Clinical and Experimental HEPATOLOGY

Clin Exp HEPATOL 2021; 7, 4: 329–336
DOI: https://doi.org/10.5114/ceh.2021.110998
Received: 14.07.2021, **Accepted:** 19.09.2021, **Published:** 26.11.2021

Review paper

Liver abscesses – from diagnosis to treatment

Dorota Maria Kozielewicz[1], Katarzyna Sikorska[2], Piotr Stalke[3]

[1]Department of Infectious Diseases and Hepatology, Faculty of Medicine, Ludwik Rydygier *Collegium Medicum* in Bydgoszcz of the Nicolaus Copernicus University in Toruń, Poland
[2]Department of Tropical and Parasitic Diseases, Institute of Maritime and Tropical Medicine, Faculty of Health Sciences, Medical University of Gdańsk, Poland
[3]Deparment of Infectious Diseases, Faculty of Medicine, Medical University of Gdańsk, Poland

Abstract

A liver abscess is a limited, often encysted, purulent inflammation with parenchymal necrosis that is a life-threatening complication of bacterial, fungal, protozoal and worm infections. Liver abscesses can be single or multiple; most of them are located in the right liver lobe. The pathogenic factor responsible for the development of abscesses can be identified by collecting the lesion's contents or the cultures of blood, bile, sometimes urine or stool samples. Diagnosis is established by serological, imaging and microscopic tests. The treatment regimen is determined mainly by the etiological factor and the size of the lesion and includes conservative treatment and mainly percutaneous procedures. The article briefly presents the epidemiology, clinical picture and the current approach to the diagnosis and treatment of liver abscesses.

Key words: pyogenic liver abscess, amoebic liver abscess, parasitic liver diseases, mycoses, invasive fungal infections.

Address for correspondence

Prof. Dorota Maria Kozielewicz, Department of Infectious Diseases and Hepatology, Faculty of Medicine, Ludwik Rydygier *Collegium Medicum* in Bydgoszcz of the Nicolaus Copernicus University in Toruń, Poland, e-mail: d.kozielewicz@wsoz.pl

Introduction

The first liver abscesses were described by Hippocrates about 400 BCE, but their etiology, methods of diagnosis and treatment were first described in the 20th century. Liver abscesses are rare but life-threatening complications of bacterial, fungal, protozoal and worm infections. The patients' age and comorbidities (diabetes, cirrhosis, malnutrition) increase the risk of their occurrence. Immune compromised people during chemotherapy, immunosuppressive treatment, inherited or acquired immunodeficiency syndrome have a higher risk of liver abscesses caused by fungi and opportunistic microorganisms [1]. Depending on the etiology, abscesses can be classified into 4 main types: 1) bacterial, 2) protozoal, 3) parasitic and 4) fungal abscess.

In most cases of liver abscesses, it is possible to identify one of five ways of spreading the infection within the liver: 1) hematogenic: through the hepatic artery in severe septic processes as a metastatic-pyemic liver abscess or through the portal vein as a liver abscess with portal vascular thrombosis and occasionally through the umbilical vein as omphalophlebitis, 2) biliary: through the biliary tract as a result of inflammation of the gall bladder or bile ducts, as well as from invasion of parasites or foreign bodies, 3) by continuation: the spread of inflammatory processes to the liver from adjacent areas, 4) post-traumatically: after liver injuries or as a result of an intrahepatic hematoma, 5) postoperatively.

Pyogenic liver abscess

Etiology

The etiological factors of pyogenic liver abscess (PLA) are Gram-negative or positive bacteria, which are mainly the intestinal microbiota. The most common pathogens are: *Escherichia coli*, *Klebsiella pneumo-*

niae, *Enterococcus faecalis*, *Staphylococcus aureus* and *Streptococcus haemolyticus* [1]. *Klebsiella pneumoniae* is often isolated in Southeast Asia in people with colorectal cancers, but its prevalence in North America and Europe is increasing. The clinical course is more severe than in other bacterial abscesses [2, 3]. Melioidosis, caused by *Burkholderia pseudomallei*, is endemic in Thailand, Cambodia and Northern Australia [4].

