



Sonographic assessment of infectious diseases of the gastrointestinal tract: from scanning to diagnosis

Margarita V. Revzin¹ · Mariam Moshiri² · Jamal Bokhari³ · John S. Pellerito⁴ · Christine Menias⁵

Published online: 20 January 2020
© Springer Science+Business Media, LLC, part of Springer Nature 2020

Abstract

Background Sonography of the gastrointestinal (GI) tract is a practical, safe, inexpensive, and reproducible diagnostic tool for the evaluation, diagnosis, and follow-up of infectious bowel disease. The modality is rapidly gaining prominence among clinicians on a global scale. In the United States, however, ultrasound of the bowel remains underutilized primarily due to insufficient experience among radiologists and sonographers in performing sonographic bowel assessment. This lack of experience and knowledge results in misinterpretations, missed diagnoses, and underutilization of this modality in patients with acute abdomen, with the majority of GI pathology on sonography discovered incidentally.

Objectives This article aims to demonstrate the characteristic sonographic findings associated with GI infectious processes as well as provide dedicated ultrasound protocols for evaluation of the GI tract.

Conclusion This article serves a twofold purpose, raising awareness of the utility of this imaging modality within the radiology community and also providing practical teaching points for sonographic evaluation of infectious disorders of the GI tract.

Keywords Bowel ultrasound · Gastrointestinal infections · Enterocolitis · Bowel perforation · Bowel physiology · Bowel anatomy · Gastrointestinal pathology

Introduction

In today's clinical practice, ultrasound is increasingly utilized for evaluation of visceral, vascular, and musculoskeletal pathologies, owing to the growing awareness of the risks of ionizing radiation, as well as recent technical advances

that have improved accuracy and imaging quality. Moreover, there has been a steady trend toward the use of more cost-effective techniques for diagnosis and treatment of patients, with ultrasound leading the way in this effort. However, in the United States (USA) the role of ultrasound has been under-represented in the evaluation of small and large bowel disorders, with most institutions utilizing the modality only for certain pediatric GI conditions or for suspected appendicitis. Bowel pathology on ultrasound is usually detected incidentally as part of a separate workup, for example during general abdominal or right upper quadrant (RUQ) examinations or during transvaginal pelvic sonography in females.

CME activity This article has been selected as the CME activity for the current month. Please visit <https://ce.mayo.edu/node/94495> and follow the instructions to complete this CME activity.

Electronic supplementary material The online version of this article (<https://doi.org/10.1007/s00261-019-02358-9>) contains supplementary material, which is available to authorized users.

✉ Margarita V. Revzin
margarita.revzin@yale.edu

Mariam Moshiri
moshiri@uw.edu

¹ Department of Radiology and Biomedical Imaging, Yale University School of Medicine, 333 Cedar Street, PO Box 208042, Room TE-2, New Haven, CT 06520, USA

² Department of Radiology, University of Washington Medical Center, Seattle, WA, USA

³ Department of Diagnostic Radiology, Yale University School of Medicine, 333 Cedar Street, PO Box 208042, Room TE-2, New Haven, CT 06520, USA

⁴ Department of Radiology, Zucker School of Medicine at Hofstra/Northwell, Northwell Health System, 300 Community Drive, Manhasset, NY 11030, USA

⁵ Radiology, Mayo Clinic Arizona, 5777 East Mayo Blvd, Phoenix, AZ 85054, USA

Ultrasound provides dynamic information regarding bowel distention and motility. Although there are some limitations associated with the use of ultrasound in evaluation of bowel pathology such as small field of view, variability in exam quality, patient body habitus, and scattered beam from bowel gas compared to other imaging modalities (computed tomography (CT) or magnetic resonance imaging (MRI)), an initial attempt at establishing a diagnosis via ultrasound should be considered.

This article aims to describe characteristic sonographic findings associated with bowel infectious processes, and hence raise awareness among radiologists to the potential utilities of this imaging modality for evaluation of disorders of the GI tract.

Anatomy and bowel mobility

The gastrointestinal tract is divided into several main segments, each of which has specific characteristics. The stomach has plicae that can become thickened when affected by infectious or inflammatory processes, or become distorted in case of ulceration or a neoplasm. The stomach is an intraperitoneal organ and is relatively mobile. The small bowel (SB) is divided into the duodenum, jejunum, and ileum. The duodenum is a predominantly retroperitoneal structure, with only its proximal portion covered with visceral pleura, making it less mobile. The jejunum and ileum contain a distinctive fold pattern, “*valvulae connivente*,” that helps to distinguish them from the large bowel on imaging studies. The proximal SB has more folds, while the distal SB has a comparatively lesser number of folds. The jejunum and ileum are entirely intraperitoneal structures and are relatively mobile, as they are attached via a long mesentery to the posterior abdominal wall. The large intestine is distinguished from the small intestine by the presence of “*teniae coli*,” which represent three thickened bands consisting of muscle, haustra, and omental appendices [1, 2].

Because the transverse colon, cecum, appendix, and sigmoid colon are intraperitoneal structures, they have longer attachments to the posterior abdominal wall and are therefore more mobile within the abdominal cavity. The ascending and descending colon and the rectum are less mobile due to their extraperitoneal location and their short attachments to the abdominal wall. Relatively immobile segments of the large bowel serve as points of reference during sonographic evaluation of the bowel, and therefore knowledge of these landmarks is essential in order to correctly interpret imaging findings [1].

Physiology and bowel motility

When fasting, the bowel is usually in a quiescent state, and demonstrates minimal motility. This significantly changes in the post-prandial state, when the bowel demonstrates more active peristalsis.

Assessment of bowel motility is of great importance, as many pathological processes affect bowel peristalsis. Bowel motility is generally divided into two main categories: the large peristaltic anterograde and retrograde waves that are observed in the pre-prandial state, and the small amplitude anterograde waves that are observed in the post-prandial state. The small bowel usually demonstrates more active peristalsis than the large bowel, although increased bowel motility (or hyperperistalsis) may be seen with colitis and enteritis. In contrast, markedly diminished bowel peristalsis may be observed in the setting of appendicitis and diverticulitis, resulting in “*quiescent bowel*” or a “*sentinel loop*,” which occurs in response to severe inflammation in order to minimize propagation and dissemination of disease mitigating the associated painful symptomatology [3].

Ultrasound technique

Currently in the USA clinical practice, with the exception of certain pediatric conditions and in those with suspected appendicitis, adult sonographic evaluation of gastrointestinal tract is not routinely performed. As stated earlier, bowel pathology is often discovered incidentally while performing other types of ultrasound exams. The patient’s presenting symptoms and laboratory abnormalities can play a pivotal role in directing the examiner to evaluate a particular area of concern increasing the likelihood of establishing a diagnosis. Requesting that the patient point to the area of pain with a single digit optimizes the ability of the sonographer to identify the site of bowel pathology.

In 2017, the European Federation of Societies for Ultrasound in Medicine and Biology (EFSUMB) published recommendations and guidelines for evaluation of the gastrointestinal system using ultrasound [4]. In their recommendations, the experts offered an overview of gastrointestinal ultrasound (GIUS) examination techniques for the assessment of the bowel in patients with variable GI conditions.

Equipment and ultrasound examination modes

The selection of an optimal transducer is of great importance for the assessment of the bowel. Imaging should initially be performed using a curvilinear probe (3.5–5.0 MHz), which provides assessment of a large field of view and offers optimal depth of penetration. Assessment of the bowel in all

four quadrants of the abdomen and pelvis allows for detection of asymmetry between different segments of the bowel wall, recognition of free fluid, assessment of inflamed perienteric/peri-colonic fat, determination of excessive or absent normal peristalsis, and presence of lymphadenopathy [5]. A higher frequency probe (7–12 MHz), owing to its better resolution capability, is utilized for evaluation of layers of the bowel wall and, if an abnormality is detected, delineating changes of the diseased segment. Attention should be paid to bowel wall thickness, length of the abnormal segment, presence of wall hyperemia, luminal distention, intraluminal contents, and the presence of solid or cystic masses related to the intestine. Bowel wall thickness should be measured only with higher frequency transducers [4].

Utilization of gray scale, color/spectral Doppler, and power Doppler modes is recommended since they play complementary roles. Color and power Doppler are utilized to evaluate bowel wall vascularity. Doppler parameters should be optimized to maximize sensitivity for the detection of vessels with low velocity flow in the bowel wall [4]. A more thorough analysis of certain bowel conditions may be performed via spectral Doppler interrogation of the mesenteric arteries and calculation of resistive indices. Sequential systematic scanning of small bowel followed by large bowel should be performed, in order to avoid misinterpretations and pitfalls in diagnosis.

Contrast-enhanced ultrasound

Contrast-enhanced ultrasound (CEUS) is an application that is also used in the assessment of gastrointestinal pathology and is most useful for differentiation of vascular from non-vascular intestinal or peri-intestinal lesions, including abscesses. The difference in perfusion between healthy and diseased bowel can be recognized by CEUS. CEUS is also used to quantify vascularity in the bowel wall. It is performed following injection of stabilized microbubbles with gaseous content into the blood stream. There are three main ways of interpreting contrast-enhancement in the bowel wall: (1) pattern of enhancement, (2) contrast quantification of peak intensity, and (3) dynamic contrast-enhanced ultrasound where intensity changes over time are analyzed [4]. An overview of the different techniques and non-hepatic applications of CEUS has been recently published by EFSUMB [6–9].

GI ultrasound elastography

With the advent of elastography as another application of ultrasound, the field of gastrointestinal elastography has emerged as an alternative approach for improved tissue characterization. Over the last decade elastography has been suggested as a tool for the assessment of diseases in the GI

tract [4, 10]. At this time the main utility of GI elastography lies in distinguishing benign from malignant lesions and in the monitoring of inflammatory bowel lesions for degree of inflammation or fibrosis. This application takes advantage of the changed elasticity, or stiffness, of soft tissues that results from specific pathological or physiological processes [11, 12]. The anatomy of the bowel, however, raises many challenges for strain or shear wave imaging, due to thin structures, non-constant boundary conditions, and intrinsic contractility. Pathological lesions tend to increase bowel wall thickness and may ease elastography imaging. Very few studies have addressed issues of bowel wall elastography to date, and both inflammatory and neoplastic lesions seem to increase tissue stiffness in the bowel wall [13].

It is important to note that the imaging-based differentiating features of infectious and non-infectious causes of bowel inflammation may not always be apparent, and the two entities commonly overlap. Usually the diagnosis relies on laboratory analysis, stool cultures, and the presence of characteristic clinical signs of an infectious process, including but not limited to fever, high white blood cell count, signs of peritonitis and sepsis, and history of prior antibiotic use (in case of *Clostridium difficile* (*C. Diff*) colitis). These features are less likely to be present in patients with inflammatory, ischemic, obstructive, or traumatic gastrointestinal processes. In some circumstances, more specific characteristic imaging features may be seen with different causative agents and may play a crucial role in establishing a correct diagnosis. For example, in the case of ascariasis, the roundworms are usually seen within the loops of small or large bowel. In cases of pancolitis due to *C. Diff* infection, the entire large bowel may be affected, and on ultrasound is characterized by marked circumferential thickening of the wall, mucosal hyperemia, and submucosal layer hypoechogenicity [14]. In contrast to Crohn's disease, in infectious bowel diseases the wall layers are almost always intact and the muscularis and serosa are almost never affected, whereas in Crohn's disease, depending on severity of inflammation, stratification of the wall may be preserved or destroyed [15]. There are usually no signs of mesenteric hypertrophy, bowel obstruction, abscess, or fistula formation seen in infectious processes (except for perforated appendicitis and complicated diverticulitis) [16]. Hyperemia may be present in both infectious and inflammatory bowel pathology. In general, each case should be assessed with a great attention to all pertinent information that will help to derive the correct diagnosis.

Ultrasound protocol for evaluation of the gastrointestinal system

A standard examination of the intestine does not need a specific bowel preparation. To reduce amount of food and air in the small bowel, a fasting period of at least 4 h is

recommended. Overnight fasting is recommended before assessing gastrointestinal motility [4]. Oral fluid contrast, either using water when evaluating the large bowel and stomach or iso-osmolar polyethylene glycol (PEG) (aka macrogol) solution when evaluating small bowel, can improve visualization of bowel disease.

The scanning technique for evaluation of bowel may vary according to the clinical problem. Based on the recommendations of EFSUMB, a general approach on how to perform the examination is described [4].

Initial assessment begins with evaluation of the small bowel with a low-frequency curved transducer placed in the mid abdomen. Scanning is performed in the transverse and sagittal planes through the entire mid abdomen and pelvis. Using orthogonal planes, the transducer should be moved from upper abdomen to the pelvis, and from the right to the left lateral margins of the abdomen (Video 1).

The large bowel is also examined in a sequential manner from a site of fixed or relatively immobile bowel in order to ensure correct identification of bowel segments. All parts of the large bowel are assessed. Scanning begins in the right upper quadrant, where the ascending colon is attached to the posterior margin of the peritoneum. Gradually the transducer is moved inferiorly along the ascending colon. The right iliac fossa is scanned in a transverse plane that helps in identification of the cecum. Appendix is located 3 cm inferior to the cecum. Subsequently, the transducer is placed to the initial starting point, and scanning is performed across the abdomen to the left, evaluating the transverse colon. Subsequently, the transducer is moved inferiorly along the descending colon to the left lower quadrant, where the sigmoid colon and proximal rectum can be identified. The rectum is assessed using a distended urinary bladder as an acoustic window. The anal region can be assessed by using either an endorectal, perianal, or in female patients via a transvaginal approach. In patients with suspected vascular injury or bowel ischemia, the mesenteric vessels, aorta, and branches can be interrogated with color and spectral Doppler ultrasound, obtaining waveforms and peak systolic velocities within splanchnic arteries and veins. Patency of the vessels, presence of any significant atherosclerosis, possible dissection, any significant stenosis/occlusion, or the presence of an aneurysm/pseudoaneurysm can also be assessed (Video 2). In general, the identification of haustrations and the location of the intestine help to differentiate the large bowel from the small bowel. Longitudinal assessment of the large bowel aids in identification of haustrations.

Using a high-frequency transducer, the thickness of the bowel wall should be measured only in the transverse plane of the bowel section, as images obtained obliquely may overestimate wall thickness [17]. The posterior bowel wall often is not possible to see due to air in the lumen; therefore, measurements should be made in the anterior wall [4].

Harmonic imaging should be activated when available as this may improve the delineation of bowel wall layers.

Graded compression technique is employed for assessment of acute appendicitis, diverticulitis, epiploic appendagitis, and other infectious/inflammatory processes. The probe is used to compress the abdomen while following the respiratory movements, enabling displacement of intra-abdominal fat and bowel segments and, thus, improving visualization of deeper located structures and mesentery. This technique will separate air-filled freely mobile bowel loops from the relatively non-compressible and non-mobile diseased bowel (Video 3) [18].

Appearance of the normal bowel on ultrasound

Healthy bowel segments are generally compressible and demonstrate normal peristalsis, with the wall having a stratified morphology. The wall consists of five concentric rings of alternating echogenicity, referred to as the “gut signature,” which can be seen when high-frequency transducers are utilized [5]. The first innermost hyperechoic layer is an echogenic line that represents the interface between the bowel lumen and the gut mucosa (referred to as mucosal layer). This is followed by a second hypoechoic layer that corresponds to muscularis mucosa, and the third layer which is an echogenic stripe corresponding to submucosa. The fourth hypoechoic layer represents muscularis propria, and finally the fifth outermost echogenic layer corresponds to the serosa (Fig. 1). Bowel wall thickness should be measured perpendicular to the wall from the interface between serosa and proper muscle to the interface between the mucosa and the lumen. If the colon is distended and filled with stool, bowel layers are very difficult to see even with high-frequency transducers. Visualization of the posterior bowel wall may be limited due to air in the lumen [4].

In the absence of pathology, the normal stratified bowel wall layers are preserved and are easily demonstrated by sonography. A bowel wall thickness less than 2 mm (not the cut-off value for pathology) could be considered as normal when measured in the normal filled state, with the exceptions of the duodenal bulb and rectum. The duodenal and rectal walls usually measure less than 3 and 4 mm, respectively [4, 19]. Bowel wall is considered thickened when measured greater than 4 mm. Normal bowel does not demonstrate increased vascularity, and therefore the detection of hyperemia on color Doppler imaging should raise concern for bowel pathology, specifically an infectious or inflammatory process. Color Doppler provides a semi-quantitative description of vessel density in the bowel wall, with normal bowel usually demonstrating 2 or fewer vessel signals per square centimeter [4]. Bowel loops are considered to be

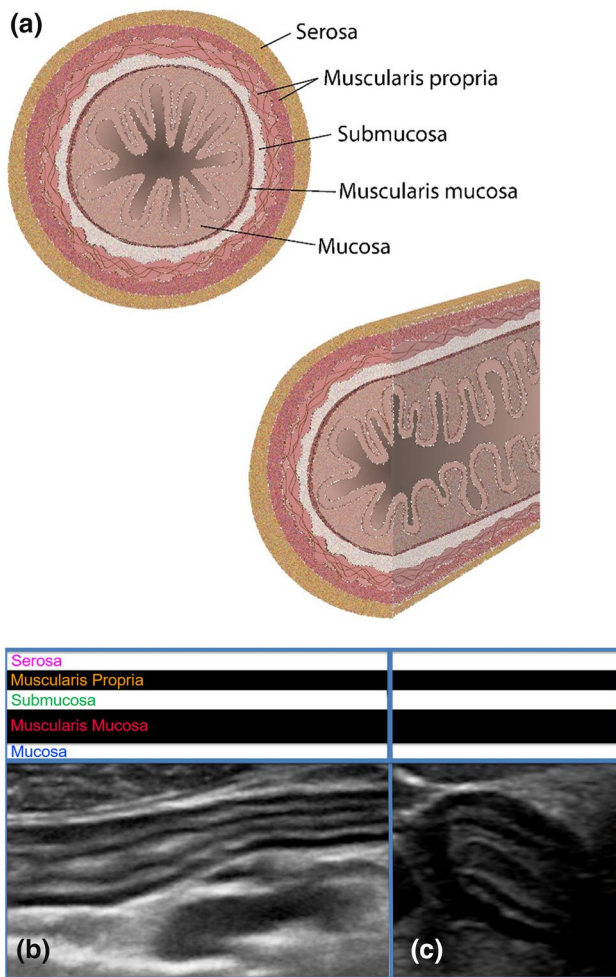


Fig. 1 **a** Schematic representation of the bowel wall anatomy in the axial and longitudinal views. Note 5 distinct layers of the bowel wall. **b, c** Gray scale of the small bowel in transverse and longitudinal planes demonstrating stratified appearance of the bowel wall on ultrasound consisting of five concentric rings of alternating echogenicity referred as the “gut signature”

dilated if the diameter of the small bowel loops is greater than 3 cm and the large bowel loops greater than 10 cm [20].

Stomach and small bowel

Gastritis

Acute gastritis is a general term that encompasses multiple causes of gastric mucosal inflammation/infection. In adults, the most common causative agent is *Helicobacter pylori*; however, gastritis may also be associated with use of non-steroidal anti-inflammatory medications (NSAIDs), autoimmune disorders, or related to an immunosuppressive state (CMV, candida albicans, histoplasmosis and more) [21].



Fig. 2 Eosinophilic gastritis in a 4-week-old male infant with projectile vomiting. Gray scale image in the transverse plane demonstrates prominent pylorus which measures 12 mm in length and has a single wall thickness of 2 mm. Gastric contents were seen passing through the pylorus on real-time examination. The echogenic gastric mucosa in the region of the gastric antrum and pylorus appear thickened and measures up to 3 mm (bracket) compatible with eosinophilic gastritis (milk allergy). *L* liver

In young children, an allergic reaction to milk may induce eosinophilic gastritis [22].

