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Literature Review

Neonatal Gastrointestinal Emergencies

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Abstract:

Background: Neonatal gastrointestinal emergencies refer to a set of life-threatening conditions affecting the digestive system of a newborn within the first 28 days of life and often associated with high morbidity and mortality. As such, these conditions require immediate and accurate diagnosis as well as proper treatment to optimize the outcomes of these patients. This condition has the potential to obstruct the flow of gastric content leading to vomiting, failure to thrive, and electrolyte imbalances.

Discussion: Gastrointestinal obstruction is one of the most common conditions causing emergency condition in neonates. This condition may occur anywhere between the upper part of gastrointestinal tract to the lower gastrointestinal tract. In most cases of neonatal gastrointestinal emergencies, patients almost always present with vomiting that may be bilious or non-bilious. Furthermore, persistent vomiting may also lead to a more severe consequences such as hypovolemic shock and electrolyte imbalances. Therefore, clinicians are expected to address this problem early while also working to find the underlying etiologies of neonatal vomiting. On the other hand, gastrointestinal bleeding is often an alarming sign that indicates a possible emergency condition in neonates. However, some non-emergency condition such as swallowed maternal blood and cow's milk allergy can also result in gastrointestinal bleeding in neonates.

Conclusion: Given the critical time window and the vulnerability of the neonate population, the proper identification and prompt treatment of neonatal gastrointestinal emergencies is crucial to minimize morbidity and mortality. Multidisciplinary management with neonatologists, pediatric surgeons, radiologists, and nursing staff working closely together can provide the best possible outcomes.

Keywords: emergency, gastrointestinal, neonates

Introduction

Based on the latest World Health Organization (WHO) data, 11 neonatal deaths per 1000 live births are reported in Indonesia.[1] Among those numbers, neonatal emergencies play a major role in contributing to the neonatal mortality rate especially in developing countries. These emergency conditions may arise from various conditions such as infection, respiratory failure, shock and gastrointestinal problems.[1,2]

Neonatal gastrointestinal emergencies refer to a set of life-threatening conditions affecting the digestive system of a newborn within the first 28 days of life and often associated with high morbidity and mortality. As such, these conditions require immediate and accurate diagnosis as well as proper treatment to optimize the outcomes of these patients. The range of gastrointestinal emergencies includes structural anomalies, such as atresia and fistula, which interfere with food intake and can cause respiratory distress. Another category is necrotizing enterocolitis, characterized by severe inflammation and necrosis of the intestines, leading to systemic illness. All of these conditions have the potential to obstruct the flow of gastric content leading to vomiting, failure to thrive, and electrolyte imbalances.

In some conditions, prenatal imaging is sensitive enough to determine the underlying cause of gastrointestinal emergencies in neonates. However, in most patients, careful history and physical examination as well as imaging modalities are crucial for an accurate diagnosis. Plain radiography, fluoroscopy, ultrasound are the most common modalities being used to diagnose gastrointestinal emergencies.[3] The aim of this review is to discuss the neonatal gastrointestinal emergencies while reviewing the appropriate imaging options as well as focusing on their radiological features and the diagnostic algorithm.

Approach to Neonatal Intestinal Obstruction

Gastrointestinal obstruction is one of the most common conditions causing emergency condition in neonates. This condition may occur anywhere between the upper part of gastrointestinal tract to the lower gastrointestinal tract. There are several features that are highly suggestive of gastrointestinal obstruction such as polyhydramnios, feeding intolerance, bilious emesis, abdominal distention and delayed passage of meconium.[4]

As neonatal gastrointestinal obstruction may be caused by various etiologies at different site along the gastrointestinal tract, an algorithm for diagnostic approach is essential especially in healthcare facilities with limited imaging modalities. This approach should start with simple history taking, physical examination and plain radiograph before moving on to a more advanced imaging modality for more

sophisticated cases. An algorithm for diagnosis of neonatal intestinal obstruction is presented in **Figure 1**.

Often times, neonates presenting with gastrointestinal obstruction also present with serious life-threatening conditions such as shock, respiratory distress or electrolyte imbalance. Therefore, resuscitation is the main focus for those presenting with emergency conditions before moving on to explore the underlying etiology. Placing nasogastric tube should also be considered in most cases with gastrointestinal obstruction in order to decompress the abdominal pressure swiftly. Inability to pass the nasogastric tube is highly suggestive of esophageal atresia or web. Plain abdominal radiograph is essential in determining the approximate site of obstructions based on the air pattern seen on the image. The complete differential diagnosis algorithm is presented in **Figure 1**.

