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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

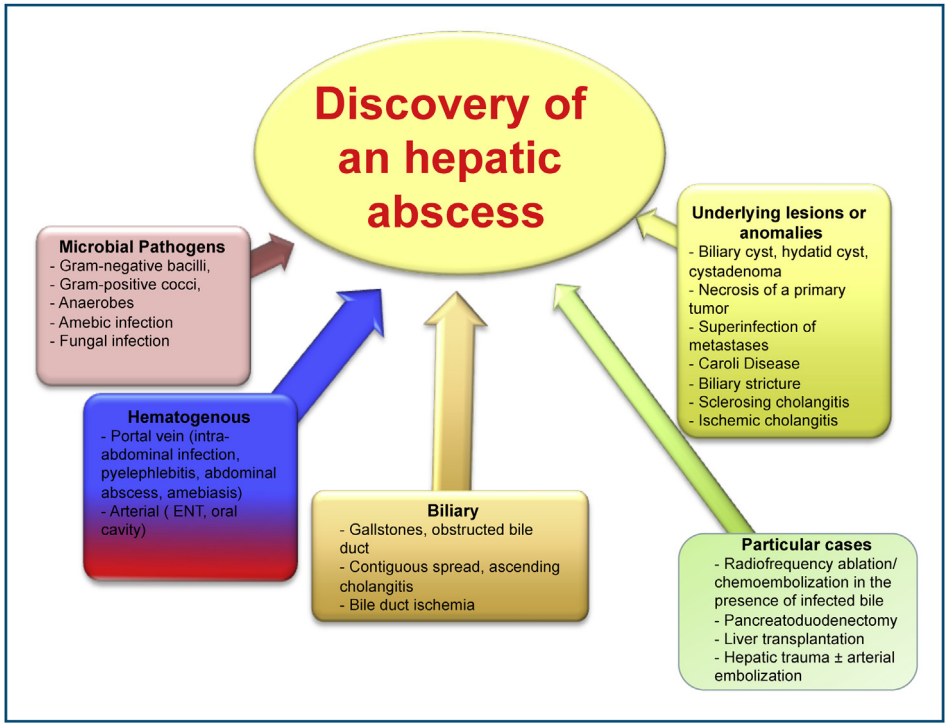

Figure 1. The different etiologies that must be considered when a hepatic abscess is discovered. ENT: ear, nose and throat.

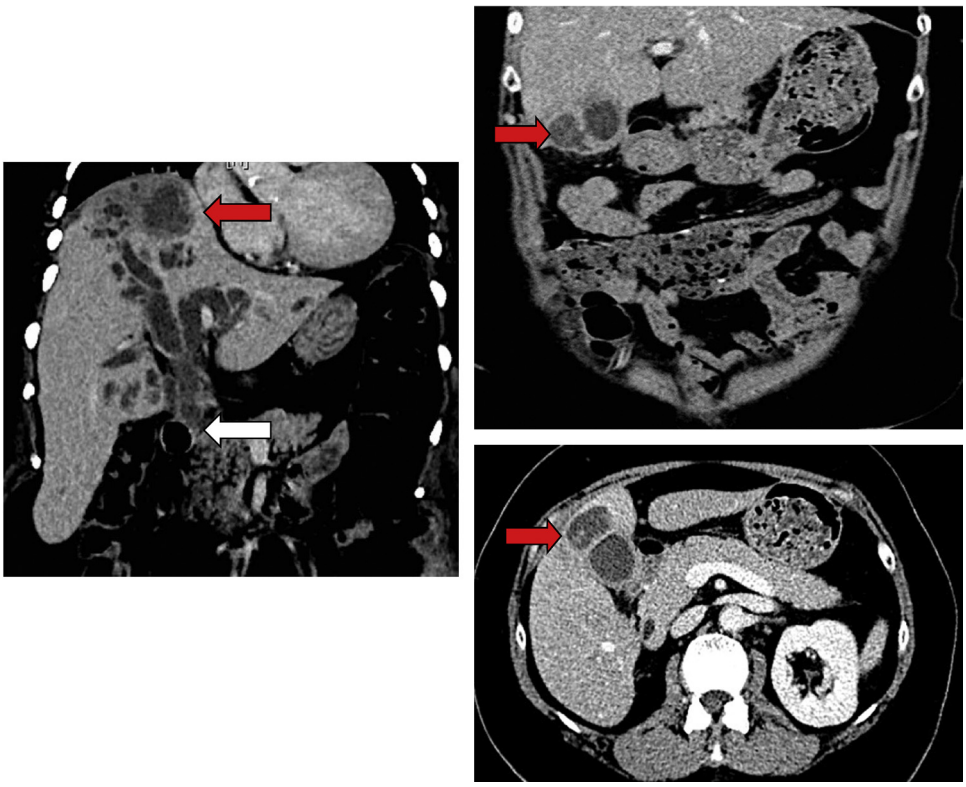

Figure 2. Left: abscess of the dome of the liver (red arrow) complicating lithiasic cholangitis: dilated bile ducts above an obstructing common duct stone (white arrow). Right: peri-cholecystic liver abscess (red arrow) as a complication of acute calculous cholecystitis.

procedures such as hepatic artery embolization without HA (*cf. infra*).

Imaging should be used to detect biliary disease (localized or diffuse), abscess or other signs of intra-abdominal infection, with or without septic porto-mesenteric thrombosis (pylephlebitis) (Fig. 3). If there is evidence of biliary disease and/or hepatic vein involvement, MRI can be useful to complete the work-up searching for biliary obstruction as a possible cause of HA. Colonoscopy can help to detect a gastrointestinal septic source in non-biliary HA (Fig. 3).

Generalities concerning the treatment of HA

Classically, management of non-parasitic HA consists of antibiotics, percutaneous abscess drainage and treatment of the underlying disease or cause (Fig. 8). Early criteria for effective treatment are apyrexia, disappearance of pain, and normalization of leukocytosis and CRP; reversal of imaging findings usually occurs somewhat later.