Differentiation of various forms of gastritis by imaging is not feasible due to significant overlap of findings. A definitive diagnosis may require clinical and laboratory correlation, and in some cases endoscopic examination with tissue biopsy [21].

On sonography, gastritis is characterized by circumferential thickening of the wall and folds of the gastric antrum, specifically affecting the mucosal and submucosal layers with significantly increased mucosal layer-to-antral wall thickness ratio (Fig. 2) [23]. Hyperemia and extra-gastric fat inflammation usually are not observed.

Eosinophilic gastritis (EG) is a relatively uncommon disease. It is characterized by diffuse infiltration of any or all layers of bowel wall by eosinophils. On ultrasound, there is marked thickening of the mucosa of the antrum and/or pylorus, with the presence of ascites and peritoneal nodules (Fig. 3) [24]. Establishing the correct diagnosis plays a pivotal role in patient management, as the symptoms improve with dietary restrictions and/or steroid therapy.

Infectious duodenitis

Duodenitis may be associated with inflammatory conditions such as pancreatitis and cholecystitis, or infectious etiologies, with the most common pathogen being

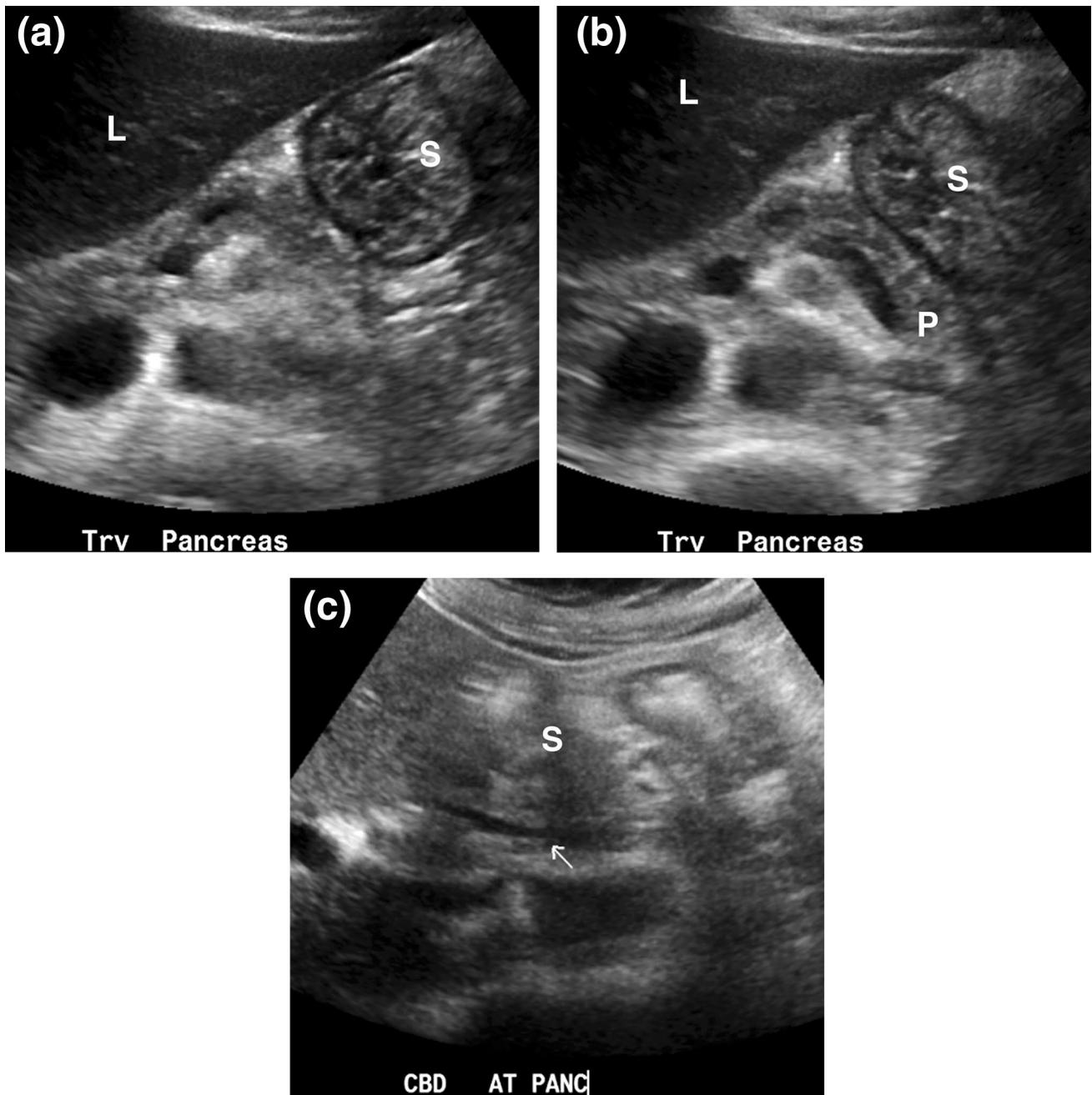


Fig. 3 Gastritis in a 65-year-old female with vomiting and epigastric pain. **a, b** Transverse gray scale image through the pancreas demonstrates circumferential thickening of the gastric folds and walls with predominant thickening of the mucosal and submucosal layers of the

gastric antrum, measuring over 5 mm (S). **c** Gray scale transverse image obtained 3 months earlier showed normal wall thickness of the gastric antrum. *P* pancreas, *L* liver

Helicobacter pylori. Less common infectious etiologies include giardiasis and tropical sprue. Wall thickening and luminal dilatation are two non-specific findings that can be observed with infectious duodenitis [25]. A small amount of peri-duodenal fluid and/or inflammation of the surrounding fat may also be seen on ultrasound imaging (Fig. 4, Video 4). Endoscopically obtained cultures usually help in establishing a definitive diagnosis.

Infectious enteritis

Infectious enteritis is defined as inflammation of the small intestine caused by a pathogen of bacterial, viral, or amoebic origin [26]. A key diagnostic feature of this entity is the distribution of the inflammatory process produced by the offending organism. The proximal small bowel is usually affected by parasitic organisms such as *Giardia* and *Strongyloides* species and *Mycobacterium avium-intracellulare* (MAI), whereas the ileum is most commonly affected by bacterial pathogens including *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia* (Fig. 5). The distal ileum is prone to infection by tuberculosis (TB), amebiasis, and Gram positive enteric commensal bacteria in the neutropenic patient that results in typhlitis (Fig. 6) (Table 1). Depending on the causative agent, management is usually conservative with supportive therapy and medications aimed at the type of infection. The diagnosis commonly relies on imaging findings, and therefore, a safe, quick, and well-tolerated imaging modality would be desirable. Ultrasound has been shown to have a comparable sensitivity in the diagnosis of enteritis when compared to CT and MRI [18, 27, 28]. The major sonographic features of enteritis include hypoechoic small bowel wall thickening and concomitant enlargement of mesenteric lymph nodes [29]. A bowel wall thickness of greater than or equal to 4 mm is considered abnormal; however, assessment of the degree of bowel wall thickening is somewhat subjective and depends on the degree of distention [4]. When only a limited segment of bowel is affected, it is recommended to compare bowel wall thickness in the diseased segment to wall thickness in clearly non-diseased segments, in order to minimize overdiagnosis. The mucosa and submucosa are primarily affected in the early stages of the disease characterized by loss of demarcation between these two layers and disproportionate prominence of echogenic

mucosa. Affected bowel loops are usually prominent and fluid-filled, and a small amount of peritoneal free fluid may also be present (Fig. 5). Inflammatory changes in the perienteric fat can manifest as increased echogenicity, signifying inflammation and edema of adjacent soft tissues [30]. In mild cases, bowel motility may be increased; however, the opposite may occur in severe forms of the disease. It is important to acknowledge that several other processes can have similar sonographic features and can therefore mimic enteritis on ultrasound. Differentiation from inflammatory bowel conditions such as active Crohn's disease may not be feasible as radiological findings can overlap. When terminal ileitis is detected, sonographic control is recommended to exclude Crohn's disease unless the patients present with fever, implying a more likely infectious etiology, or skip lesions or transmural complications such as fistula or abscesses are also present, which clinch the diagnosis of Crohn's disease. Additionally, cases of severe Crohn's flare-up are usually characterized by substantial wall thickening, up to 12 mm (Fig. 7) [15].

Parasitic infection: ascariasis

Parasitic infections may not result in thickening of the bowel wall, but rather present with intestinal obstruction or visible portions of the organism within the lumen of the bowel. For example, Ascariasis, infection by the organism *Ascaris lumbricoides*, can be diagnosed on ultrasound by detection of the adult worms with well-defined echogenic walls within the bowel lumen. They appear as avascular hypoechoic target-like structures in short axis, and tubular in long axis. Commonly, there is more than 1 worm within the bowel lumen resulting in bowel distention. Worm movements may be seen during real-time imaging (Figure 8). Imaging assessment of pulmonary parenchyma and visceral organs (liver/pancreas) is essential, as there is known involvement of these structures at different stages of the parasitic development [31].

Infected Meckel's diverticulum

Meckel's diverticulum is a true diverticulum and is the most common congenital anomaly of the gastrointestinal tract. It is found in approximately 2–3% of the population with

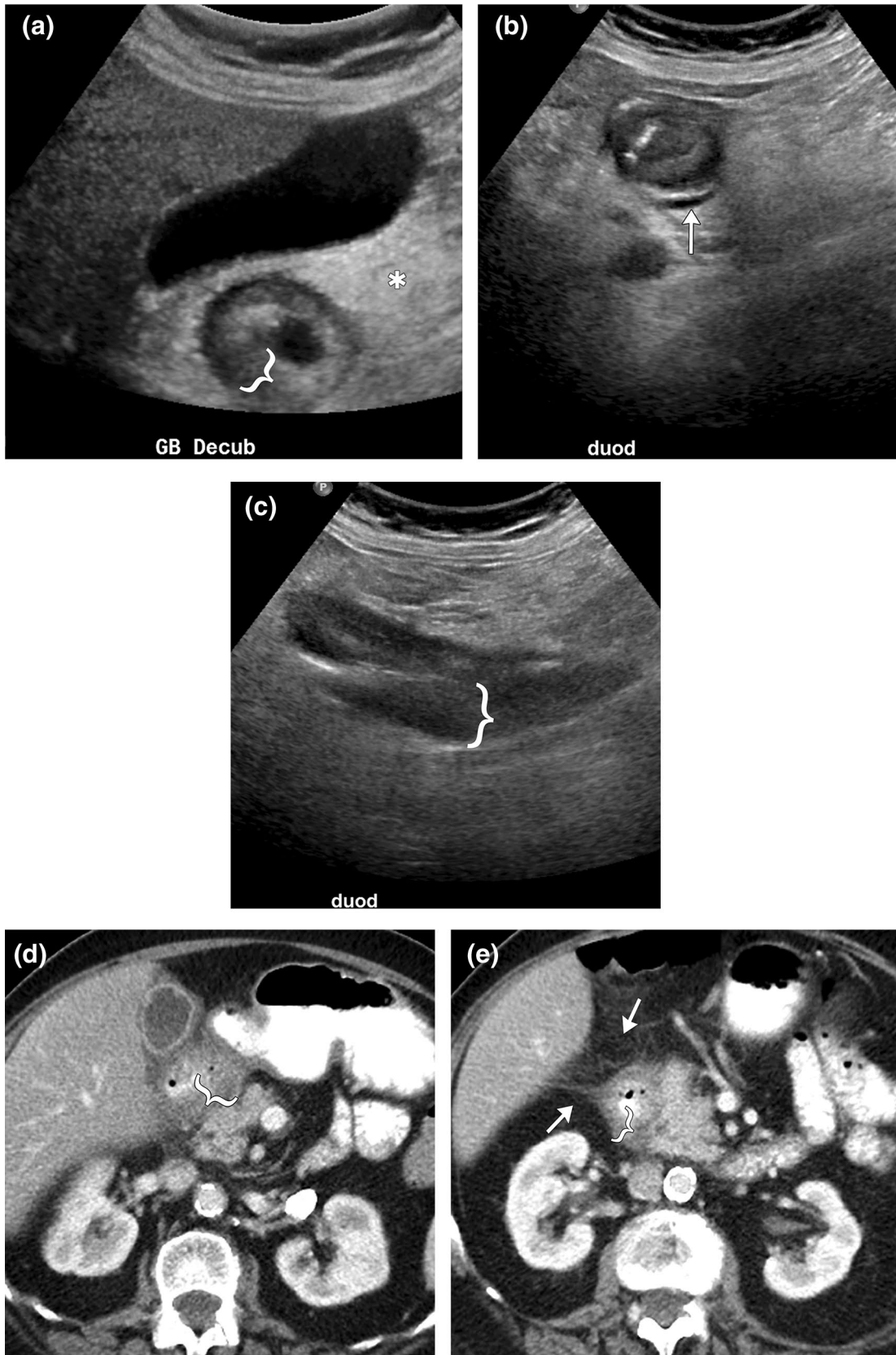


Fig. 4 Duodenitis in an 84-year-old female with epigastric pain for 1 month. **a, b** Transverse gray scale images of the descending portion of the duodenum demonstrate markedly thickened duodenal wall (brackets). A small amount of free fluid is noted adjacent to posterior wall of the duodenum (arrow in **b**). **c** Gray scale image obtained in longitudinal plane demonstrates marked thickening of the descending and part of horizontal portions of the duodenum. Note echogenic fat around the duodenum compatible with inflammation (asterisk). **d, e** Contrast-enhanced CT images in the axial plane show marked thickening of the first and second portions of the duodenal wall (brackets) and inflammatory changes in the peri-duodenal fat (arrows). Note that the pancreas is not involved (P)

no gender predilection and represents failure of the omphalomesenteric (aka vitelline) duct regression during fetal development [32, 33]. In the majority of cases, a Meckel's diverticulum is located within 100 cm of the ileocecal valve on the anti-mesenteric border of the distal ileum. Its walls are composed of all layers of the intestinal tract, and it usually measures 5 cm in length and 2 cm in diameter. Although most Meckel's diverticula are asymptomatic, complications can occur in 4–40% of patients and include superinfection (diverticulitis) as well as intestinal obstruction and ileoileal/

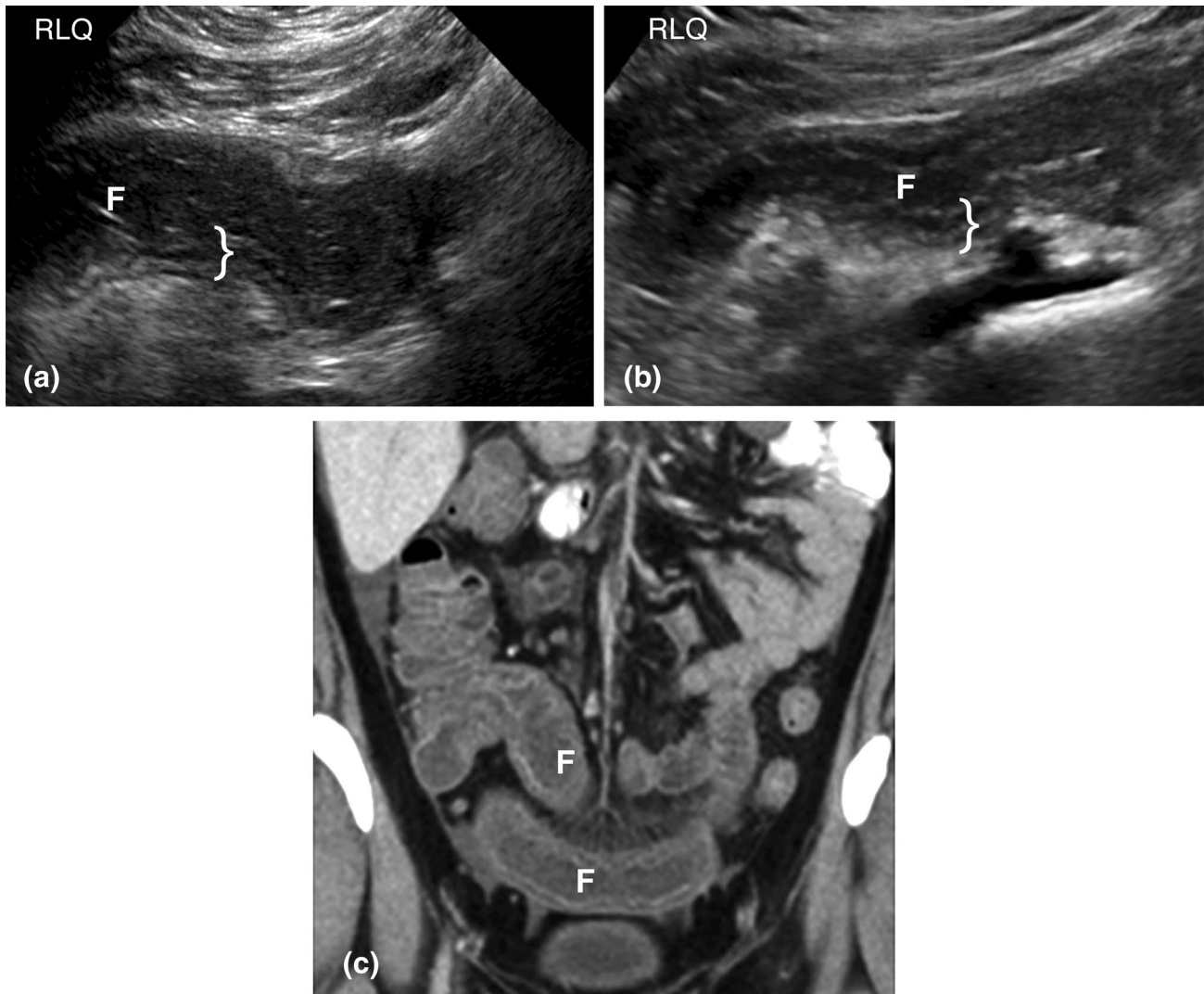


Fig. 5 Infectious enteritis in a 23-year-old-female with 1 day of abdominal pain, nausea/vomiting, and diarrhea. Multiple people in the household were suffering similar symptoms. **a, b** Gray scale longitudinal images of the right lower quadrant demonstrate fluid-filled prominent loops of small bowel (F) with mild thickening of the small bowel wall (bracket). Note preserved striation of the bowel wall with

all layers relatively proportionally thickened. Affected bowel loops were not compressible (not shown). **c** Coronal contrast-enhanced CT image shows multiple mildly dilated fluid-filled loops of small bowel (F) in the deep pelvis and right lower quadrant with mesenteric hyperemia and inflammation. Appendix was normal (not shown)

