AMOEbic liver abscess: revisited

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Amoebiasis is common in Asia, Africa, and South America, and is endemic in many regions of the Philippines. Hepatic involvement is a frequent extra-intestinal complication which is encountered in 3 to 9% of the cases of amoebiasis. In Africa, over a period of 20 years, the King Edward VIII Hospital reported 2,074 cases of hepatic abscess, while in New York City at the Mt. Sinai Hospital only 11 cases were seen in approximately 500,000 admissions. The Santo Tomas University Hospital in Manila had an incidence of one hepatic amoebic abscess per thousand admissions over a ten-year period.

Despite the advances in diagnostic tests and management, amoebic liver abscess remains to be a health problem in many parts of the world, where hygiene is not properly observed.

Etiology and pathogenesis

Amoebic liver abscess is a protozoal infestation that starts in the colon. The etiologic agent is Entamoeba histolytica. The protozoa was discovered by Losch in St. Petersburg, but named later on by Prof. Shaudinn in 1903. Its transmission is through contaminated water or vegetables. Amoebic infestation is initiated by swallowing the cysts. The cysts are spherical, 10 to 20um in diameter which rupture in the intestinal tract lumen into four metacystic trophozoites.

It is through the trophozoites that the disease evolves, often in the cecum and the ascending colon. The amebic protozoa may exist in its commensal state for years before becoming invasive. The invasiveness of the infestation is possibly related to the following: diet, host resistance, virulence of the protozoa, humoral and the cell mediated immunity. It is the protective cell mediated immunity that may be vital in restricting tissue invasiveness, as well as in aiding tissue resolution.

Pathology

The typical amoebic abscess is due to necrotic lysis of the liver tissue, which vary in size from a few centimeters to a large lesion. It is often single, usually in the posterior superior aspect of the right lobe. The abscess is well-demarcated that consists of the chocolate brown “anchovy-like” material of necrotic liver tissue, bile, fat and other products. It is usually sterile but may be secondarily infected.

Seen externally, the hepatic surface is smooth, swollen, and tender to touch. If untreated, it may rapidly progress to form fibrous reactions on the hepatic surface and the surrounding tissues. Its superior extension can lead to rupture into the pleural cavity. Its rupture inferiorly can lead to fatal peritonitis. The left-sided lesions, though infrequent, can lead to cardiac tamponade. The present study showed complete resolution with almost little residual scarring with successful nonsurgical management.

Clinical features

The duration of the illness that denotes hepatic involvement vary from a few days to several weeks. It is usually gradual in onset, with fever and progressive right hypochondriac pain. The febrile episode is intermittent, which later may be accompanied by chills. The pain may later on radiate to the epigastrum, chest and shoulders. In some cases, it may be associated with coughing but diarrhea is infrequent and loss of weight may not be significant.

An enlarged liver is usually palpable, smooth and tender. Its upward enlargement may push the right hemidiaphragm, which may be associated with breathing difficulties. Tenderness over the intercostal spaces on the right side might indicate an impending rupture of the hepatic abscess, particularly when associated with skin discoloration over a localized area. If the abscess extends downwards, there might be a bulge below the right
subcostal area, which when associated with exquisite tenderness, might indicate an unusually large abscess with impending rupture into the peritoneal cavity. An enlarged and exquisitely tender left hepatic lobe is an indication of a possible rupture towards the pericardial cavity. Jaundice which is relatively infrequent is obstructive in nature. It is important to auscultate the hepatic area for possible friction sound of hepatic amoebic abscess and differentiate it from the vascular sound of a neoplastic mass.

INVESTIGATIONS

Determination of the white blood count can be helpful and usually ranges from 10,000-20,000/cu.mm. The serum bilirubin is predominantly conjugated, associated with elevated serum alkaline phosphatase. On the other hand, the hepatic enzymes are often not significantly elevated.

The chest x-ray is important to show the unusual elevation of the right hemidiaphragm, particularly in the right lateral position, which suggests upward hepatic enlargement that may be associated with pleural effusion.

With the advent of hepatic imaging by means of the sonogram and the CT scan, the confirmation of hepatic abscess has been made relatively easy, showing its location, size, number and extent of the hepatic enlargement. Through these means, it is easier to rule out hepatic cysts, granulomas, neoplastic disease and hepatic enlargements of other causes.

Hepatic angiography as a means to differentiate problematic cases from granulomatous and neoplastic lesions has been proven to be useful in studies done at the Sto. Tomas University Hospital. Stool examinations should be done to determine the presence of the parasite. Short or long endoscopic examinations may likewise be considered to assess the extent of possible associated intestinal disease.

