Parasitic diseases as a cause of acute abdominal pain: imaging findings

Emre Ünal *, Sevtap Arslan, Mehmet Ruhi Onur and Erhan Akpinar

Abstract
Acute abdominal pain can be seen in cases with parasitic diseases delivered to emergency departments. The diagnosis of the parasitic disease can be delayed because of the similar clinical signs encountered in other frequently seen causes of acute abdomen. Nevertheless, the features detected in imaging scans can be helpful in the diagnosis. The present study aims to raise awareness about abdominal parasitosis in emergency conditions and also to underline the association between imaging findings and the life cycle of parasites with illustrative cases.

Keywords: Abdominal parasitosis, Abdominal emergency, Imaging findings, CT, Acute abdomen

Key points
- Some types of parasites are endemic for certain locations.
- Immigration and travel are responsible for worldwide cases.
- A parasite may involve a specific organ or migrate through several organs.
- A parasite may induce a cyst formation that could be complicated with rupture, superinfection, or mass effect.
- Clinical and radiological findings may vary for the different types of parasites and their site of involvement.

Background
Parasitic diseases are common. Although some types of parasites are endemic for certain locations, worldwide cases can be seen due to immigration and travel. A previously published systematic review reported that parasitic diseases resulted in 48.4 million cases annually and 59,724 deaths per year [1]. The diseases caused by most parasites results in significant morbidity and mortality among vulnerable populations. In the same study, researchers emphasize that enteric protozoa, ascariasis, and toxoplasmosis are the most common parasitic diseases; however, the global burden of disease is highest in cysticercosis. Therefore, these data indicate that there is no correlation between the incidence of the parasitic disease and the frequency and severity of symptoms [1]. Parasitic diseases may be transmitted in three different ways as (i) fecal-oral route, (ii) active penetration of the skin by larvae, and (iii) vector arthropods [2]. Infestation affects different parts of the body. Nevertheless, abdominal involvement is seen in the majority of cases. Although the clinical symptoms are usually nonspecific, patients may present with acute abdominal pain due to inflammatory changes in parenchymal organ, bowel walls, bile ducts, and peritoneal surfaces and obstructive changes in bowels and bile ducts. Acute abdominal pain may also result from complications of parasitic involvement such as abscess formation and rupture of focal parasitic cystic lesions. A parasite may be hosted by a specific intraabdominal organ, or may travel among several intraabdominal organs, or may induce a cyst formation that could be complicated with rupture, superinfection, or mass effect. Therefore, clinical and radiological findings may vary for the different types of parasites and also for their site of involvement. The present study aims to raise awareness about abdominal parasitosis that we encountered in our emergency radiology practice. A detailed literature search was also...
| Disease          | Parasite          | Epidemiology          | Geographic distribution                                                                 | Hosts/vectors  | Transmission          | Final diagnosis (routinely used method)                                                                 |
|------------------|-------------------|-----------------------|----------------------------------------------------------------------------------------|----------------|-----------------------|--------------------------------------------------------------------------------------------------------|
| Cystic echinococcosis [3–6] | E. granulosus [a] | 2–404/10,000          | High prevalence in Mediterranean regions, southern and central parts of Russia, central Asia, China, Australia, South America and Africa | 1 Definitive hosts (dogs and foxes) | Intermediate hosts (sheep and human) | Imaging based (primarily ultrasound findings) |
|                  | E. multilocularis  | 0.2–3400/100,000      | Asia, Central Europe and the northern parts of Europe, and North America               |                |                       | Aspiration of cyst content during imaging guided intervention |
|                  | E. oligarthrus     | Very rare, 106 human cases | Central and South America                                                            |                |                       |                                                                                                          |
|                  | E. vogeli          |                       |                                                                                       |                |                       |                                                                                                          |
| Fascioliasis [3, 7] | F. hepatica [a]   | 0.9–6.1%              | Worldwide, high prevalence in Europe and America                                        | 1 Intermediate hosts (Freshwater snails) |                   | Stool examination |
|                  | F. gigantica      |                       | Tropical areas of Asia and Africa                                                    |                |                       | Serologic assay |
| Ascariasis [3, 8] | Ascaris lumbricoides | 8.8–22.3%          | Asia, Africa, and South America                                                       | 1 No intermediate host |                   | Stool examination |
|                  |                   |                       |                                                                                       |                |                       |                                                                                                          |
| Toxocariasis [9]  | T. canis [a]      | 0.7–15%               | Worldwide                                                                               | 1 Intermediate hosts (rabbit, lamb, fowl) |                   | Serologic assay |
|                  | T. cati           |                       |                                                                                        |                |                       |                                                                                                          |
| Intestinal taeiniasis [10–12] | T. saginata | 0.7–4.9%              | Sub-Saharan Africa and the Middle East, Eastern Europe, the Philippines, and Latin America | 1 Intermediate hosts (pigs for T. solium, cattle for T. saginata) |                   | Stool examination |
|                  | T. solium         |                       | Worldwide; especially in Mexico, Latin America, West Africa, Russia, India, Manchuria, and Southeast Asia |                |                       |                                                                                                          |
|                  | T. asiatica      |                       |                                                                                        |                |                       |                                                                                                          |
| Amoebiasis [13]   | E. histolytica [a] | 5–42%                 | Worldwide. Endemic in developing parts of Central and South America, Africa, and Asia   | 1 Humans are the principal host |                   | Stool examination |
|                  | E. dispar         |                       |                                                                                        |                |                       | Microscopic examination of blood                                                                 |
|                  | E. moshkovskii    |                       |                                                                                        |                |                       |                                                                                                          |
| Malaria [14, 15]  | P. falciparum [a] | 5.2–75%               | Tropical Africa, South America, South-eastern Asia, and Western Pacific            | 1 Vectors (Anopheles mosquitoes) |                   |                                                                                                          |
|                  | P. vivax           |                       |                                                                                        |                |                       |                                                                                                          |
|                  | P. ovale          |                       |                                                                                        |                |                       |                                                                                                          |
|                  | P. malariae       |                       |                                                                                        |                |                       |                                                                                                          |
|                  | P. knowlesi       |                       |                                                                                        |                |                       |                                                                                                          |
| Visceral leishmaniasis [16] | Leishmania donovani | 200,000–400,000/year | Northeast of the Indian subcontinent, East Africa                                     | 1 Vectors (Phlébotomus sandflies) |                   | Bone marrow sampling |
|                  | Leishmania infantum |                       | Mediterranean region, Latin America                                                   |                |                       | Serological examination                                                                                        |
| Dientamoebiasis [17] | Dientamoeba fragilis | 0.4–42%             | Worldwide                                                                              | 1 Humans are the principal host |                   | Stool examination |
|                  |                   |                       |                                                                                        |                |                       |                                                                                                          |
| Anisakiasis [18]  | Anisakis simple   | 3/1,000,000 in Japan  | Japan, Korea, Latin America, and Europe (Scandinavia, The Netherlands, Spain, France, Britain). Over 50% of cases are from Japan | 1 Intermediate hosts (different species of crustaceans, fish, or squid) |                   | Endoscopy, histopathological examination |

