Liver abscess in children: an overview

Kirtisudha Mishra, Srikanta Basu, Subhasis Roychoudhury, Praveen Kumar
New Delhi, India

Background: **Liver abscess (LA) in the pediatric population has become relatively uncommon in developed countries but it continues to have a high incidence among children in developing countries. This article aims to review the trends in all aspects of LA in children, both temporally and geographically.**

Data sources: **The PubMed and Google Scholar database were searched with the keywords "liver abscess", "children", "predisposing causes", "clinical signs and symptoms", "treatment" from 1975 to 2009 and all kinds of retrospective and prospective studies, reviews, case series were included.**

Results: **Pyogenic LA constitutes the majority of cases, followed by amebic and fungal LA. *Staphylococcus aureus* is the most common pathogen worldwide. Ultrasonography (US) and computed tomography (CT) are widely used as diagnostic tools. There are varying opinions regarding the treatment of LA in children. The general trend is towards less invasive modalities of treatment like percutaneous drainage along with antimicrobial drug therapy. However, in selected patients, open surgical drainage still plays an important role. The mortality rate for pyogenic LA has shown a decline from about 40% before the 1980s to less than 15% in the recent years. At the same time, the mortality rate of amebic LA cases reported to be around 11%-14% before 1984 has reduced to less than 1% at present.**

Conclusions: **Etiological pattern of LA in children has remained the same over the years, and in most regions, it is associated with *Staphylococcus aureus* and amebic LA is quite uncommon. US or CT scan is the most frequently employed diagnostic modality for LA, and follow-up is usually performed by serial US scans. Antimicrobial therapy along with, if necessary, drainage of the abscess by either percutaneous or open surgical route remains the treatment of choice.**

World J Pediatr 2010;6(3):210-216

Key words: **children;**
clinical signs and symptoms;
liver abscess;
predisposing causes;
treatment

Introduction

Liver abscess (LA) is a frequently encountered disease in children from developing countries, especially those living in the tropical and subtropical zones. It has become relatively uncommon in developed countries. A review of the literature reveals that *Staphylococcus aureus* is the most common causative organism, followed by *Entamoeba histolytica*. Although much is known about the etiopathogenesis of LA, the gold standard of treatment is still debatable. There are different modes of medical and surgical treatment and it is difficult to prove the superiority of one modality over the other. Rather, a judicious application of conservative and surgical methods is what is mostly resorted to. This review attempts to study the trends in various aspects of LA in children and present the conclusions of several studies from different parts of the world.

Incidence

Pyogenic liver abscess

Overall, pyogenic LA constitutes the majority (80%) of hepatic abscesses in children.[1-3] The incidence of pyogenic LA has been reported to be 1 in 140 admissions in Brazil[4] to more than 79 per 100 000 pediatric admissions in India.[5] From the other parts of the world, the incidence varies from 28 per 100 000 in South Africa[1] to 25 per 100 000 pediatric admissions in USA,[6] 11 per 100 000 admissions in Denmark[7] and 20 per 100 000 admissions in Taiwan of China.[8]

Amebic liver abscess

There is a paucity of the literatures regarding the exact incidence of amebic LA in children. Studies have

Author Affiliations: Department of Paediatrics, Kalawati Saran Children's Hospital, Lady Hardinge Medical College, New Delhi-110001, India (Mishra K, Basu S, Roychoudhury S, Kumar P)

Corresponding Author: Srikanta Basu, # 318, Ashirwad Enclave 105, I P Extension Patpargunj, New Delhi 110092, India (Tel: 0091-11-22238229/0091-9818291595; Fax: 0091-11-23365792; Email: srikantabasu@gmail.com)

doi:10.1007/s12519-010-0220-1

suggested that it is rare in children[9] and mostly endemic in Thailand, India, Egypt and South Africa.[10] It has also been mentioned that amebic LA develops in less than 1% of patients infected with *Entamoeba histolytica*.[11] However, a study from Vietnam reported that the annual incidence of amebic LA was 21 per 100 000 inhabitants, 95% of whom were adults.[9]

