SONOGRAPHERS QUICK REFERENCE GUIDE TO

Paediatric Bowel Pathology

APPENDICITIS

PATHOPHYSIOLOGY
Appendicitis can develop when the appendiceal lumen is obstructed by an appendicolith or foreign body and the appendix distends.[1]

PRESENTATION
Nausea, vomiting, anorexia, rebound tenderness, pyrexia and elevated white cell count may be symptoms of appendicitis.[2]
The appendix can be in one of several locations.[3] The tip may be located away from the RIF.

TECHNIQUE AND ULTRASOUND FINDINGS
A graded compression technique with both linear and curvilinear frequency transducers is used.[1] RUQ to RIF and region of pain examined in both supine and decubitus positions.
The maximum outside diameter is measured (Fig 1) with the appendix under compression.[1]

An inflamed appendix is noncompressible. An appendicolith may or may not be present (Fig 2). There is usually increased colour Doppler flow within the appendiceal wall and surrounding echogenic mesentry (Fig 3). Mixed echogenic fluid in RIF/inflammatory mass/abscess may be present in the case of a perforated appendix (Fig 4).[2]
Secondary signs of appendicitis may be present:
- echogenic mesenteric fat (compare to contralateral side)
- free intraperitoneal fluid in the RLQ
- enlarged intraperitoneal lymph nodes.

In the case of a perforated appendix – secondary signs may also include thickened adjacent bowel wall and debris in the urinary bladder.

MEASUREMENT
Normal appendix: 6 mm or less
Equivocal appendix: 6–8 mm and look for secondary signs
Appendicitis: above 8 mm.[1]

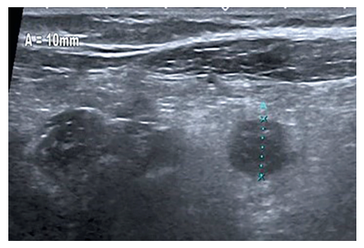

Figure 1

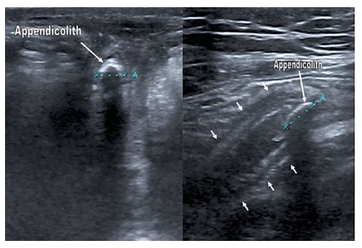

Figure 2

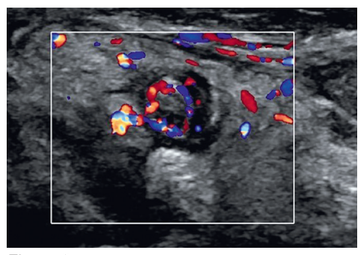
Figure 3

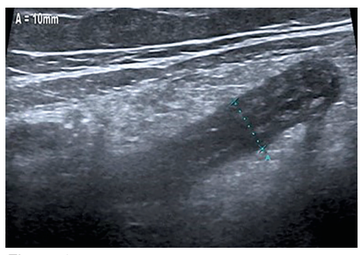

Figure 4

INTUSSUSCEPTION

PATHOPHYSIOLOGY
Intussusception is the invagination (telescoping) of a proximal bowel loop into the immediately adjacent distal bowel lumen. Can occur anywhere along the large or small bowel. If left untreated can lead to bowel obstruction, perforation and peritonitis. Intussusception can occur at any age; idiopathic cases occur most commonly between 6 months to 2 years. After 2 years suspect a pathological lead point. Small bowel intussusceptions are considered not clinically relevant unless the segment invaginated is longer than 3.5 cm.[4]

PRESENTATION
Intermittent abdominal pain with crying and drawing the knees to the abdomen, vomiting, and a right upper quadrant mass +/- lethargy and occult blood/red current, jelly-like stools.[4]

ULTRASOUND FINDINGS
Transverse appearances change along the intussusception.
- Transverse apex of the intussusception = ‘doughnut sign’ (Fig 5). Thick hypoechoic rim (oedema) and central echogenic focus (residual lumen).
- Transverse proximally = ‘target’ sign. The outer ring and inner circle are hypoechoic representing returning and entering walls. Separated by hyperechoic ring, consisting of bowel walls, mesentery and blood vessels.
- Longitudinal imaging of involved bowel = ‘hamburger’ sign, 3 parallel sections of hypoechogenicity separated by 2 echogenic lines (Fig 6). Outer sections represent returning walls, inner sections, the apex of intussusception. The 2 echogenic lines between them represent interfaces between returning and entering wall of the intussusception.