[a] Most common
[b] More severe clinical presentation
[c] References
carried out to be able to summarize the epidemiologic aspects, radiological, clinical, and laboratory findings of these particular parasitic diseases (Tables 1 and 2) [3–19, 67, 74, 78].

Hydatid cyst

Hydatid disease is a worldwide zoonosis produced by the larval stage of the Echinococcus tapeworm. There are 4 types of Echinococcus infections. E. granulosus is the most common type, whereas E. multilocularis is less common but more invasive, mimicking malignancy [20]. E. vogeli and E. oligarthrus are very rare.

In humans, hydatid disease involves the liver in approximately 75% of the cases [21]. Although the liver is the most frequent site of involvement, any part of the body may host the hydatid disease. Hepatic hydatid cyst is commonly detected incidentally and patients are asymptomatic in most of the cases. Ultrasonography (US) can be used as a screening method of choice in liver hydatidosis. Gharbi classification system and World Health Organization (WHO) classification system classify the hydatid cysts based on their gray-scale ultra-sound appearances. Unilocular or multilocular appearance, anechoic or echogenic content, multivesicular or multiseptated appearance, presence of hydatid sand, daughter cysts, floating membrane, and/or calcified wall are the sonographic features which determine the type of the hydatid cyst in Gharbi’s or WHO classification system [18, 22, 23]. Gharbi’s and WHO classification systems are helpful to determine to evaluate the cyst activity the appropriate treatment method (medical treatment, percutaneous drainage, or surgical excision) for the hydatid cyst type [22–25]. The sensitivity of computed tomography (CT) in liver hydatidosis is 94% [20]. Evaluation of cystic component, vascular and biliary tree involvement, and extrahepatic extension may be assessed with magnetic resonance imaging (MRI) [26].

Hepatic hydatid cyst may cause acute abdominal pain due to its complications. The most common complication is the rupture and the most common site of the rupture is the biliary tree [21, 27, 28]. Other sites of the rupture are peritoneal cavity, thoracic cavity, hepatic subcapsular space, hollow viscera, and abdominal wall [21, 27]. The rupture may cause superinfection and anaphylaxis [27, 28]. The imaging findings of intrabiliary rupture of the hydatid cyst are structural deformity, loss of spherical shape, dilatation of the intrahepatic bile ducts, and linear filling defects within the biliary tract (Figs. 1 and 2) [21, 27, 28]. Also, air or an air-fluid level within the cyst may be present and this may indicate super-infection (Fig. 3) [28]. Intrapерitoneal rupture is a rare complication and may cause peritoneal seeding (Fig. 4) [27, 29]. Superficial, large, and thin-walled hepatic hydatid cysts are most vulnerable to abdominal rupture [28]. The imaging findings of intraperitoneal rupture of the hydatid cyst are focal outward bulging, discontinuity of the cyst wall adjacent to the hepatic capsule, and intraperitoneal fluid collections [21, 27–29]. Also, mural thickening of the bowel loops and peritoneal fat tissue stranding may occur due to the local allergic reactions (Fig. 5). Large hydatid cysts in the liver or other organs may cause acute abdominal symptoms due to mass effect. Alveolar echinococcosis (E. multilocularis) may demonstrate an infiltrative growth pattern and patients’ symptoms are correlated with the size of the lesion. Compression of the biliary system may result in acute cholangitis and imaging findings may resemble those seen in liver malignancy (Fig. 6) [30]. Rupture or mass effect may be seen in other abdominal organ involvement (Fig. 7) [31]. In renal hydatid disease, the rupture of the hydatid cyst in the pelvicalyceal system is a rare complication (Fig. 8). Other rare complications of abdominal hydatid cyst (disease) are portal hypertension, portal vein thrombosis, and Budd-Chiari syndrome due to mass effect on the portal and hepatic veins [28].