Etiopathogenesis

Predisposing factors

Historically, liver abscess in children is secondary to perforated appendicitis. LA is seen often in children with major debilitating diseases, granulocyte dysfunction, sickle cell disease, and congenital or acquired immunosupression. Helminthic infections with larva migrating through the liver are predisposing factors for LA in children. The occurrence of LA in patients with worm infestations is due to stimulation of T2 immunity and suppression of the T1 effector limb thereby compromising the handling of bacteria and fungi by phagocytes. In addition, bacteria are trapped in the liver granulomas surrounding the parasites whose larvae and eggs act as a nidus for infection.[12,13]

The biliary tract is an important source of pyogenic LA. Congenital anomalies, biliary strictures and other anatomic abnormalities lead to obstruction of bile flow, resulting in bacterial proliferation. When the biliary tract is the source of LA, there are multiple abscesses. Biliary ascariasis associated with cholangitis and LA is common in the areas where ascariasis is endemic, like the Kashmir valley in India.[14]

Protein calorie malnutrition also predisposes to LA in children probably due to immunosupressed state. Of 84 childhood LA studied over a 10-year period by Hendricks et al,[1] 56% of the patients had protein calorie malnutrition. Similarly Kumar et al[5] reported moderate to severe malnutrition in 27.8% of their cases. Genetic disorders like Papillon-Leferve syndrome which is a rare autosomal recessive disease associated with pyogenic LA.[15] LA is also seen in children with inherited immunodeficiency disorders such as chronic granulomatous disease.[16]

Pathophysiology

Bacteria and other organism can enter the liver through various routes including the biliary tract, portal vein, and hepatic artery. Infections in organs in the portal bed can also result in localized septic thrombophlebitis, releasing septic emboli into the portal circulation which are trapped by hepatic sinusoids and become the nidus for hepatic abscess formation.

Penetrating hepatic trauma can inoculate organisms directly into the liver parenchyma, resulting in pyogenic LA. Non-penetrating hepatic trauma can also have a similar result by causing localized hepatic necrosis, intrahepatic hemorrhage and bile leakage, thus providing a suitable environment for bacterial growth. Approximately two thirds of LA occur in the right lobe of the liver and the majorities are solitary.[5,17-20] The predilection for the right hepatic lobe can be attributed to the volume of the right portal vein flow and to that the right portal vein continues the direction of the common portal vein while the left portal vein takes a more horizontal direction.

Pyogenic LA is usually solitary, located in the right lobe of the liver and is encased in the fibrous tissue. In the presence of pylephlebitis, the portal vein and its branches may contain purulent material and blood clots. Perihepatitis with formation of adhesions may be seen. On histological examination, areas remote from the abscess show portal zone infection and surrounding disintegrating hepatocytes being infiltrated by polymorphs.[21]

In case of pyogenic LA, amebic LA is frequently seen in the right lobe of the liver, often antero-superiorly, just below the diaphragm. The center of the abscess consists of a large necrotic area which has liquefied into thick, reddish-brown pus, similar to anchovy or chocolate sauce. This occurs as a result of lysis of liver cells as is evidenced by fragments of liver tissue within the area. Secondary bacterial infection may also occur and as a result the pus may become green or yellow in color. On histopathological examination, the wall of the abscess consists of degenerated liver cells, leucocytes, red blood cells, connective tissue strands and debris. Amebae may be identified in the scrapings. The rest of the liver parenchyma is usually normal.[21]

Causative organism

Pyogenic organisms account for nearly 80% of LA in children, followed by amebic LA which is responsible for about 21%-30% of the cases.[1-3] Worldwide, the most common pathogen isolated from LA in children is *Staphylococcus aureus* both in developed and developing countries.[4,22] Studies from India revealed similar results.[5,13,23] *E. Coli*, *Klebsiella* and *Enterobacter* are the other species focused in LA.[7,17,18,24] A study from Taiwan of China reported *Klebsiella* pneumoniae was the most common pathogen responsible for pyogenic LA in children.[8] Recurrent pyogenic cholangitis may be due to *Salmonella typhi*.[21] Kumar et al reported LA as an unusual complication of enteric fever in the pediatric age group.[25] In some reports, anaerobes constitute up to 30% of isolated pathogenic organisms.[6,7,26] Tubercular LA is rare.[2,27]

