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The role of radiological imaging in the identification of abdominal parasitoses

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Abstract

Background. Various parasites are found in the human body. Some of them infect the human body and form a symbiotic relationship with it. Other parasites cause serious and even fatal complications. The most frequent abdominal parasites include Echinococcus spp., liver flukes, and Entamoeba histolytica.

Aim: to review information on current radiological imaging possibilities to identificate abdominal parasitoses as reported in the literature.

Methods. Literature sources were selected from PubMed medical database using these keywords: radiological imaging, abdominal parasitosis, echinococcus granulosus, echinococcus alveolaris, liver flukes, entamoeba hystolitica.

Results. Ultrasonography, computed tomography, and magnetic resonance imaging (MRI) are the most often utilized imaging technologies in diagnosis. Furthermore, in the majority of instances, serological testing is required to confirm the diagnosis.

Conclusions. Parasitic diseases are highly prevalent worldwide and cause severe infections or death. Radiological diagnostic tests and accessible serological testing are crucial in the identification of parasitic diseases.

Keywords: radiological imaging, abdominal parasitosis, echinococcus granulosus, echinococcus alveolaris, liver flukes, entamoeba hystolitica.

1. Introduction

Parasitic diseases are among the most severe and widespread infections in the world, causing millions of morbidities and deaths each year. Although parasitosis can affect practically any organ in the human body, the majority of infections result in liver damage. Parasites in the liver cause a wide range of diseases and mortality by inducing recurrent cholangitis, cirrhosis, liver failure and cancer. The most frequent orally transmitted parasites that invade the liver following mucosal penetration or by portal-venous blood flow include *Echinococcus spp.*, liver flukes, and *Entamoeba histolytica*. However, the proper identification of a hepatic parasite is frequently delayed due to clinicians unfamiliarity with the corresponding organisms [1,2].

The liver has a unique feature of immune status, that it can promote tolerance rather than immunity. This function can be used by the parasites to avoid host immunity [2]. Hepatic immune tolerance is caused by a unique combination of anatomical and histological properties of the liver. The liver receives practically all of its blood supply directly from the portal system, making it the first organ to be exposed to gut-derived compounds such as innocuous bacterial metabolites and nutrients [3]. The liver provides the most scavenger cells in the body, primarily Kupffer cells (KCs), liver-resident macrophages, and liver sinusoidal endothelial cells (LSECs). KCs eliminate larger blood-borne particles via phagocytosis, whereas LSECs remove small particles (< 200 μm) and macromolecules via receptor-mediated endocytosis. Both of these cell types are essential for the liver's barrier effectiveness and blood filtering ability. Many of the eliminated elements are potential inflammatory inducers, but their removal by liver cells is often not associated with inflammatory immune responses. Instead, immunological responses are frequently repressed by multiple mechanisms, known as immune tolerance in the liver. Although liver tolerance is presumably an essential response to its frequent exposure to a variety of food and microbial antigens, it is one of the mechanisms that enable parasite invasion in liver cells [3,4].

2. Methods

The PubMed database was used for the literature review. Publications were collected in June and July of 2023. Keywords used: "radiological imaging", "abdominal parasitosis", "echinococcus granulosus", "echinococcus alveolaris", "liver flukes", "entamoeba hystolitica".

3. Results

3.1. Echinococcus spp.

3.1.1 Echinococcus granulosus

Cystic Echinococcosis (CE) or hydatid disease (HD) is triggered by infestation with metacestodes (larval phase) of *Echinococcus granulosus* tapeworm. Human echinococcosis is a zoonotic secondary illness [5–10]. *E. granulosus* is a common parasite in certain regions of the globe, and it can be found on every continent except Antarctica. Consequently, CE impacts a significant number of individuals [5,10].

When carnivores consume the entrails of contaminated intermediate hosts, such as pigs or sheep, they frequently become infected with *Echinococcus granulosus*. The gravid proglottids, which are eventually eliminated in the infected animal's feces, are released by the parasite once it has entered the small intestine and adheres rigidly to the mucosa. Each proglottid contains a number of eggs that can be consumed by intermediate hosts. As seen in sheep that become infected after grazing on grass contaminated with dog excrement containing the eggs, the eggs develop into cysts and offspring cysts in these hosts. Handling or eating infected

sheep's meat or organs carries no risk of human infection [5]. Humans are accidental intermediate hosts who become infected either by direct contact with a dog contaminated with egg-bearing excrement or through ingestion of contaminated water, food, or soil [5]. The asymptomatic incubation phase, during which ingested eggs release oncospheres that are capable of penetrating the human intestinal wall, is the first stage of infection. These oncospheres can access the liver, lungs, and other organs by penetrating the portal venous system. Then the oncospheres start to develop cysts, which can have a diameter between one and fifteen centimeters. Cysts are most commonly found in the liver (70 %) or lungs (20 %), 10 % of cysts, however, can be discovered anywhere in the body, including the spleen (6 %), heart (2 %), kidney (2 %), and brain (2 %) [5,7,8,11] - with the exception of hair, teeth, and fingernails, it can affect nearly any organ in the body [8].

