Fatal case of amoebic liver abscess in a child

Khuen Foong Ng¹, Kah Kee Tan¹, Romano Ngui², Yvonne AL Lim², Amirah Amir², Yamuna Rajoo², Hassan Hamimah ³, Mahmud Rohela ³*

¹Department of Pediatric, Tuanku Jaafar Hospital, 70300, Seremban, Negeri Sembilan, Malaysia
²Department of Parasitology, Faculty of Medicine, University of Malaya, 50603, Kuala Lumpur, Malaysia
³Department of Medical Microbiology, Faculty of Medicine, University of Malaya, 50603, Kuala Lumpur, Malaysia

ARTICLE INFO

Article history:
Received
Received in revised form
Accepted
Available online

Keywords:
Amoebic liver abscess
Entamoeba histolytica
Child

ABSTRACT

We reported a case of amoebic liver abscess (ALA) in a 6-year-old Malaysian boy who presented with fever, lethargy, diarrhea and right hypochondriac pain. On admission he was diagnosed with perforated acute appendicitis and a laparotomy was done. After surgery he developed acute respiratory distress. Ultrasonography, chest X-Ray and CT scan revealed two ALAs in the posterior segment of right lobe of liver, pleural effusion and collapsed consolidation of lungs bilaterally. Percutaneous liver abscesses drainage was done and intravenous Metronidazole was started. PCR carried out on the pus from the abscess was positive for Entamoeba histolytica. Patient however succumbed to the infection one week after admission.

1. Introduction

Amoebiasis is endemic in the tropical and subtropical regions of the world especially in developing countries. It is the third most common parasitic cause of mortality after malaria and schistosomiasis[1]. Amoebic liver abscess (ALA) is the most common extraintestinal manifestation of invasive amoebiasis and occurs in 3-9% of patients with intestinal amoebiasis[1]. There are few reports of ALA in children and the disease is life-threatening. It presents with high grade fever, right upper quadrant pain, leukocytosis and raised erythrocyte sedimentation rate. Jaundice and derangement of liver enzymes were unusual[2-4]. It can rupture into the pleura to cause acute respiratory distress[3]. Increase in morbidity and mortality is attributed to low index of suspicion of ALA causing a delay in diagnosis. The prognosis of ALA in children improves with early diagnosis and prompt treatment.

2. Case report

A previously healthy 6-year-old Malay boy from Seremban, Malaysia presented with fever for 4 days duration associated with right hypochondriac pain, non-bloody diarrhea and vomiting for 3 days. He was also lethargic with reduced oral intake and urine output. There was a history of swimming in a water park 1 month prior to presentation. On physical examination, the child was dehydrated but fully conscious, tachypnoeic (respiratory rate 40 breaths/minute), febrile (temperature 38.5 °C), tachycardic (heart rate 134 beats/minute) but blood pressure was normal (104/62 mmHg) and oxygen saturation was 98%. The abdomen was distended, tensed and guarded. There was a palpable vague mass and tenderness in the right upper quadrant. Respiratory examination was normal.

On admission, total white cell count was 27.3×10⁹/L (neutrophils 84.3%, 23×10⁹/L), hemoglobin 11.0 g/L, platelet 617×10⁹/L, urea 1.1 mmol/L, sodium 126 mmol/L, potassium 2.8 mmol/L, chloride 92 mmol/L, creatinine 33 mmol/L, albumin 20g/L and C-reactive protein 291.5 mg/L. Alkaline phosphatase, hepatic transaminases, bilirubin level and blood gas were normal.

*Corresponding author: Mahmud Rohela, Department of Parasitology, Faculty of Medicine, University of Malaya, 50603 Kuala Lumpur.
E-mail: rohela@ummc.edu.my
Foundation project: It is supported by University of Malaya (H-20001-00-E00651).
The patient was given intravenous boluses of resuscitation fluid to correct dehydration. He was subsequently referred to the surgeon with the suspicion of acute appendicitis presenting as acute abdomen. Pre-operative diagnosis was perforated acute appendicitis. Ampicillin-sulbactam was started before he underwent laparotomy. Operative findings were presence of 400 mL of straw-coloured ascitic fluid, hepatomegaly and a layer of slough at the upper rectum. Appendix was only mildly inflamed, most likely reactionary. The postoperative diagnosis was spontaneous bacterial peritonitis and the antibiotic was changed to penicillin and cefotaxime to cover for peritonitis.

