REVIEW Pediatric imaging

Ultrasound evaluation of infantile vomiting: what a general radiologist should be aware of

Ilianna Tsetsou[1], Nektarios Spanomanolis[1], Hippocrates Moschouris[1], Marina Papadaki[1]
Department of Radiology, Tzaneio General Hospital of Piraeus

SUBMISSION 25/11/21 -ACCEPTANCE 03/03/22

ABSTRACT

Vomiting in infancy is often a diagnostic challenge. A variety of diseases can manifest with vomiting. Gastrointestinal tract disorders, both congenital and acquired, as well as metabolic, neurological, and inflammatory causes may all present with vomiting. Early diagnosis is of utmost importance, in order to rule out possible life-threatening conditions. Imaging plays an important role in the evaluation of an infant who vomits. Ultrasonography is a safe, easy to perform, and radiation-free imaging modality that allows the radiologist to accurately evaluate the gastrointestinal tract, as well as the abdominal organs. Moreover, in more experienced hands, it allows visualization of the infantile brain in suspected neurological causes.

KEY WORDS Infant, vomiting, ultrasonography, intestinal obstruction, radiology

Introduction

Vomiting in infancy is a common clinical problem and always requires further investigation. It is symptom induced most commonly by processes involving the gastrointestinal (GI) tract, such as gastroesophageal reflux disease and hypertrophic pyloric stenosis, while other rare causes include intestinal obstruction, usually due to volvulus or intussusception, and necrotizing enterocolitis. Additionally, abdominal inflammation, metabolic and neurologic disorders, urinary tract infections, and food allergies can also produce this symptom [1].

Clinical history must include prenatal and perinatal

CORRESPONDING AUTHOR, GUARANTOR
Ilianna Tsetsou
Email: ilianna.ts@gmail.com

data. Due to the infant's inability to effectively communicate with the physician, obtaining relevant clinical information is often impossible. The investigation is further complicated by non-specific clinical findings. Therefore, imaging in combination with relevant laboratory findings will ultimately suggest the disorder.

Ultrasonography (US) has proved to be a valuable imaging tool when investigating a case of an infant who vomits, while is an accurate, reliable, and rapid screening method [2]. Nowadays US is used as the initial imaging modality in an emergency department or in hospitalized infants and outpatients. Barium studies, computed tomography (CT), and/or magnetic resonance imaging (MRI) are reserved for selected cases.

This pictorial essay describes the most common causes of neonatal vomiting and their associated ultrasonographic findings, having in mind the general radiologist. The need for further imaging evaluation will be discussed.

Discussion

Gastroesophageal reflux disease

Gastroesophageal reflux (GER) is a normal phenomenon in neonates and infants that occurs mainly after meals and usually resolves by the age of 6-12 months [3]. Pathologic GER is called gastroesophageal reflux disease (GERD) and is associated with choking, irritability, non-bilious vomiting, wheezing and recurrent pulmonary infections [4].

24h-esophageal PH monitoring is still considered the gold standard for the diagnosis of GERD, while endoscopic imaging and manometry are performed in selected cases only [5].

Barium gastrointestinal series is recommended in infants where an esophageal or post-gastric obstruction is suspected. Ultrasonography may be proposed as an additional, non-invasive, and non-irradiating procedure that can add valuable information about the number of reflux episodes, the anatomy and position of the gastroesophageal junction, the length of the subdiaphragmatic esophagus, and the gastroesophageal angle. However, it is operator-dependent and further studies are required in order for it to determine treatment decisions [6].

Thirty (n=30) minutes before the examination the infant should be fed with at least 80ml of milk. This 30-minute interval is important in order to prevent normal post-prandial reflux. Examination starts by placing the transducer under the xiphoid process with the indicator facing cranially. The transducer is then slightly tilted to the left. This orients the beam in a coronal plane and positions the image so that the abdominal esophagus is visualized using the left liver lobe as an acoustic window. Total observation time should reach ten (n=10) minutes and the total number of reflux episodes should be counted [7]. The number of reflux episodes and the length of the intra- abdominal esophagus varies based on age and normal values are available in the literature [8]. A cut-off value of 4 episodes/10 min is considered within normal limits in healthy children during the first 2 months of life. Colour Doppler increases the sensitivity of the method due to more accurate detection of small and rapid reflux episodes [9].

Accurate measurement of the subdiaphragmatic portion of the esophagus is not always easy to obtain as its length shifts during peristalsis. The gastroesophageal junction is depicted as a small hyperechoic triangle pad of gastric folds and the cardiac orifice is the proximal point of this triangle pad. Proper measurement starts from the point of the esophageal hiatus to the orifice (Fig. 1). It should be noted that the gastroesophageal junction may prove difficult to locate, therefore waiting for a peristaltic wave allows for good visualization of the cardiac orifice. The gastroesophageal angle, the so-called angle of His, is the acute angle created between the cardia at the entrance to the stomach and the esophagus. In case of a short abdominal esophagus, protrusion of gastric folds in the thorax, or an abnormally enlarged gastroesophageal angle, the radiologist should suspect a sliding hernia [7].

Treatment is conservative in the vast majority of cases, while surgery is considered if conservative treatment fails.

Infantile hypertrophic pyloric stenosis

The incidence of infantile pyloric stenosis is reported at around 2% of live births. It typically affects infants between 2-8 weeks, with a reported 4:1 male to female ratio. Some of the affected infants are proved to have a compatible family history. The pathogenesis remains elusive; prolonged spasm of the pyloric muscle is considered a possible mechanism that leads to hypertrophy and subsequent gastric outlet obstruction [10].

