be well localised, and a history of fatty food intolerance may be difficult to elicit. Jaundice as a presenting symptom of cholelithiasis in childhood is uncommon. There is probably little doubt that cholecystectomy represents the treatment of choice for children presenting with symptomatic cholelithiasis. Controversy, however, still exists in the management of asymptomatic cholelithiasis. Jacir *et al.*(10) have suggested long term observation of asymptomatic children for spontaneous resolution of gall stones while others recommend cholecystectomy because of the associated sequelae and complications of gall stones(11).

In conclusion, it can be stated that though cholecystitis and cholelithiasis is an uncommon problem in childhood, it should be considered in all patients with upper abdominal pain. Ultrasonography is diagnostic and cholecystectomy is the treatment of choice both in symptomatic and asymptomatic children. In India, long term follow up for a possible spontaneous resolution of cholelithiasis is difficult due to poor socio-economic status of the patients.

REFERENCES

1. Bhanasali SK. Cholelithiasis and cholecystitis. J Postgrad Med 1980, 26: 74-85.
2. Chatterjee A, Banerjee P. Evaluation of cholecystitis in young patients. Indian J Surg 1989, 51: 293-295.
3. MacMillan RW, Schullinger JN, Santulli TV. Cholelithiasis in childhood. Am J Surg 1974, 127: 689-692.
4. Gibson J. An extraordinary large gall bladder and hydropick cystitis. Medical essays and observations. Philosoph Soc Edin 1737, 2: 352.
5. Ariyan S, Shessel FS, Pickett LK. Cholecystitis and cholelithiasis masking as abdominal crisis in sickle cell disease. Pediatrics 1976, 58: 252-258.
6. Glenn F, Hill MR Jr. Primary gall bladder disease in children. Ann Surg 1954, 139: 302-311.
7. Whitington PF, Black DD. Cholelithiasis in premature infants treated with parenteral nutrition and furosemide. J Pediatr 1980, 97: 647-649.
8. Benjamin DR. Cholelithiasis in infants: The role of total parenteral nutrition and gastrointestinal dysfunction. J Pediatr Surg 1982, 17: 386-389.
9. Roslyn JJ, Berquist WE, Pitt HA, *et al.* Increased risk of gallstones in children receiving total parenteral nutrition. Pediatrics 1983, 71: 784-789.
10. Jacir NN, Anderson KD, Eichelberger M, Guzzetta PC. Cholelithiasis in infancy: Resolution of gallstones in three of our infants. J Pediatr Surg 1986, 21: 567-569.
11. Bailey PV, Connors RH, Tracy TF, *et al.* Changing spectrum of cholelithiasis and cholecystitis in infants and children. Am J Surg 1989, 158: 585-588.

Acute Acalculous Cholecystitis

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Acute acalculous cholecystitis is rare in children(1-4). The acute symptom complex

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of pain abdomen, vomiting and right upper quadrant tenderness and/or mass in abdomen; frequently led to exloratory laparotomy and subsequent cholecystectomy(5,6). Until recently, the diagnosis of acalculous cholecystitis was pre-operative and the recommended treatment was surgical(4,7). We present our experience of acalculous cholecystitis in a 4-year-old boy due to chloramphenicol resistant *Salmonella typhi* infection, diagnosed by ultrasound and treated conservatively.

Case Report

A 4-year-old boy presented with history of fever, loose stools, vomiting, pain abdomen and abdominal distension for 6 days. For these complaints the child was admitted in a private nursing home, where he was treated with intravous fluids, ampicillin and gentamicin. Since he did not respond to the treatment he was referred to V.S. General Hospital, Ahmedabad with diagnosis of liver absecss for further management. General physical examination revealed a toxic, irritable and febrile child. His abdomen was distended with positive bowel sounds. His liver was enlarged 3 cm below costal margin with firm consistency. A tender globular cystic swelling measuring 3 × 3 cm continuous with the lower margin of liver and mobile in horizontal plane was present in the right side of abdomen. A firm and nontender splenomegaly was present. Rest of the systemic examination was within normal limits.