Antibiotics

Antibiotics should be started promptly, as soon as blood cultures have been drawn and before drainage procedures, to limit the systemic effects of septicemia [8,9]. Small HA, less than 3—5 cm, especially when multiple, can be treated by antibiotics alone without drainage, although no general consensus exists on this point. In 2008, Hope et al. reported a 100% success rate with antibiotic therapy alone for uniocular HA < 3 cm in their series of 107 patients [10]. Similarly,

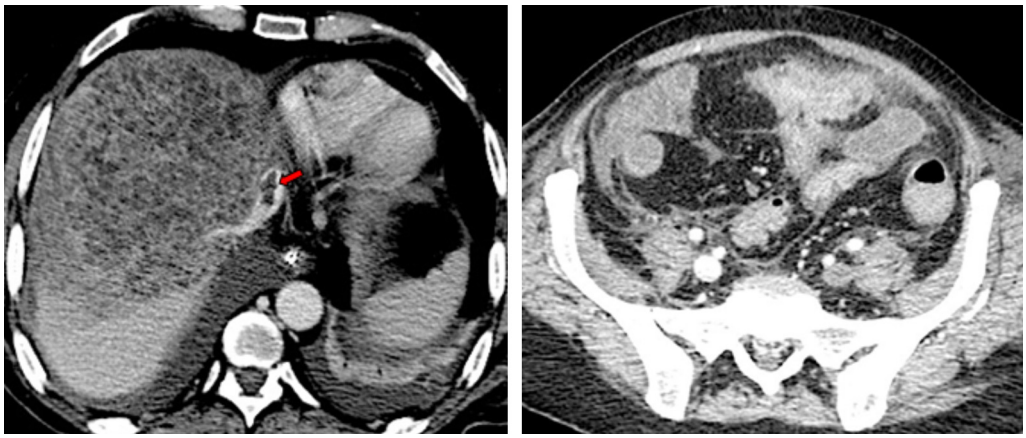

Figure 3. Left: diffuse hepatic abscess of portal vein origin in the pre-suppurative stage. The red arrow shows portal vein thrombosis, probably septic. Right: this hepatic abscess (HA) occurred during a severe inflammatory attack of ileal Crohn's disease with deep interloop mesenteric abscesses (portal origin).

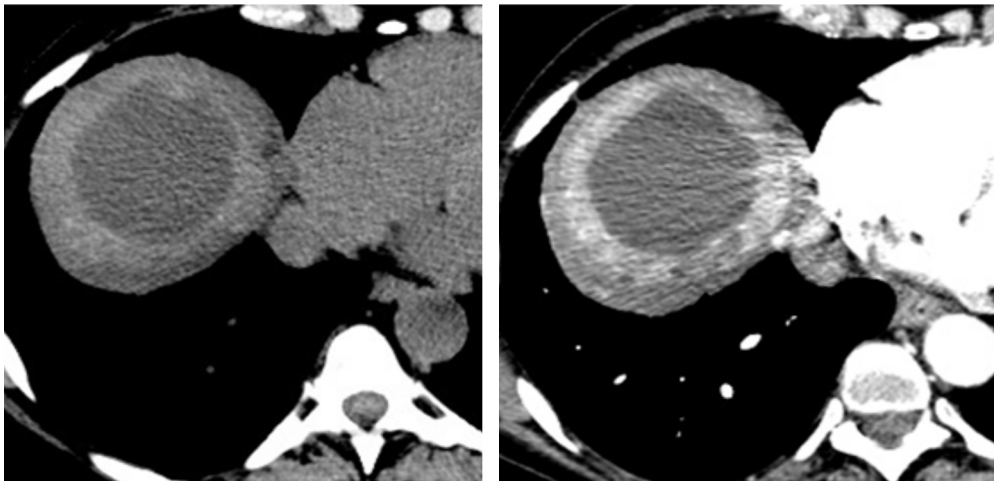

Figure 4. Biliary cyst with superinfection. Computed tomography (CT) with IV contrast shows a thickened cyst wall (left) that enhances with contrast injection (right).

in a literature review of 465 medically-treated abscesses, 176 of which were located in the liver, the 5 cm cut-off was the main factor associated with success of medical treatment alone, and the success rate for HA in this review was over 80% [11].

Empirical antibiotic choice should be directed at the microbes typically responsible, i.e. with activity against aerobic Gram-negative bacilli and Gram-positive cocci, [piperacillins, tazobactam, amoxicillin-clavulanic acid, 3rd generation cephalosporins, (cefotaxime, ceftriaxone)], in combination with an aminoglycoside (gentamicin) [9—11]. An anaerobic drug such as metronidazole can be used in combination if the chosen antibiotic is not active against anaerobes (or when an amebic abscess is a possibility) [9—11]. Antibiotic selection should be based on culture and sensitivities. The duration of antibiotic therapy is not clearly established, but is generally between 2 and 6 weeks [9—11].

Aspiration and drainage

Percutaneous drainage of HA was initially described in Hong Kong in 1953 by McFadzean et al. [12]. Sonographic or CT-guided needle puncture with aspirative drainage is currently the first line treatment of HA (Figs. 5 and 6). Needle aspiration also allows identification of the causative germ and may incidentally reveal evidence of biliary tract communication. After drain insertion, complete evacuation of the collection can proceed.

In 1998, Rajak et al. published a randomized study comparing simple needle aspiration versus catheter drainage of