Epidemiology

Pyogenic liver abscesses occur worldwide, although their prevalence varies widely geographically. A global tendency for their frequency to increase has been observed for years. PLA has an incidence rate of 1.07 to 3.59/100,000 in the West and up to 17.59/100,000 in the East [5]. Liver abscesses can be solitary or multiple. Biliary abscesses are most often multiple and affect both liver lobes, while the lesions spread through the portal vein, through continuity or as a result of trauma, are single. They are more frequently located in the right than both or left liver lobes (60-70% vs. 25% and 15% cases, respectively) [6]. The source of 30-50% of liver abscesses is biliary tract disease, but in 15-40% of patients it is unknown (Table 1) [1, 7]. Uncontrolled diabetes and colorectal cancers are risk factors for the development of cryptogenic abscesses. They are diagnosed in 15% and 24% of patients with PLA of unknown origin, respectively [1, 8]. Mortality due to PLA in developed countries is estimated at 2-19% [1]. The factors that increase the risk of death include older age, shock, acute respiratory distress syndrome, disseminated intravascular coagulation syndrome, immunodeficiency states, severe hypoalbuminemia, diabetes, neoplasms, and ineffective surgical drainage [1].

Table 1. Causes of pyogenic liver abscess

Biliary tract	Iatrogenic injury of liver/biliary tract
Cholangitis Choledocholithiasis Cholangiocarcinoma Ischemic cholangitis Congenital biliary diseases	Liver biopsy Blocked biliary stent Radiofrequency ablation/hemoembolization in the presence of infected bile Pancreatoduodenectomy Liver transplantation
Portal vein	**Hepatic artery**
Appendicitis Diverticulitis Crohn's disease	Dental infection Bacterial endocarditis Pyelonephritis Osteitis
Direct extension of:	**Secondary infection of liver**
Gall bladder empyema Perforated peptic ulcer Subphrenic abscess Kidney abscess	Liver cyst Biliary cyst Metastases
Liver trauma	**Cryptogenic**

Clinical course

Clinical signs of PLAs are often nonspecific and include fever, chills, malaise, weight loss and abdominal pain located in the upper right quadrant. The onset may be occult in the elderly. Symptoms of a primary infection may appear first, e.g. diverticulitis. Often, especially solitary PLAs are asymptomatic for a long time or cause no characteristic symptoms. Multiple abscesses are associated with an acute systemic inflammatory reaction. Sometimes, the sole symptom is fever of an unknown origin [1]. On physical examination, the liver is usually enlarged and tender. Jaundice is a late symptom of liver abscess unless there is purulent cholangitis. PLAs caused by *K. pneumoniae* are associated with septic metastatic complications affecting the eye (endophthalmitis), brain (meningoencephalitis), and fascia (necrotizing fasciitis). Unfortunately, they may persist even after successful topical treatment of the abscess [4].

Diagnosis

Laboratory test results are not characteristic, but approximately two thirds of patients have leukocytosis with anemia. Higher C-reactive protein (CRP) and procalcitonin concentrations are markers of bacterial infection. Alkaline phosphatase (ALP), bilirubin and aminotransferase activities are raised and albumin level is decreased [1]. Imaging tests are the basis of the differential diagnosis of liver abscesses. Chest X-ray imaging can show a fluid level in the abscess cavity, fluid in the right pleural cavity or elevation of the right diaphragm dome. Abdominal ultrasonography (US) remains the preferred initial method of imaging. Its advantage is the wide availability, noninvasiveness and the ability to assess both the liver in terms of the presence of focal lesions and their nature, as well as other abdominal organs in search of the source of infection. Contrast-enhanced computed tomography (CT) is helpful to identify other intra-abdominal abscesses. Due to US and CT over 90% of liver abscesses can be diagnosed and their etiology determined [9]. Magnetic resonance cholangiography (MRCP) and endoscopic retrograde cholangiopancreatography (ERCP) are used to define the localization of biliary obstruction and to allow biliary stenting and drainage (ERCP) [1]. Colonoscopy is useful in detection of large bowel infections and colorectal neoplasms [10].

Treatment

Antibiotics

Empirical broad spectrum parenteral antibiotic therapy remains one of the basic methods of treatment of PLA. It should be started immediately after suspicion of infection and modified due to pus/blood cultures drawing results. The potential source of infection and aerobic Gram-negative bacilli and Gram-positive cocci must be taken into account when an antibiotic is chosen. First line therapy includes piperacillin/tazobactam, amoxicillin/clavulanic acid or 3rd generation cephalosporins (cefotaxime, ceftriaxone) in monotherapy or in combination with an aminoglycoside (gentamicin) [11-13]. Anaerobic antibiotics, e.g. metronidazole, are used in the case of lack of effects of the current treatment or suspicion of amoebic abscess. In the elderly and those with impaired renal function a 3rd generation cephalosporin should be used. Ceftazidime is the antibiotic of choice in melioidosis [4]. Bacteria, especially from the *Enterobacteriaceae* family, may show resistance to antibiotics used in the empirical scheme; therefore it is very important to identify the microorganism early on the basis of abscess aspirate and/or blood culture and to modify the therapy. The duration of treatment varies but antibiotics should be used for a minimum of 2 to 6 weeks. After the initial intravenous therapy, the oral form can be used in most cases to complete the therapy [11-13]. A complete cure with antibiotic treatment is possible only in patients with small abscesses (the size not exceeding 3-5 cm). Most patients require, in addition to antibiotics, ultrasound or computed tomography (CT)-guided percutaneous aspiration or catheter drainage.