Hydatid cysts may be unintentionally found during radiologic workup. Because of the gradual growth and development of cysts, as well as the immune system's reaction, CE might go undiagnosed for a long period of time [5,10], because the majority of cases are asymptomatic for years [11]. In the early stages of the disease, patients may either be completely asymptomatic or exhibit vague signs and symptoms [13]. The symptoms of cystic echinococcosis differ depending on where the cyst is located.

The liver is the most affected organ in the body because it acts as the first filter for portal venous blood and obstructs roughly 75 % of ingested embryonated eggs. While hydatid cysts of the liver are often asymptomatic and found incidentally on medical imaging, they might cause symptoms due to cyst expansion, which can cause hepatomegaly or the host's inflammatory reactions. Hydatid cyst infection, biliary duct fistula, and rupture into the peritoneum or chest are the most common consequences [10].

Patients with intra-abdominal CE present with symptoms late in the course of the illness [10]. Depending on their size and location, cysts may eventually put pressure on surrounding tissues, causing stomach pain and discomfort [5,9,10]. Despite being a non-malignant ailment, the manifestation of hydatid disease can result in elevated morbidity and mortality rates [7]. Cyst rupture or leakage can cause immunologic symptoms because it triggers an immunoglobulin (Ig)E response, which can cause allergic reactions characterized by hives, flushing, and swelling of the mucous membranes. Anaphylactic shock can be lethal if there is a major rupture [5]. As per the research conducted by the World Health Organization in 2010, the fatality rate for human echinococcosis is 4 %, where the majority of deaths are caused by anaphylactic shock induced by cyst rupture [7]. Prompt diagnosis and therapy are required to avoid potentially fatal complications such as anaphylactic shock or pressure effects on essential organs [10,11].

Imaging and serologic testing is used to diagnose CE. Simple serum studies typically have low sensitivity associated with undetected immunological responses, making diagnosis challenging. About 30–40 % of patients have no antibodies at all, even in those with circulating parasite antigens. Low sensitivities of serum liver enzyme assays make them typically unreliable in determining the underlying severity of the infection [5]. Complete blood count tests might be helpful for these patients because they can have eosinophilia [5,10,12].

The field of medical imaging holds great significance in both the identification of a hydatid disease and determining the extent of its progression [12]. Depending on the cyst's stage, different

imaging results are obtained. In up to 30 % of CE cases, calcification may be seen on radiographs. During the CE process, calcification can happen at any point. The pericyst is where calcifications are typically deposited. Once a cyst has fully calcified, the pathogen is considered to be dormant or dead [5].While chest radiography is widely used as the main diagnostic tool, it is insufficient for evaluating the complications and progression of hydatid disease [8].

The detection of CE is now commonly done through ultrasound. The precision of ultrasound diagnostics can go up to 90 %, contingent upon the proficiency and skill of the user. This method is presently the preferred choice for screening due to the convenience and availability of the device, even in small, remote medical centers. Because of its lack of radiation, exceptional precision for identification, distinguishing between ailments, categorization, and screening to evaluate the pervasiveness of hydatid cysts in the abdomen, ultrasound scanning is the preferred option [10]. Ultrasound is not only valuable for diagnosis but also for tracking post-treatment progress [5,10]. Due to its potential to provide comprehensive information about the mass, including a more accurate assessment of its dimensions, exterior characteristics, the presence of a secondary cyst, and other pertinent anomalies in the area, ultrasound is particularly responsive [12]. Inside the hydatid cyst, an ultrasound can clearly show the hydatid sand, membranes, daughter cysts, and vesicles. It has 96 % sensitivity and 98 % specificity in distinguishing hydatid cyst of the liver from simple liver cysts [10]. Its inability to differentiate between hydatid disease and other conditions is one of its weaknesses [12]. While ultrasound is a superb means of preliminary diagnosis, sonography can sometimes fall short due to various factors such as obesity, excessive intestinal gas, and past surgical procedures [5].

CT has a sensitivity rate close to 94 % and is essential for detection [5]. Particular signs include wall calcification, detached inner layer, and daughter cysts (a defining characteristic). A CT scan is still a more accurate examination because it can confirm the size and location of the lesion as well as any relation or involvement with neighboring tissue. If surgical intervention is intended to be used to ascertain the parenchymal involvement of the cyst within the tissue, this can be especially helpful during the preoperative planning stage. CT imaging is important because it allows for a clear view of cystic lesions and calcification, which might appear as a thick, unilocular or multilocular heterogeneous cyst or the presence of low-density daughter cysts [12]. Calcifications, daughter cysts, and detached membranes are all uncommon hydatid cyst features [7,8]. Observations made through imaging in cases of cystic echinococcosis vary from cystic formations to formations that seem to be solid. The cyst may show up as a fluid collection that is clearly defined. Often, the endocyst may detach from the pericyst, leading to the appearance of a membrane that appears to be floating [9].

