Helminthic diseases have a worldwide distribution. They affect billions of people in endemic areas and can result in serious clinical complications. Some parasites have a human gastrointestinal life cycle with resultant abdominal manifestations. However, the symptoms of helminthic diseases are usually nonspecific. Radiologic imaging, along with the identification of risk factors, may help narrow the differential diagnosis. To avoid diagnostic delays, radiologists should be familiar with the geographic distribution, transmission cycle, and characteristic and atypical manifestations of common helminthic diseases at abdominal imaging with radiography, computed tomography, magnetic resonance imaging, and ultrasonography. Awareness of the clinical, epidemiologic, and pathogenic characteristics of these diseases also may be helpful for narrowing the diagnosis when imaging features are nonspecific.
Introduction
Helminthiasis is a highly prevalent disease worldwide that is caused by species of Platyhelminthes (flatworms) and Nematoda (roundworms). The disease may be transmitted by the fecal-oral route, active penetration of the skin by larvae from the soil, or vector arthropods. Helminthiasis is endemic in developing countries and places where sanitation is poor, but it also occurs in nonendemic areas because of immigration and travel.

According to the World Health Organization, approximately 2 billion people are affected by helminthic infections worldwide (1). These infections are responsible for high levels of morbidity and mortality, including iron-deficiency anemia, seizures, portal hypertension, and chronic diarrhea (1–3). Children exhibit high infection rates because they usually play in close contact with the soil and may put their fingers in their mouths. Children also are vulnerable to serious complications of helminthic infection, such as malnutrition, anemia, bowel obstruction, and learning disabilities (1). The clinical manifestations of helminthic disease and associated complications often are nonspecific, and familiarity with characteristic imaging features may be helpful in diagnosing and managing these conditions.

The article describes common helminthic diseases in terms of their geographic distribution, routes of transmission, and common and uncommon manifestations at abdominal radiography, computed tomography (CT), magnetic resonance (MR) imaging, and ultrasonography (US). For ease of description, the diseases are grouped according to whether their transmission is by larval penetration (as in schistosomiasis and strongyloidiasis) or the fecal-oral route (biliary parasitic diseases, ascariasis, trichuriasis, cysticercosis, echinococcosis, toxocariasis, and angiostrongylosis).

Schistosomiasis
Human schistosomiasis is a highly prevalent disease caused by flatworms, mainly Schistosoma mansoni, Schistosoma haematobium, and Schistosoma japonicum. An estimated 200 million people worldwide are affected by schistosomiasis. *S. mansoni* infections are prevalent in South America, Africa, and the Middle East. *S. japonicum* infections are frequent in China and the Philippines, and *S. haematobium* infections occur mainly in Africa and the Middle East (Fig 1) (4).

*S. mansoni* and *S. japonicum* live inside the mesenteric veins, and infections manifest as hepatic fibrosis. *S. haematobium* resides in the venules of the urinary bladder, which results in ureteral granulomatous inflammation (4).

![Figure 1. Map shows the world distribution of the three most common Schistosoma species (regions in red): S japonicum, in China and the Philippines; S haematobium, in Africa and the Middle East; and S mansoni, in South America, Africa, and the Middle East. S mansoni is the only species that has been observed to cause schistosomiasis in South America.](image1)

![Figure 2. Diagram shows the life cycle of S mansoni, which begins with the excretion of eggs in human feces (1). If the eggs reach water, miracidia hatch (2) and may infect snails (3). Inside a snail, miracidia mature into cercariae (4). After their secretion by the snail, the cercariae swim until they come into contact with and penetrate the skin of a human host (5). They migrate via blood and lymph vessels to the lungs (6) and liver (7).](image2)

Part of the life cycle of *S. mansoni* takes place inside snails, which are the intermediate hosts. For the larvae to be able to swim to and infect snails, the *S. mansoni* eggs must reach water. The entire *S. mansoni* life cycle can be completed in areas with poor sanitation (Fig 2).