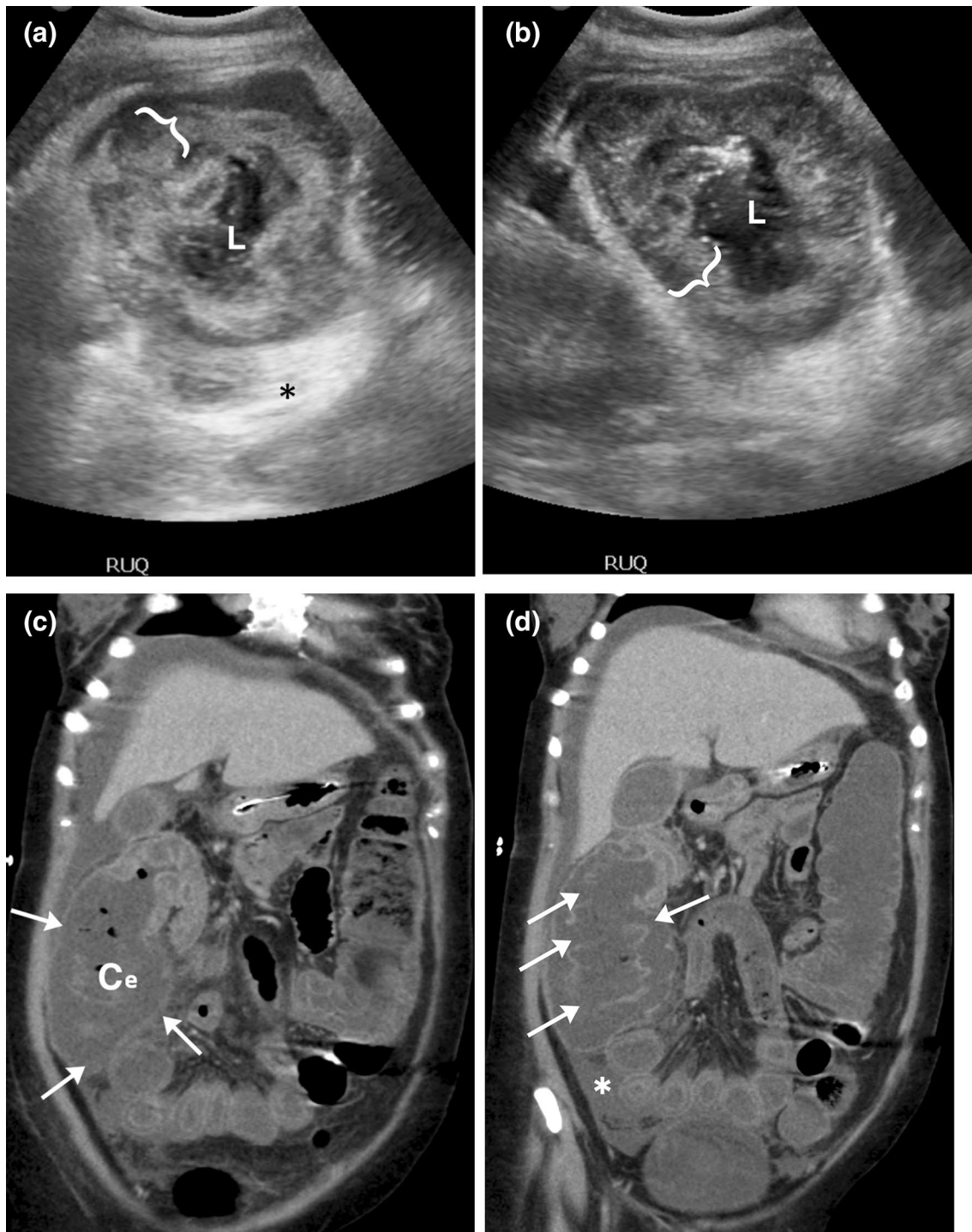


Fig. 6 Typhlitis in a 30-year-old female with history of relapsed AML presented to the emergency department with severe abdominal pain. **a** Gray scale transverse and **B** longitudinal ultrasound images demonstrate markedly thickened heterogeneous echogenicity cecal wall (brackets). The lumen of the cecum is dilated and contains complex debris and air (L). Note marked echogenicity of the inflamed

mesocolonic fat (asterisk). **c, d** Contrast-enhanced CT images of the abdomen and pelvis reveal focal marked thickening of the cecum (Ce) (arrows in **c**) with areas of no enhancement of the cecal mucosa (arrows in **d**). Small amount of free fluid is seen in the right paracolic gutter (asterisk in **d**)

Table 1 Geographic distribution of various infectious agents responsible for enterocolitis, differential diagnoses, and main differentiating features

GI tract location	Pathogenic organisms	Characteristic imaging findings	Differential Dx
Stomach	<i>Helicobacter pylori</i> —most common CMV, candida albicans, histoplasmosis	Thick gastric folds, specifically affecting the mucosal and submucosal layers Increased mucosal layer-to-antral wall thickness ratio Rarely may see hyperemia and extra-gastric fat inflammation	Eosinophilic gastritis
Duodenum	<i>Helicobacter pylori</i> —most common, Giardiasis, Tropical Sprue	Wall thickening and luminal dilatation, non-specific ± peri-duodenal fluid Inflammation of the surrounding fat	Reactive bowel wall thickening due to Pancreatitis
The proximal small bowel	Giardia, Strongyloides species Mycobacterium avium-intracellulare (MAI)	Wall thickening > 4 mm Enlargement of mesenteric lymph nodes Early disease: loss of demarcation between mucosa and submucosa—echogenic Fluid-filled loops of bowel ± peritoneal free fluid, Inflammatory changes in the peri-enteric fat → increased echogenicity Mild disease: increased bowel motility Severe disease: decreased bowel motility	Crohn's disease
The distal small bowel	Salmonella, Shigella, Campylobacter, Anisakis, Yersinia,	The same features as in the proximal small bowel	Crohn's disease
The distal ileum	Typhlitis, Tuberculosis (TB), Amebiasis	In typhlitis: Marked thickening of the wall of the distal ileum and sometimes cecum (6–18 mm) Severe mesenteric inflammation Mesenteric vascular hyperemia Loss of bowel wall striation Transmural pattern of inflammation. ± floating echogenic septa representing sloughed necrotic mucosa. Free abdominal fluid. In TB: Skip lesions and multiple strictures, ascites	Sarcoidosis, Non-specific IBD Drug-induced ileitis (NSAIDs, gold therapy, oral contraceptives, ergotamine, digoxin, diuretics, antihypertensives, potassium chloride), radiation ileitis, granulomatous inflammations (arteritis, spondyloarthropathies, actinomycosis)
Ascending colon	Yersinia, Salmonella, Entamoeba Histolytica	Mild colitis—bowel wall thickening 3–6 mm Moderate colitis—bowel wall thickening 6–9 mm Severe colitis—bowel wall thickening > 9 mm Loss of bowel wall stratification Increased echogenicity of the peri-colonic fat Lymphadenopathy	Crohn's disease
Descending colon	Shigella, Schistosomiasis	The same findings as in the ascending colon	Immunotherapy-induced colitis Radiation-induced colitis Chemotherapy-induced colitis Ischemic colitis
Sigmoid colon	Herpes simplex virus, Gonorrhea, Chlamydia	The same findings as in the ascending colon	Immunotherapy-induced colitis
Pancolitis	Clostridium Difficile, Herpes complex virus, Gonorrhea, Chlamydia	Ascites is commonly present fluid-fluid level within the bowel seen in the setting of neutropenia or extensive broad spectrum antibiotics usage	Crohn's disease

ileocolic intussusception with the diverticulum acting as a lead point. Volvulus and torsion of the diverticulum are additional potential complications [34]. Due to presence of

heterotopic gastric or pancreatic mucosa in up to 60% of the diverticula, they can also be affected by hemorrhage from

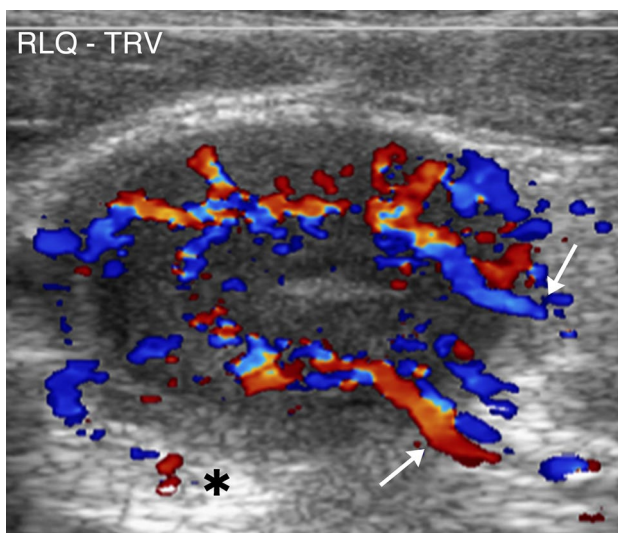


Fig. 7 Active Crohn's disease in a 20-year-old male with abdominal pain and 8-year history of Crohn's Disease. Color Doppler image of the right lower quadrant in transverse plane demonstrates marked bowel wall thickening, (measuring > 10 mm), loss of normal mucosal striation, increased echogenicity of peri-enteric fat (asterisk), and hyperemia of the bowel wall with vessels extending to the mesentery, aka comb sign (white arrows)

peptic ulceration. Foreign bodies, enteroliths, and fecaliths have been reported in the diverticular lumen.

On sonography, an uncomplicated Meckel's diverticulum is seen as a fluid-filled blind-ending tubular or rounded structure in the right lower quadrant, resembling a thick-walled loop of bowel with typical "gut signature," demonstrating clear communication with the small intestinal lumen. The echogenic mucosa can be readily recognized on gray scale ultrasound. When inflamed, the walls of the diverticulum

may become very thick. Marked inflammation of the surrounding fat can be detected as areas of increased echogenicity. Reactive decreased motility of the bowel adjacent to an inflamed diverticulum can also be noted. Echogenic foci in the diverticular lumen may represent enteroliths, fecaliths, peripheral calcifications, inflammatory debris, or foci of air. Color Doppler ultrasound may demonstrate hyperemia within the walls of the diverticulum (Fig. 9) [33].

Sonographic findings of an inflamed Meckel's diverticulum may mimic those of acute appendicitis or intestinal duplication cyst. Duplication cysts usually have smooth internal walls, in contrast to the irregular walls of a Meckel's diverticulum. An inflamed appendix is generally non-compressible and can be easily traced back to its origin from the cecum.

Complicated cases of Meckel's diverticulum require further cross-sectional imaging and are usually managed surgically.

Large bowel colon

Infectious colitis

Infectious colitis refers to inflammation of the colon due to various pathogens including bacterial, viral, fungal, or parasitic organisms, with some agents having preferential predilection to specific segments of the large bowel (Table 1) [35]. For example, *Yersinia*, *Salmonella*, and *Entamoeba Histolytica* commonly affect the ascending portion of the colon, whereas *Shigella* and schistosomiasis more frequently involve the descending colon. Herpes simplex virus, gonorrhea, and chlamydia usually affect sigmoid colon and rectum, while pancolitis is frequently seen with *C. diff* infection

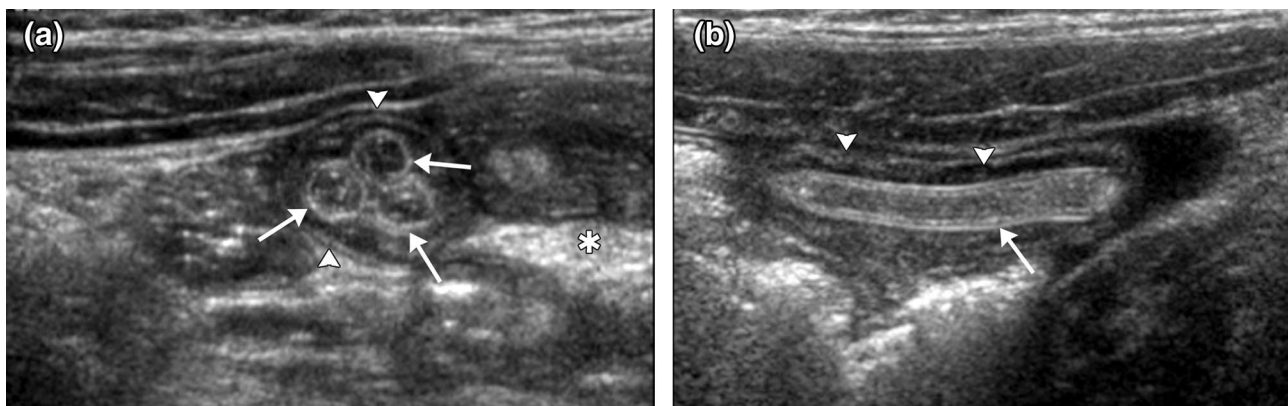


Fig. 8 Ascariasis in a 5-year-old male with abdominal pain and headaches, recently adopted from Africa. **a** Gray scale images of the mid abdomen in transverse and **B**. longitudinal planes demonstrate multiple tubular (arrow in **b**) or round-shaped (arrows in **a**) structures in the mildly distended loops of small bowel (arrowheads) compatible

with ascariasis. The tubular structures show outer and inner echogenic parallel lines compatible with enteric tube of the worm. Note echogenic mesentery compatible with inflammatory process (asterisk in **a**)

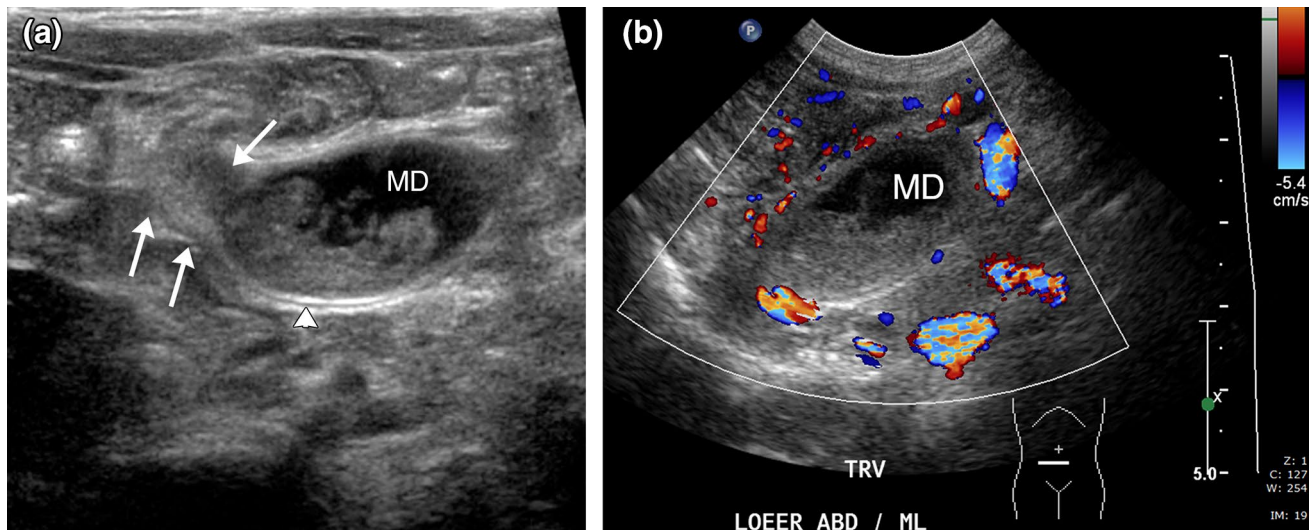


Fig. 9 Infected hemorrhagic Meckel's diverticulum in a 12-month-old female with decreased oral intake, emesis, fever, and abdominal pain. **a** Gray scale and **b** Color Doppler images of the mid/lower abdomen obtained in transverse plane demonstrate a blind-ending loop of bowel (MD) that is connected to the remaining bowel (arrows

in **a**). “Gut signature” is noted, differentiating it from a duplication cyst (arrowhead in **a**). Significant hyperemia of the surrounding soft tissues is present on color Doppler (**b**). Complex fluid is seen within the lumen of the Meckel's diverticulum compatible with hemorrhagic necrosis (confirmed on pathology evaluation)

[35, 36]. The symptoms of colitis are non-specific and commonly include fever, crampy abdominal pain, watery or bloody diarrhea, nausea, and vomiting. The diagnosis is generally made on the basis of stool analysis or colonoscopic evaluation and biopsy. There is considerable overlap in imaging findings between various types of infectious colitis, with all commonly presenting with wall thickening, pericolic fat stranding, and free intraperitoneal/peri-colonic free fluid [37, 38].

On ultrasound, abnormal bowel wall thickness of greater than 3–4 mm is usually considered abnormal; however, no established cut-off value exists at this time. Note should be made that most of the investigations have been performed to identify cut-off values for bowel wall thickness in the diagnosis of inflammatory bowel disease, specifically Crohn's disease, with latest meta-analysis showing that a cut-off value of 3mm had a sensitivity and specificity of 89% and 96%, respectively, while other cut-off values (4mm or more) yielded a sensitivity of 87% and a specificity of 98% [15]. Although bowel wall stratification is usually preserved in infectious colitis, in some cases loss of bowel wall stratification may be observed, and has been described as visualization of less than 5 bowel wall layers or lack of distinction between any layers [17]. Increased echogenicity of the pericolic fat surrounding the affected bowel loops represents inflammatory changes. In any type of colitis, prominent lymph nodes may be detected in the mesentery, porta hepatis, and along the ileocecal lymph node chains [16]. Depending on the causative agent, color Doppler ultrasound may depict mucosal or transmural bowel wall hyperemia, which

is characterized by increased vascularity of the wall of the affected bowel loop compared to a normal segment (Fig. 10, 11) [39].

The normal colon is often easily compressible. In infectious colitis, compressibility of the colon diminishes, and in severe cases becomes fully non-compressible. Infectious colitis should be differentiated from other bowel pathologies with overlapping imaging findings. These include appendicitis, intussusception, Crohn's disease, ulcerative colitis, and immune therapy, chemo or radiation-induced colitis. Knowledge of the clinical history is paramount in differentiating these entities. A few more specific findings such as presence of ascites and fluid-fluid levels within the bowel are commonly seen with diffuse colitis/pancolitis associated with infectious processes such as *Clostridium difficile* (*C. Diff*) or in chemotherapy-induced colitis, which aid in differentiating these entities from Crohn's disease where ascites is rarely seen. In ischemic colitis, the characteristic clinical presentation includes bloody bowel movements, abdominal pain, and leukocytosis, as well as involvement of bowel watershed areas with sparing of the rectum [40]. A definitive diagnosis of infectious colitis usually requires stool cultures, serology titers, or tests for specific toxins [41]. Colitis is usually managed conservatively with supportive therapy and pathogen-specific medications (Table 1).

