

Biliary Pseudolithiasis in a Neonate: A New Complication of Total Parenteral Nutrition

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To the Editor,

Cholelithiasis is uncommon in neonates and infants and is usually associated with predisposing factors such as congenital abnormalities of the biliary tract, hemolytic diseases, total parenteral nutrition (TPN) administration, congenital heart disease, and prolonged fasting. Infants with complications of prematurity are more predisposed to development of biliary calculi. Cholelithiasis may also develop due to use of some medications. Recent studies demonstrated ceftriaxone as inducing reversible precipitations in the gallbladder that mimic cholelithiasis (1). This complication is termed 'biliary pseudolithiasis' or 'reversible cholelithiasis'. Reversible biliary sludge and/or lithiasis appears to be a temporary, self-limiting phenomenon. To our knowledge, no patient has been reported in the literature with biliary pseudolithiasis due to TPN. In this paper, we describe a premature newborn that developed biliary pseudolithiasis after 30 days of TPN therapy, which completely resolved 11 days after the end of the treatment.

A premature female baby born at 30 weeks of gestational age was admitted to our neonatology department with complaints of respiratory distress and cyanosis. She was resuscitated and required mechanical ventilation. Ampicillin (100 mg/kg), amikacin (15 mg/kg) and TPN were initiated. She underwent mechanical ventilation treatment for three days. On the third day of hospitalization, oral feeding was attempted but was not tolerated. On the 12th day after birth she showed signs of a sudden septic condition. Antibiotic regimen was changed to vancomycin (15 mg/kg/dose b.i.d.) and meropenem (20 mg/kg/dose b.i.d.). Blood culture of the infant was coagulase-negative staphylococcus. Anti-biotherapy was given for 10 days. Because of septic condition and oral intolerance, TPN was continued for 30 days after birth. On the 30th day of life, due to prominent abdominal distention, we performed abdominal ultrasonography (USG), which showed biliary sludge with mobile echogenic foci with poor acoustic shadowing in the gallbladder lumen that were demonstrated as gallstones (Figure 1). Hematological (hemoglobin, white blood cell count, platelet count) and biochemical analyses of the blood (total and direct bilirubin, liver function tests, blood urea nitrogen, creatinine and lipids) were performed in the patient. Biochemical parameters were within normal ranges. TPN treatment was discontinued after the diagnosis of cholelithiasis. She was given ursodeoxycholic acid (15 mg/kg/dose b.i.d.) orally. After beginning ursodeoxycholic acid treatment, she tolerated oral nutrition and her clinical condition began to improve. After ursodeoxycholic acid treatment for 11 days, USG revealed resolution of the calculi and biliary sludge (Figure 2).

Cholelithiasis in neonates and infants has been reported only rarely. It is usually associated with known predisposing factors such as hemolysis, congenital anomalies of the biliary tree, TPN, congenital heart disease, and prolonged fasting. Reversible biliary sludge and/or lithiasis, named as pseudolithiasis, have been reported in patients treated with ceftriaxone (2,3). Araz et al. (2) observed ceftriaxone-associated pseudolithiasis in eight patients treated for meningitis. After ceftriaxone treatment was discontinued,

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Figure 1. Abdominal USG showed biliary sludge with mobile echogenic foci with poor acoustic shadowing in the gallbladder lumen that were demonstrated as gallstones.



Figure 2. Follow-up USG revealed resolution of the calculi and biliary sludge.

gallbladder sonograms were found to be normal in all patients at the follow-up sonographic examinations. Herek et al. (1) also described a patient who developed biliary pseudolithiasis after six days of ceftriaxone therapy, which completely resolved 11 days after the end of the treatment

TPN-associated biliary sludge and/or calculi usually do not resolve spontaneously and may require surgery. Our patient did not have hemolytic disease, dehydration, urinary tract infection, or cholestatic liver disease and did not take third-generation cephalosporin. She had only received TPN. The only risk factor for pseudolithiasis formation in our patient was prolonged TPN treatment and prematurity. Holcomb et al. (4) attributed most of the gallstones diagnosed from birth to 2 years of age to the use of TPN in premature infants. Infection, especially

as the result of Gram-negative bacilli, may have a lithogenic effect on bile by decreasing the independent bile salt flow. In the blood culture of our patient, coagulase-negative staphylococcus was isolated. Since gallbladder stone and sludge did not occur during septic condition but 10 days after sepsis, it was not considered as an etiologic factor for biliary pseudolithiasis.

In this case, we want to emphasize prolonged TPN treatment as an etiological factor for biliary pseudolithiasis in premature infants. It appears to be a temporary, self-limiting phenomenon, and an aggressive approach is not warranted in premature infants. Since it may disappear without surgery, in the absence of other clinical or imaging evidence of biliary tract disease, conservative management and serial sonograms are advised.

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