Infants present with a history of late-onset, worsening, projectile, non-bilious vomiting after an initial period of normal feeding. If left untreated, the condition may lead to severe weight loss, dehydration, and electrolyte imbalance. A mass often described as an "olive", may be palpable during the physical examination and it represents the

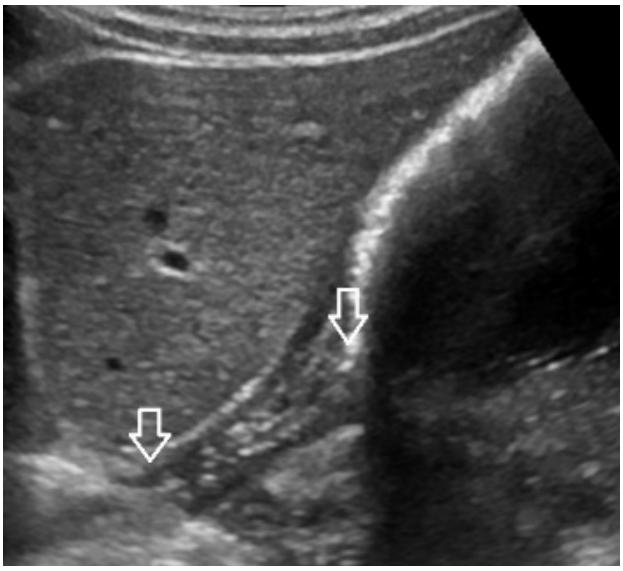

Fig. 1: *Length of the subdiaphragmatic esophagus measured between the diaphragmatic hiatus and the cardiac orifice (marked by white arrows).*

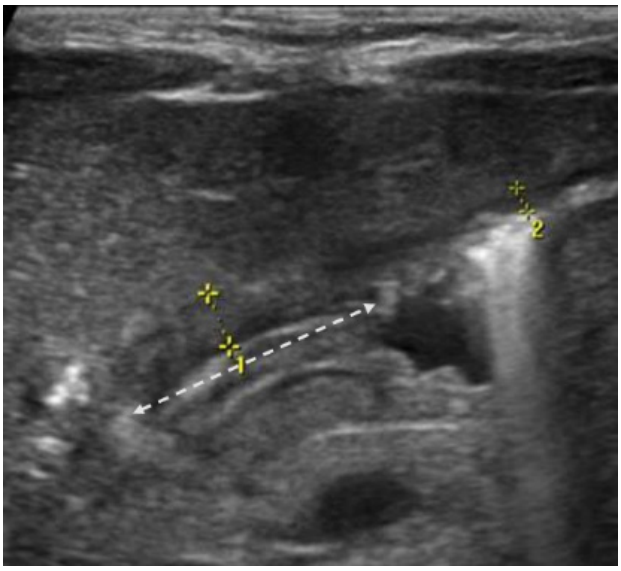

Fig. 2: *Hypertrophic pyloric stenosis. The muscle thickness is measured at 4mm (marked by 1st calliper). The 2nd calliper measures the stomach wall thickness to contrast the thickened pylorus.*

hypertrophied pylorus. Any suspicion of pyloric stenosis is a strong indication for US evaluation. No additional imaging studies are required [11]. Diagnosis is based on both morphological and dynamic criteria [12]. In an adequately hydrated child, a pyloric muscle thickness greater than 3mm, and a pyloric muscle length greater than 15-18mm are findings strongly suggestive of pyloric stenosis [13] (Fig. 2). The hypertrophied muscle appears mildly hyperechogenic, while a striated pattern may be observed during antral contractions. Colour Doppler may demonstrate hypervascularity in the axis of striations but does not provide additional diagnostic criteria [14]. A small amount of gastric content may pass through the pyloric canal during observation. However, the canal is unable to relax and the muscle remains thickened. The stomach is usually distended and a secondary GER may be observed. In borderline cases, the evaluation must be repeated within the next 24-48 hrs.

It should be mentioned that pylorospasm may sometimes mimic findings of hypertrophic pyloric stenosis. However, in pylorospasm, muscle thickness is usually less than 3mm and length is shorter than 15mm. Moreover, during the examination, there are variations in these values and findings, representing the functional aspect of this clinical entity rather than anatomic abnormalities [15].

The first step in treating an infant with hypertrophic pyloric stenosis is fluid resuscitation. After successful resuscitation, pyloromyotomy is the standard treatment of pyloric stenosis, while conservative therapy (atropine, naso-duodenal feeding) is employed in some cases [16].

Bowel obstruction

Obstruction of the small bowel and the colon is mainly due to congenital malformations. Both intra-luminal (atresia, stenosis, and webs) and external factors can lead to obstruction. Pre- and perinatal history plays a very important role in the correct identification of the etiology and in many cases, the level and cause of obstruction have already been demonstrated during prenatal imaging (US, MRI).

Bilious vomiting is the main symptom of any occlusion beyond the ampulla of Vater [1]. Delayed passage of meconium and abdominal distention may also occur. Delays in diagnosis and treatment could lead to bowel ischemia, peritonitis, and perforation. Thus, every infant who presents with vomiting should be initially evaluated for possible obstruction in order to avoid these life-threatening complications.

Imaging plays an important role in diagnosis and monitoring and sometimes it allows for therapeutic maneuvers as in uncomplicated meconium ileus and in meconial plug syndrome. Plain films, barium studies, and US are the mo-

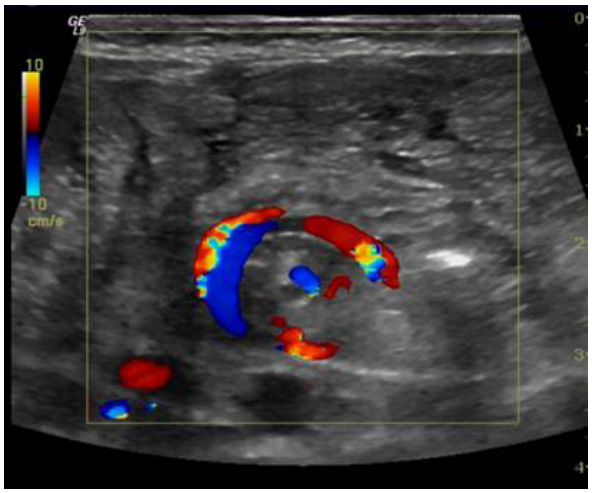

Fig. 3: *Malrotation with volvulus - whirlpool sign. Spiral twist of the superior mesenteric vein (red-blue) around the artery (blue). Colour Doppler is used to clearly demonstrate the vessels.*