Fascioliasis

Fascioliasis is an important disease caused by Fasciola hepatica and Fasciola gigantica. F. hepatica is the most common type. Freshwater snail species are the intermediate hosts for fasciola parasites while many herbivorous mammals including humans are the definitive host [32]. Fasciola parasites develop into adult flukes in the bile ducts of infected mammals, which transfer immature Fasciola eggs through their feces [36].

The infection in humans begins when watercress or contaminated water containing encysted larva are ingested. The larvae reach the liver after penetrating the duodenal wall, migrating through the peritoneal cavity and penetrating the Glisson’s capsule. The flukes then migrate through the liver parenchyma to the bile ducts and gallbladder which are their permanent residence. The migratory larval and resting adult stages correspond to two clinical stages of the life cycle, called the hepatic and biliary stages [34, 35]. Common symptoms of the hepatic phase are urticaria, right upper quadrant pain, and fever. The biliary phase is usually accompanied by intermittent right upper quadrant pain, with or without cholangitis or cholelithiasis [36].

The migration process causes multiple microabscesses in the liver. The findings of the migration process become visible on CT as a “tunnel-like tract” from the entry site at the Glisson’s capsule deep to the parenchyma and clustered small necrotic cavities arranged in serpentine fashion are seen as “caves” [34–37]. “Tunnels and caves” sign is very characteristic for fascioliasis (Figs. 9 and 10). Subcapsular hemorrhage-effusion, thickening, and enhancement of the focal liver capsule may occur due to penetration of the Glisson’s capsule by larva [36, 38].
| Disease type | Imaging findings | Clinical presentation | Lab findings |
|---------------|------------------|-----------------------|--------------|
| Cystic echinococcosis\(^\text{a}\) [2, 3, 19–31] | - Fluid collection through the course of cyst's content | RUQ or generalized abdominal pain, fever, nausea and vomiting, urticaria, and fatal anaphylaxis | Leukocytosis, elevated CRP (C reactive protein), eosinophilia, increase in serum aspartate transaminase (AST) and alanine aminotransferase (ALT) levels |
| Liver | - Mural thickening of the bowel loops and fat tissue standing adjacent to cyst contents | | |
| Biliary rupture | - Focal outward bulging, discontinuity of the cyst wall | | |
| Vascular invasion | - Structural deformity, loss of spherical shape of the cyst | RUQ pain, jaundice, pruritus, fever, nausea and vomiting, anaphylactic reaction | Leukocytosis, elevated CRP, eosinophilia, elevated serum AST, ALT, gamma-glutamyl transferase (GGT), alkaline phosphatase (ALP), bilirubin, and amylase |
| Liver | - Dilatation of the intrahepatic bile ducts with linear filling defects within the biliary tract | | |
| Biliary rupture | - Lipid-fluid level due to bile | | |
| Vascular invasion | - Total occlusion or linear filling defects within the lumen | RUQ pain, dyspnea and chest pain in case of pulmonary embolism | Leukocytosis, eosinophilia, abnormal liver function tests and, hypoxemia and hypocapnia/hypercapnia in case of pulmonary embolism |
| Liver | - Parenchymal hemodynamic change at vascular territory | | |
| Biliary compression | - Dilatation of the intrahepatic bile ducts without linear filling defects; however, increased echogenicity/decreased T2 signal could be seen due to within the biliary tract. Dilatation of the intrahepatic bile ducts, enhancement of intrahepatic biliary duct, parenchymal changes (increased signal intensity on T2-weighted images, segmental parenchymal enhancement) | RUQ, jaundice, pruritus, fever, nausea and vomiting less common compared to biliary rupture | Eosinophilia, elevated ALP, GGT, and bilirubin |
| Kidney | - Fluid collection through the course of cyst's content | Flank pain, fever, nausea and vomiting, urticaria, and fatal anaphylaxis | Leukocytosis, elevated CRP, eosinophilia, high serum blood urea nitrogen (BUN), and creatinine |
| Perirenal rupture | - Focal outward bulging, discontinuity of the cyst wall | | |
| Pelvicalyceal system rupture | - Structural deformity, loss of spherical shape of the cyst | Flank pain, fever, nausea and vomiting, urticaria, and fatal anaphylaxis | |
| Liver | - Hydroureteronephrosis, pyonephrosis may accompany | | |
| Pelvicalyceal system compression | - Filling defects within the pelvicalyceal system | | |
| Liver | - Urinoma may accompany in case of pelvicalyceal system rupture | | |
| Pelvicalyceal system | - Hydroureteronephrosis without filling defects | Flank pain, fever, nausea and vomiting, urticaria, and fatal anaphylaxis | Eosinophilia, elevated BUN, and creatinine |
| compression | - Urinary dilatation is seen above the level of compression | | |
| Fascioliasis\(^\text{a}\) [2, 3, 32–43] | Multiple microabscesses, “tunnels and caves” sign | RUQ pain, urticaria, right upper quadrant pain and fever, fatigue | Leukocytosis, eosinophilia, elevated liver enzymes |
| Liver | - Subcapsular hemorrhage-effusion, focal liver capsule thickening and enhancement | | |
| Liver | - Periportal lymphadenopathy, rarely portal vein thrombosis and wedge-shaped hemorrhagic infarction | | |