Typhlitis

Neutropenic enterocolitis is a severe complication of intensive chemotherapy, which is most commonly associated

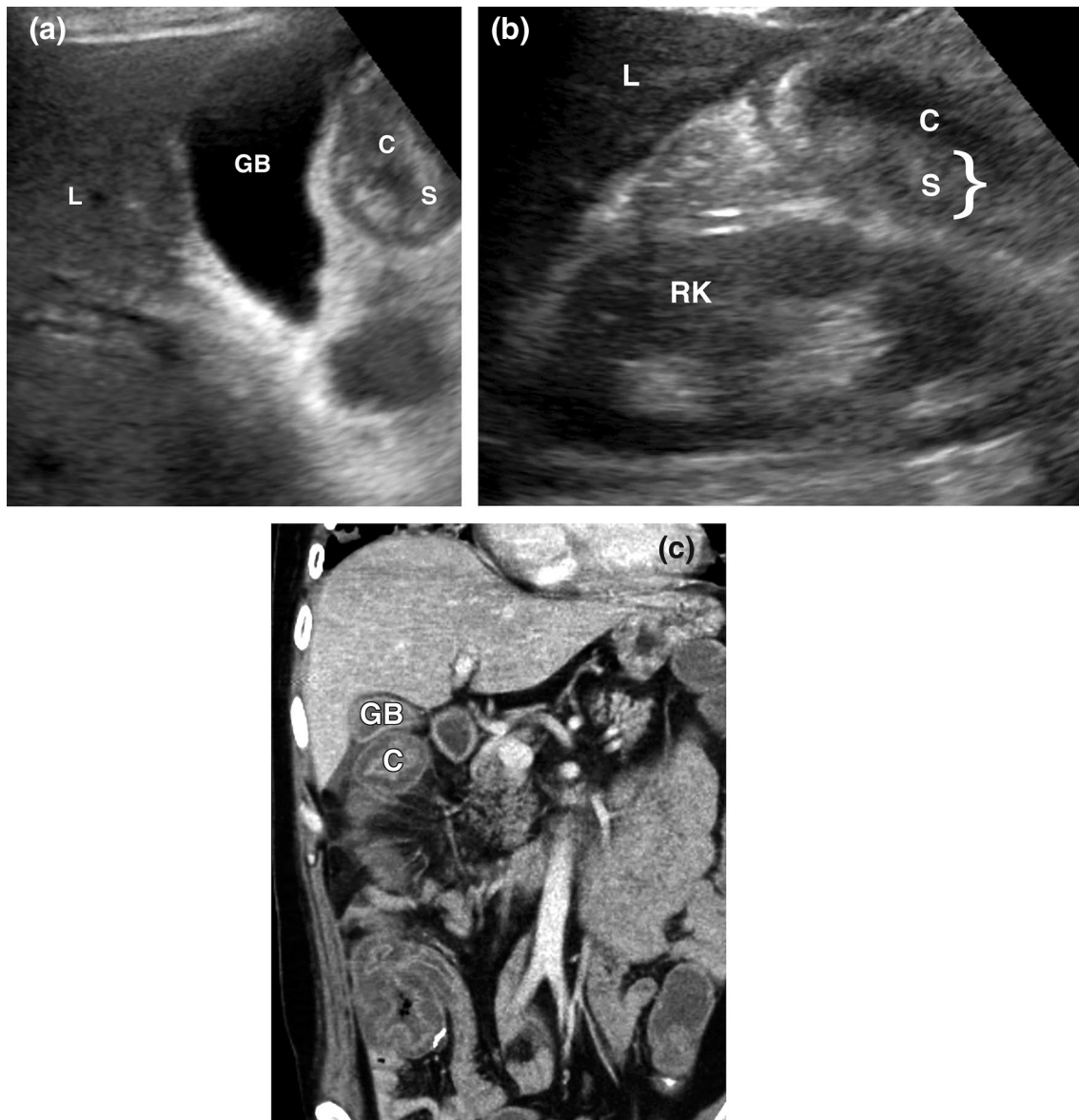


Fig. 10 Colitis of ascending colon in a 32-year-old male with right upper quadrant pain for 2 days. **a, b** Gray scale images of the RUQ obtained in transverse and longitudinal planes show partially imaged loops of transverse and ascending colon (**c**) that demonstrate marked thickening of the colonic wall, specifically submucosal layer (**S** and

bracket). **L** liver, **GB** gallbladder, **RK** right kidney. **c** Coronal CECT image shows marked edema and thickening of the hepatic flexure adjacent to the gallbladder (**GB**) that corresponds to the ultrasound. Left colon is unremarkable

with the treatment regimens of acute leukemia. This type of colitis also clinically presents with fever, abdominal pain, and diarrhea. The symptoms are due to mucosal injury from the chemotherapeutic agents in addition to secondary bacterial superinfection, leading to necrosis of the mucosa of the bowel wall. The degree of the disease is variable ranging from mucosal inflammation to transmural necrosis [42]. Typhlitis represents a localized form of neutropenic enterocolitis in immunocompromised patients, with inflammation of predominately the cecum and often terminal ileum as well as the right colon [35, 43]. Commonly, typhlitis is associated

with gram positive gut flora, including *C. diff*. There is a high mortality rate (up to 50%) associated with this disease owing to high rates of necrosis, rupture, and peritonitis. US findings of neutropenic enterocolitis/typhlitis include diffuse or localized small and/or large bowel wall thickening that is commonly greater than 10 mm (ranging from 6 to 18 mm), with either marked thickening of the mucosa and submucosa or loss of bowel wall striation and a transmural pattern of inflammation (Fig. 6, Video 5) [14]. The lumen of the bowel may contain floating echogenic septa representing sloughed necrotic mucosa. Other common findings include

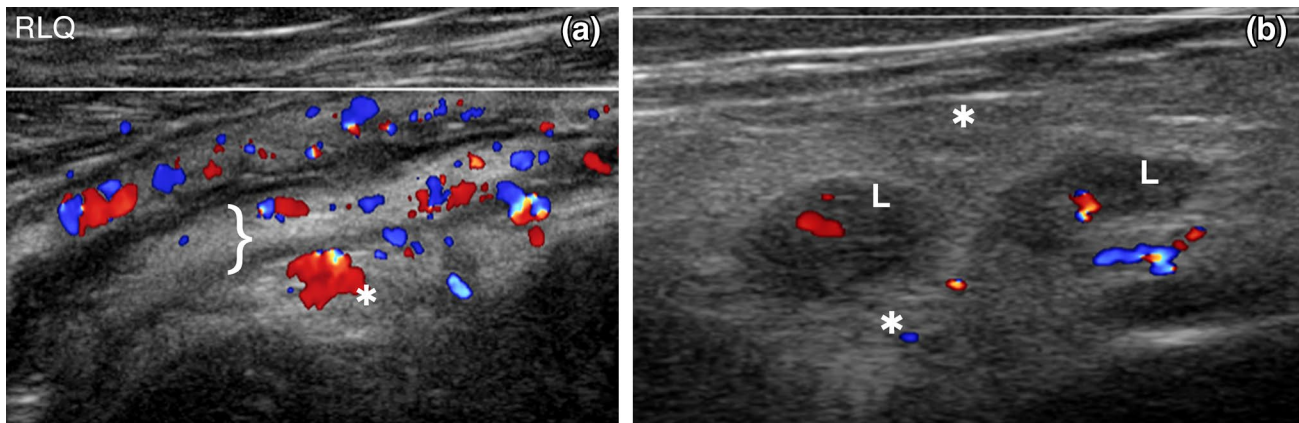


Fig. 11 Infectious colitis in a 35-year-old female with 2 days of diarrhea and RLQ abdominal pain who recently returned from vacation in the Dominican Republic. She had prior negative work up for IBD. **a** Longitudinal and **b** transverse color Doppler images of the right lower quadrant demonstrate marked thickening of the echogenic

submucosal layer of the ascending colon wall (bracket in **a**). Bowel wall hyperemia is appreciated on Color Doppler imaging. Note echogenic mesocolic fat (asterisk in **a** and **b**) and multiple prominent ileocolic reactive lymph nodes (L in **b**). Note loss of haustrations (**a**). Decreased peristalsis was observed on real-time imaging

hypervascularity of the thickened bowel wall and free abdominal fluid. The mortality rate is significantly higher in patients with bowel wall thickening > 10 mm. In patients who are able to recover from this complication, a significant reduction in bowel wall thickening will be observed. Therefore, US can be used as a surveillance imaging tool for continuous assessment and follow-up until patient recovery [44, 45] (Table 1). CT can be employed for better identification of extend of the disease and its severity and when associated complications are suspected.

Appendicitis

Acute appendicitis is the most common cause of acute abdominal pain with approximately 280,000 appendectomies performed annually in the United States [46]. The pathogenesis of appendicitis is multifactorial with a combination of ischemic mucosal damage and bacterial overgrowth, as well as some degree of luminal obstruction. *Escherichia Coli* (*E. coli*) is the most common causative bacterium in appendicitis [47]. Delay in diagnosis can lead to significant morbidity from appendiceal rupture, abscess formation, peritonitis, and septic shock, and therefore, rapid and accurate diagnosis is required. Clinical symptoms are frequently non-specific and may overlap with other etiologies of abdominal pain [48, 49]. Advanced imaging with either CT or US is commonly utilized for diagnosis of appendicitis. Due to potential deleterious effects of radiation, ultrasound should be considered as the first-line imaging modality for its diagnosis [50]. In the hands of well-trained operators and with adequate equipment, the sensitivity and specificity of ultrasound for detecting acute appendicitis are similar to CT and MRI with accuracy of ultrasound reaching values above 90% [51].

Therefore, the use of ultrasound imaging should be routine in every patient with suspected appendicitis. Complimentary CT or MRI should be limited to inconclusive findings and difficult conditions, e.g., in very obese patients or in pregnant women (MRI) [51].

Several sonographic imaging techniques are employed for evaluation of appendicitis, including graded compression with a linear transducer, resulting in decreased depth of the abdominal cavity by gradual application of anterior compression in the right lower quadrant. This technique results in displacement and compression of the bowel, minimizing artifacts produced by bowel gas, allowing better visualization of the inflamed and non-compressible appendix [18]. Additional techniques include posterior manual compression and left lateral oblique decubitus positioning that are most helpful for identification of a retrocecal appendix, while using a low-frequency convex transducer [52]. In a female patient, endovaginal sonography is helpful to localize an appendix projecting deep in the pelvis. The quality of the exam significantly increases when a combination of these techniques is utilized, especially if conventional graded compression fails to identify the appendix. Although visualization of a normal appendix as a compressible, tubular, blind-ending structure filled with fluid, gas, or feces can reliably exclude acute appendicitis, detection of normal appendix may be difficult to achieve, thus resulting in inconclusive findings. One of the most common pitfalls in the diagnosis of acute appendicitis is mistaking the terminal ileum for the appendix. Identification of the cecum and the appendiceal origin are paramount in confirmation of the appendiceal location and correct interpretation (Video 6) [53]. Anatomical variations of the location of the appendix require a

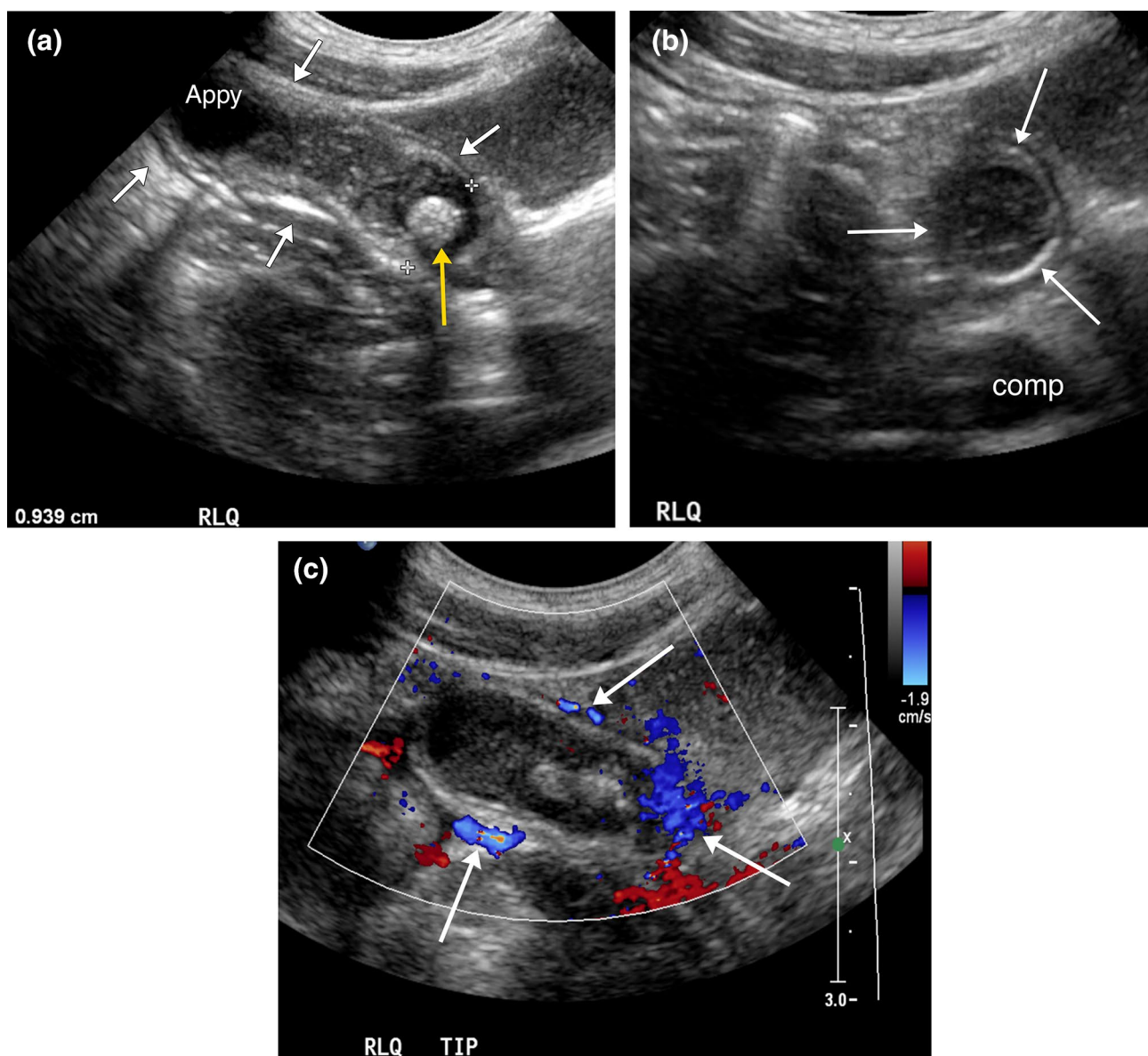


Fig. 12 Non-complicated appendicitis in a 12-year-old male who presented to the emergency department with periumbilical abdominal pain lasting for 9 h. **a** Longitudinal and **b** transverse gray scale ultrasound images demonstrate dilated appendix (appy, arrows in **a**) (measuring 9.4 mm) filled with heterogeneous complex material

and a fecalith in the distal appendix (yellow arrow). The appendix (arrows) was not compressible on manual compression (comp in **b**). **c** Color Doppler image in longitudinal plane shows marked hyperemia of the appendiceal wall (arrows). No free fluid, adjacent fluid collections, or lymphadenopathy was present on real-time images

systematic examination technique for correct identification of the appendix.

Characteristic sonographic findings supportive of appendicitis include a dilated non-compressible rounded or tubular appendix more than 6 mm in diameter, presence of an appendicolith seen as an echogenic shadowing focus especially at the base of the appendix, echogenic prominent peri-cecal

and peri-appendiceal fat, peri-appendiceal fluid collection, and increased flow in the appendiceal wall on color Doppler US (Fig. 12). Thickening of the lamina propria and the submucosal layer can be seen with lymphoid hyperplasia, in which case clinical management may be medical rather than surgical [54]. The appendiceal diameter should be measured during compression in order to minimize false

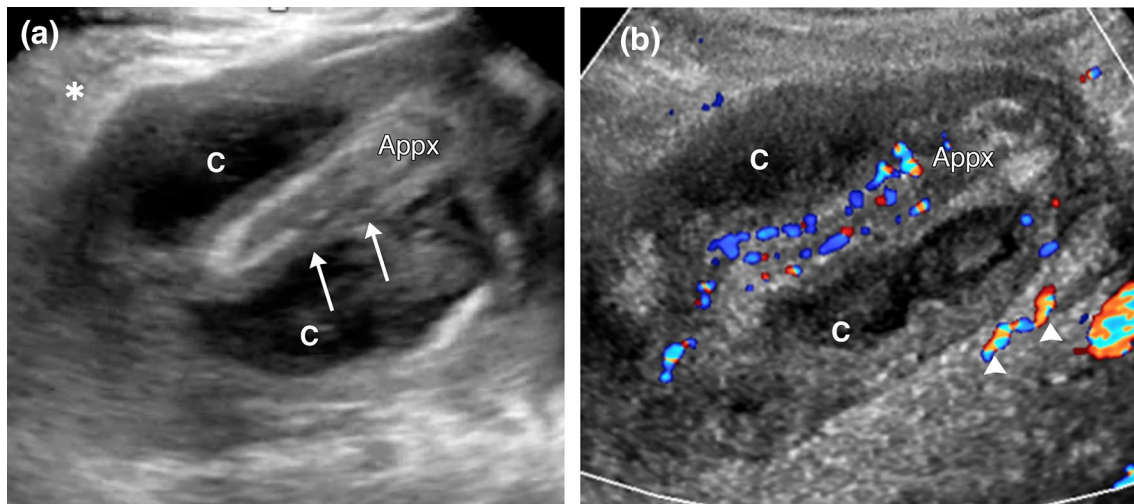


Fig. 13 Acute appendicitis complicated by perforation and abscess formation in a 7-year-old male who presented with 2 weeks of right lower quadrant abdominal pain. **a** Gray scale and **b** color Doppler images in longitudinal plane obtained in the right lower quadrant show collapsed inflamed appendix (Appx) with thick hyperemic

wall. Note focal discontinuity of the mucosal layer in the body of the appendix (arrows in **a**) compatible with perforation. Complex avascular fluid collection (C) surrounds the perforated appendix compatible with an abscess. Surrounding mesocolonic fat is echogenic (asterisk in **a**) and shows hyperemia around the abscess (arrowheads in **b**)

positive results. In appendiceal perforation, the diameter of the appendix may be normal due to release of pus into the peri-appendiceal space with a simple or complex fluid collection seen adjacent to the appendix (Fig. 13 Video 7). Findings of irregularity, disruption, or discontinuity of the appendiceal mucosa are suggestive of wall necrosis as seen in gangrenous appendicitis, which may also be seen in association with perforation (Fig. 14). When the appendix is not visualized in a symptomatic patient, the results are considered equivocal and further imaging with CT is advised [50].

Clinically, the most common differential diagnosis is mesenteric adenitis, in which a normal appendix and enlarged mesenteric lymph nodes are observed. Other differential considerations for acute right lower quadrant pain include inflammatory bowel disease which may also affect the appendix, pelvic inflammatory disease (PID), right-sided diverticulitis, Meckel's diverticulitis, acute epiploic appendagitis, and omental infarction.

Mesenteric adenitis

Mesenteric adenitis is a self-limiting inflammatory process that affects the mesenteric lymph nodes in the right lower quadrant [55]. Two distinct groups are recognized: primary and secondary mesenteric adenitis. Based on imaging

findings, primary mesenteric adenitis is defined as right-sided mesenteric lymphadenopathy without an identifiable acute inflammatory process or with only mild (<5 mm) wall thickening of the terminal ileum [55]. The cause of primary mesenteric adenitis in most cases is related to underlying infectious terminal ileitis, involving infectious agents including *Yersinia enterocolitica*, *Helicobacter jejuni*, *Campylobacter jejuni*, *Salmonella* spp., and *Shigella* spp. [2–8]. Secondary mesenteric adenitis is defined as lymphadenopathy associated with a detectable intra-abdominal inflammatory process. This condition is predominantly seen in children and is thought to be caused by either viral or bacterial infectious spread from the intestinal lymphatic tissues. Clinically, mesenteric adenitis may mimic appendicitis as patients often present with right lower quadrant abdominal pain, fever, and leukocytosis. In the setting of a normal appearing appendix, the primary sonographic finding of mesenteric adenitis is the presence of a cluster of three or more lymph nodes measuring 5 mm or greater each in the mesentery of the right lower quadrant [18, 55]. The lymph nodes can be either elongated or round and are usually adjacent to the cecum or situated along the mesentery. An associated small amount of free fluid as well as hyperemia of the mesentery may also be present. If identified, the appendix will be normal in appearance (Fig. 15) [56].

