Severe Hemolytic Disease of Fetus and Newborn Due to Anti-S Antibodies

Sir,

The relative proportion of hemolytic disease of fetus and newborn (HDFN) caused by non-D antibodies (Abs) has increased with the introduction of anti-D prophylaxis. Anti-S/s Abs constitute 2% of the alloantibodies detected in antenatal women.[1] S antigen is one of the 46 antigens constituting MNS blood group system (M, N, S, s, and U being clinically relevant high prevalence antigens).[2] Immunoglobulin G anti-S alloantibodies causing HDFN result from exposure to alloantigen through prior blood transfusion or fetomaternal hemorrhage. Anti-S Abs are a rare cause of HDFN and generally are thought to cause mild hemolytic jaundice.[3] Severe cases requiring multiple exchange transfusions[4] or resulting in fetal/neonatal deaths have been reported.[3]

A term 2.7 kg baby was born to a mother with the following antenatal history - 27-year-old G2P1, blood group A RhD positive, Kidd typing Jk (a + b–) S phenotype: s + s+, Ab screen positive for anti-Jk^b and anti-S at titers 1:1 and 1:512 at the first antenatal visit and 1:1 and 1:256 respectively at 28 weeks. Ab titer remained at that level until delivery. Her partner's blood group was A RhD negative Jk (a+b+) S phenotype: S+ s + with a 50% chance of fetus inheriting the target antigens. Close antenatal monitoring with biophysical profile and middle cerebral artery peak systolic velocity (MCA-PSV) estimation was started at 24 weeks gestation. MCA-PSV hovered about 1.5 multiples of the median (MoM) reaching a maximum of 82.5 cm/s (just under 1.5 MoM) in the 38th week suggesting possibility of mild fetal anemia. The mother was managed expectantly following the standard protocol.[5] Her Ab screen during her first pregnancy 4 years prior was positive for anti-S and anti-Jk^b at titers 1:512 and 1:2 respectively. Her first baby developed only mild jaundice with negative direct antiglobulin test (DAT).

The second baby had severe anemia and jaundice at birth. Blood results in the first hour revealed the following – cord blood bilirubin 200 µmol/L (11.7 mg/dl), blood group: A negative, S phenotype S + s+, Kidd typing: Jk (a+b–); DAT positive, anti-S Abs eluted off cord red cells; hemoglobin (Hb) 81 g/L, white blood cells (WBC) 21.4 × 10^9/L, platelets 68 × 10^9/L, nucleated red blood cells (RBC) 191/100 WBC, retic count 34%; peripheral blood picture: Marked polychromasia, occasional fragmented and contracted red cells.

With a total bilirubin (TBil) of 260 µmol/L (15.2 mg/dl) at 4.5 h of life, a double volume exchange transfusion

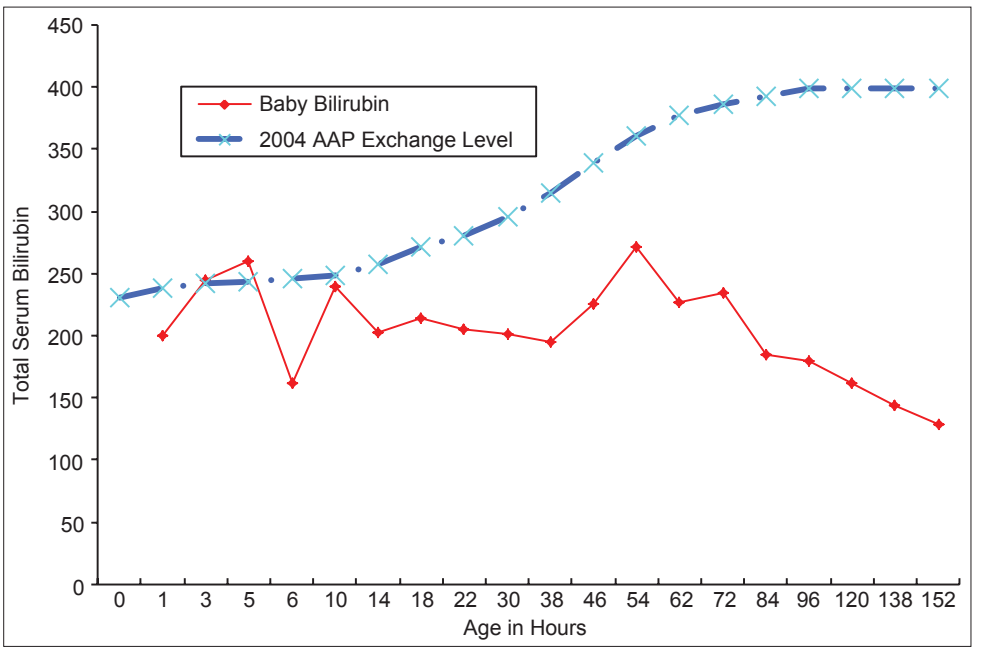

Figure 1: Total serum bilirubin (mmol/L) plotted against time in hours after birth

was performed with O negative blood (packed RBC). Postexchange, TBil was 162 μmol/L (9.5 mg/dl), Hb 199 g/L, platelet count 12 × 10^9/L, international normalized ratio 1.2, prothrombin time 16.4 s, and activated partial thromboplastin time 36.5 s. He received platelet transfusion and in an effort to prevent the need for a second exchange transfusion, 1 g/kg intravenous immunoglobulin. Subsequently, TBil rose to a maximum of 272 μmol/L (15.9 mg/dl) on day 3 [Figure 1]. His conjugated bilirubin peaked at 68 μmol/L (3.97 mg/dl) on day 5 before declining. Phototherapy was continued at maximum intensity and withdrawn by day 5. Hearing screening with automated auditory brainstem response was normal. He developed late anemia with Hb of 81 g/L on day 38, which recovered with Iron and Folate supplementation.

Antenatal monitoring by MCA-PSV did not predict the severity of the disease in this case. Continued vigilance is necessary when any Ab known to be associated with HDFN is detected in a mother, with prompt evaluation and treatment of the affected newborn.

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