Transverse measurements across large bowel – greater than 3 cm with measurements across small bowel – less than 2 cm.[4,5]

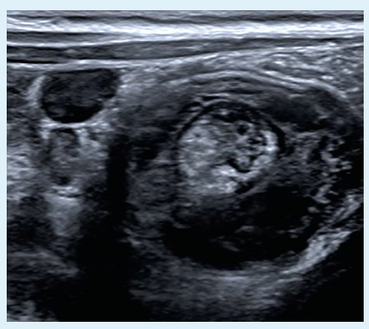
Figure 5

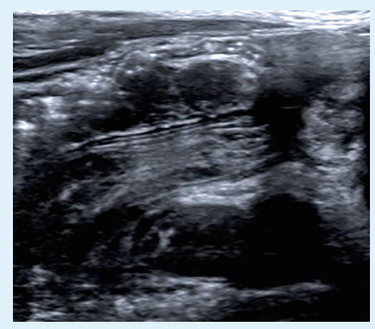
Figure 6

MALROTATION

PATHOPHYSIOLOGY
Intestinal malrotation is a congenital anatomical anomaly due to abnormal rotation of the gut as it returns to the abdominal cavity during fetal development. This predisposes the patient to the complications of midgut volvulus.

PRESENTATION
In the first week of life the baby’s symptoms may include vomiting, abdominal pain, abdomen swelling, diarrhoea, constipation, and bloody stools. These symptoms can also be seen within the first year of life for some children who avoid developing obstruction in the first week of life.

ULTRASOUND FINDINGS
The baby is usually lying supine. A high frequency linear transducer is preferable. Place the probe transversely over the portal confluence. Once it is identified, the transducer should be moved inferiorly. The normal position of the SMV is to the patient’s right of the SMA. The third part of the duodenum (D3) should be seen passing horizontally between SMA and the aorta.
Malrotation is when there is inversion of the superior mesenteric vein (SMV) and the superior mesenteric artery (SMA) (Fig 7). D3 will not be visible crossing the aorta. A volvulus is seen when the SMV is swirling around the SMA pedicle.

MALROTATION AND MIDGUT VOLVULUS
The intestine that is supplied by the superior mesenteric artery is referred to as the midgut. In this situation of midgut volvulus, the SMV twists around the SMA, referred to as the ‘whirlpool sign’ (Fig 8).

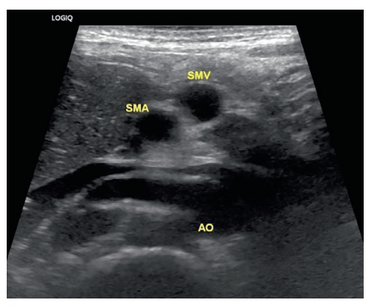

Figure 7

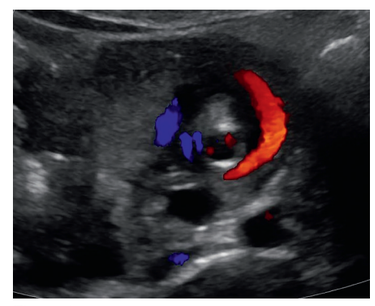
Figure 8

MECKEL’S DIVERTICULUM

PATHOPHYSIOLOGY
Meckel’s diverticulum, a remnant of the omphalomesenteric duct, is the most common congenital anomaly of the GI tract.

