Transplacental passage of anti-ś antibody without haemolysis

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Summary
The production of anti-ś antibodies during pregnancy is a rare event and usually causes haemolysis of fetal red cells. A case is presented where an IgG anti-ś antibody crossed the placenta, but did not produce any evidence of haemolysis.

Introduction
The formation of antibodies to red blood cells is usually due either to previous blood transfusions or to pregnancy. During pregnancy, transplacental haemorrhages allow fetal red cells to enter the maternal circulation. If these cells contain antigens 'foreign' to the mother, an immune response is initiated and IgG antibodies once formed, traverse the placenta producing haemolysis of fetal red cells.

Iso-immunization during pregnancy usually results from incompatibility to the rhesus (usually D antigen) and ABO blood group antigen systems. However, antibody formation against most red cell antigens have been reported and frequently cause haemolytic disease of the newborn.

Anti-ś is a very rare antibody. Only 11% of the Caucasian population lack the ś antigen and are therefore at risk of forming antibodies. Anti-ś antibodies have previously been reported as causing haemolytic disease of the newborn. In the present case, anti-ś crossed the placenta but did not produce haemolysis.

Case report
A 30-year-old woman was first seen in the antenatal clinic early in the course of her fourth pregnancy. Her previous obstetrical history was complicated by a traumatic forceps delivery of her first child 6 years previously, a spontaneous abortion one year later and a normal birth (40 weeks gestation) 2 years later. One blood transfusion had been given following the abortion. No antibodies had been detected in the patient's serum previous to this pregnancy. Her other past medical history was normal.

An anti-E antibody (titre 1:16 by enzyme technique) was detected in the patient's serum at 30 weeks' gestation, subsequently rising to a titre of 1:256 at term. Otherwise, the pregnancy was uneventful and she was admitted to hospital for delivery at term. At this time, a second antibody, anti-ś, was detected (titre 1:256 by indirect antiglobulin technique). This had not been present earlier in the pregnancy using standard laboratory techniques. The delivery was uncomplicated, and at birth, the infant appeared to be slightly jaundiced with a haemoglobin concentration of 13.5 g/dl, and a reticulocyte count of 2%. The direct antiglobulin test was strongly positive on the infant's red cells using specific IgG antisera. An antibody was eluted (ether technique) from the red cells and found to have anti-ś specificity with no anti-E specificity. The infant's serum bilirubin rose to a maximum of 5.9 mg% before falling to normal values within the first week of birth. No specific therapy was necessary. The blood groups and MS genotype of the family are shown in Table 1.

<table>
<thead>
<tr>
<th>TABLE 1.</th>
<th>ABO group, rhesus group, and MS genotype of the family</th>
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<tbody>
<tr>
<td>Father</td>
<td>A</td>
</tr>
<tr>
<td>Mother</td>
<td>A</td>
</tr>
<tr>
<td>Infant</td>
<td>O</td>
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Discussion
Since the first description of an anti-s antibody in pregnancy (Levine et al., 1951), few other reports have appeared in the literature (Giblett, Chase and Crealock, 1958; Lusher, Zuelzer and Parsons, 1966; Drachmann and Brogaard Hansen, 1969; Davie, Smith and Dyball, 1972). All cases had IgG antibodies. Clinical disease was variable. Two patients...
Case reports

had mild haemolysis not requiring treatment, and 3 were severe with one intra-uterine death. Anti-\(s\) has also been reported in pregnancy without causing haemolytic disease of the newborn (O'Riordan and Cann, 1959). This antibody was later found to be IgM.

The present patient was therefore interesting, in-so-far as an IgG anti-\(s\) antibody was present which did not produce any clinical evidence of haemolysis.

The reason why occasional patients do not destroy antibody coated red cells is not known. It is possible that either a defect in reticulo-endothelial clearance is present or that the antibody responsible may not be recognized by tissue-bound macrophages.

References
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