LIVER ABSCESS: A Review

A.A Akinkuolie *FWACS*
Department of Surgery, O.A.U Ile – Ife

ABSTRACT

Liver abscess can be defined as an area of liquefied infected infarct. Differentiating the etiology of the abscess is important in the diagnosis and management of the patient. The pathogenic route of infection determines the microbiology and loculation of the abscess.

Presentations of liver abscesses depend on the severity of the disease, its chronicity, microbiology of the abscess, age of the patient, presence or absence of vascular compression and extension of abscess into contiguous organ(s).

Investigations in a patient with liver abscess can be divided into: diagnostic; liver parenchyma status; anatomical and physiological effect of the disease on body. Management of liver abscess is dependent on available resources, expertise, disease severity and co-morbity. The prognosis of patients with liver abscess is determined solely by the functional status of the underlying liver.

INTRODUCTION

Liver abscess can be defined as an area of liquefied infected infarct. The infective organism can be bacteria or fungi; sometimes it can be secondary to infestation by protozoan such as *entamoeba histolytica*. Differentiating the etiology of the abscess is important in the diagnosis and management of the patient.[1-3]

RELEVANT SURGICAL ANATOMY

Liver is the largest organ in the body and is interposed physiologically between the gastrointestinal tract and other bodily organ systems. It receives 20% of its blood supply from oxygen rich hepatic artery and 80% from nutrient rich portal vein: moreover, vessels in ligamentum teres and perihepatic venules: though these are insignificant sources of blood supply they may be important in the pathogenesis of liver abscess. The outflow of bile from the liver is through the biliary tract to empty into the second part of the duodenum. The liver is related to right copula of the diaphragm and the right lung superiorly, rib cage to the right and inferio-laterally to the stomach and duodenum. The importance of this is the risk of extension of infection from or through the adjacent organs.

CLASSIFICATION

- Aetiology of the abscess
  - pyogenic 60%
  - fungal 8%
  - parasitic 10%
  - cyptogenic 22%
- Location of the abscess within the liver
  - right hepatic lobe 70-80%
  - left hepatic lobe 20%
  - caudate lobe 10%
- Number of loculi
  - solitary 60-80%
  - multiple 20-40%

Pyogenic liver abscess (PLA):

PLA is a disease of developing country with high incidence and prevalence in the middle age; and slight male preponderance, it is commoner on the right than the left lobe of the liver[4-7]. Microbiology and loculation of the abscess depends on the route of infection. Pathogenic route of infection can be through any of the following seven routes:

- *Umbilical vein/ligamentum teres:* this is common route of PLA in neonatal period following umbilical sepsis from umbilical vein catheterization, and also; in patients with portal hypertension: recannalisation of umbilical veins in ligamentum teres often forms a ready made route for liver abscess, more commonly in those that are immunocompromised. The abscess cavity is usually uniloculated and monomicrobial; commonest being staphylococcus aureus.[5, 8]
- *Biliary tract:* This currently forms the commonest route of PLA following ascending cholangitis from biliary tract obstruction; either from: benign or malignant causes[9-11]. Loculation and size of the abscess cavity depends on the degree of obstruction within the biliary tree: total obstruction associated with ascending cholangitis; the normal pressure (5 – 15mmHg) within the biliary tree is greatly increased overcoming the hepatic secretory pressure (25 – 30mmHg) resulting in multiple microabscesses in the

liver parenchyma, whereas, partial obstruction allowing for distal venting which normalise the biliary pressure is usually associated with macroabscesses which may be solitary or multiple. Microbiologically, PLA originating from biliary tract is polymicrobial comprising of enteric flora: mostly, enterobacteriaceas and anaerobes.[12]