The patient became increasingly dyspnoeic 12 hours after the surgery with reduction of breath sound over the right side of the chest. Oxygen requirement also increased. A chest radiograph showed massive pleural effusion and lung consolidation over the entire right hemithorax with no cardiomegaly (Figure 1A). Ultrasound thorax and abdomen later revealed 2 heterogenous collections in the posterior segment of the liver, consistent with liver abscess and right pleural effusion. Antibiotic was changed to cloxacillin and ceftriaxone. The patient was intubated prior to CT thorax and abdomen, which subsequently confirmed the discovery of 2 loculated hepatic abscesses in the right lobe (one measuring 6.9 cm × 8.3 cm × 9.3 cm and another 5.5 cm × 6.5 cm × 7.2 cm), ascites, bilateral pleural effusion and collapsed-consolidation of both lungs (right more than left) (Figure 1B).

Ultrasound guided percutaneous liver abscesses drainage was done and thick anchovy paste-like pus was aspirated (Figure 1C). Microscopy examination and staining of the pus for *Entamoeba histolytica* (*E. histolytica*) trophozoites was negative. Parenteral metronidazole was immediately started on day 5 of admission. *E. histolytica* was detected in the aspirated material by PCR technique. Serological test for *E. histolytica* was not done. There was no an *E. histolytica* cyst or trophozoites found in the stool sample and PCR test on the stool for *E. histolytica* was also negative. Pus, tracheal secretion, peritoneal fluid and blood for bacterial culture were negative. PCR of blood specimen for *Streptococcal pneumonia* and serological test for *Mycoplasma pneumonia* antibodies were negative. Histopathology examination of the appendix that was removed during laparotomy only showed early acute appendicitis which was not in agreement with patient’s clinical presentation.

While the patient was in pediatric intensive care unit, there were no seizures, no signs of pericardial tamponade or congestive cardiac failure and the pupils were equal and reactive to light. Level of consciousness was not able to be ascertained objectively as the patient was heavily sedated due to difficult ventilation secondary to severe recurrent wheezing. The patient continued to deteriorate and became hypotensive requiring 4 inotropes. Patient passed away on day 7 of admission.

### 3. Discussion

Amoebic liver abscess (ALA) is a complication of intestinal amoebiasis. It is more common in male adults and male children and is associated with severe morbidity and mortality[1,5]. There are few reports of ALA in children. In Malaysia, 20% of ALA reported was seen in children[6]. The predisposing factors to transmission of amoebiasis are malnutrition, poor sanitary conditions, low socioeconomic status and overcrowding[1,7]. Our patient came from an urban area and from a middle income group and the only apparent risk factor was a history of swimming in a water park 1 month prior to presentation. No one is spared the risk of acquiring *E. histolytica* infection[7].

ALA usually present with fever, right upper quadrant pain and tender hepatomegaly[2]. Our patient presented with an acute illness with typical clinical features of fever, lethargy, right hypochondrial pain with a palpable vague mass and tenderness in the right upper quadrant. There was also a history of diarrhoea three days before admission. A study reported diarrhoea in 20% of children with ALA[8]. Unfortunately, the diagnosis of ALA was missed in this child in the early stage of presentation.

Patients with ALA present with leukocytosis and on admission our patient's total white cell count was raised with predominant neutrophils[9] alkaline phosphatase was however normal. Since respiratory examination on admission did not reveal abnormality except for tachypnoea, which was attributed to high fever, severe abdominal distension and pain, no chest X-Ray was done on admission. He was suspected of having acute appendicitis presenting as acute abdomen and was subjected to laparatomy. At laparatomy even though appendix was found to be mildly inflamed, with signs of peritonitis and hepatomegaly, no diagnosis of ALA was made. The shortfall of this case was that ultrasonography of the abdomen should have been carried out before performing laparotomy because percutaneous catheter drainage could be carried out as a bedside procedure to drain the abscesses (depending on the size of the abscess, drainage is done if the abscess is more than 10 cm in diameter) instead of placing the child under general anaesthesia.

