Management of Pyogenic Liver Abscess

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Abstract:
Pyogenic liver abscess is a not so common clinical scenario which comes across the doctors worldwide. It is reported that these abscesses when not diagnosed in time, prove fatal.

Known since the age of Hippocrates, a lot of work has been done regarding Liver Abscesses. Infectious Etiological agents including ameba and bacteria have been identified, clearly dividing the infectious abscesses in two categories, amebic Liver Abscesses and Pyogenic Liver Abscesses. Various Therapeutic strategies both medical and interventional have been devised to manage these abscesses.

Introduction:
It is an encapsulated collection of suppurative material in the liver parenchyma, liver abscesses are of various etiologies including bacteria, protozoa, fungi, malignancy or iatrogenic. Out of these, Pyogenic Liver Abscesses are the most common ones.

Over time, the pathophysiology of PLA has changed. In the early 1990s, the condition was common as a complication of appendicitis which gave rise of pylephlebitis and consequently PLA. Later, biliary tract diseases were found to take lead as precursors of PLA and this still remains the leading cause till today.

Also, Malignancies and interventions like radiotherapy and TACE are contributing to the increasing incidence of PLA nowadays. The frequency of PLA is different in different parts of the world. Incidence is 2.3 cases per 100,000 hospital admissions in North America, whereas countries like Taiwan has incidence 274.4 per 100,000. Previously known to have high mortality i.e. 75%-80%, mortality has decreased now to 10%-40% mainly due to better antibiotics and interventional therapeutics option.

Although mortality has improved, early diagnosis of PLA is important as they are difficult to diagnose in view of variable and non specific symptoms.

Diagnostic imaging has been found useful in timely diagnosis of PLA. This article aims to describe the evolving risk factors and pathophysiology of PLA and review the latest recommendations for the management of PLA.

Risk Factors:
Various risk factors have been identified to cause and complicate PLA. These include conditions like Diabetes Mellitus, Cirrhosis, immunocompromise conditions, use of PPI, gender and age.

Diabetes Mellitus DM is a well recognized risk factor for PLA, found in 29.3%-44.3% PLA patients as concomitant disease. Diabetics are also found to have multiple abscesses usually. Several pathophysiological mechanisms are responsible for increased incidence of PLA in Diabetics, for example altered neutrophil metabolism, impaired chemotaxis and phagocytosis. This leads to impaired immunity and favors PLA.

Patients with Cirrhosis also have impaired immunity like diabetics. These unfortunate patients are 15.4 times more at risk to develop PLA as compared to general population.

Other immune-compromised states including solid organ malignancies, Chemotherapy, Immunosuppression after organ transplants and splenectomy also raise the risk for PLA.

The widespread use of PPIs also increases the PLA risk by decreasing the gastric acidity. This leads to decreased gastric immunity against bacteria. Wang et al. in a large case control study, showed a dose-response relationship between liver abscess formation and dose of PPI over a 90 days period.

Advancing age is also a risk factor for PLA. One study found a mean age > 57 years in patients with PLA suggesting increased susceptibility for PLA in older age group. Exact mechanism of this association need further research to get clarified.

In his 10 year study, Lee et al. found the ratio of males to females presenting with HA was about 2 to 1. Similar results were reported by Pang et al. and Lin et al.
Morbidity and Mortality:
Historically, PLA had high mortality, greater than 80%. Prompt diagnosis and treatment has improved the overall outcome with mortality around 15%-20%. Poor prognostic factors include:
- Age >70 years
- Multiple abscesses
- Polymicrobial infections
- Concomitant malignancy or Immunosuppressive disease
- Sepsis
PLA still has 100% mortality if left untreated.

Etiology:
The etiological agents reach liver either directly from the infected organs or through bloodstream via the portal vein or hepatic artery.
Etiological agents isolated from blood and abscesses cultures are given below:
- E. coli - 33%
- K. pneumoniae - 18%
- Bacteroides species - 24%
- Streptococcal species - 37%
- Microaerophilic streptococci - 12%

Most of the abscesses have more than one organism. The microbes are frequently of enteric or biliary in origin. Blood cultures are positive in one third to two third patients. Positive cultures are seen in 73%-100% patients when taken from abscess itself. E. coli is most common culprit in the western country. K. pneumoniae is the most common causative agent in Taiwan.

Pathophysiology:

Biliary disease:
Biliary disease is a common cause of PLA. Extrahepatic biliary obstruction usually due to bile duct stones, benign and malignant tumors, or strictures are the most common culprits. Biliary-enteric anastomoses (choledochoduodenostomy or choledochojejunostomy) have also been associated with a high incidence of liver abscesses. Bile leak after liver transplantation also causes pyogenic liver abscesses.

Infection via portal system (portal pyemia):
Intra abdominal infections reach liver through embolization or seeding of the portal vein. Appendicitis and pylephlebitis are the most common causes. It is seen that any intra-Abdominal collection e.g. acute diverticulitis, inflammatory bowel disease, perforated viscus can cause portal pyemia and consequent liver abscess.

Hematogenous (via Hepatic artery):
Bacterial endocarditis or urinary sepsis are common examples of entry through this portal. Blunt or penetrating trauma and liver injury during laparoscopic cholecystectomy also leads to PLA.

