Pseudomonas UTI Masquerading as Neonatal Jaundice: A Case Report

Abstract

The pathological jaundice in neonates develops due to hematological, infectious, surgical causes. We present an interesting case of persistent neonatal hyperbilirubinemia, who was diagnosed to have urinary tract infection (UTI) and effectively managed. Unexplained persistent neonatal jaundice, poor response to phototherapy, and higher rebound total serum bilirubin levels should be evaluated for UTI.

Keywords: *UTI, Hyperbilirubinemia, Neonatal jaundice, VUR*

Introduction

The commonest type of neonatal hyperbilirubinemia is physiological (up to 60%). Few neonates develop pathological jaundice due to ABO or Rh mismatch, hepatic problems, sepsis, metabolic diseases, glucose-6-phosphate dehydrogenase (G6PD), pyruvate kinase, galactose-1-phosphate enzyme deficiencies, hypothyroidism, polycythemia, surgical causes, and more.[1,2] Urinary tract infection (UTI) is an established causes of persistent jaundice.[1,3,4] The rates of culture-proven UTI and prolonged jaundice differ worldwide.[4] We present a case of a newborn with prolonged unexplained jaundice, where the etiology was found to be UTI due to pseudomonas aeruginosa, rarely reported as a cause of such a condition.

Case Report

A term male baby was born via Cesarean section to a G3P1L1A1 mother. He was born without complications. His mother's antenatal course was uneventful; anomaly ultrasound scan was normal, and there were no maternal risk factors for neonatal infection. Both the mother and the baby had identical blood group. He looked pink and active on clinical examination with a normal cry, muscle tone, and reflexes. There was no apparent dysmorphism, organomegaly, or clinical stigma of intrauterine infection. He was on exclusive breastfeeding with normal weight gain and urine output. He passed urine and stool on day one of life, and his stool color was normal.

Icterus was noticed on day two of his life over face and chest, which progressed to involve palms and soles on day five and total serum bilirubin (TSB) was 21.2 mg/dl (unconjugated fraction: 20 mg/dl). He was put on double-surface phototherapy for the next 48 hours and again required phototherapy for 24 hours due to a rebound rise of bilirubin with a gap of 36 hours. On evaluation, his blood reports were normal, including hemogram, renal function, urine analysis, thyroid profile, direct Coombs test negative, G6PD, and lactate dehydrogenase (LDH). Sepsis workup was negative. He was discharged with TSB of 15mg/dl. On the tenth day of his life, he was readmitted with the worsening icterus. Re-workup showed a rise in TSB of 17mg/dl (unconjugated and conjugated fraction 15mg/dl and 2mg/dl, respectively). All other blood parameters except mild transaminitis were normal, including hemogram, DCT, renal function, C-reactive protein, and blood culture. Repeat sepsis screen came negative. Ultrasound of the whole abdomen showed normal liver and biliary tracts. Ultrasound revealed thickened urinary bladder wall. Catheterized urine sample showed heavy growth of Pseudomonas aeruginosa. According to the sensitivity report, intravenous antibiotic was started, following which the child clinically improved; TSB and bilirubin fractions showed a downtrend (TSB 12.04mg/dl, unconjugated and conjugated fraction 11.7mg/dl and 0.7mg/dl, respectively) and liver enzymes normalized. Subsequently, his TSB and direct bilirubin levels decreased to 2mg/dl and 0.5mg/dl, respectively, on follow-up. After treatment of UTI, a micturating cystourethrogram was performed, which revealed right-sided grade-1 vesicoureteral reflux (VUR). The baby was discharged on oral antibiotic prophylaxis of UTI (oral Cephalexin 10mg/kg/day) on day 31 of his life.

Discussion

The prevalence of UTI in asymptomatic infants with unexplained prolonged hyperbilirubinemia varies worldwide, reported as 7% in developed countries and ranging from 0.6% to 53.9% in lower-income countries.[1,3,5] Neonatal hyperbilirubinemia is a known cause and can be the sole initial manifestation of UTI.[1,2] Male gender, preterm, low birth weight neonates are more prone to have UTI.[3] Immature local immunity, low secretary immunoglobulin A (IgA), low urothelial bactericidal activity, less renal acidification, and more colonization around the urethra are risk factors for UTI in neonates.[6] The causative organisms in UTI with prolonged unexplained jaundice in male neonates with VUR are mostly gram negative; among them, the most common is Escherichia coli, followed by Klebsiella pneumoniae. Enterobacter, serratia, Acinetobacter bacilli, Staphylococcus aureus, and Pseudomonas are other less commonly reported.[1,2,3] The proposed mechanism of hyperbilirubinemia is the production of hepatotoxins and hemolysin by gram negative organisms, which increases red blood cell (RBC) fragility and hampers intestinal excretion.[1] Studies also revealed that prolonged phototherapy requirements, the highest peak of unconjugated bilirubin levels, and higher rebound bilirubin levels are more associated with UTIs.[1,2] Indirect hyperbilirubinemia is more common, though direct hyperbilirubinemia is also reported.

A possible mechanism is hepatic microcirculatory changes by bacterial products, such as endotoxin.[7] The resolution of the condition has been proven after UTI treatment. Studies showed that 16%–55% of cases had underlying kidney urinary tract anomalies, which include hydronephrosis, VUR (10%–15%), pyelonephritis, and renal stones.[2,3,7]

Conclusion

The neonates who have unexplained persistent jaundice, including poor response to phototherapy, and develop higher rebound TSB levels should be evaluated for UTI. Early diagnosis and timely management are of utmost importance for favorable outcomes. Underlying urinary tract anomalies should be looked for.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest

There are no conflicts of interest.

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