Direct thoracic extension of ALA is unusual in children[9]. This patient developed symptomatic right pleural effusion and pneumonia which was detected 12 hours after surgery. Chest radiography, ultrasonography thorax and CT thorax revealed bilateral pleural...
effusion and collapsed consolidation of both lungs, right lung more than left. Ultrasonography abdomen revealed the abscesses in the posterior segment of the liver. CT abdomen confirmed two hepatic abscesses in the right lobe and ascites. There was no evidence of abscess rupture from the CT scan. Involvement of the lungs and pleural space may represent a sympathetic pleural effusion, contiguous with liver abscess and similarly with ascites, it may represent sympathetic reaction. This effusion serves as a warning of the proximity of the abscess to the pleural and peritoneal cavities. There is a possibility of its rupture into the serous cavities. It is not possible to differentiate effusion from ruptured abscess till tapping is carried out[10]. Study reported multiple ALAs in over 50% of pediatric patients[10]. They also reported that extrahepatic extension as a result of rupture or contiguous extension of ALA to one or more organs complicates the disease in 17% of patients. Another study reported solitary abscesses in 22 of 24 children and there were no mortality despite a delay of about 2 weeks before admission to the hospital[2]. This was attributed to a high index of suspicion, early treatment with Metronidazole and abscess aspiration of those with potential to rupture. Study also reported 8.3% of multiple ALA in children[1]. Three cases out of 48 had complication of abscess rupture but there was no mortality. In our patient, diagnosis of ALA was not made intraoperatively. It was difficult to feel the abscesses at laparotomy because of their position in the posterior segment of the liver. Case of a 2-year-old infant that was missed as ALA until it ruptured into the pleural cavity and caused respiratory distress[3].

Since diagnosis of ALA was initially missed in this case, Metronidazole was not started until on day 5 of admission. Most cases of ALA respond to intravenous Metronidazole which is the drug of choice[1]. Patient was treated in the Pediatric intensive care unit but his condition continued to deteriorate and he succumbed to the infection on day 7 of admission. Mortality in children with ALA is high (42%)[7]. The mortality rate of ALA cases which was reported to be around 11% to 14% before 1984 has reduced to less than 1% at present[11]. Factors which contribute to the severity of ALA in children are failures to establish a prompt diagnosis as revealed in this case and diagnosis may prove difficult. There may be difficulty in isolation of the trophozoites. In addition, seropositivity may be delayed/absent in very young children where humoral antibodies production is less efficient[7]. In our patient, microscopic examination and staining for E. histolytica trophozoites was negative in both stool and pus samples and no serological test for amoebiasis was carried out.

Early diagnosis and prompt treatment aid in the reduction of the morbidity and mortality rates. Ultrasonography is useful in making early diagnosis and treatment[7,12–14]. With the advancement of molecular technique, PCR is useful in diagnosing the presence of antigen in the pus obtained from liver abscess. It is a sensitive and specific method[9]. As in this case, etiology was confirmed by PCR.

Diagnosis of ALA requires a high index of suspicion especially in children. In our patient, the diagnosis was made only after he developed respiratory distress. The delay in diagnosis of ALA even though the clinical features were present could be avoided by carrying out the diagnostic radiological procedures available in most hospitals. Ultrasonography is very useful, quick, safe, cheap, non-invasive and is an accurate tool to establish the diagnosis. It aids to locate the site for abscess aspiration and follow-up after treatment. PCR is a useful test to confirm liver abscess to be due to E. histolytica.

Conflict of interest statement

We declare that we have no conflict of interest.

Acknowledgements

This laboratory work was supported by University of Malaya (H-20001-00-E00051).

References