A case report: clinically benign anti-Cs

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Abstract: Previous reports on the clinical significance of anti-Cs(Cost-Stirling) have presented conflicting data. We report our findings, over an 8-month period, of a patient whose serum contained anti-Cs and anti-Fya. Nineteen donor units of ABO and Rh-matched, Fya-negative red cells, which were crossmatch incompatible, were transfused with no clinical, serological, or biochemical evidence of a hemolytic transfusion reaction.

Anti-Cs was first described by Giles et al. in 1965.1 The Cs antigen has a frequency of approximately 98% in random populations. Prior to 1978, anti-Cs was widely accepted, through personal communications, as a clinically benign antibody based on transfusion records of patients with anti-Cs in their sera. In 1978, Shore and Steane2 demonstrated a normal in vivo survival of Cs(a+), 51Cr-labeled donor red cells in a patient who had anti-Cs. A similar finding was also obtained in another patient with anti-Cs as reported by Valko et al.3 However, Molthan et al.4 reported a case of intravascular clearance of Cs(a+) donor red cells in a patient who had anti-Cs.

The conflicting published reports led us to conduct a follow-up study of a patient who had anti-Cs.

Case Report
The patient was a 46-year-old Caucasian female with end-stage renal disease. Her past medical history included five miscarriages and multiple blood transfusions. On November 24, 1986, anti-Cs and anti-Fya were first identified in a posttransfusion blood sample. The direct antiglobulin test (DAT) was weakly positive with monospecific anti-IgG. However, no RBC antibody could be detected in an eluate prepared from her posttransfusion red cells.

Materials and Methods
Serum and eluate testing
The tests were performed using standard procedures.5 The patient's serum was tested with a commercial reagent red cell panel and Cs-negative red cells (stored in liquid nitrogen). Three Cs-negative red cells were Fy(a+) and three were Fy(a-). LISS (American Red Cross Production Laboratory, Rockville, MD) was used as an antibody enhancement medium, according to the manufacturer's instructions. All tests were read for agglutination and hemolysis after 37°C incubation, followed by the antiglobulin technique.

Red cell eluates of the patient's posttransfusion samples were prepared using the methylene chloride (dichloromethane) technique. The eluates were tested against a reagent red cell panel using the LISS antiglobulin technique.

Red cell phenotyping
The patient's red cell antigens were phenotyped using a standard tube test with known typing sera according to the manufacturers' instructions. Separation of the young red cell population was performed using a phthalate ester capillary technique.6

Hematology and chemistry profile
The patient's hemoglobin level, serum lactate dehydrogenase (LDH), and total bilirubin were tested at the referral hospital.

Identification of IgG subclass
IgG subclassing of the patient's anti-Cs was performed by the American Red Cross Special Services, Medical Operations, Holland Laboratory, Rockville, MD.6

Results
The patient's red cells were typed as group O; D+, C+, E-, c-, with a weakly positive DAT (IgG only). Since the patient had been transfused 37 days before this blood sample was drawn, a sample of the patient's 'young' red cells, separated by the phthalate ester capillary technique, were phenotyped as D+, C+, E-, c-, S+, s+, K-, Fy(a-b+); Jk(a+b+); and DAT-.
negative. Csa antigen status was inconclusive. Serum testing revealed the presence of anti-Fya and anti-Csa. The anti-Csa was reported as a mixture of IgG1 and IgG2 subclasses. No red cell antibody could be demonstrated in an eluate prepared from the DAT-positive posttransfusion red cells.

No compatible donor could be found in screening group O; D+; Fy(a-) units. Therefore, “least incompatible” group O; D+, E-, c-, Fy(a-) donor units were transfused.

Clinical and laboratory data compiled within 48 hours after each transfusion of 19 Cs(a+) units on 10 occasions, over eight months, are shown in Table 1. At no time was there evidence of a clinically adverse transfusion reaction. Posttransfusion hemoglobin increments appeared appropriate and the serum biochemical parameters (bilirubin and LDH) also revealed no evidence of clinically significant hemolysis.

**Conclusion**

Although the anti-Csa was a mixture of IgG1 and IgG2 immunoglobulins, there was no clinical, serological, or biochemical evidence of adverse reaction to the transfusion of 19 incompatible Cs(a+), Fy(a-) donor units transfused over an 8-month period. Therefore, this case study represents another example of a clinically benign anti-Csa.

**References**


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**Table 1. Clinical and serological data following transfusion of weakly incompatible, Cs(a+) units of blood**

<table>
<thead>
<tr>
<th>Transfusion dates</th>
<th>Number units transfused</th>
<th>HB (g/dL)</th>
<th>LDH (U/L)</th>
<th>Total bil. (mg/dL)</th>
<th>DAT IgG</th>
<th>Eluate</th>
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<td>post</td>
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<td>w+</td>
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<td>NT</td>
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<td>169</td>
<td>NT</td>
<td>w+</td>
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</table>

* All units transfused were weakly incompatible in the antiglobulin phase
† Normal range - 50 to 110 U/L
‡ Normal range - 0.2 to 1.3 mg/dL
§ Direct antiglobulin test
∥ Not tested