Liver Abscess in Children

M.P. Sharma and Arvind Kumar

Department of Gastroenterology, Rockland Hospital, New Delhi, India

ABSTRACT

Liver abscesses are frequently observed in pediatric clinical practice in tropics and subtropics especially in developing countries. Children have unique set of predisposing factors which have been highlighted. Expected microbiology and setting for unusual organisms are mentioned. Clinical presentations, including importance of location and number of abscesses has been discussed. Role of each modality of treatment clarified. Importance and limitations of investigations and imaging explained, and complications and mortality discussed. [Indian J Pediatr 2006; 73 (9) : 813-817] E-mail : mpsharma_s@hotmail.com

Key words : Liver abscesses; Predisposing causes; Clinical signs and symptoms

Children with liver abscesses constitute more than 79 per 100,000 pediatric admissions (<12 yrs age) in tertiary care centres in India.1 In larger series from developing nations (Brazil) it is documented in frequency of approximately 1 out of 140 admissions.2 However in developed countries it is rare with an incidence of 25 per 100,000 admissions in USA3 to 11 out of 100,000 admissions in Denmark.4 It is a cause of significant morbidity and mortality. For unknown reasons male children akin to male adults are affected by liver abscesses more than female children.2,5

Predisposing causes of Liver abscesses

Parasitic Infestations: Parasitic infestations are thought to predispose to pyogenic liver abscesses in children. Almost all parasites including ascariasis, schistosomiasis, fascioliasis, trichuris trichura, necator, ancylostoma and toxocara infestation have been reported as association. Significant increased parasitic infestation has been demonstrated in children with pyogenic liver abscesses when compared with controls from similar background.6 Experimental studies have shown that bacteremia specially from Staphylococcus species in presence of parasitic infestation predisposes to liver abscess formation.7 Salmonella infection has also been associated with parasitosis. The pathogenic mechanism is thought to be stimulation of T2 immunity in patients with worm infestations. This is hypothesized to suppress the T1 effector limb thereby compromising phagocyte handling of bacteria and also fungi. This apart, liver granulomas around parasites, their larvae and eggs are believed to trap bacteria in the granulomatous reaction around them serving as nidus. Tissues from liver biopsies or surgical and autopsy specimens in patients with liver abscesses have documented eggs, larvae, and antigens of parasites.8,9 Laboratory clue to an underlying parasitosis is often a peripheral eosinophilia (40% patients), hyper IgE levels, antibodies to various parasites and detection of eggs and larvae in stool. Sometimes tropical myositis may point towards a Toxocara infestation. Appropriate tests are therefore recommended to ascertain such infestation and follow up treatment should be done. Biliary ascariasis associated with cholangitis and liver abscesses continue to be common in endemic areas2 and in India in Kashmir valley.10

Genetic Disorders: (1) Papillon-Leferve syndrome is a rare autosomal recessive disease comprising palmoplantar keratoderma and periodontitis. Pyogenic liver abscess is an increasingly recognized complication.11 (2) Chronic Granulomatous Disease (CGD) is a rare inherited primary immunodeficiency where phagocytes cannot destroy catalase -positive bacteria and Fungi. Defect in phagocytic cells’ respiratory bursts lead to life threatening infections including liver abscesses. Fever is the most common presenting symptom. These abscesses are often recurrent, multiple and difficult to treat. Mean age of first abscesses is often mid teens. Commonest micro-organism is staphylococcus though gram negative rods, nocardia and fungal infections are also reported. Often surgical resection is more rewarding than percutaneous drainage.12 Interferon gama therapy may have a role in resistant cases.

(3). Infections and liver abscess has been reported in
children with deficiencies in C1 complement. (4). Hyper Immunoglobulin E syndrome is associated with recurrent abscesses involving several organs including liver.