Many of reports on amebic LA in children are from Latin America and Africa.[1-3] Studies from the United States suggest that 1%-7% of the population is infected with *Entamoeba histolytica* and less than 1% of the infected have extraintestinal disease.[28]

Fungal hepatic microabscesses either alone or in association with splenic microabscesses may occur in children with leukemia.[29] A large number of LA cases have been found without any apparent cause and have been labeled as cryptogenic.[23] As high as 33%-35% cases of cryptogenic LA have been reported by Donovan et al[30] and Bari et al.[23] The abscess may also be sterile because the patient has received prior antibiotic therapy. Otherwise, the report may be unreliable because of inadequate culture of anaerobic organisms.[21]

Clinical presentations

The usual features of LA in children are fever, abdominal pain and tender hepatomegaly.[23,31,32] A single abscess is often insidious in its presentation while multiple abscesses (Fig. 1, 2) usually present in a more acute fashion.[25] Subdiaphragmatic irritation or pleuropulmonary spread of LA may lead to pain in the right shoulder and/or cough. In our center, a fair number of children present with pleural effusion or empyema associated with LA. Some patients may present with nonspecific symptoms like weight loss, fatigue, often associated with a vague right upper quadrant pain.[13] On the other hand, some cases of LA may present with complications like fulminant sepsis or an acute abdomen either due to rupture of the LA or due to infection of ascitic fluid caused by transmigration of infection.[13]

Investigations

Blood counts may show anemia, leukocytosis and a raised erythrocyte sedimentation rate (ESR). Liver enzymes, especially serum alkaline phosphotase, may show altered values. Donovan et al[30] reported increased levels of serum glutamate oxaloacetate transaminase (SGOT) and serum glutamate pyruvate transaminase (SGPT) in patients with LA, while Moazam and Nazir[33] reported increased level of serum bilirubin in patients with amebic LA. However, Kaplan and Feigin[34] reported normal liver function tests in children with pyogenic LA. Blood cultures are known to be less sensitive than pus aspirate cultures.[7,24] Trophozoites are difficult to detect in aspirates of pus from amebic LA as they are usually located in the wall of the abscess. However, serologic tests like indirect hemagglutination antibody titres and enzyme linked immunosorbent assay (ELISA) are sensitive markers to diagnose amebic infection. Negative serological tests strongly point to a pyogenic etiology. Ultrasound (US) is more widely used (Fig. 3) as contrast-enhanced computed tomography (CECT) scan is more expensive and carries a risk of contrast nephropathy. On magnetic resonance imaging (MRI), LA appears hypointense on T1 weighted, and hyperintense in T2 weighted sequences.[35] Other tests

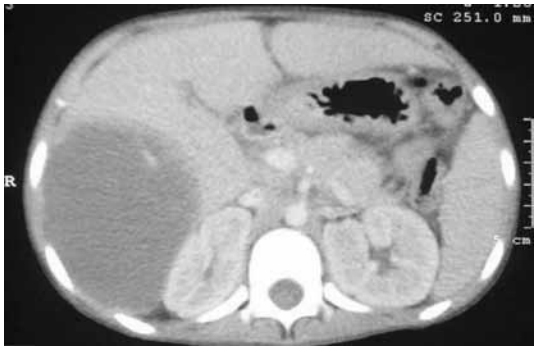

Fig. 1. CT image of an 8-year-old boy showing a large, hypodense non-enhanced area (10×5.7 cm, abscess cavity) in segments V and VI of the right liver lobe.