Although MRI plays a role in identification, the main limitation of using this technology is its exorbitant cost, especially when there is a chance of reaching the diagnosis using less expensive ultrasound or CT imaging [12]. While it isn't always necessary, MRI may offer additional information not visible on CT [5] - it is more effective at exhibiting the correlation with neighboring flexible tissues and contents of the cyst and daughter cysts [8].

Depending on the growth stage, a hydatid cyst can manifest in various dimensions and configurations, resembling both benign and malignant tumors, thereby presenting diagnostic complexities on certain occasions. When making a differential diagnosis for any cystic lesion, keep a hydatid cyst

in mind [7,8]. Hydatid liver cysts must be considered as part of the differential diagnosis as they can mimic a variety of liver lesions, such as choledochal cysts, Caroli's disease, hemangioendotheliomas, mesenchymal hamartomas, teratomas. The imaging features of hydatid cysts are different and critical for diagnosis. Hydatid cysts that are discovered in unexpected places could be misdiagnosed, leading to ineffective treatment and potentially fatal complications [11]. Radiological imaging can thus only be used to differentiate hydatid cysts from other infected or malignant tumors. Hydatid cysts are infrequent, although they might produce unexpected effects and symptoms in rare cases [7].

Radiology is critical in both getting an accurate diagnosis and offering assistance during the treatment planning phase [8,12].

Figure 1. *Echinococcus granulosus (Hydatid disease) cystic lesions viewed in ultrasonogram [13].*

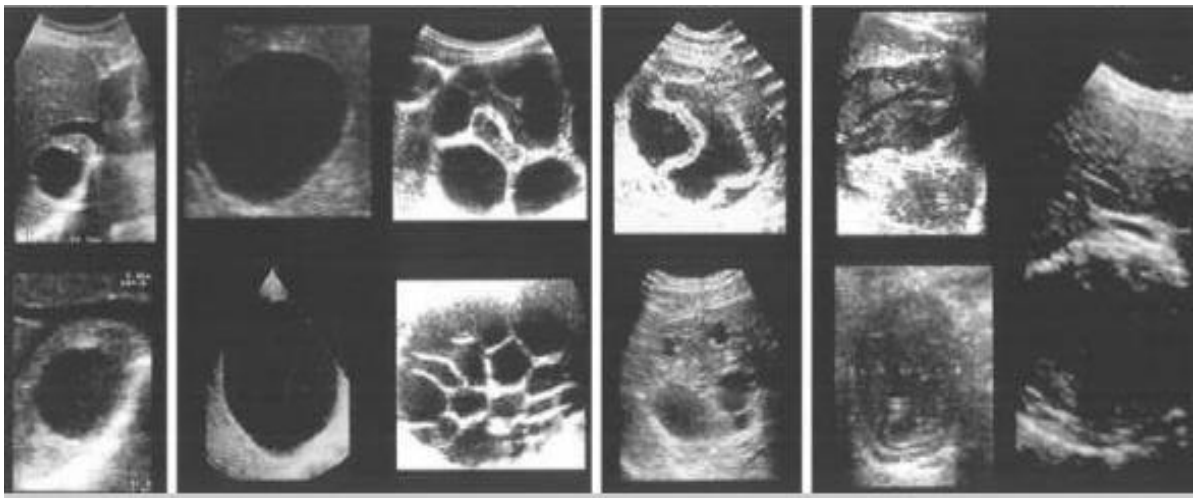
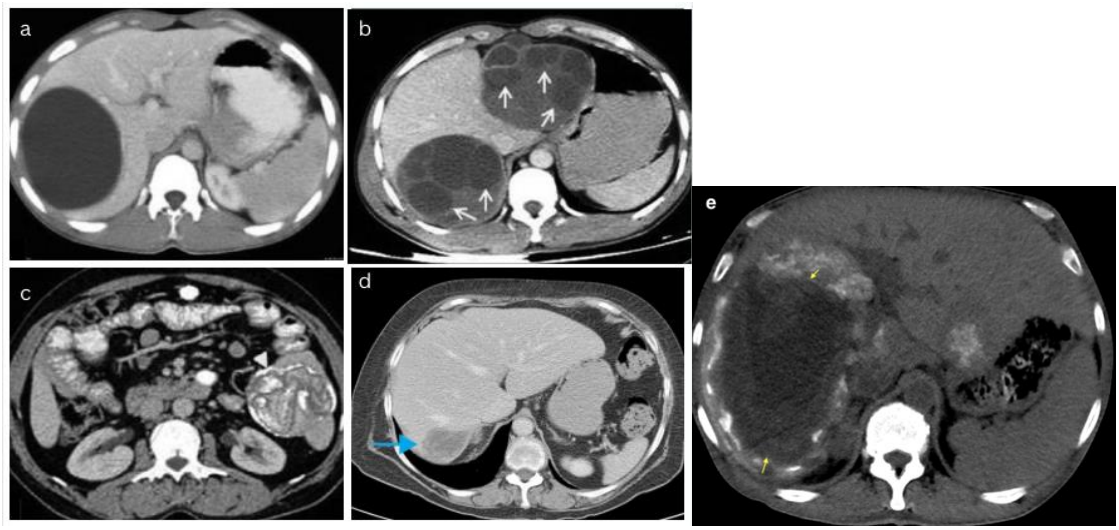