| Liver | - Splenomegaly may accompany | | |
| Biliary stage | - Dilated biliary ducts, periportal thickening | RUQ pain, pain could be disseminated in case of pancreatitis, jaundice, fever, nausea, diarrhea, pruritus | Leukocytosis, eosinophilia, elevated liver enzymes, hyperbilirubinemia, and elevated pancreatic enzymes in case of pancreatitis |
| Liver | - Floating particles in the biliary system | | |
| Disease type       | Imaging findings                                                                 | Clinical presentation                                                                 | Lab findings                                                                 |
|--------------------|----------------------------------------------------------------------------------|--------------------------------------------------------------------------------------|----------------------------------------------------------------------------|
| Ascariasis\[2, 3, 44–49\] | - Tubular structure within the lumen of stomach or bowel, associated fat tissue stranding  
- Dilated bowels in case of intestinal obstruction  
- Rarely, intraabdominal free fluid, gas, and/or fluid collection in case of GIS perforation  
- Inflamed and distended appendix vermiformis | Abdominal distension, dyspepsia, nausea and vomiting in case of GIS obstruction, generalized abdominal pain fever and sepsis in case of GIS perforation, RLQ pain in case of acute appendicitis | Anemia, leukocytosis, eosinophilia |
| Liver              | - Areas of decreased attenuation on CT, altered echogenicity on US due to parenchymal involvement  
- Filling defects within the biliary tree or gallbladder  
- Biliary dilatation, periportal inflammation  
- Gallbladder wall thickening and distention  
- Parenchymal or perihepatic abscess | RUQ pain, fatigue, fever, jaundice and pruritus in case of biliary dilatation, MURPHY sign positivity in case of acute cholecystitis, septic status can be seen in case of gallbladder perforation or liver abscess | Anemia, leukocytosis, eosinophilia, and elevated liver tests including bilirubin |
| Pancreas           | - Thickening of pancreas, peripancreatic fat tissue stranding and fluid collection may accompany | Epigastric pain and acute pancreatitis can be the sole presentation or be seen following biliary involvement | Leukocytosis, eosinophilia, elevated pancreatic enzymes, mild hyperbilirubinemia, high ALP and GGT levels |
| Toxocariasis\[2, 46–49\] | - Multiple, ill-defined, oval shaped, small lesions (< 2 cm) in parenchyma  
- Lesions may coalescence on follow-up  
- Splenomegaly may accompany | RUQ pain, fatigue, fever, nausea and vomiting | Leukocytosis, eosinophilia, mildly elevated liver enzymes |
| Taeniasis\[2, 18, 50–56\] | - Tubular structure within the lumen of stomach or bowel, associated fat tissue stranding  
- Dilated bowels in case of intestinal obstruction  
- Rarely, intraabdominal free fluid, gas and/or fluid collection in case of GIS perforation  
- Inflamed and distended appendix vermiformis | Abdominal distension, dyspepsia, nausea and vomiting in case of GIS obstruction, generalized abdominal pain fever and sepsis in case of GIS perforation, RLQ pain in case of acute appendicitis | Leukocytosis, eosinophilia, anemia |
| Liver              | - Filling defects within the biliary tree or gallbladder  
- Biliary dilatation, periportal inflammation  
- Gallbladder wall thickening and distention | RUQ pain, fatigue, fever, jaundice and pruritus in case of biliary dilatation, MURPHY sign positivity in case of acute cholecystitis, septic status can be seen in case of gallbladder perforation or liver abscess | Leukocytosis, eosinophilia, hyperbilirubinemia and elevated liver enzymes |
| Pancreas           | - Thickening of pancreas, peripancreatic fat tissue stranding and fluid collection may accompany | Epigastric pain and acute pancreatitis can be the sole presentation or be seen following biliary involvement | Leukocytosis, eosinophilia, elevated pancreatic and liver enzymes |
| Amoebiasis\[44, 57–59\] | - Colonic wall thickening, mucosal edema and increased mucosal enhancement (primarily ascending colon)  
- Rarely, intraabdominal free fluid, gas and/or fluid collection in case of colonic perforation | Bloody diarrhea, tenesmus, fever, and abdominal pain. Generalized abdominal pain and peritonitis in case of colon perforation | Leukocytosis, anemia |
| Liver              | - Parenchymal loculated fluid collection (abscess), air or an air-fluid level can be seen within the collection.  
- Perihepatic fluid and right-sided pleural effusion may accompany | RUQ pain, fever, colonic symptoms may accompany, sepsis in case of diffuse liver abscesses | Leukocytosis without eosinophilia, anemia, elevated liver enzymes, high erythrocyte sedimentation rate and CRP |
Wedge-shaped hemorrhagic infarction in the periphery of the liver was reported as a result of an experimental study on hepatobiliary fascioliasis [39]. A case presenting portal vein thrombosis has also been reported [40]. Periportal lymphadenopathy is a frequent and helpful finding which can be encountered in both hepatic and biliary stage [38].