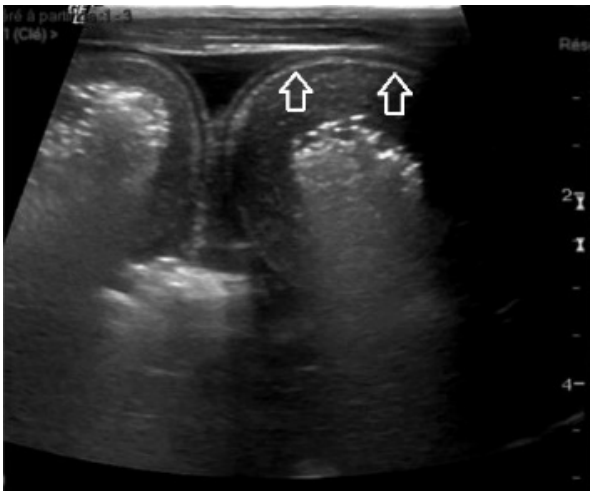

Fig. 4: *Meconium ileus. Bowel wall pseudo-thickening due to meconium layer stuck on the wall. The arrows show the true bowel wall margin.*

dalities of choice over the course of the evaluation.

Treatment of choice is surgery in the vast majority of patients, though it is not always performed on an emergency basis.

It is beyond the scope of this review to describe all possible obstructive pathologies. However, three distinct types are mentioned in detail, because they manifest with characteristic ultrasonographic findings that the general radiologist should be aware of. These are malrotation with volvulus, meconium ileus, and necrotizing enterocolitis.

Malrotation with volvulus

Malrotation is defined as an abnormal fixation of the small bowel mesentery, resulting in a short mesenteric root that is prone to twisting. Volvulus is the twisting of bowel loops around themselves and the accompanying mesentery that can lead to bowel obstruction, ischemia, and necrosis. Malrotation with volvulus mostly affects newborns, reportedly in over 75% of cases, but may occur at any age in patients with undiagnosed malrotation [17].

Typical symptoms suggesting volvulus include bilious vomiting and acute abdominal pain. In the case of intestinal ischemia, the infant may present with bloody diarrhea, abdominal distension, and lethargy. As a rule, any newborn with bilious vomiting is considered to suffer from malrotation with volvulus until proven otherwise [18].

An upper gastrointestinal study is able to indicate volvulus [19]. It is mostly of value in older, hemodynamically stable children suspected of malrotation, usually on a non-emergency basis. However, US has gained favor over the years and has demonstrated excellent diagnostic accuracy [20]. It should be carried out on an emergency basis, accompanied by an abdominal plain film in the supine position. The film is inconclusive in 30-40% of cases [21].

Some US findings are considered specific. The most important finding is the location of the superior mesenteric vein in relation to the superior mesenteric artery. Normally the vein is located right of the artery. Variations from this normal relationship may be suggestive of malrotation, although in some cases with proven malrotation the relationship is normal. Moreover, in case of complications, the dilated bowel loops may not allow for the visualization of the vessels [20]. Another sign highly suggestive of malrotation with volvulus is the "whirlpool sign" [22] (Fig. 3). A "whirlpool sign" is indicative of a spiral twist of the superior mesenteric vein around the artery. Sometimes the vessels, the mesentery, and intestinal loops may form a mass which is located right anterolaterally of the abdominal aorta. Distension of the proximal duodenum is usually present [20]. Any signs of intestinal ischemia should always be reported. These include thickened or extremely thinned small bowel walls, hypo- or aperistaltic bowel loops, free peritoneal fluid, and diminished or absent flow

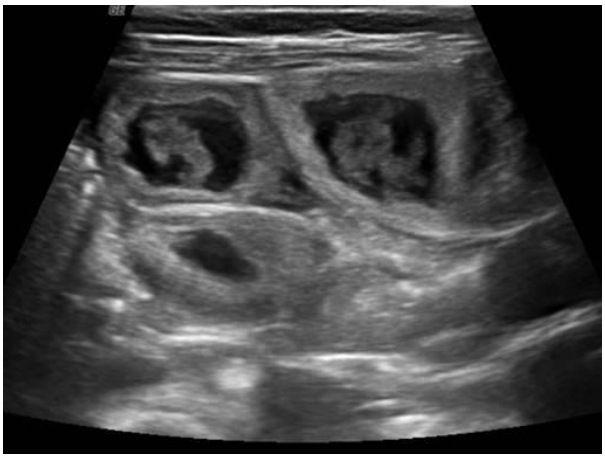

Fig. 5: Necrotizing enterocolitis with thickening of the small bowel wall due to ischemia.

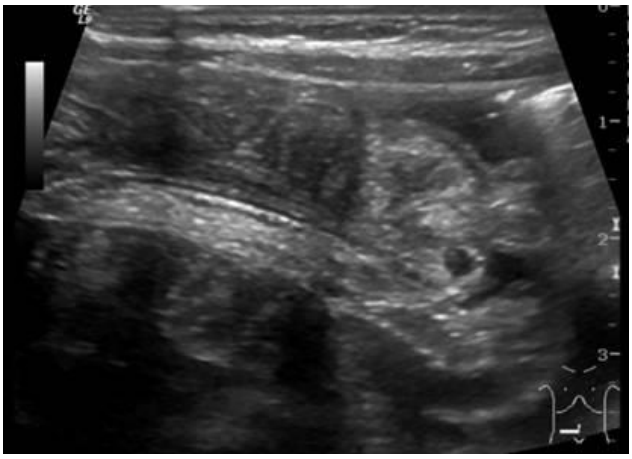

Fig. 6: Inverted Meckel's diverticulum. A tubular structure with a gut signature is demonstrated trapped inside a small bowel loop.

in the superior mesenteric artery [18].

Treatment of malrotation with volvulus is surgical.

