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REVIEW

Hepatic abscess: Diagnosis and management

S. Lardière-Deguelte[a], E. Ragot[b,c], K. Amroun[a], T. Piardi[a], S. Dokmak[b,c], O. Bruno[c,d], F. Appere[a], A. Sibert[c,d], C. Hoeffel[e], D. Sommacale[a], R. Kianmanesh[a,*]

[a] *Department of General, Digestive and Endocrine Surgery, Robert-Debré Hospital, Université de Reims Champagne-Ardenne, 51100 Reims, France*
[b] *Department of Hepatobiliopancreatic and Liver Transplantation, Beaujon Hospital, AP—HP, 92110 Clichy, France*
[c] *Université Denis Diderot, Paris 7, 75013 Paris, France*
[d] *Department of Radiology, Beaujon Hospital, AP—HP, 92110 Clichy, France*
[e] *Department of Radiology, Robert-Debré Hospital, 51100 Reims, France*

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Summary Microbial contamination of the liver parenchyma leading to hepatic abscess (HA) can occur via the bile ducts or vessels (arterial or portal) or directly, by contiguity. Infection is usually bacterial, sometimes parasitic, or very rarely fungal. In the Western world, bacterial (pyogenic) HA is most prevalent; the mortality is high approaching 15%, due mostly to patient debilitation and persistence of the underlying cause. In South-East Asia and Africa, amebic infection is the most frequent cause. The etiologies of HA are multiple including lithiasic biliary disease (cholecystitis, cholangitis), intra-abdominal collections (appendicitis, sigmoid diverticulitis, Crohn's disease), and bile duct ischemia secondary to pancreatoduodenectomy, liver transplantation, interventional techniques (radio-frequency ablation, intra-arterial chemo-embolization), and/or liver trauma. More rarely, HA occurs in the wake of septicemia either on healthy or preexisting liver diseases (biliary cysts, hydatid cyst, cystic or necrotic metastases). The incidence of HA secondary to *Klebsiella pneumoniae* is increasing and can give rise to other distant septic metastases. The diagnosis of HA depends mainly on imaging (sonography and/or CT scan), with confirmation by needle aspiration for bacteriology studies. The therapeutic strategy consists of bactericidal antibiotics, adapted to the germs, sometimes in combination with percutaneous or surgical drainage, and control of the primary source. The presence of bile in the aspirate or drainage fluid attests to communication with the biliary tree and calls for biliary MRI looking for obstruction. When faced with HA, the attending physician should seek advice from a multi-specialty team including an interventional radiologist, a hepatobiliary surgeon and an infectious disease specialist. This should help to determine the origin and mechanisms responsible for the abscess, and to then propose the best appropriate treatment. The presence of chronic enteric biliary contamination (i.e., sphincterotomy, bilio-enterostomy) should be determined before performing radio-frequency ablation and/or chemo-embolization; substantial stenosis of the celiac trunk should be detected before performing pancreatoduodenectomy to help avoid iatrogenic HA.

* Corresponding author.
E-mail address: rkianmanesh@chu-reims.fr (R. Kianmanesh).

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Introduction

Hepatic abscess (HA) can be defined as a suppurated cavity caused by the invasion and multiplication of microorganisms within healthy or diseased liver parenchyma [1]. Microbes can invade the liver parenchyma by way of the bile ducts, blood stream (hematogenic, most often portal), or by contiguous spread, especially via the gallbladder bed. HA is rare: the incidence is difficult to define, and it varies from one country to another. For Huang et al. in an American hospital during the 1990's [2], the incidence was 20/100,000 admissions. The incidence of HA seems to increase with age and co-morbidities (diabetes, denutrition, immunosuppression).

The etiology of HA can be bacterial, parasitic (amebic essentially), mixed (pyogenic superinfection of parasitic abscess) or more rarely fungal [3]. The main causes of HA are summarized on Fig. 1. Causal frequency varies according to geographic regions. In South-East Asia and in Africa, amebic contamination is the most frequent cause. In Western countries, 80% of HA are bacterial. They can occur in the course of intra-abdominal biliary infections that contaminate the biliary tract at the same time or can be secondary to seeding via the portal venous system of non-biliary infections (appendicitis or sigmoiditis). HA can also complicate surgical procedures (pancreatoduodenectomy, or liver transplantation) or hepatobiliary procedures (radio-frequency ablation and/or intra-arterial chemo-embolization). More rarely, HA develops after liver trauma or arterial embolization for trauma. Some HA are secondary to extra-abdominal infections that contaminate the liver parenchyma or pre-existing liver lesions (biliary cysts, hydatid cysts or necrotic metastases), most often via the hematogenous route. Unlike HA complicating abdominal infection, HA of arterial origin is most often monomicrobial with positive blood cultures.

