize long-term nutritional outcomes.

First principles: go see the baby

Evaluation begins at the bedside. The clinician should see the infant promptly, obtain a focused history, and perform a meticulous physical examination before over-relying on imaging. History targets gestational age, birth weight, prenatal findings, and particularly maternal conditions and peripartum exposures—diabetes, preeclampsia, drug use, and medications—because these correlate with functional obstructions such as small left colon syndrome and meconium plug states as well as with specific mechanical lesions.

On physical exam, confirm the position and patency of tubes (NG/OG) and all orifices; an NG tube that will not pass raises suspicion for an esophageal lesion, while an absent or ectopic anus redirects the workup to anorectal malformations. In neonates, the abdominal wall

is a "window": progressive mottling or color change (red, green, blue, black) can herald perforation or ischemia and should lower the threshold for urgent operative exploration. Look deliberately for hernias. Screen for associated congenital anomalies given their high co-occurrence with certain obstructions (e.g., trisomy 21 and congenital heart disease in duodenal atresia).

Red flags and the initial decision tree

Three presenting clusters focus the triage: vomiting (especially bilious), abdominal distension, and failure to pass meconium. The combination of bilious emesis and clinical instability is a surgical emergency until proven otherwise; malrotation with volvulus must be presumed and promptly excluded or treated. The diagnostic tree starts with attempts to pass an NG tube; if unsuccessful, investigate esophageal obstruction. If the anus is imperforate or malpositioned, prioritize anorectal malformation pathways. If both are patent, obtain plain radiographs to judge the extent and distribution of intraluminal gas. Predominantly proximal gas with absence distally suggests a high obstruction (e.g., duodenal atresia or volvulus), whereas diffuse gas with distal air–fluid levels suggests a distal obstruction for which a contrast enema is often the most informative next step.

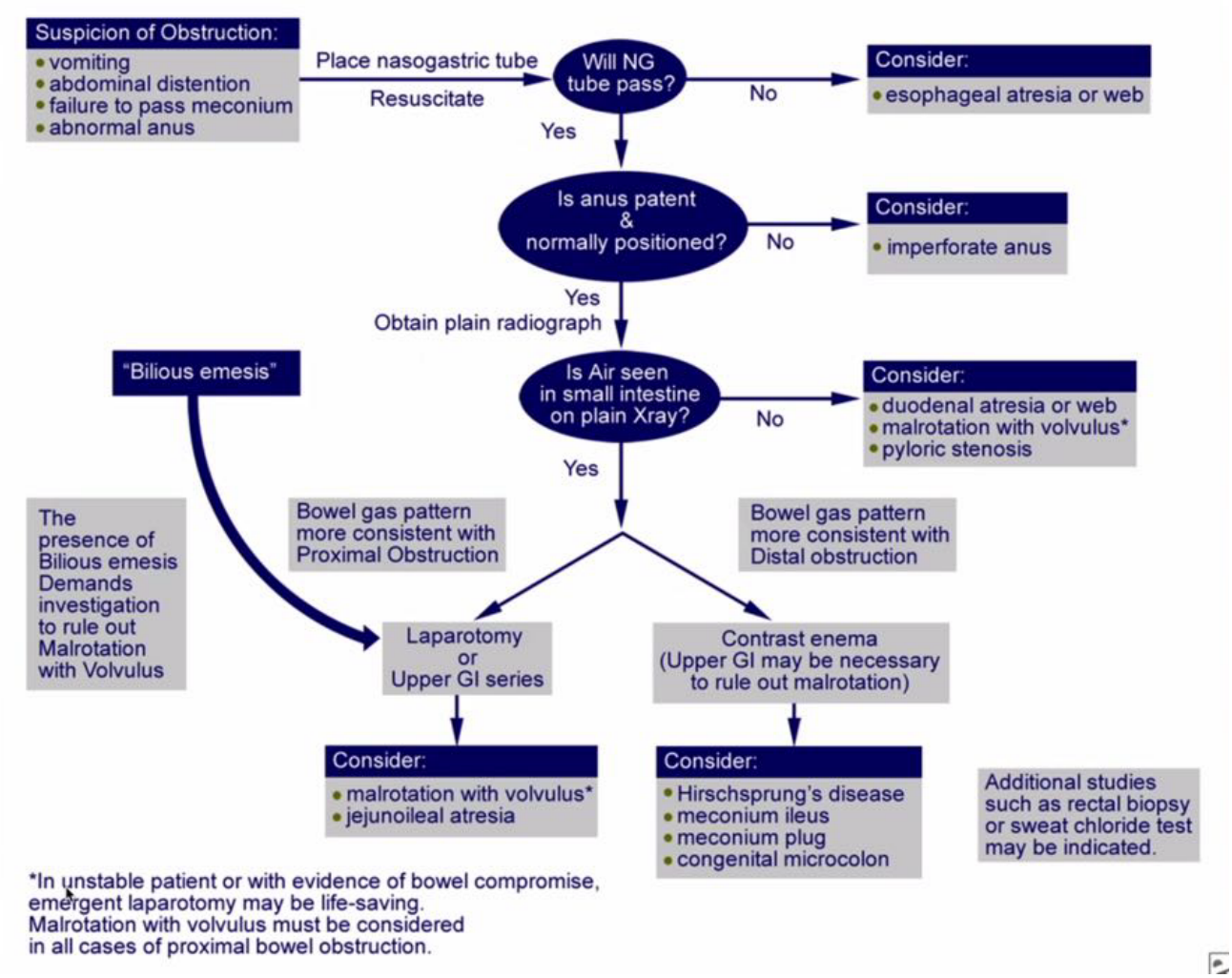

Fig. 1. Algorithm for Diagnosis of Neonatal Intestinal Obstruction

At this stage, avoid algorithmic tunnel vision: "proximal vs distal" remains helpful, but unstable physiology or evolving abdominal wall changes trump imaging—go to the operating room when the baby declares peritonitis or ischemia. In stable infants with suspected proximal disease, an upper GI series can both confirm malrotation and demonstrate specific signs of volvulus (e.g., failure of the duodenal sweep to cross midline, a "bird's beak" termination).