- *Portal vein:* Until the mid 1980s, this used to be the commonest route of PLA from portal pyemia complicating appendicitis[13, 14], however, with better management; appendicitis as a cause of PLA is uncommon. PLA from portal vein route tend to occur more in the right lobe of the liver, seemingly, the current of superior mesenteric vein portion of portal vein flow differentially to the right lobe of the liver. The abscess cavity is usually uniloculated and polymicrobial, though; the commonest reported organisms being K. pnuemoniae and E.coli.[15]
- *Hepatic artery:* PLA from this route originate from systemic sources, the infection can either be primary or secondary. such as: infective endocarditis,bacterial endopthalmitis[16],[17]
- *Traumatic:* Truamatic liver injuries can be blunt or penetrating. Most blunt hepatic trauma produces venous injuries that are low pressure and devoid of outside contamination, in contract, penetrating trauma produces both venous and arterial injuries with disruption and contamination of liver parenchyma along the trajectory of the offending object. Therefore, PLA is commonly associated with penetrating liver injuries; the abscess cavity is dependent on the pattern of heamotoma formation and inoculated organism(s) from the offending object[18].
- *Peri-hepatic infection and perihepatic venules:* Peritonitis with perhepatic collection can result in septic thrombophlebitis of the perihepatic venules. Contaminated clots with organism(s) showering hepatic vascular bed can result in multiple infracted areas with abscess formation[19]. This is commoner in immunocompromised host.
- *Miscellaneous:* Chronic granulomatous diseases[20-23], ascariasis[5, 24-26], papillon lefevre syndrome[27, 28], colonic carcinoma[29-33], colonic polypectomy[34-36] and swallowed foreign bodies.[37, 38] are known to be aetiological factor in pyogenic abscess.

Amoebic liver abscess (ALA):

ALA is the most common extra- intestinal manifestation of intestinal amoebiasis[39, 40]. It is a disease of the young adult, with male preponderance and it is the third leading cause of parasitic death in developing country.[41] ALA is assumed to be due to microemboli of trophozoites carried in portal circulation from intestinal focus of infestation resulting in multiple microabscesses in the liver which may eventually coalesce into macroabscesses.[42]

The primary sites of intestinal amoebiasis are: ceacum, appendix and ascending colon; which are drained by the superior mesenteric vein- a tributary of portal vein. Differential flow of superior mesenteric vein to the right lobe of the liver makes amoebic liver abscess to be commoner in the right hepatic lobe. ALA is progressive, non-suppurative with the abscess bacteriologically sterile - the characteristic “anchovy paste” abscess[43]; active trophozoites being confined to the abscess wall although there are variants of this in which there is involvement of the left lobe or sometimes multiple liver abscesses.

CLINICAL FEATURES

Presentations of liver abscesses depend on the microbiology of the abscess, age of the patient, presence or absence of vascular compression and extension of abscess into contiguous organ(s).

Generally, patients with PLA present with a pentad of symptoms- right hypochondrial pain, fever, jaundice, hypotension and central nervous system depression- especially, when the route of infection is through the biliary tree; though, many patients have prodromal symptoms of malaise, weight loss and anorexia.

Patients with ALA tend to present more acutely than patients with PLA and usually, with a preceding history of diarrhea which may be transient. Associated symptoms include: right hypochondrial pain, low grade fever, weight loss and anorexia. Patients with inferior vena cava obstruction or hepatic outflow obstruction might present with pedal oedema with/without ascites. Features of portal hypertension can be seen in those patients with chronic hepatic vein outflow obstruction from the posteriorly situated ALA in the right lobe. Symptoms of extension of the abscess depend on the organ involved: copious expectoration is suggestive of rupture into the lungs and rupture into the peritoneal cavity manifest with peritonitis.

Physical findings depend on the severity of the disease and its chronicity. The patient might be pale with clinical evidences of weight loss; jaundice may be

apparent: especially, in those with large abscesses obstructing the outflow of bile from the liver. Fever and tender hepatomegally are common features of liver abscess. Pulmonary or peritoneal signs are suggestive of extension of the abscess into the surrounding organ(s).

INVESTIGATIONS

Investigations in a patient with liver abscess can be divided into:

- Diagnostic
- Anatomical and physiological effect of the disease
- Liver parenchyma status

Diagnostic investigations:

High index of suspicion is required clinically to diagnose pyogenic liver abscess (ALA or PLA) in a patient presenting with fever, right hypochodrial pain and signs of acute/chronic liver disease, especially; in ALA, when a history of living or being to a known endemic area is important.