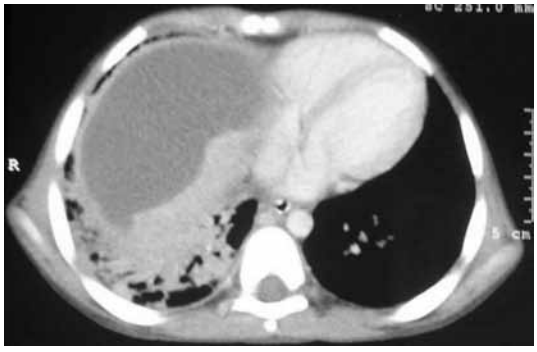

Fig. 2. CT image of an 8-year-old boy showing a lenticular shaped hypodense infracapsular collection of size (approximately 10×5 cm) on the anterosuperior surface of the right liver lobe.

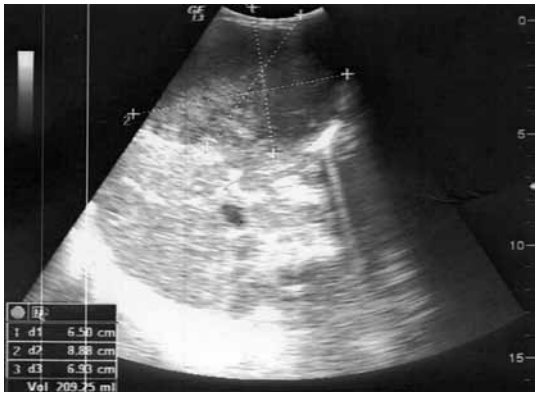

Fig. 3. Ultrasonography of an 2.5-year-old boy showing a hypoechoic lesion with well-defined margins (size: 88×69×65 mm; volume: 209 ml) in the left lobe of the liver, suggestive of a liver abscess.

include scintigraphy with labeled leukocytes, which shows abscesses as "hot spots" whereas abscesses appear as "cold spots" on 99mTc labeled scintigraphy. Halvorsen et al[36] evaluated the imaging results of 63 patients with hepatic abscess, and found that CECT detected 57 (97%) of 59 patients with LA with false-negative results in two patients; US detected 33 (79%) of 42 patients, missing abscesses in the dome of the liver, small abscesses, and 2 large early abscesses; radionuclide (RN) examination detected 16 (80%) of 20 patients, which could not detect abscesses less than 2 cm in diameter. In this retrospective study CECT was the most sensitive imaging modality available for the detection of LA. Similarly, Pearce et al[37] from UK reported that the sensitivity of CECT in detecting LA was 100%, whereas that of US was 96%. A study from India reported that US was the first imaging technique used in 130 suspected cases of amebic LA and that the sensitivity and specificity of US were 96.5% and 61.36%, respectively. In high-resolution imaging, ultrasound is still the mainstay in diagnosis of LA with a sensitivity as high as 80%-95%.[38]

Treatment

Untreated LA is almost always fatal because of complications including sepsis, empyema or peritonitis from rupture into the pleural or peritoneal spaces, and retroperitoneal extension. There have been significant advances and changes in the management of pediatric LA over the last 15 years.[31]

Treatment of pyogenic liver abscess

Traditionally patients with pyogenic LA are treated by open surgical drainage and antibiotic therapy. Over the years, however, operative drainage has been increasingly replaced by accurate imaging and percutaneous drainage.

Medical treatment

Antibiotic therapy as a sole treatment modality has been successful. Antimicrobial treatment is a common adjunct to percutaneous or open surgical drainage. Kumar et al[5] treated 66% of cases of pyogenic LA conservatively by antibiotics alone, but 16.7% of them subsequently required drainage. In another study, non-operative treatment was successful in 60% of cases of multiple LA.[3] A report of 38 children with LA in children from Pakistan concluded that the conservative management was effective for smaller abscesses (<5 cm) and percutaneous needle aspiration under US guidance was the best treatment modality for non-complicated larger abscesses (>5 cm).[39] However, studies have suggested that 80%-90% of pyogenic LA cases require drainage.[18,40] When abscess aspirate culture reports are available, the treatment with antimicrobial agents should be directed toward the most commonly involved pathogens. An anti-staphylococcal, anti-anerobic or anti-amebic drug combined with an aminoglycoside or cephalosporin is an initial choice for antimicrobial therapy.[13]

Ultimately, isolated organisms and their antibiotic sensitivities are detected to make final choice of antimicrobial therapy. Currently 4-6 weeks of antimicrobial therapy is recommended for solitary lesions that have been adequately drained. Multiple LA may require up to 12 weeks of therapy. The clinical and radiographic progresses of the patients are also used to define duration of antimicrobial therapy.