Skin infections: Skin infections are common in children. These provide a source of bacteremia and inoculation of infection in liver. Significant co-relations have been documented between pyogenic liver abscesses and skin infections in some large series of children with liver abscesses.2,8 Predominant organism is Staphylococcus aureus. Chromobacterium violaceum sepsis in tropics and sub-tropics can present as skin and liver infection.

Protein Calorie malnutrition: There is likelihood of co-relation with liver abscesses and malnutrition in children. Liver abscesses in children are rare in developed countries whereas they are common in developing countries. Though studies have observed significant proportion of malnutrition in children with liver abscesses,1,13 comparison with disease controls in same centres have not given a clear association.14

Abdominal infections and Liver abscesses: At the beginning of this century portal route of entry of infections constituted a large proportion of patients with liver abscesses. However this has declined, with increasing ease of diagnosing such infections and treating them. However case reports15 and series16 are still published, emphasizing their importance. Previous portal vein catheterization also can predispose to portal pyaemia and liver abscesses. Choledochal cysts can predispose to recurrent cholangitis and liver abscesses in children.16 Similarly congenital hepatic fibrosis can predispose to cholangitis and liver abscesses.

Post trauma: Trauma predisposes to liver abscesses both by direct injury to liver or by providing habitat for proliferation of organisms elsewhere. Child abuse can sometimes be the cause of sepsis and liver abscesses.17 Unusual organisms like clostridia can flourish and this needs to be kept in mind.18

Microbiology of Liver abscesses

Most of the liver abscesses in children are pyogenic in nature with amoebic liver abscesses constituting 21-30% of cases13,20,25 or more (50%).22 However in an appropriate epidemiological setting one may see cases of amoebic liver abscesses presenting as neonatal sepsis.23 Among cases of pyogenic liver abscesses, Staphylococcus is the leading cause in most series.1,5,7,12,13,21 Anaerobes constitute an important proportion of up to 30% of organisms3,4,24 and include microaerophilic streptococci. Gram negative rods like E. coli,5,22 and klebsiella25 are common isolates as are other enterobacter species4.

Unusual micro-organisms: Fungal hepatic microabscesses either alone or in association with splenic microabscesses may occur in children with acute leukemia.26

Tubercular liver abscesses are rare but are known to occur.20,27

Typhoid fever can have associated hepatic abscesses28 and in an appropriate setting, cat scratch disease, brucella, meliodosis, can all be suspected as cause of hepatic and splenic abscesses usually associated with a systemic dissemination of infection.

Location and number of Liver abscesses: Irrespective of any etiology most of liver abscesses (approx 2/3rd) occur in the right lobe of liver and majority (approx 2/3rd) are solitary.1,3,22,29,30,31,32 Left lobe abscesses should be treated with caution as they are frequently associated with complications like rupture into peritoneum and pericardium and cause pericardial effusions29 each of which may be life threatening. Left sided liver abscesses require drainage far more often (85% cases) when compared to right sided lesions31,20 even when aetiology is amoebic.31 Multiple liver abscesses constitute 20-25% of all cases.1,2,24 Majority ie 2/3rd of multiple pyogenic liver abscesses are confined to right side of liver.23 With multiple liver abscesses mortality may be almost twice that in solitary cases.24 Even patients with amoebic multiple liver abscesses have more complications and mortality.23 We found in our study that severe clinical presentation, jaundice and left lobe hepatomegaly were markers for multiple amoebic liver abscesses.35

Clinical Presentation Signs and Symptoms

Irrespective of pyogenic or amoebic aetiology, fever often with chills, abdominal pain specially in right upper quadrant and tender hepatomegaly are common presenting signs and symptoms.1,3,22,25,36 Nausea and vomittings, anorexia, unexplained anemia, and cough with breathing difficulty37 or simply fever are other common complaints. Whereas fever is the most common symptom in children, afebrile presentations of liver abscesses specially of amoebic aetiology are well known in series of mixed age groups.29 In one series of amebic liver abscesses, majority i.e 58% of patients had subacute presentation with right upper quadrant dull ache, weight loss, fatigue, anemia and low to moderate grade fever.29 On the other hand liver abscesses may present as fulminant sepsis23 or acute abdomen.35 Clinical presentations do not distinguish amoebic from a bacterial etiology unless there is an obvious precipitating source for eg. abdominal infections. It is uncommon to get a positive history of colitic diarrhoea from patients of amebic liver abscesses.