Meconium ileus and complications

Abnormally thick and impissated meconium can lead to obstruction at the level of the distal ileum. This pathology, named meconium ileus, is strongly associated with cystic fibrosis and around 15% of infants diagnosed with cystic fibrosis present with meconium ileus [23]. Newer reports have indicated that it can be a rare occurrence among very low birth weight infants [24].

Vomiting, abdominal distention, and inability to pass meconium are the most typical symptoms and usually appear during the first days of life. Complications such as perforation and peritonitis can occur in half of the affected infants. Other associated findings are meconium pseudo-cysts and peritoneal calcifications [25].

An abdominal plain film is performed initially which may show distension of proximal loops or calcifications [26]. Enema with a water-soluble, hyperosmolar contrast agent is usually performed. Rehydration of meconium may be achieved, allowing it to pass the ileum with a rate of success is up to 60% of uncomplicated cases [27].

Ultrasound imaging is of great value in depicting the distended loops. They typically contain hyperechogenic content with pseudo-thickened walls due to the meconium sticking on them. Small air bubbles trapped within the abnormal meconium often create a granular pattern (Fig. 4). Another US finding is that of microcolon. A microcolon is defined as a colon of tiny diameter and it is indicative of an obstruction that happens above it. Moreover, US can spot complications such as ascites containing punctuate echoes or calcifications [28].

Necrotizing enterocolitis

Necrotizing enterocolitis is the most common surgical emergency in low-weight premature neonates. Its mortality rate is high, up to 50%, and mostly depends on the percentage of intestinal involvement [29]. Its aetiology is still unclear but it is considered to be due to a combination of bacterial infection and ischemia or hypoxia in an immature immune system.

Vomiting, abdominal distension, bloody stools, diarrhea, bradycardia, feeding intolerance, lethargy, sepsis, and apnoea are among the most frequently described signs and symptoms. Prompt diagnosis, even though difficult in most cases, is of paramount importance and leads to better clinical outcomes [30].

Plain abdominal film is normal in many cases or it may reveal some findings that raise the suspicion of necrotizing enterocolitis. These are pneumatosis intestinalis, portal venous gas, and pneumoperitoneum [31]. Ultrasonography may reveal important features and contribute to early diagnosis. Ascites and gaseous bowel dilatation are among the most common early findings. Intestinal and portal pneumatosis are also easily demonstrated [32]. Intestinal pneumatosis appears as echogenic foci in the bowel wall, typically in a submucosal location but can sometimes be subserosal. When echogenic foci are seen inside the portal vein and its branches they are indicative of portal pneumatosis. Thickening and increased vascularity of the bowel wall may be observed early in the course of enterocolitis

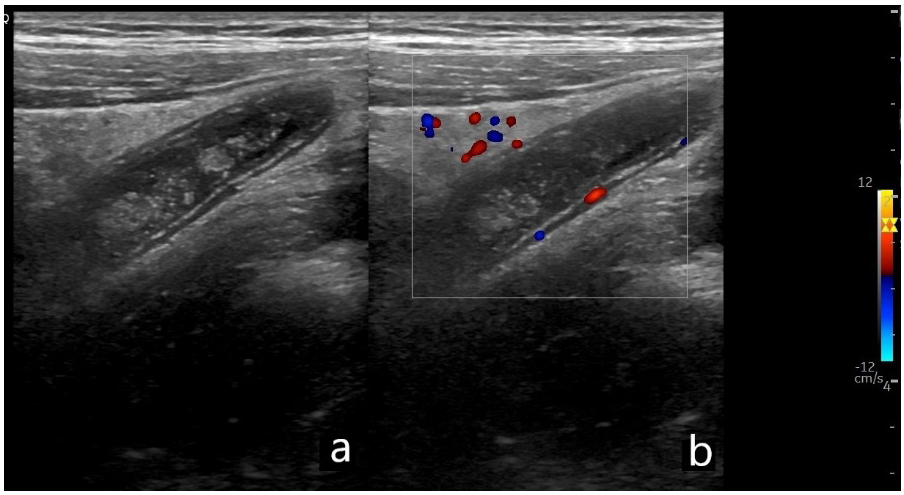

Fig. 7: *Appendicitis. Fig.7a: Inflammation of the appendix with hyperechoic surrounding fat. Fig.7b: Same patient with Colour Doppler imaging which demonstrates vascularity of the surrounding tissues.*

(Fig. 5). Thinned avascular bowel walls, together with ascites and extra-intestinal gas bubbles are all signs of perforation and severe disease that needs immediate surgical treatment [33].

Meckel's Diverticulum

Meckel's diverticulum (MD) is a congenital diverticulum, located in the distal ileum. It is the result of incomplete regression of the omphalomesenteric duct and it is the most common congenital anomaly of the gastrointestinal tract, with a reported prevalence in the general population around 2%. Most of the symptomatic patients are usually younger than 2 years old and the incidence of symptomatic disease decreases with age, thus being extremely rare in adults. There is a male predilection when it comes to symptomatic patients [34].

Typical complications of MD include intestinal obstruction, gastrointestinal bleeding or inflammation. Therefore, the most common symptoms include fever, abdominal pain, abdominal distention, vomiting, and bleeding from the rectum or melena. However, these symptoms are not specific for MD and they are often attributed to other, most common aetiologies, while MD is mostly found incidentally, especially during surgical exploration [35]. In younger children, the most common symptom is gastrointestinal bleeding and/or anemia, so the radiologist should always consider MD in those cases [36].

Ultrasonography is used for the initial workup of an infant with the aforementioned symptoms. An inflamed MD (MD diverticulitis) appears as a non-compressible, tubular structure, usually in the right iliac fossa with a "gut signature" sign, meaning with visualization of the layers of a typical bowel wall. Its wall may appear thickened and inflammatory changes in the surrounding mesenteric fat tissues are present [37]. It is usually misdiagnosed as an inflamed appendix or rarely a duplication cyst, thus it is important to look for its connection to the GI tract. MD should show a clear connection to ileal loops, while the appendix is connected to the cecum and a duplication cyst is typically not connected to the gastrointestinal tract [36]. Another possible US finding is an inverted MD, which is depicted as a "double target sign" due to alternating diverticular and small bowel walls with mesenteric fat between them [37] (Fig. 6). Other, non-specific findings of a complicated MD are those of ileo-colic intussusception, volvulus and obstructed bowel loops due to a mesodiverticular band [38].

