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Abscesses in the Liver: Amoebic or Pyogenic?

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Liver abscess is a common condition in our part of the world. This is not surprising if one looks at the epidemiology of amoebiasis. The prevalence of infection with *E. histolytica* and *E. dispar* is as high as 50% in the underdeveloped areas, where asymptomatic intestinal infection occurs in 90 to 99% of infected individuals and confers a 10% risk per year of symptomatic invasive amoebiasis¹. However, apart from *Entamoeba histolytica*, liver abscess may also be caused by other pathogens, including enteric gram-negative bacteria and pyogenic gram-positive cocci. Occasionally, salmonella, brucella, mycobacteria and even fungi have been known to produce abscesses in the liver. Pyogenic liver abscess, however, is an uncommon entity. Not unexpectedly, it is more common than amoebic liver abscess in the developed world: a direct reflection of prevalence of *E. histolytica* in these areas^{2,3}. How does a liver abscess evolve? The liver is prone to microbial invasion by virtue of its anatomical location and function. Most of the blood from the large gut drains into the right lobe of the liver through the portal system. Organisms passing through the hepatic sinusoids are trapped, phagocytosed and killed by the hepatic mononuclear-macrophage system. An abscess develops when these phagocytic cells fail to eradicate the microbes, either because of some intrinsic defect in their function or because of the virulence of the infecting organisms. Microorganisms that evade phagocytosis and intracellular killing, subsequently inflict cellular injury, directly and/or through release of lytic enzymes and toxins.

Conditions like appendicitis, diverticulitis or inflammatory bowel disease, where the infectious process is in the bed of the portal venous circulation, lead to pyogenic liver abscesses through suppurative thrombophlebitis. Apart from portal circulation, the liver may be infected from the biliary tract, secondary to a calculus, stricture or malignancy. Biliary disease is now the most common source of pyogenic abscess⁴. Liver abscess can also arise from infection of the gall bladder, through the blood stream from infected foci anywhere in the body, and following direct trauma. About one-fourth of the cases are cryptogenic, i.e., no source can be identified and are probably caused by infection of infarcted portions of the liver. Pyogenic liver abscesses may develop in patients with sickle cell anemia⁵ and as an infectious complication of liver transplantation⁶.

Liver abscesses caused by *E. histolytica* complicate 3-9% of cases of amoebic colitis². Although there is no sex predominance in bacterial liver abscesses, more than 90% of amoebic liver abscesses occur in men; patients with amoebic liver abscesses are generally younger than those with bacterial abscesses¹. Pyogenic liver abscess is frequently polymicrobial and enteric gram-negative bacilli are generally cultured from the aspirated pus. The specific types of microorganisms that cause liver abscess probably vary with the underlying disease. The high frequency of "sterile" abscesses, reported in some series to be about 50% is probably a result of inadequate anaerobic cultivation. As a result of modern anaerobic bacteriologic techniques, anaerobic bacteria are now recognized as a major cause of liver abscesses. When modern anaerobic culture techniques are used, about 50% of all pyogenic liver abscesses have been found to be caused by anaerobes⁷ with positive blood cultures in 54% of these cases. *Staphylococcus aureus* and group A streptococcus account for 20% or less of cases. *Candida* may invade the liver as part of a systemic infection in patients with acute leukemia and produce microabscesses.

In the past, it was believed that 10-20% of amoebic liver abscesses were secondarily infected with bacteria, usually of enteric origin. However, in more recent series, superinfection was found to have

occurred in only upto 4% of cases⁸.

Both amoebic and pyogenic abscesses can be single or multiple. Multiple pyogenic abscesses are more apt to be caused by biliary tract disease whereas abscesses arising via the portal vein are usually solitary; the right lobe is more commonly involved than the left.

Clinical differentiation of amoebic from a pyogenic liver abscess is based on the clinical presentation and recognition of epidemiologic risk factors, a lack of predisposing conditions for pyogenic liver abscess, and early use of noninvasive imaging studies. History of dysentery may not be available. Concomitant diarrhea occurs in 30 to 40% of patients; amoebae are found in stool by microscopic examination even less frequently¹. Although older studies emphasize that an amoebic liver abscess presents as a single large lesion in the right lobe of the liver, studies using modern imaging technology demonstrate a high frequency of multiple lesions⁹. Abscess may be aspirated if there is a high suspicion of pyogenic infection, in which case aspiration is both diagnostic and therapeutic.