- Ultrasound (USS) is the usually the first investigation done in hepatic abscess because it is not invasive; it diffentiates biliary tract diseases from hepatic parenchyma lesions and can demonstrate space occupying lesion with echogenic architecture of the liver. Moreover, ultrasound guided aspiration can also be done at the same time.
- Microbiology of the aspirate and its sensitivity pattern is essential in determining the aetiology of the abscess.
- Serological screening for antibodies to *E. histolytica* usually develops after about a week following invasive amoebiasis; however, since antibodies persist for years: ELISA cannot differentiate acute from remote disease. However, serum antibody to 170KD subunit of galactose inhibitable adherence lectin is highly specific for differentiating acute from convalescent disease especially in endemic area
- Computed tomography (CT) is an alternative to ultrasound; though, more sensitive, is more expensive and not ready available in the developing countries. CT has the added advantage of being able to detect microabscess as small 5mm in diameter and moreover, can identify the source of infection in the abdomen.

Other imaging modalities such as: radionuclide scanning, magnetic resonance imaging and hepatic artiriography offers no added advantage over USS and CT; and they are not suitable for interventional procedures.

Anatomical and physiological:

- Chest x-rays might show consolidation in the lower lobe of the right lung or sympathetic effusion on the right.
- Clotting profile should be determined because of risk of bleeding during interventional procedures. Bleeding and clotting time; partial thromboplastin time and prothrombin time are essential before any invasive procedure.
- Heamogram determination to detect anaemia and pattern of leucocytosis.
- Renal function studies are most indicated in those that are jaundiced to know the baseline renal function and to prevent the risk of hepatorenal syndrome.

Liver parenchyma status:

Liver function studies to detect patients with hypoalbuminemia especially as a defferential in those with pedal oedema and ascites from the posteriorly located ALA obstructing inferior vena cava.

Management:

Management of liver abscess is in three stages:

- Resuscitation
- Therapeutic
  - Physiological preparation
  - Psychological preparation
  - Legal preparation
  - Treatment
- Prevention of recurrence
  - Health education (ALA).
  - Anatomical correction of predisposing lesion (PLA).

Resuscitation is mandatory to maintain steady state in acutely/chronically ill patient(s) with unstable vital signs: this entails maintaining/ensuring adequate cardiac output and end organ perfusion, and also, starting the patient empirically on broad spectrum antibiotic.

Physiological preparation involves correction of clotting profile, anaemia, electrolyte imbalance and other abnormalities discovered in the course of investigations.

Psychological preparation is explanation of the disease to patient and the available therapeutic modalities and allows the patient to come to term with the disease.

Legal preparation is gaining informed written consent about the planned intervention in the presence of a witness.

Treatment

- Medical: following adequate resuscitation, investigations and due preparation; appropriate antibiotics is commenced intravenously based on the suspected organism(s) adjudged from aetiology and route of infection. Pyogenic liver abscess less than 5cm in diameter, combination of antibiotics active against enterobacteriaceae and anaerobes should be first line choice of treatment; and should be continued for 3 -4weeks.

  Amoebic liver abscess responds solely to amoebicides in about 90% of cases. Single agent or combination of amoebicides have equal effectiveness, however, the choice amoebicide(s) should be continued for a minimum of ten days.

- Surgical
  - Percutaneous: this is the current mode of management of patients requiring surgical treatment. It is indicated in the following circumstances[8]:
    - Failure of medical treatment
    - Large abscess cavity in PLA (> 5cm)
    - Thin rim liver parenchyma surrounding (< 10mm) the abscess cavity
    - Ruptured abscess
    - Evidence of liver failure
    - Lack of clinical improvement in ALA after 48 to 72 hours of amoebicides.
    - Seronegative abscess in ALA.

