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RESEARCH ARTICLE

AMOEBIC LIVER ABCESS-A CLINICIANS APPROACH

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ABSTRACT

Amebic liver abscess is the most frequent extraintestinal manifestation of Entamoeba histolytica infection. This infection is caused by the protozoa E histolytica, which enter the portal venous system from the colon. Amebic liver abscess is an important cause of space-occupying lesions of the liver, mainly in developing countries. Prompt recognition and appropriate treatment of amebic liver abscess lead to improved morbidity and mortality.

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INTRODUCTION

Amebic liver abscess is the most frequent extraintestinal manifestation of Entamoeba histolytica infection. This infection is caused by the protozoa E histolytica, which enter the portal venous system from the colon. Amebic liver abscess is an important cause of space-occupying lesions of the liver, mainly in developing countries. Prompt recognition and appropriate treatment of amebic liver abscess lead to improved morbidity and mortality. E histolytica exists in 2 forms. The cyst stage is the infective form, and the trophozoite stage causes invasive disease. People who chronically carry E histolytica shed cysts in their feces; these cysts are transmitted primarily by food and water contamination. Rare cases of transmission via oral and anal sex or direct colonic inoculation through colonic irrigation devices have occurred. Cysts are resistant to gastric acid, but the wall is broken down by trypsin in the small intestine. Trophozoites are released and colonize the cecum. To initiate symptomatic infection, E histolytica trophozoites present in the lumen must adhere to the underlying mucosa and penetrate the mucosal layer. Liver involvement occurs following invasion of E histolytica into mesenteric venules. Amebae then enter the portal circulation and travel to the liver where they typically form large abscesses. The Gal/GalNAc lectin is an adhesion protein complex that sustains tissue invasion (Blazquez, 2007). The abscess contains acellular proteinaceous debris, which is thought to be a consequence of induced apoptosis (Stanley, 2003) and is surrounded by a rim of amebic trophozoites invading the tissue. The right lobe of the liver is more commonly affected than the left lobe. This has been attributed to the fact that the right lobe portal laminar blood flow is supplied predominantly by the superior mesenteric vein, whereas the left lobe portal blood flow is supplied by the splenic vein. Amebic liver abscess is rare and is currently seen almost exclusively in immigrants or travelers. In 1994, 2,983 cases of amebiasis were reported to the Centers for Disease Control (CDC). The disease was removed from the National Notifiable Diseases Surveillance System in 1995. An estimated 4% of patients with amebic colitis develop an amebic liver abscess. An estimated 10% of the population is infected with Entamoeba dispar. Previously thought to be a nonpathogenic strain of E histolytica, this type of amoeba does not produce clinical symptoms even in the immunocompromised host. All races can be affected by amebic liver abscess. Risk factors for infection include travel or residence in endemic areas. Amebic liver abscess is marked by a 7-12–fold higher incidence in males than in females despite an equal sex distribution of noninvasive colonic amebic disease among adults (Acuna-Soto, 2000). However, no sexual preponderance exists among children. Peak incidence of amebic liver abscess occurs in people in their third, fourth, and fifth decades of life, although it can occur in any age group Worldwide, approximately 40-50 million people are infected annually, with the majority of infections occurring in developing countries. The prevalence of infection is higher than 5-10% in endemic areas (Blessmann, 2003), and sometimes as high as 55% (Haque *et al*., 2002). The highest prevalence is found in developing countries in the tropics, particularly in Mexico, India, Central and South America, and tropical areas of Asia and Africa. Infection with E

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histolytica ranks second worldwide among parasitic causes of death, following malaria. Annually, 40,000-100,000 deaths are caused by infection with E histolytica. Per year, a 10% risk of developing symptomatic invasive amebiasis exists after the acquisition of a pathogenic strain (Ralston, 2011).

Complications

Pleuropulmonary infection is the most common complication. Mechanisms of infection include development of a sympathetic serous effusion; rupture of a liver abscess into the chest cavity, leading to empyema; or hematogenous spread, resulting in parenchymal infection. Bronchopleural fistula may occur in rare instances when patients expectorate a substance that resembles anchovy paste. Trophozoites may be demonstrated in the fluid. Occasionally, this complication may be followed by a spontaneous cure of the amebic liver abscess. Cardiac involvement results following the rupture of an abscess involving the left lobe of the liver. It usually is associated with very high mortality. Intraperitoneal rupture occurs in 2-7% of patients. Left lobe abscesses are more likely to progress to rupture because of their later clinical presentation.