PRESENTATION
The most common clinical presentation is painless rectal bleeding. The rule of 2s is important to remember.[9]
- 2% of the population
- 2 inches (5 cm) in length
- 2 feet (60 cm) from ileocecal valve
- 2% become symptomatic.

ULTRASOUND FINDINGS
Meckel’s diverticulum most commonly appears as a tubular blind ending, mass-like structure arising from the ileum with a typical gut signature sign. The wall is generally irregular and thickened (> 3 mm). Meckel’s diverticulum has a clear communication with the small bowel. It can also appear as a cystic structure in the right iliac fossa with a differential diagnosis of a duplication cyst; however, unlike a duplication cyst the mucosal layer is irregular (Figs 9 and10). Colour Doppler often demonstrates anomalous vessels and hyperaemia of the walls.[9]

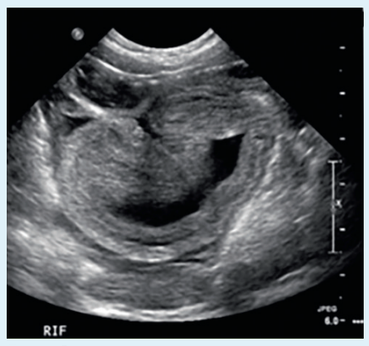

Figure 9

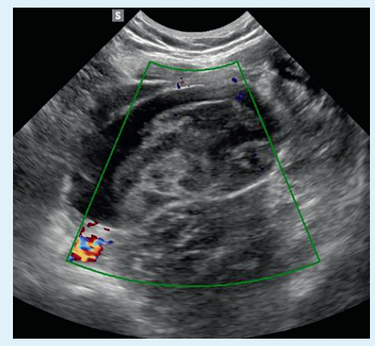
Figure 10

MESENTERIC AND DUPLICATION CYSTS

PATHOPHYSIOLOGY
Mesenteric cysts are lymphatic malformations of mesentery thought to arise from the embryonic retroperitoneal lymph sac during gestation. The lesions are composed of dysplastic lymphatic channels separated by thin septa. The channels contain chylous or serous fluid and lack connection with the normal lymphatic system. These lesions can occur anywhere in the mesentery.[10]
Duplication cysts occur as a result of duplication of the normal gastric/enteric wall due to abnormal embryological development (theories include persistent embryologic diverticulum and abnormal gut recanalisation). They can arise anywhere along the GI tract but most commonly occur at the ileum and foregut (oesophagus, stomach and duodenum).[11]

PRESENTATION
Often asymptomatic; however, symptoms may arise if the mass compresses on the adjacent organs or bowel. This can lead to abdominal distension, nausea, pain, bowel obstruction and/or change in bowel motions.

ULTRASOUND FINDINGS
Mesenteric cysts appear as loculated cystic mesenteric masses within the abdominal cavity. The internal fluid is commonly anechoic; however, can contain low level echoes or sedimentation (Fig 11). A rapid increase in size, pain, and internal echogenic material with fluid-fluid levels may indicate bleeding into the lesion.[10,12]
Duplication cysts present as unilocular cystic structures attached to the bowel, sharing a wall of hypoechoic smooth muscle, and internally lined with an echogenic mucosal membrane (Fig 12). The differential diagnosis to exclude in a female infant is an ovarian cyst.[11,12]

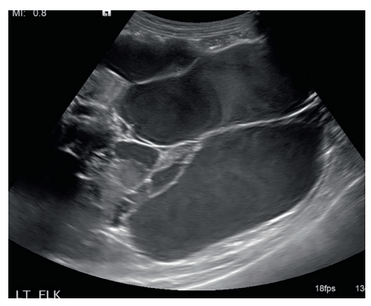

Figure 11

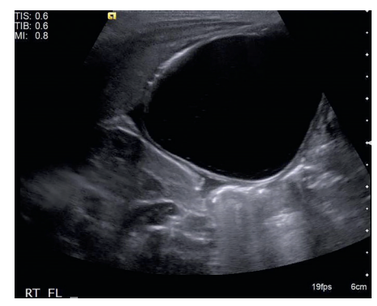

Figure 12

PYLORIC STENOSIS

PATHOPHYSIOLOGY
Hypertrophic pyloric stenosis (HPS) occurs due to hyperplasia and hypertrophy of the pyloric circular muscle; the aetiology of the condition is unknown.