Surgical treatment

Open surgical drainage was the gold standard of care until the introduction of percutaneous drainage techniques in the mid 1970s. With the refinement of image-guided techniques, percutaneous drainage and aspiration have been accepted as the standard management of LA.[41,42]

Percutaneous drainage (PD)

Compared to open surgical drainage, PD eliminates the need of general anesthesia, except a short period of hospitalization. PD is indicated if the following are met. First, the volume of the abscess is large and there is risk of spontaneous rupture (specially in cases of left lobe LA); Second, after 48-72 hours of medical therapy, there is lack of clinical response along with signs of persistent sepsis; Third, there are clinical or US features suggestive of enlarged abscess with impending rupture; Fourth, there is evidence of liver failure; Fifth, there is uncorrected primary pathology in critically ill patients as a temporal measure to improve their conditions before surgery.[23,31]

Percutaneous aspiration in conjunction with antibiotics has been recommended for unilocular LA.[23,31] In our center percutaneous needle aspirations along with antimicrobial therapy constitute the first line treatment which is effective in a large number of patients.

PD complications include bacteremia, hemorrhage of the peritoneal cavity and intestine (hemobilia), and iatrogenic infection.[13] A review of various studies shows that the overall failure rate of PD ranges from 5% to 28%.[43-45] Herman et al[46] analyzed 48 patients with pyogenic LA and found that the failure rate of PD was 30.8%, whereas the failure rate of open surgical drainage was only 8.5%. Percutaneous management failed in patients with thick-walled

abscess or containing viscid pus, and in the presence of loculations.[46] PD is not indicated for ascites or when LA is close to the pleura.[46]

Open surgical drainage

In spite of the great enthusiasm for percutaneous drainage, open surgical treatment plays an important role in the management of LA, primarily in patients with (a) ruptured abscess on presentation, (b) multiloculated abscess, (c) abscess with thick pus, (d) multiple abscesses, (e) left lobe abscess, (f) other intra-abdominal pathology, and (g) failure of percutaneous drainage.[13,23] In our experience, the two common practical indications for open surgery are ruptured abscess with peritonitis and failed percutaneous drainage, especially in severely sick patients. Open surgery can be performed by two approaches. (1) A transperitoneal approach allows drainage of LA including multiple abscesses and may identify any etiology or associated pathology. Good results including a low mortality have been reported.[3] (2) For high, posteriorly located LA, a posterior transpleural approach can be used. It is apparent that a laparoscopic approach may be a useful minimally invasive technique for drainage of LA. However, this approach has not been reported.

Of 129 children with LA reported by Bari et al,[23] 55 were treated by open surgical drainage, whereas 27 children were subjected to percutaneous aspiration under US guidance. Percutaneous aspiration failed in 5 patients, who later underwent open surgical drainage. It was concluded that open surgical drainage was still the best modality of treatment, and that although percutaneous aspiration was safe and effective, expertise was required.[23] Moore et al[3] studied 124 children with LA for a period of 16 years and suggested that patients with a solitary left-sided LA warrant early operation. Other researches have also advocated early surgical treatment of LA in children.[47,48]

Treatment of amebic liver abscess

According to several studies, 95% of amebic LA cases received medical therapy, such as therapy with nitroimidazoles for a total duration of ten days.[38,49] In a randomized controlled trial on 39 consecutive patients with amebic LA, Sharma et al[50] concluded that chemotherapy with potent tissue-amebicidal drugs such as metronidazole is optimally effective in treating amebic LA, and that in uncomplicated cases routine aspiration is not required. Most studies including those conducted by Bari et al[23] and Sharma et al[50] have suggested that uncomplicated amebic LA can be managed by medical therapy instead of aspiration. However, Meng et al[51] reported that needle aspiration combined with drug therapy was superior to drug therapy. In 20 children with LA treated by de Kolster et al,[18] 60% of amebic LA cases received only medical treatment. Amebic LA with secondary pyogenic infection may require antimicrobial therapy (antibiotics plus metronidazole) along with drainage of the abscess.