- Bacterial superinfection can occur.
- Rupture into peritoneal organs (eg, stomach) and mediastinum can occur.
- Cases of hepatic artery pseudoaneurysm have been reported.

History

The signs and symptoms of amebic liver abscess often are nonspecific, resembling those of pyogenic liver abscess or other febrile diseases (Hoffner, 1999; Hughes, 2000; Ravdin, 1995 and Ravdin *et al*., 2005).

Time of onset

Patients with amebic liver abscess usually present acutely (duration of symptoms < 14 d), with the most frequent complaints being fever and abdominal pain. This presentation is characteristic of younger patients. The subacute presentation is characterized by weight loss, and, in less than half the cases, abdominal pain and fever are present.

Abdominal pain

Abdominal pain is the most common element in the history and is present in 90-93% of patients. The pain is usually constant, dull, and aching, and it is most frequently located in the right upper quadrant (54-67%) and may radiate to the right shoulder or scapular area. The pain increases with coughing, walking, and deep breathing, as well as when patients rest on their right side.

Constitutional symptoms

Fever is present in 87-100% of cases, and rigors are present in 36-69% of cases. Nausea and vomiting are present in 32-85% of cases, and weight loss is present in 33-64% of cases.

Diarrhea

Diarrhea is present in less than one third of patients at the time of diagnosis. Some patients describe a history of having had dysentery within the previous few months. Bloody diarrhea is present in 7% of cases.

Pulmonary symptoms

Pulmonary symptoms are present in 18-26% of cases. The most frequent symptoms are cough and chest pain, which may represent a sign of secondary pulmonary involvement by abscess rupture in the pleural cavity. When coughing produces an odorless brown substance similar to anchovy paste, a bronchopleural fistula has developed (Mbaye, 1998).

Recent travel to endemic areas

Onset of symptoms usually occurs within 8-12 weeks from the date of travel. In 95% of cases, onset occurs within 5 months of returning from travel to an endemic area. A remote travel history of as many as 12 years has been reported.

Physical

Fever is the most common sign and is found in as many as 99% of cases. Hepatomegaly is present in some cases. The frequency varies widely in different series published, reporting as high as 63% in one series and as low as 18% in another. Hepatomegaly with pain upon palpation is one of the most important signs of amebic liver abscess. Point tenderness over the liver, below the ribs, or in the intercostal spaces is a typical finding. Right upper abdominal quadrant tenderness is present in 55-75% of cases. When the abscess is located in the left lobe (28% of cases), epigastric tenderness is noted. Pulmonary abnormalities are present in 20-45% of cases, and they consist of dullness and rales at the right lung base and nonproductive cough. Breath sounds over the right lung base may be diminished. Pleural rub may be audible. Jaundice (< 10% of cases) most often occurs in complicated cases with multiple abscesses or a large abscess compressing the biliary tract.

Signs of complications include the following

- Signs of peritoneal irritation, such as rebound tenderness, guarding, and absence of bowel sounds, are present when the abscess ruptures into the peritoneal cavity. Peritonitis occurs in 2-7% of cases.
- Pericardial friction rub can be audible when the abscess extends into the pericardium. This sign is associated with a very high mortality.
- Signs of pleural effusion are present when the abscess ruptures into the pleural cavity

Causes

The following are the risk factors associated with amebic liver abscess:

- Immigrants from endemic areas
- Institutionalized persons, especially people with mental retardation
- Crowding and poor hygiene
- Men who have sex with men (secondary to sexually acquired amebic colitis)
- Presence of immunosuppression (eg, HIV infection, malnutrition with hypoalbuminemia, alcohol abuse, chronic infections, posttraumatic splenectomy, steroid use)

Laboratory Studies

Hematology

Approximately three fourths of patients with an amebic liver abscess have leukocytosis. This most likely will appear if symptoms are acute or complications have developed. However, eosinophilia is rare. Anemia may be present, but the cause usually is multifactorial.