In the biliary stage, adult flukes in the extrahepatic bile ducts and gallbladder cause biliary epithelial hyperplasia, hypertrophy, and partially or complete biliary obstruction [34–36]. The obstruction causes acute abdominal pain due to complications such as cholestasis, cholecystitis, cholangitis, and pancreatitis [41–43]. Imaging features of acute abdominal pain due to biliary obstruction

### Table 2  Parasitosis and affected abdominal organs. Associated radiological, clinical, and laboratory findings (Continued)

| Disease type | Imaging findings | Clinical presentation | Lab findings |
|--------------|------------------|-----------------------|--------------|
| **Malara** [14, 60–66] | | | |
| Liver | - Hepatomegaly, periportal edema, perihepatic or intraabdominal ascites - Gallbladder wall thickening and distention due to acalculous cholecystitis | RUQ pain, fatigue and fever, MURPHY sign positivity in case of acute cholecystitis | Anemia, thrombocytopenia, leukocytosis, mildly elevated liver enzymes |
| Spleen | - Areas of decreased attenuation on CT, altered echogenicity on US due to infarction, splenomegaly - Parenchymal or perisplenic hemorrhage/ hematoma in case of spontaneous splenic rupture | LUQ pain Rapid clinical deterioration tachycardia and hemorrhagic shock in case of splenic rupture | |
| Gastrointestinal system (GIS) | - Dilated bowels in case of intestinal obstruction - Rarely, intraabdominal free fluid, gas, and/or fluid collection in case of GIS perforation | Nausea and vomiting in case of GIS obstruction, generalized abdominal pain, fever and sepsis in case ofGIS perforation | |
| Pancreas | - Thickening of pancreas, peripancreatic fat tissue stranding and fluid collection may accompany | Epigastric pain and symptoms related to acute pancreatitis | Anemia, thrombocytopenia, leukocytosis, hyperbilirubinemia, elevated liver and pancreatic enzymes |
| **Visceral leishmaniasis** [16, 67–72] | | | |
| Liver | - Hepatomegaly, ascites, right-sided pleural effusion - Nodular shaped focal parenchymal lesions | RUQ pain, fatigue and fever, Pancytopenia, elevated CRP, hypergammaglobulinemia | |
| Spleen | - Splenomegaly, ascites, left sided pleural effusion - Nodular shaped focal parenchymal lesions - Parenchymal or perisplenic hemorrhage/ hematoma in case of spontaneous splenic rupture | LUQ pain, fatigue and fever, rapid clinical deterioration tachycardia, and hemorrhagic shock in case of splenic rupture | |
| Lymph nodes | - Enlarged and heterogeneous lymph nodes, central cystic changes can be seen in case of necrosis | Painful and palpable peripherally located lymphadenopathy | |
| **Dientamoebiasis** [17, 73] | | | |
| Gastrointestinal system (GIS) | - Bowel wall thickening, mucosal edema, associated intra-abdominal free fluid - Dilated bowels in case of intestinal obstruction | Abdominal pain, diarrhea, anorexia | Leukocytosis, rarely eosinophilia |
| Liver | - Periportal fat tissue stranding and/or edema | RUQ or epigastric pain, fever | |
| Anisakiasis [74–77] | | | |
| Gastrointestinal system (GIS) | - Severe submucosal edema of the involved gastrointestinal area, adjacent fat tissue stranding, enlargement of lymph nodes, intraabdominal free fluid may accompany. Rarely intestinal obstruction due to intussusception | Abdominal pain, nausea, vomiting, diarrhea, signs of peritoneal irritation ileus/intestinal obstruction with or without intussusception | Leukocytosis |

**References**

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by adult flukes include dilated biliary ducts, linear echogenic floating particles in the biliary system on ultrasonography (US), and irregular thickening of bile duct walls, contrast enhancement of duct walls, and intermediate signal filling defects in the dilated ducts on T2-weighted MR images. Imaging features of accompanying micro-abscesses in the liver parenchyma such as heterogeneous echotexture due to focal small hypo- or hyperechoic lesions on US and round or oval clustered hypodense lesions with peripheral contrast enhancement on CT and MRI may be helpful in the differential diagnosis [34, 35, 38, 79]. A large cavitary lesion may occur rarely as a result of reinvasion of the hepatic parenchyma by an adult worm and rupture of the bile duct. As a rare complication of fascioliasis, acute hemobilia related to the bleed-
ing ulcer in the bile duct may be observed [36].

**Ascariasis**

*Ascaris lumbricoides* is the most common and the largest roundworm parasite of the human intestine [2]. *A. lumbricoides* are transmitted through the ingestion of contaminated food. After eggs are dissolved in the stomach to initiate larva formation, the larvae reach the cecum and migrate through the liver via penetrating portal vein branches. They reach hepatic veins via hepatic sinusoids and finally, the right heart and lungs are involved. They travel to bronchi and trachea and may be seen in sputum at this stage. When sputum is swallowed again, they reach the gastrointestinal system where they become adult worms [44].

