



Pyogenic Liver Abscess

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8.1 Introduction

A hepatic abscess is a suppurated cavity caused by the invasion and multiplication of microorganisms within the liver parenchyma [1]. It can be bacterial (pyogenic), parasitic (amebic), mixed (pyogenic superinfection of parasitic abscess), or rarely fungal. Adult pyogenic liver abscess (PLA) is a major hepatobiliary infection with mortality of up to 46% [2–4]. The recent improvement in mortality can be attributed to improved understanding of sepsis with advances in interventional radiology and critical care. The cornerstone of PLA treatment remains antibiotic therapy with percutaneous drainage/aspiration. The first significant study on PLAs was performed by Ochsner in 1938, and he reported association with young men, portal pyemia, and 77% mortality [5].

8.2 Epidemiology

Incidence of PLA is increasing globally [2]. Prevalence is more common in Asia than in the West. Taiwan has a prevalence of 18/100,000 compared to up to 4/100,000 in the West [6–8]. The characteristics of PLA in the Asian and Western population also differ [9]. A greater proportion of Western patients have underlying malignancy or hepatobiliary-pancreatic pathologies, while Asian patients present with cryptogenic PLA or biliary pathologies. *Staphylococcus* or *Streptococcus* species were the more common causative organisms in the West, as opposed to *Klebsiella pneumoniae* in Asia. Alarmingly, there is an increasing prevalence of

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extended-spectrum beta-lactamases (ESBL)-producing *Klebsiella pneumoniae*, from 1.6% of initial blood cultures in 2001 to 14.3% in 2011 [2]. This increase in resistance is likely to be due to widespread use of broad-spectrum third-generation cephalosporins. PLA patients are more likely to have type 2 diabetes mellitus (DM), and there is a significant rise in malignancy as an etiology [2]. A recent case control study from Taiwan has reported that proton pump inhibitor use is associated with greater risk of PLA [10].

8.3 Etiology

PLA arises due to the invasion of the liver parenchyma by microorganisms via the blood stream (hematogenous, most often portal), bile ducts, or contiguously especially via the gallbladder bed [11]. Hematogenous spread can occur via the portal venous system infections such as appendicitis, enteritis, or colitis. PLA may also arise from extra-abdominal infections or preexisting liver lesions (biliary cysts, hydatid cysts, or necrotic metastases). Biliary spread may result in the course of intra-abdominal biliary infections that contaminate the biliary tract. PLA may complicate surgical procedures that involve bilio-enteric anastomosis (pancreaticoduodenectomy) and immunosuppression (liver transplantation). Widespread adoption of interventional radiology procedures such as radiofrequency ablation (RFA) or trans-arterial chemo-embolization (TACE) for hepatocellular carcinoma (HCC) is also associated with PLA. In a local study reporting on 103 patients with acute cholecystitis treated with percutaneous cholecystostomy, 12.6% of patients had contiguous PLA on imaging [12]. PLA is also known to complicate patients with recurrent pyogenic cholangitis (RPC) [13]. In a study reporting on 319 patients with RPC, Law ST et al. reported that PLA in patients with RPC has distinct clinical, microbiologic, and radiologic patterns; however, outcomes with antibiotics and percutaneous drainage are comparable [14].

8.4 Bacteriology

The mainstay of the treatment of PLA is antibiotics. Ideally all patients should receive targeted antibiotic therapy [15]. In PLA patients, blood culture is the prime source for microbial identification. The pus from percutaneous aspiration or drainage also allows for microbial isolation.

8.4.1 *Klebsiella pneumoniae* Pyogenic Liver Abscess (KPPLA)

The past two decades have seen an increasing prevalence of *Klebsiella pneumoniae* as the leading pathogen, especially in Asian countries [16]. KPPLA is reported to be associated with larger size of PLA [17, 18]. In a study involving 288 patients, we have reported that KPPLA is ten times more common as compared to *Escherichia coli* PLA. Also, KPPLA tends to be larger in size (6 cm vs. 4 cm, $p = 0.006$). Due to large size, KPPLA was more likely treated with percutaneous drainage [16].