Chemistry

Hyperbilirubinemia is present in only a small proportion of cases. In acute liver abscess, the aspartate aminotransferase (AST) levels are high. In chronic liver abscess, the alkaline phosphatase level tends to be elevated and the AST level tends to be within normal limits. Overall, the alkaline phosphatase level is elevated in about 70% of cases of amebic liver abscess. Similar CBC count and liver test abnormalities are found in patients with pyogenic liver abscesses and are not specific.
Stool Studies

Stool examination

The role of microscopic stool examination is limited. Less than 30-40% of patients with amebic liver abscess have concomitant intestinal amebiasis, and 10% of the population is infected with the nonpathogenic strain of E dispar. Hence, the microscopic examination of the stool for the identification of cysts is of little value. If positive, it may suggest the diagnosis. Fecal findings suggestive of amebic colitis include a positive test for heme, a paucity of neutrophils, and the presence of Charcot-Leyden crystal protein. The stool examination is still of value if the serologic and antigen identification tests are not available. Examination of the stool for hematophagous trophozoites of E histolytica must be made on at least 3 fresh specimens because the trophozoites are very sensitive and may be excreted intermittently. A combination of wet mount, iodine-stained concentrates, and trichrome-stained preparations is used. Upon examination of the stool, trophozoites may be confused with neutrophils. Cysts must be differentiated morphologically from nonpathogenic Entamoeba hartmanni, Entamoeba coli, and Endolimax nana. Nonpathogenic **E** dispar cannot be differentiated morphologically and require fecal antigen detection.

Stool antigen detection

Stool antigen detection facilitates early diagnosis before an antibody response occurs (< 7 d) and differentiates pathogenic from nonpathogenic Entamoeba infection. The primary drawbacks are the requirement for fresh, unpreserved stool specimens (Tanyuksel, 2003), and the lack of intestinal amebiasis in as many as 60% of patients with amebic liver abscess. Stool antigen detection kits based on enzyme immunoassay (EIA) are most common and still quite sensitive compared to polymerase chain reaction (PCR)-based methods (Solaymani-Mohammadi, 2006). The PCR stool test shows high sensitivity for detecting E histolytica and for distinguishing nonpathogenic amoebas (Hamzah, 2006; Khairnar, 2007 and Roy, 2005). However, this test is expensive. Real-time (rapid) PCR is sensitive but not well standardized (Qvarnstrom, 2005) and is not widely available.

Stool culture

Stool culture for amoeba is sensitive but has limited availability.

Serologic testing

Serologic testing is the most widely used method of diagnosis for amebic liver abscess. In general, the test result should be positive, even in cases when the result of the stool test is negative (only extraintestinal disease) (Otto, 2013).

EIA

EIA has now largely replaced indirect hemagglutination (IHA) testing and counter immunoelectrophoresis (CIE) testing. EIA is relatively simple and easy to perform, rapid, inexpensive, and more sensitive (Knobloch, 1983 and Restrepo, 1996). The EIA test detects antibodies specific for E histolytica in approximately 95% of patients with extraintestinal amebiasis, in 70% of patients with active intestinal infection, and in 10% of persons who are asymptomatic cyst passers. The EIA serology findings revert to negative in 6-12 months following eradication of infection. Even in highly endemic areas, fewer than 10% of patients who are asymptomatic have positive amebic serology findings. Initial negative test results may appear in as many as 10% of patients with amebic liver abscess. Under these circumstances, order repeat serology testing in 1 week. This test result will usually be positive.

Serum antigen detection

E histolytica galactose lectin antigen is detectable by enzyme-linked immunosorbent assay (ELISA) in at least 75% of serum samples obtained from patients with amebic liver abscess. Studies reported an antigen seropositivity of 96% with a reversal rate of 82% after 1 week of treatment with metronidazole. This test may be useful for patients who present acutely, before an antibody response occurs. The sample needs to be obtained before starting the treatment, as the treatment leads to rapid antigen loss. This test can be used for rapid diagnosis in highly endemic areas, where serology can be misleading, but it is not widely available (Tanyuksel, 2003). Rapid antigen and antibody tests are currently being evaluated and seem very promising (Leo, 2006).

Imaging Studies

None of the imaging tests can definitively differentiate among a pyogenic liver abscess, an amebic abscess, and malignant disease. Clinical, epidemiologic, and serologic correlation is needed for diagnosis.