Percutaneous needle aspiration

Ultrasound or CT-guided percutaneous needle aspiration (PNA) is used to collect pus for microbiological examination (establishment of etiology and antibiogram). When pus contains bile it is necessary to look for a biliary tract obstruction (choledocholithiasis, neoplasms) or previously unrecognized diseases, such as sclerosing or ischemic cholangitis [14]. PNA is also used for treatment of small abscesses less than 5 cm.

Percutaneous catheter drainage

A meta-analysis of five randomized controlled trials covering 306 patients showed that outcomes in patients treated with percutaneous catheter drainage (PCD) were superior to those in patients treated with PNA in terms of clinical improvement and days to achieve a 50% reduction in abscess cavity size. No significant differences were found in duration of hospitalization or procedure-related complications [15]. Irregularly shaped abscesses, including hourglass-shaped abscesses, are effectively drained with multisite drainage to empty each cavity [16]. Hybrid drainage, which combines percutaneous and endoscopic drainage, is the method of choice in the treatment of abscesses resulting from biliary duct obstruction. In the first step, abscess drainage is performed, followed by ERCP to decompress the biliary tract [16].

Surgical drainage

The importance of surgical drainage in the treatment of PLA has decreased in the last decades. However, there are still indications to perform it: very large (> 5 cm) or multilocular abscesses, unsuccessful percutaneous drainage, presence of intra-abdominal infection (peritonitis), ruptured abscess [11, 17].

Parasitic abscesses

The most common parasitic liver abscesses are amoebic abscesses, caused by the invasion of human roundworm and liver flukes. Suppurating echinococcal cysts are sometimes also classified as parasitic abscesses. Most parasitic infections in the liver do not cause liver abscesses, but liver inflammation.

Amoebic liver abscess

Epidemiology

Entamoeba histolytica (*Eh*) affects about 50 million people, and 40,000-100,000 die due to invasive disease complications – amoebic colitis and liver abscess. The prevalence of amoebiasis is the highest in India, Mexico, Africa, and South and Central America [18-21]. Data from developed countries confirm the relationship between risky sexual behavior and amoebic transmission and a higher risk of infection in men who have sexual contacts with men compared to the general population [22-24].

Etiology

Humans, the only hosts of *Eh*, become infected after cysts enter the gastrointestinal tract. In the terminal section of the ileum, invasive, mobile trophozoites are released, migrating to the colon and dividing there. Malnutrition, immune disorders, chronic liver damage, pregnancy, alcohol intoxication, male gender, and coinfections with other pathogenic microorganisms increase the risk of a severe course of infestation and complications. The dissemination of trophozoites

to other organs occurs through the bloodstream, lymphatic drainage and the invasion of adjacent tissues [18, 25-29]. Amoebic liver abscess (ALA) develops in about 1% of infected people; it is the most common complication of intestinal amoebiasis and the most common parenteral manifestation of the infection. Men aged 18-50 who come from or travel to endemic areas develop this disease 10 times more frequently than women. It is rarely diagnosed in children [29].

Clinical course

Prolonged diarrhea occurs in 20-50% of cases. ALA can also be diagnosed in a person without previous gastrointestinal symptoms or with a subclinical course of intestinal amoebiasis. About 80% of patients with ALA will develop symptoms within a few weeks of infection. The incubation period is usually 2-6 months, although cases of ALA have been reported even several years after staying in the endemic region [18, 28]. Most acute patients report fever with chills and pain in the right upper abdomen. Less common are diarrhea, nausea, vomiting, and loss of appetite. Other symptoms include liver enlargement, tenderness in the right hypochondrium during palpation, radiating pain to the right shoulder, pain in the right side of the chest, cough, weight loss, and jaundice. An abscess in the form of a single lesion is most often located in the right liver lobe, while in the case of multiple lesions, the clinical course is more severe, with symptoms of increased toxemia. Complications of ALA include pulmonary-pleural form of amoebiasis, secondary lesions in the peritoneal cavity and pericardium, hepato-intestinal fistula, bacterial coinfections, and biliary dysfunction. Hepatic vein thrombosis and portal vein thrombosis are rare complications of ALA [18, 26].