8.4.2 *Escherichia coli* Pyogenic Liver Abscess (ECPLA)

ECPLA occurs more commonly in older patients with ischemic heart disease and is more likely to be associated with an underlying biliary disease [16, 17]. Hence the management of ECPLA poses distinct challenges. We have reported on adversaries associated with ECPLA and concluded that despite demographic and clinical differences, in the era of multimodal care, the outcomes of ECPLA are comparable to KPPLA [16]. Due to increased association with gallstone disease and biliary pathology, we advocate routine abdominal ultrasound scan of patients with ECPLA.

8.4.3 Culture-Negative Pyogenic Liver Abscess (CNPLA)

While blood cultures are the main source for microbial identification, the sensitivity to isolate the culprit microorganism is less than 40% [2, 19, 20]. In patients who undergo PLA aspiration or drainage, only 60% have positive pus cultures [2]. This is likely due to the starting of antibiotics prior to drainage. Other methods such as polymerase chain reaction (PCR) can be used to improve the diagnostic yield or obtain results earlier.

CNPLA is often underreported, yet is not uncommon. There is limited data on the outcomes of CNPLA. The management of CNPLA poses unique challenges when patients fail to respond to treatment. We have reported that the prevalence of CNPLA was the same as KPPLA over a 9-year period [20]. CNPLA patients were treated with the same empirical antibiotics targeted to *Klebsiella pneumoniae*, and the overall outcomes (length of hospital stay and 30-day mortality) of CNPLA patients were similar to KPPLA patients. CNPLA patients who had undergone percutaneous drainage have better outcomes compared to those treated with antibiotics alone. The empirical antibiotic of choice in CNPLA patients should be tailored to the most prevalent microorganism in local geography and antibiogram of the institution.

8.5 Clinical Presentation

The diagnosis of PLA requires a high index of clinical suspicion, as symptoms are often nonspecific and highly variable. The common symptoms of PLA are fever with chills, abdominal pain, and malaise. The common signs are pyrexia and right upper quadrant tenderness (two thirds of PLA are located in the right hemiliver). In a study by Chen et al., there was no difference in the presentation of KPPLA as compared to PLA from infection with other microbes [21]. PLA can occur on background of abdominal sepsis, and patient may manifest with clinical symptoms and signs of primary underlying disease. In patients treated with RFA or TACE for HCC, persistent pyrexia and abdominal pain should be investigated for possible PLA. In patient with TACE, high index of suspicion is essential as fever can be discounted as post-embolization syndrome. Possible complications of PLA include rupture, endophthalmitis, and multi-organ failure [21].

8.5.1 Rupture

Rupture of PLA is uncommon, occurring in up to 6% of PLA [22, 23]. Risk factors for rupture include DM, liver cirrhosis, *Klebsiella pneumoniae*, gas formation, size >6 cm, and left lobe involvement [24, 25]. Diabetes mellitus and liver cirrhosis compromise phagocytic and bactericidal functions with severe local inflammation and predisposing to rupture. PLA rupture is a surgical emergency. Free rupture with peritonitis requires surgical intervention, while localized rupture without peritonitis can be treated with percutaneous drainage and antibiotics [26]. Patients with ruptured PLA will have longer duration of hospital stay and use of antibiotics, higher rates of intervention (percutaneous or surgical drainage), and risk of metastatic infection. In a study reporting on 23 patients with ruptured PLA, Jun CH et al. have reported 4.3% mortality [24].

8.6 Investigations

8.6.1 Biochemistry

There is no specific serum biochemical test for establishing diagnosis of PLA. Routine serum biochemistry aids in diagnosis of sepsis, and on a background of clinical presentation, PLA can be suspected. Raised total white blood cell count, elevated urea, coagulopathy, and deranged liver enzymes aid in clinical judgment. Serum biochemical tests assist in monitoring the response to treatment and also guide duration of treatment. In the process of abscess formation, hepatocytes undergo necrosis and secrete cytokines to stimulate the growth of adjacent fibrous stroma tissue to form the abscess wall [27]. C-reactive protein (CRP) is an acute-phase protein synthesized primarily in the liver and is stimulated by cytokine release, especially interleukin-6. Law et al. calculated the CRP ratio in relation to the CRP concentration at week 1. They showed that a CRP ratio of 0.278 or less at week 3 is a marker of response and the PLA will likely be eradicated by a 5-week antibiotic regime, while a CRP ratio of greater than 0.57 at week 3 indicates possible treatment failure, progression of abscess, and a higher risk of mortality [27]. This results need to be validated.