Ultrasonography

Ultrasonography is the preferable initial diagnostic test. It is rapid, inexpensive, and is only slightly less sensitive than CT scan (75-80% sensitivity vs 88-95% for CT scan). Ultrasonography simultaneously evaluates the gallbladder and avoids radiation exposure. As opposed to scanning with technetium-99m, ultrasonography often can distinguish an abscess from a tumor or other solid focal lesion. The lesions tend to be round or oval, with well-defined margins, and hypoechoic.

Computed tomography scanning

CT scanning is sensitive but the findings are not specific. The abscess typically appears low density with smooth margins and a contrast-enhancing peripheral rim. The use of injected contrast may differentiate hepatic abscesses from vascular tumors.

Magnetic resonance imaging: MRI is sensitive, but the findings are not specific. This imaging modality provides information comparable with less expensive imaging procedures.

Nuclear imaging studies: Technetium-99m liver scanning is useful for differentiating an amebic liver abscess from a pyogenic abscess; however, it is not used as a first-line test. Because amebic liver abscesses do not contain leukocytes, they appear as cold lesions on hepatic nuclear scanning, with a typical hot halo or a rim of radioactivity surrounding the abscess. In contrast, pyogenic liver abscesses contain leukocytes and, therefore, typically appear as hot lesions on nuclear scanning. Gallium scanning is helpful in differentiating pyogenic abscess (similar to technetium-99m nuclear hepatic scanning) but requires delayed images, which makes the test less helpful.

Other imaging studies: Hepatic angiography is only useful to differentiate liver abscesses from vascular lesions. Plain chest or abdominal films may show elevation and limitation of motion of the right diaphragm, basilar atelectasis, and right pleural effusion or gas within the abscess cavity.

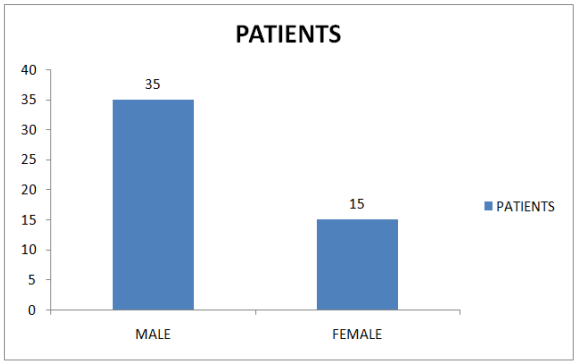
MATERIALS AND METHODS

The study was conducted in the Department of General Surgery, Nair Hospital from 16th March 2015 to 15TH March 2016 on patients admitted in Nair Hospital. The patients consent was taken for the procedures they underwent. There was no inclusion or exclusion criteria specified in the study.

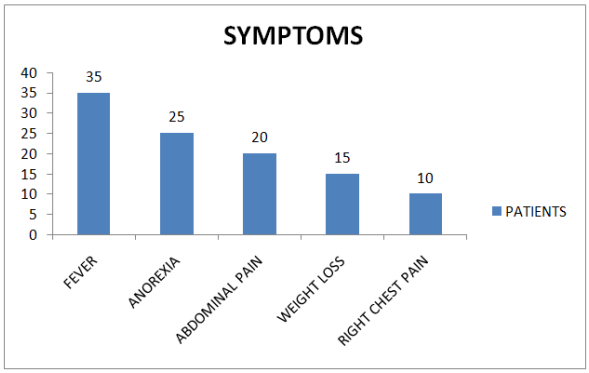
Observation and Results

Following observations were made:

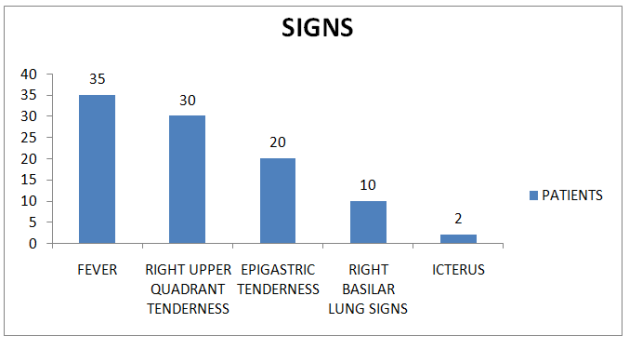
1. Sex of patient



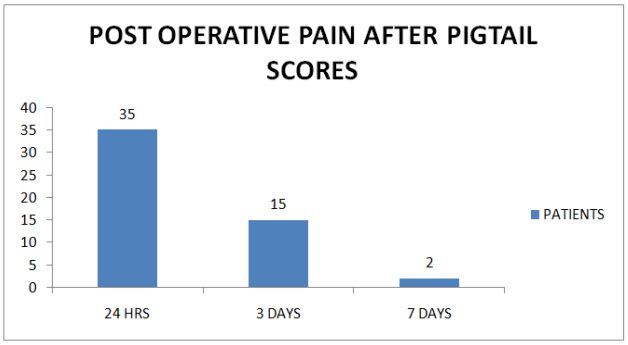
2. Symptoms



3. Sign



4. Post-operative pain scores (24hrs, 3 days, 7 days) after pigtail



5. Complications Nil

6. Number of days to return to work 25-30 days

DISCUSSION

Management of amoebic liver abscess (ALA) Four groups of treatment modalities are effective: 1. Drug therapy only 2. USG guided aspiration and drug 3. Percutaneous catheter drainage and drug 4. Laparatomy, drainage and drug. In our study drug therapy alone was given for 10 patients. Usg guided aspiration with drug therapy was done for only one patient. Percutaneous catheter drainage with drug therapy were given to maximum number of patients in all 39.Laparatomy with drainage was not performed.

Medical Therapy: Medical therapy may be instituted using either a single agent or a combination of drugs for the extraluminal parasite. Pharmacotherapy of E. histolytica infection in adults with side effects.

- Metronidazole 750 mg orally three times a day for 5–10 days; 500 mg IV every 6 hours for 5–10 days 30–50 mg/kg/d for 5–10 days orally in three divided doses; 15 mg/kg IV load followed by 7.5 mg/ kg every 6 hours (maximum, 2250 mg/d) Psychosis, seizures, Peripheral neuropathy and a metallic test
- Chloroquine (base) (used as an alternative or adjuvant) 600 mg/d orally for 2 days, then 300 mg/d orally for 14 days 10 mg /kg of chloroquine base Diarrhoea, abdominal cramps, cardiotoxicity, seizures and hypotension
- Tinidazole (Preferable to Chloroquine) 2 gm/ day for 3-5 days

Luminal agents (Used to eradicate intestinal colonization after Amoebicidal treatment)

- Paromomycin 25-30 mg/kg/d orally for 7 days in three divided doses 25 mg/kg/d orally for 7 days in three divided doses (maximum, 2 g/d) Diarrhoea,
- Iodoquinol 650 mg orally three times a day for 20 days 30–40 mg/kg/d for 20 days in three divided doses (maximum, 2 g/d) Contra indicated in patients with Hepatic insufficiency or hypersensitivity to Iodine
- Diloxanide furoate (Indicated in patients who fail to respond to Iodoquinol and Paromomycin) 500 mg orally three times a day for 10 days 20 mg/kg/d in three divided doses

Criteria for medical management

- All non-complicated abscess
- No features of rupture / impending rupture
- No compression effect

Nitroimidazoles including metronidazole are effective in over 90%.[22] Therapy should continue for at least 10 days. Relapses have been reported with this duration and the drug may be administered for up to 3 weeks. Single agent therapy with metronidazole yields excellent results and the alternative toxic drugs are indicated rarely and used probably in seriously ill patients where the risk of failure of therapy is unacceptable. The response to anti-amoebic drug is usually evident within 48 to 72 hours with the subsidence of toxemia (Thompson, 2003). Aspiration or drainage of the abscess: Routine aspiration of liver abscess is not indicated for diagnostic or therapeutic purposes (Stanley, 2003). A combination of USG findings with a positive serology in the appropriate clinical setting is adequate to start drug therapy.

Aspiration has been indicated in the following circumstances (Ralls, 1982)

- Lack of clinical improvement in 48 to 72 hours
- Left lobe abscess
- Large abscess having impending rupture / compression sign
- Thin rim of liver tissue around the abscess(<10mm)
- Seronegative abscesses
- Failure in the improvement following non-invasive treatment after 4 to 5 days.