Diagnosis

Laboratory abnormalities: increased leukocytosis, usually up to 20,000/mm^3, CRP concentration, and ALP activity may be accompanied by increased activity of aminotransferases, γ-glutamyltranspeptidase and anemia.

Abdominal imaging examinations are used in diagnosis and monitoring of the treatment effect. Abdominal ultrasound (US) imaging shows hypoechoic liver changes; contrast enhanced CT shows a heterogeneous low-density mass with fluid content and peripheral enhancement. Liver magnetic resonance imaging (MRI) shows low signal intensity of the mass in the T1 dependent image and high signal intensity with perifocal edema in the T2 dependent image.

The detection of parasites in stools confirms the diagnosis, but it is positive only in 20% of ALA cases. Direct microscopy has limited sensitivity and specify (60% and 50%) and does not enable *Eh* to be distinguished from non-pathogenic *E. dispar.* Serological assay is used to detect *Entamoeba* coproantigen (*Eh* specific Gal/GalNac lectin). Molecular identification of *Eh* DNA with a sensitivity and specificity of more than 90% is the preferred method. Parasitological and molecular examination of the aspirate from the abscess consists of direct microscopy (which frequently fails to identify trophozoites in the aspirate; few of them are concentrated on the edges of the mass) and detection of *Eh* DNA. Serological tests detect antibodies to *Entamoeba* antigens with a sensitivity of 65-92% and a specificity of > 90%. Their weakness is the possible persistence of positive results many years after the active infection has resolved [18, 29, 30].

Puncture of the abscess with aspirate collection is of diagnostic and therapeutic importance. The high density and chocolate color of the obtained aspirate are characteristic. Culture both of blood and aspirate for bacterial infections with aerobic and anaerobic flora and fungi are necessary.

Treatment

Any case of amoebiasis, including ALA, requires causal treatment. Treatment should include nitroimidazole derivatives with activity against invasive trophozoites and an active drug against cysts in the intestinal lumen, e.g. paromomycin, to eradicate the infection. Metronidazole 750 mg t.i.d. orally for 10 days, alternatively tinidazole 2.0 g daily for 5 days. In the case of severe clinical course and intolerance of oral treatment, intravenous metronidazole 500 mg t.i.d. or paromomycin 25-35 mg/kg/daily. Antiparasitic treatment implemented under strong epidemiological conditions, usually before confirming the etiology, is associated with broad-spectrum antibiotic therapy [28]. Abscess drainage is used when no improvement occurs within 72 hours of antiparasitic treatment, in suspicion of bacterial co-infection, in large lesions (> 5-10 cm) or the presence of an abscess in the left lobe due to a risk of perforation and involvement of the pleura, peritoneum and pericardium [31]. After the end of causal treatment, slow regression of lesions in the liver is observed, with the presence of residual cavernous lesions for up to 6 months [26, 29, 31].

Hepatobiliary ascariasis – *Ascaris lumbricoides* (roundworm)

Etiology

Ascaris lumbricoides is the most common human intestine parasite, infecting approximately 800 million people.

The infestation occurs via the fecal-oral route after ingestion of the invasive egg. The larvae hatching in the intestine migrate to the liver through the intestinal wall and the lymphatic and blood vessels. Most of the larvae die in the liver, and the surviving larvae reach the small intestine through the alveoli and bronchi. In the small intestine they mature and parasitize [32]. Adult worms are sometimes able to wander up the bile duct, which may result in biliary obstruction, cholangitis and formation of an abscess.

Clinical course

Abdominal pain and recurrent biliary colic with fever dominate in hepatobiliary ascariasis. Pain is not relieved by spasmolytics, as the nematode enters the bile ducts, migrating deeply into the liver parenchyma. The consequence of the process may be thrombosis of the portal vessels and the formation of abscesses. Adult nematodes may also cause pancreatitis as a result of pancreatic duct obstruction, or appendicitis.

During the migration of larval forms through the liver, granulomas with giant cells and eosinophilic margins and an inflammatory infiltrate in the portal space are formed. The extrahepatic symptoms of infection include pneumonia, exacerbation of bronchial asthma [32].