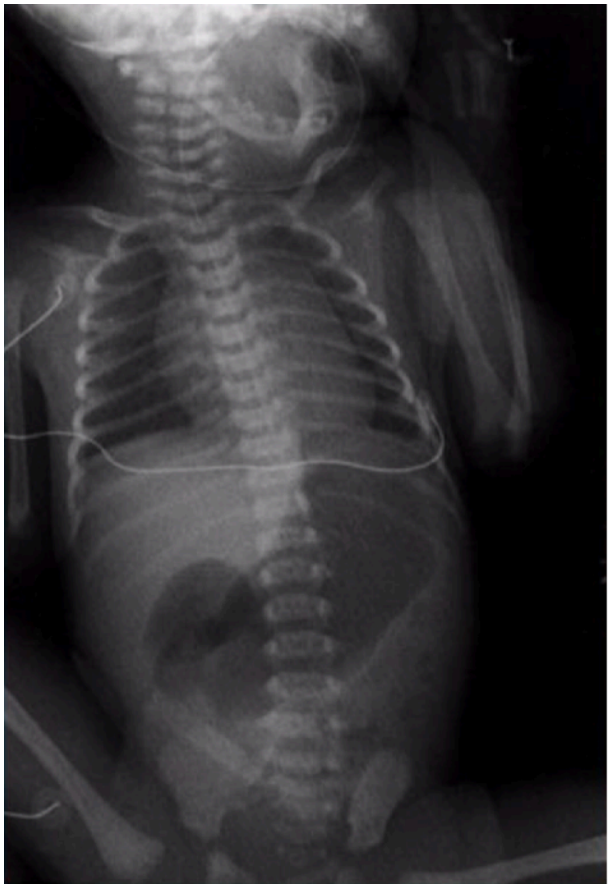

Fig. 2. Example plain film and "double-bubble" sign for proximal obstruction

Preoperative stabilization and lines

Once an operative lesion is likely—or the infant demonstrates significant obstruction—standardize preoperative care: keep NPO, institute nasogastric decompression to low intermittent suction, begin intravenous fluids for resuscitation, and start broad-spectrum antibiotics. Anticipate prolonged parenteral support by placing a PICC or UVC for total parenteral nutrition (TPN), especially when atresia is suspected or the differential includes NEC.

Differential diagnosis: mechanical vs functional

The differential divides usefully into mechanical and functional etiologies. Mechanical causes include malrotation with volvulus, duodenal atresia (intrinsic or extrinsic), jejunoileal atresia, colonic atresia/stenosis, and incarcerated hernias; functional causes include sepsis-related ileus, necrotizing enterocolitis (NEC), meconium ileus, meconium plug syndrome, Hirschsprung disease, and small left colon syndrome. Several cross-links matter for early decisions: bilious vomiting in a neonate must raise concern for malrotation/volvulus; a classic "double-bubble" without distal gas points to duodenal atresia; a microcolon on contrast enema suggests nonuse from a small-bowel process such as jejunoileal atresia or meconium ileus; and delayed strictures after NEC should be anticipated and timed for interval imaging.

Why speed and sequence matter

Time-sensitive recognition preserves bowel and life. For malrotation with volvulus, the clock to ischemia is short; stabilization cannot delay detorsion and Ladd's procedure when clinical and imaging data align with volvulus. For atresias, early decompression and TPN reduce aspiration, distension, and metabolic derangements while the operative plan is optimized. In suspected NEC, adherence to conservative measures and judicious thresholds for operative intervention (perforation, fixed loops, abdominal wall cellulitis, clinical decline) reduce mortality while balancing the risks of short bowel syndrome.

Malrotation with Volvulus

Embryologic background and pathophysiology

Intestinal malrotation arises from failure of the normal 270° counterclockwise rotation of the

midgut around the superior mesenteric artery (SMA) during embryogenesis. Between the fifth and eleventh weeks of gestation, the midgut herniates into the umbilical cord, undergoes partial rotation, and then returns to the abdominal cavity, fixing the duodenojejunal loop to the left of the midline and the cecocolic loop to the right lower quadrant. When this sequence is disrupted, the mesenteric base remains abnormally narrow, predisposing the small bowel to twist around the SMA axis, producing **midgut volvulus**.

Epidemiologically, malrotation occurs in approximately 1 in 200 individuals, though most remain asymptomatic. Only 1 in 6,000 develops volvulus, and about one in thirty patients with malrotation will experience this catastrophic event at some point.

Clinical presentation

Malrotation with volvulus most commonly presents in the neonatal period, though it may manifest at any age. The hallmark symptom is **bilious emesis**, often accompanied by abdominal distension and tenderness. Because venous and lymphatic compromise precede arterial occlusion, early signs may be subtle—vomiting without severe pain or distension—until ischemia progresses. Late findings include abdominal wall color changes, hemodynamic instability, and metabolic acidosis, reflecting bowel necrosis. It should be emphasized that **bilious vomiting in a neonate is a surgical emergency until proven otherwise.** This must not be missed in a newborn or infant, and the surgeon should proceed to immediate imaging or surgery depending on stability.

Diagnostic imaging

The diagnosis may be made clinically when a newborn presents with bilious emesis and peritoneal signs, in which case delay for imaging can be fatal. In stable infants, an **upper gastrointestinal (UGI) series** remains the gold standard. Normally, contrast outlines a duodenal C-loop that crosses midline and ascends retroperitoneally to the ligament of Treitz. In malrotation, the duodenum fails to cross midline, descending vertically on the right side of the spine. When volvulus is present, the contrast tapers abruptly in a "**bird's beak"** configuration at the site of torsion, often with a corkscrew appearance of the twisted bowel distal to the obstruction.

Plain abdominal films may demonstrate a gasless abdomen or a single dilated loop, but are nonspecific. Ultrasonography, where available, can reveal inversion of the superior mesenteric artery and vein relationship or a whirlpool sign of twisted mesentery. However, if the baby is sick and has that kind of picture, don't wait for further studies—go to the OR.

Operative management: the Ladd's procedure

Once the diagnosis is made—or in any infant with strong suspicion and clinical deterioration—the operative standard is the **Ladd's procedure**. The guiding principle is to detorse the bowel—turn back time. The key steps include:

1. **Detorsion**: Rotate the bowel counterclockwise (from the surgeon's perspective) to unwind the volvulus and restore perfusion.
2. **Resection**: Excise any frankly necrotic bowel. If viability is uncertain, avoid excessive resection and plan a "second look" within 24–48 hours.
3. **Division of Ladd's bands**: These peritoneal folds tether the cecum to the right upper quadrant and cross over the duodenum, producing extrinsic obstruction; dividing them releases the duodenum.
4. **Widening the mesenteric base**: Dissection continues to broaden the attachment of the small-bowel mesentery to prevent recurrent volvulus.

5. **Reorientation**: The small bowel is placed on the right and the colon on the left, minimizing the risk of retorsion.
6. **Appendectomy**: Removing the appendix prevents future diagnostic confusion, since its new left-sided position could obscure appendicitis.