Like PLA, percutaneous drainage is indicated when thick collection is not getting aspirated by needle or there is failure of USG-guided aspiration. Antiamoebic therapy alone is as effective as routine needle aspiration combined with antiamoebic therapy in the treatment of patients with uncomplicated liver abscess. (Chavez-Tapia *et al*., 2009) Surgical intervention: Open surgical drainage is rarely indicated and may be required in the setting of: (Sarda *et al*., 1989)

- Large abscess with a poor yield on needle aspiration or percutaneous drainage
- Clinical deterioration despite attempted needle aspiration
- Complicated ala (like ruptured abscess in peritoneal cavity with features of peritonitis)
- Complicated ala (ruptured in the pleural cavity / pericardial cavity/ adjacent viscera)

Surgical mortality is, however, very high. Hence, in clinical practice, it is only used when the cavity has ruptured into adjacent viscera or body cavities. Long term follow up of ALA patients: After clinical cure, patients show few symptoms and sonographic follow up demonstrates evidence of persistent hypoechoic lesion. The mean time for the disappearance of the sonographic abnormality is 6-9 months. (Sharma *et al*., 1995) In our study the patients took an average of 25-30 days o return to work. Relapses are very uncommon and the sonographic abnormality does not warrant continued therapy. The patterns of resolution seen on sonographic follow up include: 29 Type I: where complete disappearance of the cavity occurs within 3 months (29.8%), Type II: a rapid reduction till 25% of the original cavity size and then a delayed resolution (5.9%). Factors influencing healing time include: the size of abscess cavity at admission, hypoalbuminaemia and anaemia. The type of clinical presentation, nature of therapy, number of location of abscesses and time for clinical resolution pattern of multiple liver abscesses is similar to solitary abscess. The total abscess volume of all the cavities is the most important factor that influences resolution time in multiple abscesses. As clinical resolution does not correlate with ultrasonographic resolution it is suggested that the results of the therapy should be monitored by clinical criteria rather than USG. Prognostic markers: Independent risk factors for mortality in ALA are: (Sharma *et al*., 1996)

- Bilirubin level > 3.5 mg/dl
- Encephalopathy,
- Volume of abscess cavity
- Hypoalbuminaemia (serum albumin<2.0g/dl).

The duration of symptom and the type of treatment does not influence mortality.

REFERENCES

Acuna-Soto R, Maguire JH, Wirth DF. 2000. Gender distribution in asymptomatic and invasive amebiasis. *Am J Gastroenterol*., May. 95(5):1277-83. [Medline].

Blazquez S, Rigothier MC, Huerre M, *et al*. 2007. Initiation of inflammation and cell death during liver abscess formation by Entamoeba histolytica depends on activity of the galactose/N-acetyl-D-galactosamine lectin. *Int J Parasitol*., Mar. 37(3-4):425-33. [Medline].

Blessmann J, Ali IK, Nu PA, *et al*. 2003. Longitudinal study of intestinal Entamoeba histolytica infections in asymptomatic adult carriers. *J Clin Microbiol*., Oct. 41(10):4745-50. [Medline].

Chavez-Tapia NC, *et al*. 2009. Image guided percutaneous procedure plus metronidazole versus metronidazole alone for uncomplicated amoebic liver abscess. *Cochrane Data Syst Rev*., 209; Jan 1:CD004886.

Hamzah Z, Petmitr S, Mungthin M, *et al*. 2006. Differential detection of Entamoeba histolytica, Entamoeba dispar, and Entamoeba moshkovskii by a single-round PCR assay. *J Clin Microbiol*., Sep. 44(9):3196-200. [Medline].

Haque R, Duggal P, Ali IM, *et al*. 2002. Innate and acquired resistance to amebiasis in bangladeshi children. *J Infect Dis*., Aug 15. 186(4):547-52. [Medline].

Hoffner RJ, Kilaghbian T, Esekogwu VI, *et al*. 1999. Common presentations of amebic liver abscess. *Ann Emerg Med*., Sep. 34(3):351-5. [Medline].

Hughes MA, Petri WA Jr. 2000. Amebic liver abscess. *Infect Dis Clin North Am*., Sep. 14(3):565-82, viii. [Medline].