Diagnosis

Laboratory tests reveal eosinophilia and extrahepatic cholestasis. The dead nematode remains in the bile duct and may promote the formation of gallstones [32, 33]. The diagnosis is confirmed by the detection of invasive eggs in the stool or bile. It is also possible to detect the presence of parasites by contrastive X-ray examination of the gastrointestinal tract, and occasionally by abdominal US or ERCP [33, 34].

Treatment

The treatment includes albendazole in a single dose of 400 mg, or alternatively mebendazole (2 × 100 mg for 3 days or 500 mg single dose), or pyrantel pamoate 10 mg/kg in pregnancy [32].

Large liver fluke (*Fasciola hepatica*)

Etiology

It is a sheep and cattle bile duct worldwide parasite, causing sporadic infections in humans. The eggs are excreted in the feces. The larvae (miracidia) emerge from the eggs and penetrate freshwater snails (*Lymnaea* species), which, in turn, release cercarias. As metacercarias, they attach themselves to water plants, especially watercress. When eaten with the plant, they are released in the intestine and through its wall with blood they reach the liver parenchyma. After a few weeks, they settle in the bile ducts [35].

Clinical course

By migrating through the liver parenchyma, they cause inflammatory infiltration, bleeding and necrosis of hepatocytes. Their presence in the bile ducts promotes the proliferation of epithelial cells, fibrosis and constriction of the inflamed bile ducts. As a consequence, a liver abscess is formed. In the phase of invasion and migration to the liver, there is pain in the right upper abdomen, hepatosplenomegaly, fever, vomiting, diarrhea, loss of appetite, urticaria, and slight jaundice. Years later, chronic cholangitis and secondary biliary cirrhosis develop [35].

Diagnosis

Laboratory tests show leukocytosis with eosinophilia, increased activity of transaminases, bilirubin concentration, and acceleration of ESR [35]. Diagnosis is based on the detection of eggs in the stool or bile. Nodular changes (4-10 mm), small liver abscesses and biliary tract abnormalities can be diagnosed by abdominal ultrasound or CT, or by cholangiography [36, 37].

Treatment

Treatment is based on the administration of triclabendazole or alternatively with nitazoxanide, possibly also with the addition of praziquantel [38]. If oral therapy has been unsuccessful, endoscopic lavage of the biliary tract with povidone-iodine solution is recommended.

Chinese liver fluke (*Clonorchis sinensis*) and *Opisthorchis viverrini*

Etiology

Both flukes with a similar life cycle are found mainly in China, Japan and Southeast Asia. It is transmitted by the consumption of raw or undercooked freshwater fish. The main hosts are pigs, dogs, cats and humans. They eggs are excreted in the feces; they contain a miracidium, and are eaten by water snails. The latter excrete cercarias, which penetrate fish as metacercarias and encyst in their muscles. After ingestion cercaria migrate through the duodenum to the biliary tract, where they mature and live for the next several years [39].

Clinical course

Epigastric pain and fever predominate, but diarrhea, weight loss, enlarged liver, eosinophilia and mild hyperbilirubinemia with cholestasis are also common. Flukes in the bile ducts, sucking blood, stimulate the proliferation of the epithelium and the formation of adenomatous changes with accompanying peridural fibrosis. The consequences of chronic cholangitis are minor liver abscesses, cholelithiasis, and secondary biliary cirrhosis or cholangiocarcinoma [39].

Diagnosis

The diagnosis is established by stool examination for the presence of eggs, but it is more reliable to detect them in the bile. Moreover, flukes can be detected by ultrasound, MRI, and ERCP examinations.

Treatment

It consists of the administration of praziquantel (3 × 25 mg/kg) for 2 days.

Large intestinal fluke

Etiology

A disease caused by the *Fasciolopsis buski* fluke living in the jejunum and duodenum of East Asian inhabitants. The intermediate host is freshwater snails. Infection occurs through consumption of metacercariae-infected aquatic plants, especially raw water nuts.

Clinical course

The infection leads to fever, weight loss, vomiting, abdominal pain, bloody diarrhea, anemia and ascites, as well as cholangitis and liver abscess with appropriate laboratory test results [36-38].

Treatment

See Chinese liver fluke.

Siberian liver fluke (*Opisthorchis felineus*)

Etiology

The other name is cat fluke; it occurs mainly in Asia and to an increasing extent also in Europe. The egg development process and life cycle are similar to the Chinese liver fluke.

Clinical course and treatment

See Chinese liver fluke.