8.6.2 Imaging

Ultrasound (US), computerized tomography (CT), and magnetic resonance imaging (MRI) scans are highly sensitive in the diagnosis of PLA. However, imaging findings are often nonspecific and may mimic hepatic cysts or necrotic tumors. US is simple and readily available and, in addition to diagnosis of PLA, assists in detection of gallstones. In patients presenting with septic shock and acute kidney injury, US can help reduce the risk of contrast-induced nephropathy. CT scan is helpful in detailing the enhancement pattern, as well as the presence of gas or calcifications.

MRI scan has multiplanar capability and has high specificity for differentiating hydatid cysts [4, 28]. On MRI scan, PLA may have perilesional edema and increased signal intensity seen on T2-weighted images and exhibit variable signal intensity on T1- and T2-weighted images, depending on their protein content. Imaging features can assist in the identification of underlying microorganism. Widely scattered microabscesses are seen in staphylococcal infections and usually involve both the liver and spleen. PLA may also manifest as a cluster of microabscesses that appear to coalesce focally. The cluster pattern is associated with coliform bacteria and enteric organisms and may represent an early stage in the evolution of a large PLA cavity. Gas formation is suggestive of clostridial infection; however, even in patients with gas formation, *Klebsiella pneumoniae* remains the predominant pathogen. In a study by Alsaif et al., gas was only seen in 17% of PLA on CT scan [29]. KPPLA is more likely solitary and appear solid and multilocular as compared to PLA caused by other bacteria. Imaging is typically considered the treatment end point and is a core component of diagnosis and treatment monitoring.

8.6.3 Size of the Abscess

Antibiotics are the mainstay of treatment of PLA; however, parenteral antibiotics alone may not be sufficient to treat large PLA because of the higher bacterial load, inadequate penetration of antibiotics, and ineffective medium for bacterial elimination [30, 31]. Drainage of PLA may shorten the duration of parenteral antibiotics. The size of an abscess that necessitates drainage and the modality of drainage are subject to much debate. Liao et al. [32] showed that an abscess larger than 7.3 cm predicts failure of percutaneous drainage. Other authors advocate operative drainage for larger PLA [30, 33]. There is currently no consensus on what defines a “large” abscess. We have defined a “giant” PLA as an abscess that is equal to or greater than 10 cm in diameter [34]. In that retrospective study, only 7% of PLA were giant and only 2.6% of giant PLA failed percutaneous drainage. This study demonstrated that percutaneous drainage with parenteral antibiotics is a safe and sufficient treatment for giant PLA, and operative drainage is only rarely needed. Size of the abscess does not affect the overall mortality [34]. Size is not the only factor that determines the success of percutaneous drainage. Multiloculation also leads to higher failure rates of percutaneous drainage because of the compartmentalization of the abscess. Multiloculated PLA is associated with increased morbidity and hospital stay [35, 36]. Only 55% of giant PLA were found to be multiloculated in the series by Ahmed et al. [34]. The relatively lower rates of multiloculated PLA in this series could have contributed to the high success rate of percutaneous drainage.