Khairnar K, Parija SC. 2007. A novel nested multiplex polymerase chain reaction (PCR) assay for differential detection of Entamoeba histolytica, E. moshkovskii and E. dispar DNA in stool samples. *BMC Microbiol*., May 24. 7:47. [Medline].

Knobloch J, Mannweiler E. 1983. Development and persistence of antibodies to Entamoeba histolytica in patients with amebic liver abscess. Analysis of 216 cases. *Am J Trop Med Hyg*., Jul. 32(4):727-32. [Medline].

Leo M, Haque R, Kabir M, *et al*. 2006. Evaluation of Entamoeba histolytica antigen and antibody point-of-care tests for the rapid diagnosis of amebiasis. *J Clin Microbiol*., Dec. 44(12):4569-71. [Medline].

Mbaye PS, Koffi N, Camara P, *et al*. 1998. [Pleuropulmonary manifestations of amebiasis]. *Rev Pneumol Clin*., Dec. 54(6):346-52. [Medline].

Otto MP, Gerome P, Rapp C, *et al*. 2013. False-negative serologies in amebic liver abscess: report of two cases. *J Travel Med*., Mar-Apr. 20(2):131-3. [Medline].

Qvarnstrom Y, James C, Xayavong M, *et al*. 2005. Comparison of real-time PCR protocols for differential laboratory diagnosis of amebiasis. *J Clin Microbiol*., Nov. 43(11):5491-7. [Medline].

Ralls PW, *et al*. 1982. Sonographic findings in hepatic amoebic abscess. *Radiology*, 145:123-6.

Ralston KS, Petri WA Jr. 2011. Tissue destruction and invasion by Entamoeba histolytica. *Trends Parasitol*., Jun. 27(6):254-63. [Medline]. [Full Text].

Ravdin JI, Stauffer W. 2005. Entamoeba histolytica (amebiasis). Mandell Gl, Bennett J, Dolin R eds. Principles and Practice of Infectious Diseases. 6th ed. Philadelphia, PA: Elsevier; Vol 2: Part III, sect H, 3097-3111.

Ravdin JI. 1995. Amebiasis. *Clin Infect Dis*., Jun. 20(6):1453-64; quiz 1465-6. [Medline].

Restrepo MI, Restrepo Z, Elsa Villareal CL, *et al*. 1996. Diagnostic tests for amoebic liver abscess: comparison of enzyme-linked immunosorbent assay (ELISA) and counterimmunoelectrophoresis (CIE). *Rev Soc Bras Med Trop*., Jan-Feb. 29(1):27-32. [Medline].

Roy S, Kabir M, Mondal D, *et al*. 2005. Real-time-PCR assay for diagnosis of Entamoeba histolytica infection. *J Clin Microbiol*., May. 43(5):2168-72. [Medline].

Sarda AK, *et al*. 1989. Intraperitoneal rupture of amoebic liver abscess. *Br J Surg*., 76:202-3.

Sharma MP, *et al*. 1995. Long term follow up of amoebic liver abscess: clinical and ultrasound patterns of resolution. *Trop Gastroenterol*., 16:24-8.

Sharma MP, *et al*. 1996. Prognostic markers in amoebic liver abscess: a prospective study. *Am J Gastroenterol*., 91:2584-8.

Sharma MP, *et al*. 2000. Management of amoebic liver abscess. *Arch Med Res*., 31:S4-5.

Solaymani-Mohammadi S, Rezaian M, Babaei Z, *et al*. 2006. Comparison of a stool antigen detection kit and PCR for diagnosis of Entamoeba histolytica and Entamoeba dispar infections in asymptomatic cyst passers in Iran. *J Clin Microbiol*., Jun. 44(6):2258-61. [Medline].

Stanley SL Jr. 2003. Amoebiasis. *Lancet*, Mar 22. 361(9362): 1025-34. [Medline].

Stanley, SL Jr. 2003. Amoebiasis. *Lancet*, 361:1025-34.

Tanyuksel M. and Petri WA Jr. 2003. Laboratory diagnosis of amebiasis. *Clin Microbiol Rev*., Oct. 16(4):713-29. [Medline].

Thompson JE Jr, *et al*. 1985. Amoebic liver abscess: a therapeutic approach. Rev Infect Dis., 7:171-9. 24.

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