As mentioned above, a definite diagnosis is made during abdominal exploration. Nevertheless, some imaging methods are more specific and can guide the radiologist to the correct diagnosis. Angiography can indicate the vitelline artery branching from the superior mesenteric artery towards the right iliac fossa. This finding is pathognomonic for MD. Technetium-99m scintigraphy is positive when an MD contains ectopic gastric tissue, with a reported sensitivity in pediatric populations of 85-90%. It has been proposed that certain medications, such as H2-antagonists, can increase the diagnostic accuracy by preventing the tracer's secretion from the gastric cells. Another utilized method is small bowel endoscopy [34,37]. However, all

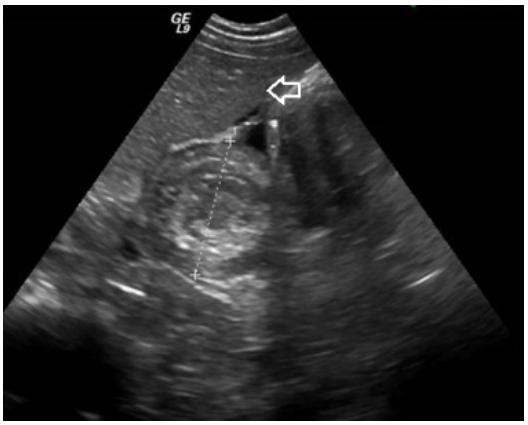

Fig. 8: *Ileo-colic intussusception. A typical target sign is shown (between markers). The white arrow indicates the liver.*

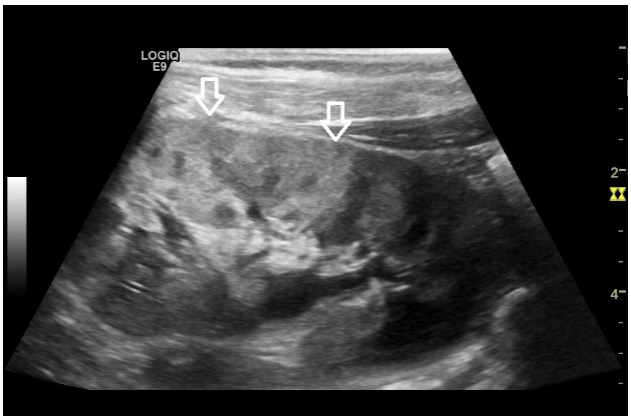

Fig. 9: *Pyelonephritis in an infant. The arrows demonstrate inflammatory changes of renal cortex with increased echogenicity and disruption of normal corticomedullary differentiation.*

these methods are not typically performed in infants.

Treatment of a complicated MD is surgical resection, whereas the removal of an asymptomatic, incidentally found MD is still controversial.

Acute Appendicitis

Acute appendicitis (AA) in infancy is relatively rare but it is associated with more complications and a higher morbidity and mortality rate. The reason for this is the atypical clinical manifestation, anatomical differences between older children and infants, as well as difficulty in communication and delayed diagnosis due to low suspicion. Thus, most infants with AA present with late complications, such as perforation or peritonitis [39]. Most infants with AA present with vomiting, pain, fever, diarrhea, irritability, cough, abdominal distention, or lethargy. Right lower quadrant pain is rare and most children present with diffuse abdominal tenderness.

The neonatal appendix is funnel-shaped, in contrast with the finger-like appendix of older children and adults. Thus, it is less prone to obstruction, and this is probably one of the reasons the incidence of AA in this age group is low, reportedly around 0,2%. Another possible protective factor is the liquid diet, which prevents the formation of fecaliths [40]. However, its wall is thinner, and the omentum is less developed and, in case of perforation, it is easier for intestinal content to spread rapidly and cause peritonitis [39]. Other associated conditions that can cause AA are Hirschsprung's disease, meconium plug syndrome, cystic fibrosis, gastroenteritis, and necrotizing enterocolitis. All those conditions can cause obstruction of the appendiceal lumen [40].

Ultrasonographic evaluation of the pediatric appendix can be challenging for the general radiologist. It is typically located anterior of the iliac vessels. If it is not visualized at its typical location, the radiologist should identify the cecum and the ileocecal valve and then search inferiorly [41]. A well-established technique is that of graded compression to displace the overlying bowel loops and to visualize the appendix. A normal appendix changes shape upon compression from circular to an oval shape, whereas when inflamed it is uncompressible. The maximum outer diameter is then measured while applying pressure. It is usually less than 6mm [42]. Some studies suggest that a cut-off value in the pediatric population could lead to diagnostic errors, because the diameter can vary depending on the age and comorbidities, such as cystic fibrosis which can cause a normal appendix to appear enlarged due to the mucus content. Other findings are mural thickening, which is defined as a wall thicker than 3mm, as well as increased vascularity (Fig. 7). Clinical entities that can mimic AA are mucocele, Meckel's diverticulitis, or reactive lymphoid hyperplasia of the appendix [41].

When the appendix is not clearly visualized, some additional indirect findings can indicate inflammation. Increased echogenicity of mesenteric fat, free fluid, enlarged mesenteric lymph nodes, or thickening of the adjacent ileal loops are some secondary findings [42].

Treatment of choice is usually surgical resection, although in some cases the child can be treated conservatively with antibiotics and careful observation [39].

Intussusception

Intussusception is the invagination of one part of the intestine into the section next to it. It mostly affects children between the age of 3-12 months, with more than 50% of cases concerning children younger than 1-year-old. It is the most common cause of intestinal obstruction [43]. In the majority of cases in infancy, it is idiopathic. However, some factors have been associated with intussusception, such as hyperplastic lymphoid tissue, anatomical variants, infections, a Meckel's diverticulum, tumours, intestinal duplication cyst, etc [44].