    Drainage catheter should be discontinued when daily drainage is less than 10ml/day and the patient is apyrexic. Percutaneous drainage is contraindicated when there is no secure route.
  - Endoscopic biliary drainage: [44] is indicated in ALA if:
    - Percutaneous drainage > 25ml/day for 2weeks
    - Presence of bile in the draining fluid
    - Failure of medical treatment
    - Thin rim of liver parenchyma (< 10mm) surrounding the abscess cavity.
  - Laparascopy:is indicated in the following circumstances:
    - Failed percutaneous drainage.
    - Those with associated intra-abdominal pathology needing surgery
    - Those with multilocuted abscesses with the advantage of laparascopic US.
    - Ruptured intra-abdominal abscess
  - Open: is an alternative where facilities for laparascopic surgery is not available or is contraindicated. Open surgical treatment of liver abscess is necessary those with ascites.

Prevention of recurrence

The treatment of liver abscess is incomplete without the effort at preventing recurrence. Patients successfully treated for ALA should be health educated about the predisposing conditions and need for personal, food and environmental hygiene. In PLA it is mandatory to surgically correct the predisposing anatomical anomally where feasible.

Prognosis

The outcome of liver abscess is dependent on:

- Presence or absence of liver decompensation at presentation.[45]
- Associated comorbidity [46]

REFERENCES

1. Kar P, Kapoor S, Jain A. Pyogenic liver abscess: aetiology, clinical manifestations and management. *Trop Gastroenterol* 1998;**19**(4): 136-140.
2. Karatassas A, Williams JA. Review of pyogenic liver abscess at the Royal Adelaide Hospital 1980-1987. *Aust N Z J Surg* 1990;**60**(11): 893-897.
3. Mohsen AH, Green ST, Read RC, McKendrick MW. Liver abscess in adults: ten years experience in a UK centre. *Qjm* 2002;**95**(12): 797-802.
4. Kaplan GG, Gregson DB, Laupland KB. Population-based study of the epidemiology of and the risk factors for pyogenic liver abscess. *Clin Gastroenterol Hepatol* 2004;**2**(11): 1032-1038.
5. Kumar A, Srinivasan S, Sharma AK. Pyogenic liver abscess in children--South Indian experiences. *J Pediatr Surg* 1998;**33**(3): 417-421.
6. Hendricks MK, Moore SW, Millar AJ. Epidemiological aspects of liver abscesses in children in the Western Cape Province of South Africa. *J Trop Pediatr* 1997;**43**(2): 103-105.