Figure 2. *Contrast-enhanced computed tomography of the abdomen show unenhanced hypodense mass with well-defined borders and no internal architecture without septa (a). Contrast-enhanced computed tomography of abdomen (b) shows liver hydatids with multiple daughter cysts (arrows). Contrast-enhanced computed tomography of the abdomen shows splenic hydatid cysts (c) well-defined lesions with peripheral wall calcification and membranes appear as lamellated calcification (arrow head). Complex fluid collection with curvilinear densities (blue arrow), consistent with detachment of the laminated membranes of the endocyst from the pericyst of a hepatic hydatid cyst (d) [14]. [13]. The CT scan (e) reveals a patient from Kaunas Clinics with echinococcus granulosus cysts in the liver.*



3.2 Echinococcus alveolaris

Echinococcus multilocularis life cycle involves two separate mammalian organisms, the definitive host and the natural intermediate host. In the life cycle of *E. multilocularis*, foxes are the definitive hosts while rodents serve as intermediate hosts. The fox releases the eggs that the adult parasite produces into the environment, and the cycle is then completed when the intermediate host digests tainted food. Humans are either unintentional or atypical intermediate hosts. The invasion of different organs by metacestodes, primarily the liver and lungs, causes severe problems. Alveolar echinococcosis is caused by cestodes of *Echinococcus multilocularis*. AE is a manifestation that can only be observed in the northern hemisphere [15,16]. Echinococcus multilocularis metacestodes have the ability to infect multiple organs in people and exhibit neoplastic-like growth and infiltration patterns. AE is also referred to as "worm cancer" - it is easily mistaken for cancer because it can metastasize to distant organs or invade nearby ones [16]. Furthermore, because the condition is uncommon, particularly in non-endemic locations, diagnosing it can be difficult [15,16].

The illness typically progresses with an asymptomatic incubation period lasting 5-15 years, followed by a chronic phase. In more than 33 % of cases, the condition is identified by chance. The clinical symptoms displayed depend on the individual organ affected and the amount of the invasion. Other abdominal organs are damaged by the direct invasion or metastatic spread. There have been reports of invasions of the diaphragm, perirenal region, abdominal lymph nodes, peritoneum, mesenteric tissues, spleen, pancreas, adrenal glands, kidneys, gallbladder, retroperitoneum, abdominal wall, and stomach. Primary organ involvement outside of the liver is extremely rare. The lungs are the organs that are most frequently involved in secondary AE [15].

The larval stage typically manifests in the liver, inducing infiltrative mass lesions with numerous vesicles that range in diameter from submillimeter to 20 cm [16]. The most common symptoms of liver invasion are jaundice, pain in the upper abdomen, exhaustion and weight loss. The transition from healthy tissue to diseased tissue is indistinct, and the masses are further characterized by diffuse fibrosis, calcified foci, and necrotic regions, primarily located in central zones. The infiltration of the biliary ducts and vessels, combined with tissue death at the core of the injury, can result in critical issues such as bile duct inflammation, abscesses in the liver, portal vein hypertension, Budd-Chiari syndrome, biliary cirrhosis, or additional infections, all of which can increase morbidity or mortality. AE is extremely harmful to the human body, damaging the liver in 100 % of cases and causing substantial damage to hepatic functioning. Lung involvement is typically discovered by chance. The primary symptoms include hemoptysis, dyspnea, coughing, and chest pain [15].

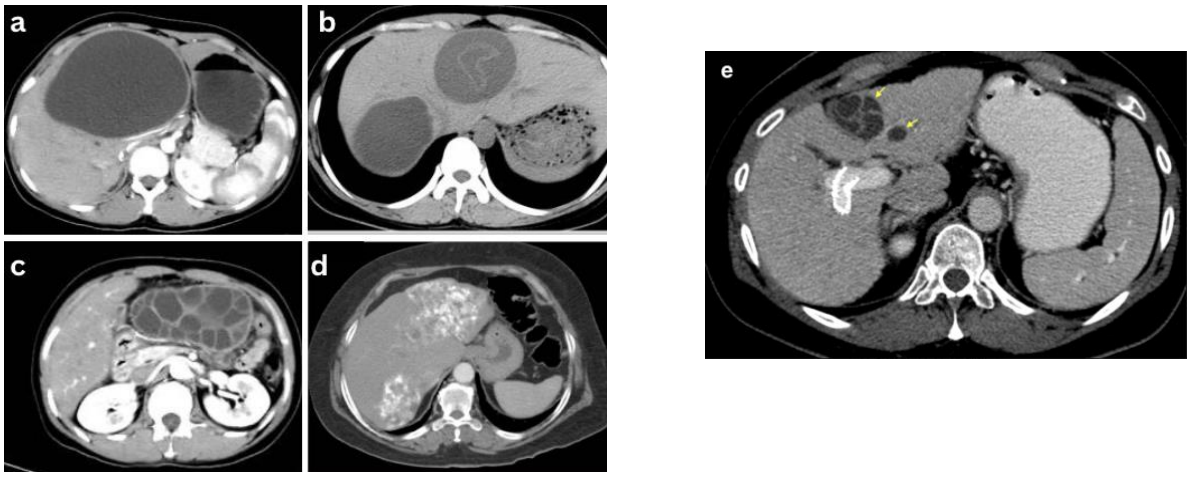
AE is a chronic tumor-like condition that, if untreated or treated insufficiently, can be fatal. More than 90 % of patients with untreated or insufficiently treated AE die within 10 to 15 year [15,16]. Death can result from problems with the hepatobiliary system and associated infections, secondary biliary cirrhosis and related complications, issues caused by vascular structure involvement, and invasion of distant organs. Primary involvement beyond the liver is exceedingly uncommon (1 % of cases), with direct invasion of other intra-abdominal organs or secondary spread to more distant organs such as the brain, lungs, or bones via hematogenous or lymphatic pathways [15,16]. Multi-organ invasion is observed in 13 % of cases [15].

