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Bilious vomiting in the newborn

Lourdes Cohen *

Department of Department of Pediatric, Flushing Hospital Medical Center, USA

Abstract

Bilious vomiting in a newborn is a sign of bowel obstruction and can present with or without abdominal distention. The more proximal the obstruction is, the less distended the abdomen will be. A thorough physical exam followed by plain abdominal films often gives the diagnosis. Dilated loops of bowel or air fluid levels suggest the diagnosis of obstruction. Upper gastrointestinal or contrast enema study is often necessary to make a diagnosis. The causes of bilious vomiting in the newborn are duodenal, jejunoileal, and colonic atresias, meconium ileus, meconium plug, hypoplastic left colon, necrotizing enterocolitis, Hirschsprung disease and malrotation with midgut volvulus. The latter represents a neonatal emergency and prompt diagnosis and treatment are necessary to prevent rapid bowel necrosis.

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*Email: lcohen.flushing@jhmc.org

Introduction

Bilious vomiting in a newborn has numerous causes and it is a reason for admission to the neonatal intensive care unit. This short review describes the differential diagnosis of bilious vomiting in the newborn. It is important that any health care practitioner caring for newborns recognize the signs and symptoms of bowel obstruction, differentiate the various causes, and manage them in a timely manner to prevent significant morbidity or death.

Bilious vomiting in a newborn can be due to numerous reasons, and among them to be ruled out immediately is intestinal malrotation with midgut volvulus. This condition occurs when the gut failed to make the 270° rotation as it returns to the abdominal cavity. There is a spectrum of anatomic variants known as intestinal rotation anomalies [1]. The classic one refers to cases in which the duodenum does not cross over the spine with abnormal attachment of the retroperitoneum, resulting in a narrow mesenteric vascular pedicle, which is prone to twist, and causing midgut volvulus. It is difficult to determine the true incidence of intestinal malrotation since many cases are asymptomatic. Generally, the more severe the anatomic variant is the more prone to volvulus and the earlier it will present. Bilious vomiting is the most common presentation occurring in 95% of patients with malrotation and midgut volvulus. The abdominal exam initially is performed on a flat abdomen, in a previously asymptomatic baby. Abdominal distention, jittery, tachycardia, pale color, rectal bleeding and shock are late signs [2].

The diagnosis is made by an upper gastrointestinal (GI) contrast study. In malrotation, the duodenum and jejunum lie to the right of the spine. The duodenum may cross the spine, but travel downward rather than be positioned upward and to the left at the ligament of Treitz. The diagnosis might also be made by a contrast enema demonstrating malposition of the cecum.

When there is a volvulus associated with this condition, there is usually duodenal obstruction and a “corkscrew” appearance of the proximal duodenum at the point of the obstruction. This condition represents a neonatal emergency, and failure to make a timely diagnosis results in bowel ischemia very quickly. Once the diagnosis of volvulus is considered, the patient should be taken to the operating room (OR) immediately.

Other causes of bilious vomiting include intestinal atresias (duodenal, jejuno-ileal and colonic).

Duodenal atresia is due to failure of recanalization of the bowel lumen at the second portion of the duodenum. The prevalence is 1 per 5000 births. It is commonly associated with intraintestinal malformations such as annular pancreas, biliary atresia, choledocal cysts and anorectal malformations. In addition, duodenal atresia is associated in 30% of the cases with trisomy 21, VACTERL associations (Vertebral, Anal, Cardiac, Trachea, Esophagus, Renal and Limb anomalies) and congenital cardiac anomalies [3].

The diagnosis can be made prenatally by findings of polyhydramnios and ultrasound showing a classic "double bubble" sign. Postnatally, it can be diagnosed in a newborn who presents very early in life with vomiting. Duodenal atresia can present with bilious vomiting if the obstruction is distal to the ampulla of Vater or non- bilious vomiting if the obstruction is proximal to the ampulla of Vater and no abdominal distention. A plain abdominal film demonstrates the "double bubble" sign.

There are three major types of duodenal atresia. A mucosal diaphragmatic membrane with intact muscle wall characterizes type I, the most common. The proximal duodenum is dilated and the distal duodenum is narrowed. Type II has a short fibrous cord connecting the two ends of the atretic duodenum. Type III has a complete separation of the two ends of atretic duodenum.

The treatment of duodenal atresia is surgical correction, but in contrast with intestinal malrotation with volvulus, it is not a surgical emergency. The newborn does not need to be rushed to the operating room, but rather stabilized with nasogastric decompression and correct the electrolytes imbalances prior to entering the OR.

Jejunoileal and colonic atresia are also causes of bilious vomiting due to intrauterine vascular accident. The prevalence is reported some where between 1.6 to 2.8 per 10,000 births [4]. There is no sex predominance. Jejunoileal atresia is associated in one third of cases with prematurity, but it is not associated with other congenital malformations [2]. The clinical manifestations include maternal polyhydramnios, bilious vomiting and abdominal distention developing usually 12-24 hours after birth. There is delayed passage of meconium in the first 24 hours, however, 20% of the babies with jejunoileal atresia pass meconium within 24 hours of age.