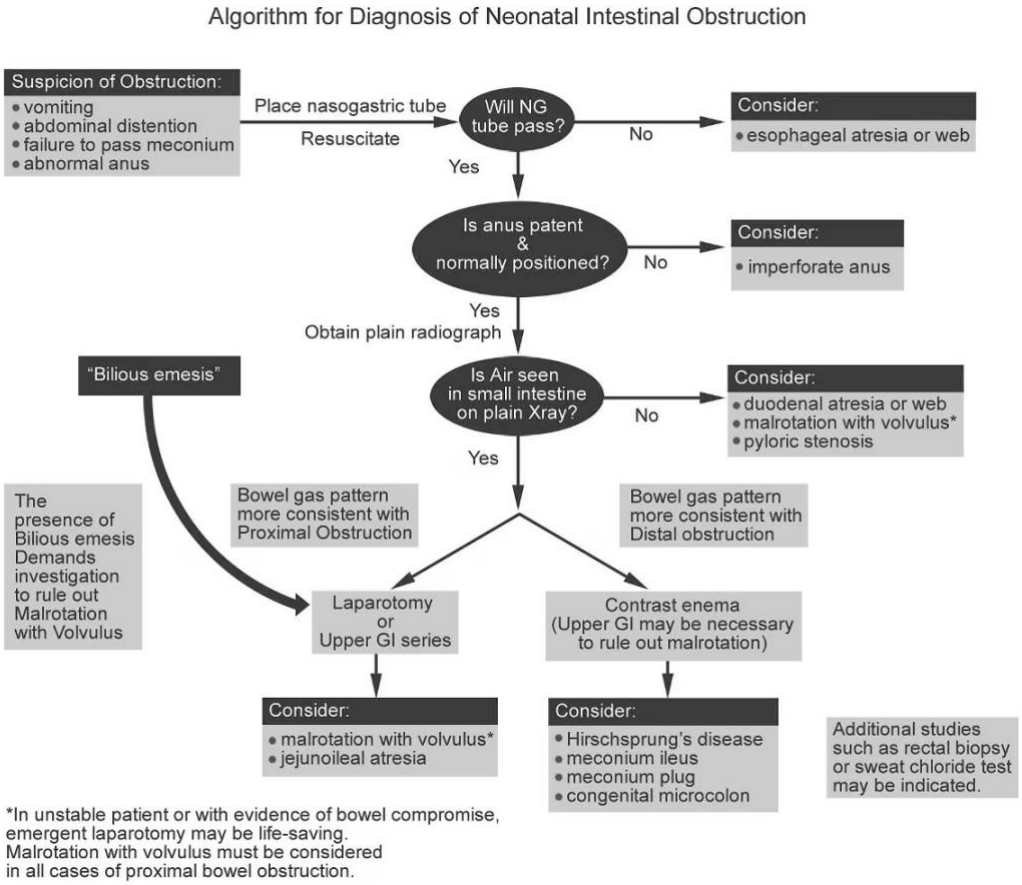


Figure 1. Diagnostic approach for neonates presenting with intestinal obstruction.

Approach to Neonatal Vomiting

In most cases of neonatal gastrointestinal emergencies, patients almost always present with vomiting that may be bilious or non-bilious. Furthermore, persistent vomiting may also lead to a more severe consequences such as hypovolemic shock and

electrolyte imbalances. Therefore, clinicians are expected to address this problem early while also working to find the underlying etiologies of neonatal vomiting.[5] Infants who present with bilious emesis should always be evaluated for possible surgical problems particularly midgut volvulus, intussusception or any other possible cause of intestinal obstruction below the ampulla of Vater. Meanwhile, acute non-bilious emesis can be caused by gastrointestinal diseases such as intestinal obstruction above the ampulla of Vater or systemic diseases such as infection. On the other hand, chronic non-bilious vomiting should be assessed for any red flag to determine the underlying etiologies. The complete algorithm of diagnostic approach for neonatal vomiting can be seen in **Figure 2**.[5]

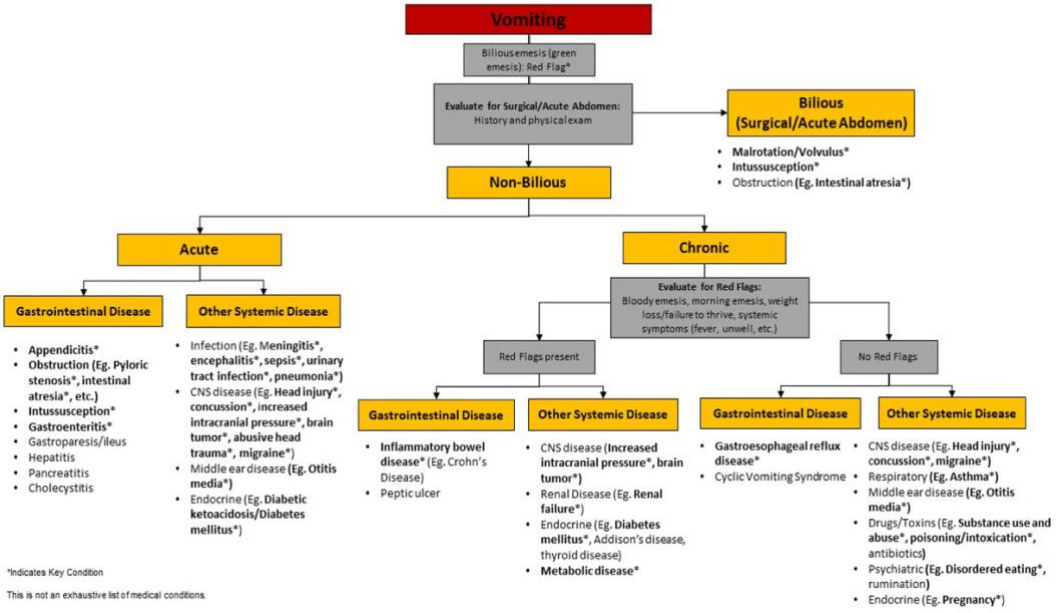


Figure 2. Diagnostic approach for neonates presenting with vomiting.[5]

Approach to Neonatal Gastrointestinal Bleeding

Gastrointestinal bleeding is often an alarming sign that indicates a possible emergency condition in neonates. However, some non-emergency condition such as swallowed maternal blood and cow's milk allergy can also result in gastrointestinal bleeding in neonates.[6] Patients with gastrointestinal bleeding but with a normal abdominal radiograph should be evaluated for fissure or colitis by using proctosigmoidoscopy.[6] Meanwhile, if patients have distended abdomen, work up for midgut volvulus and necrotizing enterocolitis should be evaluated as depicted in **Figure 3**.