Intussusception is classified according to the affected intestinal segments and ileo-colic represents the majority of cases to a percentage of 80%. Other types of intussusceptions are ileo-ileal or colo-colic, with the last being extremely rare. Ileo-ileal intussusception is mostly idiopathic and transient [43]. At first, an infant presented with crampy abdominal pain, vomiting, and bloody stool. Later symptoms include those of acute abdomen, and they are a result of venous congestion, bowel ischemia, and possibly necrosis and perforation [44].

Ultrasonography is the test of choice in suspected intussusception, with high sensitivity and specificity and real-time evaluation of intestinal motility. Recent research suggests that the point-of-care US offers similar accuracy in the detection of intussusception while reducing the time needed for diagnosis [45]. A "target" sign represents the visualization of the intussusceptum in the intussuscipiens with its accompanying mesenteric fat and shows layers of intestine within the intestine at the axial plane. The same findings in the longitudinal plane make up the "pseudokidney" sign. In ileo-colic intussusception, this sign is observed in the right iliac fossa and usually measures 2-4cm. (Fig. 8). In the case of a different location, an ileo-ileal intussusception should be suspected. The presence of lymph nodes suggests ileo-colic intussusception. It is important to use Colour Doppler because the absence of blood flow in the affected segment indicates ischemia. Findings such as free fluid, trapped fluid, dilated bowel loops, or pneumatosis point towards obstruction.

Treatment of uncomplicated ileo-colic intussusception is air or hydrostatic enema reduction, while idiopathic, transient ileo-ileal intussusception usually resolves by itself and does not require medical intervention. Reduction is contraindicated in complicated cases due to the high risk of perforation [43].

Disorders not associated with the GI tract

Apart from GI tract disorders, many other clinical entities could lead to vomiting. Depending on the presentation and accompanying laboratory findings, the differential diagnosis may include urinary tract infections, food allergies, increased intracranial pressure, and inborn metabolism disorders.

Urinary tract infections (UTIs)

UTIs in infants may manifest with non-specific symptoms, such as fever, irritability, vomiting, and poor feeding. Other typical symptoms include ill appearance, a previous history of UTI, and suprapubic tenderness. There is a predilection for girls and uncircumcised boys. A positive urine culture is the gold standard of diagnosis and urinalysis provides additional information [46].

Current literature suggests that sonographic evaluation of the urinary tract should be conducted in all febrile infants with UTI [47]. Its role is to demonstrate findings of pyelonephritis, possible complications, and congenital malformations such as megaureter, collecting system duplication, and ureteroceles (Fig. 9).

Moreover, with the addition of contrast media, US is able to detect vesicoureteral reflux and other disorders. The technique is called contrast-enhanced voiding urosonography and should be performed if US reveals concerning findings, such as hydronephrosis and ureter dilatation. Its main advantage over voiding cystourethrography is the lack of ionizing radiation [48] (Fig. 10).

Food allergies

Food allergies typically cause diarrhea, intermittent vomiting, and malabsorption, but sometimes lead to enteropathy or food protein-induced enterocolitis syndrome (FPIES). It may manifest as profuse vomiting, dehydration, and lethargy. The diagnosis is established through supportive history in relation to food and these children rarely need imaging evaluation for diagnosis. FPIES can sometimes resemble necrotizing enterocolitis and differentiating between the two entities relies on history. Anaphylactic reactions are more severe and can also cause vomiting, although their diagnosis is out of the scope of this article [49].

Increased intracranial pressure (IICP)

Vomiting in infants can be a result of IICP. Infants with chronically elevated ICP usually present with macro-

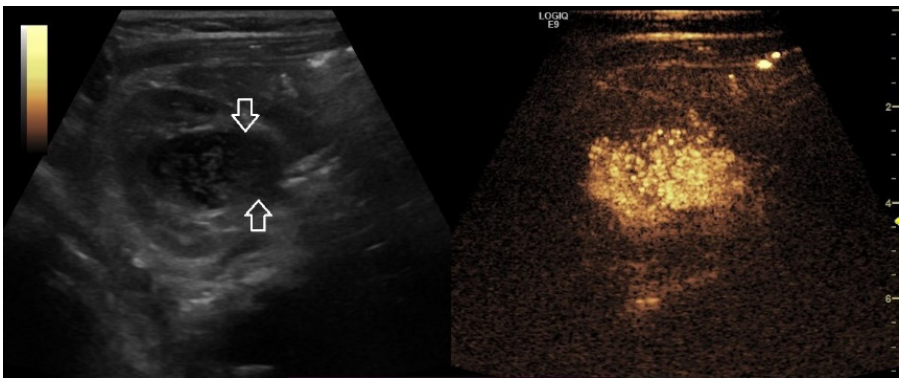

Fig. 10: *Grayscale ultrasound (left) and contrast-enhanced voiding urosonography (right) that clearly indicates reflux of contrast media in a dilated renal pelvis (shown between arrows in grayscale).*

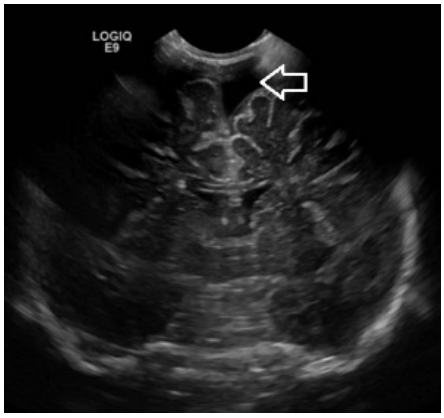

Fig. 11: *Cranial ultrasound in an infant with projectile vomiting where subdural hematoma is shown (arrow). This was a case of child abuse.*

cephaly and/or a bulging anterior fontanelle, because the cranial sutures are not yet fused in this age group. They may also present with lethargy and poor feeding. Acutely elevated ICP typically manifests with projectile vomiting, hypertension, altered mental status, and various other neurological symptoms. Trauma and/or abuse, hydrocephalus, brain tumours, and infections are the most common diseases that can cause IICP.

