

Hepatic Abscess in a Preterm Neonate

Shailesh Sharma

A. Mohta*

Prakash Sharma**

We present a preterm (32 weeks; weight: 1300g) neonate, with solitary hepatic abscess. The causative organism was Klebsiella. The child presented with septicemia, hepatomegaly and abdominal lump. Radiological investigations were inconclusive. Surgical drainage and prolonged antibiotic treatment led to resolution of the hepatic abscess.

Key words: Hepatic abscess, Klebsiella, Neonate.

Hepatic abscess is rare in the neonatal period, and usually not taken into consideration in the differential diagnosis of obscure masses in the upper abdomen.

Case Report

A preterm, 32 weeks, appropriate for gestational age baby with birth weight of 1300 g was delivered vaginally to a primigravida. Apgar scores at 1 and 5 minutes were 8 and 9 respectively. The mother had leaking per vaginum for more than 12 hrs prior to delivery. The neonate was referred to our hospital at 36 hours of life in a stable state. Initial investigations including septic work up were normal.

Nasogastric feeding was introduced on day 4 of life and increased over the subsequent days. The child developed abdominal distension with bilious aspirates and decreased bowel sounds on day 9.

From the Departments of Pediatrics, Surgery, and Anesthesiology**, University College of Medical Sciences and Guru Teg Bahadur Hospital, Delhi 110 095, India.*

Correspondence to: Dr. Shailesh Sharma, 39, Bharat Apartments, Sector 13, Plot No. 20, Rohini, New Delhi 110 085, India. E-mail: drprakashsharma@rediffmail.com

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Enteral feeding was discontinued. The liver was enlarged, 3 cm below costal margin, firm, smooth surface with well defined margins. Another lump was palpated in the right hypochondrium 5 × 6 cm, firm, smooth, immobile and was felt separately from the liver. Investigations revealed Hb = 10.8 g/dL, TLC=11,900 mm³, platelet count = 58,000 per mm³; micro ESR=10 mm, peripheral smear revealed toxic granules, arterial blood gas analysis was normal and blood culture showed a pure growth of Klebsiella species sensitive to ciprofloxacin and amikacin. Amebic serology was non reactive.

Ultrasonography was inconclusive about the character of the lump; CT of the abdomen revealed a cystic mass in the liver (Fig.1). The character of the lump outside the liver could not be commented but revealed leakage of the dye from the terminal ileum. On day 12, exploratory laprotomy under general anesthesia was done. At surgery, small bowel was seen to be adherent to the under surface of right lobe of liver. On separation, a perforation of 7mm size was seen in terminal ileum. Right lobe of liver had a large abscess filled with organized pus that was evacuated. There was no bilious leak from the raw surface of abscess cavity. Rest of the abdomen was normal without any evidence of peritonitis. Bowel perforation was closed and abscess cavity was drained. The infant was put on injection vancomycin, ceftriaxone and metronidazole, which were changed to ciprofloxacin and amikacin after the culture reports. The post

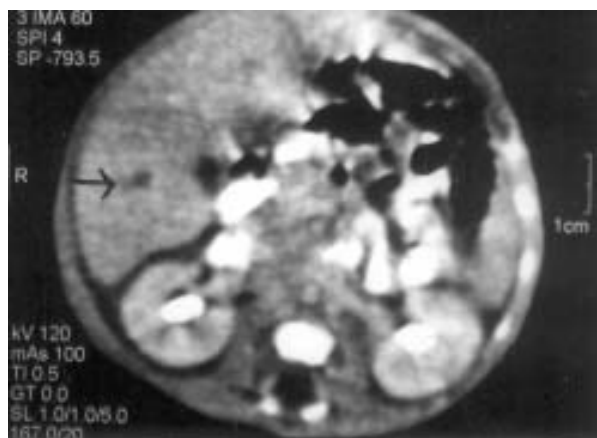


Fig. 1. CT showing the hepatic abscess as marked by the arrow

operative period was uneventful. Serial USG of the abdomen after 7 days showed that the size of the abscess had reduced to $1.7 \times 1.9 \times 1.6$ cm. The child was discharged after 6 week.

Discussion

The first review of liver abscess appeared in 1936 by Kutsunak(1) who reported 2 infants with fatal peritonitis with solitary liver abscess at necropsy. Since then, about 50 cases of solitary neonatal liver abscess have been reported(2). Of the the 18 cases reviewed by Doerr, *et al.*(3) 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(4). Another 6 cases of neonatal liver abscess were reported by Tan, *et al.*(5).

