CASE REPORT

NEONATAL HEPATIC ABSCESS WITH PORTAL VEIN THROMBOSIS

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Abstract
Hepatic abscesses are rare in newborns and their diagnosis is difficult and delayed, as the symptoms are non-specific. Portal vein thrombosis and cavernoma formation are rare complications following neonatal liver abscess. We report a neonate who presented to us as ongoing sepsis who on ultrasonography was found to have hepatic abscess with portal vein thrombosis. Prolonged antibiotic treatment led to resolution of the hepatic abscess.

Key words: hepatic abscess, neonate, portal vein thrombosis

Introduction

Hepatic abscess is rare in the neonatal period and usually not taken into consideration in the differential diagnosis of sepsis. Liver abscess leading to portal vein thrombosis is very rarely reported with few case reports (1-5). However, over half of these children had umbilical vein catheterization as a predisposing factor.

We present a neonate who presented to us as sepsis without any predisposing factor and on ultrasonography (USG) was detected to have liver abscess with portal vein thrombosis. We present this case for its rarity.

Case Report

A 37 weeks term, appropriate for gestational age baby with birth weight 2800g was delivered vaginally at our hospital to a 25 year old second gravid mother without any risk factors. The Apgar scores at 1 and 5 minutes were 8 and 9 respectively. The baby did not require any interventions or neonatal intensive care unit admission and was discharged on the third postnatal day. The baby came back to us on the eighth postnatal day with fever, abdominal distension and excessive cry. Initial examination on admission showed fever (39^0C), irritability, tachypnea, and tachycardia. There was no cyanosis or pallor, all peripheral pulses were well palpable. Abdomen was distended and tense. Liver was firm and palpable 5 cm below costal margin in right mid clavicular line with a span of 9 cm. Abdominal examination also showed sluggish bowel sounds and periumbilical redness. On investigation, C-reactive protein (CRP) was 48 mg/l, complete blood count showed hemoglobin of 14.6 g/dl, total leukocyte count of 17,904/cumm with a neutrophilic predominance. Liver function tests were normal. After sending blood culture baby was started on Cefotaxime and Amikacin. Baby responded to treatment clinically, so nasogastric feeding was introduced on the third day of admission and increased over the subsequent days. The child developed abdominal distension with decreased bowel sounds on fifth day. Diagnosis of necrotizing enterocolitis was thought of and USG was done which revealed multiple hypoechoic lesions in the liver suggestive of septic foci with thrombosis of left branch of portal vein. Diagnosis of liver abscess was made and blood culture also revealed growth of methicillin resistant staphylococcus aureus (MRSA) sensitive to injection Vancomycin and Clindamycin. The baby was given injectable antibiotics Vancomycin and Clindamycin for fourteen days. A repeat ultrasound after 2 weeks revealed resolution of the hepatic lesions and the portal vein thrombus without collaterals. The baby was discharged after two weeks. Repeat investigations after 14 days showed CRP<6mg/l, hemoglobin of 8.1gm% with total leukocyte count of 10,100/cumm. Liver function test showed serum bilirubin of 12.03mg/dl, serum glutamic pyruvate transaminase (SGPT) was 49U/l, serum glutamic oxaloacetic transaminase (SGOT) was 50U/l. In view of normal liver enzymes, breast milk jaundice is considered as cause of hyperbilirubinemia. The baby is under regular follow up and growing well.

Discussion

Liver abscess is very rarely present in neonatal period and it differs from older children. The first review of liver abscess appeared in 1936 by Kutsunai (6) who reported 2 infants with fatal peritonitis with solitary liver abscess at necropsy. Since then, about 100 cases of solitary neonatal liver abscess have been reported. (5) Of the 18 cases reviewed by Doerr et al (7) nine patients survived. Four of the survivors were preterm and low birth weight and had a history of umbilical venous catheterization. One of the survivors, 2.2 kg, 34 wks, developed solitary hepatic abscess as a delayed complication of neonatal bacteremia .(8) In the case series by Tan et al (9) all were premature with a median gestational age of 24.5 weeks and birth weight of 743 grams. Our patient was full term baby without any interventions.

Majority of liver abscesses result from an ascending infection via the umbilical and portal veins, hematogenous spread, or via the biliary tree, or via direct contiguous spread from neighboring structures. (10) The causative agent of neonatal pyogenic liver abscess is variable. Although S. aureus and gram-negative enteric bacteria are the most common pathogens isolated from a neonatal liver abscess, any organism can potentially cause an abscess. Other reports have isolated klebsiella pneumonia, amoebiasis, candida species, chryseobacterium gluem and acinetobacter baumani from blood culture and liver abscess culture. (9) Reports from literature has proved that the risk factors for development of liver abscess includes blood culture proven sepsis, umbilical cannulation, central parental nutrition, necrotizing enterocolitis , surgery, immunodeficiency in form of chronic granulomatous diseases and prematurity. (9)

Computed tomography scan (CT) and ultrasound are the most sensitive diagnostic modalities for detecting hepatic abscess. (11) USG has a sensitivity of 80-90% and CT has a sensitivity of 97%. But due to high resolution imaging, lack of need of prior preparation and easy availability USG remains the investigation of choice for the pediatric liver abscesses. (9) The characteristic CT appearance is that of a well-circumscribed low attenuation mass with a contrast-enhancing rim (3).

Pyogenic neonatal liver abscess remains uniformly fatal if untreated. The high mortality reported in the literature in the era before percutaneous aspiration or drainage indicates that percutaneous aspiration or drainage should be seriously considered in solitary liver abscesses whenever possible, even in high-risk patients because antibiotic treatment alone may be insufficient and unsuccessful. Lee et al (12) showed in their series of 8 neonates with liver abscesses, imaging-guided percutaneous aspiration or drainage performed with long-term antibiotic coverage have good long-term results and minimal complications. Percutaneous aspiration offers reduced risk along without need for general anesthesia.

Complications of liver abscess include bacteremia and rupture of the abscess into the peritoneal cavity. Other rare complications reported include metastatic septic emboli to the brain, empyema and acute glomerulonephritis, portal vein thrombophlebitis and portal cavernoma formation. (9)

Known factors associated with the initiation and propagation of thrombosis, in general, include endothelial damage during catheter placement, composition of the infusate, catheter characteristics, and the duration and location of catheter placement. (13) In neonates, factors predisposing to thrombus formation include low birth weight, birth hypoxia, and sepsis. Many reports have determined that umbilical catheterization increases the risk of portal vein thrombosis but in our case there were no such risk factors or any interventions done. But the patient had a culture proven sepsis along with periumbilical redness indicating umbilical sepsis which could have lead to liver abscess and subsequently to portal vein thrombosis.

Obstruction of portal vein leads to development of collateral channels around it, which tend to maintain the hepatoportal flow but progressively lead to development of portal hypertension by 3-5 years of age which are seen as cavernous transformation of portal vein on doppler ultrasound.

Conclusion

Though portal vein thrombosis is an extremely rare complication of hepatic abscess in neonates, these case reports indicate the need for repeated ultrasound supervision of neonatal hepatic abscess for portal vein patency and regular follow up.

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