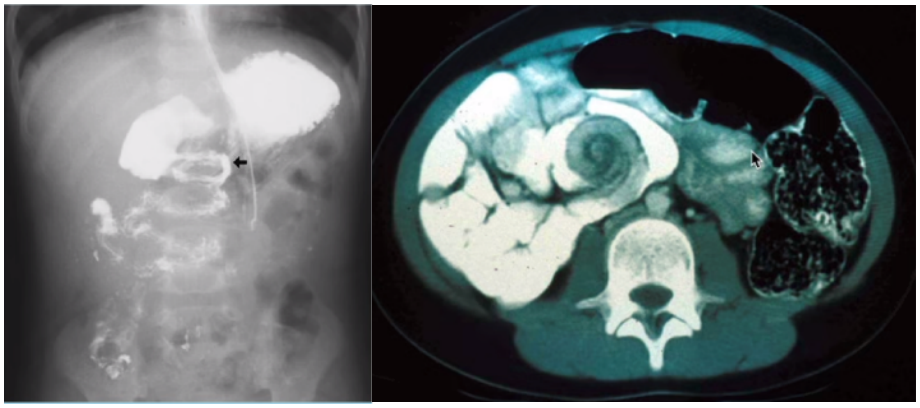

Fig. 3. Fluoroscopic UGI and CT images illustrating malrotation

Intraoperatively, the surgeon assesses bowel viability after detorsion. Dusky or cyanotic loops may recover after a period of warm saline packing; if improvement is seen, a **second-look laparotomy** the next day allows confirmation before committing to resection. For infants in whom the abdomen cannot be closed without tension, a temporary silo or negative-pressure dressing may be employed.

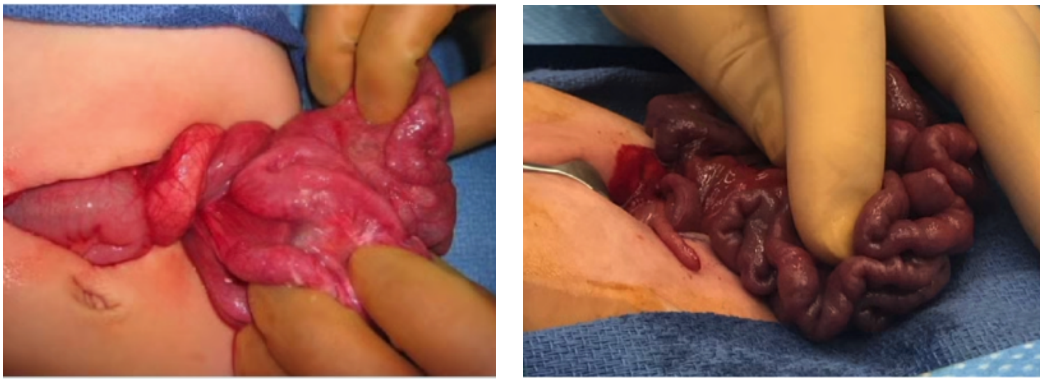

Fig. 4. Intra-op photos and diagram of intestinal repositioning

Laparoscopic versus open approach

While laparoscopic Ladd's procedures have gained traction in older or stable patients, neonates generally undergo open repair. Laparoscopy may create insufficient adhesion formation; paradoxically, this is one setting where scar tissue is desirable since mild adhesions help fix the bowel in a non-torsed position.

Postoperative care and outcomes

After Ladd's procedure, neonates are typically admitted to the neonatal intensive care unit for close monitoring. They remain NPO with nasogastric decompression until bowel sounds and stool passage return, which may take several days. TPN is maintained throughout this period. Antibiotics are continued for 48–72 hours postoperatively or longer if ischemia was present. Electrolytes, urine output, and abdominal girth are closely followed.

If a second look was performed, primary closure or ostomy creation depends on intraoperative findings. Long-term outcomes are favorable when intervention occurs before irreversible ischemia: mortality is near zero in uncomplicated cases, but rises sharply once necrosis develops. Adhesive small-bowel obstruction and recurrent volvulus are rare but recognized late complications.

Feature	Typical Findings / Management
Incidence	1 in 200 malrotation; 1 in 6,000 with volvulus
Age at presentation	Neonatal (most common), but any age
Key symptom	Bilious vomiting
Imaging hallmark	UGI: duodenal sweep fails to cross midline; "bird's beak"
Surgical procedure	Ladd's: detorsion, division of bands, mesenteric widening, appendectomy
Second look	24–48 hours if viability uncertain
Outcome	Excellent if diagnosed pre-necrosis; mortality due to bowel loss

Table 1. Diagnostic and management summary of malrotation with volvulus

Duodenal Atresia and Extrinsic Duodenal Obstruction

Overview and clinical presentation

Duodenal atresia represents the second most common cause of neonatal intestinal obstruction, with a reported incidence ranging between 1 in 6,000 and 1 in 40,000 live births. Advances in prenatal ultrasonography have allowed many cases to be diagnosed in utero, often through detection of the characteristic "double-bubble" sign. Approximately two-thirds of affected infants have associated congenital anomalies, most notably congenital heart disease and trisomy 21, each occurring in roughly one-third of patients. Because of these associations, a detailed preoperative echocardiogram is essential once the diagnosis is suspected or confirmed.

Clinically, most neonates present within the first 24 hours of life with nonbilious or bilious emesis depending on whether the obstruction lies proximal or distal to the ampulla of Vater. The abdomen may appear scaphoid or only mildly distended due to decompression of the distal bowel. The absence of meconium passage is common but nonspecific. In the absence of timely diagnosis and decompression, aspiration and electrolyte derangements may occur rapidly.

Embryology and pathology

Duodenal atresia results from failure or incomplete recanalization of the duodenal lumen between the fifth and tenth weeks of gestation. This defect is distinct from the vascular accidents that underlie jejunoileal and colonic atresias. Three morphologic subtypes are recognized:

1. **Type I** – an intact bowel wall with an intraluminal mucosal web or diaphragm that obstructs the lumen ("windsock" deformity if distally prolapsed);
2. **Type II** – blind proximal and distal segments connected by a fibrous cord; and
3. **Type III** – complete separation with no tissue connection between the blind ends.

The majority of lesions (approximately 80%) occur distal to the ampulla of Vater, explaining

the predominance of bilious vomiting in these infants. Because of the high rate of concomitant anomalies—particularly cardiac and chromosomal—meticulous preoperative assessment is required.

External (extrinsic) duodenal obstruction

Not all duodenal obstructions are intrinsic. Several extrinsic lesions may compress the duodenum and mimic atresia. The most common of these are **annular pancreas** and **preduodenal portal vein**, both of which arise from aberrant rotation or migration of embryologic structures during development.