While radiologic imaging techniques are important, the diagnosis must be validated by further support of histopathologic verification or detection of parasite

nucleic acid in the clinical sample. For some years, conventional imaging modalities such as x-ray, ultrasonography (US), computed tomography (CT), and magnetic resonance imaging (MRI) have been used in the diagnosis, follow-up, and therapy of AE. New investigations on the diagnosis and follow-up of the condition using contrast-enhanced ultrasonography (CEUS), diffusion-weighted imaging (DWI), magnetic resonance spectroscopy (MRS), and positron emission tomography-computed tomography (PET-CT) have recently been published [15].

Ultrasound is often the chosen imaging modality for diagnosis and follow-up. It is an appropriate and efficient imaging approach for detecting AE lesions. When there are irregular contoured mass lesions, a mixed heterogeneous echogenic pattern, cystic necrotic areas, and multiple scattered calcific foci, the liver is involved. A less typical sonographic appearance is a hailstorm pattern with several hyperechogenic solid lesions. This look is thought to represent early AE lesions. Radiologists in endemic areas must precisely identify this less common presenting form, which might be confounded with venous malformations and other lesions of a similar character [15].

Figure 3. *CT scan (a) reveals a single giant echinococcosis cyst in the liver. CT image (b) shows CE of the detached inner capsule type: the inner capsule is folded into a floating band symbol after being deflated and separated. CT image (c) reveals multi-daughter cysts are visible inside the big cyst. The CT image (d) acquired after intravenous contrast shows two hepatic masses with unclear borders and diffuse hyperdense foci of coarse calcification [13]. The CT scan (e) reveals a patient from Kaunas Clinics with echinococcus alveolaris cysts in the liver.*



The most effective imaging modality for demonstrating the distinctive morphologic aspects of AE lesions in intra-abdominal organs is CT. It is helpful in determining how the hepatic lesions interact with the bile ducts and vascular systems. It is also possible to demonstrate involvement of extrahepatic organs. AE lesions resemble tumors and have erratic borders, diverse internal structures, and numerous, dispersed calcific foci. The affected liver lobe exhibits atrophy and capsular retraction as a result of vascular and biliary involvement [15].

The best imaging technique for identifying the different parts of a parasite lesion and showing invasion of the vascular and biliary structures is magnetic resonance imaging (MRI). Since MRI is so helpful in revealing expansion to nearby organs, it should be used in preoperative imaging. The typical MRI finding of AE in the liver is a mass lesion with infiltrative characteristics, uneven boundaries, internal heterogeneity, and necrotic regions in the center. The inner edge of the fibrous zone appears as a slightly hyperdense signal with localized invagination forming a peninsula sign and internal necrosis and liquefaction forming a fluid retention

cavity. These exhibit hypo- or isointensity on T1-weighted images and hypo-, iso-, or hyperintense signal characteristics on T2-weighted images [15].

Particularly in nonendemic areas, AE may be easily misdiagnosed by inexperienced doctors as a metastatic malignant tumor [15,16]. The radiologist must be knowledgeable about the multimodality imaging findings of this disease. Ultrasonography can be used for scanning as well as for the initial imaging process. Additionally, ultrasound can direct the interventional techniques. The use of CT and MRI is essential for making sure the right diagnosis is made, for organizing surgical procedures, and for monitoring patients with AE. Radiology plays a crucial role in assisting the clinician in making an early diagnosis and selecting the best course of action [15].

3.3 Liver flukes

Fascioliasis is a parasitic infection of the hepatobiliary system resulting from digenean flatworms known as liver flukes, *Fasciola hepatica* or *Fasciola gigantica*. Whenever parasites infiltrate the liver, the bile ducts may become obstructed. *F. gigantica* survives in tropical climates, meanwhile *F. hepatica* is mostly found in temperate climate regions. Mature *Fasciola hepatica* flukes are around 30 mm by 15 mm in size and are visible with the human eye. Parasites have two suckers, including a big one on the ventral side known as the acetabulum, which allows the fluke to attach itself to the bile duct wall and stay in position, while the smaller anterior sucker feeds from the fluid inside. *Fasciola gigantica* flatworms, as the name implies, can grow to be 75 mm long but resemble smaller versions [6]

The worldwide count of *Fasciola* infection is believed to be between 2.4 and 17 million, with most cases going unreported and undiagnosed. *Fasciola hepatica* is indigenous to Europe and Asia, with infrequent occurrences appearing in Northern Africa, Central and South America, and the Middle East, as well as the United States and the Caribbean. Domestic livestock in Asia, the Pacific Islands, and areas of Northern Africa are infected with *Fasciola gigantica* [6,17].