Procedures like trans arterial embolization (TACE) therapy and cryoablation for liver masses are also now recognized causes of PLA.

Cryptogenic:
In nearly 50% of cases, no definitive cause is identified. Such patients should be properly investigated for any GI or biliary cause.

Presentation:

History:
Patient gives a relatively longer history of ill health usually weeks or may be months. Fever which may be associated with rigors and chills and right upper quadrant pain are found in most of the patients. Pleuritic chest pain or referred right shoulder pain may also be the presenting clinical features. General ill health features like loss of appetite or weight loss may also be accompanied with the classical clinical features.

Physical Examination:
Right upper quadrant tenderness is a common clinical finding. Hepatomegaly and jaundice are also common clinical signs. At times patients also have associated chest pathology in the form of pleural effusion or consolidation. Peritonitis may be the presenting feature if there is an intra peritoneal rupture of the abscess.

Complications:
PLA may rupture into adjacent organs and body cavities to give rise to complications. These complications may be broadly divided into pleuro-pulmonary and intra-abdominal types. Pleuro-pulmonary complications are the most common and have been reported in 15-20% of early series. These include pleurisy and pleural effusion, empyema, and bronchohepatic fistula.

Intra-abdominal complications include subphrenic abscess and rupture into the peritoneal cavity, stomach, colon, vena cava, or kidney. A large abscess compressing the inferior vena cava and the hepatic veins may result in Budd-Chiari syndrome. Rupture into the pericardium or brain abscess from hematogenous spread is rare.

Pyogenic liver abscess has been associated with increased risk of acute kidney injury and acute pancreatitis.
Workup:

Laboratory studies:
CBC shows anemia in nearly 50-80% patients with leukocytosis >10,000/mcl in 75-96% patients. Bands>40% are seen in nearly 40% patients. The ESR is commonly elevated. LFTs show raised Alkaline phosphatase in 95-100% patients. ALT and AST are also increased. Serum Bilirubin is raised in 28-73% patients. A decreased serum albumin and increased globulin are also frequently seen. The prothrombin time is prolonged in 71-87% of patients.

Imaging Studies:
Initial evaluation involves chest and abdominal Xrays. However, they mostly show non specific findings. Example include raised right hemidiaphragm, Subdiaphragmatic air-fluid level, pneumonitis, and pleural effusion. In case of gas forming organisms, AXR may show intrahepatic air, portal venous gas, air-fluid levels or air in the biliary tree. Ultrasound findings are 80-100% sensitive. A round or oval hypoechoic mass is suggestive of PLA. CT is the imaging study of choice. However, PLA is not enhanced on imaging by the contrast. Triphasic CT scanning helps define the proximity of abscess to the major branches of the portal and hepatic veins. These findings are sensitive equally to Ultrasound but not specific.

Diagnostic procedure:
Daignostic aspiration under Ultrasound and Guidance is done followed by drainage catheter placement. The aspirate is cultured and examined for cytology.

Treatment and management:
The current accepted approach includes the following three steps:
- Initiation of antibiotic therapy
- Diagnostic aspiration and drainage of the abscess
- Surgical drainage in selected patients

Antibiotic therapy:
The antibiotic used should be active against gram-negative bacilli, microaerophilic streptococci, and anaerobic organisms, including Bacteroides fragilis. A combination of two or more antibiotics is preffered. Metronidazole and clindamycin have excellent anaerobic coverage. A third-generation cephalosporin or an aminoglycoside provides coverage against most gram-negative organisms. Fluoroquinolones are an acceptable alternative in patients who are allergic to penicillin.

Percutaneous drainage:
Percutaneous aspiration usually done under Ultrasound or CT guidance and is followed by the placement of a drainage catheter. The drain is removed when the abscess cavity collapse. Presence of Ascites and proximity to vital structures are contraindications for drainage. Percutaneous drainage is successful in 80-87% cases. Failure is evident if there is no clinical improvement or the condition worsens in 72 hours or if abscess recurs after initial drainage. Failure of percutaneous drainage is managed by placement of a second catheter or surgical drainage.

Surgical Therapy:
Surgical drainage is indicated in the following conditions:
- Abscesses larger than 5 cm
- Abscesses that are not amenable to percutaneous drainage secondary to location
- Coexistence of intra-abdominal disease that requires operative management
- Concomitant biliary/intra-abdominal disease
- Failure of antibiotic therapy
- Failure of percutaneous aspiration / drainage

Presence of peritoneal signs requires emergency laprotomy as these signs usually arise due to perforation of hepatic abscess.

Liver resection should be considered when the following are present:
- Liver carbuncle
- Hepatolithiasis
- Suspicious lesion that would require control of sepsis before a surgical procedure

Operative details:
Open drainage may be accomplished via one of the following three approaches,
- Transpleural
- Extraperitoneal
- Transperitoneal

Currently, with the availability of broad-spectrum antibiotics, the transperitoneal approach is preferred because it allows thorough inspection of the peritoneal cavity.

Laparoscopic drainage:
With the advancement in laparoscopic techniques, laparoscopic drainage of PLA is gaining
popularity. The availability of intraoperative ultrasonography also aids in the localization of the predisposing lesion.\textsuperscript{50,51}
References:


