A NEW EXAMPLE OF THE ANTI-S AGGLUTININ

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According to the latest information of Race and Sanger (1950) seven examples of the antibody anti-S are known. In two cases this antibody was apparently caused by iso-immunization during pregnancy. The first example was described by Walsh and Montgomery (1947), and the other by Mary Pickles (1948). A naturally occurring antibody anti-S was found in the serum of a patient by Coombs *et al.* (1951) and in the blood of a donor by Vogel and Rosenfield (see Race and Sanger, 1950). In three cases the formation of anti-S was the result of multiple transfusions (Cutbush and Mollison, 1949; Race *et al.*, 1949; Collins *et al.*, 1950).

If the antibody was formed by immunization during pregnancy or blood transfusion one or more other immune antibodies, mostly anti-D, were present in the serum.

In our example the antibody was found in the serum of a woman suffering from a severe chronic anaemia, apparently due to osteosclerosis of the bone marrow. The anaemia was treated for the first time in 1933 after delivery of her first child. The administration of medicaments (iron, copper, liver extracts) was not successful. After delivery of the second child she received her first transfusion of 350 ml. of blood from her husband. This was followed by a rise in temperature and chills. The clinical effect of a second transfusion with 175 ml. of

blood, given one month afterwards, is unknown. The first and second pregnancies ended with delivery of a normal child. The third pregnancy, in 1940, also terminated with the birth of a healthy child.

In February, 1940, the patient was treated again for her anaemic condition (see Table). She received eight intramuscular injections of blood and seven transfusions. The effect of one of these transfusions is unknown. The third did not cause any ill effect, but all the others resulted in mild febrile or allergic reactions (Quincke's oedema or urticaria). The last transfusion was followed by a haemolytic reaction with rise of temperature (40° C.), chill's, dyspnoea, and haemoglobinuria. During that time difficulties were observed in determining the blood group owing to the presence of an irregular antibody in the patient's serum.

In 1950 the patient was readmitted to the hospital (Wageningen) and treated with blood transfusions. The haemoglobin level was 34%, red cell count 1,400,000, diameter of red cells 6.9 μ , white cells 2,400; the bilirubin content was slightly increased (method of Hijmans van den Bergh) 1.6 units. Sternal puncture failed as in previous trials. This was apparently due to the fact that the corticallis was enlarged, as was found in 1937 by x-ray examination of other bones (upper and lower extremities).

The first transfusion with group A blood, given in October, 1950, was stopped after infusion of 100 ml. because of severe abdominal pain and chills. Three days later a second transfusion of 250 ml. of group A blood was administered, which was followed, about one hour later, by chills.

As the red cells of different donors of group A agglutinated the serum of the patient, the blood was more thoroughly investigated in the Blood Grouping Laboratory in Amsterdam.

The patient's blood group was: A MMss cde/cde Fya+. In the patient's serum were found the following antibodies: anti-C in saline 1:512; anti-D in saline 1:512; anti-S in saline 1:16. The titres of these anti-

Record of Blood Transfusions and Injections

Date	Quantity of Blood ml.	Type of Blood	Method of Admin.	Reaction	Hb Level %	Antibodies	
						Anti-Rh	Anti-S
19/11/36	350	O CDe/cdE ss (husband)	I.V.	Chill	60	n.i.	n.i.
18/12/36	175	O Rh?	I.V.	?	60	n.i.	n.i.
1/2/40	20	?	I.M.		23		
3/2/40	20	?	I.M.		?		
5/2/40	20	?	I.M.		30		
7/2/40	20	?	I.M.	Febrile reactions (39-5° C.) and abdominal complaints	?		
9/2/40	20	?	I.M.		30		
11/2/40	20	?	I.M.		?		
13/2/40	20	?	I.M.		24		
15/2/40	20	?	I.M.		22		
17/2/40	100	A Rh?	I.V.	Mild febrile	21	n.i.	n.i.
28/2/40	450	A Rh?	I.V.	" "	25	n.i.	n.i.
8/3/40	450	A Rh?	I.V.	None	27	n.i.	n.i.
14/3/40	300	A Rh?	I.V.	Quincke's oedema	30	n.i.	n.i.
22/3/40	300	A Rh?	I.V.	Urticaria	40	n.i.	n.i.
1/4/40	?	?	I.V.	?	?	n.i.	n.i.
10/4/40		?	I.V.	Chill and fever, dyspnoea, haemoglobinuria	28	n.i.	n.i.
4/10/50	100	A Rh?	I.V.	Chill, malaise	35	n.i.	n.i.
9/10/50	250	A Rh?	I.V.	" "	35	Anti-D 1:512 Anti-C 1:512	1:16
26/10/50	500	A cde/cde ss	I.V.	None	30		
26/10/50	20	A cde/cde S	I.V.	(Febrile 39° C.)	40		
27/10/50	1,000	A cde/cde ss	I.V.	None	45		1:4
31/10/50	500	A cde/cde ss	I.V.	"	50		1:32
3/11/50	500	A cde/cde ss	I.V.	"	57		
4/11/50	1,000	A cde/cde ss	I.V.	"	68		
7/11/50	1,000	A cde/cde ss	I.V.	" General condition ameliorated			
7/11/50	10	A cde/cde S	I.V.	None	60		
9/11/50	1,000	A cde/cde ss	I.V.	"			1:64
12/11/50							
21/12/50							1:4