In **annular pancreas**, a portion of the pancreatic head encircles the second portion of the duodenum, forming a ring of pancreatic tissue that constricts the lumen. In **preduodenal portal vein**, the portal vein passes anterior to the duodenum rather than posterior, similarly producing an external constriction. Although these anomalies are rare, they must be anticipated during surgical exposure to avoid vascular injury. The key principle is not to disturb or divide the anomalous structure; instead, the duodenum is bypassed around it.

Operative management: Duodenoduodenostomy

The operation of choice for both intrinsic and extrinsic duodenal obstructions is a **diamond-shaped duodenoduodenostomy**. After exposure of the dilated proximal duodenum and decompressed distal segment, transverse and longitudinal incisions are fashioned respectively in the proximal and distal limbs. These are oriented such that, when approximated, they form a diamond configuration. This orientation provides a wide, tension-free anastomosis with minimal risk of narrowing as the child grows.

The anastomosis is performed using fine absorbable sutures, beginning with the posterior wall. A small red rubber catheter may be inserted proximally and distally to ensure patency and confirm that no additional webs or windsock deformities exist beyond the visible obstruction. The anastomosis should incorporate healthy tissue from each limb, with care to maintain alignment and avoid torsion.

In cases of annular pancreas or preduodenal portal vein, the bypass is created without dissecting or mobilizing the anomalous structure. If a mucosal web is encountered and can be accessed without extensive dissection, it may be excised or incised longitudinally and closed transversely.

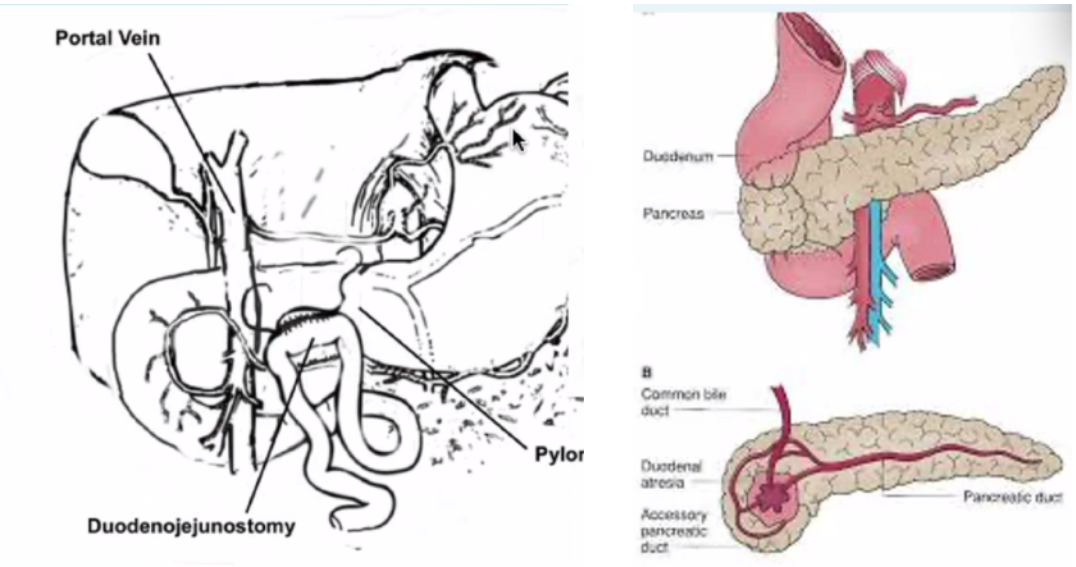

Fig. 5. Annular pancreas and preduodenal portal vein anatomy—pancreatic tissue encircling duodenum and portal vein anterior to the duodenal loop

Laparoscopic duodenoduodenostomy has been successfully performed in select centers, primarily for larger neonates with stable physiology and adequate intra-abdominal working space. However, most newborns still undergo open repair due to their small size and the need for delicate anastomotic precision. The laparoscopic approach offers cosmetic and recovery advantages but remains technically demanding..

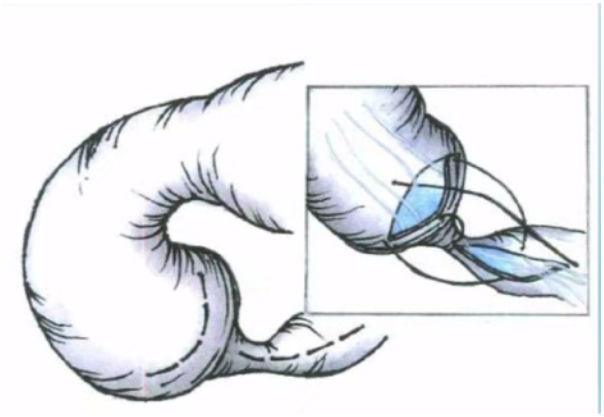

Fig. 6 Diagram of diamond-shaped duodenoduodenostomy

Postoperative management and outcomes

Postoperatively, the nasogastric tube remains in place for gastric decompression until bowel function resumes. Because the proximal duodenum and stomach are often massively dilated and atonic, it may take several days for motility to return. Once output decreases and the abdomen softens, feeds are gradually advanced.

Parenteral nutrition is continued until full enteral feeding is tolerated. Most infants demonstrate excellent recovery and feeding progression, with shorter hospitalization and earlier time to feeds compared to more distal atresias. Complications are uncommon but may include anastomotic leak, delayed gastric emptying, or, rarely, stricture. Long-term prognosis is excellent, with survival rates exceeding 95%; most mortality is related to associated anomalies rather than the obstruction itself.

Teaching pearls

In clinical practice, the “double-bubble” radiograph remains a reliable visual cue for duodenal atresia, but subtle differentiation from malrotation or midgut volvulus may require an upper GI series when the presentation or gas pattern is atypical. During surgery, one must remember the possibility of an extrinsic constriction rather than an intrinsic one—do not cut through what may be a preduodenal portal vein. Finally, the choice of diamond-shaped anastomosis is not arbitrary: it shortens recovery time, lowers leak rates, and reduces the risk of delayed gastric emptying compared to end-to-end repair.

Jejunoileal and Colonic Atresia

Overview and embryology

Jejunoileal atresia represents the most common congenital anomaly of the small intestine, occurring in approximately one in 5,000 live births with equal frequency in the jejunum and ileum. In contrast to duodenal atresia, which results from failed recanalization, jejunoileal and colonic atresias are caused by **intrauterine vascular accidents** leading to ischemic necrosis and resorption of a segment of bowel. Such vascular events may arise from midgut volvulus, intussusception, internal herniation, or compromise of segmental mesenteric vessels. Maternal use of vasoconstrictive agents—including pseudoephedrine, ephedrine, cocaine, or even nicotine—has been implicated as a potential risk factor.