The differentiation of amoebic from a pyogenic liver abscess is greatly assisted by amoebic serology, since laboratory tests like liver function tests and stool microscopy are non-discriminatory, facilities for culture are lacking and the positivity rate of bacterial culture (aspirate, blood) is only around 50%. For this reason, the anti-amoebic antibodies, in significant titres, constitute the definitive diagnostic test of amoebic liver abscess. The antibodies are positive in up to 99% of patients with invasive amoebiasis¹. Sensitivity of serology is generally quoted to be approx. 90% for amoebic liver abscess¹⁰. In a local study on 100 liver abscesses, the indirect haemagglutination test (IHA) was found to be 100% sensitive and 94% specific for amoebic liver abscess¹¹. The cut off point of antibody titre between normal population and patients with invasive amoebiasis has been found to be 1:128 in an endemic population". Antibodies persist for a long time after resolution of infection, the titre and duration of persistence may not be influenced by early institution of specific chemotherapy¹². Furthermore, IHA test has been shown to remain positive for 20 years after infection¹³.

Therefore, serologic tests for amoebiasis should be interpreted with caution. A physician practising in an endemic area must bear in mind the frequent occurrence of asymptomatic infection with pathogenic *E. histolytica*, which leads to the high prevalence (10-35% of population) of serum anti:amoebic antibodies¹⁰. Another possible pitfall in interpretation would be in patients with an acute presentation of less than 7 days, when serologic studies may be negative.

Based on relevant clinical data, most cases of amoebic liver abscess can be diagnosed and treated without aspiration. In some cases, antimicrobials directed against enteric gram negative organisms may be added to metronidazole therapy, pending the results of serologic studies. Therapeutic trials of specific anti-amoebic therapy can be helpful diagnostically; most patients respond within 3 days with decreased pain and fever, The liver abscess cavity usually resolves gradually over months but persistent cystic lesions are not unusual. Serial ultrasonography has shown that at 6 months after successful treatment, only one-third to two-thirds of amoebic liver abscesses resolve completely¹⁰. Persistence of cystic lesions, presence of *E. histolytica* cysts in stool in carriers and high titres of anti-amoebic antibodies in endemic population are some of the confounding factors, which should be kept in mind in the work-up of a patient suspected to have liver abscess.

References

- 1.Mandell GL, Bennett JE, Dolin R. Principles and Practice of Infectious Diseases. 5th ed. Edinburg: Churchill Livingstone, 2000, PP. 840-42.
- 2.Barbour GL, Juniper K Jr. A clinical comparison of amoebic and pyogenic abscesses of the liver in sixty-six patients. Am. J. Med., 1972;53:323-34.
- 3.Ribaudo JM, Ochsner A. Intrahepatic abscesses: Amebic and pyogenic. Am. J. Surg., 1973; 125:570-

74.

4.Miedema BW, Dineen P. The diagnosis and treatment of pyogenic liver abscesses. *Ann Surg.*, 1984;200:328-35.

5.Shulman ST, Beem MO. A unique presentation of sickle cell disease: pyogenic hepatic abscess. *Pediatrics*, 1971; 47:1019-22.

6.Kusne S, Dummer JS, Singh N, et al. Infections after liver transplantation: An analysis of 101 consecutive cases. *Medicine, Baltimore*, 1988;67:132-43.

7.Perera MR, Kirk A, Noone P. Presentation, diagnosis and management of liver abscess. *Lancet*,1980;2:629-32.

8.Barnes PF, DeLock KM. Reynolds TN, et al. A comparison of amoebic and pyogenic abscess of the liver. *Medicine (Baltimore)*. 1987;66:472-83.

9.Rails PW, Henley DS, Colletti PM, et al. Amoebic liver abscess: MRI imaging. *Radiology*, 1987;165:801-4.

10.Petri WA Jr., Singh U. Diagnosis and management of amoebiasis. *Clin. Inf. Dis.*, 1999;29: 1117-25.

11.Khan MH, Qamar R, Shaikh Z. Serodiagnosis of amoebic liver abscess by IHA method. *J. Pak. Med. Assoc.*, 1989;39:262-64.

12.Knobloch J, Mannweiler E. Development and persistence of antibodies to *Entamoeba histolytica* in patients with amoebic liver abscess. Analysis of 216 cases. *Am. J. Trop. Med. Hyg.*, 1983;4:727-32.

13.Patterson M, Healy GR, Shabot JM. Serologic testing for amoebiasis. *Gastroenterology*, 1980;78: 136-41.