Case Report

Exchange transfusion for severe jaundice: Time is brain

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Abstract

Background: Neonatal hyperbilirubinemia is a common clinical problem and is usually managed successfully with phototherapy. However, severe hyperbilirubinemia approaching or exceeding exchange transfusion thresholds, particularly when associated with neurological dysfunction, constitutes a neonatal emergency. Although exchange transfusion has become less frequent in many settings, it remains a critical rescue intervention in selected infants. Current American Academy of Pediatrics (AAP) recommendations emphasize that decisions regarding exchange transfusion should be based on gestation- and age-specific bilirubin thresholds integrated with clinical neurological assessment. We report a late preterm infant with very high bilirubin levels and early neurological signs who required urgent double-volume exchange transfusion, underscoring the importance of timely escalation of care in an appropriately equipped neonatal intensive care unit (NICU).

Key words: Neonatal hyperbilirubinemia; Neonatal Intensive Care Unit (NICU); Bilirubin-Induced Neurological Dysfunction (BIND)

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1. Case Presentation

A late preterm neonate born at 36 weeks' gestation with a birth weight of 3.24 kg was delivered by emergency caesarean section for non-progress of labor. The infant cried immediately after birth and did not require resuscitation. All neonates delivered in hospital undergo pre-discharge bilirubin assessment. At 40 hours of life, total serum bilirubin (TSB) was 16.9 mg/dL, and double-surface phototherapy was initiated.

After 24 hours of phototherapy, TSB decreased to 11.13 mg/dL at 60 hours of life, and the infant was discharged with advice for follow-up.

At the first postnatal follow-up on day 7 of life, the infant was noted to have deep jaundice extending to the palms and soles. Neurological examination revealed a high-pitched cry, poor suck, incomplete Moro reflex, and variable hypotonia. Serum bilirubin at presentation was 23.83 mg/dL, and the bilirubin-induced neurological dysfunction (BIND) score was 3, consistent with early acute bilirubin encephalopathy.

2. Intervention

The infant was immediately admitted to the NICU and initiated on intensive triple-surface phototherapy. Given the TSB approaching exchange transfusion thresholds in association with neurological signs, an urgent double-volume exchange transfusion was planned. An umbilical venous catheter was placed, and exchange transfusion was performed using 10 mL aliquots over 41 cycles, completed within one hour. We used fresh O Negative Packed RBC suspended in AB plasma cross matched to the neonate. The procedure was uneventful.

Post-exchange, triple-surface phototherapy was continued. Serum bilirubin levels declined progressively from 23.83 mg/dL to 14 mg/dL, and subsequently to 9.92 mg/dL. Evaluation for other causes of hyperbilirubinemia revealed a negative direct Coombs test, normal liver and renal function tests, normal reticulocyte count, and no evidence of hemolysis on peripheral smear. The G6PD screen was also negative. The infant was discharged in stable condition with a normal neurological examination at discharge.

3. Discussion

This case highlights the need to integrate bilirubin levels with neurological assessment when deciding on escalation of therapy in neonatal hyperbilirubinemia. As per AAP recommendations, urgent exchange transfusion is indicated when TSB is at or above exchange thresholds or when signs of intermediate or advanced acute bilirubin encephalopathy are present. In this infant, the combination of very high bilirubin levels and early neurological manifestations justified prompt exchange transfusion.

Infants requiring exchange transfusion should be managed in a tertiary-level NICU with trained personnel and appropriate blood product support. When such facilities are unavailable, early consultation and urgent transfer, with continuation of intensive phototherapy and supportive care during transport, are essential. Timely recognition and appropriate escalation of care remain critical to preventing bilirubin-related neurological injury.