

Pyogenic liver abscess mimicking pleural effusion

Abiodun M, MBBS(Benin), Senior Registrar

Osarogiagbon W, MBBS, FWACP, Consultant Paediatrician

Oviawe O, MBBS, FWACP, FMCP, Professor of Child Health, Consultant Pulmonologist

Director, Institute of Child Health, University of Benin Teaching Hospital

Correspondence to: Moses Abiodun, e-mail: biodunmt@yahoo.com

Keywords: pyogenic liver abscess, pleural effusion, ultrasound-guided percutaneous drainage

Abstract

Pyogenic liver abscess is a major visceral abscess that may pose a diagnostic dilemma in a febrile child with prominent extra-abdominal symptoms. We present a case of a well-nourished, immunocompetent four-year-old girl who had none of the common predispositions to a liver abscess. Hence, in this unusual setting, pyogenic liver abscess was not considered at the initial assessment, until closer evaluation and futile efforts to drain a seemingly large "pleural effusion" eventually revealed the diagnosis, which was confirmed by imaging. She underwent ultrasound-guided percutaneous drainage of the pyogenic liver abscess to avert the sequelae of this potentially fatal disorder. This underscores the need to think laterally when considering differential diagnoses for the sick child.

Peer reviewed. (Submitted: 2011-07-02. Accepted: 2011-11-04.) © SAAFP

S Afr Fam Pract 2012;54(5):459-462

Introduction

Pyogenic liver abscess can present both diagnostic and therapeutic challenges to healthcare providers. Based on aetiology, liver abscesses can be classified as pyogenic, which accounts for about 80% of cases; amoebic, which constitutes 10% of cases; and fungal (*Candida* species), which accounts for less than 10% of cases.¹ A liver abscess is twice as common in the right lobe than in the left lobe of the liver.² The annual incidence of liver abscess in children varies widely in different regions of the world, occurring more commonly in developing countries.² The incidence ranges from 28 per 100 000 in South African children, to more than 79 per 100 000 paediatric admissions in India.^{3,4} Risk factors for liver abscesses in children include malnutrition, sickle cell disease, acquired immunodeficiency syndrome and congenital immunodeficiencies, such as chronic granulomatous disease.^{2,5}

Liver abscesses can result from local spread via the portal circulation of an intra-abdominal infection focus (such as appendicitis), or from an ascending biliary infection.⁶ It may also be due to the haematogenous spread of a systemic infection via the hepatic artery. Other potential sources of infection include surgical wounds or penetrating injuries, while about 15% of cases are cryptogenic.^{1,2}

Clinical features of liver abscesses in children include fever and right hypochondrial tenderness in about 100% of cases, hepatomegaly in 85%, and vomiting in 25%.⁷ Symptoms such as cough, dyspnoea and severe chest pain suggest

intrathoracic involvement due to sympathetic pleural effusion or pneumonia. Rarely, pyogenic liver abscess may present with thoracic empyema or pericardiac tamponade, due to rupture of an abscess in the left lobe of the liver.⁸ Other metastatic complications of pyogenic liver abscess include peritonitis, pericarditis, splenic abscess, necrotising fasciitis, endophthalmitis, and brain abscesses, occurring more commonly with *Klebsiella pneumoniae* than with other bacteria (14.6 vs. 3.8%).⁹ The diagnosis of a liver abscess is usually confirmed by imaging studies.²

The treatment of a liver abscess can be medical, surgical, or a combination of both. Currently, ultrasound-guided percutaneous drainage is the treatment option of choice.² Prognosis depends on the underlying pathology and complications. With appropriate culture-sensitive antibiotics and drainage, the mortality rate is less than 15% in children with uncomplicated pyogenic liver abscess, but as high as 40% in complicated cases.²⁻⁴

We managed a child with a liver abscess who presented in an atypical manner, which led to treatment by pleural aspiration and chest tube insertion. Important lessons, in both the diagnosis and management of this important condition, are presented in the case study.