The adult *S. mansoni* worm inhabits the inferior mesenteric vein and the portal venous system, releasing eggs into the blood flow (4).
At this stage, infection may produce intestinal or hepatosplenic manifestations. The hepatosplenic form of disease results in more serious clinical complications than the intestinal form. Advanced-stage hepatosplenic schistosomiasis results in marked morphologic changes in the liver, typical periporal fibrosis, and signs of portal hypertension. Severe splenomegaly with siderotic nodules is common, and observation of these features may aid in diagnosis (5). Isolated intestinal schistosomiasis may be indistinguishable from other forms of proctocolitis (6).

In the hepatosplenic form of schistosomiasis, the parasitic eggs cause granuloma formation and periporal fibrosis (Symmers fibrosis) in the liver (6,7). Fibrosis in turn leads to hepatic morphologic changes such as right lobe atrophy and fissure widening (Fig 3) (8). The presence of the eggs also incites a chronic endothelial inflammatory response, which, in association with portal hypertension, may lead to venous thrombosis. In some cases, parietal calcification may be observed in the portal system.

Typical imaging findings in the presence of periporal fibrosis include gallbladder wall thickening that extends to the porta hepatis and periporal hyperechogenicity, both of which may be observed at US (Fig 4) (7). At MR imaging, periporal fibrosis is depicted as high-signal-intensity bands along the portal tracts on T2-weighted images (Fig 5a). The bands, which have low signal...
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Figure 6. Axial CT image shows exuberant para-esophageal collateral circulation due to thrombosis and cavernous transformation of the portal vein and thrombosis of the splenic vein in a patient with schistosomiasis.

Figure 7. Hepatosplenic schistosomiasis after splenectomy. (a) MR cholangiopancreatographic image shows intrahepatic biliary dilatation and gallstones. (b) Axial contrast-enhanced T1-weighted MR image obtained during the venous phase shows compression of the main hepatic duct with associated cholangiopathy (arrow) due to thrombosis and cavernous transformation of the portal vein.

Imaging findings may aid in the diagnosis of schistosomiasis, but a definitive diagnosis is made when eggs of the parasite are identified at stool specimen examination or rectal biopsy. A positive serologic test result is considered diagnostic in endemic areas (4). Follow-up imaging studies are necessary to detect clinical complications of portal hypertension. Acute disease is treatable with medication. In some cases, surgery is necessary for the control of portal hypertension.

Strongyloidiasis

Strongyloides stercoralis is a roundworm species that infects more than 30 million people worldwide, mostly in tropical and subtropical regions. Strongyloidiasis is endemic in the southeastern United States (Fig 8) (10,11). It is considered...
Figure 8. Map shows the world distribution of *S. stercoralis*, which is endemic in many tropical and subtropical regions of Asia, Africa, and South America and prevalent in the southeastern United States.

Figure 9. Diagram shows the life cycle of *S. stercoralis*, which begins when eggs eliminated in human feces (1) reach the soil (2), where the larvae hatch. At contact with a human host, the infective larvae penetrate the skin (3), migrate to the lungs (4), and ascend the bronchial tree to the esophagus, at which point they are swallowed. In the small intestine, the larvae mature into worms (5). A unique pattern of continuous autoinfection may become established (6).

Strongyloidiasis may manifest as a clinical syndrome involving the skin, lungs, or gastrointestinal tract alone or in combination (10). Such syndromes include acute infection with Loeffler syndrome (pneumonitis during the pulmonary stage of larval development), chronic intestinal infection, asymptomatic autoinfection, symptomatic autoinfection, and hyperinfection (10). Corticosteroid therapy is the most common risk factor for the development of a hyperinfection syndrome, because corticosteroids reduce the levels of circulating eosinophils (10).

The symptoms of intestinal strongyloidiasis vary and may include abdominal pain, diarrhea, nausea, vomiting, and, rarely, malabsorption (10). Imaging findings in patients with the chronic gastrointestinal syndrome are usually nonspecific and may mimic those of other types of intestinal infection. Markers of intestinal strongyloidiasis include mucosal congestion, edema of the intestinal wall with tumefaction of the intestinal folds and flattening and atrophy of the overlying mucosa (Fig 10) (12).