The gold standard for diagnosing elevated ICP is by directly measuring the ICP using an external ventricular drain or intraparenchymal monitor, although many non-invasive methods are utilized. Brain CT is the modality of choice but US methods play an important role in providing additional information.

Transorbital ultrasound is used both by adequately trained pediatric radiologists and physicians to measure the optic nerve sheath diameter and assess ICP [50].

Cranial ultrasound (CUS) is a valuable imaging modality during the first year of life due to its ability to monitor hydrocephalus and point out its cause, as well as other entities as haemorrhage, masses, and malformations. It is performed when an infant presents with increased brain circumference, neurological symptoms, or trauma and it is also utilized as a screening modality in selected cases. If any of the above cases is suspected, the child should be referred to a trained pediatric radiologist [51] (Fig. 11).

Metabolism disorders

Inborn metabolic disorders of the infant usually result in encephalopathy and manifest as vomiting, lethargy, and sepsis.

Many of these disorders can lead to organomegaly which can be detected by US. Glycogen, lysosomal, and lipid storage disorders such as Gaucher disease and Nieman-Pick disease can cause hepatomegaly and/or splenomegaly. Other diseases, such as neonatal hemochromatosis can lead to infantile cirrhosis [52]. R

Conflict of interest

The authors declared no conflicts of interest.

Acknowledgements

We thank Professor Stephanie Franchi-Abella (Department of Pediatric Radiology, Kremlin-Bicetre Hospital, Paris, France) who gave us permission to use figures 3,4,5,6 and 11.

REFERENCES

1. Alazraki AL, Rigsby CK, Iyer RS, et al. ACR Appropriateness Criteria® Vomiting in Infants. *J Am Coll Radiol* 2020; 17(11): S505–S515.
2. Gale HI, Gee MS, Westra SJ, et al. Abdominal ultrasonography of the pediatric gastrointestinal tract. *World J Radiol*. 2016; 8(7): 656.
3. Swischuk LE, Fawcett HD, Hayden CK, et al. Gastroesophageal reflux: how much imaging is required? *Radiographics*. 1988; 8(6): 1137–1145.
4. Tolia V, Wuerth A, Thomas R. Gastroesophageal reflux disease: Review of presenting symptoms, evaluation, management, and outcome in infants. *Dig Dis Sci*. 2003; 48(9): 1723–1729.
5. Vandenplas Y, Badriul H, Verghote M, et al. Oesophageal pH monitoring and reflux oesophagitis in irritable infants. *Eur J Pediatr*. 2004; 163(6): 300–304.
6. Minella R, Minelli R, Rossi E, et al. Gastroesophageal and gastric ultrasound in children: the state of the art. *J Ultrasound*. 2021; 24(1): 11–14.
7. Catherine Baud, Alain Couture, Jean Louis Ferran, et al. Ultrasonographic Imaging of the Esophago-Gastric Junction. In: *Gastrointestinal Tract Sonography in Fetuses and Children*. Springer, Berlin Heidelberg, 2008, pp 85-98
8. Koumanidou C, Vakaki M, Pitsoulakis G, et al. Sonographic Measurement of the Abdominal Esophagus Length in Infancy: A Diagnostic Tool for Gastroesophageal Reflux. *Am J Roentgenol* 2004; 183(3): 801–807.
9. Jang HS, Lee JS, Lim GY, et al. Correlation of color Doppler sonographic findings with pH measurements in gastroesophageal reflux in children. *J Clin Ultrasound* 2001; 29(4): 212–217.
10. Hernanz-Schulman M. Infantile Hypertrophic Pyloric Stenosis. *Radiology* 2003; 227(2): 319–331.
11. Hiorns MP. Gastrointestinal tract imaging in children: current techniques. *Pediatr Radiol* 2011; 41(1): 42–54.
12. Hsu P, Klimek J, Nanan R. Infantile hypertrophic pyloric stenosis: Does size really matter? *J Paediatr Child Health*. 2014; 50(10): 827–828.
13. Iqbal CW, Rivard DC, Mortellaro VE, et al. Evaluation of ultrasonographic parameters in the diagnosis of pyloric stenosis relative to patient age and size. *J Pediatr Surg*. 2012; 47(8): 1542–1547.
14. Catherine Baud, Alain Couture, Jean Louis Ferran, et al. Antro-Pyloric Abnormalities. In: *Gastrointestinal Tract Sonography in Fetuses and Children*. Springer, Berlin Heidelberg, 2008, pp 99-129
15. Cohen HL, Zinn HL, Haller JO, et al. Ultrasonography of pylorospasm: findings may simulate hypertrophic pyloric stenosis. *J Ultrasound Med* 1998; 17(11): 705–711.
16. Taghavi K, Powell E, Patel B, et al. The treatment of pyloric stenosis: Evolution in practice. *J Paediatr Child Health* 2017; 53(11): 1105–1110.
17. Stanescu AL, Liszewski MC, Lee EY, et al. Neonatal Gastrointestinal Emergencies: Step-by-Step Approach. *Radiol Clin North Am*. 2017; 55(4): 717–739.
18. Azarow K, Connolly B, Babyn P, et al. Multidisciplinary evaluation of the distended abdomen in critically ill infants and children: The role of bedside sonography. *Pediatr Surg Int*. 1998; 13(5–6): 355–359.
19. Seashore JH. Midgut Volvulus. *Arch Pediatr Adolesc Med* 1994; 148(1): 43.
20. Nguyen HN, Navarro OM, Guillerman RP, et al. Untwisting the complexity of midgut malrotation and volvulus ultrasound. *Pediatr Radiol* 2021; 51(4): 658–668.
21. Strouse PJ. Disorders of intestinal rotation and fixation ('malrotation'). *Pediatr Radiol*. 2004; 34(11): 837–851.
22. Patino MO, Munden MM. Utility of the Sonographic Whirlpool Sign in Diagnosing Midgut Volvulus in Patients With Atypical Clinical Presentations. *J Ultrasound Med* 2004; 23(3): 397–401.
23. Waldhausen J, Richards M. Meconium Ileus. *Clin Colon Rectal Surg* 2018; 31(02): 121–126.
24. Byun J, Han JW, Youn JK, et al. Risk factors of meconium-related ileus in very low birth weight infants: patients-control study. *Sci Rep*. 2020; 10(1): 4674.
25. Sathe M, Houwen R. Meconium ileus in Cystic Fibrosis. *J Cyst Fibros*. 2017; 16: S32–S39.
26. Leonidas JC, Berdon WE, Baker DH, et al. Meconium ileus and its complications. A reappraisal of plain film roentgen diagnostic criteria. *Am J Roentgenol Radium Ther Nucl Med* 1970; 108(3): 598–609.
27. Burke MS, Ragi JM, Karamanoukian HL, et al. New strategies in nonoperative management of meconium ileus. *J Pediatr Surg* 2002; 37(5): 760–764.
28. Veyrac C, Baud C, Prodhomme O, et al. US assessment

