An example of anti-s causing mild haemolytic disease of the newborn

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Synopsis

An example of anti-s found during routine antenatal tests and causing mild haemolytic disease of the newborn is described. The serological and immunological properties of the antibody are described.

Case Report

The patient, Mrs Ba, had a live birth in June 1969. She received 2 pints of compatible group O Rh-negative blood for a postpartum haemorrhage and an intramuscular injection of anti-D immunoglobulin following the birth of the baby. There was a miscarriage in 1970 and she started her third pregnancy in 1971.

She was investigated for the first time at the Wessex Transfusion Centre in March 1972 when the patient came to live in Southampton. At this time she was 38 weeks pregnant.

Methods

All antenatal serum samples received in this laboratory are screened for atypical blood group antibodies against two different batches of fully typed group O cells by a manual cysteine-activated papain technique and by AutoAnalyzer. Antibodies detected by either of these methods are then tested manually against a standard panel of fully typed red cells so that their specificity can be determined.

The AutoAnalyzer method was modified from the procedure using bromelin methycellulose (BMC) described by Marsh, Nicholls, and Jenkins (1968). Group AB serum was omitted and the cell mixture consisted of 10 ml of washed packed cells, 10 ml of 30% bovine albumin, 20 ml of 0.3% aqueous methyl cellulose, and 25 ml of 0.9% sodium chloride. A range expander similar to the one described by Annan and Fisher (1971) was fitted to improve the readability.

The maternal serum was fractionated by ion exchange column chromatography using DEAE-Sephadex. The antibody level in the fractions was estimated on the AutoAnalyzer, and expressed in units of activity with reference to the international standard (anti-D) incomplete blood-typing serum. This standard has a concentration of 64 International Units per ml (Goldsmith, Mourant, and Bangham, 1967).

Other than the techniques used above, standard serological methods were used (Stratton and Renton, 1958).

Results

The patient was group O rr, MSMS, P1+, K+, Le(a−b−), Fy(a+), Jk(a+b+), direct antiglobulin test negative. Routine screening tests revealed an atypical antibody by the AutoAnalyzer technique only.

By subsequent manual techniques using a panel of 'fully typed' group O cells the antibody was identified as anti-s (p = 0.0083). Strong reactions were obtained in the indirect antiglobulin test (titre 1 in 16) using a broad-spectrum reagent, a specific IgG antihuman, and an anti-complement Coombs reagent. Negative results were obtained with anti-IgM. Weak reactions were observed against saline suspensions of cells at 16°C and 37°C and by an albumin displacement method at 37°C. Negative results were obtained with cells treated with ficin and cysteine-activated papain; however, agglutination of bromelin-treated cells was found. The titre of the antibody was 1 in 80 using Ss cells on the AutoAnalyzer.

Following separation on a DEAE-Sephadex column, antibody activity was almost entirely confined in the first IgG protein peak (Table). It was evident that the anti-s was predominantly IgG immunoglobulin and would therefore be expected to cross the placenta. When the cord serum was
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<table>
<thead>
<tr>
<th>Units of Antibody Activity</th>
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<tbody>
<tr>
<td>Maternal serum</td>
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<tr>
<td>IgG fractions</td>
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<tr>
<td>IgM containing fractions</td>
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<tr>
<td>Cord serum</td>
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Table: Antibody levels in maternal and cord serum and fractions of maternal serum following DEAE-Sephadex chromatography

tested anti-s was detected at 0-44 units of antibody activity. The husband was group O Rh (D) positive, probable Rh genotype R1R2, ss.

At birth the infant appeared clinically normal, the total bilirubin level of the cord blood was 3-0 mg/100ml, and the haemoglobin was 16-3 g/100 ml. The direct antiglobulin test was only moderately strongly positive (+ + +) and anti-s was detectable in the cord serum. The infant was group O Rh (D) positive, Ss.

Discussion

Anti-s is an extremely rare cause of haemolytic disease of the newborn. Cases have been reported by Levine, Kuhmichel, Wigod, and Koch (1951), Giblett, Chase, and Crealock (1958), Lusher, Zuelzer, and Parsons (1966) and Drachmann and Brogaard Hansen (1969).

This case report underlines the usefulness of the single-channel AutoAnalyzer for screening antenatal sera for atypical blood group antibodies. Marsh et al (1968) found that their BMC system reacted well with anti-S but anti-s was not tested. While Perrault and Hogman (1971) observed that the detection of anti-s on the AutoAnalyzer was less sensitive than the most appropriate manual technique, the anti-s described in this case was detected at a much higher titre on the AutoAnalyzer than by the indirect antiglobulin technique. The results indicate that the antibody was mainly an IgG immunoglobulin which could have been stimulated by the previous pregnancy but is more likely to have been formed following the previous blood transfusion. The case emphasizes the importance of testing antenatal sera using the indirect antiglobulin technique in addition to an enzyme method.

Anti-s may produce severe haemolytic disease of the newborn as occurred in three of the previously reported cases. In our case the IgG anti-s antibody was transferred across the placenta; the baby had mild haemolytic disease of the newborn and fortunately no treatment was required.

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References