Figure 10. Intestinal strongyloidiasis. Image from a barium study of the upper digestive tract shows diffuse effacement of the mucosal folds in the duodenum and proximal jejunum (arrow). (Courtesy of Luiz Nunes de Oliveira, MD, Instituto da Criança do HCFMUSP, São Paulo, Brazil.)

an emerging global infectious disease because of its increased incidence after immunosuppressive therapy and in transplant recipients (10).

*S. stercoralis* has both free-living and parasitic modes of life (11). Humans become infected either when they ingest the parasitic larvae or when the larvae actively penetrate the skin. The larvae may have a pulmonary development stage (Fig 9). After the larvae reach the small intestine, they mature into female adult worms, which produce eggs through parthenogenesis. The eggs then hatch into larvae (the first larval stage), which are eliminated in feces. If larvae remain in the intestine and achieve the next stage of development, they become capable of invading the intestinal mucosa (11). Thus, *S. stercoralis* larvae may establish a unique chronic pathway of continuous autoinfection in the host (10,11).
Figure 13. Diagram shows the life cycle of *A. lumbricoides*. Eggs eliminated in the feces (1) become embryonate and infective within 18 days. After the eggs are swallowed (2), larvae hatch, invade the intestinal mucosa (3), and migrate through the portal (4) and systemic circulation to the lungs (5), where they ascend the bronchial tree, enter the esophagus, and are again swallowed. In the small intestine (6), the larvae mature into adult worms.
Figure 14. Image from a barium study in a child with abdominal pain shows elongated radiolucent filling defects (arrow). The final diagnosis was ascariasis. (Courtesy of Luiz Nunes de Oliveira, MD.)

Figure 15. US image obtained in a patient with nonspecific abdominal pain shows the digestive tract of a worm as two parallel echogenic lines (arrows). Ascaris was identified at stool analysis. (Courtesy of Miguel José Francisco Neto, MD, Departamento de Radiologia da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil.)

Figure 16. Radiograph obtained in a child with acute abdominal symptoms shows a dilated bowel loop containing an aggregation of thick tubular structures (arrow), a feature that represents an Ascaris worm bolus. (Courtesy of Luiz Nunes de Oliveira, MD.)

formation of intrahepatic biliary stones. Chronic cholangitis and cholangiocarcinoma are associated late complications (14).

Ascariasis

Ascaris is the most common and the largest roundworm parasite of the human intestine (15,16). Approximately 1 billion people worldwide are infected with this parasite (15,17). Ascariasis has a global distribution but is most prevalent in tropical countries, where the temperature and humidity favor the development of eggs in the soil (Fig 12) (16). Ascariasis is transmitted through the fecal-oral route (Fig 13). Respiratory symptoms are common, since the larvae have a pulmonary developmental stage. For example, Loeffler syndrome is a frequent manifestation of ascariasis.

Most of those affected are asymptomatic or present with nonspecific abdominal symptoms. However, because of the high prevalence of Ascaris infection, complications and mortality are frequent (18). On images from barium studies, Ascaris worms may be depicted as elongated filling defects (Fig 14), and the intestinal lumen of the worm may be opacified by the contrast material. At US, too, it may be possible to distinguish the worm’s digestive tract, which appears as two echogenic parallel lines (Fig 15).

Complications such as bowel obstruction are usually related to a massive infestation (15). Intestinal obstruction due to ascariasis is not an uncommon complication in children. Radiographs in such cases show a mass of thick tubular structures inside the bowel lumen (Fig 16). The differential diagnosis includes a trichobezoar.

Pancreatic or biliary manifestations occur when the adult worm migrates from the small bowel into the main pancreatic duct or the biliary tree (Fig 17). Related complications include biliary colic, gallstone formation, cholecystitis, liver abscesses, and pancreatitis (15,17,19,20). Recurrent pyogenic cholangitis is attributable to ascariasis in highly prevalent areas (19).
Figure 18. Diagram shows the life cycle of *Trichuris* trichiura. Eggs eliminated in feces (1) become embryonate in the soil. After the eggs are swallowed (2), larvae hatch in the small intestine and migrate to the cecum and ascending colon (3).