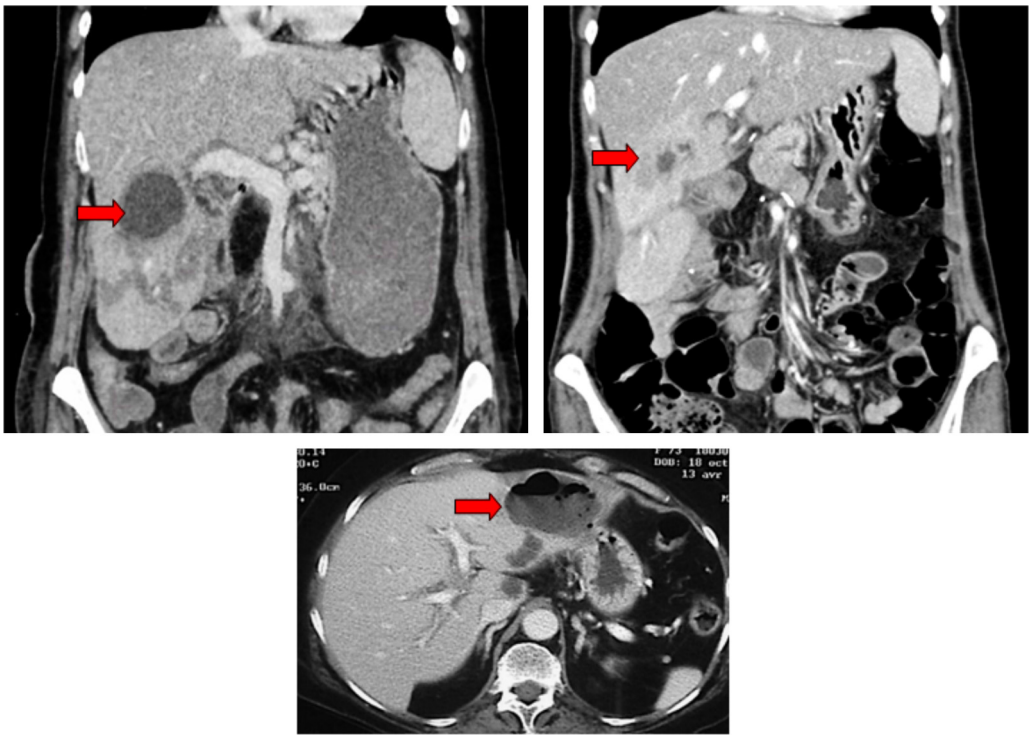

Figure 5. Upper images: hepatic abscess (HA) complicating pancreatoduodenectomy with zones of necrosis and developing abscess (arrow) due to the intraoperative ligation of the right branch of the hepatic artery with preservation of a patent left hepatic artery. Lower image: necrosis of the left lobe and segment 1 with abscess (arrow) following a pancreatoduodenectomy with ligation of the left hepatic artery.

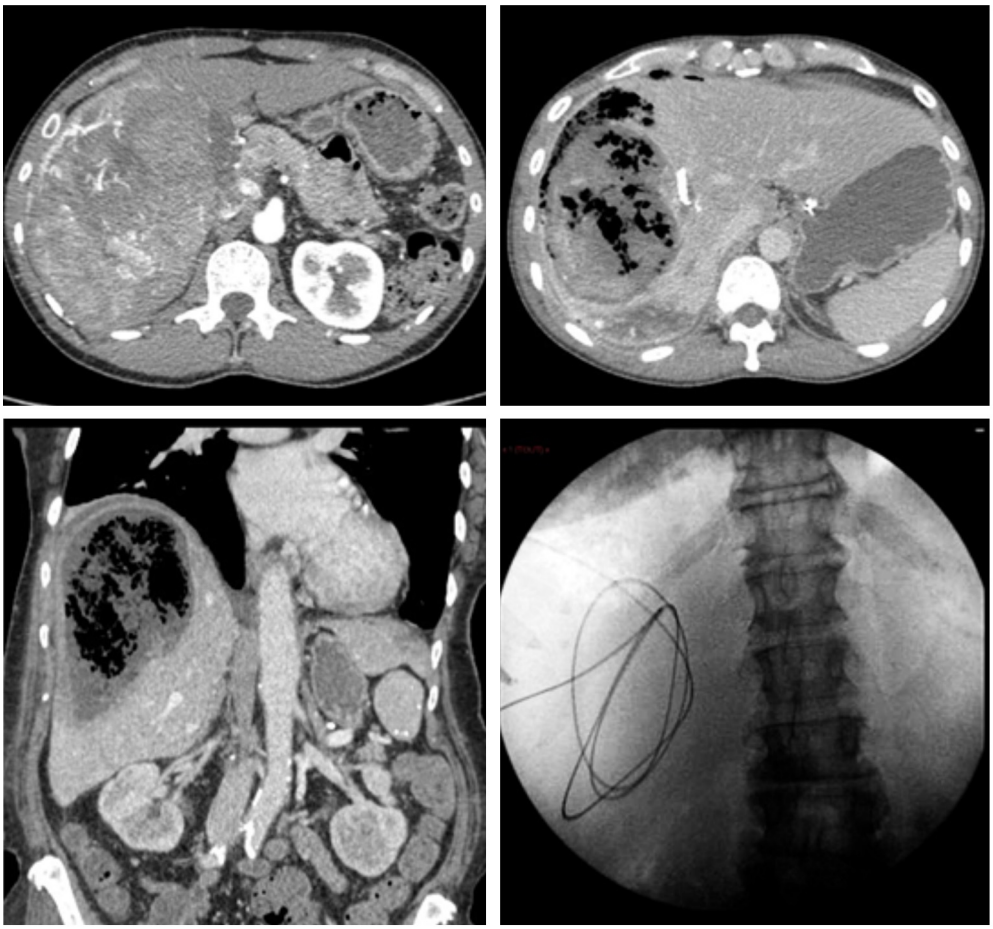

Figure 6. Above left: hypervascularized right lobe hepatocellular carcinoma. Treatment by transarterial chemo-embolization (TACE). Above right and lower left: tumoral necrosis with abscess formation 21 days after TACE. Below right: treatment by radiology-guided percutaneous drainage.