Investigations

Anemia, leucocytosis and raised sedimentation rates are usual findings on hemogram. Altered liver enzymes specially alkaline phosphatase point to liver as an involved organ. Majority of children have a prolonged prothrombin time.22 All of these set of investigations do not differentiate amoebic from pyogenic. Blood cultures must be sent but they have lower sensitivity than pus aspirate cultures in identification of culprit organism in

Indian Journal of Pediatrics, Volume 73—September, 2006
Liver Abscess in Children

pyogenic abscesses. Aspiration of pus for amebic trophozoites is often not helpful in clinching an amebic etiology. However, indirect hemagglutination antibody titres in blood is sensitive in detecting systemic amoebic infection. Indirect fluorescent antibody and ELISA tests are also very useful. Negative amebic serology points strongly to a pyogenic source of infection.

**IMAGING**

*Role of USG:* USG is the imaging of first choice. It is quick, safe, cheap, and accurate in picking a liver lesion. Appearances of an abscess may be a rounded or an oval lesion which is usually hypechoic but may have heterogeneous echotexture. A solid or heterogeneous lesion often evolves into a hypechoic lesion on subsequent examination. Majority of the abscess have a well defined wall which may be thin or irregular. USG may reveal an intra-abdominal precipitating cause for liver abscess. However USG may miss very small abscesses or abscesses lying in areas of liver difficult to examine when patient cooperation is poor.

*Role of CT:* CECT is more sensitive in detecting even small abscesses anywhere in liver. Yet it is inconvenient and expensive with risk of contrast nephropathy being always there. A hypo dense lesion with low attenuation areas and an enhancing rim is a classical CECT image. Small hypo echoic lesions in cluster may suggest a beginning of process of coalescence into a single large abscess later.

*Role of MRI:* Liver abscesses on MRI appear hypo intense on T1 weighted and hyper intense in T2 weighted sequences. On gadolinium enhanced sequences there is early and continued enhancement of wall which persists on delayed images. There is increased peri-abscess tissue enhancement in immediate post-gadolinium images. With all the inconveniences of time and cost attached to MRI, it is at its best of same capability as CECT.

*Role of Scintigraphy:* If ambiguity arises in diagnosis in an occasional patient with conventional imaging, scintigraphy may help. In labeled leukocyte scintigraphy (ILLS) which shows abscesses as ‘hot spots’ superimposed on 99mTc labelled scintigraphy which shows abscesses as ‘cold spots’ are of value in establishing a diagnosis. Resolution and follow up of abscess on imaging: Of all imaging modalities, USG is best for follow up, although it is not routinely necessary. The abscess cavity takes many months to finally resolve and lags behind clinical resolution by months. Resolution time depends on abscess size, but hypoalbuminemia and anemia at presentation were also found important in our study. It is not at all necessary to retreat or continue treating a clinically quiescent patient with a visible cavity on imaging. Recurrences are rare for amoebic liver abscesses. Recurrences are observed with pyogenic liver abscesses, most of which occur within 3 months of treatment. There may be an underlying problem like CGD predisposing recurrence.

**TREATMENT**

*Medical therapy:* At presentation, it is important to resuscitate a sick septic patient with I.V. fluids and other supportive measures. Appropriate analgesics may be essential but NSAIDs should be avoided if intervention is planned. A combination of anti-staphylococcal drug like cloxacinil, an anti-anerobic and anti-amebic drug like metronidazole and an aminoglycoside or cephalosporin for gram negative bacilli is a good initial choice. Therapeutic drainage is not a must in all cases of pyogenic liver abscesses. However experiences with most series suggest that 80-90% pyogenic liver abscesses require some form of drainage. Medical antibiotic cover is additionally required for a period of 3-4 weeks.