Over 90% of cases involve a single atresia; multiple atresias, though rare, portend a more complex operative course and prolonged postoperative recovery. Isolated stenoses account for less than 5% of cases. The vascular etiology explains why jejunoileal atresia frequently coexists with other ischemic sequelae, such as short mesentery or focal intestinal necrosis.

Classification and morphology

The atresias are commonly categorized into four major types based on anatomic configuration:

1. **Type I (mucosal web)** — The bowel is in continuity, but an intraluminal diaphragm obstructs passage; the serosa remains intact.
2. **Type II** — The blind proximal and distal segments are connected by a fibrous cord through which the mesentery remains intact.
3. **Type IIIa** — A mesenteric defect separates two blind ends, with complete discontinuity of the bowel and mesentery.
4. **Type IIIb ("apple-peel" or "Christmas tree" deformity)** — The distal small bowel is coiled around a single arterial arcade arising from the ileocolic or right colic vessel. This configuration is often associated with a markedly shortened small bowel and risk of malabsorption.
5. **Type IV** — Multiple atresias involving several segments, giving a "string of sausages" appearance on gross examination..

Clinical presentation and diagnosis

Newborns typically present within the first 24 to 48 hours of life with **bilious emesis**, abdominal distension, and failure to pass meconium. The degree of distension correlates with the distance of the atresia from the stomach: the more distal the obstruction, the greater the distension. In contrast to duodenal atresia, which produces the "double-bubble" sign, jejunoileal obstruction results in multiple dilated loops of small bowel with air–fluid levels on plain radiographs.

Contrast enema frequently demonstrates a **microcolon**, representing disuse of the distal large bowel and confirming the obstruction's proximal origin. When atresia is suspected, further studies such as upper GI are unnecessary and may only delay surgery. Once the diagnosis is established radiographically, prompt surgical exploration is indicated.

Operative management

The goals of surgery are threefold: (1) resect the atretic or necrotic segment, (2) establish bowel continuity for early feeding, and (3) preserve maximal bowel length to prevent short bowel syndrome.

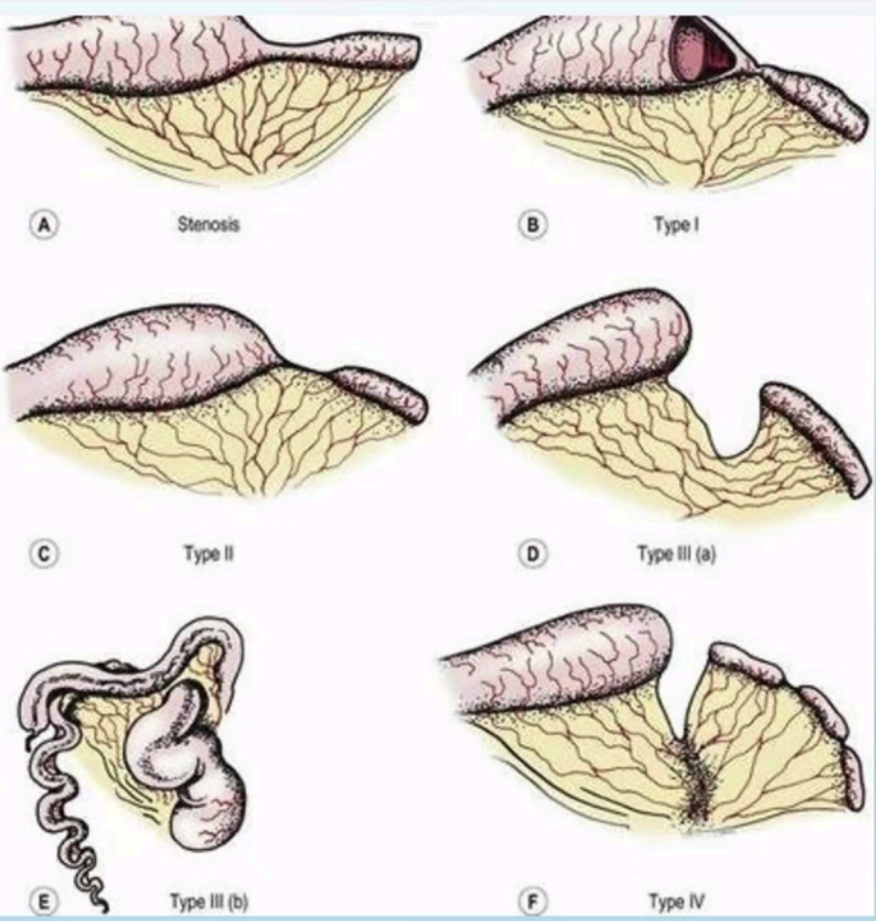

Fig. 7. Types of jejunoileal atresia

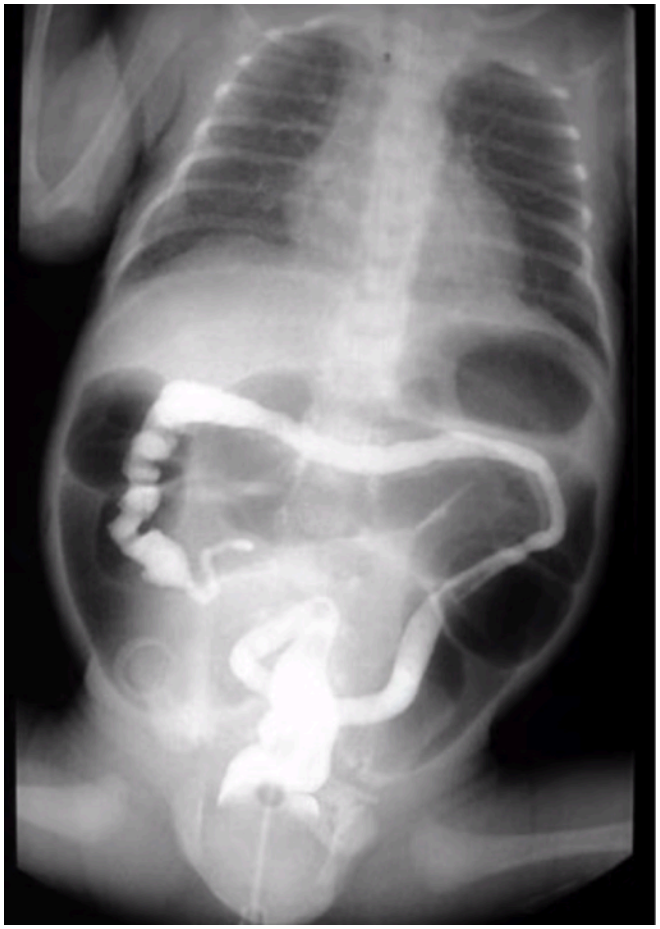

Fig. 8. Contrast enema showing microcolon in jejunoileal atresia

At laparotomy, the proximal dilated bowel and distal decompressed segment are identified. The surgeon must inspect the entire length of the intestine to exclude multiple atresias, particularly in types IIIb and IV. The atretic segment is excised with limited margins of healthy bowel. When size discrepancy between segments is marked, several strategies may be employed:

