Does ABO incompatibility matter?

Sir,

Quinn et al show with the progression from ABO compatibility to ABO incompatibility and then to ABO incompatibility with a positive Coombs test that there is an increasing incidence of jaundice, but the jaundice decreases in severity (see table). About half of the ABO incompatible babies who had a positive Coombs test had a peak bilirubin concentration less than the lower limit of bilirubin in the jaundiced ABO compatible babies.

It is clear that jaundice, a positive Coombs test, and ABO blood group incompatibility are not sufficient criteria on which to base a diagnosis of ABO incompatible jaundice. The data are entirely compatible with the assumption that ABO incompatible jaundice is a figment of the paediatricians' vivid imagination.

Reference


Dr Quinn, Weindling, and Davidson comment:

We concur with Dr Williams' interpretation of our data. There is little doubt that bias was introduced into the ascertainment of jaundice because of the paediatricians' prior knowledge of the result of the elution and Coombs test. This would explain increased frequency of detection with lesser severity in elution and Coombs positive cases. We are not sure whether ABO incompatible jaundice is a figment of the paediatricians' imagination or whether the elution and Coombs test are inappropriate for its detection. Brouwers et al recently published data suggesting that a combination of the antibody dependent cell mediated cytotoxicity assay and antigen density of A or B antigens on the red cells together provide a good screening test for ABO incompatibility. Unfortunately these investigations are not widely available.

Table Details of infants from Quinn et al

<table>
<thead>
<tr>
<th></th>
<th>ABO compatible (control group) (n=110)</th>
<th>ABO incompatible (n=110)</th>
<th>ABO incompatible with positive Coombs test (n=36)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No (%) with jaundice</td>
<td>18 (16)</td>
<td>29 (26)</td>
<td>14 (39)</td>
</tr>
<tr>
<td>Median peak serum bilirubin of jaundiced babies (µmol/l)</td>
<td>190</td>
<td>171</td>
<td>162</td>
</tr>
<tr>
<td>Range</td>
<td>159–295</td>
<td>111–282</td>
<td>111–236</td>
</tr>
</tbody>
</table>

Rectal nifedipine in acute severe hypertension in young children

Sir,

We read with interest the paper by Evans et al on sublingual nifedipine for the treatment of acute severe hypertension. They found that sublingual nifedipine rapidly lowers blood pressure in children with acute severe hypertension and we agree with them.

Evans et al did not experience hypertensive children under the age of 5 years. It is difficult to administer a liquid medicine sublingually to young infants. We, therefore, attempted rectal administration of nifedipine in hypertensive children including infants.

The volume inside a 10 mg capsule of nifedipine is 0.34 ml. Based on our dose range (0.2 to 0.5 mg/kg), we aspirated the extra contents from the capsule with a syringe. We made another hole in the capsule which we then administered rectally. We treated nine severely hypertensive children aged 10 months to 15 years, five of whom were under the age of 5. The hypertension was secondary to glomerulonephritis in three, renovascular hypertension in three, and other causes in three. We observed successful effects of rectal nifedipine in all cases, although Evans et al failed to control blood pressure in some patients. Its effect in lowering blood pressure appeared within 10 minutes after administration, and lasted more than three hours. Side effects were mild and infrequent; they included asymptomatic tachycardia and mild flushing.

Nifedipine is an excellent drug for the treatment of acute severe hypertension as reported. We are sure that rectal administration is reliable in young children.

References

2. Uchiyama M, Hayakawa H, Ogawa T, Hayashi M, Sakai K.