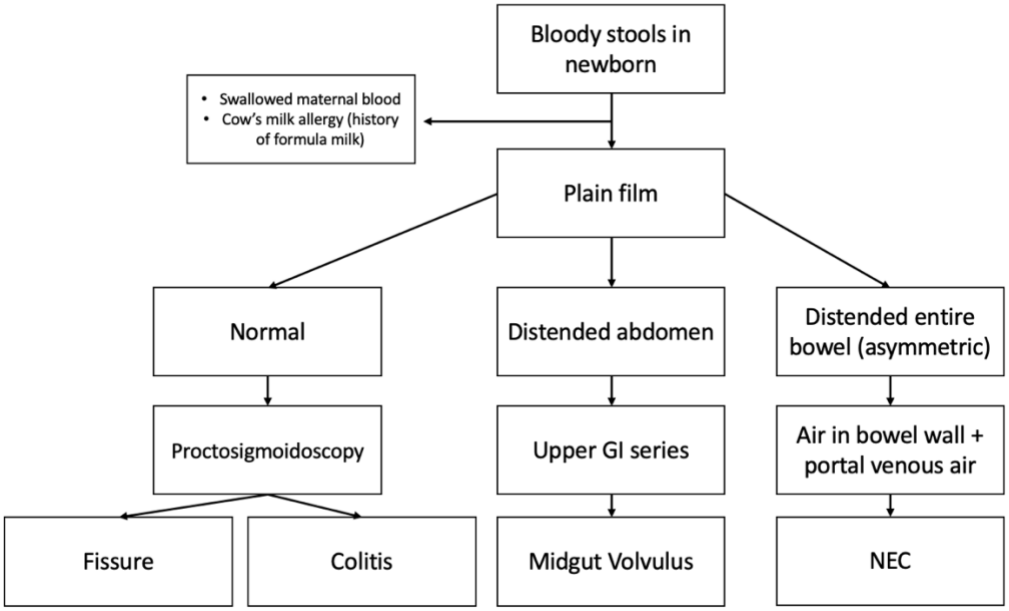


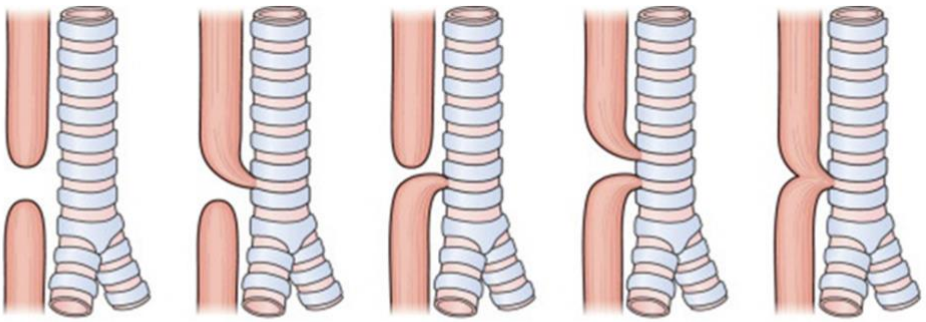
Figure 3. Diagnostic approach for neonates presenting with gastrointestinal bleeding.

Upper Gastrointestinal Emergencies

Esophageal Atresia

Esophageal atresia is a congenital anomaly of the upper gastrointestinal tract with a prevalence of 1 in 2500-4500 live births.[7] During early fetal development, the common foregut separates to form the trachea and esophagus. Failure of this separation or complete development of foregut tube leads to esophageal atresia with or without tracheoesophageal fistula.[8]

The majority of patients with esophageal atresia often present with associated anomalies, commonly known as VACTERL (vertebral defects, anal atresia, cardiac defects, tracheoesophageal fistula, renal and limb anomalies) or CHARGE (coloboma, heart defects, atresia choanae, growth retardation, genital and ear abnormalities).[7] Based on the anatomical configuration, esophageal atresia can be classified into several types as depicted in **Figure 4**.[9] Type A has a prevalence of 7%, characterized by isolated esophageal atresia without any tracheoesophageal fistula. Type B is characterized by proximal tracheoesophageal fistula and has a rather low prevalence of 2%. Type C is the most common and involves a proximal esophageal atresia with distal tracheoesophageal fistula. Type D is characterized by the presence of both proximal and distal tracheoesophageal fistula with a prevalence of 1%. Lastly, type E which is an isolated tracheoesophageal fistula with a prevalence of 4%.



Gross	A	B	C	D	E
Vogt	II	III	IIIb	IIIa	H-type
Frequency	7%	2%	86%	1%	4%

Figure 4. Classification of esophageal atresia.[9]

Prenatal ultrasound is often able to diagnose one-third of esophageal atresia cases which usually present with polyhydramnios and absent stomach bubble during ultrasound. However, many cases are not diagnosed before birth especially those in developing countries with limited healthcare facilities. Babies with esophageal atresia are symptomatic shortly after birth with inability to feed and excessive drooling, which may lead to choking and cyanotic episodes during feeding. Typically, esophageal atresia is diagnosed after fail attempt to pass an orogastric tube. On chest radiograph, the tube can be seen coiling above the level of esophageal atresia. Abdominal radiograph may show an absence of bowel gas in isolated esophageal atresia or esophageal atresia with proximal tracheoesophageal fistula (Gross type A and B).[3] However, in cases with distal tracheoesophageal fistula, bowel gas can be seen within 4 hours of life.[3] In addition, other modalities such as bronchoscopy is used as the gold standard to look for tracheoesophageal fistula.[3]