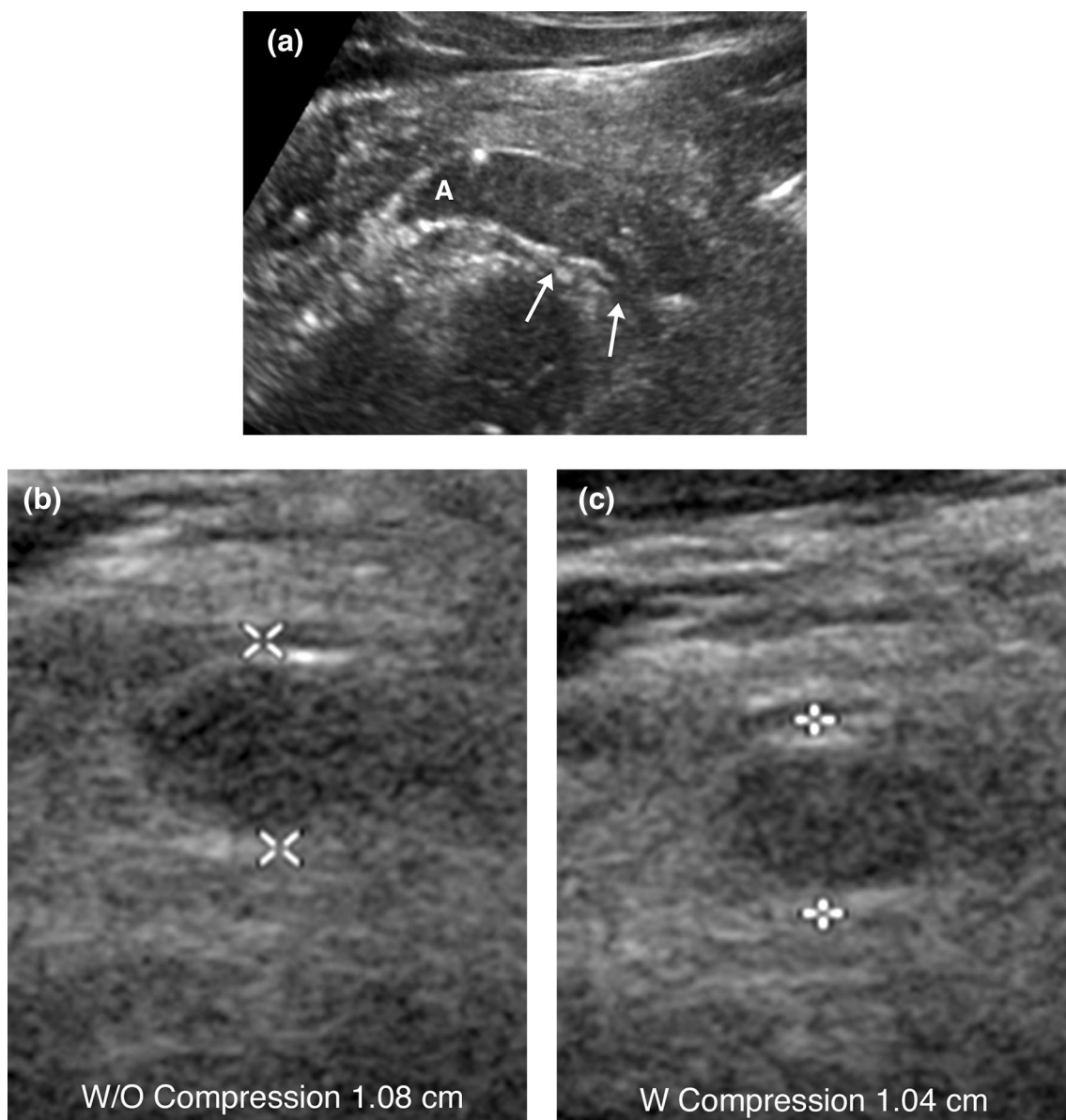


Fig. 14 Acute appendicitis with mucosal necrosis and microperforation in a 14-year-old female with 3-day history of abdominal pain localizing to right lower quadrant with vomiting and fever. **a** Longitudinal gray scale image of the right lower quadrant shows a blind-ending loop of bowel filled with fluid compatible with an inflamed

appendix (**a**). Note mucosal discontinuity of the appendix compatible with focal ulceration and perforation (arrows). **b, c** Longitudinal gray scale image with and without compression show that the appendix lumen is not collapsed with compression due to very viscous material within the appendix minimizing leak in the surrounding soft tissues

Infected mucocele

A mucocele represents dilatation of an obstructed appendiceal lumen that is filled with mucoid material. It is a rare disease found in only 0.3% of all appendectomies [57–59]. Females and those over 50 years of age are more commonly

affected. Mucoceles can range in size. An appendicular diameter of 15 mm or more has been determined as a threshold for diagnosis of mucocele with a sensitivity of 83% and a specificity of 92% [60]. Four histologic types exist: retention mucinous cyst, mucosal hyperplasia, mucinous cystadenoma, and mucinous cystadenocarcinoma [61]. In mucinous

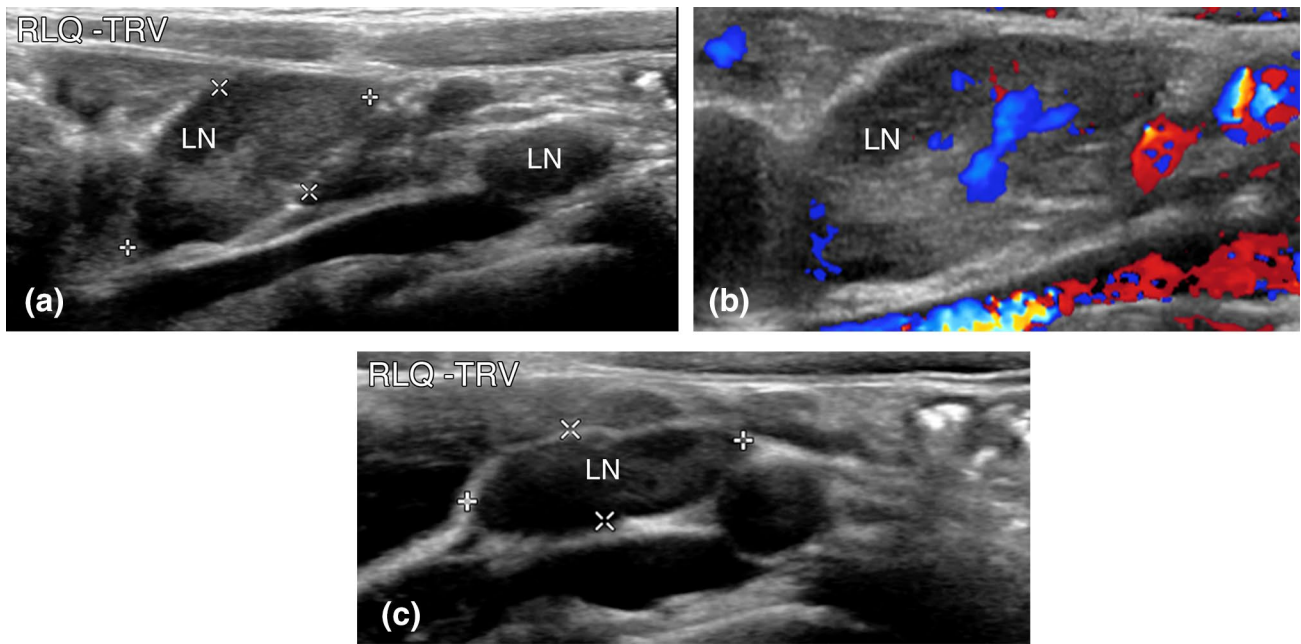


Fig. 15 Mesenteric adenitis in a 5-year-old male who presents to the Emergency room with fever and generalized abdominal pain without any other significant medical history. **a, b** Gray scale images in transverse plane show a cluster of prominent (>5 mm) lymph nodes

(LN) in the right lower quadrant. **c** Color Doppler ultrasound shows blood flow at the level of the hyperechoic lymph node hilum (arrow). Appendix was normal on the same day examination (not shown)

cystadenomas, the luminal dilatation can reach up to 6 cm. Malignant transformation may be seen in up to 36% of cases. Association with more remote neoplasms such as colon or ovarian cancer has also been reported [62].

Clinically, up to 64% of patients present with right lower quadrant abdominal tenderness and a mass on physical palpation [63], while others remain asymptomatic. Symptoms are usually related to superimposed infection or intussusception with an inverted mucocele as a lead point.

On ultrasound, mucoceles appear as a right lower quadrant or midline abdominal fluid collection with internal echoes and a thin wall. The wall of the mucocele may be thickened if it is inflamed or has undergone malignant transformation [64, 65]. The *onion skin sign*, defined as echogenic layers or a layered appearance of the internal contents of the mass, has been described as specific for appendicular mucocele [66]. Polypoid projections representing epithelial proliferation, absent peristalsis, peripheral or rim calcification, or thin septations may also be present. Intraluminal gas bubbles or an air-fluid level within an appendix mucocele

suggests the presence of infection, which needs to be differentiated from an appendicular abscess (Fig. 16, Video 8). Due to the loculated nature of the fluid collection, no appreciable changes in its configuration should be detected with alterations in patient positioning.

Complications associated with spillage of mucinous contents from the mucocele into the peritoneal cavity may result in pseudomyxoma peritonei. Mucoceles are managed surgically.

Colonic diverticulitis

Acute diverticulitis is caused by inflammation of colonic diverticula. In the United States, diverticular disease is the third most common gastrointestinal illness that requires hospitalization, with approximately 312,000 hospital admissions annually and an estimated annual cost of nearly 2.6 billion dollars [67–69]. Deficiency in dietary fiber has long been implicated as a causative factor.

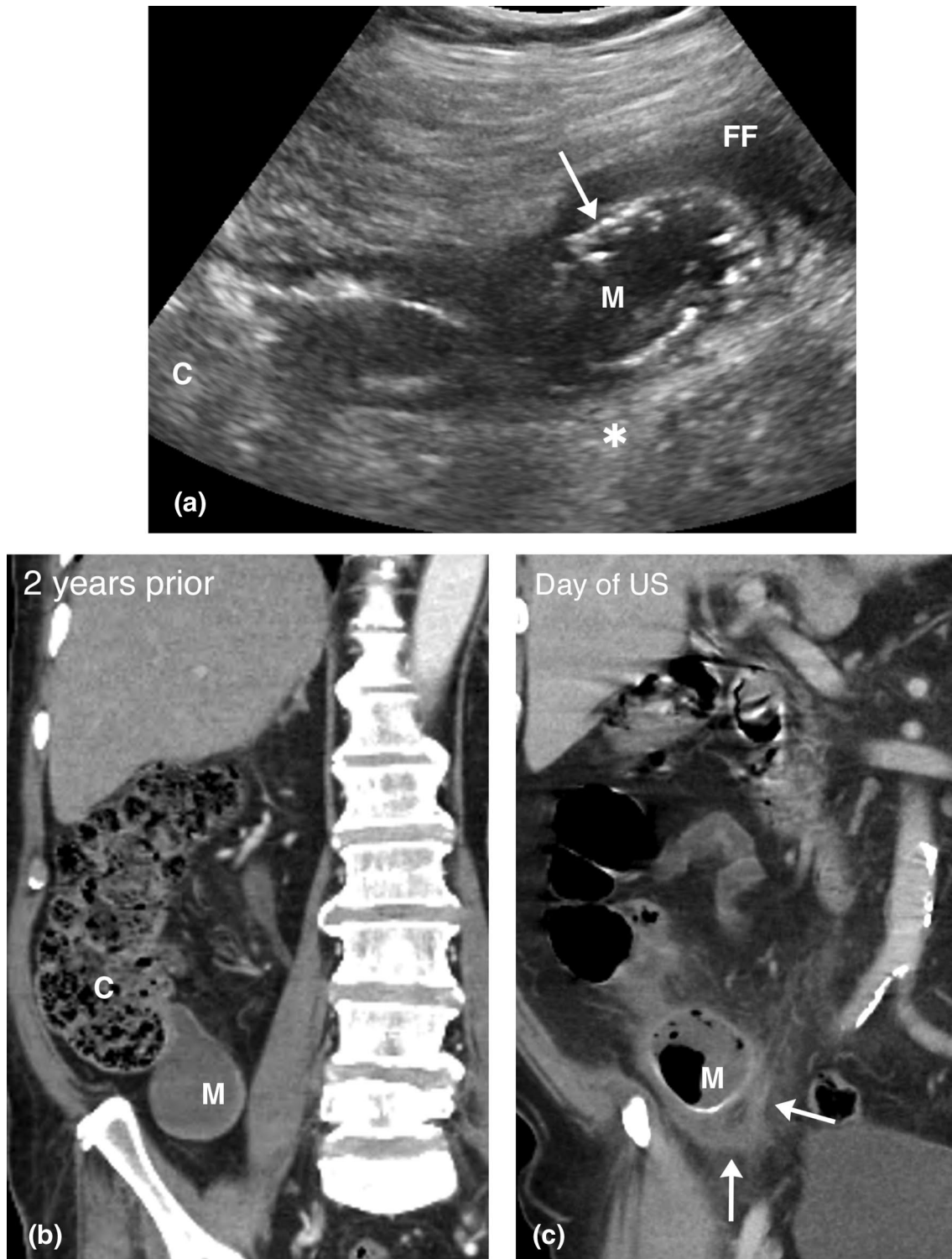


Fig. 16 Infected mucocoele in a 87-year-old male with 2-day history of abdominal pain, fever, and elevated WBC. **a** Gray scale image of the right lower quadrant in longitudinal plane shows an elongated complex air and fluid containing structure (M) with rim calcification (arrow) which is continuous with the cecum (C). No normal appendix is seen. The structure did not show significant hyperemia (not shown), but there was increased echogenicity of the peri-cecal fat

(asterisk) and small amount of free fluid (FF) compatible with inflammation. **b** Coronal CECT image obtained 2 years earlier shows an uncomplicated mucocoele (M). **c** Coronal CECT image obtained the same day as ultrasound shows air within a mucocoele and marked surrounding inflammatory changes (arrows) compatible with an infected appendiceal mucocoele (M)

Traditionally, diverticular disease was thought to primarily affect the elderly; however, there has been increasing incidence of this disease among individuals younger than 40 years of age. It is suggested that over 50% of patients over 60 years of age have diverticulosis and approximately 10–25% of them will develop complications such as diverticulitis with a fivefold increase in the risk of complications such as fistula formation [70].

Acute diverticulitis is associated with a wide spectrum of clinical presentations, ranging from mild inflammation to potentially life-threatening complications such as abscess formation, bowel wall macro-perforation, fistula formation, and hemorrhage. In approximately one-third of patients, the disease is recurrent following an initial episode of uncomplicated diverticulitis. Uncomplicated diverticulitis is usually managed conservatively with a 10-day course of oral antibiotic therapy, with repeated episodes culminating consideration of surgical management [71].

Although CT imaging is the mainstay modality in the evaluation of diverticular disease in the United States, recent literature has emphasized the comparability of ultrasound to CT for establishing this diagnosis [67]. The reported sensitivity and specificity for US is 92% and 90%, and for CT 94% and 99%, respectively, with both modalities demonstrating similar accuracies [72]. The need for CT imaging in the clinical setting of complicated diverticulitis is indisputable; however, if mild or uncomplicated diverticulitis is suspected, then ultrasound should be offered first as an alternate modality. This strategy must be followed by CT whenever US is inconclusive or unreliable [51, 73].

Characteristically, intact diverticula on ultrasound are seen as small outpouchings of colonic mucosa, often filled with air producing substantial posterior shadowing (Fig. 17). Mild forms of uncomplicated diverticulitis can be diagnosed if the following sonographic features are observed: 1. Preservation of the “gut signature” of an unaffected large bowel; 2. Thickening of the bowel wall up to 4–5 mm in the affected colonic segment, resulting in a “pseudokidney sign” or “target” sign” which refers to the hypoechoic thickened colonic wall surrounding a hyperechoic lumen and mucosa; 3. An inflamed diverticulum is usually fluid-filled with minimal or no air remaining in its lumen, and is emanating from the thickened colonic wall, which refers to the “dome sign”; 4. Hyperechoic inflammatory peri-colonic fat (“pericolitis”)

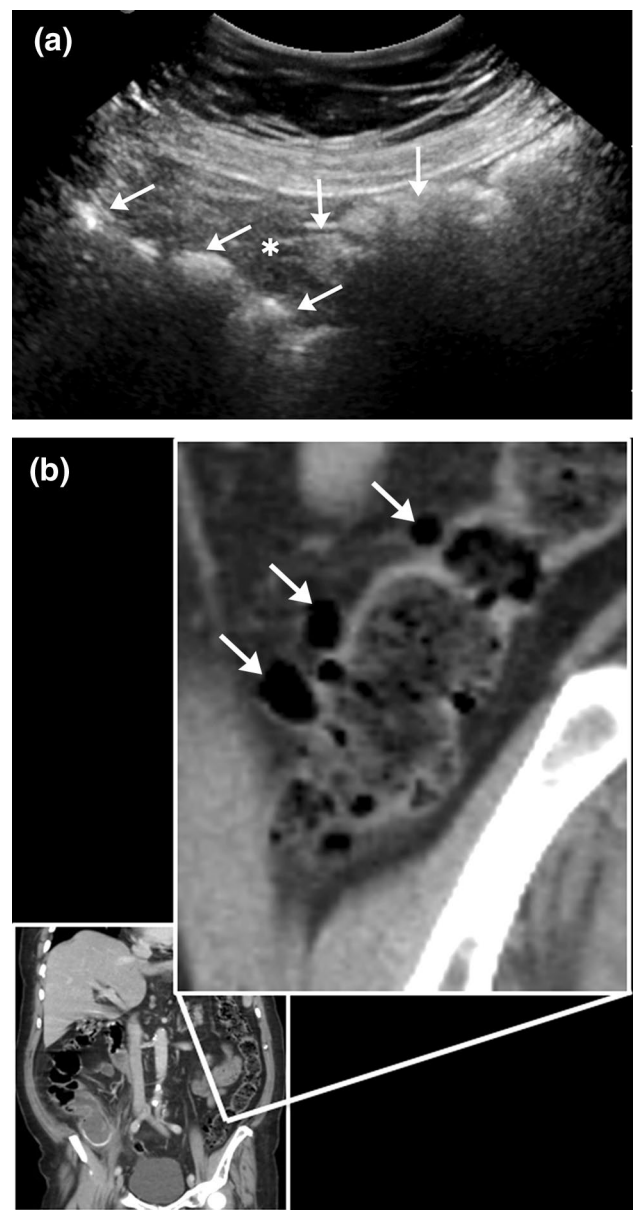


Fig. 17 **a** Colonic diverticulosis in a 60-year-old male presenting for screening of the abdominal aorta. There are innumerable curvilinear echogenic structures in the non-dependent colonic wall with dirty posterior acoustic shadowing compatible with air-filled diverticula of the sigmoid colon (arrows). Note normal hypoechoogenicity of the mesocolonic fat (asterisk). **b** Magnified coronal image of the left colon shows multiple air-filled diverticula without any evidence of diverticulitis (arrows)

