



Invited Review

Liver abscess: diagnostic and management issues found in the low resource setting

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Abstract

Introduction: Liver abscesses are mainly caused by parasitic or bacterial infection and are an important cause of hospitalization in low-middle income countries (LMIC). The pathophysiology of abscesses is different depending on the etiology and requires different strategies for diagnosis and management. This paper discusses pathophysiology and epidemiology, the current diagnostic approach and its limitations and management of liver abscess in low resource settings.

Sources of data: We searched PubMed for relevant reviews by typing the following keywords: ‘amoebic liver abscess’ and ‘pyogenic liver abscess’.

Areas of agreement: Amoebic liver abscess can be treated medically while pyogenic liver abscess usually needs to be percutaneously drained and treated with effective antibiotics.

Areas of controversy: In an LMIC setting, where misuse of antibiotics is a recognized issue, liver abscesses are a therapeutic conundrum, leaving little choices for treatment for physicians in low capacity settings.

Growing points: As antimicrobial resistance awareness and antibiotic stewardship programs are put into place, liver abscess management will likely improve in LMICs provided that systematic adapted guidelines are established and practiced.

Areas timely for developing research: The lack of a quick and reliable

diagnostic strategy in the majority of LMIC makes selection of appropriate treatment challenging.

Key words: liver abscess, amoebic, pyogenic, resource limited settings, low-middle income countries

Pathophysiology

Liver abscesses can be broadly divided into two categories: amoebic and pyogenic (see Table 1). The pathogenesis of amoebic liver abscess (ALA) is different from pyogenic liver abscess (PLA).¹ In the former, *Entamoeba histolytica* induces hepatic apoptosis and the latter is a suppurative infection of the liver parenchyma. Confirmatory diagnosis is important, albeit difficult in resource limited settings, as it leads to appropriate management.

Epidemiology

PLAs have a global distribution, although incidence varies significantly between different countries from more than 900 cases in a 10-year period in Asian countries such as Taiwan, Singapore and South Korea to 23 cases in the same timeframe in non-Asian regions.² In the US,³ the incidence of PLA is 2.3 per 100 000, predominantly in older men and diabetes and cancer are considered risk factors to the development of PLA. The most common pathogen isolated in this setting was *Streptococcus milleri* followed by *Klebsiella pneumoniae*. This differs from South Korea and Taiwan, where *K. pneumoniae* is the most common pathogen found in PLA.^{1, 4}

Entamoeba histolytica is a protozoan that causes amebiasis (gastrointestinal infection) and the most common cause of intestinal parasite infection in returned travelers.⁵ *Entamoeba histolytica* is globally distributed with higher rates of infection in low-middle income countries (LMIC) settings compared to high income countries (HIC). Furthermore, significant proportion of cases in HIC is usually imported, while non-imported cases usually affect immunosuppressed patients.⁶ Infection is associated

to poor living conditions and contamination of drinking water. A good example of this was shown by high amoebiasis rates (63/1000 children) in Thai-Cambodian border refugees between 1987 and 1989.⁷

The most common extra-intestinal manifestation is liver abscess, with parasite being carried to the liver via the portal vein. The incidence of the disease is highest in Asia, where rates can be as high as 21 per 100 000 inhabitants per year.⁸ ALA predominantly affects middle age (30–60 years old) men. Risk factors include alcohol consumption and malnutrition (low body mass and hypoalbuminemia).⁹

Pathogenesis of liver abscess

A pyogenic abscess is defined as a collection of pus consisting of numerous inflammatory cells, notably neutrophils and tissue debris.¹⁰ Infection is associated with necrosis from inflammation of surrounding tissue.

The word abscess may represent a misnomer when it is used to define the pathologic process caused by *E. histolytica* in the liver. In the case of ALA, there is hepatocyte cell death either by apoptosis or necrosis.^{11,12} It is generally agreed that there is an absence of inflammatory cells due to lysis of neutrophils by the protozoan forming the typically described non-purulent ‘anchovy paste’ abscess.¹ Cell death will continue to occur with expansion of the abscess until patient receives appropriate treatment. Of note, a hamster study revealed that soon after seeding *E. histolytica* into liver parenchyma, inflammatory cells mainly consisting of polymorphonuclear surrounded the parasite and were subsequently lysed along with hepatocytes.¹³