bodies were not higher with the albumin/serum method and the indirect anti-human-globulin test. After heating the serum for 10 minutes at 70° C. incomplete antibodies anti-C and anti-D were found, titre 1:256, but no incomplete antibodies of the type anti-S could be detected with the albumin/serum method, indirect anti-human-globulin, or trypsin technique.

In the period from October 26 to November 7, 1950, the patient received 4,500 ml. of A, Rh-negative, S-negative blood. No reaction occurred, and the haemoglobin level increased from 35 to 60%. The titre of the antibody anti-S had decreased from 1:16 to 1:4.

Apart from the transfusions of A, Rh-negative, S-negative blood, the patient received on October 26 20 ml. of A, Rh-negative, S-positive blood, which resulted in a febrile reaction (39° C.). Four days later the titre was 1:32. Twelve days after this first attempt of artificial immunization 10 ml. of Rh-negative S-positive blood was given again, this time without causing any reaction. Five days later the titre had increased to 1:64.

Reinvestigation of the serum on December 21 revealed a decrease in titre to 1:4.

Determination of the blood groups of husband and children revealed the following facts.

Husband		O CDe/cdE ss
1st child, born in 1933		O CDe/cde ss
2nd " " " 1936		O cdE/cde ss
3rd " " " 1944		O cdE/cde ss

From these findings it was concluded that the formation of anti-S was not due to immunization during pregnancy but to blood transfusions.

It is highly probable that the reaction following the first transfusion with blood of the husband was caused by Rh antibody formation during the first pregnancy, although this first child showed no symptoms of haemolytic disease. The birth of two normal children afterwards was apparently due to the lack of D (and C) antigen in their red cells.

Summary

A patient suffering from a severe chronic anaemia, probably due to osteosclerosis of the bone marrow, received in a period of 14 years 11 blood transfusions, most of them causing mild or severe reactions, and 8 intramuscular injections of whole blood.

In the serum was found, besides complete and incomplete antibodies anti-C and anti-D, a saline-agglutinating antibody of the type anti-S. This agglutinin is apparently of the immune type, because the titre, after administration of two intravenous injections of S-positive blood (respectively 20 and 10 ml.) increased from 1:4 to 1:64, and fell again after the artificial immunization was stopped. The first injection with S-positive blood was followed by a febrile reaction. Rh-negative S-negative blood was well tolerated.

No incomplete anti-S antibodies were detected.

This is the eighth case in which the anti-S agglutinin was found and the fourth case in which the formation was due to immunization by blood transfusion.

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Medical Memoranda

Enteroliths of Small Intestine

True enteroliths of the small intestine are rare. De Witt *et al.* (1943) described a case and mentioned seven others reported in the literature. Since that date Dearing *et al.* (1944) reported a case which was the only one in the records of the Mayo Clinic, while Sten Grettve (1947) has since reported another.

Case Report

A spinster aged 44 was admitted as an emergency case complaining of abdominal pain of about five hours' duration, which had begun in the region of the umbilicus and then became localized to the right iliac fossa. Vomiting had occurred after the onset of the pain and there had been a little diarrhoea. She stated that she had had a number of similar attacks over the past few years, but that they had become more frequent in the last 12 months.

Photograph of the enteroliths.

On admission her temperature and pulse were normal, and there was tenderness and slight guarding in the right iliac fossa.

The abdomen was explored through a right lower paramedian incision. The small intestine about 2 ft. (60 cm.) from the caecum was found to be dilated to the size of a normal stomach, whilst the wall was hypertrophied to a far greater thickness than that of a stomach. At each end of the dilatation there was a slight constriction of the gut, and this prevented three enteroliths enclosed in this section from being passed proximally or distally. The mesentery to this area of gut was heavily infiltrated with fat, as was the adjacent subserosa. The enteroliths were removed through the hypertrophied wall of the gut and the patient made an uneventful recovery. The largest stone was oval in shape with biconcave lateral surfaces, into which the two smaller ones fitted accurately, thus suggesting that they had originated by splitting off from the larger one.

On further questioning the patient stated that from the time she first started going to school at the age of 5 she could remember having similar attacks of abdominal pain, for which her mother used to put her to bed for a few days, following which she would be perfectly well for a year or so. These attacks had become more frequent in recent years.