8.6.3.1 Gas Formation

Gas-forming PLA has higher rates of septic shock, occurring in 32.5% as compared to 11.7% in non-gas-forming PLA [37]. Gas formation is also an independent risk factor for spontaneous rupture [24]. The presence of gas on imaging is

associated with high mortality [22, 32]. The threshold to escalate therapy should be lowered in patients with gas-forming PLA that do not respond to percutaneous drainage. While percutaneous drainage is not contraindicated in gas-forming PLA, a study by Liao et al. [32] showed that the presence of gas was the most important radiological predictor for percutaneous drainage failure. *Klebsiella pneumoniae* is the most common bacteria isolated from gas-forming PLA, both from liver pus aspirates and from blood cultures [29]. *Klebsiella pneumoniae* is found in 81–100% of positive liver pus cultures in gas-forming PLA compared to 28–86% of non-gas-forming PLA [38–41]. Gas formation may be reliably diagnosed with either US or CT scan, with detection rates up to 100% [40]. On US, gas formation may appear as diffuse hyperechoic spots with acoustic shadowing [38] or hyperechoic lesions with reverberation [37]. On CT scan, gas formation may be recognized as low attenuation areas with Hounsfield units similar to that of the lungs [38]. Gas-forming PLA is increasingly reported to be associated with TACE [42, 43].

8.7 Management

8.7.1 Pharmacologic

The mainstay of PLA treatment is antibiotic therapy, with or without percutaneous aspiration/drainage. Antibiotic therapy must be targeted according to the locally prevalent organism and to specimen culture and sensitivity. It may not always work due to the bacterial load and poor penetration of antibiotics. Factors that predict failure of antibiotics-only therapy include age ≥ 55 years, multiple abscesses, malignant etiology, and patients who underwent endoscopic intervention. Antibiotic duration and abscess size were not predictive of failure [2]. In a recent study, we have concluded that empiric treatment of patients with CNPLA is safe if the treatment is tailored according to local antibiogram [20]. Our local algorithm of management of PLA is showed in Fig. 8.1.

8.7.2 Percutaneous Aspiration/Drainage

While some authors believe that percutaneous drainage may be inadequate for large PLA [30], more recent evidence has shown that large size of the abscess is not a contraindication to percutaneous drainage. In a study by Ahmed et al., percutaneous drainage was successful in 97.4% of PLA greater than 10 cm [34]. Figure 8.2 shows a CT scan image of an elderly gentleman with gas-forming PLA. Figure 8.3 is the CT scan image of the same patient following treatment with antibiotics and percutaneous drainage. Percutaneous drainage requires local anesthesia and minimal sedation and can be performed under radiological guidance. Percutaneous drainage