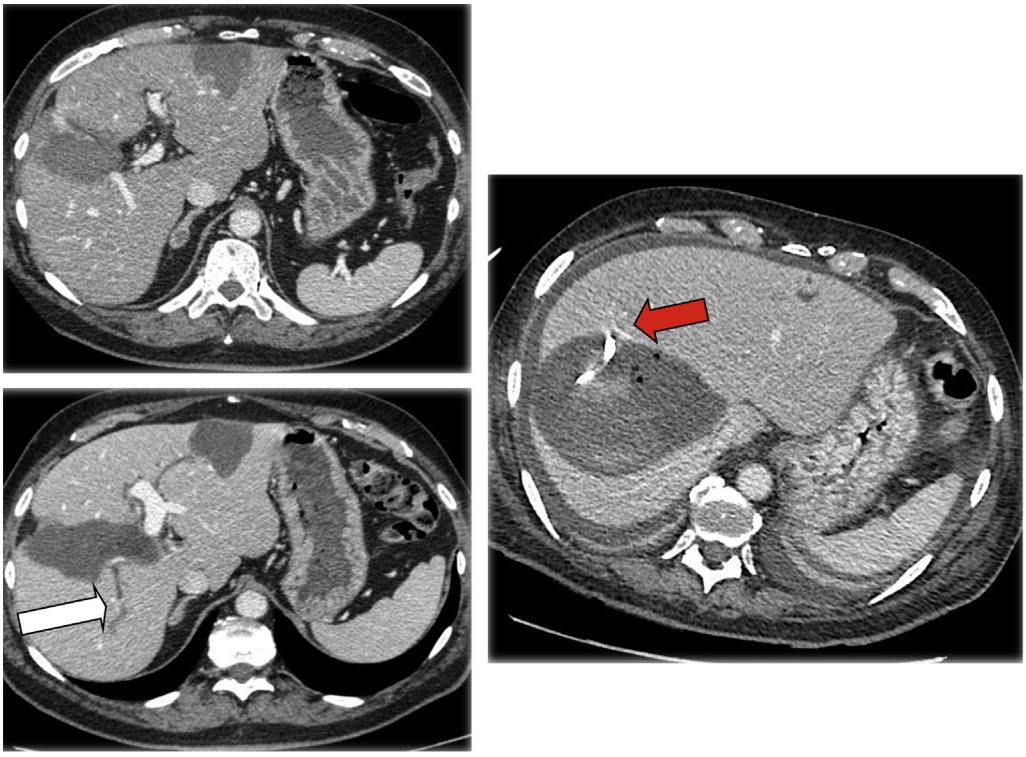

Figure 7. Progressive development of a right lobe hepatic abscess (HA) late after radio-frequency ablation (RF) for bilobar hepatic metastases (left images). A slight dilatation of the biliary ducts was noted (white arrow) 1 month after RF. The post-RF HA was treated with radiology-guided percutaneous drainage (red arrow).

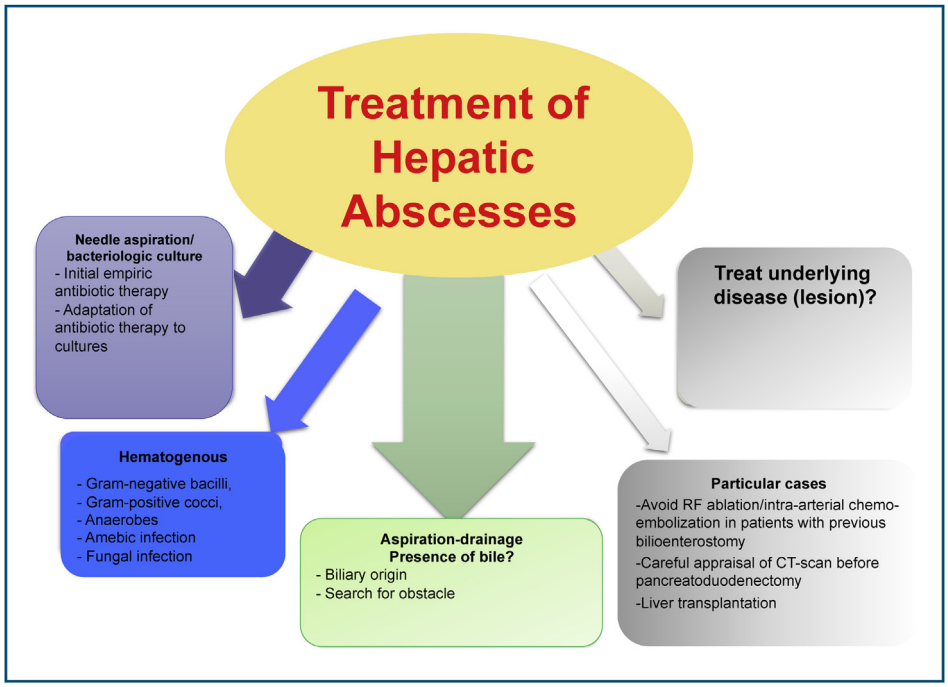

Figure 8. Management schema for hepatic abscess (HA) depending on the etiology.

HA [13]. In this study of 50 patients, with either bacterial or amebic abscess, catheter drainage was found to be more effective than needle aspiration alone (success rate 100% vs. 60%) [13,14]. Another randomized study, published in 2004 by Yu et al., compared repeated needle aspiration versus catheter aspiration in 64 patients with bacterial HA larger than 3 cm in diameter [15]. The authors concluded that the two techniques were equivalent in terms of success (positive needle aspiration rate = 97%), morbidity and mortality, and duration of hospital stay [15]. Of note, however, 59% of patients required more than one procedure [15].

In countries where interventional radiology is readily available, surgical drainage is rarely indicated. However, for some authors, there is a role for surgical drainage if percutaneous treatment for HA fails, for large abscesses > 5 cm, and/or multilocular HA [10,16,17], or when surgical treatment of the underlying cause of HA is necessary. Nonetheless, experienced radiologists can usually manage complex situations by ''re-drainage'' and/or improvement of existing drainage systems (larger drains, irrigation/lavage systems...), or even by innovative endoscopic wall collapsing techniques [18]. If surgery is planned, superficial HA can be treated with simple fenestration and drainage while deep abscesses can be treated either by intra-operative echo-guided drainage or limited hepatic resection [10,16,17].

Bile in drainage fluid is an essential element for classifying HA

The presence of bile is an *essential diagnostic element that helps to define the mechanism of onset of HA*, as it attests to communication with the biliary tract, either by contiguity, or because of ischemia/necrosis of the bile ducts [19,20]. HA may be classified as either communicating or non-communicating with the biliary tree, according to the presence or absence of bile in the drainage [21] (Fig. 1).

When a HA contains bile, one must search for:

- a biliary tract obstruction, usually due to choledocholithiasis or more rarely tumoral stricture;
- bile duct disease, often unrecognized (i.e., sclerosing or ischemic cholangitis).

Biliary MRI or cholangiography can visualize not only the communication but also the entire biliary tree to assess associated disease. When communication is found, specific treatment of the HA and eventual underlying cause can be entertained. HA secondary to ''ischemic cholangitis'' is all the more serious since communication leads to retrograde enteric contamination, e.g., after bilio-enterostomy or biliary sphincterotomy.