The life cycle began when humans and other fish-eating mammals, the definitive hosts, transferred eggs into surroundings via their feces. When the eggs appear in freshwater, the miracidia infect their first intermediate host, a freshwater snail, and change into sporocysts, rediae, and cercariae. Cercariae elude the snail and infect freshwater fish, the second intermediate host. The cercariae encyst as metacercariae in the muscles or under the scales and can infect humans through the consumption of undercooked or raw cyprinoid fish products. The metacercariae enter the human small intestine uninjured and move until they reach the bile ducts, where they grow into adult worms and deposit eggs within 4 weeks. The parasites can live in the human liver for up to 25 years [6].

The migration of metacercariae induces parenchymal liver damage, triggering a chain reaction of inflammatory and immunological responses that results in acute symptoms. Adult flukes can clog the bile ducts either entirely or partially, causing fibrosis, hypertrophy, and eventually dilatation of the proximal biliary system. The level of liver damage is often positively associated with parasite load [17].

The fascioliasis infection has two different phases. The acute (hepatic) stage typically begins 6 to 12 weeks after ingesting metacercariae from polluted water. The first symptom is frequently a high temperature, discomfort in the right upper quadrant, hepatomegaly, and jaundice. The presence of peripheral eosinophilia can be detected by a complete blood count (CBC). Myalgias, urticarial rash, nausea, anorexia, and diarrhea are common symptoms. The above symptoms are caused by

Fasciola flatworms spreading into the liver parenchyma and triggering inflammatory and immune system reactions. Most acute signs disappear after 6 weeks, and the infection expands to a chronic stage [17]. However, extrahepatic signs such as reactive eosinophilic pneumonitis, vasculitis, myocarditis, cerebral vasculitis and generalised lymphadenopathy may also be present [17,18]. Once the flukes are located inside the bile ducts, the chronic (biliary) phase can remain for up to a decade. This stage is normally asymptomatic, although it can cause chronic epigastric and right upper quadrant pain, nausea, vomiting, diarrhea, hepatomegaly, and jaundice. Chronic common bile duct obstruction can cause recurrent jaundice, cholelithiasis, pancreatitis, and, more dangerously, ascending cholangitis. Chronic biliary cirrhosis, sclerosing cholangitis, and even cholangio-carcinoma can arise from long-term infection and/or a high parasite load [17,18].

Fascioliasis is a pretty rare illness, but it should be evaluated in any individual who has abdominal pain, fever, pruritus, skin rashes, dyspepsia, vomiting, nausea, transaminitis, and significant peripheral eosinophilia. If a patient has gone to endemic locations and their food habits include watercress intake or eating of raw vegetables washed in possible contaminated water, suspicion must be raised [19].

Misdiagnosis and late diagnosis are still common since the disease is rarely observed in nonendemic locations and has an unclear clinical appearance. Hepatobiliary fascioliasis in humans is diagnosed with direct parasitological tests, indirect immunological tests, and imaging methods like ultrasound, CT, and MRI. Radiological tests are useful in differential diagnosis when liver flukes are suspected, as is the history of recent travel to the endemic region [19]. On CT imaging, the leading sign of hepatic fascioliasis is groups of irregular ill-defined darkish patches, mainly immediately under the liver surface, which may progress to inner tissue and areas around the bile duct. Liver parenchyma can reveal nodular, tiny, branching, and subcapsular lesions - migrating parasites leave these wounds. Necrosis may be visible during intravenous contrast scanning. It may be possible to see subcapsular hematoma, capsular thickening, or parenchymal calcifications as well [15,19].

MRI is preferable to CT imaging because it can detect a greater number of lesions, also it may show the disease's typical evolutionary pattern, representing its life cycle during the early parenchymal phase, even without the use of a contrast agent. Furthermore, in fascioliasis, MRI can provide more detailed information regarding the development of complications such as hemorrhagic lesions and abscess formation [9,19].

Sonographic findings in hepatic fascioliasis are frequently nonspecific. During the parenchymal phase, they include focal hypoechoic or anechoic liver lesions and diffuse liver involvement (heterogeneous echotexture). During the ductal phase of ultrasound, ductal thickening, intrahepatic or common bile duct dilatation, and tortuousness are frequently seen due to intraluminal parasites or the hemorrhage/inflammatory response they cause. It might even show a mobile fluke in the gallbladder or dilated bile ducts [19].