- **Tapering enteroplasty** of the proximal segment to reduce diameter and improve peristaltic efficiency.
- **Antimesenteric incision and spatulation** of the distal segment to enlarge its lumen.
- **End-to-oblique or side-to-side anastomosis** to equalize caliber differences.

In situations with questionable viability or severely shortened bowel, a **staged approach** is preferable. A proximal stoma with distal mucous fistula allows decompression and eventual re-anastomosis after several weeks of growth and adaptation. When multiple small atresias are present, fine red rubber catheters can be threaded through the segments to attempt to preserve as much bowel as possible.

Preservation of bowel length is paramount. Any decision to taper, resect, or create a stoma must balance the need for continuity with the long-term risk of intestinal failure. A "second look" operation is advisable when viability is uncertain or when significant edema precludes safe anastomosis.

Postoperative management

After surgery, nasogastric decompression continues until bowel motility returns. Parenteral nutrition supports hydration and caloric needs during this interval. Gradual introduction of enteral feeds begins once stool or gas is observed.

Potential complications include anastomotic leak, strictures, short bowel syndrome, and bacterial overgrowth. Early recognition and aggressive nutritional support can markedly improve long-term outcomes. Survival now approaches 90–100%, with mortality primarily related to severe bowel loss or comorbid conditions.

Colonic atresia and acquired colonic stenosis

Colonic atresia is rare, estimated at 1 in 20,000 births. The pathogenesis parallels that of jejunoileal atresia—an in utero vascular insult leading to segmental resorption. Roughly 15–20% of affected neonates also have a proximal small-bowel atresia, classifying them as type IV in the small-bowel scheme. About 2% have concurrent Hirschsprung disease, making a **suction rectal biopsy** mandatory either intraoperatively or postoperatively.

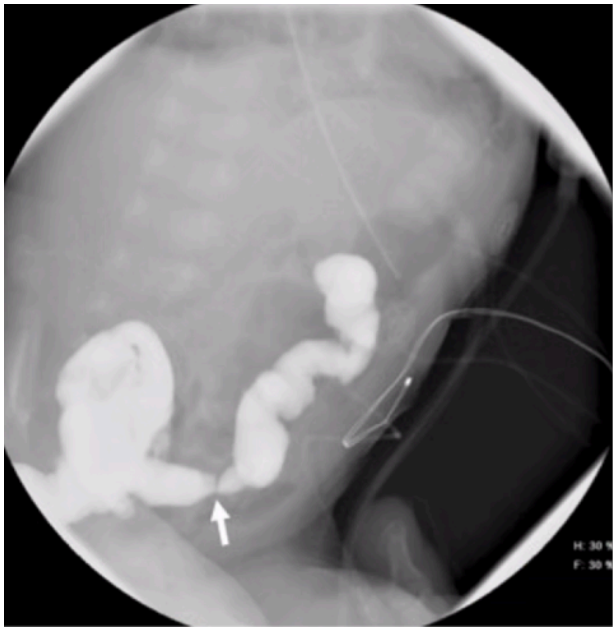

Fig. 9. Contrast enema demonstrating sigmoid stricture after NEC

Clinical features include marked abdominal distension, bilious vomiting, and failure to pass meconium. Contrast enema is diagnostic, revealing abrupt cutoff and proximal colonic dilation. Because perforation can occur rapidly—especially if the ileocecal valve is competent—timely operative intervention is critical.

Surgical management involves resection of the atretic segment with either primary anastomosis or staged diversion, depending on the infant's condition and the bowel's viability. Intraoperative biopsy is essential to exclude Hirschsprung disease.

A subset of infants develop **acquired colonic strictures** secondary to **necrotizing enterocolitis (NEC)**. Between 11% and 35% of NEC survivors develop post-inflammatory intestinal strictures, most commonly in the sigmoid colon. These strictures typically become evident four to six weeks after resolution of the acute episode and are identified on contrast enema. Surgical resection with primary anastomosis is curative in most cases.

Key operative considerations

For both jejunoileal and colonic atresias, three intraoperative questions guide decision-making:

1. What is the **best way to re-establish continuity** to enable early enteral feeding?
2. How can we **maximize bowel length and absorptive capacity**?
3. What is the **optimal strategy** to manage dilated or dysmotile proximal bowel?

Careful adherence to these principles not only reduces morbidity but also improves nutritional autonomy and quality of life.

Functional Obstruction and Postoperative Care

Necrotizing Enterocolitis (NEC)

Among functional causes of neonatal obstruction, **necrotizing enterocolitis (NEC)** remains the most feared and continues to be the leading surgical cause of death in the neonatal intensive care unit. Although most cases occur in premature and very low birth weight infants, NEC can appear in term neonates as well, and paradoxically, full-term infants often experience worse outcomes. The incidence rises sharply as birth weight falls—approximately 14% in infants weighing 500–750 g and 9% in those between 751 and 1,000 g.

The pathophysiology involves mucosal injury, bacterial invasion, and an exaggerated inflammatory cascade. Reduced mucosal blood flow results in cellular hypoxia, epithelial necrosis, and subsequent perforation. The disease most commonly affects the terminal ileum and proximal colon but may involve multiple areas. Metabolic acidosis, thrombocytopenia, and neutropenia reflect systemic compromise.

Bell's classification remains the standard clinical guide.

- *Stage I (suspected)*: nonspecific findings such as abdominal distension, occult blood in stools, or ileus.
- *Stage II (definitive)*: pneumatosis intestinalis or portal venous gas on radiographs, often accompanied by metabolic acidosis.
- *Stage III (advanced)*: pneumoperitoneum, abdominal wall cellulitis or edema, and hemodynamic instability.