NEEDLE ASPIRATION

The availability of imaging techniques makes needle aspiration a relatively simple procedure. Aspiration may be advised in certain lesions as part of the treatment as well as for the histologic confirmation of the lesion, particularly in places where liver cancer and hepatobiliary tuberculosis are common. If the abscess is large or a point of exquisite tenderness is elicited externally, aspiration is mandatory to preempt rupture. Aspiration is done using a medium bore needle about 1.5 mm. in diameter attached via a three-way stop-cock to a 50 ml. syringe. The selected site for aspiration is the point of localized tenderness between the intercostal spaces with dullness on percussion. Left-sided lesions should be aspirated only if they are superficially located. Sonographic monitoring is recommended whenever available. Surgical drainage is recommended when the abscess is unusually large, there are impending signs of rupture or when rupture has occurred.

Examination of the aspirated material is important to confirm or exclude the diagnosis of amoebiasis. However, the E. histolytica is usually difficult to demonstrate in the aspirated material, which may require immunofluorescent techniques followed by conjugated anti-human globulin examination.

SEROLOGY

Obviously, the traditional and reliable method for the diagnosis of amoebiasis is the microscopic identification of E. histolytica. Unless proper technique is followed, the isolation of the protozoa can be frustrating.

To confirm the diagnosis of amoebiasis with certainty, there are serologic tests which include indirect immunofluorescence, indirect hemagglutination, enzyme-linked immunosorbent assay, and counter-immunoelectrophoresis. The indirect hemagglutination assay is widely used for its sensitivity in recognizing the invasiveness of the disease. Factors that may affect the result of the serologic test would include the length of exposure, severity of infestation and immunologic competency. There are, however, simpler tests that can be done by the bedside, like the gel diffusion in capillary tubes and the slide agglutination of antigen-coated latex particles. These tests have been found to show a positive result in more than 95% of the patients with amoebic liver abscess.

DIFFERENTIAL DIAGNOSIS

Amoebic liver abscess should be differentiated from pyogenic liver abscess in view of their similarities in clinical behavior. Amoebic liver abscess is usually seen in the 2nd to 5th decade of life, as a tender, smooth hepatic mass with associated pulmonary symptoms. In large right hepatic lobe abscesses, localized points of tenderness along the intercostal spaces might be elicited. A third of these patients may have a history of bloody diarrhea. Chest x-ray findings are common, manifesting as elevated right
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hemidiaphragm, effusion or atelectasis. The hepatic sonogram and the CT scan are often classic. The serologic tests are mandatory to differentiate lesions from pyogenic abscesses with utmost care in interpreting the results in endemic areas 5,13.

Pyogenic abscess is more common in the elderly with known predisposing diseases, such as biliary disorders, immunosuppressive conditions, diabetes and malignancies. It has an insidious onset of malaise, epigastric pain, fever and chills. Often the abscesses are multiple, the jaundice more obvious and the fever spiking. The general appearance of the patient is more toxic as a rule. Often, blood culture is necessary to show the presence of bacterial infection, usually E. coli or Klebsiella, to confirm the etiology of the disease.

Cystic liver disease, including those due to hydatid cysts, should be considered in patients from the tropics. Indirect hemagglutination and complement fixation tests for antibodies are positive in at least 80%.

Of course, in regions where hepatocellular carcinoma and other granulomatous diseases are prevalent, the histologic confirmation by means of percutaneous or open biopsy may be necessary as its clinical manifestation may simulate that seen in hepatic amoebic abscess 4,5,8,9.

MANAGEMENT

As late as in the '70s, the treatment of amoebic liver abscess remained debatable. There was no unanimity as to what was best, whether treatment should be with the use of anti-amoebic drugs alone or should it be combined with aspiration. When the abscess is very large or is showing signs of possible rupture, needle aspiration is deemed mandatory.

Currently, the most commonly used agent for amoebic liver abscess is metronidazole at a dose of 500 to 750 mg orally, three times a day for 7 to 10 days, providing a cure rate of more than 90%.

Metronidazole is well absorbed from the gastrointestinal tract and the use of the parenteral form offers no significant advantage. If the patient shows slow response or relapse after treatment, aspiration and/or a prolonged course of metronidazole may be considered. Metronidazole-resistant E. histolytica trophozoites had not been reported 14.

In some places, where tinidazole and/or nidazol are available, they can be considered as alternative agents, administered only for a few days, with a reported success rate of 94%.

Other alternatives for eradicating liver trophozoites include chloroquine, at 600 mg daily for 2 days, then 300 mg daily for 2-3 weeks. Dihydroemetine and emetine are no longer recommended in view of their significant potential side effects including cardiac toxicity and should only be administered in hospitalized patients 14,15.

The use of luminal agents are recommended for the possible associated intestinal infestation, even if the stool is negative for the organism. One of the following may be used: paromomycin, at 30 mg/kg/day orally in three divided doses for 10 days; diiodohydroxyquin at 650 mg orally three times a day for 20 days; or diloxanide furoate at 500 mg orally three times daily for 10 days 14,15.

Uncomplicated amoebic liver abscess has a mortality rate of <1% if diagnosed early. However, for the complicated disease, the mortality can be as high as 17 to 20%. Untreated, the mortality is 100% 6.

REFERENCES

8. Sharma, MP; Amebic liver abscess. Trop Gastroenterol 1993; 14: 3.