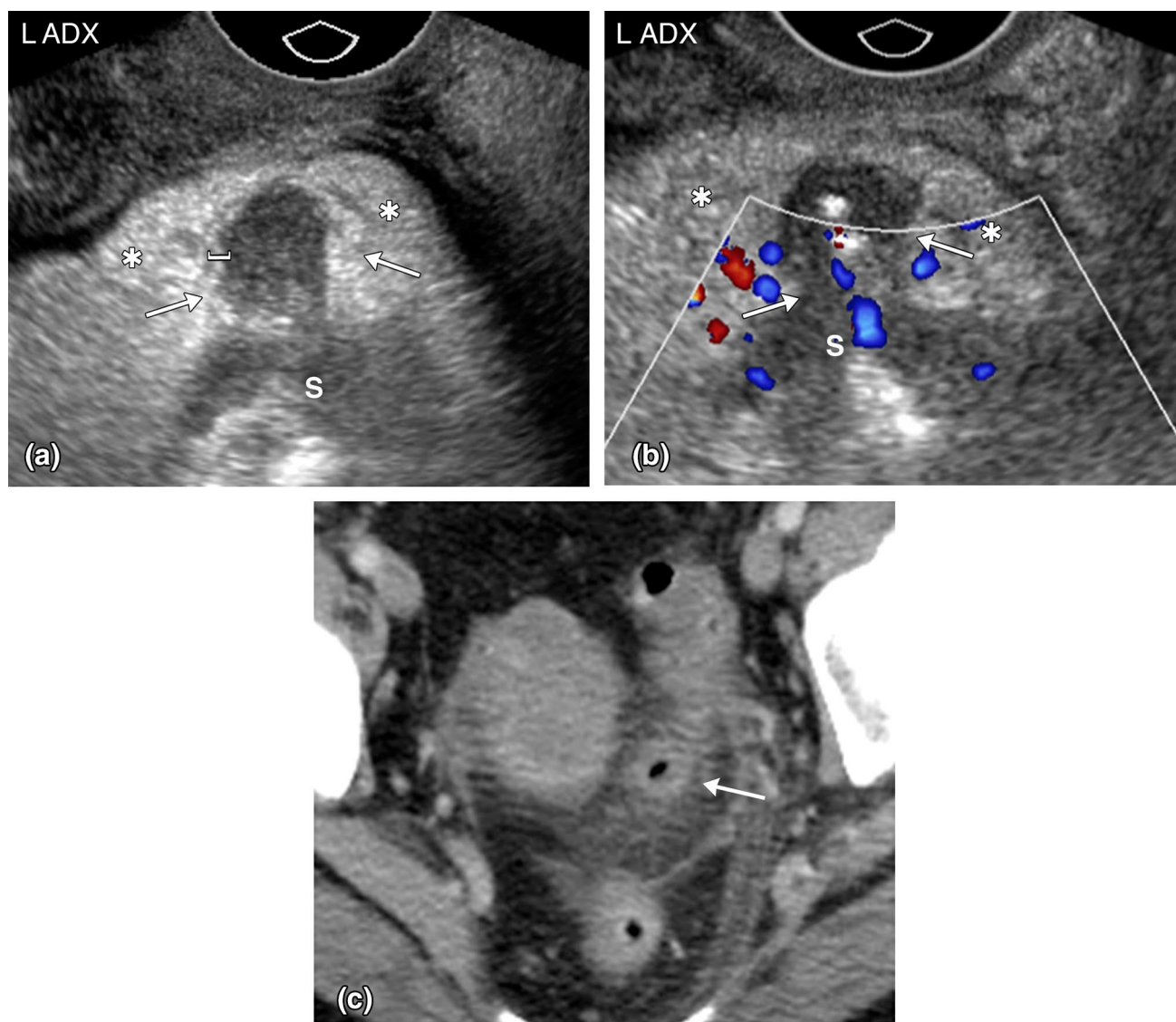


Fig. 18 Acute diverticulitis in a 55-year-old female with severe left lower quadrant abdominal pain. Ovarian torsion was clinically suspected. **a** Gray scale and **b** color Doppler transverse images via transvaginal approach demonstrate inflamed sigmoid diverticulum in the left adnexal region (arrows). The diverticulum shows thick wall (brace in **a**) and lumen almost entirely filled with fluid and only mini-

mal air content (**b**). Marked echogenicity of the adjacent mesocolonic fat (asterisk) compatible with ongoing inflammation. No fluid collections were seen in the surrounding soft tissues. **c** Coronal CECT image shows inflamed diverticulum of the sigmoid colon (arrow) in the left adnexa. *S* sigmoid colon

surrounding the affected diverticulum signifies active inflammation of the surrounding mesocolon; 5. Focal hyperemia of the affected segment of bowel (Fig. 18,) [74, 75]. The point of maximal tenderness is frequently at the site of the affected diverticulum. In complicated cases of acute diverticulitis, where there is bowel wall macro-perforation and abscess formation, fluid collections can be seen in the vicinity of the infected diverticulum (Fig. 19, Video 9). The detection of

peritoneal extraluminal air may be difficult on ultrasound, particularly if the amount of extraluminal air is relatively insignificant or located deep in the pelvis. Decreased motility (hypoperistalsis) of adjacent bowel loops represents an important marker for the presence of an ongoing infectious process. In female patients, a transvaginal approach may be particularly helpful to diagnose sigmoid or deep pelvic diverticulitis. Differential diagnosis may include other forms

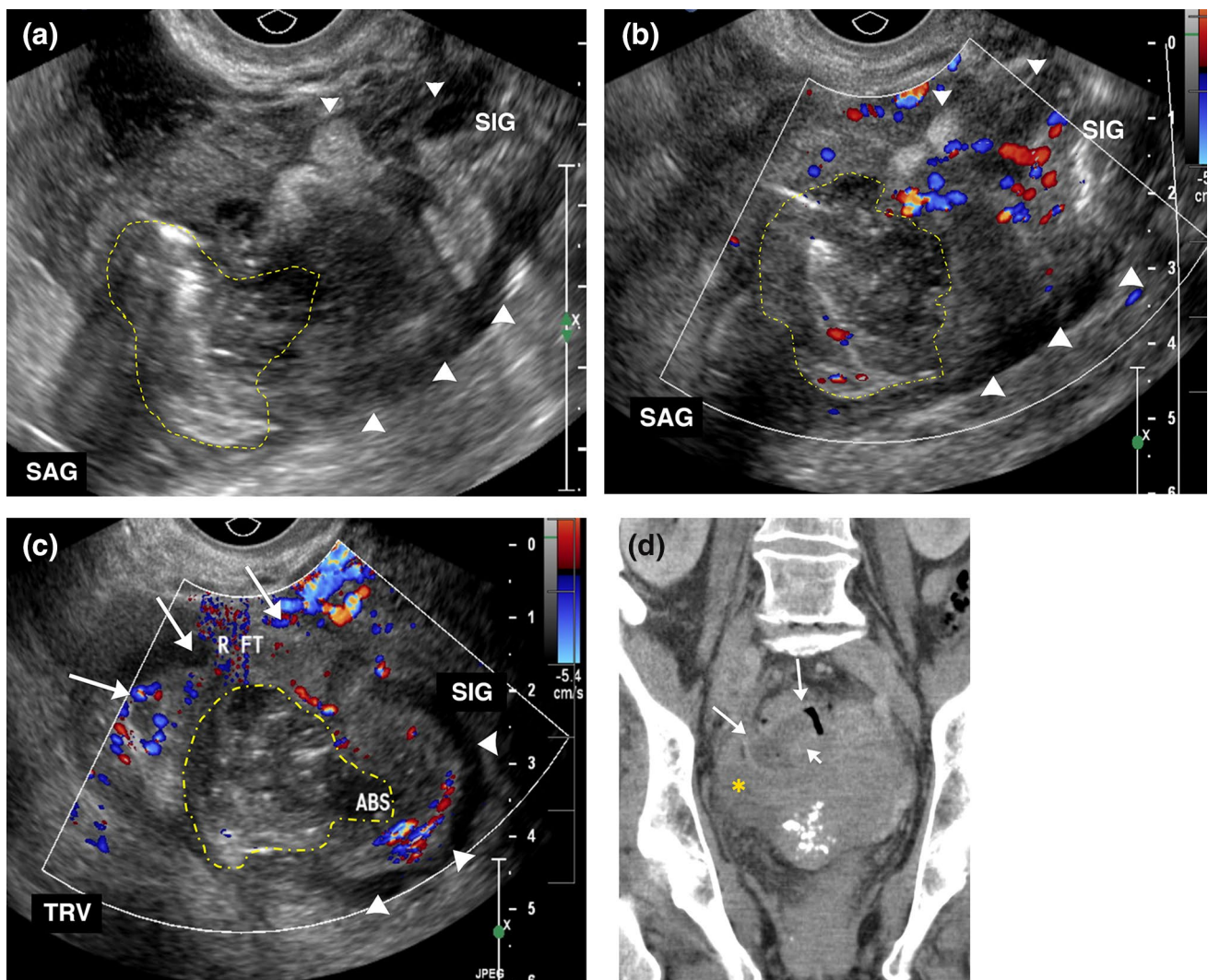


Fig. 19 Acute diverticulitis with perforation and abscess formation in a 74-year-old female who presents to the emergency department with vague right pelvic pain. **a** Gray scale and **b** Color Doppler ultrasound images obtained in sagittal plane and **c** color Doppler ultrasound image in oblique/transverse plane through a transvaginal approach demonstrate a complex fluid collection (yellow dash lines in **a–c**) with echogenic foci within it representing gas. These findings are compatible with an abscess. Peripheral hyperemia is noted around

the collection (**b, c**). The collection extends from the thickened sigmoid bowel wall (SIG, arrowheads). The right fallopian tube (R FT in **c**) is thickened and inflamed. **D**. Coronal CECT of the abdomen and pelvis obtained shows a large complex fluid collection with air in the right adnexa (arrows) extending from the adjacent sigmoid colon (not shown). The right adnexa is very poorly defined with free fluid within (asterisk). The right fallopian tube is very inflamed (not shown)

of infectious or inflammatory colitis; however, in such cases a large portion of the bowel wall will be affected.

Diverticulitis complicated by fistula formation may also be recognized on ultrasound when a hypoechoic fistulous tract filled with air is found originating from the site of infection to communicate with an adjacent visceral structure (Fig. 20, Video 10).

Mimics of diverticulitis

Intraperitoneal focal fat infarction-epiploic appendagitis and omental infarction

Epiploic appendagitis is a rare self-limiting inflammatory/ischemic process involving the appendix epiploica of the



Fig. 20 Colovesical fistula from a complicated diverticulitis in a 67-year-old male who was found in a trash container, brought to the ER, and complained of abdominal pain. **a** Gray scale transverse image through the pelvis showed large amount of air in the urinary bladder (**b**) and an echogenic tract (arrows) from the bladder to the

adjacent bowel loop compatible with colovesical fistula. **b** Axial contrast-enhanced CT through the pelvis shows air in the colovesical fistula tract (arrow) that provides communication between the urinary bladder (**B**) and the sigmoid colon (**S**). Note large amount of air in the urinary bladder (**B**)

colon caused by either a torsion of the epiploic appendage or venous occlusion. This condition affects patients in their 2nd to 5th decades with a predilection for women and obese individuals, presumably due to larger appendages [76]. This entity has a benign self-limiting course.

Clinically, patients present with abdominal pain and possible guarding, symptoms that mimic those of diverticulitis as well as acute appendicitis. The most commonly affected colonic segments are the sigmoid, descending, and ascending colon, with symptoms of abdominal pain usually more focal and on the left.

Ultrasound performed in the area of maximal tenderness may reveal a rounded, non-compressible, hyperechoic mass without internal vascularity, surrounded by a subtle hypoechoic halo (Fig. 21) [77]. The lesion is firmly attached to the anterior abdominal wall, a feature that can easily be visible on sonography during deep patient respiration. The size of the fatty central core ranges from 1.5 cm to over 5 cm. Occasionally a very small hypoechoic center, representing a thrombosed vein, may be identified. The adjacent colonic wall is usually not affected [76, 78]. Depending on its size,

acute appendagitis-related changes may exert local mass effect on adjacent structures. Ascites is not a finding associated with this pathology, which helps differentiate it from other gastrointestinal processes. Serial follow-up ultrasound evaluations can be performed in order to observe response to conservative therapy.

In omental infarction, the hyperechoic mass is greater in size and located medial to the colon, as opposed to epiploic appendagitis where the mass is located laterally or anteriorly. In addition, the previously described hypoechoic halo is absent in omental infarcts. The management and differential considerations are otherwise similar to those of epiploic appendagitis.

Proctitis

Proctitis is a condition in which the lining tissue of the inner rectum becomes inflamed. It is confined to the distal 15 cm of the large bowel. The causes usually are either inflammatory, related to inflammatory bowel disease such as Crohn's

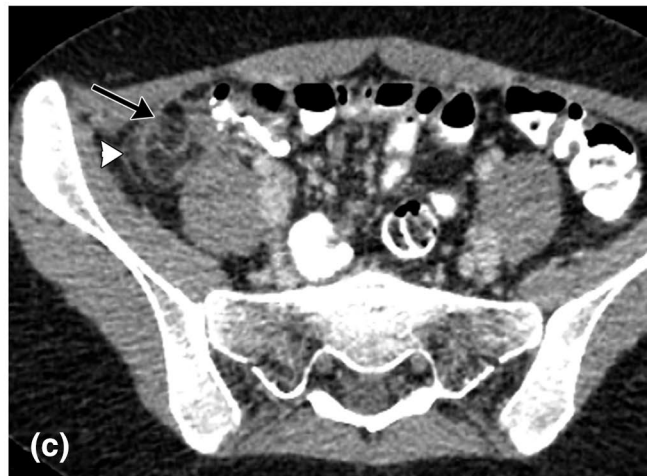
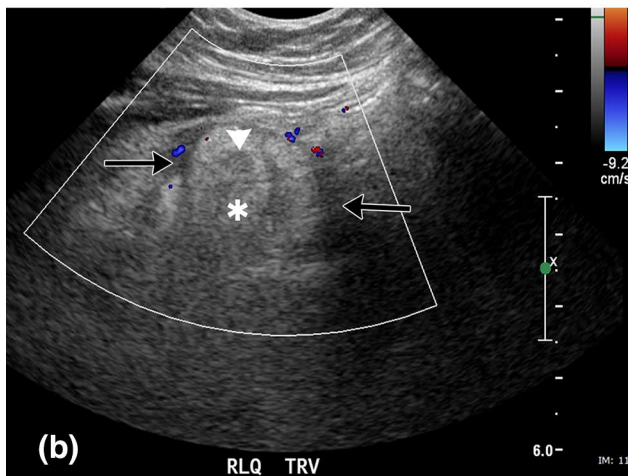
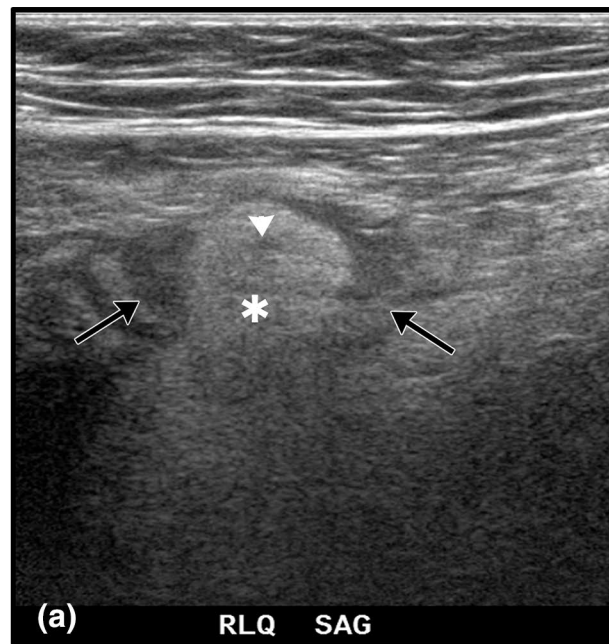


Fig. 21 Epiploic appendagitis in a 18-year-old female with right lower quadrant (RLQ) pain. **a** Gray scale and **b** Color Doppler transverse images of the right lower quadrant demonstrate a focal area of echogenic fat (asterisk) with surrounding hypoechoic halo (black arrows), inseparable from the adjacent bowel. Central hypoechoic-ity represents venous congestion (arrow head in **a** and **b**). Patient had

point tenderness at this level during real-time scanning. **c** Contrast-enhanced CT abdomen and pelvis in axial plane shows an area of hypodensity with hyperdense rim on the anti-mesenteric side of the right colon (black arrows). Note inflammatory changes in the surrounding soft tissues (arrow head)

disease or Ulcerative colitis, trauma related, or infectious in etiology. Among infectious causes Salmonella, Shigella, C. Diff, and sexually transmitted infectious (gonorrhea, chlamydia, herpesvirus) are the most common causative agents [79]. Symptoms of proctitis include tenesmus, which

refers to frequent urge to have a bowel movement, pain in the rectum, anus, and abdominal region, rectal bleeding (hematochezia), development of ulcers, passing of mucus or discharge from the rectum, very loose stools, and watery diarrhea. Transperineal, and in female patients transvaginal

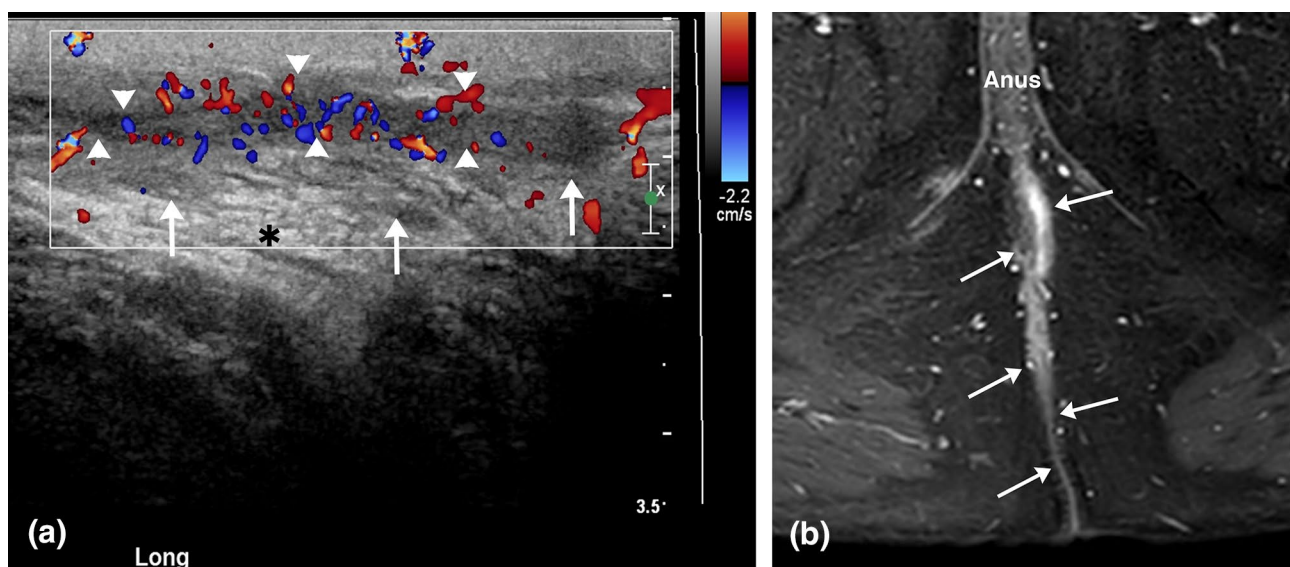


Fig. 22 Perianal fistula in a 43-year-old male with tenderness and erythema of the perineum. **a** Longitudinal color Doppler image of the left perineum obtained via perineal approach demonstrates a hypoechoic complex tubular structure (arrow heads) representing a fistula track. The track is extending from the subcutaneous tissues of the perineum to the anal canal (not shown). Note marked stranding

along the track (arrows), and substantial hyperemia of the fistula wall (arrows). Inflammation of the surrounding fat is also noted (asterisk). **b** T2* MRI image obtained in axial plane demonstrates a hyperintense fistulous track extending to the anus (Anus) with trace amount of fluid within the track (arrows)

ultrasound, can be utilized for evaluation of suspected proctitis. On ultrasound, proctitis may be diagnosed on the basis of diffuse rectal wall thickening, edema, and hyperemia. Other segments of the bowel are not involved [80].