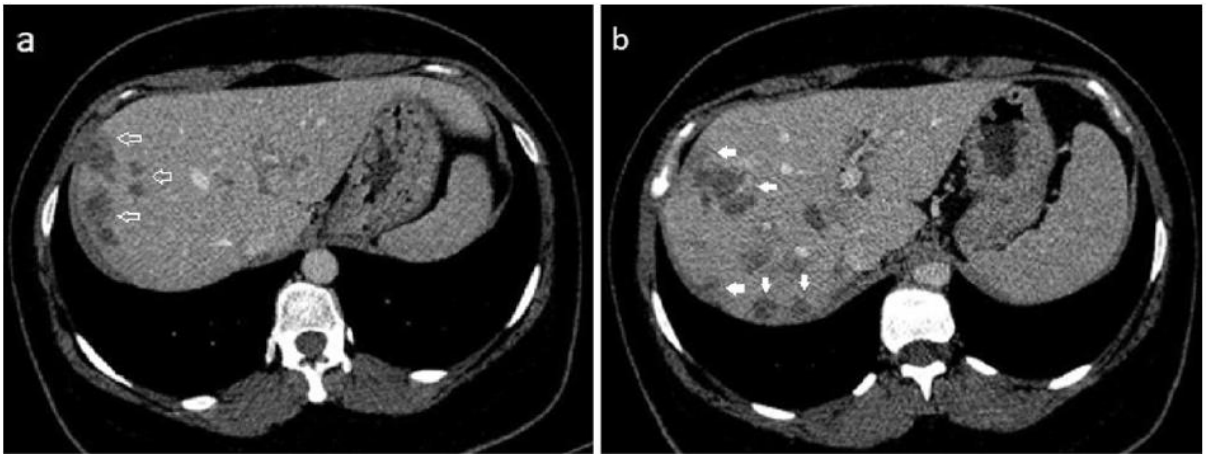
During the biliary stage, ultrasound and cholangiogram can detect mobile, leaf-like flukes in the biliary ducts or gallbladder, which are frequently linked with stones. Abnormal common bile duct wall thickening, hepatomegaly, splenomegaly, and/or periportal lymphadenopathy are frequently observed, particularly during the acute stage [19,20].

However, in recent years, serology has emerged as the quickest and most efficient method of testing for fascioliasis. Titers against *Fasciola* antigens become positive during the early stages of parasite migration

and are detectable for 2 to 4 weeks after initial immune system activation [20].

Identification of *Fasciola hepatica* tends to be challenging, especially in areas where it is not endemic, because clinical and laboratory signs are non-specific. Cross-sectional imaging is commonly used to evaluate probable causes of these confusing findings. On CT/MRI scans, clusters of twisted subcapsular lesions with exterior contrast improvement that extends into the deeper parenchyma and periportal regions are significant signs that strongly suggest hepatic fascioliasis. When relevant radiological indicators are discovered, the potential of hepatic fascioliasis must always be considered in a proper clinical context [19].

Figure 4. *Contrast-enhanced CT picture (a) with patchy areas of decreased attenuation migrating from the liver capsule into deeper parenchyma and periportal regions with modest peripheral contrast enhancement (arrows). Contrast-enhanced CT-image (b) revealing clusters of hypodense lesions extending from the liver capsule into deeper parenchyma and periportal regions with slight peripheral contrast enhancement (arrows) [21].*



3.4 Entamoeba histolytica

The most prevalent cause of liver abscess in tropical countries is amebic liver abscess (ALA), a parasite illness caused by the protozoan *Entamoeba histolytica*. Amebic liver abscess (ALA) occurs in 3-9 % of amebiasis patients, with complications occurring in 20-40 % of cases and a death incidence of 2-18 % [22]. After malaria and schistosomiasis, it is the third leading parasite cause of mortality, killing approximately 100,000 individuals per year [23]. Although the number of cases has decreased in recent years, ALAs remain a major public health concern in endemic areas [24]. Amoebiasis is most prevalent in tropical countries with poor sanitation and lower socio-economic status. This parasite has been detected to infect around 20 % of the Indian population. Homosexual men, people with acquired immunodeficiency syndrome or HIV, cirrhotic patients, and people living in group homes or mental health facilities are all at high risk of getting amoebiasis [24]. Malnutrition and alcohol use are also risk factors. The majority of ALAs appear with mild to moderate symptoms and respond to antiamebic medication within three days. However, reports from endemic areas show that the disease can be severe in up to 40% of patients and is not controllable with the medicine alone. These abscesses frequently appear rapidly with rupture, organ failure, and abnormal laboratory results - additional percutaneous drainage is required. Amebic liver abscesses have a 1% fatality rate [21]. Infection begins with the intake of *E. histolytica* cysts found in water or food that is contaminated. The vast majority of infected people (90 %) are asymptomatic carriers, meaning the parasite lives securely inside the lumen of the colon and excretes

cysts in feces to complete the entire life cycle. *E. histolytica* breaks the intestinal mucosa and invades the underlying lamina propria, where it contacts with host immune cells, producing a raging proinflammatory cytokine response that causes tissue damage in 10 % of infected individuals (symptomatic). It is unknown why a high number of *E. histolytica*-colonized people do not develop invasive illness. The complicated pathogenesis is thought to be driven by a combination of parasite virulence factors, host genetics, microbiome, and immunological responses [21].