Treatment of fungal abscess

Systemic antifungal agents should be initiated if fungal abscess is suspected or after the abscess has been drained percutaneously or surgically. Initial medication for fungal LA is currently amphotericin B. Lipid formulations may be better as the complexing of drug to lipid moieties allows for concentration in hepatocytes. Successful treatment with flucanazole for fungal LA after failure of amphotericin treatment has been reported; however, the use of flucanazole as the initial therapeutic drug is under investigation.[52]

Prognosis

If LA is untreated, the prognosis is fatal. The mortality rate of LA cases was as high as 40% until the 1980s.[53,54] Since then, more potent antibiotics, improvement in imaging techniques with image-guided percutaneous drainage, and appropriate use of surgical intervention have reduced the mortality rate to less than 15%.[1,5,6,19] Jaundice, liver failure, acute abdomen and sepsis, bilirubin levels >3.5 mg/dl, encephalopathy, large volume of abscess, multiple abscesses, and hypoalbuminemia (<2 mg/dl) are indicators of poor prognosis. These may be the markers of complications like intraperitoneal rupture of LA associated with peritonitis, hepatopleural or hepatobronchial fistulae, intrapericardial rupture of the abscess, or Budd-Chiari syndrome.[53,54]

In conclusion, LA in children is still very common in developing countries. Pyogenic LA is more common than amebic, fungal or other etiologies. Imaging with US and/or CT is diagnostic. Antimicrobial therapy along with percutaneous drainage constitutes the mainstay of treatment, whereas open surgical drainage should be reserved for selected cases.

Funding: None.
Ethical approval: Not required.
Competing interest: None.
Contributors: Mishra K collected the data, reviewed the literature and drafted the main body of the article under the supervision of Basu S. Roychoudhury S critically reviewed the whole article and provided pediatric surgical inputs to the article. Kumar P critically revised the article for important intellectual content. Basu S provided the concept and design of the review and is the guarantor for the article.