Trichuriasis

*Trichuris* trichiura is the third most common roundworm found in humans. This species, along with *A. lumbricoides* and the hookworms *Ancylostoma duodenale* and *Necator americanus*, infects millions of people worldwide. The world distribution of *T. trichiura* is similar to that of *A. lumbricoides* (Fig 12) (18). Unlike *Ascaris* species and hookworms, *Trichuris* species inhabit the colon, where a massive infestation may lead to dysentery and rectal prolapse (21).

*Trichuris* eggs are transmitted through the fecal-oral route. After their elimination in feces, *Trichuris* eggs become embryonate and infective within 15 to 30 days. Upon ingestion, the eggs release larvae in the small intestine. The larvae mature during their migration to the cecum and ascending colon, where the adult worms attach themselves to the mucosa (Fig 18). Images obtained in a barium enema examination may depict the worms as small elongated filling defects (Fig 19). The differential diagnosis of such findings includes lymphoid hyperplasia (21) and aphthous ulcers. In trichuriasis, as in ascariasis, a definitive diagnosis is achieved when the worms or eggs are identified in stool samples.

Cysticercosis (Taeniasis)

Cysticercosis is caused by the flatworm species *Taenia solium*. The disease is associated with poverty and, because pigs are involved in the parasite life cycle, it occurs in areas where traditional pig husbandry is practiced and people eat pork (Fig 20) (2,3). Cysticercosis is acquired by ingesting *T. solium* eggs (Fig 21), and taeniasis is acquired by ingesting *T. solium* larvae, which are called cysticerci (2,22). The most important clinical manifestation of *T. solium* infection is neurocysticercosis (2,3), which is the leading cause of acquired epilepsy worldwide (2).
Abdominal symptoms of taeniasis that are nonspecific include altered appetite, weight loss, abdominal pain, vomiting, diarrhea, and constipation. The presence of fever and eosinophilia helps narrow the diagnosis (2,22). Unusual manifestations of taeniasis include pleural effusion, ascites, and dyspnea (22). Taeniasis is seldom observed at cross-sectional imaging because of the thickness of the worms. When the cysticerci migrate to the muscles, rice grain–shaped calcifications that correspond to calcified granulomas may be detected (Fig 22). Cysticercous cysts may develop anywhere in the body (22).
A definitive diagnosis of taeniasis is achieved with stool analysis or serologic testing (2,22). The diagnosis of cysticercosis is based on clinical, serologic, and imaging findings. An MR imaging finding of central nervous system cysts containing scolecites is diagnostic (3).

Echinococcosis

Four species of *Echinococcus* are known to be pathogenic to humans. *E granulosus* and *E multilocularis* are the most prevalent species, whereas *E vogeli* and *E oligarthrus* infections occur less frequently. *E granulosus* is found in regions where dogs and livestock are raised together, including the Mediterranean, North Africa, Eastern Europe, South America, the Middle East, and Oceania. *E multilocularis* is found in the Arctic and in Asia, and *E vogeli* is found in Central and South America. The intermediate hosts of *E vogeli* are pacas, whose habitat is in South America (Fig 23) (23–25).

The adult *E granulosus* tapeworm inhabits the intestines of its definitive hosts, mainly dogs and foxes, and releases eggs with larvae, called oncospheres (hexacanths), into feces (24). Sheep are the most common intermediate hosts. After oncospheres are ingested, the larval hatch, penetrate the intestinal mucosa of the intermediate host, and migrate to the visceral organs, where they form cysts (the metacestode stage). When the definitive host eats the viscera of the intermediate host, the *E granulosus* life cycle is completed (25). Humans may become incidental intermediate hosts by ingesting *E granulosus* eggs (Fig 24) (24,25). The clinical manifestations of *E vogeli* infection in humans are similar to those of *E granulosus*. Epide-
miologic data and immunohistochemical and histologic analyses are useful for differentiating these two entities (23).

When a human ingests oncospheres, the larvae may migrate from the intestines to other organs, such as the liver, spleen, and lungs, where they form hydatid cysts. The hydatid cyst consists of the endocyst, where the scoleces are produced, and the pericyst, which is composed of host cells that form a peripheral layer of reactive fibrous tissue (Fig 25) (23–25). Daughter cysts develop from the inner germinal layer.