Once diagnosed with esophageal atresia, patients should be intubated to maintain airway patency as well as to prevent choking. Suction catheter should also be placed gently to eliminate excessive secretions. Patients should be given nothing per oral and nutrition should be given through parenteral route. The definitive treatment for esophageal atresia is surgical repair and should be done only after careful evaluation of other associated abnormalities. Neonates with less than 1500 grams birthweight should undergo staged approach with ligation of fistula initially before proceeding for atresia repair once the neonate is larger.[10] Complications are quite common after surgical repair of esophageal atresia. Early complications such as anastomosis leakage occurs in 15-20% of patients.[11] Meanwhile, late complications such as stricture formation occurs in 30-40% of patients.[11] Therefore, patients should be closely monitored to identify any complications should they arise.

Pyloric Atresia and Hypertrophic Pyloric Stenosis

Pyloric atresia is a rare entity with a prevalence of 1 in 100.000 live births and only contribute to 1% of all intestinal atresia.[12] Up to 55% of pyloric atresia is associated with other anomalies such as epidermolysis bullosa and multiple intestinal atresia, which worsen patients' prognosis.[12] Prenatal ultrasound often shows polyhydramnios with dilated stomach bubble without duodenal or other intestinal dilation. Upon birth, patients may present with non-bilious vomiting, feeding intolerance and upper abdominal distention.[3] On plain abdominal radiograph, a single gastric bubble with a complete absence of distal (single bubble appearance) is highly suggestive of pyloric atresia.[3]

Hypertrophic pyloric stenosis occurs due to abnormal thickening and elongation of the pyloric sphincter musculature, obstructing the gastric outlet. The incidence of this abnormality is approximately 2-5 per 1000 live births and is more predominantly in males.[13] Patients with hypertrophic pyloric stenosis usually present with projectile non-bilious emesis. Upon physical examination, a firm, non-tender, hard pylorus measuring 1 to 2 cm in diameter in the right upper quadrant (resembles an 'olive') is palpable. Upper gastrointestinal contrast study was historically used to diagnose hypertrophic pyloric stenosis based on the finding of string sign (narrowed pyloric canal due to compression from the enlarged pyloric sphincter musculature) with no egress of contrast (**Figure 5**).[3] However, with ultrasound device is more readily available nowadays, this modality has started to replace upper gastrointestinal contrast study in diagnosing hypertrophic pyloric stenosis. Moreover, ultrasound also has the advantage that it doesn't produce any ionizing radiation and has a sensitivity that reaches almost 100%[3]. Based on ultrasound finding, diagnostic criteria for pyloric stenosis include single muscular wall thickness greater than or equal to 3 mm, and a pyloric length of greater than or equal to 15 mm.[14] Right later decubitus positioning may also help visualization in case of overdistended stomach.[15]

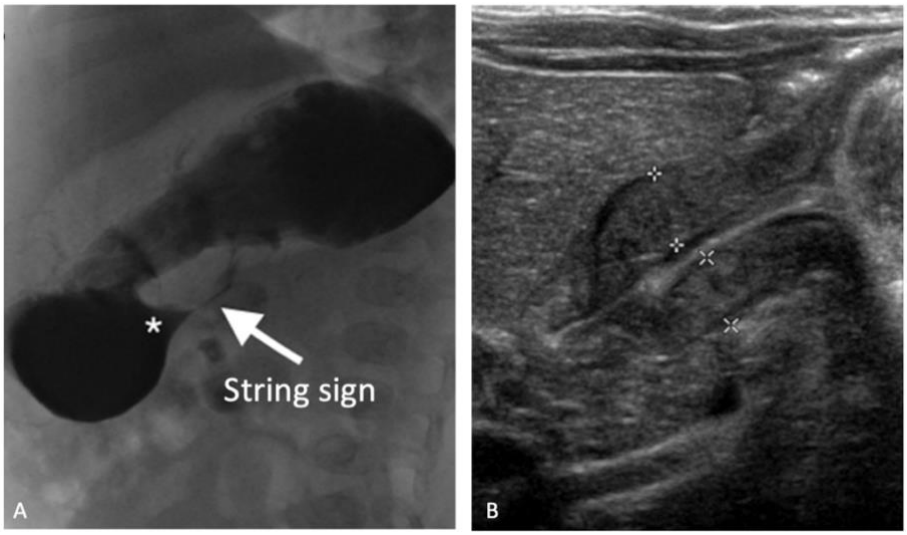


Figure 5. (A) Upper gastrointestinal contrast study demonstrates string sign in patient with hypertrophic pyloric stenosis. (B) Ultrasound finding: thickening of the pyloric musculature and elongation of pyloric outlet.[3]

The main focus for treatment of pyloric atresia/ stenosis should be rehydration and correction of any electrolyte imbalances due to excessive vomiting.[16] Nasogastric tube should also be placed for decompression.[16] Lastly, surgical procedure called pyloromyotomy can be considered after rehydration and correction of other metabolic imbalances.[16]