Case study

A four-year-old girl was admitted to the paediatric ward of University of Benin Teaching Hospital, Nigeria, on 29 December 2010. She was the youngest of three children,

and lived with their parents in a three-bedroom flat. She presented with high-grade fever that had lasted five weeks, with associated chills and rigors, a cough of four-week duration and right lower chest pain. She had had difficulty breathing for a week. She also complained of appetite loss. There was no history of trauma, and a systematic review of the other systems was not contributory.

On examination, she looked acutely ill, febrile (39°C), dyspnoeic, tachypnoeic, not pale, and not jaundiced. She was well nourished. Her weight was 15 kg (> 50th percentile), height 104 cm (75th percentile), and mid-upper arm circumference (MUAC) 15 cm. Her body mass index (BMI) was 14 kg/m². Her chest was asymmetrical, with slight bulging of the right hemithorax. The trachea was deviated to the left, and the apex beat was displaced 3 cm lateral to the left mid-clavicular line. On percussion, there was stony dullness and reduced breathing sounds in the right middle and lower lung zones. The abdomen was flat and soft. There was a 4 cm hepatomegaly below the right costal margin, which was tender. There was no ascites, and all other organ systems were normal.

Initial assessment was for community-acquired lobar pneumonia with a right-sided pleural effusion. Pulmonary tuberculosis was considered to form part of the differential diagnosis. A chest X-ray showed a homogenous opacity in the right middle and lower lung zones, with mediastinal shift (Figure 1a).

This was reported as a right-sided pleural effusion. Diagnostic thoracentesis yielded 3 ml of serous fluid. A chest tube was successfully inserted, but had to be removed, because it was not draining any fluid. Post-extubation chest X-ray did not show any improvement. Pleural fluid microscopy revealed the absence of acid-fast bacilli (AFB). Gastric aspirate was also negative for AFBs, and the Mantoux test was negative. A full blood count showed the following: white blood count 12.4 x 10³/μl, neutrophil 7.6 x 10³/μl (61.2%), lymphocytes 3.4 x 10³/μl (27.2%), monocytes 0.9 x 10³/μl (7.6%), eosinophil 0.5 x 10³/μl (4.0%). The erythrocyte sedimentation rate (ESR) was 18 mm/hour. Retroviral screen results were negative, haemoglobin genotype was AA, a stool microscopy showed no ova or parasite, and the blood culture yielded no growth.

Despite treatment with intravenous cefuroxime (100 mg/kg/day), she continued to deteriorate, and by day three post-admission, the liver was markedly tender and 6 cm below the right costal margin. A review of the chest X-ray showed a clear costophrenic angle without a meniscus sign (Figures 1a and 1b).

Immediate abdominal and pleural ultrasonography revealed a solitary liver abscess (13.4 cm x 9.03 cm) in the postero-superior aspect of the right lobe of the liver, with no evidence of biliary system dilatation, subdiaphragmatic,

supradiaphragmatic, or pleural fluid collections (Figure 2). Liver function tests were essentially normal: alkaline phosphatase (ALP) 25 IU/l, aspartate aminotransferase (AST) 36 IU/l, alanine aminotransferase (ALT) 17 IU/l, total bilirubin 0.4 mg/dl, conjugated bilirubin 0.3 mg/dl, total protein 5.7 g/dl, albumin 3.4 g/dl, and globulin 2.3 g/dl.

Ultrasound-guided percutaneous drainage was performed, and 280 ml of cream-coloured purulent fluid was aspirated (Figure 3). The cavity was irrigated with metronidazole and gentamicin, and intravenous metronidazole was added to her treatment. Post-aspiration, the cavity decreased to 4.3 x 4.7 cm. A two-week post-aspiration chest X-ray showed considerable descent of the elevated right hemidiaphragm. The liver abscess aspirate showed both Gram-negative



Figure 1a: Chest radiograph showing homogenous opacity in the right hemithorax, a clear right costophrenic angle, and absence of meniscus sign. **Figure 1b:** A sketch of Figure 1a, highlighting the elevated right hemidiaphragm "a", normal left hemidiaphragm "b", clear costophrenic angles, and mediastinal shift to the left.