Hepatic abscess related to cholelithiasis

While HA is rarely the presenting complication of cholecystitis or lithiasic cholangitis, biliary disease is one of the most common causes of HA in the world (Figs. 2 and 3). The incidence of HA related to cholelithiasis is difficult to determine with precision, but according to Huang et al., 50 to 60% of HA were of biliary origin (benign or malignant) depending on the period of study [2]. Moreover, the authors of this study found that mortality decreased from over 80% prior to 1972 to approximately 10% after this date [2].

HA associated with complicated cholelithiasis characteristically presents with symptoms of persistent fever, right upper quadrant pain or laboratory findings of inflammatory syndrome despite well-conducted antibiotic therapy. The diagnosis is confirmed by imaging (sonography, CT scan or MRI).

HA complicating cholangitis

HA may originate from communication with bile infected by enteric bacteria upstream from an obstruction [1,2]. Imaging suggests the etiology when ductal dilatation (Fig. 2), biliary obstruction or underlying disease (intrahepatic stones, inflammatory and/or ischemic cholangitis or Caroli's disease) is evident. Of note, bile duct dilatation can disappear

if percutaneous abscess drainage results in high-output biliocutaneous fistula.

When cholangitis is complicated by HA, the biliary obstruction must be resolved (essentially via endoscopic sphincterotomy) because management of the HA will fail if obstruction persists [22]. When high-output bile drainage persists after drainage of HA, even in the absence of bile duct dilatation, a bile duct obstruction should be sought with CT (Fig. 2) or MRI cholangiography. Occasionally, direct opacification by cholangiography after drain insertion can define the obstruction.

HA complicating cholecystitis

Contiguous septic spread to involve the liver parenchyma may occur when perforation of an infected gallbladder extends into the lower segment 4 and/or segment 5 (Fig. 2). The onset of contiguous HA can be misleading, suggesting gallbladder carcinoma. In reality, intrahepatic perforation of cholecystitis is quite rare accounting for < 1% of all gallbladder complications [23].

With regard to treatment, the 2013 Tokyo consensus conference advised that Grade II acute cholecystitis (moderate acute cholecystitis) associated with severe local complications such as HA should be treated by emergency cholecystectomy (via laparoscopy or open approach) combined with systemic antibiotics [24]. For patients with grade III cholecystitis (acute cholecystitis complicated by organ failure) treatment should consist of percutaneous drainage of the gallbladder and/or the abscess plus medical treatment (antibiotics and management of organ failure), followed by secondary cholecystectomy [24].

''Non-lithiasic/non-biliary'' hepatic abscess

Abscess of portal origin

Non-biliary HA is usually secondary to portal bacteremia or pylephlebitis; these represent 10 to 20% of all bacterial HA [2]. While appendicitis was the most frequent cause of portal pylephlebitis in the 1970's, complicated sigmoid diverticulitis has taken over the first place today. Air in the portal system is a rare finding and is considered indicative of poor prognosis [25]. Other diseases that can result in portal HA include supra-infected gastrointestinal tumors (colon, small intestines, stomach) and chronic inflammatory bowel disease (Crohn's, Ulcerative Colitis) (Fig. 3) [5,26]. Performance of a CT scan and colonoscopy is therefore appropriate in the etiologic work-up of HA, especially when infection is polymicrobial.

Abscess by contiguity

These abscesses can be the consequence of any infection near the liver such as hepatic flexure cancer, perforated right colonic diverticulitis, Crohn's colitis, or perforated gastroduodenal ulcer. Polymicrobial infection and high levels of amylase in drainage fluid suggest this diagnosis.

Another rare but classical cause of contiguous HA is foreign body migration into the liver (left lobe) through the gastrointestinal wall (fish or chicken bone, toothpick) [27]. The diagnosis is generally made on CT scan and under these conditions, surgical treatment with removal of the foreign body and drainage of the HA are appropriate measures [27].

HA of arterial origin

Bacteremia arising from the lungs, urinary tract, upper airway or mouth (teeth), infectious endocarditits or suppurative peripheral thrombophlebitis in IV drug users may result in arterial seeding of the liver parenchyma [28]. HA of arterial origin is usually monolocular, monomicrobial (Gram-positive: staphylococcus or steptococcus), or fungal (*Candida albicans*) and is more likely to develop in patients with co-morbidities such as diabetes or immunosuppression. In the patient with fungal HA, associated valvular, splenic and renal primary foci should be specifically searched for [28].

The particular case of HA due to *Klebsiella pneumoniae*

HA due to *K. pneumoniae* was described in the 1990's in Taiwan and is occurring with increasing frequency in Asian series of HA but also in Western reports [29—31]. HA due to *K. pneumoniae* has the particularity of developing distant ''septic metastases'' (ocular and/or neurologic in 12% vs. 0% for other bacterial HA's); it is associated with intra-abdominal disease in less than 1% of cases [32], and is most often cryptogenic. HA due to *K. pneumoniae* is being described more and more frequently in Europe [30,31].

HA on preexisting hepatic disease (biliary cyst, metastasis, hydatidosis)

Superinfected biliary cysts

Superinfection of biliary cysts occurs in < 2% of patients with such disease, and usually involves cysts greater than 5 cm [33]. Contamination usually occurs via the hematogenous route, unlike superinfection of renal cysts. On sonography, the infected cyst has heterogenous contents with thickening of the typically thin cyst wall. Diagnosis requires identification of the germ(s) in the cyst contents, usually mixed with hemorrhagic deposits. Clinical signs vary with the localization of the cyst. Superinfection of a biliary cyst can modify the typical radiologic appearance of the cyst and can mimic other more serious disorders, especially with minimally symptomatic infectious and/or hemorrhagic episodes [34,35].

In patients with polycystic disease of the kidneys and liver, the risk of superinfection is higher in patients undergoing hemodialysis or transplantation (immunosuppression). Imaging plays an important role in diagnosis, demonstrating a cyst or group of cysts with heterogenous contents or air, or with thickened walls that enhance on CT scan after contrast injection (Fig. 4). MRI and PET scan (for certain authors) are the two most important investigations to localize infected cysts [34,35].