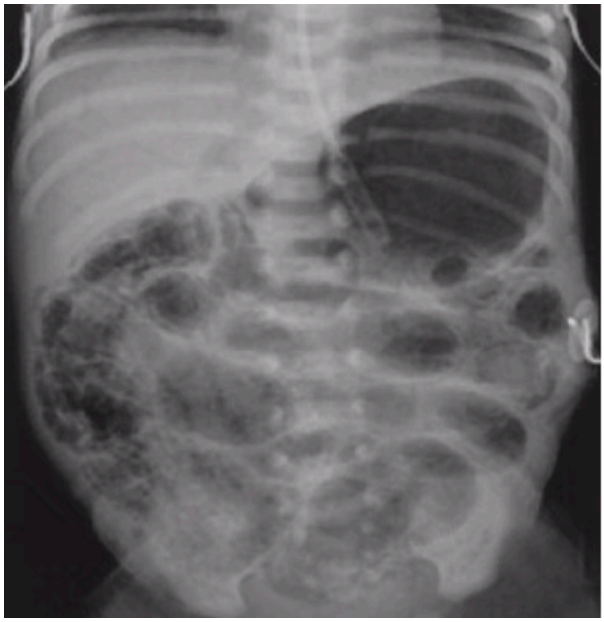

Fig. 10. Abdominal radiograph showing pneumatosis and portal venous gas

Initial management is medical: immediate bowel rest with nasogastric decompression, broad-spectrum antibiotics, hemodynamic stabilization, and meticulous fluid and electrolyte replacement. Surgery is indicated for **perforation**—the only absolute indication—or for progressive deterioration despite maximal medical therapy. Relative indications include a fixed loop on serial films, abdominal wall erythema or edema, palpable mass, or persistent metabolic acidosis.

For infants under 1 kg, bedside peritoneal drainage may serve as a temporizing or definitive measure; larger infants usually undergo exploratory laparotomy with resection of necrotic segments and stoma creation. If extensive ischemia precludes resection, damage-control strategies and staged reassessment after stabilization are appropriate. Survivors require follow-up imaging several weeks later to detect strictures, which develop in up to one-third of cases, most commonly in the sigmoid colon.

Meconium Ileus

Meconium ileus is the earliest clinical manifestation of **cystic fibrosis (CF)** and presents in roughly 20 % of affected neonates. Thick, inspissated meconium obstructs the terminal ileum, sometimes complicated by perforation or pseudocyst formation. Uncomplicated cases present with bilious vomiting, abdominal distension, and failure to pass stool. In utero perforation may produce cystic masses palpable at birth, and polyhydramnios with intrauterine growth restriction is observed in approximately one-fifth of pregnancies.

Physical examination may reveal the "**putty sign"**—a soft indentation left by thumb pressure on the abdominal wall—and localized erythema indicating inflammation. Radiographs show variable bowel calibers with a granular or "soap-bubble" pattern over the right abdomen, reflecting gas mixed with tenacious meconium.

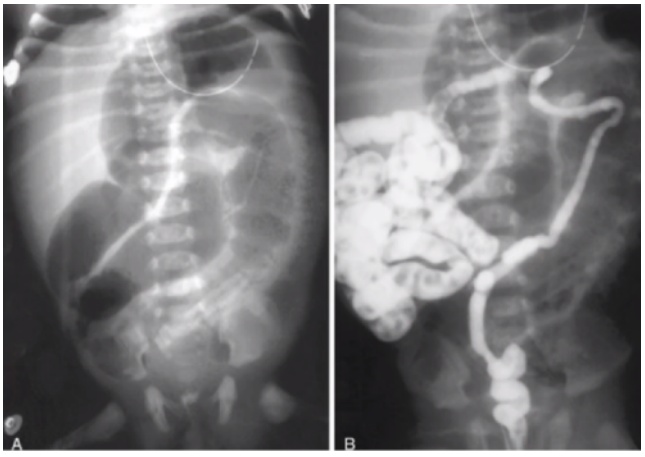

Fig. 11. Contrast enema demonstrating microcolon in meconium ileus

Contrast enema demonstrates a **microcolon**, confirming obstruction.

Treatment begins with a **water-soluble contrast enema** using agents such as Gastrografin™ or Omnipaque™ . The hyperosmolar solution draws water into the lumen, softening and expelling the meconium; success rates reach 50–65 % in stable infants. If obstruction persists, 2–4 % N-acetylcysteine can be instilled via nasogastric tube to further solubilize material.

Surgery is required for complicated or refractory cases. Operative options include enterotomy with irrigation using saline or pancreatic enzyme solution, formation of an appendicostomy or cecostomy for postoperative irrigation, or proximal diversion if perforation or necrosis is present. Intraoperative findings typically reveal very dilated proximal bowel with thick green meconium and decompressed distal bowel filled with distal pellet-like concretions. After surgery, cystic fibrosis testing is mandatory. Pancreatic enzyme supplementation and careful nutritional management follow.

Meconium Plug Syndrome

Meconium plug syndrome is a more benign and transient cause of neonatal obstruction, most often seen in premature infants. It results from immaturity of the myenteric plexus leading to ineffective peristalsis and excessive water absorption, producing thick plugs of meconium within the distal colon.

Clinically, the newborn fails to pass meconium within 24 hours and may develop mild abdominal distension. Contrast enema reveals one or more filling defects in the sigmoid or descending colon, often followed by spontaneous evacuation of the plugs during the procedure—both diagnostic and therapeutic. Persistent or recurrent symptoms mandate evaluation for **cystic fibrosis** or **Hirschsprung disease**, as up to 14% of CF patients and 13% of those with Hirschsprung's present initially with this pattern.

Meconium plug syndrome is frequently associated with maternal factors such as preeclampsia treated with magnesium sulfate, diabetes, or hypothyroidism, all of which can

