

REFERENCES

1. Ramanan A, Pandit N, Yeshwanth M. Unusual complications in a multidrug resistant *Salmonella typhi* outbreak. *Indian Pediatr* 1992, 29: 118-120.
2. Christi AB. Typhoid and paratyphoid fevers. *In: Infectious Diseases—Epidemiology and Clinical Practice*, 3rd edn. Ed Cristi AB. Edinburg, Churchill Livingstone, 1980, pp 47-102.
3. Jasudasan MV, Jacob John T. Multidrug resistant *Salmonella typhi* in India. *Lancet* 1990, 336: 252.
4. Meghan JP, Phillips KG, Cox KL. Sonography of normal pediatric gallbladder and biliary tract. *Radiology* 1982, 144: 873-875.
5. Silvert M, Vakil NB. Emergence of biliary tract. *Gastroenterol Clin North Am* 1988, 17: 245-247.
6. Shaffer EA. Gallbladder disease. *In: Pediatric Gastrointestinal Disease*. Eds Walker WA, Durie PR, Hamilton JR, Smith JA, Watkin JB. Philadelphia, B.C. Decker Inc., 1991, pp 1152-1170.
7. Glenn F. Acute acalculous cholecystitis. *Ann Surg* 1979, 189: 458-465.
8. Glenn F, Becker CG. Acute acalculous cholecystitis. *Ann Surg* 1982, 195: 131-136.
9. Brenner RW, Stewart CF. Cholecystitis in children. *Curr Surg* 1964, 21: 327-335.
10. Stuart BM, Pullen RL. Typhoid. *Arch Intern Med* 1946, 78: 629-661.
11. Gupta SK, Gupta V. Cholecystitis and cholelithiasis in children. *Indian Pediatr* 1991, 28: 801-803.
12. Kabra SK, Taiati A, Shah R, Desai KD, Modi RR. Acute acalculous cholecystitis. *Indian Pediatr* 1991, 28: 803-805.
13. Ternberg JL, Keating JP. Acute acalculous cholecystitis—Complication of other illnesses of childhood. *Arch Surg* 1975, 110: 543-547.
14. Sherlock S. Gallstones and inflammatory gallbladder diseases. *In: Diseases of liver and Biliary System*, 8th edn. Eds Sherlock S. Oxford, Blackwell Scientific Publications, 1989, pp 671-674.
15. Sanghameshwaran KV, Chaturvedi AK, Mathai SS, Purkaystha SB, Dhall A. Role of abdominal ultrasound in evaluation and followup of enteric fever. *Indian J Radiol* 1991, 1: 29-34.
16. Sachdev HPS, Sharma S, Khandpur SC, Kulshresta R. Sonography in diagnosis and management of acute acalculous cholecystitis. *Indian Pediatr* 1987, 24: 379-383.

Conservative Treatment of Bile Ascites in a Neonate

J.R. Sharma
S.R. Dave
V.K. Gandhi
H.C. Shah

Ascites of a greater degree which is not associated with generalized edema, is a rare occurrence in a neonate(1). Till now, less than 50 cases of bile ascites have been

From the Department of Pediatrics, V.S. General Hospital, K.M. School of Post Graduate Medicine and Research, Ahmedabad 380 006.

Reprint requests: Dr. Jayendra R. Sharma, Assistant Professor of Pediatrics, Government Medical College, Surat 395 001.

*Received for publication: December 31, 1991;
Accepted: July 9, 1992*

reported in the literature. Once the diagnosis is made, operation is mandatory for exploration of the biliary tract. With the advent of non-invasive imaging techniques and better understanding of the entity, bile ascites can be managed conservatively in some cases which is being exemplified by our retrospective observation in this case.