Imaging features vary according to the stage of cyst development: In the early stages, Echinococcus cysts have the appearance of simple cysts; after daughter cysts develop, multiseptate wheel-like structures are visualized. When the parasite dies, the septa disintegrate and, finally, wall calcification appears (Fig 26) (26).

Echinococcosis symptoms are usually related to hydatid cyst enlargement (23,26). Abdominal pain or palpable masses are common manifestations (Fig 27). The liver is the organ most frequently affected, but the spleen, lungs, brain, and kidneys also may be involved. Complications of hydatid cysts include bile duct compression (Fig 28), biliary communication, and peritoneal seeding and rupture (Fig 29), which may lead to fever, pruritus, and anaphylaxis (26).
Clinical diagnosis of echinococcosis is based on epidemiologic data, the depiction of cysts at radiologic imaging, and the results of immunologic and other laboratory tests, which may include analysis of fluid from a cyst puncture if percutaneous needle aspiration is not contraindicated (23,24).

Toxocariasis

Toxocariasis in humans is caused by the larvae of *Toxocara canis* or *Toxocara cati*. In humans, the larvae do not develop into adult worms, but migrate through host tissues; therefore, the disease is also called visceral larva migrans (27). Toxocariasis is distributed worldwide, and humans can be accidental hosts (Fig 30).

The larvae induce eosinophilic inflammation in tissues (28). The most common clinical features of the disease in humans are peripheral eosinophilia, abdominal pain, hepatosplenomegaly, fever, and hypergammaglobulinemia (27,28). The abdominal imaging findings of toxocariasis are nonspecific and related to abscess and granuloma formation (Fig 31). Abscesses and granulomas may lead to complications such as endophthalmitis, hepatomegaly, and brain disturbances (28,29). The differential imaging-based diagnosis includes fascioliasis, ascariasis, ancylostomiasis, and angiostrongylosis. The final diagnosis is usually based on the results of serologic assays (28).

Angiostrongylosis

Angiostrongylosis is caused by *Angiostrongylus costaricensis* and occurs mainly in Central America, with sporadic cases found in South America (Fig 32). The parasite is normally transmitted from snails to rats. People may become incidental hosts when they ingest contaminated food, but they do not transmit the parasite because its eggs are not eliminated in human feces (Fig 33).

The adult worm resides inside mesenteric artery branches, where its presence evokes an inflammatory response; marked eosinophilic vasculitis is commonly found in tissues samples. *A. costaricensis* may migrate through the lymphatic system to reach the mesenteric veins and the hepatic portal venous system; therefore, it also may be found in the liver (Fig 34a) (30).

Abdominal pain, anorexia, eosinophilia, and fever are common clinical manifestations of the disease (30). The terminal ileum, appendix, and cecum are the bowel segments most often affected by angiostrongylosis, which produces bowel wall edema (Fig 34b) and, occasionally, perforation. However, the imaging findings are nonspecific. The diagnosis of angiostrongylosis is based on histopathologic findings of tissue eosinophilia, edema of the intestinal wall, and...
granulomas with eggs and larvae within blood vessels (Fig 34c) (31).

**Summary**

Helminthic diseases have a worldwide distribution, with increased prevalence in areas with poor sanitation. Although these diseases occur much more frequently in developing countries, sporadic cases occur in nonendemic areas. Children are at an increased risk of infection because of their play habits. Prevention of helminthic diseases mainly involves the provision of high-quality water supplies and sanitation. In addition, education about hygiene is a powerful means for decreasing infection rates.
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Knowledge of epidemiologic factors, such as living conditions and occupational and environmental exposures (eg, animal husbandry, travel), and data from laboratory testing are essential to narrow the differential diagnosis. Familiarity with typical radiologic patterns may help determine specific diagnoses (Table). When imaging findings are atypical but a patient has eosinophilia and risk factors, a diagnosis of helminthic infection should be considered.

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References

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Helminthic Diseases in the Abdomen: An Epidemiologic and Radiologic Overview

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