Amebic liver abscesses are characterized by right upper quadrant (RUQ) discomfort and fever. Symptoms commonly appear subacutely. Less than one-third of patients had diarrhea, while some may have had dysentery in the previous months. Jaundice affects less than 10 % of the affected ones. Patients returning from an endemic location usually get the condition within 8-20 weeks, with a 12-week average. Typically, there is a leukocytosis without eosinophilia and an increased alkaline phosphatase that is out of proportion to any increase in aminotransferases [20,21].

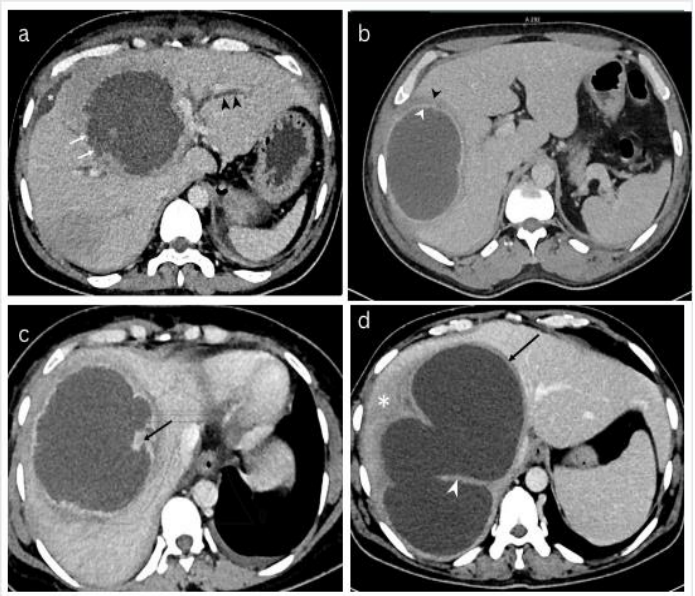


Figure 5. *CT scan (a) shows the abscess's non-enhancing and ragged edge in the absence of a defined wall (arrows). Pay attention to the dilated intrahepatic biliary ducts (arrowheads) due to the mass effect. CT image (b) of an abscess showing a double-target sign with an inner enhancing ring (white arrowhead) and an outer hypodense ring (black arrowhead). CT scan (c) of an abscess. Take note of the abscess's rim-enhancing wall and the perilesional halo-like hypodensity, as well as the focal nodularity in the wall (arrow) reflecting partially resorbed septa. The CT image (d) displays a well-defined smooth wall (arrow), although there is no contrast enhancement. The abscess wall is surrounded by an ill-defined hypodensity (asterisk). Take note of the huge cavity formed by the fusion of several abscesses, as well as the intervening wall that mimics internal septations [20].*

The diagnosis of an amebic liver abscess is obtained using a combination of imaging features and serologic testing. A cystic intrahepatic cavity is visible on imaging, which is usually indistinguishable from other types of liver abscesses. Most amoebic liver abscesses are single lesions, however multiple lesions may occur on occasion, and they are more commonly detected in the right lobe than the left. On ultrasound, the lesion shows as a spherical, well-defined hypoechoic mass. After healing, the margin of the abscess may calcify and form a round, thin rim [20,21,23].

ALAs have a non-specific CT appearance that has been described as a round or oval hypodense lesion with a thick enhancing wall and peripheral edema. Although these characteristics are considered classical, they are suggestive of resolving ALAs. Wall creation and rim enhancement are late results discovered as the healing process progresses - this appearance is likely to suggest modest drug-responsive illness. These characteristics are rarely seen in acute abscesses - they are clinically accompanied with significant symptoms or a distorted test profile. As a result, it is critical to distinguish aggressive abscesses from ones with modest manifestations [22].

The typical radiographic features are insufficient to determine a diagnosis and must be evaluated in conjunction with serological or serum antigenic evidence. Although the test may be negative in the first seven days of illness, most of the patients with amebic liver abscesses will produce detectable antibodies to *E. histolytica*. Antibodies are detected in 92- 97 % of patients with amebic liver abscesses. The aspiration of amebic liver abscesses is not required for diagnosis - trophozoites are only found in a small percentage of aspirates. Furthermore, when patients arrive with amebic liver abscesses, stool microscopy for *E. histolytica* is commonly negative, demonstrating that stool microscopy cannot be relied on to make the correct diagnosis[21,23].

4. Conclusion

The most common parasitosis that affects abdominal organs are mainly caused by *Echinococcus spp*., liver flukes and *Entamoeba histolytica*. Orally transmitted parasitic infections frequently cause liver damage, due to the liver's role as the primary organ to filter portal venous blood. Additionally, it has the potential to spread to other organs like the lungs, kidneys, and pancreas via blood flow or mucosal penetration. Parasitic infections are typically asymptomatic and diagnosis is often made by chance. However radiological imaging plays a crucial role in diagnostics - the most frequently used imaging methods include ultrasonography, computed tomography, and MRI. Moreover, in the vast majority of cases, the diagnosis must be clarified by serological tests.

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