While HA of bacterial origin is rare, it is extremely morbid with mortality reaching 15% in a series of 431 patients reported by Kuo et al. in 2013 [4]. The circumstances of onset, the signs of severity related to co-morbidity, the existence of underlying biliary disease and delays in management are some of the elements that can explain such high mortality [5,6].

Clinical and biologic signs of HA are non-specific and can include abdominal pain, fever and an inflammatory syndrome. Liver function tests can be more or less abnormal depending on the extent of the abscess, its cause (existence of underlying biliary disease with cholestasis with or without hyperbilirubinemia, increased transaminases in case of liver parenchymal ischemia) and severity of sepsis. The diagnosis relies essentially on imaging.

Imaging of HA

Sonography and CT scan lead to diagnosis in more than 90% of cases, and can often pinpoint the etiology (Figs. 2–8) [7]. The sensitivity of tri-phasic enhanced multi-slice CT is superior to that of sonography. The imaging appearance of HA and its evolution over time are variable, but can be schematically broken down to two phases: pre-suppurative and suppurative. In the pre-suppurative phase, images are heterogenous, hypodense, with irregular contours, poorly demarginated, and may simulate tumor, especially when HA are multiple and small. During the suppurative phase, images are hypo- or anechogenic, sometimes multiloculated, with rounded contours, that are clearly delineated by a more or less thick capsule. During this phase, sonographic images may have a typical ''target'' appearance. After contrast injection, peripheral enhancement forms a hyperdense border, the so-called ''ring sign'' without central enhancement. Sometimes this border is outlined by another hypodense ring, giving rise to the ''target'' image. During the arterial phase, the surrounding liver enhances transiently, and sometimes segmentally. The only sign that is quasi-pathognomonic of HA is the presence of internal gas, although air can sometimes be seen several days after

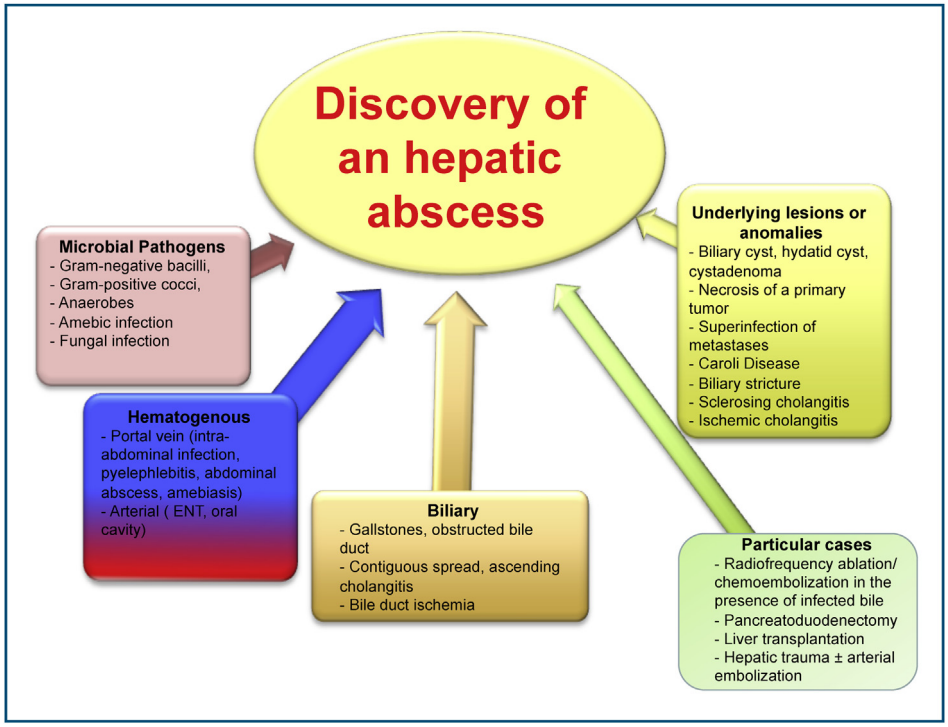

Figure 1. The different etiologies that must be considered when a hepatic abscess is discovered. ENT: ear, nose and throat.