of neonatal bowel (necrotizing enterocolitis excluded). *Pediatr Radiol* 2012; 42(S1): 107–114.
29. Henry MC., Moss RL. Current issues in the management of necrotizing enterocolitis. *Semin Perinatol* 2004; 28(3): 221–233.
30. Rich BS, Dolgin SE. Necrotizing Enterocolitis. *Pediatr Rev* 2017; 38(12): 552–559.
31. D'Angelo G, Impellizzeri P, Marseglia L, et al. Current status of laboratory and imaging diagnosis of neonatal necrotizing enterocolitis. *Ital J Pediatr* 2018; 44(1): 84.
32. Kim W-Y, Kim WS, Kim I-O, et al. Sonographic evaluation of neonates with early-stage necrotizing enterocolitis. *Pediatr Radiol* 2005; 35(11): 1056–1061.
33. Alexander KM, Chan SS, Opfer E, et al. Implementation of bowel ultrasound practice for the diagnosis and management of necrotising enterocolitis. *Arch Dis Child - Fetal Neonatal Ed* 2021; 106(1): 96–103.
34. Hansen C-C, Søreide K. Systematic review of epidemiology, presentation, and management of Meckel's diverticulum in the 21st century. *Medicine (Baltimore)* 2018; 97(35): e12154.
35. Chen Q, Gao Z, Zhang L, et al. Multifaceted behavior of Meckel's diverticulum in children. *J Pediatr Surg* 2018; 53(4): 676–681.
36. Hu Y, Wang X, Jia L, et al. Diagnostic accuracy of high-frequency ultrasound in bleeding Meckel diverticulum in children. *Pediatr Radiol* 2020; 50(6): 833–839.
37. Kotha VK, Khandelwal A, Saboo SS, et al. Radiologist's perspective for the Meckel's diverticulum and its complications. *Br J Radiol* 2014; 87(1037): 20130743.
38. Elsayes KM, Menias CO, Harvin HJ, et al. Imaging Manifestations of Meckel's Diverticulum. *Am J Roentgenol* 2007; 189(1): 81–88.
39. Almaramhy HH. Acute appendicitis in young children less than 5 years: review article. *Ital J Pediatr* 2017; 43(1): 15.
40. Khan R, Menon P, Rao KLN. Beware of neonatal appendicitis. *J Indian Assoc Pediatr Surg* 2010; 15(2): 67.
41. Gongidi P, Bellah RD. Ultrasound of the pediatric appendix. *Pediatr Radiol* 2017; 47(9): 1091–1100.
42. Reddan T, Corness J, Mengersen K et al. Ultrasound of paediatric appendicitis and its secondary sonographic signs: providing a more meaningful finding. *J Med Radiat Sci* 2016; 63(1): 59–66.
43. Edwards EA, Pigg N, Courtier J, et al. Intussusception: past, present and future. *Pediatr Radiol* 2017; 47(9): 1101–1108.
44. Treasure Island: StatPearls Publishing. Child Intussusception. Available via https://www.ncbi.nlm.nih.gov/books/NBK431078/. Published January, 2021. Updated July 17, 2021. Accessed January 1, 2022
45. Lin-Martore M, Kornblith A, Kohn M, et al. Diagnostic Accuracy of Point-of-Care Ultrasound for Intussusception in Children Presenting to the Emergency Department: A Systematic Review and Meta-analysis. *West J Emerg Med* 2020; 21(4): 1008-1016
46. Zorc JJ, Kiddoo DA, Shaw KN. Diagnosis and management of pediatric urinary tract infections. *Clin Microbiol Rev*. 2005; 18(2): 417–422.
47. Mattoo TK, Shaikh N, Nelson CP. Contemporary Management of Urinary Tract Infection in Children. *Pediatrics* 2021; 147(2): e2020012138.
48. Duran C, Beltrán VP, González A, et al. Contrast-enhanced voiding urosonography for vesicoureteral reflux diagnosis in children. *Radiographics*. 2017; 37(6): 1854–1869.
49. Ovadia A, Nahum A, Tasher D, et al. Guidelines for the Diagnosis and Management of Food Allergy in the United States: Report of the NIAID-Sponsored Expert Panel. *J Allergy Clin Immunol* 2010; 126(6): S1–S58.
50. Yildizdas D, Aslan N. Is Ocular Sonography a Reliable Method for the Assessment of Elevated Intracranial Pressure in Children? *J Pediatr Intensive Care* 2021; 10(01): 014–022.
51. Caro-Domínguez P, Lecacheux C, Hernandez-Herrera C, et al. Cranial ultrasound for beginners. *Transl Pediatr* 2021; 10(4): 1117–1137.
52. Burton BK. Inborn Errors of Metabolism in Infancy: A Guide to Diagnosis. *Pediatrics* 1998; 102(6): e69–e69.

READY - MADE CITATION

Tsetsou I, Spanomanolis N, Moschouris H, Papadaki M. Ultrasound evaluation of infantile vomiting: what a general radiologist should be aware of. *Hell J Radiol* 2022; 7(2): 24-34.