7. Abdelouafi A, Ousehal A, Ouzidane L, Kadiri R. [Ultrasonography in the diagnosis of liver abscesses. Apropos of 32 cases]. *Ann Radiol (Paris)* 1993;**36**(4): 286-292.
8. Sharma MP, Kumar A. Liver abscess in children. *Indian J Pediatr* 2006;**73**(9): 813-817.
9. Chen SC, Yen CH, Tsao SM, Huang CC, Chen CC, Lee MC, Bell WR. Comparison of pyogenic liver abscesses of biliary and cryptogenic origin. An eight-year analysis in a University Hospital. *Swiss Med Wkly* 2005;**135**(23-24): 344-351.
10. Rockey DC. Heptaobiliary infections. *Curr Opin Gastroenterol* 2000;**16**(3): 251-254.
11. Bertel CK, van Heerden JA, Sheedy PF, 2nd. Treatment of pyogenic hepatic abscesses. Surgical vs percutaneous drainage. *Arch Surg* 1986;**121**(5): 554-558.
12. Claesson BE. Microflora of the biliary tree and liver--clinical correlates. *Dig Dis* 1986;**4**(2): 93-118.
13. Shfir R, Dinbar A, Wolfstein I, Tulcinsky DB. [Pyogenic liver abscesses complicating acute appendicitis]. *Harefuah* 1975;**88**(2): 73-74.
14. De Arteaga E, Maldonado Allende I. [Pyogenic abscesses of the liver following appendicular peritonitis.]. *Prensa Med Argent* 1961;**48**: 2363-2367.
15. Chen SC, Wu WY, Yeh CH, Lai KC, Cheng KS, Jeng LB, Wang PH, Lin DB, Chen CC, Lee MC, Bell WR. Comparison of Escherichia coli and Klebsiella pneumoniae liver abscesses. *Am J Med Sci* 2007;**334**(2): 97-105.
16. Wann SR, Liu YC, Yen MY, Wang JH, Chen YS, Wang JH, Cheng DL. Endogenous Escherichia coli endophthalmitis. *J Formos Med Assoc* 1996;**95**(1): 56-60.
17. Chou FF, Kou HK. Endogenous endophthalmitis associated with pyogenic hepatic abscess. *J Am Coll Surg* 1996;**182**(1): 33-36.
18. Eng RH, Tecson-Tumang F, Corrado ML. Blunt trauma and liver abscess. *Am J Gastroenterol* 1981;**76**(3): 252-255.
19. Perez MJ, Carrillo JL, Montin IM, Castellano AD. Liver abscess due to ileocecal perforation: a case report with literature review. *Emerg Radiol* 2007;**14**(4): 241-243.
20. Brunner J, Dockter G, Rosen-Wolff A, Roesler J. X-linked chronic granulomatous disease (CGD) caused by an intra-exonic splice mutation (CYBB exon 3, c.262G->A) is mimicking juvenile sarcoidosis. *Clin Exp Rheumatol* 2007;**25**(2): 336-338.
21. Feld JJ, Hussain N, Wright EC, Kleiner DE, Hoofnagle JH, Ahlawat S, Anderson V, Hilligoss D, Gallin JI, Liang TJ, Malech HL, Holland SM, Heller T. Hepatic Involvement and Portal Hypertension Predict Mortality in Chronic Granulomatous Disease. *Gastroenterology* 2008.
22. Hussain N, Feld JJ, Kleiner DE, Hoofnagle JH, Garcia-Eulate R, Ahlawat S, Koziel DE, Anderson V, Hilligoss D, Choyke P, Gallin JI, Liang TJ, Malech HL, Holland SM, Heller T. Hepatic abnormalities in patients with chronic granulomatous disease. *Hepatology* 2007;**45**(3): 675-683.
23. Kobayashi S, Murayama S, Takanashi S, Takahashi K, Miyatsuka S, Fujita T, Ichinohe S, Koike Y, Kohagizawa T, Mori H, Deguchi Y, Higuchi K, Wakasugi H, Sato T, Wada Y, Nagata M, Okabe N, Tatsuzawa O. Clinical features and prognoses of 23 patients with chronic granulomatous disease followed for 21 years by a single hospital in Japan. *Eur J Pediatr* 2008.
24. Bari S, Sheikh KA, Ashraf M, Hussain Z, Hamid A, Mufti GN. Ascaris liver abscess in children. *J Gastroenterol* 2007;**42**(3): 236-240.
25. Robbani I, Shah OJ, Zargar SA. Worms in liver abscess: Extensive hepatobiliary ascariasis. *Dig Liver Dis* 2008.
26. Slesak G, Phanthavong P, Rasphone O, Luangxay K, Anoulakkham P, Pahatsalang V, Soumphonphakdy B, White JA, Newton PN. Obstructive biliary ascariasis with cholangitis and hepatic abscesses in Laos: a case report with gall bladder ultrasound video. *J Infect* 2007;**54**(4): e233-235.