Investigations revealed a hemoglobin of 11 g/dl, total leucocyte count of 6500/cu mm with 65% polymorphs and peripheral smear showed normocytic hypochromic anemia, serum bilirubin was 0.8 mg/dl with direct of 0.6 mg/dl. SGOT, SGPT levels were 35 and 45 units/L, respectively. Serum alkaline phosphatase was 20 KA units/dl. Widal test was positive with titres of 1: 240 against TO and TH antigens Renal function tests, and urine examination were within normal limits. Blood culture revealed *Salmonella typhi* which was sensitive to cephazoline, gentamicin and ciprofloxacin; however, it was resistant to chloramphenicol, ampicillin and cotrimoxazole. Radiological investigations of abdomen showed hepatomegaly with soft tissue shadow in right side. Ultra sound abdomen revealed enlarged distended gall bladder with edematous wall, sludge in its lumen which was moving with posture and absence of calculi (*Fig.*). Apart form hepatomegaly rest of the hepatobilliary system and other viscera were normal.

He was treated with intravenous fluids, cephazoline and gentamicin. By 3rd day oral fluids were started and intravenous fluids stopped; antibiotics were continued for 14 days. His hospital course was uneventful. He became afebrile by 3rd day, lump abdomen was not palpable by 4th day and there was no pain abdomen even on deep palpation by 5th day. Repeat ultrasound on day 14 revealed a normal gall bladder with mild hepatomegaly. On follow up at 3 months he is a completely normal child with no problems

Discussion

Gall bladder diseases are rare in children and acute acalculous cholecystitis (AAC) is extremely rare(1,5,8). In upto 30-60% of cases AAC occurs secondary to systemic infections such as typhoid fever, gastrointestinal infections, scarlet fever, erysepelas, urinary tract infections, bacterial pneumonias and leptospirosis(9). In our patient it occurred secondary to *Salmonela typhi* which was resistant to routine

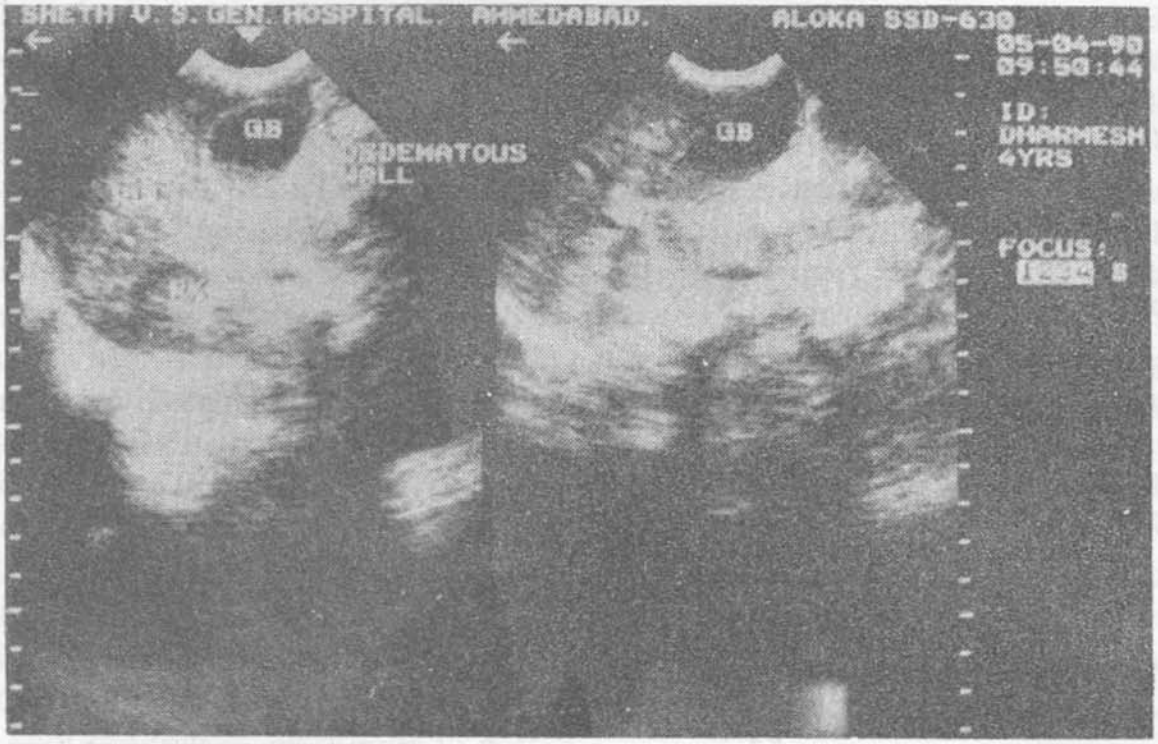

Fig. 1. Ultrasound abdomen showing enlarged, distended gall bladder with edematous walls and sludge in it's lumen which is moving with posture and absence of calculus.

drugs. The clinical observations were similar to those, described in cases confirmed preoperatively(4,5,8). The ultrasonographic findings were similar to those described earlier for diagnosis of AAC(3). In the earlier literature the recommended treatment for cholecystitis was cholecystectomy or tube cholecystostomy or simple aspiration(1,6). Experience with conservative management of acute acalculous cholecystitis has been reported recently(3). Our patient responded to conservative therapy. In the present state of experience it can be concluded that if surgical complications such as gangrene of gall bladder, perforation, *etc*. are absent; a trial of conservative treatment is one of the modality for management of AAC.

REFERENCES

1. Shandling B. Cholecystitis. *In:* The Liver and Biliary system in Infants and Children. Ed Chandra RK. Edinbugh, Churchill Livingston, 1979, pp 306-307.
2. Andrassy RJ, Treadwell TA, Ratner TA, Buckley CJ. Gall bladder diseases in children and adolescents. Amer J Surg 1976, 132: 19-21.
3. Sachdev HPS, Sharma S, Khandpur SC, Kulshrestha R. Sonography in the diagnosis and management of acute acalculous cholecystitis. Indian Pediatr 1987, 24: 379-383.
4. Chandra J. Asharaf SM, Fakhir S, Manazir S, Ahmad SH. Acute acalculous cholecystitis in a young child. Indian J Pediatr 1989, 56: 533-534.
5. Ulin AW, Nosal JL, Martin WL. Cholecystitis in childhood: Associated obstructive jaundice. Surgery 1952, 31: 312-326.
6. Terneberg JL, Keating JP. Acute acalculous cholecystitis. Arch Surg 1975, 110: 543-547.

7. Hays DM. Cholecystitis and cholelithiasis. *In:* Pediatric Surgery. Eds Holder TM, Ashkraf KW. Philadelphia, WB Saunders Co 1980, pp 524-525.

8. Dutta T, George V, Sharma GD, Meenakshi DK. Gall bladder disease in infancy and childhood. Prog Pediatr Surg 1975, 8: 109-118.

9. Silverman A, Roy CC. Acute and chronic biliary tract disease. *In:* Pediatric Clinical Gastroenterology, Ed. Bergeik, St Louis, Missouri, Mosby Company, 1983, pp 97-99.

Hypomagnesemic Hypocalcemia as a Cause of Persistent Upper Airway Obstruction in Acute Bacterial Tracheitis

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Bacterial tracheitis is not an uncommon cause of acute airway obstruction in infancy, characterized by moderate to high grade fever, marked inspiratory stridor, respiratory distress and polymorphonuclear leucocytosis(1). We present here an infant with acute bacterial trancheitis in whom intractable hypocalcemia secondary to an underlying hypomagnesemia lead to persistent laryngospasm and airway obstruction. It is known that 10-15% of patients in an ICU setting develop hypocalcemia(2). While cardiovascular manifestations are the common mode of presentation, laryngospasm has also been reported(2). This may lead to persistent upper airway obstruction despite resolution of underlying disease. Therefore, if not looked for, hypocalcemia may lead to diagnostic and management problems.

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Case Report

A, 2-month-old exclusively breast fed male infant, was admitted to Intensive Care Unit with history of moderate grade fever, stridor and respiratory distress of a few hours duration. The antenatal and early neonatal period had been uneventful.

The baby weighed 4.9 kg which was appropriate for age. On examination, the child was febrile, toxic and had mild cyanosis with severe respiratory distress, a respiratory rate of 68/min, inspiratory stridor and suprasternal, intercostal and subcostal recessions. Direct laryngoscopy revealed thick pus exuding from the laryngeal inlet. The epiglottis was normal and the rest of the systemic examination was non-contributory. A diagnosis of acute bacterial tracheitis was made. Investigations revealed a hemoglobin of 11.8 g/dl, total leucocyte count of 13,500/cumm with polymorphonuclear leucocytosis. The CSF examination was normal and blood and CSF cultures were sterile. Pus from trachea revealed Gram positive cocci, but culture was sterile. Lateral *X*-ray neck showed subepiglottic narrowing while *X*-ray chest was normal with a normal thymic shadow.