PRESENTATION
Babies present usually between 3–6 weeks of age with a history of projectile vomiting. There is often some weight loss and an ‘olive’ mass may be felt in the upper abdomen.

ULTRASOUND FINDINGS
The baby is positioned supine and rolled towards the right. A high frequency, small, curved array or linear transducer should be used. The pylorus is located in the right upper quadrant adjacent to the gallbladder and imaged in transverse and longitudinal planes.
Opening of the pylorus and the passage of fluid through the duodenum should be demonstrated.
The ultrasound diagnosis is made from measurement of the muscle thickness, length of the pyloric canal and dynamic assessment of fluid transit.

MEASUREMENT CRITERIA FOR HPS
Length ≥ 17 mm[13] (Fig 13).
Wall thickness ≥ 3 mm[13,14] (Fig 14).

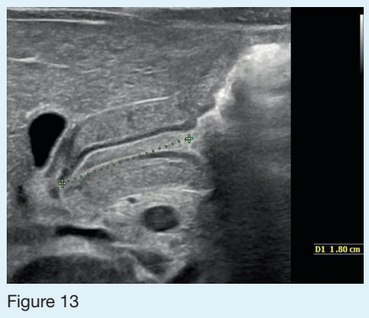

Figure 13


Figure 14

REFERENCES

1. Reddan T, Corness J, Mengersen K, Harden F. Ultrasound of paediatric appendicitis and its secondary sonographic signs: providing a more meaningful finding. *J Med Radiat Sci* 2016;63(1):59–66. doi:10.1002/jmrs.154
2. Quigley AJ, Stafrace S. Ultrasound assessment of acute appendicitis in paediatric patients: methodology and pictorial overview of findings seen. *Insights Imaging* 2013;4:741–751.
3. Hacking C. Appendix position (diagram). Case study, Radiopaedia.org. Accessed on 12 Nov 2021. https://doi.org/10.53347/rID-62911
4. Cogley J, O’Connor S, Houshyar R, et al. Emergent pediatric US: What every radiologist should know. *Radiographics* 2012;32:651–665.
5. Edwards E, Pigg N, Courtier J, et al. Intussusception: past, present and future. Pediatr Radiol 2017;47:1101–1108.
6. Applegate K & Anderson J. Radiographics intestinal malrotation in children: 1 Sept 2006.
7. Intestinal malrotation in children – Up to date. *Am J Roentgenol* 2002;179:1429.
8. Bensard D. Paediatrics: General medicine intestinal malrotation. 2018; Dec 19.
9. Kotha, et al. Radiologist’s perspective for the Meckel’s diverticulum and its complications. *BR J Radiol* 2014.
10. Wohlgemuth WA, Brill R, et al. Abdominal lymphatic malformations. Radiologe 2018;58(1):529–533.
11. Di Serafino, et al. *Ultrasound evaluation of the enteric duplication cyst: the gut signature*. *J Ultrasound* 2016;19:131–133.
12. Arraiza M, Metser U, et al. *Primary cystic peritoneal masses and mimickers: spectrum of diseases with pathologic correlation*. *Abdominal Imaging* 2015;40:875–906.
13. Forster N, Haddad RL, Choroomi S, Dilley AV, Pereira J. *Use of ultrasound in 187 infants with suspected infantile hypertrophic pyloric stenosis*. *Australasian Radiology* 2007;51:560–563.
14. Cascio S, Steven M, Livingstone H, Young D, Carachi R. *Hypertrophic pyloric stenosis in premature infants: evaluation of sonographic criteria and short-term outcomes*. *Pediatric surgery international* 2013 Jul 1;29(7):697–702.