27. Dhadke SV, Kulkarni PM, Dhadke VN, Deshpande NS, Wattamwar PR. Papillon Lefevre syndrome. *J Assoc Physicians India* 2006;**54**: 246-247.
28. Sanchez-Fernandez P, Sanchez-Reyes K, Blanco-Benavides R. [Clinical images in gastroenterology. Pyogenic liver abscess complicated to pleura and Papillon-Lefevre syndrome]. *Rev Gastroenterol Mex* 2007;**72**(3): 249.
29. Fernandez Ruiz M, Guerra Vales JM, Castelbon Fernandez FJ, Llenas Garcia J. [Pyogenic liver abscess as presenting manifestation of silent colon adenocarcinoma]. *Rev Esp Enferm Dig* 2007;**99**(5): 303-305.
30. Giuliani A, Caporale A, Demoro M, Scimo M, Galati F, Galati G. Silent colon carcinoma presenting as a hepatic abscess. *Tumori* 2007;**93**(6): 616-618.
31. Hiraoka A, Yamashita Y, Uesugi K, Koizumi Y, Yamamoto Y, Doi H, Hasebe A, Ichikawa S, Yano M, Miyamoto Y, Ninomiya T, Matsuura B, Horiike N, Michitaka K, Hiasa Y, Nishikage S, Onji M. Three cases of liver abscesses complicated with colon cancer without liver metastasis: importance of screening for digestive disease. *Intern Med* 2007;**46**(24): 2013-2017.
32. Pisano M, Cogoni GF, Meloni A, Gromo C, Piga S, Saliu A, Ottonello R. [Pyogenic abscess in hepatic metastasis: unusual first manifestation of colorectal cancer. A case report]. *Chir Ital* 2007;**59**(3): 423-427.
33. Yokota T, Iwamoto K, Watanabe Y, Yamauchi H, Kikuchi S, Hatori M. Pyogenic liver abscesses secondary to carcinoma of the sigmoid colon: a case report and clinical features of 20 cases in Japan. *Ups J Med Sci* 2005;**110**(3): 241-244.
34. Farmer AD, Browett K, Rusius V, Bhalerao S, Anderson MR. Pyogenic liver abscess as a complication of sigmoid polypectomy. *Endoscopy* 2007;**39 Suppl 1**: E261.
35. Gross RG, Reiter B, Korsten MA. Pyogenic liver abscess complicating colonoscopic polypectomy. *Gastrointest Endosc* 2008;**67**(4): 767-768.
36. Harnik IG. Pyogenic liver abscess presenting after malignant polypectomy. *Dig Dis Sci* 2007;**52**(12): 3524-3525.
37. Kim IH, Kim HC, Koh KH, Kim SH, Kim SW, Lee SO, Lee ST. Journey of a swallowed toothbrush to the colon. *Korean J Intern Med* 2007;**22**(2): 106-108.
38. Serwe S, Weber J, Strock P, Lens V. Liver abscess caused by an unnoticed swallowed toothpick perforating the colonic wall. *Z Gastroenterol* 2007;**45**(10): 1060-1062.
39. Andersen PL. [Amebiasis]. *Ugeskr Laeger* 2000;**162**(11): 1537-1541.
40. Ventura-Juarez J, Jarillo-Luna RA, Fuentes-Aguilar E, Pineda-Vazquez A, Munoz-Fernandez L, Madrid-Reyes JI, Campos-Rodriguez R. Human amoebic hepatic abscess: in situ interactions between trophozoites, macrophages, neutrophils and T cells. *Parasite Immunol* 2003;**25**(10): 503-511.
41. Hughes MA, Petri WA, Jr. Amebic liver abscess. *Infect Dis Clin North Am* 2000;**14**(3): 565-582, viii.
42. Stanley SL. Pathophysiology of amoebiasis. *Trends Parasitol* 2001;**17**(6): 280-285.

43. Salles JM, Moraes LA, Salles MC. Hepatic amebiasis. *Braz J Infect Dis* 2003;**7**(2): 96-110.

44. Sandeep SM, Banait VS, Thakur SK, Bapat MR, Rathi PM, Abraham P. Endoscopic biliary drainage in patients with amebic liver abscess and biliary communication. *Indian J Gastroenterol* 2006;**25**(3): 125-127.

45. Chen SC, Tsai SJ, Chen CH, Huang CC, Lin DB, Wang PH, Chen CC, Lee MC. Predictors of mortality in patients with pyogenic liver abscess. *Neth J Med* 2008;**66**(5): 196-203.

46. Rockey D. Hepatobiliary infections. *Curr Opin Gastroenterol* 1999;**15**(3): 229-233.