Table 1 Differences between amoebic and pyogenic abscess

	Amoebic abscess	Pyogenic abscess
Pathogen	<i>Entamoeba histolytica</i>	<i>Klebsiella pneumoniae</i> , <i>Streptococcus milleri</i> , <i>Escheria coli</i> , <i>Burkholderia pseudomallei</i> , <i>Staphylococcus aureus</i> , Polymicrobial including anaerobes
Distribution	Globally, higher rates in LMICs, typically males 30–50 years	Globally, older patients
Acquisition	Poor sanitation, contaminated drinking water	Biliary source, e.g. impacted gall stone Systemic infection
Pathogenesis	Inflammation—abundant neutrophils	Necrosis—absence of neutrophils
Imaging	Usually single (can be multiple) Typically in right lobe (can be in left lobe) Cold appearance on sulfur colloid scan	Either single or multiple Any lobe can be involved Hot appearance on sulfur colloid scan
Fine needle aspirate	Macroscopic—thick, chocolate brown, odourless, ‘anchovy paste’ Microscopy for trophozoites—insensitive (25%) Antigen testing—sensitive and specific, generally not available in LMICs PCR—sensitive and specific, generally not available in LMICs	Macroscopic—purulent, may be foul smelling Culture—limited availability in LMICs
Other diagnostic modalities	Serology—useful in returned travelers, limited role in residents of high endemicity Antigen testing of serum—sensitive and specific, generally not available in LMICs	Blood cultures—sensitivity 50%, limited availability in LMICs, in LMICs patients often pre-treated with antimicrobials prior to specimen collection.
Treatment	Medical therapy with metronidazole usually sufficient. (May require drainage in co-infection or impending rupture.)	Percutaneous drainage along with antibiotics is mainstay of therapy. Antibiotic treatment in small responsive abscesses.

Causative organisms of pyogenic liver abscess

PLA may be caused by a variety of organism, including *K. pneumoniae*, *Escherichia coli* and *Burkholderia pseudomallei*.¹⁴ The microbiology differs according to the presumed route of hepatic invasion. Infections may arise from the biliary tree (usually from an impacted gallstone), circulation (portal vein, hepatic artery), a contiguous focus of infection and penetrating trauma. In the South–East Asian region, patients working with soil and water with comorbidities such as diabetes, liver and renal failure and hazardous consumption of alcohol are also at risk of infection with *B. pseudomallei*.¹⁵

There are challenges in defining the different microbiological pathogens, which cause PLA. One reason for this problem is that it is common that pus from a liver abscess is collected after administration of antibiotics.¹ This may lead to an under-estimation of bacteria causing liver abscess and may also contribute to a gap in physician knowledge to determine which antibiotic is most appropriate for treatment. This raises another issue of selection bias, where the positive culture results may have a more resistant profile if patients had received antimicrobial therapy prior to culture. It may also under-estimate the number of susceptible pathogens

that have been rendered culture negative by prior treatment. Laboratory capacity in culture and identification may be limited in LMICs. Examples include inability to test due to lack of culture capabilities and lack of anaerobic culture facilities such that a negative culture may not equate an absence of growth. Studies have identified that gram-negative rods such as *E. coli*, *K. pneumoniae*, anaerobes, *S. milleri* and *Staphylococcus aureus* are important causative pathogen.¹⁶ The source of infection usually arises from the biliary, intestinal tract or portal system with subsequent seeding of the liver.

In Taiwan,¹⁷ *K. pneumoniae* is an important pathogen that is frequently isolated. Although multi-resistant strains of *K. pneumoniae* have been increasingly observed in these settings, the *K. pneumoniae* isolates responsible for liver abscess have generally remained susceptible. The study described 182 cases of liver abscesses between 1990 and 1996, 88% ($n = 160$) were caused by *K. pneumoniae*, diabetes was a frequent risk factor. It has been observed that gas-forming *K. pneumoniae* liver abscess is thought to be associated with worse prognosis.¹⁸ Patients with diabetes mellitus are at increased risk of developing gas-forming primary liver abscess and infectious metastatic disease. The study hypothesizes that the gas formation process may be caused by high level of glucose in tissues, which allows for vigorous metabolism and growth of *K. pneumoniae*. Toxic by-products of inflammation accumulate with delayed clearance by circulation due to microangiopathy, which delays the transport of end products out of the lesion. This would suggest that good glycemic control is also important for controlling the infection and improved clinical outcome.¹⁸

Melioidosis is an important cause of liver abscess in Southeast Asia.¹⁵ The infection is caused by *B. pseudomallei*, a saprophytic gram-negative bacillus found in the environment. Patients working in close contact with soil and water such as rice farmers and people with weakened immune systems, such as diabetics, renal or liver impairment or thalassemia are at most risk of contracting infection. Transmis-

sion of the pathogen is either via ingestion, inhalation or inoculation and can cause various types of infections such a sepsis, pneumonia and deep-seated abscesses. In a northeast Thailand study, 33% ($n = 77/230$) cases had deep-seated abscesses, liver abscess only 26% ($n = 20/77$) liver and spleen abscess 31% ($n = 24/77$). It was observed that the majority (70% $n = 31/44$) of liver abscesses had multiple lesions. Over one-third ($n = 16$) cases underwent percutaneous incision and drainage and splenectomy was performed in two cases.¹⁵ This infection has also been recognized in Cambodia, an LMIC in SE Asia.¹⁴ Although the disease is still under-recognized, increased utilization of the microbiology services shows that the disease is endemic throughout Cambodia.