Figure 2: A well-defined, rounded area of mixed echogenicity, measuring 13.4 x 9.03 cm in the posterosuperior aspect of the right lobe, in keeping with a liver abscess



Figure 3: Purulent aspirate (280 ml) obtained from the liver abscess via ultrasound-guided percutaneous drainage

bacilli and Gram-positive cocci following Gram stain, but culture (both aerobic and anaerobic) yielded no growth. An acid-fast smear of the liver aspirate was also negative. Neither fungal hyphae, nor yeast cells, were seen on microscopy. Specimens from the abscess wall was negative for *Entamoeba histolytica*. The patient recovered fully, and was discharged with oral cefixime and metronidazole, which she had to take for four weeks. She is being followed up in our clinic.

Discussion

Pyogenic liver abscess is a major visceral abscess that may pose a diagnostic dilemma in a febrile child who presents

with prominent extra-abdominal symptoms and no risk factors for liver abscess, such as malnutrition, sickle cell disease, hepatobiliary disease, or immunosuppression.⁵ The prominent respiratory features in our patient resulted from the pressure effect of the liver abscess on the right hemidiaphragm causing pulmonary compression and cardiomeastinal shift (Figures 1a and 1b). In this case, the solitary right lobe abscess that was found is the usual trend. Zibari et al reported 95% right lobe involvement, and 70% of liver abscesses being solitary, in a large case series.¹⁰ Similarly, in a meta-analysis, Mishra et al reported that approximately two-thirds of liver abscesses occur in the right lobe of the liver, the majority of which are solitary.² The predilection for abscesses to occur in the right hepatic lobe could be due to the fact that it has a larger blood supply than the left and caudate lobes. Also, unlike the left portal vein which branches at an angle, the right portal vein follows a straight course in the direction of the common portal vein, encouraging direct spread of infection from pylephlebitis.²

The most common organism isolated from pyogenic liver abscess in children is *Staphylococcus aureus*, both in developed and developing countries.² Other species implicated in pyogenic liver abscess are *E. coli*, *Klebsiella*, *Enterobacter* and anaerobes.² In this case, liver aspirate microscopy showed both Gram-positive cocci and Gram-negative bacilli, but culture yielded no growth. Polymicrobial aetiology is common, and blood culture may be negative in about half of cases.^{7,11} Eosinophilia, found in this case, may be due to previous helminthic infestation. Helminthiasis has been implicated in pyogenic liver abscess in children without pre-existing liver lesions, by inducing eosinophilia and granulomatous changes while passing through the liver.¹²

Clinical manifestations and sonographic findings of pyogenic liver abscess, as found in our patient, are often distinct from those of other cystic lesions of the liver, such as a simple cyst, cystadenoma, echinococcal cyst, liver metastasis and polycystic liver disease.^{12,13} On ultrasonography, simple cysts appear as an anechoic unilocular fluid-filled space with an imperceptible wall; cystadenoma as an hypoechoic loculated lesion with thickened irregular walls; and an echinococcal cyst as an anechoic smooth, round lesion with calcification.^{12,13} The unifocal nature of the pyogenic liver abscess, and the absence of a debilitating underlying disease, are good prognostic factors in our patient.

As illustrated in this case report, thorough evaluation of children without obvious risk factors for a liver abscess, and a high index of suspicion, are crucial to enable prompt diagnosis and management of this potentially fatal disease.

Acknowledgements

We thank Drs Iseh, Venn and Olaiya, who participated in the management of this patient.

