Hydrops fetalis due to ABO incompatibility

Maura McDonnell, Simon Hannam, SP Devane

Abstract
Antenatal haemolysis in association with ABO incompatibility occurs very rarely. Two cases of hydrops fetalis in black infants caused by anti-B haemolysins are reported. The greater severity of ABO incompatibility in black African peoples may have important implications for antibody screening in this ethnic group.

(Keywords: ABO incompatibility; hydrops fetalis; black Africans)

ABO incompatibility is the most common materno-fetal blood group incompatibility which, unlike rhesus disease, is usually a problem of the neonate rather than the fetus. Anaemia is rare; the main clinical problem is jaundice. The incidence in the United Kingdom is about 2% of all births, but severe haemolytic disease occurs in only 0.03% of births. Hydrops fetalis in association with ABO incompatibility is extremely rare, with single case reports only. We report two cases of hydrops caused by ABO incompatibility.

Case reports
CASE 1
A girl was born at term to a Nigerian couple. The mother’s blood group was O rhesus positive. An emergency caesarean section was performed for fetal distress. At delivery the baby was cyanosed, limp, and bradycardic, requiring intubation and ventilation. Her birthweight was 2740 g. She was pale and oedematous with a petechial rash over the trunk and had a distended abdomen with marked hepatosplenomegaly. Initial investigations showed features of haemolytic anaemia (table 1). A double volume exchange transfusion was performed at 2 hours of age.

CASE 2
This boy was the second of twins born at term to an Afro-Caribbean woman whose blood group was O positive. Her partner was Nigerian. During pregnancy both twins grew along the 50th percentile. The second twin was limp and bradycardic, requiring intubation and ventilation. On admission to the nursery he was pale, jaundiced, and oedematous, with abdominal distension and hepatosplenomegaly. His birthweight was 2032 g. Initial investigations again showed features of haemolytic anaemia (table 1). A double volume exchange transfusion was required.

In both cases full infection and metabolic screens were carried out and were normal. The jaundice and hepatosplenomegaly gradually resolved over a period of 8 weeks.

Discussion
There is a 1 in 5 chance of ABO incompatibility between fetal red cells and maternal serum, yet ABO haemolytic disease of the newborn (HDN) is relatively uncommon, occurring in about 2% of all births. Only three cases of hydrops fetalis with neonatal survival have been described in association with ABO incompatibility.1-3 This condition is rare because anti-A and anti-B antibodies that develop during the first few months of life are usually IgM immunoglobulins that cannot cross the placenta. HDN can only be caused by IgG (immune) maternal antibody. A high titre of immune antibody will not necessarily cause problems in utero as A and B antigens are present on cells of all other tissues and body fluids, and not only on red cells. These fetal antigens help to protect the incompatible fetal red cells by neutralising transferred maternal antibody.

The incidence of severe HDN varies among racial groups. A higher incidence and severity has been observed among Latin American4 and Black peoples.5 In a study of 509 Nigerian volunteers 53.6% of group O individuals had anti-A haemolysins, 62.7% had anti-B haemolysins and 47.9% had both. Thus Nigerians have a high level of anti-A and anti-B haemolysins compared with Europeans, and unlike Europeans, many group O individuals in Nigeria are “dangerous” rather than “safe” universal blood donors.6

The cases reported here show that severe haemolysis can occur as a result of ABO incompatibility and that this diagnosis should be considered in the antenatal evaluation of hydrops fetalis. It is not cost effective to screen for ABO incompatibility as there is no test that is of high predictive value for severe HDN. It is known, however, that haemolysis can be more severe in certain racial groups and so there may be a case for screening for immune antibodies in these groups to monitor fetuses that may be at risk. This may be combined with antenatal scanning to look for early signs of hydrops. Cord blood testing could then be considered in

Table 1 Clinical data at presentation

<table>
<thead>
<tr>
<th></th>
<th>Case 1</th>
<th>Case 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin (g/l)</td>
<td>58</td>
<td>99</td>
</tr>
<tr>
<td>Platelet count (x 10^9/l)</td>
<td>47</td>
<td>110</td>
</tr>
<tr>
<td>Reticulocyte count (x 10^7/l)</td>
<td>160</td>
<td>272</td>
</tr>
<tr>
<td>Initial / peak bilirubin (µmol/l)</td>
<td>290 / 450</td>
<td>214 / 232</td>
</tr>
<tr>
<td>Conjugated SBR</td>
<td>165</td>
<td>152</td>
</tr>
<tr>
<td>AST (IU/l)</td>
<td>404</td>
<td>645</td>
</tr>
<tr>
<td>Blood group</td>
<td>B positive</td>
<td>B positive</td>
</tr>
<tr>
<td>Direct Coomb’s test</td>
<td>Positive</td>
<td>Positive</td>
</tr>
<tr>
<td>Maternal anti B titre</td>
<td>1:1280</td>
<td>1:1280</td>
</tr>
<tr>
<td>Blood film</td>
<td>Polychromasia</td>
<td>Polychromasia</td>
</tr>
</tbody>
</table>
selected cases. This would be of particular importance in areas with a high ethnic mix. Finally, early neonatal discharge may be risky for babies of group O women with high antibody titre, or with a history of ABO incompatibility in a previous pregnancy.

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