Case Report

A male baby was delivered normally without significant antenatal events. He presented at the age of 14 days with complaints of abdominal distension which had developed over 5 days and constipation for 2 days. Physical examination showed a jaundiced newborn, weighing 2.5 kg with significant ascites, umbilical hernia without organomegaly. He passed light yellow stool after admission and was accepting feeds well throughout the hospital stay. Investigative workup revealed a Hb of 12.5 g/dl, TLC 9000/cu mm, (DLC P-55, L-40), the sepsis screen was negative, total serum bilirubin was 11 mg/dl (direct 4.6 mg/dl, indirect 6.4 mg/dl), SGPT was 20 IU, serum alkaline phosphatase 16 KA units, serum VDRL was negative, blood urea was 30 mg/dl, serum creatinine was 0.8 mg/dl, and serum electrolytes were Na⁺ 130 meq/L, K⁺ 3 meq/L and Cl-102 meq/L. X-ray chest, X-ray abdomen and micturating cystourethrogram were normal. Abdominal sonography showed normal viscera and biliary tract but septae were present in between the ascitic fluid. Ascitic fluid was bile stained, containing 3.3 g/dl of protein, plenty of polymorphs and bilirubin content was 3.2 mg/dl. Gram stain and culture of ascitic fluid were negative. The child was given broad spectrum parenteral antibiotics until the sepsis was ruled out. An exploratory laparotomy was planned which was refused by relatives. So ascitic tapping

was done thrice with proper aseptic precautions at weekly intervals for the drainage. A significant decrease in abdominal girth was noted and the child was discharged with mild to moderate ascites at request. The parents did not turn up for followup and incidentally they reported after 10 months for respiratory tract infection. On examination, the child was anicteric without ascites and organomegaly. According to parents, the abdominal distension cleared up in 4 weeks time after discharge from hospital. Repeat liver function test and abdominal sonography were normal.

Discussion

Bile ascites in neonate is an extremely rare condition. Caulfield in 1936, for the first time, reported a case of "bile peritonitis"(2). The term "bile ascites" in the place of previously used term "bile peritonitis" was introduced by Davies and Elliot-Smith in 1955 because the condition is essentially an escape of bile following the rupture of some component of extrahepatic biliary system, forming a collection in the peritoneal cavity(3).

Most of the cases have their onset at the end of neonatal period(6,7) and the clinical course is similar in most cases regardless of the etiology(8). Bile ascites classically presents with a gradual onset, characterized by mild fluctuating jaundice, acholic pale stools, fluid hernias and ascites due to leak of bile from the biliary system to the peritoneal cavity. The combination of clay colored stool indicating obstruction to the flow of bile with minimal jaundice and slight bilirubinuria suggest that bilirubin is escaping elsewhere instead of being reabsorbed in to the blood stream(3). The bilious peritoneal fluid indicates that bile has leaked in the peritoneal

cavity. Bonde reviewed 4 cases of his own and 41 cases from the literature. Among these, 36 had perforation of the bile duct, 6 had anomaly of ductal system and 11 had cholelithiasis(4). Thus, perforation of the bile duct is the commonest cause. The site of perforation is relatively constant at the junction of cystic and common hepatic duct in most cases. Johnston(7) pointed out that relative constancy of the site of perforation would argue a developmental *locus minoris resistentiae* which might rupture as a result of raised intraductal pressure as obstruction was found in 60% cases. In patients where obstruction was not evident, it is conceivable that inspissated bile may cause transitory obstruction which may resolve or lead to raised intraductal pressure within subsequent perforation. Once perforation has occurred, inspissation might no longer be detectable. Lee and Mitchell(9) have also supported this view.

Jaundice drew the attention to the biliary tract pathology in the present case but certain peculiarities of this case merit attention: (i) Early presentation at the end of second week with sudden onset; (ii) Lack of the objective evidence of biliary obstruction as reflected by yellow stool and normal alkaline phosphatase value; (iii) Comparatively high serum bilirubin level on admission which lasted for a short period; (iv) Ascitic fluid analysis showed high protein, polymorphonuclear predominance and significant bilirubin content; and (v) Uneventful subsequent course and ultimate outcome. The spontaneous perforation of extrahepatic part of biliary tract is the likely pathology in a present case. The pathogenesis of bile ascites can be explained on the basis of latter part of above mentioned Johnston hypothesis. The second week of neonatal period is most vulnerable for bile inspissation with gradual

maturation of liver enzymes. The bile inspissation was a probable cause for spontaneous and sudden onset of ascites which was marked by raised bilirubin level. The hyperbilirubinemia subsided after a short period without active intervention. The fluid collection in peritoneal cavity in bile ascites initially occurs as a result of chemical peritonitis which is often painless in the neonatal period and infancy in contrast to adults. Some of the fluid is drawn from the various peritoneal and intestinal tissues by relatively high osmotic pressure of escaping bile(6). These explain the ascitic fluid picture(9). Retrospectively, with the uneventful course and gradual subsidence of ascites without surgical intervention in our case, one can reasonably assume that the biliary leak would have healed with repeated drainage as refilling of ascites was not there and the ascites showed definite regression during the hospital stay after repeated tapping.