References

1. Krige JEJ. Pyogenic liver abscess. In: Kirsch R, Robson S, Trey C, editors. *Diagnosis and management of liver disease*. London: Chapman and Hall, 1995; p. 196-202.
2. Mishra K, Basu S, Roychoudhury S, Kumar P. Liver abscess in children: an overview. *World J Pediatr*. 2010;6(3):210-216.
3. 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.
4. Kumar A, Srinivasan S, Sharma AK. Pyogenic liver abscess in children: South Indian experiences. *J Pediatr Surg*. 1998;33(3):417-421.
5. Lublin M, Bartlett DL, Danforth DN, et al. Hepatic abscess in patients with chronic granulomatous disease. *Ann Surg*. 2002;235(3):383-391.
6. Rahimian J, Wilson T, Oram V, Holzman RS. Pyogenic liver abscess: Recent trends in etiology and mortality. *Clin Infect Dis*. 2004;39(11):1654-1659.
7. Bari S, Sheikh KA, Malik AA, et al. Percutaneous aspiration versus open drainage of liver abscess in children. *Pediatr Surg Int*. 2007;23(1):69-74.
8. Vong SC, Guo HR, Lin HJ, Foo NP. Cardiac tamponade secondary to pyogenic liver abscess. *J Clin Gastroenterol*. 2007;41(6):635-636.
9. Yang CC, Yen CH, Ho MW, Wang JH. Comparison of pyogenic liver abscess caused by non-Klebsiella pneumoniae and Klebsiella pneumoniae. *J Microbiol Immunol Infect*. 2004;37(3):176-184.
10. Zibari GB, Maguire S, Aultman DF, et al. Pyogenic liver abscess. *Surg Infect*. 2000;1(1):15-21.
11. Chemaly RF, Hall GS, Keys TF, Procop GW. Microbiology of liver abscesses gram stain and associated blood cultures. *Diagn Microbiol Infect Dis*. 2003;46(4):245-248.
12. Pereira FE, Musso C, Castelo JC. Pathology of pyogenic liver abscess in children. *Pediatr Dev Pathol*. 1999;2(6):537-543.
13. Nisenbaum HL, Rowling SE. Ultrasound of focal hepatic lesions. *Semin Roentgenol*. 1995;30(4):324-346.

Press Release

Rabies: 100% Fatal, 100% Preventable

In a bid to save thousands of lives, the Department of Health (DoH), Sanofi Pasteur (the vaccines division of Sanofi), and Merial South Africa (the animal Health Division of Sanofi), have partnered with numerous human and animal health organisations worldwide to support World Rabies Day on 28 September, creating awareness that rabies is 100% preventable.

World Rabies Day, the single largest rabies education and outreach initiative, is observed each year in about 135 countries. It aims to educate people about the risk of rabies, to promote global awareness of its prevention, and to mobilise resources to support local rabies prevention programmes. Rabies is a viral disease that attacks the central nervous system. It can be transmitted to animals and humans mainly through a bite, but also through contamination of broken skin or mucous membranes with saliva from an infected animal. Rabies has been reported throughout South Africa and in almost all types of domestic animals and a number of wild animals. However, 99% of cases are transmitted through contact with infected dogs.

"The WHO estimates that more than 55 000 people die each year, more than half of them in Africa, with the most vulnerable being children. Children are likely to suffer multiple bites and scratches due to them being in close contact with dogs and cats, which carry a higher risk of contracting rabies, and are less likely to tell their parents when a bite, lick, or scratch has occurred" says Jean-Antoine Zinsou, Country Manager, Sanofi Pasteur.

Jean-Antoine Zinsou continues, "If not treated immediately - before the virus reaches the central nervous system and the symptoms of the disease appear - rabies is fatal to both animals and humans. The symptoms in humans include headaches, fever, anxiety, muscle pains, vomiting, paralysis and hydrophobia (fear of water). The symptoms of rabies in animals include behavioural changes such as restlessness, irritability, excitability and shyness."

"Prevention is better than cure; rabies prevention starts with the animal owner" notes Theunis de Bruyn, Business Unit Manager, Merial South Africa. "We recommend that people vaccinate dogs, cats and any other animal that has regular contact with humans, such as horses."

Vaccination remains the only effective treatment against rabies and acts by neutralising the virus before it actually reaches the central nervous system. The vaccine can be administered before exposure to the virus (pre-exposure prophylaxis), or immediately after contact with an infected animal (post-exposure prophylaxis). The World Health Organization recommends that pre-exposure vaccination should be promoted for children living in areas where canine rabies is endemic, including South Africa. It is also recommended for people who face increased risk of exposure, such as laboratory staff, vets, animal handlers, game rangers and travellers to high risk areas.