Patients with ascariasis are asymptomatic or present with nonspecific abdominal symptoms. Acute abdominal pain may occur due to complications [2]. Small bowel obstruction is an expected complication in the massive infestation. Massive worm aggregates may cause bowel infarction and gangrene. The worms have a predilection for the orifices and the ampulla of Vater is frequently involved. Thus, biliary and pancreatic complications are also common [45]. Biliary colic, acute cholecystitis, recurrent cholangitis, liver abscess, and acute pancreatitis are related complications [45, 46]. Rarely the worm causes gastrointestinal perforation [47]. On sonography, the roundworm may be seen as single or multiple, long, linear, and thick echogenic strips without acoustic shadowing [48]. A central, longitudinal anechoic tube between two parallel echogenic lines, known as “triple line sign,” represents the digestive tract of the Ascaris worm [46, 49]. US may reveal the movement of the worm during real-time scanning. Acute intestinal obstruction secondary to ascariasis may present on CT as dilated bowel loops and elongated or rounded filling defects in the contrast filled lumen of the bowel [48]. Ascaris worms manifest as relatively hyperattenuating tubular structures surrounded by less attenuated bile in the setting of bile duct obstruction [48]. Acute pancreatitis caused by Ascaris worms presents on CT as diffusely dilated pancreatic duct and peripancreatic edema. (Fig. 11).

**Toxocariasis**

Toxocariasis is the infection in the human host with *Toxocara canis* or *Toxocara cati*. Their definitive hosts are the domestic dog and cat. Humans (especially children) are accidentally infected hosts when they ingest the eggs from contaminated foods. The larvae hatch in the small intestine, penetrate the wall, and migrate to all organs via the bloodstream (visceral larva migrans) [80]. The liver, lungs, central nervous system, and eyes are the most affected organs. Cardiac involvement may rarely be encountered (Fig. 12). Tissue damage is largely dependent upon eosinophilic inflammation as a response of the host immune system to the dead larvae [80]. Clinically, most of the patients are asymptomatic. Heavy infection can cause fever, abdominal pain, general weakness, weight loss, and pulmonary and neurological symptoms [81, 82]. Liver involvement appears as small, oval, multiple hypechoic lesions on US [81, 82]. On CT, the most common imaging finding of the hepatic toxocariasis is multiple, ill-defined, oval or sometimes angular or trapezoid in shape, low-attenuating nodules along the portal vein branches that are measured less than 2 cm, best seen on the portal venous phase. An enhancing rim may be seen on arterial phase images. When the infection becomes severe, the small lesions fuse to form large lesions, some of which are crossed by portal vein branches (Fig. 12) [81, 82]. Toxocariasis appears as single or multiple lesions with low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Portal phase images after...
intravenous (IV) gadolinium administration are the most helpful method to demonstrate poorly defined low-signal intensity lesions. Concomitant lung lesions may also be seen in severe infection [82]. Cardiac involvement including myocarditis, pericarditis, and Loeffler’s endocarditis may be encountered in rare cases with Toxocariasis [83].

**Intestinal taeniasis**

Human taeniasis is a parasitic infection caused by three tapeworm species, *Taenia saginata*, *Taenia solium*, and *Taenia asiatica*. Humans are the only definitive hosts for these *Taenia* tapeworms. Humans become infected with tapeworms when they eat raw or undercooked beef or pork containing infective cysticerci [50]. Intestinal taeniasis is acquired by ingesting *T. solium* or *Tsasigata* larvae, whereas ingestion of *T. solium* eggs causes cysticercosis [50]. Following *Taenia solium* ingestion, the embryos may penetrate the bowel wall and enter portal circulation. Afterward, they reach distal capillaries (cysticercosis) of richly perfused tissues (central nervous system, skeletal muscle, eyes, and subcutaneous tissue) [18, 50].
Most people with tapeworm infections have no symptoms or mild symptoms. But rare complications such as intestinal obstruction, intestinal perforation, gallbladder perforation, acute pancreatitis, acute appendicitis, and acute cholecystitis may cause acute abdominal pain [51–55]. Adult worms in gallbladder lumen, bile ducts, and pancreatic duct become visible as linear hyperechoic materials on US with dilatation of the involved ducts [53]. Intestinal taeniasis appears as a fine, tubular structure with hyperechoic walls in the intestinal lumen on US (Fig. 15). Taenia worms cause double-reflective, ribbon-like echo in the lumen of the bowels (Fig. 13) [56]. MRCP images demonstrate hypointense linear structures within the dilated intra- and extrahepatic bile ducts in biliary system involvement [59]. The presence of abundant free fluid and free air on imaging studies suggests perforation of luminal organs involved by the parasite [51, 52].
Amoebiasis

Amoebiasis is a parasitic disease caused by Entamoeba histolytica. Humans are the only natural hosts. Ingestion of the cyst from contaminated food or water initiates the infection. The cyst forms the trophozoite stage within the terminal ileum or colon [57]. The trophozoite form causes disease and may spread to the extraintestinal localizations via the portal vein, the lymphatics, or by direct extension through the peritoneum [58].