Cestodes

Etiology

Apart from echinococcosis caused by 4 species – *Echinococcus granulosus sensu lato*, *E. multilocularis*, *E. oligarthrus* and *E. vogeli*, tapeworm invasions do not damage the liver and bile ducts. Liver abscesses develop as a result of infection of the echinococcal cyst (bacterial and fungal abscesses) or the death of the parasite as a result of pharmacological treatment or their natural evolution. The occurrence of a liver abscess in the course of echinococcosis is more often observed in the case of *E. granulosus s.l.* invasion than *E. multilocularis* [40].

Clinical course

Clinical symptoms appear at the earliest after 3-5 years; the peak of their manifestation is in the decade(s) after infection. Patients occasionally complain of fatigue, food intolerance, itching, weight loss and episodes of fever.

Diagnosis

Echinococcosis diagnosis is based on abdominal ultrasound, CT and MRI, ELISA and Western Blots tests. Except eosinophilia (30-40% of cases) and occasional increases of γ-glutamyl transpeptidase (GGTP), ALP or transaminases, laboratory parameters are normal.

Treatment

Patients with echinococcal liver abscess require antibiotic therapy due to bacterial superinfection or even surgical treatment. Preoperative use of albendazole is recommended when there is a risk of dissemination of protoscoleces to surrounding tissues during invasive procedures [40].

Fungal abscesses

Etiology

Fungal infections of the liver and bile ducts are classified as opportunistic infections, most often caused by the optional pathogenic *Candida* (*C. albicans*, *C. glabrata*, *C. krusei*, *C. parapsilosis*, *C. tropicalis*) or *Aspergillus* (*A. flavus*, *A. fumigatus*, *A. niger*). Among other factors, fungi of the genus *Trichosporon* (*T. beigelii*,

T. dermatis), *Coccidioides immitis*, *Torulopsis glabrata* and *Mucormycosis indicus* should be mentioned [41].

Clinical course

Factors predisposing to fungal invasions are congenital or acquired immune deficiencies, but some cases without noticeable immunosuppression have also been reported. The most common symptoms of fungal infections in the liver are the loss of appetite, worsening malaise, tenderness in the right upper abdomen or clinical symptoms such as fever of unknown origin, especially in the absence of response to antibiotics, enlargement of the liver and/or spleen, and jaundice.

Initially, the invasion of *Candida* spp. and *Aspergillus* spp. affects the mucous membranes, but further progression of the disease through the circulatory system (fungemia) involves subsequent internal organs, including the liver, leading to the formation of micro-abscesses [41, 42]. Sepsis is a rare form of trichosporosis and coccidioidomycosis when liver granulomas and micro-abscesses may be formed [43, 44]. Hepatic mucormycosis is rare and a case of liver abscesses in an immunocompromised patient has been reported as a result of dissemination of the fungus from ileocecal mucormycosis [45].

Diagnosis

Particularly important in the diagnosis of fungal liver abscesses are abdominal US, CT and MRI imaging [46]. US may reveal numerous tiny lesions that are usually detectable as hypoechoic areas. The most characteristic image is the "circle in a circle" (a hypoechoic center containing necrotic tissues surrounded by a hyperechoic layer of inflammatory cells) or a "shield" symptom [47]. It is very important to combine ultrasound image analysis with clinical and laboratory data (abnormal value CRP, aminotransferases, ALP, bilirubin). Eosinophilia is found in coccidioidomycosis [44]. Sometimes US-guided fine-needle aspiration biopsy (aspiration of contents for microbiological examination) or contrast enhanced CT or MRI examinations are helpful [44-46].

Treatment

Pharmacotherapy of fungal liver abscesses does not differ from the treatment of organ mycoses. The primary drug is liposomal amphotericin B 5 mg/kg not less than 2 weeks (possibly in combination with 5-fluorocytosine 100 mg/kg, fluconazole 800 mg next 400 mg daily and itraconazole 3 × 200 mg – 2-5 months!) [48].

Disclosure

The authors declare no conflict of interest.

