

CASE REPORT

Liver abscesses in a neonate

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Abstract

Liver abscess is rare in newborns and fewer than 100 cases have been reported in the literature. In general, they are common in preterm infants with certain risk factors like umbilical vein catheterization, immunodeficiency, sepsis and so on. A possibility of hepatic abscess needs to be considered in a neonate presenting with sepsis and tender hepatomegaly.

Majority of liver abscesses respond to intravenous antibiotics and does not need needle aspiration or surgical exploration. Here we report a case of the multiple liver abscess in a term baby secondary to sepsis.

Keywords: Liver Abscess, sepsis, umbilical vein catheterization

INTRODUCTION

Liver abscess (LA) is an uncommon, life-threatening, medical emergency, particularly in a newborn (1). A LA is rarely reported in newborns. Newborns with prematurity or low birth weight are at particular risk for LA because of the poorly developed immune defense system and presence of several predisposing factor such as central venous catheterization during intensive care, umbilical vein catheterization, necrotizing enterocolitis, and umbilical sepsis. Here we report a case of 28 days old baby with multiple liver abscesses.

CASE REPORT

A 28 days old baby girl presented with complaints of decreased feeding, abdominal distension and excessive crying for past 10 days. There were no history of fever, vomiting, and loose stools. Antenatal period was uneventful and the baby was delivered per vaginum at term and birth weight of 2.8 kg. Perinatal period was uneventful. Bothe, baby and the mother, were discharged after 48 hours of delivery. Baby was exclusively on breast feeding. The baby had no history of hospitalization or umbilical sepsis.

Physical examination revealed normal temperature, excessive cry, normal neonatal rooting and sucking reflexes, fontanelles were not full, and no feeding difficulties. Baby had no icterus. Abdominal examination showed abdominal distension, tender hepatomegaly, and normal bowel sounds. Liver span was 5.5 below the right costal margin in mid-clavicular line.

Laboratory investigations showed total leucocyte count 13000 per cubic millimeter with 52% polymorphs. Blood C-reactive protein (CRP) was >90 (normal <5). Coagulase negative staphylococcus (CONS) was grown on blood culture. Ultrasound abdomen showed multiple, well defined, hypodense space occupying lesions in both lobes of liver. The largest lesion measured 3.7x4.5x3.4cm with approximately 30ml volume. One of the abscesses in right lobe was ruptured into subcapsular space.

The baby was treated with intravenous antibiotics (Piperacillin and tazobactam) for 21 days along with other supportive measures. treatment appropriate iv antibiotics for 3 weeks. No invasive intervention were needed for abscess drainage.



Figure 1 . CT abdomen showing multiple hypodense lesions, largest measuring approximately 9.2x2.6cm in size

DISCUSSION

LA is commonly encountered among children and adult population. Till date very few cases of LA are reported in newborns. Literature suggests that in newborns, most of the LAs occur in premature and low birth weight babies. Their increased risk for LA could be secondary to frequent invasive procedures and placement of multiple venous lines in neonatal intensive care units (NICUs) such as umbilical vein catheterization, central venous line, and parenteral nutrition.

A LA can be either solitary or multiple. Solitary liver abscess, among children and adults, are most commonly caused by infection with *Entamoeba histolytica* which is uncommon in newborn. The majority of multiple liver abscesses are of bacterial in etiology. Multiple LA usually follows either bacteremia or portal pyemia secondary to bacterial infection localized to either abdominal wall (such as umbilical sepsis) or inside the abdominal cavity such as appendicitis, bacterial dysentery, bowel perforation, diverticulitis or biliary obstruction. Because of varied possible etiologies, multiple liver abscesses are much more common than single amebic abscess.

Neonatal liver abscess can be caused by a variety of organisms. Polymicrobial infection is found in up to 50% of abscesses. The most common bacteria cultured from neonatal liver abscess are *Staphylococcus aureus*, *Streptococcus pyogenes*, *Escherichia coli*, *Klebsiella* spp., and *Pseudomonas* spp.(2,3). Case reports of neonatal liver abscess caused by *Corynebacterium acnes*, anaerobic bacteria, and *Candida* spp. have also been published (4). These microorganisms may enter the liver and produce an abscess by four possible mechanisms: (1) an ascending infection via the umbilical and portal veins; (2) hematogenous spread; (3) ascending infection via the biliary tract; and (4) by direct contiguous spread from neighboring organs(3-6).

Clinical features are non-specific and include septic appearance, fever, intolerance to feeding, vomiting, abdominal distention, abdominal tenderness, and hepatomegaly. A raised diaphragm is the only useful sign on plain thoracic and abdominal X-rays. Abdominal USG is usually the first investigation that can detect, locate, and define a liver abscess with a sensitivity of 80–90%.(7) It is useful for diagnosis, monitoring, guiding the invasive treatment.

A non-cystic LA on USG could mimic infantile hemangioendothelioma, hepatic hamartoma or hepatoblastoma. A contrast enhanced CT scan

abdomen confirms the diagnosis and also defines the number, size, location, distribution, and its rupture into adjacent organs such as subhepatic space. Typical CT appearance is that of a well-circumscribed, low-attenuation mass with a contrast-enhancing rim.

Portal vein thrombosis, portal cavernoma formation, and portal hypertension are known complications of LA(8-10) which could be confirmed with doppler US or contrast enhanced CT. Babies who develop portal vein thrombosis, should be followed up during the first 5 years of life with US and Doppler studies to look for development of portal cavernoma and portal hypertension. Neonatal portal vein thrombosis can be managed conservatively with intravenous low molecular weight heparin (enoxaparin) 1.5 mg/kg subcutaneously every 12 h. Singh et al(9), reported successful conservative management of portal vein thrombosis in their case with enoxaparin with complete resolution and no long-term complications. Their case had multiple small liver abscesses that also resolved completely with conservative management with antibiotics.

Liver abscess could solely present as tender hepatomegaly in newborns. The main stay of conservative management is appropriate antibiotic therapy for prolonged duration. It can easily be diagnosed with ultrasound examination. With prompt diagnosis and appropriate treatment, the outcome seems to be better.

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