Duodenal Atresia

Duodenal atresia occurs due to embryologic failure of recanalization, resulting in complete obstruction of the duodenum. There is also an association of this abnormality with trisomy 21 syndrome.[17] Polyhydramnios and double bubble appearance (dilated stomach and duodenum) during antenatal ultrasound are highly suggestive of duodenal atresia.[3] Upon birth and first feeding, infants may develop symptoms such as bilious or non-bilious emesis, depending on the site of obstruction distal or proximal of the ampulla Vater.[3] Plain abdominal radiograph is diagnostic when "double bubble" sign (gas distension of dilated stomach and duodenum and absence of distal air) is seen as shown in **Figure 6.**[3]

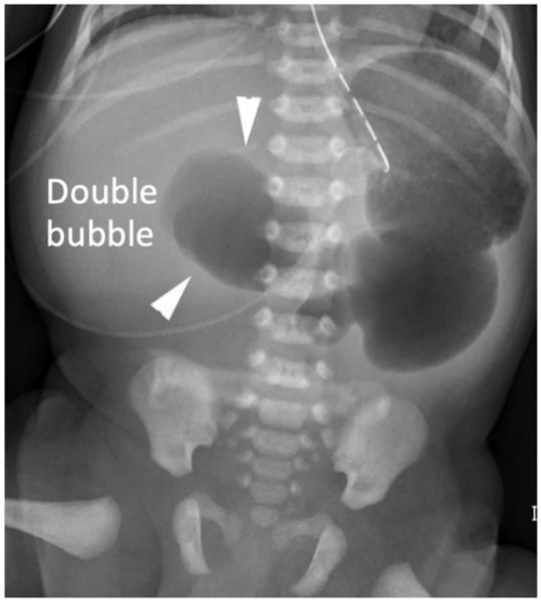


Figure 6. Abdominal radiograph showing "double bubble" sign in patient with duodenal atresia.[3]

Duodenal Stenosis and Duodenal Web

Duodenal stenosis occurs due to incomplete recanalization of the duodenum, resulting in persistent narrowing of duodenal lumen particularly on the second segment. Meanwhile, duodenal web refers to a condition in which a persistent membrane is partially obstructing the duodenal canal, with a predilection at the second part of the duodenum.[18] Hence, both duodenal stenosis and duodenal web are partial duodenal obstructions.

By the aid of the upper gastrointestinal contrast study, "windsock" deformity is pathognomonic to both duodenal stenosis and duodenal web.[3] "Windsock" deformity refers to a dilation of segment proximal to the stenosis and bulging of web into the non-dilated segment, as shown in **Figure 7**.[3] Furthermore, upper gastrointestinal contrast study is able to differentiate duodenal obstruction and midgut volvulus, which requires emergency surgery.[3]

Nasogastric tube should be gently placed when suspecting duodenal obstruction in order for decompression of the abdominal pressure. Furthermore, clinicians should prioritize in treating emergency conditions that may arise due to this abnormality, such as dehydration and electrolyte imbalances. The definitive treatment of duodenal atresia/stenosis/web is surgical repair (duodenoduodenostomy).

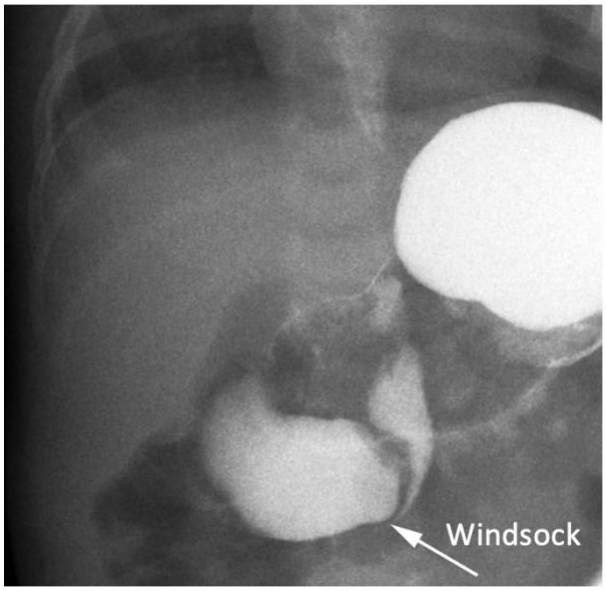


Figure 7. Upper gastrointestinal series showing "windsock" sign in patient with duodenal web.[3]

Malrotation and Midgut Volvulus

During the fourth to eight weeks of gestation, the small bowel rotates counterclockwise around the superior mesenteric artery axis. In this particular period, the bowel also protrudes through the yolk sac, elongates and rotates 90 degrees counterclockwise before retracting back into the abdominal cavity where another 180 degrees counterclockwise rotation occurs.[19] Malrotation occurs due to an error in this rotational process of the bowel during embryologic development, leading to abnormal fixation of the duodenojejunal junction and/or cecum within the peritoneum.[19] Patients with malrotation have a higher risk to develop midgut volvulus, resulting in bowel ischemia. Furthermore, in these patients, the associated Ladd's bands or peritoneal bands that attempt to fix the cecum might also cause duodenal obstruction.[3]

Classical symptoms of midgut volvulus are bilious emesis and abdominal distention with 75% of cases present as newborns.[20] However, in some cases, non-bilious emesis, hypovolemia and gastrointestinal bleeding may occur.[20] Over time, patients may also develop hemodynamic instability (hypovolemic shock), peritonitis (inflammation associated with volvulus) and hematochezia (bowel ischemia and necrosis).[3]

