

## Haemolytic disease of the newborn caused by high frequency Antigen- "U" identified in Asmara, Eritrea: a case report

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### Abstract

A 24-hour baby boy presented with jaundice on the first day of life. His total bilirubin was 19gm% and direct bilirubin-0.9gm%. By 4<sup>th</sup> day of life despite continuous phototherapy the total bilirubin was 40.8gm% and direct bilirubin-2.3gm%. Both the mother's and the baby's blood group typed B positive. Investigations at the National laboratory centre revealed the presence of Anti-U in both the mother's serum and baby's eluate using the 16-Panocell for antibody identification panel. Exchange transfusion was immediately done using the mixed mother's red blood cells and plasma from a fresh B positive donor.

### Introduction

The "U" antigen is in the MNS blood group system. The MNS blood group system is associated with the M, N, S, s, and U antigens and 32 other rare antigens.

The U antigen is located on a well-characterized glycoprotein called glyophorin B (GPB) on the red cells membrane<sup>2</sup>. This high-frequency antigen is found in all individuals except in 1 percent of African blacks who lack GPB.

Usually Anti-U is associated with mild cases of Haemolytic Disease of newborn and Exchange Transfusion is not indicated.

### Case report

A 24- hour baby boy who was delivered by SVD with the help of vacuum in a national referral hospital Asmara, Eritrea presented with jaundice on admission and was put immediately under continuous phototherapy. He was a product of a term gestation born to a 23- year old G II, PII mother whose pregnancy was uneventful. His Apgar score was 7/10, 8/10 at birth and 5 minutes after birth respectively. His birth weight was 4.2 kg and body temperature on admission-36.8°C. He was sucking vigorously and there was no sign of respiratory distress. At first day of life the total and the direct bilirubins were 19 mg% and 0.9mg% respectively. In the hospital's laboratory CBC was within normal range, CRP was non- reactive and both mother and baby blood group was typed B positive. So Jaundice due to RH and ABO incompatibility was effectively ruled out. By the third day of life despite continuous phototherapy the jaundice increased and the palms and soles were yellowish. Desperate what to do, we sent more specimen and urged the national central laboratory to further investigate the cause of this severe jaundice. By 4<sup>th</sup> day of life the baby has already signs of kernicterus, soon after the National Central Laboratory reported that the baby's total bilirubin was 40.8mg% and direct bilirubin was 2.3mg%. The baby's direct Coombs test was 3+. Both the mother's serum and the baby's eluate reacted positive with all but one cell on the antibody identification panel

(16-Panocell). Then using two U negative cells from separate stored panels, the National Central

laboratory was able to identify the cell. The cell that gave no reaction to the mother's serum and the baby's eluate was U negative that was confirmed latter by the Reference Laboratory of Immucor Inc,USA.

The probability of finding a U negative donor at the local blood bank was very remote and donor inventory was less than 50 units. There was no anti-U typing sera. There was a limited amount of serum from the mother to screen ABO compatible donors. The mother's one sibling living in the country side was ABO compatible but Incompatible with his sister's antibody. So after intensive discussion it was decided to look to the mother as the source of blood and use her red cells for transfusion. The mother's red cells were compatible with the baby's serum and eluate. Fresh B positive donor was the source for plasma. Total Exchange transfusion was done using the mixed mother's red blood cells and fresh B positive donor's plasma after calculation of the total blood volume and hematocrit appropriate for the age of that baby. The baby survived but with minimal neurological sequel. This was the first time that this type of transfusion had taken place in Eritrea.

The child (in case report) at age 2 years and 6 months



### Discussion

A neonate presented with jaundice on the first day of life. Neonatal jaundice, while a normal transitional phenomenon in most babies, can occasionally become more pronounced. Blood group incompatibilities (Rh, ABO, and others) may increase bilirubin production through increased haemolysis. Historically, Rh isoimmunization was an important cause of severe jaundice, often resulting in the development of kernicterus<sup>1</sup>. The usual causes of this presentation such as ABO, RH incompatibilities and others were excluded. Absence of the U antigen, a very rare occurrence estimated at < 1% of Afro Americans who lack GPB<sup>2,3</sup> on their red blood cell membranes was confirmed to be the cause of haemolytic disease of the newborn in our setting.

Some of the causes of jaundice of the newborn include infections especially sepsis and genetically predetermined incompatibility of red blood cells. The severity of the signs and symptoms depend on the extent of the red blood cell destruction and hepatic excretory capacity of the patient. In general the clinical manifestations of haemolytic disease of new born due to U antigen incompatibility are usually so mild that do not require exchange transfusion and resolve spontaneously or through phototherapy.

The occurrence of neonatal jaundice within the first day of life prompted our investigation into the possible

causes of this problem. After the exclusion of the common causes of incompatibility consideration was made of other possible causes. The confirmation of U antigen was a very surprising finding that prompted the definitive management through exchange transfusion. The delay incurred through the diagnostic process contributed to the development in kernicterus which was however minimal as could be testified by the improved condition of the child after two years of age.

There is limited literature written on severe haemolytic disease of newborn due to U antigen incompatibility that necessitates exchange blood transfusion. This case study underscores the need to consider a wide differential diagnosis when dealing with haemolytic disease of the newborn in order to improve neonatal outcomes through appropriate treatment modalities.

### References

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