Superinfection of metastases or tumors

Superinfection of primary and metastatic liver malignancy is possible. Lesions include sarcoma, gallbladder cancer, breast cancer, neuroendocrine tumors or mucous colloid colorectal metastases. Rarely, the tumor is a primary hepatoma [36]. It is important not to miss underlying malignant disease when dealing with an apparently banal HA. In case of doubt, repeat imaging work-up is recommended after treatment of the abscess. Law and Li described the signs

that should evoke the potential diagnosis of superinfected hepatic malignancy including:
- thickened wall;
- presence of septations;
- aerobilia (fistula with the biliary tree);
- portal thrombosis, and/or presence of gas in the abscess [36].

In their series, Law and Li also described the treatment of 35 patients with HA complicating malignant disease [36]. These lesions were treated in the same way as ''benign'' HA by aspiration (± drainage) and antibiotics; aspiration did not appear to result in tumor dissemination [36]. Conversely, prognosis was less favorable in patients with malignancy because of the underlying disease, but also because of increased frequency of septic complications [36].

Superinfection of hydatid cysts

Hydatid cysts can fistulize into the biliary tree, causing symptoms of pain and/or cholangitis and cholestasis. The communication between the cyst and the biliary tree can introduce secondary superinfection leading to abscess formation within the cyst. Biliary MRI or retrograde cholangiography allows detection of scolices in the biliary tree and shows the bilio-cystic fistula. Treatment consists of endoscopic sphincterotomy to evacuate the scolices with subsequent surgical treatment of the cyst(s). Of note, alveolar echinococcosis can also present as a complex bilio-hepatic fistula (unpublished data: Pr G. Mantion, Pr B. Heyd, University Hospital of Besançon, France).

Intrahepatic abscess complicating pancreatoduodenectomy

HA that develops in the wake of pancreatoduodenectomy can be related either to:
- stenosis or obstruction of the hepatic artery;
- stricture of the choledocho-enterostomy or, more rarely;
- biliary reflux secondary to faulty surgery (short Roux-Y limb), stricture of one of the limbs or of the jejuno-jejunostomy, or obstruction downstream from the jejuno-jejunostomy.

Post-pancreatoduodenectomy HA is related to arterial stenosis resulting in ischemia of the biliary tree; it is associated with severe morbidity and mortality reaching 80% [37]. The patency of the hepatic artery must be assessed when HA occurs after pancreatoduodenectomy. Occlusion of the hepatic artery can be related to undetected celiac axis stenosis (hemodynamically significant in 5% of the overall population), intra-operative injury to the hepatic artery or one of its branches (Fig. 5), or to the consequences of postoperative embolization of the hepatic or gastroduodenal artery to treat bleeding or pseudo-aneurysm. Rarely, a non- or slightly significant stenosis of the celiac axis due to arcuate ligament impingement or atheroma can become symptomatic during a postoperative low-flow state, resulting in secondary ischemia of the biliary tract; infected bile due to the choledocho-enterostomy may be responsible for onset of HA.

Management of post-pancreatoduodenectomy HA with hepatic artery occlusion

These abscesses are generally multiple and polymicrobial. Bacterial culture should either be obtained by direct puncture of the abscess or by culture of bile obtained at surgery. Antibiotic treatment for several weeks or months as well as multiple percutaneous drainages may be necessary. For HA localized in a single lobe, partial hepatectomy can be considered in expert centers. If compression by an arcuate ligament is found postoperatively, it is usually too late to divide it surgically, and interventional radiology with dilatation and insertion of a stent is the best alternative. If there is partial stenosis of the hepatic artery, liver revascularization may occur via development of collateral vessels serving the bilio-enterostomy or the arterial anastomoses between the right and left livers and the diaphragm. If hepatic artery stenosis develops after embolization, surgical revascularization can be discussed but is usually difficult if not impossible in the inflammatory field associated with pancreatic fistula.

Management of post-pancreatoduodenectomy HA with a patent hepatic artery

This HA is typically solitary and is usually diagnosed within a few weeks of surgery. Theoretically, it arises as the consequence of one or several bouts of cholangitis. The frequency of post-pancreatoduodenectomy cholangitis is approximately 5% [38], although this is probably an underestimate. It is often minimally symptomatic. Its origin stems from stricture of the choledocho-enterostomy or, more rarely, from enteric reflux through the choledocho-enteric anastomosis. The prognosis is generally favorable as long as antibiotic choice is based on bacterial sensitivities. The anastomotic stricture can be resolved by percutaneous dilatation, with or without definitive stent placement, or by re-operation. In the case of enteric reflux, a downstream obstruction (jejuno-jejunal anastomosis, intestinal stricture, or peritoneal carcinomatosis) is usually responsible.

Hepatic abscess after chemo-embolization or radio-frequency ablation

Factors that may promote the development of HA after intra-arterial chemo-embolization (CE) or radio-frequency ablation (RF) include:
- the presence of a choledocho-enterostomy;
- antecedent history of sphincterotomy or biliary drainage (ascending colonization);
- but also associated diabetes or immunosuppression [39].

The diagnosis of HA that develops after RF or CE is usually based on clinical and laboratory findings. When early post-therapy imaging is performed, it can show air within the area of ''aseptic'' necrosis resulting from lesion treatment without any superinfection involved. An increased quantity of air or its appearance at a distance from RF or CE, should lead to the suspicion of post-RF or post-CE HA (Figs. 6 and 7).

HA occurs in less than 5% of patients undergoing CE, but is the principal cause of post-CE 30-day mortality. Of note, this complication may occur more frequently after CE for hepatic metastases from neuroendocrine tumors, perhaps because of greater sensitivity of the underlying ''healthy'' hepatic parenchyma [40]. Most specialty teams feel that the presence of a bilio-enterostomy is a contra-indication for CE [41].

The prevalence (< 1% of cases) and the severity of HA after RF seem to be lower than after CE [42,43]. HA after RF is similarly more prevalent and more severe in patients with bilio-enterostomy, biliary prosthesis, or endoscopic

sphincterotomy where enteric germs continually contaminate the bile. Treatment may require drainage, especially when antibiotic therapy has failed (Fig. 8).