Diagnosis of liver abscess

The clinical presentation of both amoebic and PLA is indistinguishable. Patients usually present with fever and right upper quadrant tenderness. Although laboratory tests, such as leukocytosis (predominantly neutrophils), raised inflammatory markers (e.g. C-reactive protein), increased alkaline phosphatase and abnormal liver function tests are often present they have no real value in differentiating amoebic versus PLA.¹

Imaging techniques, such as ultrasonography and computed tomography (CT) scanning, are useful tools to demonstrate a space occupying lesion and confirm presence or absence of a liver abscess, it may not reliably differentiate between PLA and ALA.¹⁹ Traditionally, ALA most commonly occurs as a single lesion in the right lobe but can be present in the left lobe and be multiple.¹ CT scanning has a higher sensitivity (97% sensitive) compared to ultrasound (85% sensitive) for detection of liver abscess,²⁰ although this modality may not always be accessible in an LMIC setting.

Fine needle aspiration for culture is the gold standard for diagnosis of PLA. This is not the case for ALA as parasite culture is insensitive and not routinely available in clinical laboratories. Microscopy also lacks sensitivity as trophozoites

are seen in <25% of cases.¹ The macroscopic aspect of the aspirate may provide some preliminary information on the cause of the liver abscess. Traditionally, ALA is odourless, chocolate brown and thick, and commonly referred to as anchovy paste⁹ while PLA is usually purulent and foul smelling, particularly as a result of infection with anaerobes. Although this may be helpful, its role in differentiation for the purpose of diagnosis remains uncertain.

Blood cultures are an important adjunct to the diagnosis of pyogenic abscess and although their yield is usually lower than pus aspirate of liver abscess, they may provide helpful information in patients before they receive antimicrobials or aspiration of their abscess. It is recommended to perform a blood culture for any patient suspected of liver abscess on entry.¹

Serology can be useful in returned travelers who have visited areas of high endemicity and reside in low endemicity settings. Due to long-term positivity following exposure, it is of less value in high endemicity settings where patients may have been previously exposed.¹⁹ The test can also be falsely negative in case of acute presentations, patient's immune response, the type of serologic test or the pathogen strain.²¹

Antigen testing may be useful in LMICs. The TechLab *E. histolytica* II Antigen Detection test detects the presence of Gal/GalNAc antigen in serum and is both sensitive ($\geq 95\%$) and specific (100%, $n = 70$ controls including nine PLA).¹⁹ Sensitivity decreases significantly in patients who have been pre-treated with metronidazole prior to testing. The accessibility of the antigen detection testing may also be a potential barrier to its access in an LMIC.

Another new potential marker such as pyruvate phosphate dikinase in the form of a lateral flow assay shows potential in the diagnosis of ALA.²² There remains a need for tests that are non-invasive, accurate, readily available and affordable in the field of diagnostics for ALA.

As most patients with ALA have no bowel symptom, examination of stool for ova and parasite and antigen testing is insensitive and not recommended.

Stool testing therefore has no real value in diagnosis of liver abscess.

Molecular testing of liver abscess contents is reliable for the diagnosis of ALA.²³ Although this test offers the possibility to accurately diagnose *Entamoeba* infection, the availability of molecular test in LMIC settings is limited as it requires dedicated equipment and costly consumables.

In HIC, cause of liver abscess is usually determined using multiple diagnostic strategies, including blood cultures, *Entamoeba* serology, liver abscess aspirate for culture and molecular and antigen testing. Each of these individual options is challenged in the LMIC setting. In the LMIC setting, a patient will usually present, following failure to respond to initial antibiotic therapy, imaging reveals an abscess and the cause remains undifferentiated, due to limited testing capacity. LMICs often lack essential microbiology services and where available utilization of services is often poor.²⁴ Specimen collection should be performed prior to antibiotics, if clinical presentation allows, however in LMICs collection of specimens often occurs late and is generally reserved for patients who have failed to respond to antimicrobial treatment. In LMICs, where it is common for patients to receive medication prior to hospitalization, from either pharmacies or private clinics ($\geq 50\%$ of all transactions in Asia),^{25,26} extends to a wide variety of medication including antimicrobials.²⁷ The reasons for patients to favor receiving medications from the pharmacy are multiple, including easy accessibility, possibility to purchase medication in small quantities and familiarity with the dispenser.²⁶ Insufficient training in staff working in pharmacies result in restrictions in terms of their knowledge and availability of products.²⁸ It is also recognized²⁸ that dispensing of medication has insufficient regulation resulting in uncontrolled dispensing.