95% of amebic abscesses do well on medical therapy alone and require therapy with nitroimidazoles for a total duration of ten days only. Metronidazole is the drug of choice. Oral dose is 30-50mg/kg/d, and i.v. dose is 7.5 mg/Kg/dose 6th hourly.

*Role of aspiration:* Aspiration is safe, is helpful in diagnosis of abscess and provides material for microbiological assessment. Amebic abscess (sometimes repeated) in combination with antibiotics has been successfully tried worldwide. In pyogenic liver abscesses, aspiration along with antibiotics help and reasonable results can be achieved. This strategy has been reported to be helpful in amoebic liver abscesses too but in a randomized trial conducted by us, we did not find any added benefit of aspiration in uncomplicated amoebic abscesses. Aspiration must be attempted in solitary, unilocular lesions and on carefully selected patients but if sepsis persists, prompt drainage is required.

*Role of percutaneous drainage:* Percutaneous drainage has now come to a centrestage in management of liver abscesses that require more than just a medical management. Safety and efficacy of percutaneous abscess drainage in selected patients is now well established. Even multiloculated liver abscesses can be managed with aggressive percutaneous techniques that include disruption of loculations and placement of large bore sump catheters. Percutaneous drainage is indicated when (a) Volume of abscess is large and there is risk of spontaneous rupture (specially left lobe abscesses). (b) When actual rupture has occurred, then along with abscess cavity drainage of extraneous collection. (c) When there is lack of response to medical therapy with clinical signs of persistent sepsis or enlarging abscesses, or persistent symptoms. (d) When there is evidence of liver failure. Absence of a secure route is the only contraindication.

Indian Journal of Pediatrics, Volume 73—September, 2006
Good results have been reported in series on children. Failure is mostly due to technical causes like (a) inappropriate approach into a non-dependent portion of cavity, (b) failure to recognize and respond to sepsis, and (c) premature withdrawal of drains. Catheters should be withdrawn once there is negligible (<10 ml) pus drain per day and when patient is afebrile. Complications are bacteremia, and bleed externally or into the peritoneum or GIT (hemobilia), and sometimes iatrogenic superinfection.

Role of surgery: Presently surgical drainage is usually reserved for patients (a) who have failed percutaneous drainage, (b) those who require management for an underlying abdominal problem, (c) selected patients with multiple macroscopic abscesses, (d) those on steroids and (e) patients with ascites.

Transperitoneal approach is the usual method used. This is because the entire liver can be exposed, best drainage site can be determined, multiple abscesses identified with an intraoperative USG, and entire abdomen explored for the primary source of infection. Good results with a low mortality has been reported in literature. Morbidity and Mortality

Complications: List of possible complications are long. However pleuro-pulmonary complications are the commonest. It may be in form of pleural effusion, empyema, pneumonitis, and hepatopleural or hepatobronchial fistula. Other complications may be in form of ascites, Budd-chiari syndrome, intra-peritoneal rupture of abscess and peritonitis, intrapericardial rupture, pericardial effusion, septic shock, hemobilia and Jaundice. Rare complication like perforations into a hollow viscus like colon, stomach and duodenum and cerebral amoebiasis are also reported.

Markers of early detection of complications: Patients who present with jaundice, large or multiple abscesses, acute abdomen, liver failure and sepsis have complications more often.

Mortality: Facility of prompt diagnosis with imaging, percutaneous drainage, and better antibiotics have remarkably improved survival in the last three decades. With modern management most of the series have a mortality of less than 15%.

Markers for mortality: In our study on patients with amoebic liver abscesses, bilirubin levels >3.5 mg/dl, encephalopathy, volume of abscess cavity, number of abscesses, and hypoalbuminemia (<2 gm/dl) were independent risk factors for mortality.

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Liver Abscess in Children


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