HIDA scan is an excellent tool to demonstrate the biliary leak in a case with serum bilirubin level less than 4 mg/dl. The first followup sonography is very important in a conservatively managed case as a choledochal cyst can be a long term complication.

REFERENCES

1. Lord JM. Fetal ascites. Arch Dis Child 1953, 28: 398-403.
2. Caulfield E. Bile peritonitis in infancy. Am J Dis Child 1956, 85: 694-697.
3. Davies PA, Elliot-Smith A. Bile peritonitis in infancy. Arch Dis Child 1955, 30: 174-178.
4. Bonde J, Cotany A. Peritoneal biliary effusion and perforation of biliary tract in infant. Ann Child Infant 1966, 72: 257-314.
5. Lele RD. Principle and Practice of

- Nuclear Medicine. London, Arnold Heinemann Publishers 1984, pp 311-333.
6. Gupta BD, Miglani N, Gupta GL. Bile ascites: A case report with review of literature. *Indian Pediatr* 1979, 16: 91-94.
 7. Johnston JH. Spontaneous perforation of common bile duct in infancy. *Br J Surg* 1961, 48: 532-535.
 8. Patterson A. Spontaneous perforation of common bile duct in an infant. *Acta Clinic Scanda* 1955, 110: 192-195.
 9. Lees W, Mitchell JE. Bile peritonitis in infancy. *Arch Dis Child* 1966, 41: 188-192.

Intraperitoneal CSF Pseudocysts Following Ventriculo-Peritoneal Shunts

S. Chandra
V. Bhatnagar
D.K. Mitra

During the last two decades ventriculo-peritoneal (VP) shunts have been increasingly used for the management of hydrocephalus in children. A wide spectrum of complications have been noted after this procedure. This communication deals with intraperitoneal pseudocyst formation following VP shunts.

From the Department of Pediatric Surgery, All India Institute of Medical Sciences, New Delhi 110 029.

Reprint requests: Dr. D.K. Mitra, Additional Professor, Department of Pediatric Surgery, All India Institute of Medical Sciences, New Delhi 110 029.

*Received for publication: July 4, 1991;
Accepted: September 3, 1992*

Case Reports

Case 1: This 6-year-old girl had been initially treated for congenital hydrocephalus at the age of 45 days by a ventriculoatrial (VA) shunt. Subsequently, she underwent revision of the lower end at 2½ years of age and the VA shunt was converted to VP shunt using the Upadhyaya shunt valve with placement of the peritoneal end in the sub-hepatic space. Six months following this revision, the patient presented with complaints of headache and non-bilious vomiting. On examination, the child was irritable and a non-tender cystic lump could be felt occupying the right hypochondrium and epigastric region. There was no guarding or rigidity. Clinically, the shunt reservoir required very high pressure to empty it. Routine laboratory investigations were normal; there was no leucocytosis. On abdominal exploration a large fluid filled cyst was found situated between the liver and stomach, its antero-inferior wall being formed by the omentum. The peritoneal end of the shunt was within this cyst. The cyst was partially excised and as the cerebro-spinal fluid (CSF) drainage was normal the distal end of the shunt was resited in the pouch of Douglas. The examination of CSF from the shunt and the cyst was biochemically, microscopically and bacteriologically normal. Seven years later she is well and has not had recurrence of CSF pseudocyst.

Case 2: This 5-year-old boy had presented with congenital hydrocephalus and a VP shunt was done at the age of 1 month using the Upadhyaya shunt valve. Four years later the child was brought to the hospital with complaints of headache, non-bilious vomiting, and lethargy for 2 weeks. On examination there was a large non-tender cystic swelling occupying most of the right side of the abdomen. The distal