Title: SEVERE ACUTE HEMOLYSIS IN G6PD DEFICIENT NEWBORN SECONDARY TO MEFENAMIC ACID

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BACKGROUND: G6PD deficiency is a genetic disorder, which is common in the Middle East. Several drugs are known to cause hemolysis and increase in serum bilirubin. We previously documented increased risk of hyperbilirubinemia and kernicterus in newborns with G6PD deficiency. Mefenamic acid causes a significant increase in the osmotic fragility of red cells and a significant decrease in the activity of AChE and hemoglobin. To our knowledge mefenamic acid has not been reported as a cause of hemolysis in G6PD deficient newborns. We report a breast-fed newborn infant who had severe hemolysis and hyperbilirubinemia whose mother was taking mefenamic acid (Ponstan) as an analgesic.

CASE REPORT: Mustafa is a full term baby boy delivered by Cesarean section with birth weight 3.350 kg. Left hydronephrosis was diagnosed antenataly which was confirmed after birth in addition to pelvi-uretric junction stenosis. Rest of the physical examination was normal. Total serum bilirubin (TSB) was 14.5 mg/dL in the second day of life which decreased by phototherapy to 11.7 mg/dL. Laboratory investigations was unremarkable except for G6PD which was deficient 100 mU/10⁹ RBC (normal for NB 219-564 mU/10⁹ RBC ).

The baby was on breast feeding and routine care waiting for repair operation. The mother took mefenamic acid (Ponstan) 500 mg three times daily as analgesic. One day following Ponstan ingestion, at 6 days of age, TSB increased suddenly to 22 mg/dL, and PCV dropped to 41% from 47%. He was placed under triple phototherapy, breast milk was held, and formula with supplemental intravenous fluid were given. Inspite of that TSB increased to 25.4 mg/dl. Double volume exchange transfusion was performed. Pre and post exchange transfusion investigation were respectively as follows: Hg: 9.9 and 15 g/dL, PCV 27.9 and 43%, TSB 25.4 and 14 mg/dL, and direct bilirubin 3.1 mg/dL. Bilirubin dropped to 6.5 mg/dL before discharge. Mefenamic acid levels in the breast milk and the patient could not be measured. The mother stopped taking mefenamic acid, and resumed breast-feeding, the baby’s bilirubin continued to be normal after that.

SPECULATION: Acute hemolysis secondary to mefenamic acid secreted in the breast milk was most likely the cause of acute and severe hemolysis, which led to acute severe hyperbilirubinemia since no other obvious cause was found.

RECOMMENDATION: Caution should be taken when prescribing any medication for breast feeding mothers in areas with high incidence of G6PD deficiency since a growing list of offending drugs are reported to cause severe hemolysis in G6PD deficient subjects. Screening all newborns for G6PD deficiency in high risk areas is recommended.