Plain abdominal radiograph is less useful in diagnosing midgut volvulus as "double bubble sign" produced by abdominal radiograph is not specific only for volvulus.[3] However, this modality may aid in demonstrating pneumoperitoneum in case of bowel perforation.[3] The gold standard modality to diagnose midgut volvulus is upper gastrointestinal series. However, this imaging should only be performed in hemodynamic stable patients. There are several signs that strongly suggest midgut

volvulus during upper gastrointestinal series such as abnormal position of duodenojejunal junction, dilated duodenum and corkscrew appearance of the duodenum (**Figure 8**).[3] On the other hand, even though ultrasound has lower sensitivity and specificity in diagnosing midgut volvulus, this device is more accessible and relatively easier to perform compared to the upper gastrointestinal series. The signs of midgut volvulus based on ultrasound are inversion of the superior mesenteric vein and the superior mesenteric artery and "whirlpool" sign due to twisted mesenteric vessels around the base of mesenteric pedicle.[3]

Nasogastric or orogastric tube should be placed to decompress the abdominal pressure in case of midgut volvulus.[21] Simultaneously, fluid resuscitation and correction of any electrolyte imbalances as well as administration of broad-spectrum antibiotics should be conducted before surgery.[21] Midgut volvulus is an emergency situation and need for immediate surgical intervention to reduce the volvulus.[21] Moreover, appendectomy is also typically performed during the procedure as the malposition of the appendix may make the diagnosis of appendicitis atypical and challenging to diagnose in the future.[22]

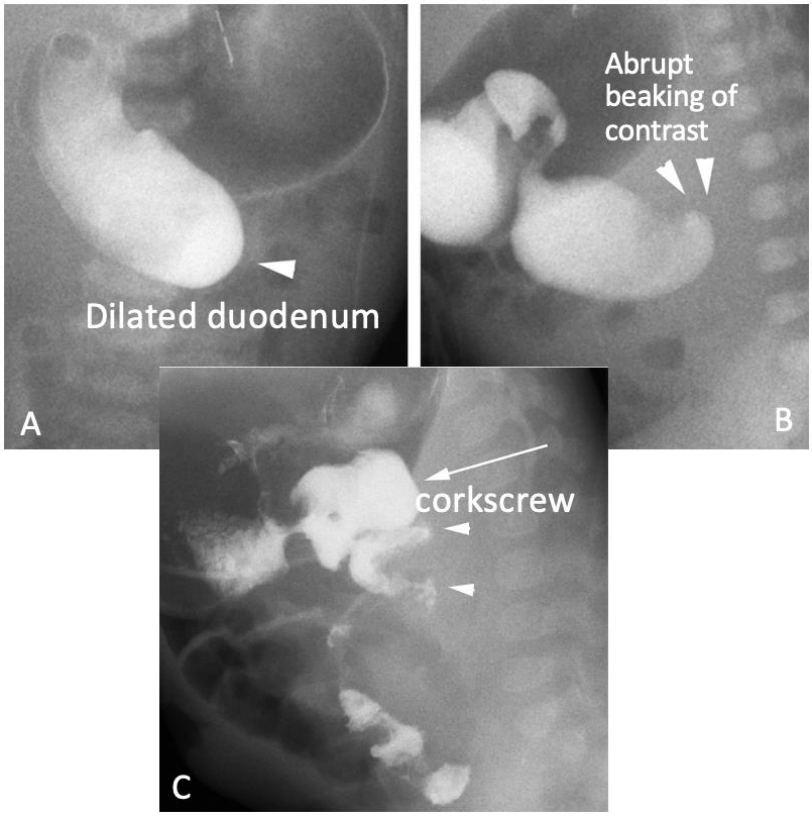


Figure 8. Upper gastrointestinal series of patient with midgut volvulus. (A) Dilated duodenum and abrupt cutoff of contrast from frontal view. (B) Abrupt beaking of contrast from lateral view. (C) "Corkscrew" appearance of distal duodenum.[3]

Lower Gastrointestinal Emergencies

Necrotizing Enterocolitis

Necrotizing enterocolitis (NEC) is the most common etiology of neonatal gastrointestinal emergencies with high mortality rate.[23] Primarily, this disease affects premature or low birthweight infants.[23] Various etiologies are known to be associated with NEC such as immature bowel function and disruption of the gut microbiota.[23] The pathophysiology of NEC is bacterial invasion due to inflammation of the intestinal wall that leads to necrosis of the colon and intestine and ultimately will also result in perforation and peritonitis.[23]

Symptoms of NEC often times are vague, variable and subtle, making clinical diagnosis become more challenging.[24] Patients can present with various complaints such as lethargy, mottling, bradycardia, abdominal distension, feeding intolerance and bloody stools.[24] In more severe cases, respiratory and circulatory failure may occur.[24] Therefore, imaging modalities play a crucial role in helping clinicians not only diagnosing but also detecting any complications. On abdominal radiograph, several signs can be found in case of NEC such as abnormal gas pattern, pneumatosis and portal venous gas (**Figure 9**).[3] In early stage, diffuse nonspecific gaseous pattern can be seen while in the later stage, fixed dilation or "persistent loop sign" can be observed, indicating an imminent bowel perforation.[3] Pneumatosis, which occurs as a bubbly appearance as gas enters the submucosa or subserosa layer, is a pathognomonic sign of NEC. Meanwhile, the presence portal venous gas is an indication of more severe disease that may require surgical intervention.[3] Lastly, pneumoperitoneum is a sign of full thickness bowel necrosis with perforation and is an absolute indication for surgery.[3]