References

1 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;43:103-105.
2 Guittet V, Ménager C, Missotte I, Duparc B, Verhaegen F, Duhamel JF. Hepatic abscesses in childhood: retrospective study about 33 cases observed in New-Caledonia between 1985 and 2003. Arch Pediatr 2004;11:1046-1053.
3 Moore SW, Millar AJ, Cywes S. Conservative initial treatment for liver abscesses in children. Br J Surg 1994;81:872-874.
4 Ferreira MA, Pereira FE, Musso C, Dettogni RV. Pyogenic liver abscess in children: some observations in the Espírito Santo State, Brazil. Arq Gastroenterol 1997;34:49-54.
5 Kumar A, Srinivasan S, Sharma AK. Pyogenic liver abscess in children—South Indian experiences. J Pediatr Surg 1998;33:417-421.
6 Pineiro-Carrero VM, Andres JM. Morbidity and mortality in children with pyogenic liver Abscess. Am J Dis Child 1989;143:1424-1427.
7 Hansen PS, Schønheyder HC. Pyogenic hepatic abscess. A 10-year population-based retrospective study. APMIS 1998;106:396-402.
8 Kong MS, Lin JN. Pyogenic liver abscess in children. J Formos Med Assoc 1994;93:45-50.
9 Haque R, Huston CD, Hughes M, Houpt E, Petri WA Jr. Amebiasis. N Engl J Med 2003;348:1565-1573.
10 Salles JM, Moraes LA, Salles MC. Hepatic amebiasis. Braz J Infect Dis 2003;7:96-110.
11 Wells CD, Arguedas M. Amebic liver abscess. South Med J 2004;97:673-682.
12 Pereira FE, Musso C, Castelo JS. Pathology of pyogenic liver abscess in children. Pediatr Dev Pathol 1999;2:537-543.
13 Sharma MP, Kumar A. Liver abscess in children. Indian J Pediatr 2006;73:813-817.
14 Javid G, Wani NA, Gulzar GM, Khan BA, Shah AH, Shah OJ, et al. Ascaris-induced liver abscess. World J Surg 1999;23:1191-1194.
15 Oğuzkurt P, Tanyel FC, Büyükpamukçu N, Hiçsönmez A. Increased risk of pyogenic liver abscess in children with Papillon-Lefevre syndrome. J Pediatr Surg 1996;31:955-956.
16 Lublin M, Bartlett DL, Danforth DN, Kauffman H, Gallin JI, Malech HL,et al. Hepatic abscess in patients with chronic granulomatous disease. Ann Surg 2002;235:383-391.
17 Wang DS, Chen DS, Wang YZ, Li JS. Bacterial liver abscess in children. J Singapore Paediatr Soc 1989;31:75-78.
18 de Kolster CE, Guerreiro N, de Escalona L, Perdomo G, Márquez R, de Laurentin N. Hepatic abscess in children: analysis of 20 cases. G E N 1990;44:221-226.
19 Moore SW, Lakhoo K, Millar AJ, Cywes S. Left-sided liver abscess in childhood. S Afr J Surg 1994;32:145-148.
20 Ahmed L, el Rooby A, Kassem MI, Salama ZA, Strickland GT. Ultrasonography in the diagnosis and management of 52 patients with amebic liver abscess in Cairo. Rev Infect Dis 1990;12:330-337.
21 The Liver in infections. In: Sherlock S, Dooley J, eds. Disease of the liver and Biliary System. Oxford: Blackwell Science, 2002: 495-497.
22 Muorah M, Hinds R, Verma A, Yu D, Samyn M, Mieli-Vergani G, et al. Liver abscesses in children: a single center experience in the developed world. J Pediatr Gastroenterol Nutr 2006;42:201-206.
23 Bari S, Sheikh KA, Malik AA, Wani RA, Naqash SH. Percutaneous aspiration versus open drainage of liver abscess in children. Pediatr Surg Int 2007;23:69-74.
24 Tsai CC, Chung JH, Ko SF, Liu PM, Su CT, Li WC, et al. Liver abscess in children: a single institutional experience in southern Taiwan. Acta Paediatr Taiwan 2003;44:282-286.
25 Kumar A, Kapoor R, Chopra K, Sethi GR, Saha MM. Typhoid fever. Unusual hepatic manifestations. Clin Pediatr (Phila) 1989;28:99-100.
26 Brook I, Fraizer EH. Role of anaerobic bacteria in liver abscesses in children. Pediatr Infect Dis J 1993;12:743-747.
27 Essop AR, Segal I, Posen J, Noormohamed N. Tuberculous abscess of the liver. A case report. S Afr Med J 1983;63:825-826.
28 Seidel J. Diagnosis and management of amebic liver abscess in children. West J Med 1984;140:932-933.
29 Maxwell AJ, Mamtora H. Fungal liver abscesses in acute leukaemia—a report of two cases. Clin Radiol 1988;39:197-201.
30 Donovan AJ, Yellin AE, Rall PW. Hepatic abscess. World J Surg 1991;15:162-169.
31 Barakate MS, Stephen MS, Waugh RC, Gallagher PJ, Solomon MJ, Storey DW, et al. Pyogenic liver abscess: a review of 10 years' experience in management. Aust N Z J Surg 1999;69:205-209.
32 Moulds-Merritt C, Frazee RC. Therapeutic approach to hepatic abscesses. South Med J 1994;87:884-888.
33 Moazam F, Nazir Z. Amebic liver abscess: spare the knife but save the child. J Pediatr Surg 1998;33:119-122.
34 Kaplan SL, Feigin RD. Pyogenic liver abscess in normal children with fever of unknown origin. Pediatrics 1976;58:614-616.
35 Balci NC, Semelka RC, Noone TC, Siegelman ES, de Beeck BO, Brown JJ, et al. Pyogenic hepatic abscesses: MRI findings on T1- and T2-weighted and serial gadolinium-enhanced gradient-echo images. J Magn Reson Imaging 1999;9:285-290.
36 Halvorsen RA Jr, Foster WL Jr, Wilkinson RH Jr, Silverman PM, Thompson WM. Hepatic abscess: sensitivity of imaging tests and clinical findings. Gastrointest Radiol 1988;13:135-141.
37 Pearce N, Knight R, Irving H, Menon K, Prasad K, Pollard S, et al. Non-operative management of pyogenic liver abscess. HPB (Oxford) 2003;5:91-95.
38 Yeoh KG, Yap I, Wong ST, Wee A, Guan R, Kang JY. Tropical liver abscess. Postgrad Med J 1997;73:89-92.
39 Cheema HA, Saeed A. Etiology, presentation and management of liver abscesses at the Children's Hospital Lahore. Annals 2008;14:148-150.
40 Huang CJ, Pitt HA, Lipsett PA, Osterman FA Jr, Lillemoe KD, Cameron JL, et al. Pyogenic hepatic abscess. Changing trends over 42 years. Ann Surg 1996;223:600-607.
41 Giorgio A, Tarantino L, Mariniello N, Francica G, Scala E, Amoroso P, et al. Pyogenic liver abscesses: 13 years of experience in percutaneous needle aspiration with US guidance. Radiology 1995;195:122-124.
42 Rintoul R, O'Riordain MG, Laurenson IF, Crosbie JL, Allan PL, Garden OJ. Changing management of pyogenic liver abscess. Br J Surg 1996;83:1215-1218.
43 Gerzof SG, Johnson WC, Robbins AH, Nabseth DC. Intrahepatic pyogenic abscesses: treatment by percutaneous drainage. Am J Surg 1985;149:487-494.
44 Bissada AA, Bateman J. Pyogenic liver abscess: a 7-year experience in a large community hospital.