Treatment

In LMICs, antimicrobial guidelines generally recommend empiric therapy targeting both amoebic and pyogenic causes of liver abscess. As treatment is

often administered prior to collection of appropriate specimens, the causative pathogen and prevalence of either disease remain unclear. Development of empirical antibiotic guidelines, with selection of the most appropriate antimicrobials for the treatment of liver abscess, is hindered by a lack of local microbiology data. As a result, recommendations are often not tailored to the local setting and taken from other settings.

ALA is managed medically, while combined infections and PLA require both drainage either by repeated needle aspiration or percutaneous catheter drainage^{9,29} and appropriate antimicrobial treatment. Surgical drainage is usually reserved for complicated cases and has now been replaced by less invasive methods as the standard of care.⁹

The mainstay of treatment for ALA is either metronidazole or tinidazole orally for a period of 10 days or 5 days, respectively. This is followed by treatment with a luminal agent such as paromomycin for a period of 5–10 days to eradicate any remaining cysts in the intestinal tract. Most cases of ALA respond to medical treatment, while patients not responding to medical treatment should undergo drainage.^{1,19} Drainage is required for complications of infection, which include patients who have secondary bacterial infection (either *de novo* or secondary to drainage) and patients who are considered high risk of ALA rupture.

The treatment for PLA has evolved over the years, from open surgical drainage to percutaneous drainage aided by imagery. There is uncertainty regarding which type of liver abscess should receive antimicrobials only versus drainage.³⁰ Current recommendations are that liver abscesses less than 3 cm can be treated medically.³¹ Aspirations of liver abscesses are effective and lead to resolution in a high percentage of patients. Repeated aspiration incrementally increases the likelihood of management success following each aspiration.³² The use of needle aspiration is an attractive option for low-middle income settings where availability of materials is limited. In LMICs, it is preferable to avoid insertion of a drain, as they can be difficult to manage and be a source of secondary infections. More studies

are needed to confirm the optimal approach of liver abscess management in LMIC setting.

The selection of an appropriate antibiotic will differ according to the isolated pathogen, susceptibility pattern and local epidemiology. For example, the recommended treatment for melioidosis is ceftazidime,¹⁵ while meropenem might be recommended for infection with ESBL producing *K. pneumoniae*.

Prognosis of patients affected by liver abscess

The prognosis of PLA is dependent on the time to diagnosis.² Patient's with delayed diagnosis are more likely to need medical treatment with drainage procedure. Patients who present with shock acute renal failure and acute respiratory failure were likely to have poor outcomes.

Worldwide, *E. histolytica* is an important cause of mortality, only second to malaria as a cause of death from parasitic disease.¹ ALA is a progressive and uniformly fatal disease if left untreated. Patients with ALA have a favorable outcome when treatment is commenced in a timely manner.¹¹ Complex and ruptured abscesses are associated with increased mortality.

Conclusions

In the LMIC setting, both amoebic and pyogenic abscesses are prevalent and have similar clinical presentations. Current diagnostic testing strategies have limitations in relation to implementation in LMIC settings, and as a result, it can be challenging to accurately identify the causal pathogen. This leads to issues regarding the optimal management of liver abscesses in LMICs.

Despite limitations with sensitivity and availability, blood cultures should be collected in all patients presenting with a liver abscess. Large pyogenic abscesses require drainage; aspirations (repeated if necessary) are an appropriate treatment modality for LMICs. Culture of aspirated liver contents

should always be performed to ensure targeted antimicrobial therapy.

There are currently few tests available for rapid and affordable diagnosis of ALA in countries where infection is common. Introduction of a reliable bedside diagnostic test, e.g. serum antigen testing, for ALA in LMICs would increase detection rates of ALA. Treatment of ALA is in the most part medical and so improved diagnostics would avoid unnecessary drainage procedures and subsequent complications. This would also allow a reduction in the empiric use of antimicrobials, for the treatment of PLA, and reduce selection pressure for the development of antimicrobial resistance.

An area of consideration for research could be to rely on systematic blood cultures and aspiration of abscesses that are amenable to drainage. Although this would not be feasible for small abscesses, macroscopic observation and testing of content aspirate for *E. histolytica*, by antigen or molecular testing and microbiology, could be helpful in stratifying patients and deciding which treatment protocol would be most appropriate. This invasive approach would mean that patients with *E. histolytica* infection would have an aspirate to eliminate co-infection.

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Conflict of interest statement

The authors have no potential conflicts of interest.

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