The treatment modalities of NEC include stabilization of the patient's airway, breathing by intubating, fluid resuscitation if hypotension occurs, as well as nasogastric tube placement with nothing per oral.[25] Intravenous antibiotics should be given and should cover gram negative and anaerob bacteria. Surgical intervention or laparotomy is indicated when patients have worsening conditions despite medical treatments or patients have bowel perforation.[25] Recent systematic review and meta-analysis found an overall beneficial effect of probiotics in the prevention of NEC.[26] However, implementation in clinical practice has been difficult because of concerns about the efficacy and safety of probiotics due to variability of probiotic strains across the studies.[26]

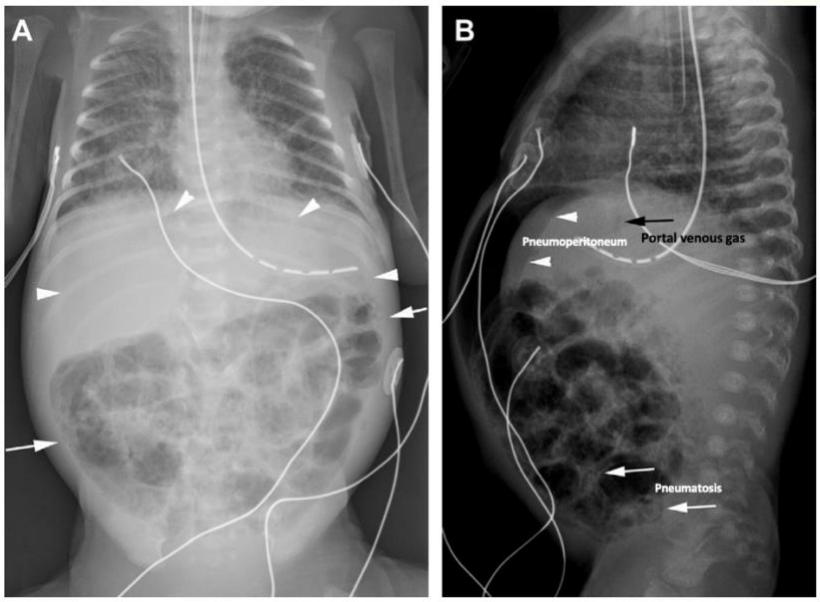


Figure 9. Abdominal radiograph of patient with NEC in (A) supine (B) lateral view showing multiple dilated bowel loops with pneumatosis (white arrow), pneumoperitoneum (white arrowhead) and portal venous gas (black arrow).[3]

Meconium-related Disorder

Meconium ileus occurs due to lumen obstruction by thick adhesive meconium and may be an early manifestation of cystic fibrosis in neonates.[27] In simple meconium ileus, thick meconium obstructs the terminal ileum causing the small intestine proximal to the obstruction site to dilate and fill with meconium, gas and fluid.[27] On the other hand, complex meconium occurs when the meconium-distended segment develops into volvulus, necrosis, ischemia or even perforation and spillage of meconium to the peritoneum, leading to meconium peritonitis.[27]

Infants usually present with intestinal obstruction with bilious emesis and abdominal distension within hours of birth and first feeding.[27] If meconium peritonitis has occurred, patients may present with abdominal tenderness, fever and even shock.[27] On plain abdominal radiograph, multiple dilated bowel loops with "soap bubble" appearance (meconium mixed with swallowed air) can be observed (Figure 10).[3] Typical obstruction sign such as air-fluid level sign is not usually seen in case of meconium ileus due to the thick consistency of meconium. Water-soluble contrast enema with hyperosmolar agents may be diagnostic as well as therapeutic and may show multiple filling defects consistent with meconium (**Figure 10**).[3] Meanwhile in meconium peritonitis, abdominal radiograph will show diffuse calcification throughout the peritoneal cavity and pseudocyst formation of the persistent spillage of meconium, which are highly predictive of the need for surgical intervention. [3]

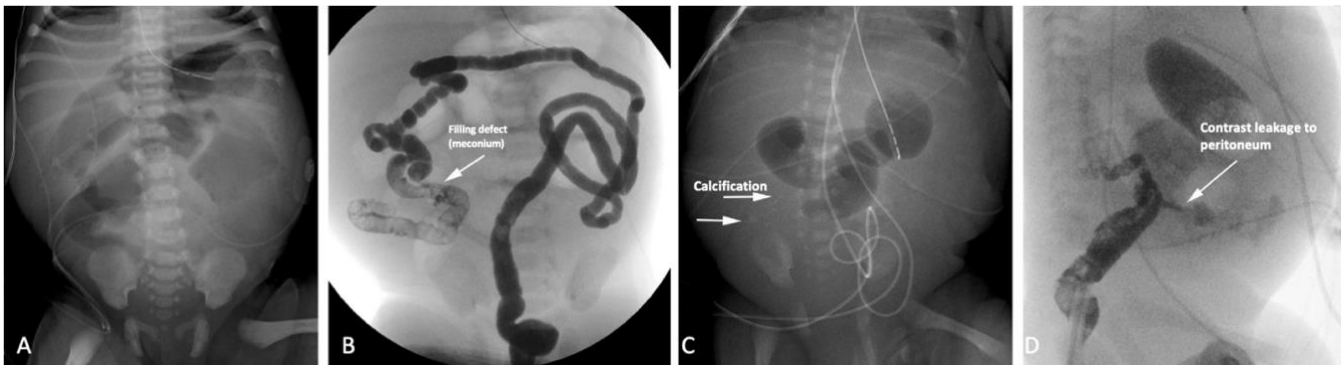


Figure 10. (A) Abdominal radiograph of patient with meconium ileus showing multiple dilated bowel loops and "soap bubble" appearance. (B) Water-soluble contrast radiograph demonstrating multiple filling defects, consistent with meconium. (C) Abdominal radiograph of patient with meconium peritonitis showing multiple scattered calcifications on the abdomen. (D) Water-soluble contrast radiograph demonstrating contrast leakage to the peritoneum, suggesting a perforation. [3]