Hepatic abscess after liver trauma

Since the inception of nonoperative management (NOM) of liver trauma, the overall mortality has decreased but morbidity remains elevated. HA occurs less frequently after NOM than after surgical treatment (4 vs. 7%) [44]. Two factors generally seem to increase the risk of developing HA after hepatic trauma:

- the severity grading of the trauma;
- arterial embolization.

The odds ratio (OR) for developing HA is 16 for grade 4 or higher trauma, while the incidence of HA varies from < 1% for grade I to almost 20% for grade 3 trauma. The incidence of HA after arterial embolization for traumatic bleeding is 27% (OR = 15).

The principal factor predisposing to HA formation after trauma is the extent of liver necrosis [45]. Necrosis may be either directly related to the trauma, or aggravated by intra-arterial embolization. One possible mechanism for developing HA in this context is superinfection of the necrotic areas by direct contiguous spread or by bacteremia with hematogenous seeding. Dabbs et al. [45] demonstrated bacteremia by blood culture in 31% of patients when necrosis was diagnosed after NOM. Most (90%) of the bacteria isolated from the necrotic zones were anaerobes. In their series of 37 patients, Mohr et al. found that HA was seen in 4% of patients with complicated necrosis [46]. The increased frequency of HA after intra-arterial embolization to control trauma-related hepatic bleeding and after CE for liver malignancy is probably related to associated lesions of the biliary tree and the arterio-portal network and/or to more aggressive and less selective ligation or embolization to stop bleeding. Moreover, patients undergoing embolization techniques for trauma often have multiple lesions, suffer from other stressful situations such as intensive care, particularly hypotension or low-flow states that can aggravate the ischemia and increase the extent of hepatic necrosis in this context.

Another less frequent mechanism for post-traumatic HA is superinfection of a biloma, which represents less than 7% of cases. The need for prophylactic antibiotic therapy in liver trauma is not clear and the reports in the literature are discordant [45,46]. No specific subgroup of patients who might benefit from prophylactic antibiotics has been defined. HA develops an average of 15 days after trauma (range 1—90 days) [47]. But, for anaerobic HA, and in particular, for *Clostridium* infection, the incubation time can be extremely short, less than 24—48 hours [47]. Clostridial HA has a rapid, potentially life-threatening course, probably related to initial parenchymal ischemia in association with enteric injury, promoting the rapid development of anaerobic infection [47,48]. Among patients with post-traumatic HA, a subgroup of patients that could benefit from limited surgical resection has not been well defined.

Post-transplantation hepatic abscess

Improvements in immunosuppression, peri-operative management, surgical technique, anesthesia, and intensive care techniques have improved survival rates after liver transplantation (LT) to a range of 82 to 90% at 1 year and from 62 to 80% at 10 years; however, infectious complications remain one of the major causes of morbidity and mortality [49,50].

Bacterial infections after LT have an incidence ranging from 35% to 68% and usually occur during the first month [51,52]. The responsible pathogens are those usually found in biliary infections: Gram-negative bacilli (*Escherichia coli*, *Enterobacter*), or Gram-positive cocci (*Staphylococcus aureus*, *Streptococcus*). In immunosuppressed patients, other bacteria can be found: Lactobacillus acidophilus (Gram-positive) as well as fungi (*Candidiasia*, *Aspergillosis*) or viruses (*Cytomegalovirus*) that can give rise to multiple HA. Guckelberger et al. [53] have suggested that the risk factors for infection after LT are: age < 20-years-old, biliary atresia, preoperative hypoalbuminemia, onset of gastrointestinal or vascular complications, need for postoperative hemodialysis and duration of stay in intensive care.

HA develops fairly rarely after LT, the incidence ranging from 0.5% to 1% [54]. However, this is an extremely severe complication since mortality can reach 45%, essentially because of the risk of graft loss [54]. While HA can occur early after LT, the median interval to onset is 60 days (although intervals of more than 1 year have been reported) [55].

The causes of HA after LT are noted in Table 1. The incidence of a biliary etiology varies between 6% and 34% (biliary fistula, stricture, or ischemic cholangitis) and remains the primary cause of morbidity. Mortality varies among series, ranging from 3% to 8% [56]. Approximately 20% of these complications are associated with arterial thrombosis, which also complicates the management [57] (hepatic artery thrombosis complicates LT in 3—7% of cases [58]). Onset of hepatic artery thrombosis is associated with a significant risk of HA. Kaplan et al. described a mean interval from onset of thrombosis to HA of 128 days in adults [59]. Thrombosis induces ischemia of the graft biliary tract, promoting hepatobiliary infection (ischemic cholangitis, cholangitis, abscess) [58].

The limited supply of donor livers for transplantation has led to an increased use of so-called ''marginal'' grafts, i.e. livers originating from donors who are elderly, hemodynamically unstable, in cardiac arrest, post-trauma or who have positive viral serology (presence of anti-HBc antibodies, positive hepatitis C serology). Grafts originating from donors in cardiac arrest or trauma victims seem to have a greater propensity to develop HA [60,61]. In donors with cardiac arrest, the warm ischemia phase between the cardiac arrest and cooling of the graft probably explains the onset of ischemic cholangitis with stenosis and dilatation of the intrahepatic bile ducts.

The risk of HA secondary to endoscopic maneuvers after LT (ERCP, endoscopic stent placement for biliary strictures or bile duct leaks) can attain 26% [48]. After percutaneous radiological maneuvers (biliary opacification or drainage, liver biopsy), bacteremia occurs 12 times more often in patients with a bilio-enterostomy compared with choledocho-choledochostomy [62]. In the series reported by Tachopoulou et al. [55], two of 14 transplanted patients (14%) developed HA after percutaneous liver biopsy.

Aside from split-liver grafts, non-reconstruction of an accessory hepatic artery (usually right or left hepatic artery) or accidental occlusion of bile ducts during graft preparation increases the risk of HA, usually limited [63].

Initial treatment of HA is the same as for patients who have not had a liver transplant, combining broad spectrum antibiotics with needle aspiration and/or drainage in case of unilocular HA. Repeat LT is often necessary to treat multiple

Table 1 Causes of hepatic abscess after liver transplantation. Combinations of causes are possible.