Infection of the liver in the neonate differs from that in the older children in that opportunistic and parasitic infections are unusual. Congenital tuberculosis, congenital syphilis, and listeriosis have been reported as etiological causes(1). The hepatic abscess can be either multiple or solitary. Multiple liver abscess as the name suggests are multiple, small in size, not drainable, usually not due to umbilical infection and have a fulminant course. Whereas, solitary liver abscess are larger, well localized, can be drained by surgical methods, usually due to multiple infection and have a subacute course.

There can be multiple routes of infection to liver(6). It can be direct invasion via contagious structure or hepatic artery through systemic circulation or portal vein via umbilical vein, mesenteric or splenic vein. Lymph channels along with umbilical vessels can also contribute. Major risk factors for hepatic abscess are sepsis, umbilical catheterization and omphalitis(7). Minor risk factors include necrotizing enterocolitis, abdominal surgery, maternal infections, infant of diabetic mother, gastroschisis requiring surgery, exchange transfusion, VP shunt and asphyxia neonatorum.

The classical presentation of liver abscess with fever, hepatomegaly and right upper abdominal

pain is generally not present in neonates. The diagnosis of liver abscess in the neonate cannot be established from the clinical picture alone. The signs and symptoms are nonspecific and are essentially those of sepsis(6). In one of the reports, out of the 24 reported case of neonates with solitary hepatic abscess the diagnosis was made at necropsy in 75%(1,7).

Both the rarity of solitary hepatic abscess in the neonate and its indolent course require a high index of suspicion for early diagnosis(4). In our case also, the correct diagnosis was delayed until an exploratory laprotomy was performed. Abdominal ultrasonography, CT scan, liver scan with technetium are all useful studies to visualise a possible hepatic abscess(6). However, in our patient abdominal USG and CT scan were inconclusive and revealed just the leakage of dye from terminal ileum.

Staphylococcus aureus, *Streptococcus pyogenes* and *E. coli* are the most common organisms isolated from solitary hepatic abscess in neonates(8). However, *Klebsiella*, *Pseudomonas*, *Corynebacterium acnes*, anaerobes and even *Candida* have been reported(9). Polymicrobial infection is found in up to 50% of infants(3).

Early surgical exploration, adequate drainage and adjunctive antimicrobial therapy led to a favorable outcome in this preterm, VLBW neonate.

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Biotin Responsive Limb Weakness

B. Adhisivam
Deonath Mahto
S. Mahadevan

We report acute onset quadriplegia in a ten year old boy associated with basal ganglia lesions due to biotin deficiency. Prolonged raw egg consumption was identified as predisposing factor for biotin deficiency. Treatment with biotin resulted in remarkable recovery.

Key words: Biotin, Basal ganglia, Egg, Quadriplegia.

Acute onset quadriplegia is a frequent neurological problem in children. Guillain Barre syndrome and poliomyelitis present as acute flaccid quadriplegia while injury to head and cervical spine, meningoencephalitis and cerebrovascular accidents lead to quadriplegia with upper motor neuron signs. Biotin deficiency as a cause

From the Department of Pediatrics, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry 605 006, India.

*Correspondence to: Dr. B. Adhisivam, Senior Resident, Department of Pediatrics, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry 605 006, India.
 Email adhisivam1975@yahoo.co.uk*

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of quadriplegia is extremely rare. We describe a 10-year-old boy with biotin responsive basal ganglia disease (BBGD) presenting as spastic quadriplegia.

Case Report

A developmentally normal and well nourished 10 year old boy was admitted for weakness of all four limbs. Initially, he had frequent falls and an unsteady gait due to weakness of the distal muscles of his lower limbs. Within two weeks, there was rapid progression of the weakness to involve his proximal and trunk muscles so that he could neither move his limbs nor get up from his bed. He was anorexic, lethargic and not conversing. The bladder and bowel functions were normal. There was no history of preceding trauma, fever or seizures.

On examination, the child appeared dull and had mask like facies. He had emotional lability and responded only to painful stimuli. The cranial nerves and optic fundii were normal. He had generalized hypotonia for the first two days followed by rigidity. Deep tendon reflexes were exaggerated. Ankle clonus and Babinski's sign were noted bilaterally. His sensory system was normal. Examination of the other systems including the skin was normal.

Investigations revealed the following: Hb 11.8 g/dL, TLC $13,300 \times 10^3/\mu\text{L}$, DLC N 55% L42% M1% E2%. The peripheral smear and serum cholesterol were normal. CSF examination was