Hirschsprung's Disease

Hirschsprung's Disease is once considered deadly with a prevalence of 1 in every 5000 live births and predominantly occurs in male infants.[28] This disease is characterized by the absence of ganglion cells at both the Meissner and Auerbach plexus in the terminal rectum and may extend proximally to a variable distance.[29] Hirschsprung's disease occurs due to a sudden arrest of migration and differentiation process of the neural crest cells at the enteric nervous system, leading to the absence of parasympathetic plexuses and overactivity of the intestine with persistent acetylcholine release.[29] As the consequence, continuous contraction causes narrowing of the colon while dilation occurs on the healthy proximal colon.

Hirschsprung's disease can be classified into 4 different types based on the length of the affected colon.[3] First is the ultrashort segment which only involves up to 4 cm of the distal rectum and can be easily missed by rectal suction biopsy if placed too deep.[3] Second is the short-segment Hirschsprung's disease which is the most common form with the aganglionic section extending to the mid-sigmoid colon.[3] On the other hand, long-segment Hirschsprung's disease usually affects colon segment proximal to the mid-sigmoid without involving the whole colon. Last is the total colonic aganglionosis if the aganglionic segment affects the whole part of the colon.[3]

The diagnosis of Hirschsprung's disease involves the combination of clinical signs and symptoms as well as radiological findings and histopathological evaluation of the biopsied sample. Infants with this disease typically present with polyhydramnios, bilious vomiting, failure to pass meconium in the first 48 hours and abdominal distention. Plain abdominal radiographs in case of Hirschsprung's disease shows multiple dilated bowel loops which is consistent with the description of distal bowel

obstructions. By using water-soluble contrast enema, a transition zone and altered rectosigmoid ratio can be observed as cone-shaped area of transition between aganglionic narrow segment and distended bowel segment proximally (**Figure 11**).[3] Furthermore, irregular contraction and mucosal irregularity is pathognomonic signs of Hirschsprung's disease but only present in 20% of cases.[3] Gold standard for diagnosing Hirschsprung's disease is rectal suction biopsy which shows the absence of ganglion cells in myenteric and submucosal plexus as well as hypertrophy of nerve fibers in the aganglionic colon segment.[3] The International Gastroenterology Committee recommends at least 2 biopsies including submucosal and mucosa layer with a minimum diameter of 3 mm.[30] Biopsies should also be performed at least 2 cm above dentine line and should be well oriented.[30] Furthermore, acetylcholinesterase staining can also demonstrate the increased parasympathetic activity of the nerve fibers.[30]

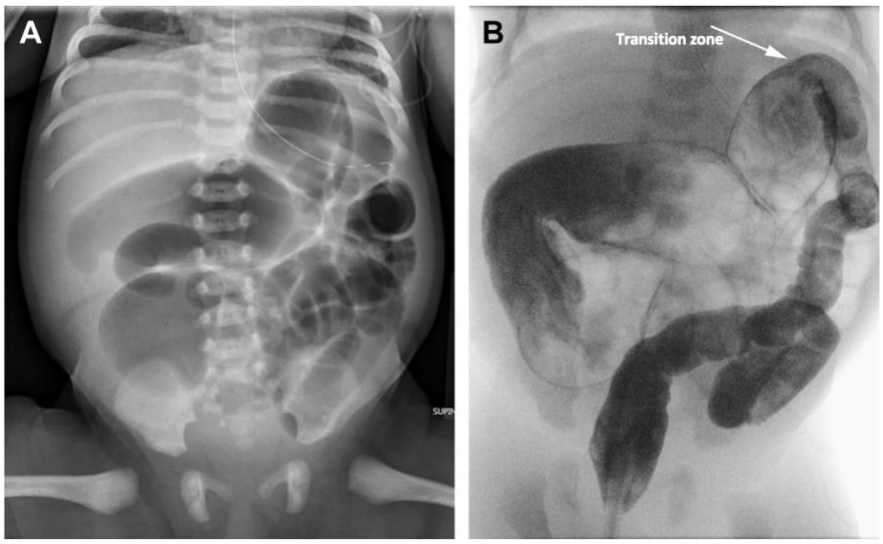


Figure 11. (A) Plain abdominal radiograph showing multiple dilated bowel loop without gas in the rectum. (B) Water-soluble contrast enema demonstrating transition zone and multiple filling defects.

Hirschsprung's disease usually requires staged reconstruction with an initial placement of temporary decompressive colostomy. Definitive pull-through surgery is typically performed four to six months after the colostomy placement. Rectal irrigation before the surgery is highly recommended to reduce the dilated colon size as well as to prevent the devastating complication of enterocolitis.[31] In case of Hirschsprung associated enterocolitis, administration of broad-spectrum antibiotics, fluid and electrolytes as well as rectal irrigation should be performed.[32]

Conclusion

Given the critical time window and the vulnerability of the neonate population, the proper identification and prompt treatment of neonatal gastrointestinal emergencies is crucial to minimize morbidity and mortality. Multidisciplinary management with neonatologists, pediatric surgeons, radiologists, and nursing staff working closely together can provide the best possible outcomes.

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Conflict of Interest

None declared.

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