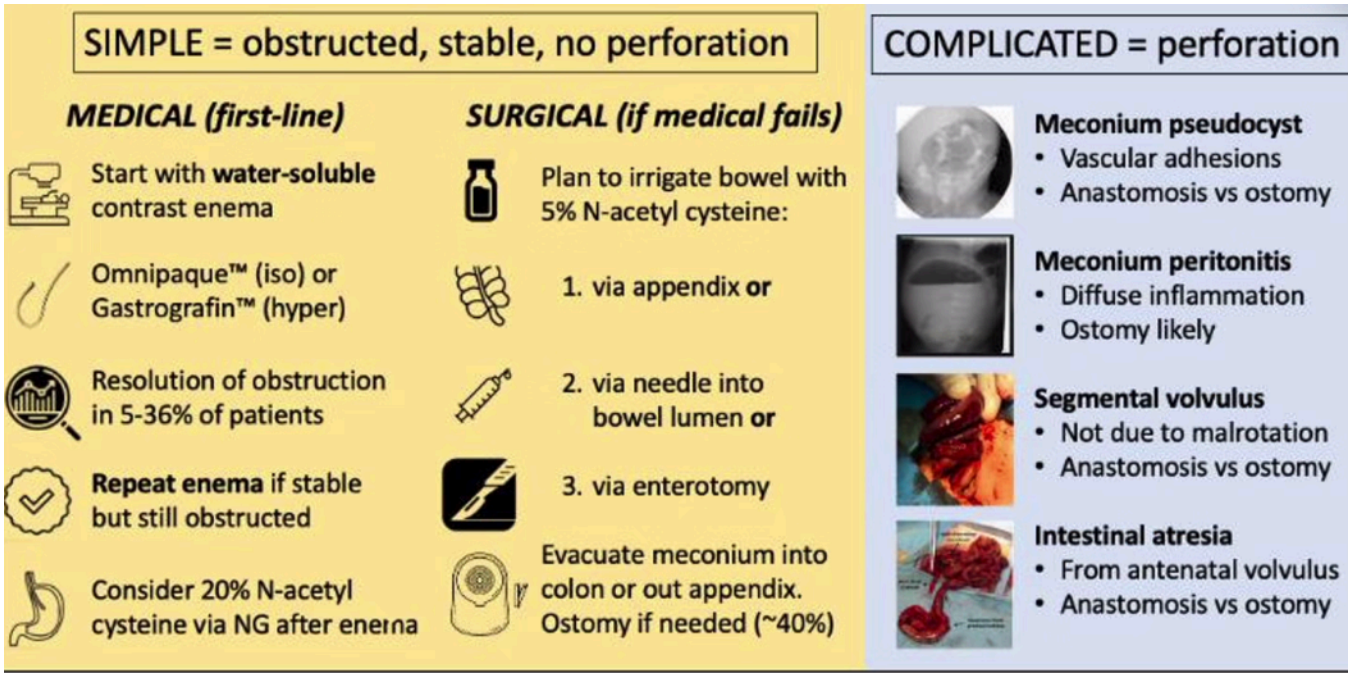

Fig. 12. Operative irrigation for meconium ileus; enterotomy and stoma options

depress neonatal motility. Most cases resolve after one enema, and surgery is rarely required.

Small Left Colon Syndrome

Small left colon syndrome (SLCS) is another transient functional disorder characterized radiographically by a funnel-shaped narrowing of the left colon terminating at the splenic flexure. It is strongly associated with maternal diabetes, eclampsia, hyperthyroidism, or exposure to magnesium sulfate during pregnancy.

Infants present with mild abdominal distension and failure to pass meconium; contrast enema typically demonstrates a sharply demarcated small-caliber descending and sigmoid colon that returns to normal caliber more proximally. The condition resolves spontaneously within 24–48 hours after birth as motility normalizes. In rare cases, persistent obstruction or perforation necessitates surgical decompression.

As with meconium plug syndrome, persistence or recurrence of symptoms after apparent resolution should prompt testing for cystic fibrosis and Hirschsprung disease. In general, no surgical intervention is required, and outcomes are excellent.

Postoperative and Supportive Care

Regardless of etiology, postoperative management of neonatal bowel obstruction shares common principles. All infants remain **NPO** with **nasogastric decompression** until bowel motility returns. In cases of atresia, this can take several weeks; in NEC survivors, at least two weeks of bowel rest are customary. **Total parenteral nutrition (TPN)** supports caloric and protein requirements, with early modifications to prevent cholestasis when prolonged use is anticipated.

Broad-spectrum antibiotics are continued for 2–14 days depending on the underlying pathology. Fluid balance, electrolytes, urine output, and weight are monitored closely. Gradual reintroduction of feeds—starting with small volumes of breast milk or elemental formula—should be guided by clinical tolerance.

Infants with significant bowel resection require long-term multidisciplinary follow-up for **short bowel syndrome**, including nutrition, growth, and developmental monitoring. Those with cystic fibrosis need coordination with pulmonary and genetic specialists for enzyme replacement and ongoing surveillance.

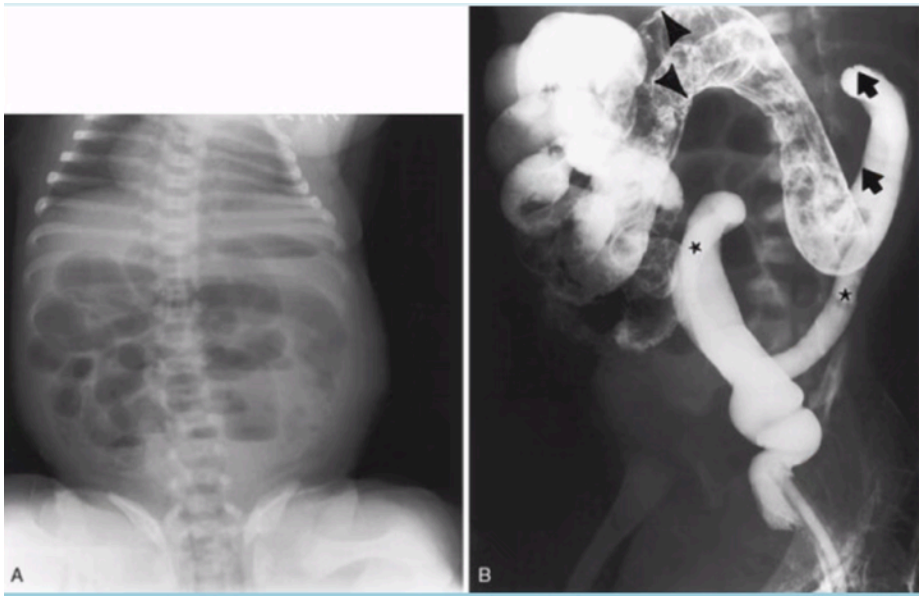

Fig. 13. Funnel-shaped tapering of left colon on contrast enema in SLCS

Conclusion

Vomiting in the neonate is never trivial. A structured approach—beginning with careful physical examination and progressing through targeted imaging—permits rapid differentiation between benign and life-threatening causes. Functional conditions such as NEC, meconium disorders, and transient left-colon syndromes demand nuanced management balancing conservative therapy with timely surgical intervention when indicated. Mechanical obstructions, conversely, require decisive operative correction guided by principles of bowel preservation and meticulous anastomotic technique.

Prompt recognition, interdisciplinary collaboration, and vigilant postoperative care remain the cornerstones of survival. With these practices, most newborns with gastrointestinal obstruction—whether functional or mechanical—can look forward to a normal, thriving childhood.

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