Perianal fistula and abscess

Perianal abscess and fistula are thought to arise from infection in small intersphincteric anal glands [81]. In those with no history of Crohn's disease, the causative mechanism involves cryptoglandular (aka intersphincteric) anal gland obstruction resulting in perianal inflammatory disease, with abscess representing the acute manifestation and fistula as a chronic condition [82, 83]. The obstruction could be a result of fecal material, foreign bodies, or trauma, with subsequent stasis and infection (primary perianal inflammatory process) or could be due to infection with TB, fungal, or viral infection [81]. The infection can spread either to the skin at the anal margin (caudal spread), or to the rectal wall (cranial spread), or can be spread to the ischioanal space by lateral extension of the infection through the external anal sphincter (lateral spread) [84].

Endorectal, endoanal, and perianal ultrasound (ERUS, EAUS, PNUS) as well as transvaginal sonography (TVUS) are useful techniques for evaluation of perianal inflammatory disease, with specific emphasis on the course of fistulous and sinus tracts (Figure 22). While performing these examinations, it is important to document presence or absence of

fluid collections (likely representing abscesses), number and the relationship of fistulous and sinus tracts to the internal and external sphincter. By convention, similarly to MRI techniques for evaluation of perianal fistulas, the openings of any fistulous tract in the rectum, anal canal, vagina, or perineal skin is described with the reference to anal clock [85, 86]. An internal opening is defined as an opening into the rectum or anus, whereas an external opening is defined as those on the perineum at physical examination [81]. By definition, a fistula is an abnormal communication between any two epithelial lined surfaces; therefore, any tract which is blind-ending and does not have both an external and internal opening is considered to be a sinus tract. Parks classification of fistulas is used for reporting the fistulas, their complexity, presence or absence of secondary fistula tracts, and abscesses [87, 88]. On perianal ultrasound, both transverse and sagittal planes are utilized. A fistula track is usually hypoechoic and contains a small amount of fluid and sometimes air. Hyperemia of the fistulous track wall may signify acute active inflammation (Fig. 22, Video 11). Most fistula tracts have an irregular shape with a blind end or with connection to other organs such as the uterus, urinary bladder, skin, or bowel. Examination of the fistula track by PNUS, especially the detection of the internal opening, can be better performed by the injection of contrast agents using a blunt cannula. Several studies have showed that perianal ultrasound has a high sensitivity and specificity in detecting perianal disease, comparable to those of MRI and ERUS/

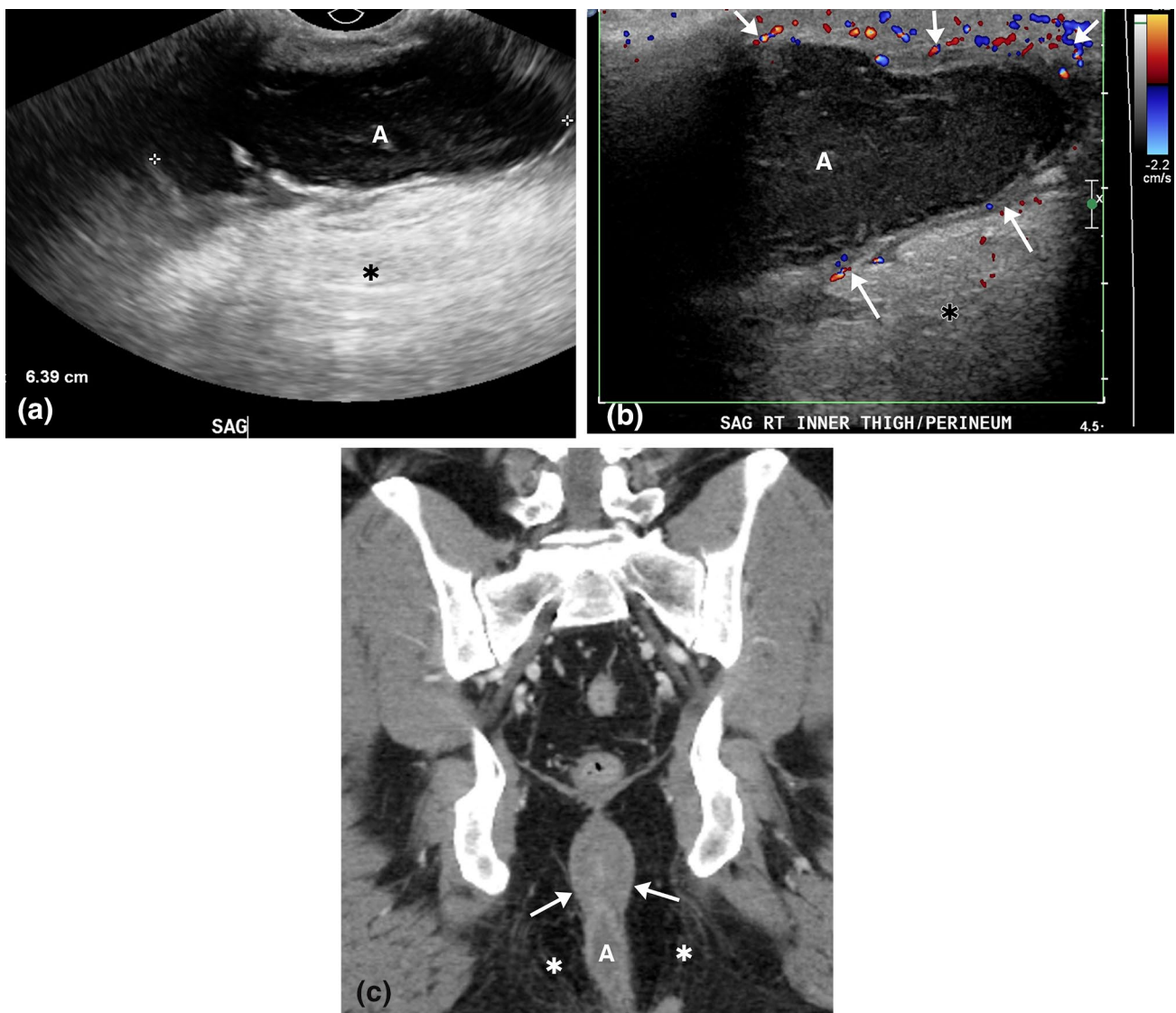


Fig. 23 Large abscess in the right inner thigh/perineum with associated fistula track extending to the anus in a 43-year-old male presenting with pain in the perineum. **a, b** Gray scale and color Doppler images obtained in sagittal plane via transperineal approach demonstrate a complex fluid collection compatible with an abscess (**a**). Marked peripheral hyperemia is noted in the soft tissues adjacent to

the abscess (arrows in **b**). Note inflammation of fat tissues adjacent to the abscess (asterisk in **a, b**). **c** Contrast-enhanced CT obtained in coronal plane demonstrates a fluid collection with peripherally enhancing rim compatible with an abscess (**a** in **c**). The abscess communicates with the adjacent anus (arrows). Fat stranding is noted in the ilioischial fossa (white asterisk in **c**)

EAUS. However, complex fistulas involving the external anal sphincter (EAS), levator and/or obturator muscles cannot be evaluated by PNUS alone. Endorectal and endoanal US and MRI examination should also be performed. Additionally, acute inflammation of the fistula tract can be

differentiated from sclerosing (chronic, non-acute) fistulas using sono-elastography, in which acute inflammation of the fistula will be seen as less stiff than the fibrotic tissue of a sclerosing fistula [51]. Fluid collections along the walls of the tract are compatible with small abscesses (Figs. 23, 24,

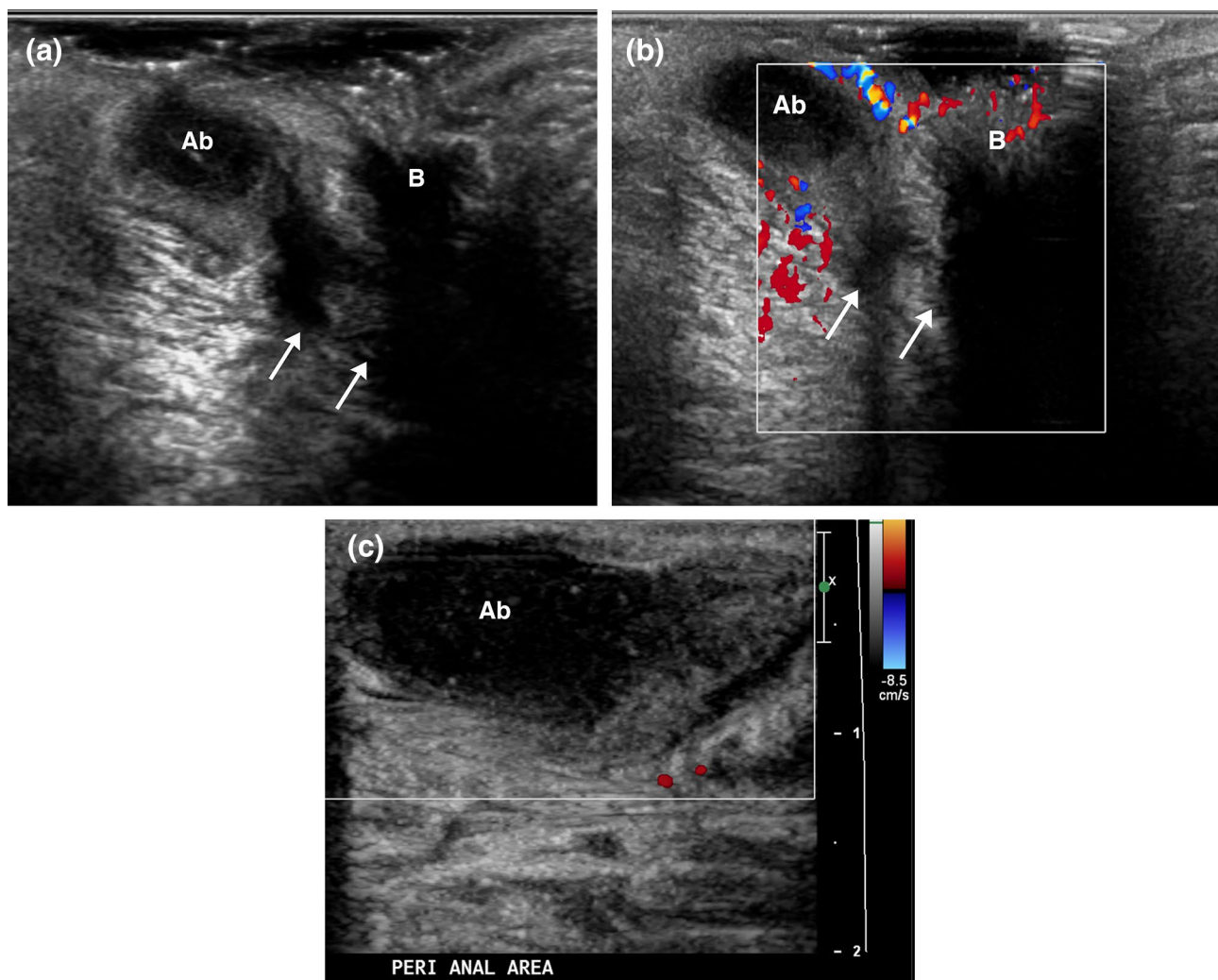


Fig. 24 Perianal abscess in an 11-year-old male who presented to the emergency department after foreign body removal. **a, b** Gray scale and color Doppler images obtained in the oblique plane via transperineal approach demonstrate complex fluid collection compatible with

an abscess (Ab) with peripheral hyperemia extending via the fistulous track (arrows) to the anus wall (**b**). **c** Magnified color Doppler image through the abscess demonstrates complex debris within the abscess (Ab)

Video 12). Based on Parks classification, 4 types of fistulas are recognized: transsphincteric and intersphincteric which are most common, suprasphincteric, and extrasphincteric. PNUS showed to have high sensitivity in classifying fistulas (92.8%) [89]. Superficial fistulas that do not communicate with underlying rectum or anus are not part of Parks classification and are usually associated with Crohn's disease. Management of the perianal fistulas and associated abscesses is based on the type of the fistula.

Conclusion

Ultrasound imaging that employs gray scale, duplex Doppler, CEUS, elastography, and real-time imaging are extremely useful and cost-effective non-invasive methods for evaluation of patients with infectious diseases of the bowel. Ultrasound imaging is fully capable of diagnosing such conditions and guiding patient management, and additionally plays a crucial role in disease surveillance and

assessment of treatment response. With the widespread use of ultrasound as well as improved recognition of the pertinent findings of bowel pathology, we are hopeful that the utilization of this application for assessment of infectious conditions of the bowel will increase dramatically.

Acknowledgements The authors thank Henry Douglas for his help with images, Anjel Mahon and Lei Wang for their help with video-recording, Mark Saba for his help with illustrations, Melody Polio, and Sasha Avenger for their help with obtaining images.

Funding None.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

- Moore KL (2018) Chapter 5 Abdomen In: Agur AMR, Dalley AF, Moore KL (eds) Clinically oriented anatomy. 8th edition. edn. Wolters Kluwer, Philadelphia, pp 439–487
- Osman S, Lehnert BE, Elojeimy S, Cruite I, Mannelli L, Bhargava P, Moshiri M (2013) A comprehensive review of the retroperitoneal anatomy, neoplasms, and pattern of disease spread. *Curr Probl Diagn Radiol* 42 (5):191–208. <https://doi.org/10.1067/j.cpradiol.2013.02.001>
- Kutchai HC (1998) Chapter 37 GastroIntestinal Motility In: Berne RM, Levy M (eds) Physiology, vol Section VI The Gastrointestinal System 4th ed. edn. Mosby, St. Louis, pp 589–617
- Nylund K, Maconi G, Hollerweger A, Ripolles T, Pallotta N, Higginson A, Serra C, Dietrich CF, Sporea I, Saftoiu A, Dirks K, Hausken T, Calabrese E, Romanini L, Maaser C, Nuernberg D, Gilja OH (2017) EFSUMB Recommendations and Guidelines for Gastrointestinal Ultrasound. *Ultraschall Med* 38 (3):e1–e15. <https://doi.org/10.1055/s-0042-115853>
- Muradali D, Goldberg DR (2015) US of gastrointestinal tract disease. *Radiographics: a review publication of the Radiological Society of North America, Inc* 35 (1):50–68. <https://doi.org/10.1148/rg.351140003>
- Sidhu PS, Cantisani V, Dietrich CF, Gilja OH, Saftoiu A, Bartels E, Bertolotto M, Calliada F, Clevert DA, Cosgrove D, Deganello A, D’Onofrio M, Drudi FM, Freeman S, Harvey C, Jenssen C, Jung EM, Klauser AS, Lassau N, Meloni MF, Leen E, Nicolau C, Nolsoe C, Piscaglia F, Prada F, Prosch H, Radzina M, Savelli L, Weskott HP, Wijkstra H (2018) The EFSUMB Guidelines and Recommendations for the Clinical Practice of Contrast-Enhanced Ultrasound (CEUS) in Non-Hepatic Applications: Update 2017 (Short Version). *Ultraschall Med* 39 (2):154–180. <https://doi.org/10.1055/s-0044-101254>
- Quaia E, Sozzi M, Angileri R, Gennari AG, Cova MA (2016) Time-Intensity Curves Obtained after Microbubble Injection Can Be Used to Differentiate Responders from Nonresponders among Patients with Clinically Active Crohn Disease after 6 Weeks of Pharmacologic Treatment. *Radiology* 281 (2):606–616. <https://doi.org/10.1148/radiol.2016152461>
- Medellin-Kowalewski A, Wilkens R, Wilson A, Ruan J, Wilson SR (2016) Quantitative Contrast-Enhanced Ultrasound Parameters in Crohn Disease: Their Role in Disease Activity Determination With Ultrasound. *AJR Am J Roentgenol* 206 (1):64–73. <https://doi.org/10.2214/AJR.15.14506>
- Medellin A, Merrill C, Wilson SR (2018) Role of contrast-enhanced ultrasound in evaluation of the bowel. *Abdom Radiol (NY)* 43 (4):918–933. <https://doi.org/10.1007/s00261-017-1399-6>
- Cosgrove D, Piscaglia F, Bamber J, Bojunga J, Correas JM, Gilja OH, Klauser AS, Sporea I, Calliada F, Cantisani V, D’Onofrio M, Drakonaki EE, Fink M, Friedrich-Rust M, Fromageau J, Havre RF, Jenssen C, Ohlinger R, Saftoiu A, Schaefer F, Dietrich CF, EfsUMB (2013) EFSUMB guidelines and recommendations on the clinical use of ultrasound elastography. Part 2: Clinical applications. *Ultraschall Med* 34 (3):238–253. <https://doi.org/10.1055/s-0033-1335375>
- Shiina T, Nightingale KR, Palmeri ML, Hall TJ, Bamber JC, Barr RG, Castera L, Choi BI, Chou YH, Cosgrove D, Dietrich CF, Ding H, Amy D, Farrokh A, Ferraioli G, Filice C, Friedrich-Rust M, Nakashima K, Schafer F, Sporea I, Suzuki S, Wilson S, Kudo M (2015) WFUMB guidelines and recommendations for clinical use of ultrasound elastography: Part 1: basic principles and terminology. *Ultrasound Med Biol* 41 (5):1126–1147. <https://doi.org/10.1016/j.ultrasmedbio.2015.03.009>
- Dietrich CF, Bamber J, Berzigotti A, Bota S, Cantisani V, Castera L, Cosgrove D, Ferraioli G, Friedrich-Rust M, Gilja OH, Goertz RS, Karlas T, de Knecht R, de Ledinghen V, Piscaglia F, Procopet B, Saftoiu A, Sidhu PS, Sporea I, Thiele M (2017) EFSUMB Guidelines and Recommendations on the Clinical Use of Liver Ultrasound Elastography, Update 2017 (Long Version). *Ultraschall Med* 38 (4):e48. <https://doi.org/10.1055/a-0641-0076>
- Havre R, Gilja OH (2014) Elastography and strain rate imaging of the gastrointestinal tract. *Eur J Radiol* 83 (3):438–441. <https://doi.org/10.1016/j.ejrad.2013.05.018>
- Bolondi L, Ferrentino M, Trevisani F, Bernardi M, Gasbarrini G (1985) Sonographic appearance of pseudomembranous colitis. *J Ultrasound Med* 4 (9):489–492. <https://doi.org/10.7863/jum.1985.4.9.489>
- Maconi G, Nylund K, Ripolles T, Calabrese E, Dirks K, Dietrich CF, Hollerweger A, Sporea I, Saftoiu A, Maaser C, Hausken T, Higginson AP, Nurnberg D, Pallotta N, Romanini L, Serra C, Gilja OH (2018) EFSUMB Recommendations and Clinical Guidelines for Intestinal Ultrasound (GIUS) in Inflammatory Bowel Diseases. *Ultraschall Med* 39 (3):304–317. <https://doi.org/10.1055/s-0043-125329>
- Puylaert JB (2003) Ultrasonography of the acute abdomen: gastrointestinal conditions. *Radiol Clin North Am* 41 (6):1227–1242, vii. [https://doi.org/10.1016/s0033-8389\(03\)00120-9](https://doi.org/10.1016/s0033-8389(03)00120-9)
- Fleischer AC, Muhletaler CA, James AE, Jr. (1981) Sonographic assessment of the bowel wall. *AJR Am J Roentgenol* 136 (5):887–891. <https://doi.org/10.2214/ajr.136.5.887>
- Puylaert JB (1986) Mesenteric adenitis and acute terminal ileitis: US evaluation using graded compression. *Radiology* 161 (3):691–695. <https://doi.org/10.1148/radiology.161.3.3538138>
- Anupindi SA, Halverson M, Khwaja A, Jeckovic M, Wang X, Bellah RD (2014) Common and uncommon applications of bowel ultrasound with pathologic correlation in children. *AJR American journal of roentgenology* 202 (5):946–959. <https://doi.org/10.2214/AJR.13.11661>
- Silva AC, Pimenta M, Guimaraes LS (2009) Small bowel obstruction: what to look for. *Radiographics* 29 (2):423–439. <https://doi.org/10.1148/rg.292085514>
- Azer SA, Akhondi H (2019) Gastritis. In: StatPearls. Treasure Island (FL)
- Walker MM, Potter M, Talley NJ (2018) Eosinophilic gastroenteritis and other eosinophilic gut diseases distal to the oesophagus. *Lancet Gastroenterol Hepatol* 3 (4):271–280. [https://doi.org/10.1016/S2468-1253\(18\)30005-0](https://doi.org/10.1016/S2468-1253(18)30005-0)