Most of the infected patients are asymptomatic; however, about 10% may show with clinical symptoms. The right-side colon tends to be more severely involved. The terminal ileum may be involved up to 10% [44]. Acute amebic colitis presents with bloody diarrhea, tenesmus,
and abdominal pain [57, 58]. In amebic colitis, CT may demonstrate wall thickening, mucosal edema, and increased mucosal enhancement (Fig. 14) [44]. Fulminant amebic colitis is characterized by transmural extension of amebic colitis which may result in total colonic gangrene and bowel perforation [44, 59]. Imaging features of fulminant amebic colitis include deep ulcerations, submucosal and intramural tracking of contrast agent, and discontinuous bowel necrosis seen as alternating enhancing and nonenhancing bowel wall [84].

The most common extraintestinal manifestation of the disease is a liver abscess. Symptoms of liver abscess are fever, right upper quadrant pain, and tenderness [57]. Amebic abscesses are predominantly solitary, oval or round shaped, and located near the liver capsule in the right lobe of the liver. Sonographic features of amebic abscess consist of a hypoechoic lesion with low-level internal echoes and the absence of significant wall echoes. On CT scan, it is seen as a low-density lesion with a
peripheral enhancing rim thickened wall. A mild contrast enhancement around the abscess in the adjacent liver parenchyma occurs due to the peripheral zone of edema [85]. Amebic liver abscess is frequently together with right-sided pleural effusion and perihepatic fluid [44].

**Malaria**

Malaria is a serious parasitic infectious disease that is caused by *Plasmodium* species. Plasmodium species are transmitted through infected female mosquitoes that bite humans. These parasites grow within erythrocytes and are released by cyclic hemolysis [60].

The main symptom of malaria is episodic fever [60]. One of the most commonly involved systems during acute malaria is the gastrointestinal tract. Gastrointestinal symptoms are common in children with falciparum malaria. Abdominal symptoms of malaria are vomiting, dyspepsia, diarrhea, abdominal pain, and intestinal hemorrhage [61]. Acute pancreatitis, acalculous cholecystitis, and subacute intestinal obstruction are rare complications of the falciparum malaria. The precise mechanism of these complications is not clearly defined. *Plasmodium falciparum* infection was stipulated to have resulted in microvascular obstruction and consequently result in affected organ ischemia [14]. Rarely perforation can also occur [62]. Splenomegaly is the most common finding on CT examination in patients with gastrointestinal symptoms (Fig. 15). Other findings are hepatomegaly, splenic infarction, spontaneous splenic rupture and hemorrhage, ascites, and periportal edema [60, 63]. In patients with malaria and acute abdominal pain, CT is the mainstay imaging technique in the differentiation between splenic infarction and spontaneous rupture. Discontinuation of splenic contours, heterogeneous enhancement of splenic parenchyma, and perisplenic and intraabdominal free fluid with density levels ranging between 35 and 60 HU that suggests hemoperitoneum and presence of highest attenuation of the intraabdominal free fluid (sentinel clot) nearest to spleen should suggest splenic rupture in patients with suspicion of malaria due to travel history to the endemic areas [64]. Patients with malaria have also been reported to develop non-cardiogenic pulmonary edema and acute respiratory distress syndrome (ARDS) due to increased capillary permeability or endothelial damage (Fig. 15) [65, 66].

**Visceral leishmaniasis**

Leishmaniasis is a vector-borne parasitic disease caused by *Leishmania* species. There are 3 main forms of leishmaniasis: cutaneous, mucocutaneous, and visceral [68]. Persistent irregular fever and splenomegaly are characteristic findings for visceral leishmaniasis. Besides, lymphadenopathy, hepatomegaly, pallor, night sweats, weakness, anorexia, asthenia, cutaneous pigmentation, and weight loss may be seen. Left upper quadrant pain may be caused by massive splenomegaly [68]. Gastrointestinal hemorrhage may be encountered in leishmaniasis due to the involvement of bowels as the duodenum being the most frequently involved segment [69].

Regarding US findings in the patients, splenomegaly, lymphadenopathy, and hepatomegaly are common. Less common sonographic findings include ascites, pleural effusion, portal vein dilatation, and inferior vena cava dilatation. Increased echogenicity of the kidneys and decreased echogenicity of the pancreas may be seen very rarely [70]. On contrast-enhanced CT, hepatosplenomegaly, hypodense nodular lesions...
in the liver, and spleen and lymphadenopathy may be detected (Fig. 16). CT angiography may reveal intraluminal contrast extravasation in cases with gastrointestinal hemorrhage. In the differential diagnosis, lymphoma should be kept in mind due to the above-mentioned mimicking findings [71, 72]. Rarely, splenic infarction and splenic rupture may occur.

**Anisakiasis**

Ingestion of raw or undercooked fish or other seafood may cause a rare parasitic disease, Anisakiasis [75]. There is a controversy in previous reports about the ability of the larvae of genus Anisakis during penetration of the gastrointestinal wall. Some reports indicate that the larva can only be stuck on the gastrointestinal wall
while others emphasize the penetrating ability of the parasite [75–77]. Attachment of larvae in the gastrointestinal wall causes local tissue damage including ulceration, granulomatous inflammation, or perforation while an allergic reaction of the gastrointestinal wall or IgE-mediated systemic allergic reaction may also occur [76]. Gastric involvement forms 90–95% of cases [77]. Symptoms include abrupt onset of abdominal pain, nausea, vomiting, fever in gastric anisakiasis with additional diarrhea, peritoneal irritation, and intestinal obstruction caused by intussusception in intestinal anisakiasis [75, 77]. Diagnosis of anisakiasis with imaging findings solely is usually difficult if one neglects the history of raw or undercooked seafood consumption. US, as a frequently used imaging technique in epigastric or right upper quadrant pain, may demonstrate diffuse concentric wall thickening in gastric or intestinal walls with hypoechoic submucosal edema, irregularity of the lumen surface due to edema of the Kerckring’s folds (corn sign), and accompanying free fluid around involved segment [75].