References

1. Kriege JE, Beckingham IJ. ABC of diseases of liver, pancreas, and biliary system. BMJ 2001; 322: 537-540.
2. Qu K, Liu C, Wang ZX, et al. Pyogenic liver abscesses associated with nonmetastatic colorectal cancers: an increasing problem in Eastern Asia. World J Gastroenterol 2021; 18: 2948-2955.
3. Fang CT, Lai SY, Yi WC, et al. Klebsiella pneumoniae genotype K1: an emerging pathogen that causes septic ocular or central nervous system complications from pyogenic liver abscess. Clin Infect Dis 2007; 45: 284-293.
4. Maude RR, Vatcharapreechasakul T, Ariyaprasert P, et al. Prospective observational study of the frequency and features of intra-abdominal abscesses in patients with melioidosis in northeast Thailand. Trans R Soc Trop Med Hyg 2012; 106: 629-631.
5. Xu S, Shi B, Chao LM, et al. Prognostic normogram for the combination therapy of percutaneous catheter drainage and antibiotics in pyogenic liver abscess patients. Abdom Radiol (NY) 2020; 45: 393-402.
6. Palczewski P, Gołębiowski M, Cieszanowski A, et al. Zapalne zmiany ogniskowe. Ropień wątroby. W: Diagnostyka obrazowa guzów wątroby. Rozpoznanie i kwalifikacja do leczenia. Gołębiowski M, Małkowski P, Zieniewicz K (red.). Wyd. Naukowe PWN, Warszawa 2011; 186-193.
7. Mavilia MG, Molina M, Wu GY. The evolving nature of hepatic abscess: a review. J Clin Transl Hepatol 2016; 4: 158-168.
8. Jeong SW, Jang JY, Lee TH, et al. Cryptogenic pyogenic liver abscess as the herold of colon cancer. J Gastroenterol Hepatol 2012; 27: 248-255.
9. Halvorsen RA, Korobkin M, Foster WL, et al. The variable CT appearance of hepatic abscesses. Am J Roentgenol 1984; 142: 941-946.
10. Lardiére-Deguelte S, Ragot E, Amroun K, et al. Hepatic abscess: diagnosis and management. J Visc Surg 2015; 152: 231-243.
11. Hope WW, Vrochides DV, Newcomb WL, et al. Optimal treatment of hepatic abscess. Am Surg 2008; 74: 178-182.
12. Bamberger DM. Outcome of medical treatment of bacterial abscesses without therapeutic drainage: review of cases reported in the literature. Clin Infect Dis 1996; 23: 592-603.
13, Solomkin JS, Mazuski JE, Bradley JS, et al. Diagnosis and management of complicated intra-abdominal infection in adults and children: guidelines by the Surgical Infection Society and the Infectious Diseases Society of America. Clin Infect Dis 2010; 50: 133-164.
14. Sugiyama M, Atomi Y. Pyogenic hepatic abscess with biliary communication. Am J Surg 2002; 183: 205-208.
15. Cai YL, Xiong XZ, Lu J, et al. Percutaneous needle aspiration versus catheter drainage in the management of liver abscess: a systematic review and meta-analysis. HPB (Oxford) 2015; 17: 195-201.
16. Zerem E, Hadzic A. Sonographically guided percutaneous catheter drainage versus needle aspiration in the management of pyogenic liver abscess. Am J Roentgenol 2007; 189: 138-142.
17. Tan YM, Yaw-Fui Chung A, Kah-Hoe Chow P, et al. An appraisal of surgical and percutaneous drainage for pyogenic liver abscesses larger than 5 cm. Ann Surg 2005; 241: 485-490.
18. Stanley SL. Amoebiasis. Lancet 2003; 361: 1025-1034.

