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Association between the specific UGT1A1 promoter sequence variant (c-3279T>G) and unconjugated neonatal hyperbilirubinemia

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Background: Neonatal hyperbilirubinemia is a common presentation, yet the diagnosis is not reached in many cases. We investigated the association between UGT1A1 promoter sequence variant (c-3279T>G) and unconjugated neonatal hyperbilirubinemia.

Methods: 141 neonates 36 weeks gestation were recruited; 63 were included in the jaundice group and 78 age and gender matched neonates with bilirubin < 7 mg/dl were included in the non-jaundice (control) group. Inclusion criteria in the jaundice group were newborns having indirect hyperbilirubinemia necessitating treatment. Newborns presenting with sepsis, asphyxia, cephalohematoma, polycythemia, viral hepatitis and prior blood transfusion or exchange transfusion were excluded.

Results: The frequency of occurrence of c.-3279T>G Allele was significantly higher in the jaundice group (49.2%) than in the non-jaundice group (25.6%). The homozygous state (p=0.001, OR=17.7 & CI 3.9–79.3) rather than the heterozygous state (P=0.3, OR=0.7 & CI=0.3-1.6) was associated with development of hyperbilirubinemia. Among the jaundice group, comparison between the three genotypes; homozygous mutation, heterozygous mutation and the normal allele, revealed that the former represented a major morbidity as reflected by the significantly higher mean peak total serum bilirubin (mean±SD: 33.7±8.2, 26.9±2.8 and 21± 2.7 respectively, p-value 0.0001), higher bilirubin/albumin ratio (p=0.000) and a longer duration of hospital stay (p=0.001). All the homozygous cases presented with an exchange level of bilirubin and 55% had bilirubin induced encephalopathy.

Conclusion: Homozygous c.-3279T>G mutation represents an important risk factor for the development of neonatal jaundice and its presence is associated with high bilirubin levels.

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