The features of anisakiasis in CT imaging during gastric and intestinal involvement include gastric or intestinal wall thickening due to submucosal edema, perigastric fat stranding in gastric anisakiasis, and trace ascites. Prompt diagnosis of gastric anisakiasis can be accomplished with endoscopy followed by endoscopic removal of larvae. Management of intestinal anisakiasis depends on conservative medical treatment with anthelmintic drugs such as albendazole [77].

Fig. 12 A 49-year-old woman was admitted to the emergency department with a 1-week history of fever, shortness of breath, and weight loss. Physical examination revealed tachycardia. Increased serum level of acute phase reactants, eosinophilia, and increased level of liver enzymes were evident at blood analysis. a–c Axial contrast-enhanced CT images demonstrate periporal fat tissue stranding (arrowheads, a) and parenchymal hypodensity affecting right liver lobe (arrows, a, b). Areas of decreased parenchymal attenuation scattered at the periphery of the liver were also noted on CT (asterisks, a–c). The presence of complex pleural effusion (black asterisks) containing nodular (arrowheads, b) and linear (long arrows, b) areas of contrast enhancement, was consistent with pleural involvement. Liver biopsy revealed liver injury and the presence of eosinophilic infiltration. CT image at the level of liver dome demonstrates the parenchymal involvement (asterisks, c, d). Sagittal reformatted T1-weighted inversion-recovery MR image obtained 10 min following IV gadolinium-based contrast agent injection, shows biventricular apical thrombus (asterisks) and subendocardial contrast enhancement (arrows) suggestive of Loeffler’s endocarditis. The definitive diagnosis of the Toxocara infection was made by serological tests.
Dientamoebiasis

*Dientamoeba fragilis* is a protozoan parasite of the human bowel. It has been considered for years to be a non-pathogenic organism but more recent reports throughout the world show association of this parasite with gastrointestinal symptoms [86]. The most common symptoms of dientamoebiasis are diarrhea, abdominal pain, loose stools, and anorexia [87]. Acute abdominal pain is a rare symptom [73, 88]. CT may demonstrate nonspecific inflammatory changes due to involvement (Fig. 17).

**Conclusion**

Acute abdominal pain may be seen in parasitic diseases. The diagnosis can be delayed because of the similar clinical signs encountered in other frequently seen causes of acute abdomen. Therefore, parasitic infections should be included in the differential diagnosis of acute abdominal pain particularly in patients from known endemic areas. Awareness of imaging findings in abdominal parasitic involvement is triggering to raise the suspicion for parasitic disease as a cause of acute abdominal pain.
A 39-year-old man was admitted to the emergency department with a 1-week history of mild abdominal pain and fever. Physical examination was unremarkable except for splenomegaly. Blood analysis demonstrated thrombocytopenia. **Fig. 15** Axial contrast-enhanced CT image shows splenomegaly (asterisk). There was no sign of splenic infarction, hematoma, and rupture at the time of diagnosis. 

**b**. CT scan of the chest showed bilateral mild pleural effusion (asterisks, **b**) and centrally located ground-glass opacities (arrows, **c**). The findings raised suspicion for non-cardiogenic pulmonary edema. The imaging findings were not specific for any particular type of disorder. Nevertheless, the final diagnosis was made by a peripheral blood smear examination showing the plasmodium falciparum infection.

A 21-old man was admitted to the emergency department with a 2-week history of left upper quadrant abdominal pain and fever. Physical examination revealed splenomegaly and inguinal lymphadenopathy. Increased serum levels of acute-phase reactants, leukocytosis, and high sedimentation were evident at blood analysis. 

**Fig. 16** Axial contrast-enhanced portal venous phase CT images demonstrate patchy areas of hypoattenuation (arrows, **a**, **b**) within the enlarged spleen and bilateral inguinal lymphadenopathy (asterisks, **c**). The liver was normal except for mild hepatomegaly (18 cm). The diagnosis of Leishmaniasis was made by histopathological examination following splenectomy.
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Fig. 17 A 23-year-old man was admitted to the emergency department with a 1-week history of abdominal pain and diarrhea. Physical examination and blood analysis were unremarkable except for mild abdominal tenderness and eosinophilia, respectively. a, b Axial contrast-enhanced CT images demonstrate the presence of intra-abdominal free fluid (asterisk) and peripancreatic edema (arrows). The imaging findings did not raise suspicion for an individual pathology. There was no definitive proof that this organism was the cause of the symptoms or imaging manifestation. Nevertheless, the patient's symptoms and imaging findings were completely resolved following the treatment for Dientamoeba fragilis which was found on the patient's stool examination.

Abbreviations
CT: Computed tomography; MRI: Magnetic resonance imaging; US: Ultrasonography

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