19. Liu J, Platts-Mills JA, Juma J, et al. Use of quantitative molecular diagnostic methods to identify causes of diarrhoea in children: a reanalysis of the GEMS case-control study. Lancet 2016; 388: 1291-1301.
20. Kumanan T, Sujanitha V, Sreeharan N. Amoebic liver abscess: a neglected tropical disease. Lancet Infect Dis 2020; 20: 160-162.
21. Leder K, Torresi J, Libman MD, et al. GeoSentinel surveillance of illness in returned travelers, 2007-2011. GeoSentinel Surveillance Network. Ann Intern Med 2013; 158: 456-468.
22. Weinke T, Friedrich-Jaenicke B, Hopp P, et al. Prevalence and clinical importance of Entamoeba histolytica in two high-risk groups: travelers returning from the tropics and male homosexuals. J Infect Dis 1990; 161: 1029-1031.
23. Huang SH, Tsai MS, Lee CY, et al. Ongoing transmission of Entamoeba histolytica among newly diagnosed people living with HIV in Taiwan, 2009-2018. PLoS Negl Trop Dis 2020; 14: e0008400.
24. Watanabe K, Gatanaga H, Escueta-de Cadiz A, et al. Amebiasis in HIV-1-infected Japanese men: clinical features and response to therapy. PLoS Negl Trop Dis 2011; 5: e1318.
25. Kantor M, Abrantes A, Estevez A, et al. Entamoeba histolytica: updates in clinical manifestation, pathogenesis, and vaccine development. Can J Gastroenterol Hepatol 2018; 2018: 4601420.
26. Wuerz T, Kane JB, Boggild AK, et al. A review of amoebic liver abscess for clinicians in a nonendemic setting. Can J Gastroenterol 2012; 26: 729-733.
27. Shirley DT, Farr L, Watanabe K, et al. A review of the global burden, new diagnostics, and current therapeutics for amebiasis. Open Forum Infect Dis 2018; 5: 161.
28. Mukhopadhyay M, Saha AK, Sarkar A, et al. Amoebic liver abscess: presentation and complications. Indian J Surg 2010; 72: 37-41.
29. Kannathasan S, Murugananthan A, Kumanan T, et al. Epidemiology and factors associated with amoebic liver abscess in northern Sri Lanka. BMC Public Health 2018; 18: 118.
30. Elizondo G, Weissleder R, Stark DD, et al. Amebic liver abscess: diagnosis and treatment evaluation with MR imaging. Radiology 1987; 165: 795-800.
31. Cai YL, Xiong XZ, Lu J, et al. Percutaneous needle aspiration versus catheter drainage in the management of liver abscess: a systematic review and meta-analysis. HPB (Oxford) 2015; 17: 195-201.
32. Fogaca HS, Oliveir CS, Barbosa HT, et al. Liver pseudotumor: a rare manifestation of hepatic granulomata caused by Ascaris lumbricoides ova. Am J Gastroenterol 2000; 95: 2099-2101.
33. Ferreyra NP, Cerri GG. Ascariasis of the alimentary tract, liver, pancreas and biliary system: its diagnosis by ultrasonography. Hepatogastroenterology 1998; 45: 932-937.
34. Al-Karawi M, Sanai FM, Yasawy MI, et al. Biliary strictures and cholangitis secondary to ascaris: endoscopic management. Gastrointest Endosc 1999; 50: 695-697.
35. Teichmann D, Grobusch MP, Gobels K, et al. Acute fascioliasis with multiple liver abscesses. Scand J Gastroenterol 2000; 32: 558-560.
36. Beers van B, Pringot J, Geubel A, et al. Hepatobiliary fascioliasis: non-invasive imaging findings. Radiology 1990; 174: 809-810.
37. Condomines J, Rene-Espinet JM, Espinos-Perez JC, et al. Percutaneous cholangiography in the diagnosis of hepatic fascioliasis. Am J Gastroenterol 1985; 80: 384-386.
38. Schiappacasse RH, Mohammadi D, Christie AJ. Successful treatment of severe infection with Fasciola hepatica with praziquantel. Trop Med Parasit 1985; 36: 88-90.
39. Ona FV, Dytoc JN. Clonorchis-associated cholangiocarcinoma: a report of two cases with unusual manifestations. Gastroenterology 1991; 101: 831-839.
40. Bosanac ZB, Lisanin L. Percutaneous drainage of hydatid cyst in the liver as a primary treatment: Review of 52 consecutive cases with long-term follow-up. Clin Radiol 2000; 55: 839-848.
41. Castaldo P, Stratta RJ, Wood RP, et al. Clinical spectrum of fungal infections after orthotopic liver transplantation. Arch Surg 1991; 126: 149-156.
42. Ow C, Maldjian C, Shires GT, et al. CT, US and MR imaging of hepatic aspergilloma. J Comput Assist Tomogr 1991; 15: 852-854.
43. Korinek JK, Guarda LA, Bolivar R, et al. Trichosporon hepatitis. Gastroenterology 1983; 85: 732-734.
44. Coodley EL. Disseminated coccidioidomycosis: diagnosis by liver biopsy. Gastroenterology 1967; 53: 947-952.
45. Borg Ter F, Kuijper EJ, van der Lebe H. Fatal mucormycosis presenting as an appendiceal mass with metastatic spread to the liver during chemotherapy-induced granulocytopenia. Scand J Infect Dis 1990; 22: 499-501.
46. Berlow ME, Spirt BA, Wen L. CT follow-up of hepatic and splenic fungal microabscesses. J Comput Assist Tomogr 1984; 8: 42-45.
47. Pastakia B, Shawker TH, Thaler M, et al. Hepatosplenic candidiasis: wheels within wheels. Radiology 1988; 166: 417-421.
48. Tsaousis G, Koutsouri A, Gatsiou C, et al. Liver and brain mucormycosis in a diabetic patient type 2 successfully treated with liposomial amphotericin B. Scand J Inf Dis 2000; 32: 335-337.