Hepatogastroenterology 1991;38:317-320.
45 Gyorffy EJ, Frey CF, Silva J Jr, McGahan J. Pyogenic liver abscess. Diagnostic and therapeutic strategies. Ann Surg 1987;206:699-705.
46 Herman P, Pugliese V, Montagnini AL, Salem MZ, Machado MA, da Cunha JE, et al. Pyogenic liver abscess: the role of surgical treatment. Int Surg 1997;82:98-101.
47 Hansen N, Vargish T. Pyogenic hepatic abscess: a case for open drainage. Am Surg 1993;59:219-222.
48 Chou FF, Sheen-Chen SM, Chen YS, Lee TY. The comparison of clinical course and results of treatment between gas-forming and non-gas-forming pyogenic liver abscess. Arch Surg 1995;130:401-405.
49 Omanga U, Mashako M. Amoebic liver abscess in children (study of 47 cases observed from 1964 to 1979 in the clinic of pediatrics of Kinshasa University). Med Trop (Mars) 1981;41:425-430.
50 Sharma MP, Rai RR, Acharya SK, Ray JC, Tandon BN. Needle aspiration of amoebic liver abscess. BMJ 1989;299:1308-1309.
51 Meng XY, Wu JX. Perforated amebic liver abscess: clinical analysis of 110 cases. South Med J 1994;87:985-990.
52 Anaissie E, Bodey GP, Kantarjian H, David C, Barnett K, Bow E, et al. Fluconazole therapy for chronic disseminated candidiasis in patients with leukemia and prior amphotericin B therapy. Am J Med 1991;91:142-150.
53 Muñoz LE, Botello MA, Carrillo O, Martínez AM. Early detection of complications in amebic liver abscess. Arch Med Res 1992;23:251-253.
54 Sharma MP, Dasarathy S, Verma N, Saksena S, Shukla DK. Prognostic markers in amebic liver abscess: a prospective study. Am J Gastroenterol 1996;91:2584-2588.

Received November 19, 2009
Accepted after revision April 13, 2010