presented with lethargy. This neonate was born at term after a normal pregnancy by normal spontaneous vaginal delivery and weighed 2700 grams. He had received 1 mg of vitamin K intramuscularly at birth. The neonate was well until day nine when presented with lethargy and refusal of feeds. All the routine markers of sepsis were negative including blood culture and cerebrospinal fluid analysis. He received a compatible blood transfusion during his hospital stay for blood stained gastric aspirate. Clinically the baby improved after five days of antibiotics although he developed progressive icterus and high coloured urine. He was then referred to our hospital for the management of his progressive jaundice.

On examination, the infant was icteric. There were no bleeding tendencies, no gastric aspirates. Liver was palpable 2 cm below the right costal margin with a span of 6 cms. There was no splenomegaly or any other masses in the abdomen. Urine was high coloured and stools were normal. His haemoglobin was 9.8 gm/dl. Direct Coombs’s Test was negative which ruled out mismatched transfusion. C-reactive protein level was 24 mg/dl (Normal <6 mg/dl). Aspartate transaminase was 118 units (Normal 15-40 units) alanine transaminase was 98 units (Normal 10-40 units). Prothrombin time was prolonged to 48 seconds control being 16 seconds. Activated partial thromboplastin time was 60 seconds control being 44 seconds. Alkaline phosphatase was 1194 units/dl (Normal 10-40 units). Total bilirubin was 44 mg/dl with direct component of 34 mg/dl. Ultrasonography abdomen showed normal echo texture of liver with biliary sludging at gall bladder. No other masses were seen. With these reports, a diagnosis of Ceftriaxone induced cholestasis was made. On applying Naranjo’s algorithm, a score of 4 is obtained hence this can be regarded as a possible adverse effect of Ceftriaxone.³

Ceftriaxone was stopped. Icterus subsided in 3 days, from bilirubin of 44/34 mg/dl at admission to normal on day 3 of stopping Ceftriaxone. The infant was discharged. Follow up ultrasonography of abdomen was normal on day 7 of discharge.

Ceftriaxone is well tolerated and safe in neonates according to a study by Mulhall et al.⁴ It is eliminated equally in bile and urine. Papadapaulov et al demonstrated biliary concerns, which completely disappeared after 2-63 days of stopping treatment.¹ Zinberg et al reported a case of biliary obstruction and secondary pancreatitis in association with reversible Ceftriaxone induced pseudolithiasis.⁵ The most important point to note in our case is quick normalisation of bilirubin levels, liver enzymes and prothrombin time confirmed by ultrasonography ruling out mismatched transfusion. C-reactive protein level was 24 mg/dl which eliminated post infectious jaundice. Normal prothrombin time eliminated the possibility of deep vein thrombosis. Normal bilirubin levels ruled out hemolytic jaundice.

Though mentioned in literature as a common adverse effect, Ceftriaxone induced cholestasis is uncommonly seen in clinical practice especially in paediatric age group. This report is a reminder to the clinicians to be aware of the possible adverse effect of this extensively used drug in routine clinical practice.

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CEFTRIAXONE INDUCED CHOLESTASIS IN A NEONATE:  
A CASE REPORT

Sir,

Ceftriaxone is a third generation cephalosporin frequently used in clinical practice for various infections in all age groups. However, it is associated with life threatening complications such as anaphylaxis and potentially dangerous complications like cholestasis and hypoprothrombinemia.¹² We report a case of Ceftriaxone induced reversible cholestasis in a neonate.

17-days-old male infant was referred to our hospital with direct hyperbilirubinemia after being treated with Ceftriaxone 50 mg/kg/dose twice daily and amikacin 7.5 mg/kg/dose twice daily for 7 days for suspected sepsis on day 9 when presented with lethargy. This neonate was born...