23. Cakmakci E, Ucan B, Colak B, Cinar HG (2014) Novel sonographic clues for diagnosis of antral gastritis and Helicobacter pylori infection: a clinical study. *Journal of ultrasound in medicine : official journal of the American Institute of Ultrasound in Medicine* 33 (9):1605-1610. <https://doi.org/10.7863/ultra.33.9.1605>
24. Savino A, Salvatore R, Cafarotti A, Cecamore C, De Sanctis S, Angelucci D, Mohn A, Chiarelli F, Pelliccia P (2011) Role of ultrasonography in the diagnosis and follow-up of pediatric eosinophilic gastroenteritis: a case report and review of the literature. *Ultraschall Med* 32 Suppl 2:E57-62. <https://doi.org/10.1055/s-0031-1281841>
25. Jayaraman MV, Mayo-Smith WW, Movson JS, Dupuy DE, Wallach MT (2001) CT of the duodenum: an overlooked segment gets its due. *Radiographics: a review publication of the Radiological Society of North America, Inc* 21 Spec No:S147-160. https://doi.org/10.1148/radiographics.21.suppl_1.g01oc01s147
26. Magliani W, Somenzi P, Valcavi P, Tcherassen M, Fanti F, Moccia G, Chezzi C (1985) Epidemiological survey on bacterial, viral and parasitic agents in patients affected by acute enteritis. *Eur J Epidemiol* 1 (2):127-130. <https://doi.org/10.1007/bf00141805>
27. Matsumoto T, Iida M, Sakai T, Kimura Y, Fujishima M (1991) Yersinia terminal ileitis: sonographic findings in eight patients. *AJR American journal of roentgenology* 156 (5):965-967. <https://doi.org/10.2214/ajr.156.5.2017961>
28. Puylaert JB, Van der Zant FM, Mutsaers JA (1997) Infectious ileocecolitis caused by Yersinia, Campylobacter, and Salmonella: clinical, radiological and US findings. *Eur Radiol* 7 (1):3-9
29. Ledermann HP, Borner N, Strunk H, Bongartz G, Zollikofer C, Stuckmann G (2000) Bowel wall thickening on transabdominal sonography. *AJR Am J Roentgenol* 174 (1):107-117. <https://doi.org/10.2214/ajr.174.1.1740107>
30. Maconi G, Imbesi V, Bianchi Porro G (1996) Doppler ultrasound measurement of intestinal blood flow in inflammatory bowel disease. *Scand J Gastroenterol* 31 (6):590-593. <https://doi.org/10.3109/00365529609009132>
31. Umetsu S, Sogo T, Iwasawa K, Kondo T, Tsunoda T, Oikawa-Kawamoto M, Komatsu H, Inui A, Fujisawa T (2014) Intestinal ascariasis at pediatric emergency room in a developed country. *World journal of gastroenterology : WJG* 20 (38):14058-14062. <https://doi.org/10.3748/wjg.v20.i38.14058>
32. Elsayes KM, Menias CO, Harvin HJ, Francis IR (2007) Imaging manifestations of Meckel's diverticulum. *AJR American journal of roentgenology* 189 (1):81-88. <https://doi.org/10.2214/AJR.06.1257>
33. Levy AD, Hobbs CM (2004) From the archives of the AFIP. Meckel diverticulum: radiologic features with pathologic Correlation. *Radiographics: a review publication of the Radiological Society of North America, Inc* 24 (2):565-587. <https://doi.org/10.1148/rg.242035187>
34. Fink AM, Alexopoulou E, Carty H (1995) Bleeding Meckel's diverticulum in infancy: unusual scintigraphic and ultrasound appearances. *Pediatr Radiol* 25 (2):155-156
35. Horton KM, Corl FM, Fishman EK (2000) CT evaluation of the colon: inflammatory disease. *Radiographics* 20 (2):399-418. <https://doi.org/10.1148/radiographics.20.2.g00mc15399>
36. Gritzmann N, Hollerweger A, Macheiner P, Rettenbacher T (2002) Transabdominal sonography of the gastrointestinal tract. *Eur Radiol* 12 (7):1748-1761. <https://doi.org/10.1007/s00330-001-1201-5>
37. Hollerweger A (2007) Colonic diseases: the value of US examination. *Eur J Radiol* 64 (2):239-249. <https://doi.org/10.1016/j.ejrad.2007.06.038>
38. Thoeni RF, Cello JP (2006) CT imaging of colitis. *Radiology* 240 (3):623-638. <https://doi.org/10.1148/radiol.2403050818>
39. Quillin SP, Siegel MJ (1994) Gastrointestinal inflammation in children: color Doppler ultrasonography. *J Ultrasound Med* 13 (10):751-756. <https://doi.org/10.7863/jum.1994.13.10.751>
40. FitzGerald JF, Hernandez Iii LO (2015) Ischemic colitis. *Clin Colon Rectal Surg* 28 (2):93-98. <https://doi.org/10.1055/s-0035-1549099>
41. Gardiner R, Smith C (1987) Infective enterocolitides. *Radiol Clin North Am* 25 (1):67-78
42. Torrisi JM, Schwartz LH, Gollub MJ, Ginsberg MS, Bosl GJ, Hricak H (2011) CT findings of chemotherapy-induced toxicity: what radiologists need to know about the clinical and radiologic manifestations of chemotherapy toxicity. *Radiology* 258 (1):41-56. <https://doi.org/10.1148/radiol.10092129>
43. Wagner ML, Rosenberg HS, Fernbach DJ, Singleton EB (1970) Typhlitis: a complication of leukemia in childhood. *Am J Roentgenol Radium Ther Nucl Med* 109 (2):341-350
44. Dietrich CF, Hermann S, Klein S, Braden B (2006) Sonographic signs of neutropenic enterocolitis. *World journal of gastroenterology : WJG* 12 (9):1397-1402
45. Cartoni C, Dragoni F, Micozzi A, Pescarmona E, Mecarocci S, Chirletti P, Petti MC, Meloni G, Mandelli F (2001) Neutropenic enterocolitis in patients with acute leukemia: prognostic significance of bowel wall thickening detected by ultrasonography. *J Clin Oncol* 19 (3):756-761. <https://doi.org/10.1200/JCO.2001.19.3.756>
46. Brown MA (2008) Imaging acute appendicitis. *Semin Ultrasound CT MR* 29 (5):293-307
47. Puig S, Staudenherz A, Felder-Puig R, Paya K (2008) Imaging of appendicitis in children and adolescents: useful or useless? A comparison of imaging techniques and a critical review of the current literature. *Semin Roentgenol* 43 (1):22-28. <https://doi.org/10.1053/j.ro.2007.08.004>
48. Humes DJ, Simpson J (2006) Acute appendicitis. *BMJ* 333 (7567):530-534. <https://doi.org/10.1136/bmj.38940.664363.AE>
49. Shogilev DJ, Duus N, Odom SR, Shapiro NI (2014) Diagnosing appendicitis: evidence-based review of the diagnostic approach in 2014. *West J Emerg Med* 15 (7):859-871. <https://doi.org/10.5811/westjem.2014.9.21568>
50. Expert Panel on Gastrointestinal I, Peterson CM, McNamara MM, Kamel IR, Al-Refaie WB, Arif-Tiwari H, Cash BD, Chernyak V, Goldstein A, Grajo JR, Hindman NM, Horowitz JM, Noto RB, Porter KK, Srivastava PK, Zaheer A, Carucci LR (2019) ACR Appropriateness Criteria(R) Right Upper Quadrant Pain. *J Am Coll Radiol* 16 (5S):S235-S243. <https://doi.org/10.1016/j.jacr.2019.02.013>
51. Dirks K, Calabrese E, Dietrich CF, Gilja OH, Hausken T, Higinson A, Hollerweger A, Maconi G, Maaser C, Nuernberg D, Nylund K, Pallotta N, Ripolles T, Romanini L, Saftoiu A, Serra C, Wustner M, Sporea I (2019) EFSUMB Position Paper: Recommendations for Gastrointestinal Ultrasound (GIUS) in Acute Appendicitis and Diverticulitis. *Ultraschall Med* 40 (2):163-175. <https://doi.org/10.1055/a-0824-6952>
52. Lee JH, Jeong YK, Hwang JC, Ham SY, Yang SO (2002) Graded compression sonography with adjuvant use of a posterior manual compression technique in the sonographic diagnosis of acute appendicitis. *AJR Am J Roentgenol* 178 (4):863-868. <https://doi.org/10.2214/ajr.178.4.1780863>
53. Chang ST, Jeffrey RB, Olcott EW (2014) Three-step sequential positioning algorithm during sonographic evaluation for appendicitis increases appendiceal visualization rate and reduces CT use. *AJR American journal of roentgenology* 203 (5):1006-1012. <https://doi.org/10.2214/AJR.13.12334>
54. Xu Y, Jeffrey RB, DiMaio MA, Olcott EW (2016) Lymphoid Hyperplasia of the Appendix: A Potential Pitfall in the Sonographic Diagnosis of Appendicitis. *AJR Am J Roentgenol* 206 (1):189-194. <https://doi.org/10.2214/AJR.15.14846>

55. Macari M, Hines J, Balthazar E, Megibow A (2002) Mesenteric adenitis: CT diagnosis of primary versus secondary causes, incidence, and clinical significance in pediatric and adult patients. *AJR Am J Roentgenol* 178 (4):853-858. <https://doi.org/10.2214/ajr.178.4.1780853>
56. Tarantino L, Giorgio A, de Stefano G, Scala V, Esposito F, Liorre G, Farella N, Ferraioli G (2003) Acute appendicitis mimicking infectious enteritis: diagnostic value of sonography. *Journal of ultrasound in medicine : official journal of the American Institute of Ultrasound in Medicine* 22 (9):945-950
57. Stocchi L, Wolff BG, Larson DR, Harrington JR (2003) Surgical treatment of appendiceal mucocele. *Arch Surg* 138 (6):585-589; discussion 589-590. <https://doi.org/10.1001/archsurg.138.6.585>
58. Deans GT, Spence RA (1995) Neoplastic lesions of the appendix. *Br J Surg* 82 (3):299-306
59. Connor SJ, Hanna GB, Frizelle FA (1998) Appendiceal tumors: retrospective clinicopathologic analysis of appendiceal tumors from 7,970 appendectomies. *Dis Colon Rectum* 41 (1):75-80
60. Lien WC, Huang SP, Chi CL, Liu KL, Lin MT, Lai TI, Liu YP, Wang HP (2006) Appendiceal outer diameter as an indicator for differentiating appendiceal mucocele from appendicitis. *Am J Emerg Med* 24 (7):801-805. <https://doi.org/10.1016/j.ajem.2006.04.003>
61. Saad EA, Elsamani EY, AbdElrahim WE, Elsiddig KE, Khalil EAG (2018) Surgical treatment of mucocele of the appendix: a systematic review and case report. *J Surg Case Rep* 2018 (6):rjy102. <https://doi.org/10.1093/jscr/rjy102>
62. Bennett GL, Tanpitukpongse TP, Macari M, Cho KC, Babb JS (2009) CT diagnosis of mucocele of the appendix in patients with acute appendicitis. *AJR Am J Roentgenol* 192 (3):W103-110. <https://doi.org/10.2214/AJR.08.1572>
63. Bahia JO, Wilson MH (1989) Mucocele of the appendix presenting as an adnexal mass. *Journal of clinical ultrasound : JCU* 17 (1):62-66
64. Kim SH, Lim HK, Lee WJ, Lim JH, Byun JY (1998) Mucocele of the appendix: ultrasonographic and CT findings. *Abdom Imaging* 23 (3):292-296. <https://doi.org/10.1007/s002619900343>
65. Sasaki K, Ishida H, Komatsuda T, Suzuki T, Konno K, Ohtaka M, Sato M, Ishida J, Sakai T, Watanabe S (2003) Appendiceal mucocele: sonographic findings. *Abdom Imaging* 28 (1):15-18. <https://doi.org/10.1007/s00261-001-0175-8>
66. Kameda T, Kawai F, Taniguchi N, Omoto K, Kobori Y, Arakawa K (2014) Evaluation of whether the ultrasonographic onion skin sign is specific for the diagnosis of an appendiceal mucocele. *J Med Ultrason* (2001) 41 (4):439-443. <https://doi.org/10.1007/s10396-014-0527-y>
67. Abboud ME, Frasure SE, Stone MB (2016) Ultrasound diagnosis of diverticulitis. *World J Emerg Med* 7 (1):74-76. <https://doi.org/10.5847/wjem.j.1920-8642.2016.01.015>
68. Kozak LJ, DeFrances CJ, Hall MJ (2006) National hospital discharge survey: 2004 annual summary with detailed diagnosis and procedure data. *Vital Health Stat* 13 (162):1-209
69. Sandler RS, Everhart JE, Donowitz M, Adams E, Cronin K, Goodman C, Gemmen E, Shah S, Avdic A, Rubin R (2002) The burden of selected digestive diseases in the United States. *Gastroenterology* 122 (5):1500-1511
70. Weizman AV, Nguyen GC (2011) Diverticular disease: epidemiology and management. *Can J Gastroenterol* 25 (7):385-389
71. Cuomo R, Barbara G, Pace F, Annese V, Bassotti G, Binda GA, Casetti T, Colecchia A, Festi D, Fiocca R, Laghi A, Maconi G, Nascimbeni R, Scarpignato C, Villanacci V, Annibale B (2014) Italian consensus conference for colonic diverticulosis and diverticular disease. *United European Gastroenterol J* 2 (5):413-442. <https://doi.org/10.1177/2050640614547068>
72. Lameris W, van Randen A, Bipat S, Bossuyt PM, Boermeester MA, Stoker J (2008) Graded compression ultrasonography and computed tomography in acute colonic diverticulitis: meta-analysis of test accuracy. *Eur Radiol* 18 (11):2498-2511. <https://doi.org/10.1007/s00330-008-1018-6>
73. Andeweg CS, Mulder IM, Felt-Bersma RJ, Verbon A, van der Wilt GJ, van Goor H, Lange JF, Stoker J, Boermeester MA, Bleichrodt RP, Netherlands Society of S, Working group from Netherlands Societies of Internal Medicine GRHeA, Dieticians (2013) Guidelines of diagnostics and treatment of acute left-sided colonic diverticulitis. *Dig Surg* 30 (4-6):278-292. <https://doi.org/10.1159/000354035>
74. Rodgers PM, Verma R (2013) Transabdominal ultrasound for bowel evaluation. *Radiol Clin North Am* 51 (1):133-148. <https://doi.org/10.1016/j.rcl.2012.09.008>
75. Mazzei MA, Cioffi Squitieri N, Guerrini S, Stabile Ianora AA, Cagini L, Macarini L, Giganti M, Volterrani L (2013) Sigmoid diverticulitis: US findings. *Crit Ultrasound J* 5 Suppl 1:S5. <https://doi.org/10.1186/2036-7902-5-S1-S5>
76. Singh AK, Gervais DA, Hahn PF, Rhea J, Mueller PR (2004) CT appearance of acute appendagitis. *AJR American journal of roentgenology* 183 (5):1303-1307. <https://doi.org/10.2214/ajr.183.5.1831303>
77. Molla E, Ripolles T, Martinez MJ, Morote V, Rosello-Sastre E (1998) Primary epiploic appendagitis: US and CT findings. *Eur Radiol* 8 (3):435-438. <https://doi.org/10.1007/s003300050408>
78. Singh AK, Alhilali LM, Gervais DA, Mueller PR (2004) Omental infarct: an unusual CT appearance after superior mesenteric artery occlusion. *Emerg Radiol* 10 (5):276-278. <https://doi.org/10.1007/s10140-004-0339-2>
79. Maddu KK, Mittal P, Shuaib W, Tewari A, Ibraheem O, Khosa F (2014) Colorectal emergencies and related complications: a comprehensive imaging review—imaging of colitis and complications. *AJR Am J Roentgenol* 203 (6):1205-1216. <https://doi.org/10.2214/AJR.13.12250>
80. Herlihy JD, Beasley S, Simmelink A, Maddukuri V, Amin A, Kamionek M, Jacobs C, Bossi K, Scobey M (2019) Flexible Sigmoidoscopy Rather than Colonoscopy Is Adequate for the Diagnosis of Ipilimumab-Associated Colitis. *South Med J* 112 (3):154-158. <https://doi.org/10.14423/SMJ.0000000000000944>
81. Stewart LK, McGee J, Wilson SR (2001) Transperineal and transvaginal sonography of perianal inflammatory disease. *AJR Am J Roentgenol* 177 (3):627-632. <https://doi.org/10.2214/ajr.177.3.1770627>
82. Sheikh MA, Ashraf SA (2007) Terminal ileum schistosomiasis with perianal fistula mimicking Crohn's disease. *Saudi Med J* 28 (9):1449-1452
83. Siddiqui MR, Ashrafian H, Tozer P, Daulatzai N, Burling D, Hart A, Athanasiou T, Phillips RK (2012) A diagnostic accuracy meta-analysis of endoanal ultrasound and MRI for perianal fistula assessment. *Dis Colon Rectum* 55 (5):576-585. <https://doi.org/10.1097/DCR.0b013e318249d26c>
84. Vasilevsky CA, Gordon PH (1984) The incidence of recurrent abscesses or fistula-in-ano following anorectal suppuration. *Dis Colon Rectum* 27 (2):126-130
85. Halligan S (1998) Imaging fistula-in-ano. *Clin Radiol* 53 (2):85-95
86. Halligan S, Buchanan G (2003) MR imaging of fistula-in-ano. *Eur J Radiol* 47 (2):98-107
87. Parks AG, Gordon PH, Hardcastle JD (1976) A classification of fistula-in-ano. *Br J Surg* 63 (1):1-12. <https://doi.org/10.1002/bjs.1800630102>
88. Nuernberg D, Saftoiu A, Barreiros AP, Burmester E, Ivan ET, Clevert DA, Dietrich CF, Gilja OH, Lorentzen T, Maconi G, Mihmanli I, Nolsoe CP, Pfeffer F, Rafaelsen SR, Sparchez Z,

- Vilman P, Waage JER (2019) EFSUMB Recommendations for Gastrointestinal Ultrasound Part 3: Endorectal, Endoanal and Perineal Ultrasound. *Ultrasound Int Open* 5 (1):E34-E51. <https://doi.org/10.1055/a-0825-6708>
89. Maconi G, Greco MT, Asthana AK (2017) Transperineal Ultrasound for Perianal Fistulas and Abscesses - A Systematic Review and Meta-Analysis. *Ultraschall Med* 38 (3):265-272. <https://doi.org/10.1055/s-0043-103954>

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.