Cause	Type	Frequency
Biliary	Stricture, fistula Ischemic cholangitis	+++++
Arterial	Thrombosis Stricture	+++
Marginal quality grafts	Trauma victim donors Donors in cardiac arrest	++
Endoscopy (ERCP) Interventional radiology (TCA)	Endoprosthesis(s) Prosthesis, biopsies	+
Technical errors	No reconstruction of accessory arteries Biliary duct injury (posterior sector)	++

ERCP: Endoscopic retrograde pancreaticocholangiography; TCA: transcutaneous aspiration.

HAs secondary to biliary tract ischemia [53,64,65]. Exceptionally, if conservative treatment fails, hepatic resection may be indicated. The decision depends on the localization and the extent of the HA, and the severity of the underlying liver disease. It is therefore necessary to routinely search for biliary complications such as ischemic cholangitis when post-transplantation HA occurs. Surgical treatment of post-LT HA is associated with high morbidity, ranging from 20—62% for liver resection, and 63—79% for repeat LT. Mortality ranges from 0—29% for resection and 30—67% for repeat LT [53,64,65].

Amebic abscesses

The WHO has estimated that *Entamoeba histolytica* (EH) is responsible for 50 million infections and 100,000 deaths per year worldwide [66—68]. While amebic HA (AHA) is the most frequent extra-intestinal complication of EH infection, it only affects 3—9% of patients with the disease [66]. AHA can occur several years after enteric infection, underscoring the need for careful history taking.

After oral ingestion, EH transits into the intestines where it causes tissue destruction and mucosal ulcerations with sub-mucosal micro-abscesses. From there, the amoebae gain access to the portal system and the liver via local invasion of intestinal venules. Hepatic lesions arise from leukocyte and macrophage lysis by EH with release of toxic cytokines that lead to liver necrosis, producing the typical ''odorless chocolate pus'' [67,68].

AHA due to EH must always be considered when the patient has lived or travelled in endemic areas, since intestinal infection often passes unnoticed or is forgotten. Classic AHA presents with hepatalgia, hepatomegaly and fever [67]. Other symptoms such as deterioration of general health, pleuro-pulmonary manifestations or jaundice can be present depending on the localization of the HA. Complications include rupture of a voluminous HA into nearby organs (pleura, peritoneum, pericardium) or into the biliary tract or bronchus [67].

The diagnosis of EH reposes on identification of specific antibodies in the serum by a variety of serologic tests: indirect hemagglutination, ELISA, indirect immunofluorescence, and the Latex agglutination technique. The combination of two different techniques increases sensitivity and specificity to nearly 100% [67]. In case of initial negative serology, a second sample should be drawn 8 to 10 days later. While serology allows confirmation of diagnosis, treatment should be started as soon as possible, even before definitive diagnosis is confirmed [67].

Sonography is the best imaging modality for the diagnosis of amebic HA. Sonography can visualize a solitary lesion or multiple abscesses; in 80% of cases, HA occurs in the right lobe, but the volume and aspect vary according to the stage of maturation. Echo-guided needle aspiration can show sterile ''chocolate'' pus, and should be used freely to rule out bacterial pyogenic abscesses. CT scan is a more sensitive test in the early stages, but does not appear to be better than sonography in the later phases [67].

Metronidazole is the standard therapeutic drug: 1.5 to 2 g/day orally or intravenously for 10 days. Tinidazole or ornidazole (1.5 g/day for 5 days) is an alternative. Treatment should be completed by local intestinal amebic eradication: tiliquinol-tilbroquinol (4 gelules/day for 10 days) [67].

Echo-guided needle aspiration should be performed if the abscess is accessible, as well as percutaneous drainage if the abscess is large (> 10 cm diameter), sub-capsular, pre-rupture status, superinfected, or if there is resistance to medical treatment [67,68].

Conclusions

Hepatic abscess is a severe condition that can be life-threatening. Diagnosis relies mainly on imaging (CT scan). The frequency of etiologies varies from one geographical region to another. In Europe, most classical HA is bacterial, arising as a complication of biliary disease. In Asia, but also more and more frequently in Europe, HA due to *K. pneumoniae* is being found, possibly arising from hematogenous seeding and capable of giving rise to ocular and/or meningeal septic metastases.

For bacterial HA less than 3—5 cm in diameter, antibiotics alone should be adequate therapy, but needle aspiration helps to identify the responsible germ(s) and evacuate the contents as well as to identify biliary communication. For larger bacterial HA, antibiotics should be combined with radiological interventional or, more rarely, surgical management. Irrespective of the size of bacterial HA, it is important to determine the etiologic mechanism. The presence of bile in aspirates and/or drainage from the HA should lead to a search for associated biliary disease. A certain number of HA could be avoided by better selection of

patients, notably HA that occur after radio-frequency ablation, chemo-embolization or pancreatoduodenectomy.

Essential points

- Consider the possibility of biliary disease whenever bile is found in the aspirate or drainage fluid from an HA and perform a biliary MRI to search for associated biliary disease and/or an obstruction of the biliary tree.
- When HA complicates cholangitis, an obstruction on the common bile duct should be sought and treated in addition to treating the abscess itself.
- When an HA containing enteric tract germs occurs without any obvious cause, a gastrointestinal entry portal should be sought by CT scan and colonoscopy.
- The incidence of HA of hematogenous (arterial) origin due to *K. pneumoniae* is rising. This germ can lead to distant septic metastases.
- In case of doubt as to superinfection of hepatic metastasis, imaging or percutaneous biopsy is required.
- The mortality rate of hepatic abscess occurring after pancreatoduodenectomy in association with an occluded hepatic artery is high. Before performing pancreatoduodenectomy, celiac trunk stenosis due to arcuate ligament compression or atheroma should be eliminated.
- The presence of choledocho-enterostomy in patients with primary and/or metastatic liver tumors represents a contra-indication for intra-arterial chemo-embolization and aggravates the risks associated with radio-frequency ablation.
- Amebic abscess should be considered in all patients who have spent time in endemic areas, even many years before.
- Hepatic abscess less than 3 to 5 cm in diameter can be treated with antibiotics alone.
- Needle aspiration is more effective than one or several evacuations for certain hepatic abscesses.

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Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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