There are classified as type I, which is a mucosal web or diaphragm with intact bowel wall and mesentery. Type II has a fibrous cord between the two blind ends of the atretic bowel and intact mesentery. Type III is sub classified as IIIa atresia where there is a complete separation of the atretic blind ends by a V-shape mesenteric gap defect. Type IIIb have an apple peel or Christmas tree deformity and the distal bowel receives a retrograde blood supply from the ileocolic or right colic artery. There is a type IV with multiple atresias with "string of sausage" or "string-of beads" appearance. A plain abdominal film confirms the diagnosis. High jejunal atresia is present with air-fluid levels and no further gas beyond that point and dilated small bowel loops. When there are calcifications present on the plain abdominal film, it is indicative of meconium peritonitis, which is a sign of intrauterine intestinal perforation. Microcolon is a common finding in babies with jejunoileal atresias and is due to lack of colonic distention. It is not necessary to do upper GI series in babies with radiographic evidence of complete obstruction. The treatment of this condition is surgical correction [5].

Colonic atresia without an imperforate anus or cloacal exstrophy is rare. The classification of colonic atresia is similar to jejunoileal atresia, but the apple peel type does not occur in the colon. It is rarely associated with other anomalies. It presents with failure to pass meconium in the first 24 hours of life, abdominal distention and bilious vomiting. Plain abdominal film reveals dilated intestine with air- fluid levels, there might be a "soap bubble" appearance due to mixture of meconium with air. The diagnosis is confirmed by contrast enema showing distal microcolon with dilated proximal colon. Treatment is surgical correction [6].

Meconium ileus is a form of distal bowel obstruction that presents in the neonatal period as bilious vomiting. It is due to thick viscid meconium. 10-20% of babies with meconium ileus are diagnosed with cystic fibrosis. The thicken meconium produces a form of mechanical intestinal obstruction. Usually the distal ileum and proximal colon contain inspissated pellets of meconium and the proximal dilated small bowel contains thick, tenacious meconium. The entire colon is of small

caliber due to lack of use. The abdominal exam will show a distended abdomen and pellets of meconium could be palpable. A plain abdominal film shows a "soap bubble" or "ground glass" appearance also known as Neuhauser's sign due to the mixture of air in the dilated loops of bowel with thick meconium. This is usually present in the right lower abdominal quadrant. A contrast enema usually releases the obstruction in 90% of the cases; the remainder cases might need exploratory laparotomy to extract manually the pellets of meconium causing the obstruction. Sometimes the obstruction can lead to intestinal perforation and peritonitis [7].

Meconium plug syndrome is another cause of bilious vomiting in the neonate that presents with abdominal distention and delay passage of meconium. It is caused by obstructing plug of meconium; the etiology is not well understood, but is believed to be due to immature myenteric nervous system producing ineffective peristalsis, with increase water absorption in the colon, leading to the development of thick meconium that causes the obstruction. The most common location of the plug is in the sigmoid and descending colon and therefore, it is not associated with microcolon, but the obstructed portion of the colon has a small caliber and it may look like small left colon syndrome. A contrast enema releases the obstruction in 50-60% of cases [8].

Small left colon syndrome is a dysfunctional, atrophied left colon that causes transient colonic obstruction with bilious vomiting and abdominal distention. It is observed in 50% of the infants of diabetic mothers and it is believed to be due to hypoglycemia induced glucagon release by the fetus. Glucagon produces smooth muscle constriction leading to transient partial colonic obstruction involving the descended and sigmoid colon. This condition is usually transient, resolves spontaneously in 48 hours as the infant's endogenous insulin normalizes. The diagnosis is made by contrast enema demonstrating small left hypoplastic colon [9].

Hirschprung disease presents itself in the newborn period with bilious vomiting, abdominal distention and delayed passage of meconium. It results from failure of the parasympathetic nervous system to migrate caudally in the intestine resulting in lack of relaxation and propagation of the peristaltic waves beyond the arrest. There is a functional obstruction at the point of farthest migration of the ganglion cells. It usually involves the rectum and sigmoid colon, rarely; about 10% of cases involves the entire colon [10]. It might present as an isolated defect or associated with chromosomes anomalies in 20% of cases or syndromes [11]. It is the most common form of neonatal intestinal obstruction. The transition zone is usually the sigmoid colon. The diagnosis is suggested by a contrast enema showing the transition zone, but it only can be made by rectal suction biopsy demonstrating lack of ganglia cells. The treatment is surgical and involves the extirpation of the aganglionic colonic segment with a "pull through" of the normally innervated colon. The procedure can be done in multiple stages with an initial colostomy and then 'pull-through' or in a single stag; primary "pull through".

Sepsis and necrotizing enterocolitis are other causes of bilious vomiting in the newborn period.

Neonatal sepsis presents with nonspecific signs such as apneas, respiratory distress, grunting, lethargy, hypotonia, hypothermia, unexplained tachycardia and abdominal distention. The plain abdominal film shows ileus and symptoms usually subside with antibiotics treatment.

Necrotizing enterocolitis, a disease on unknown origin, affects premature infants. It has a broad spectrum of manifestations with abdominal distention, hematochezia, bilious vomiting, signs of sepsis, shock and death. Plain abdominal film shows the pathognomonic sign of pneumatosis intestinalis, bowel distention, portal vein air and in severe cases pneumoperitoneum. The initial treatment is nonoperative with bowel rest, decompression, broad spectrum antibiotics and parenteral nutrition. In cases of intestinal necrosis or perforation, surgical treatment is indicated.

Conclusion

The differential diagnosis of a neonate with bilious vomiting is very broad. Proximal bowel obstruction includes duodenal, jejunoileal atresias. Distal bowel obstruction includes colonic atresia, meconium ileus, meconium plug, hypoplastic left colon and Hirschprung disease. Intestinal malrotation with midgut volvulus represents a neonatal emergency requiring prompt diagnosis.

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