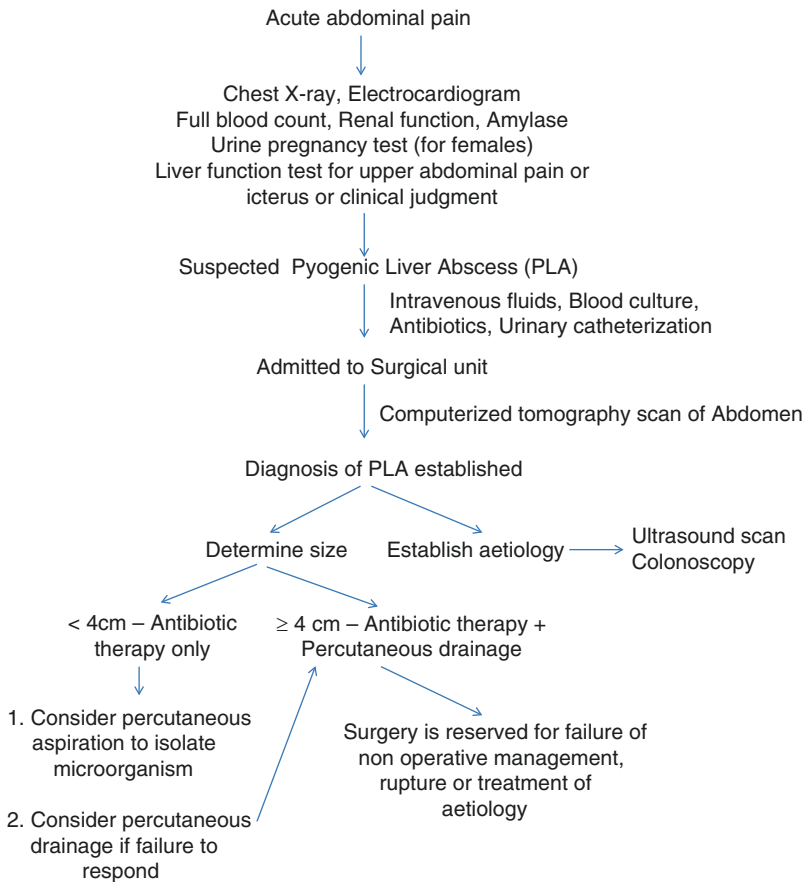


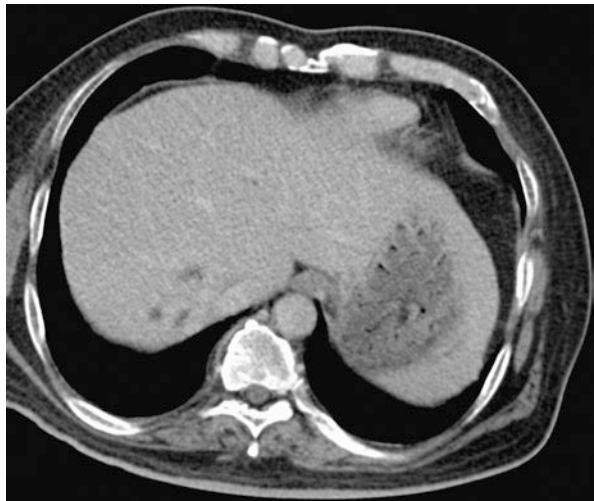
Fig. 8.1 Tan Tock Seng Hospital algorithm of management of pyogenic liver abscess

has a higher success rate compared to percutaneous aspiration and allows for controlled drainage of large abscesses with minimal hemodynamic and physiological stress to the patient [30, 44]. Factors that predicted the failure of antibiotics and percutaneous therapy include ECOG (Eastern Cooperative Oncology Group) performance status ≥ 2 , hypertension, and raised serum bilirubin [2]. The presence of multiple abscesses and size were not predictive of failure of percutaneous therapy [2]. Failure of percutaneous drainage can lead to uncontrolled sepsis and eventually death. In giant or multiloculated PLA, sometimes multiple drainage catheters are warranted. Catheter site discomfort with pain, superficial infection, and dislodgement are common drawbacks. In patients with minimal drainage, it is safer to flush the catheter to ensure patency rather than prematurely remove it. It is authors practice to remove catheter only when clinical and biochemical improvement is established and drainage is < 10 ml/24 h for 2 consecutive days.

Fig. 8.2 Computerized tomography scan image showing gas-forming pyogenic liver abscess



Fig. 8.3 Computerized tomography scan image of the same patient showing resolution of liver abscess following antibiotics and percutaneous drainage



8.7.3 Surgical Drainage

Surgery is indicated in patients with ruptured abscesses. Surgical drainage can be performed either open or laparoscopically. In most studies comparing percutaneous drainage to surgical drainage, percutaneous drainage has lower morbidity with comparable mortality [45, 46]. While laparoscopic drainage has a higher treatment success rate, operative and anesthesia-related morbidity remains high and hence only indicated in patients with failure of percutaneous drainage [47]. Open drainage allows the surgeon to use his fingers to break down the locules of the abscess effectively and subsequently places large-bore drains into the cavity. Open drainage may be better suited for abscess at difficult sites, such as the dome of the liver, as it allows more effective hemostasis in patients with severe coagulopathy [30].

However, with improvements in laparoscopic techniques and instruments, laparoscopic drainage may be as effective as open drainage. Recently, video-assisted drainage of PLA is reported in patients with failure of percutaneous drainage [48]. Hepatic resectional procedures are restricted to PLA in patients with recurrent pyogenic cholangitis.

Conclusion

PLA is a severe hepatobiliary infection with substantial morbidity and mortality. Widespread application of interventional radiology procedures for treatment of HCC has partly contributed to changing geographic trends. Imaging remains the cornerstone for diagnosis and treatment monitoring. Percutaneous drainage is essential in patients with large size and even sufficient in patients with multiloculated or giant PLA. Surgical therapy is indicated in patients with rupture, failure of percutaneous drainage, or for treatment of underlying etiology. Multimodal care is integral to ensure good outcomes.

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