KURZE MITTEILUNG

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**Immunoadherence and Post-Transfusion Reactions** *

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Summary

In blood samples taken during or right after transfusion reactions not caused by blood group incompatibility, it is possible to isolate in 5 out of 43 cases blood cells that were IA-reactive after treatment with guinea-pig complement and that were able to agglutinate human indicator O-erythrocytes. In contrast it is impossible to show IA-reactive complexes in plasma. This indicates that at least with part of the post-transfusion reactions IA is involved.

Zusammenfassung


Immunoadherence (IA) is defined by Nelson (1953) as the attachment of any antigen antibody complement (AG-AB-C')-complexes either to erythrocytes of primates or to thrombocytes of non-primates. The latter reaction is first described by Rieckenberg (1917) and also called 'Rieckenberg reaction'. (Bibliographic review see: Nelson (1963) and Nelson and Uhlenbruck (1967)).

The nature and biological meaning of IA is not yet completely clear. Nelson (1953, 1956) and Robineaux and Nelson (1955) have demonstrated that IA in vitro and in vivo increases the phagocytotic activity and Nelson (1963) has presumed that IA is involved in certain allergic reactions, e.g. the generalized Schwartzman reaction.

Since all components necessary for IA reaction are contained or can be formed in the blood it is reasonable to assume interrelations not only between immunohemopathies and IA but also between IA and blood transfusion.


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After transfusions especially multiple or mass transfusions it is not only possible but also probable that antibodies are formed against corpuscular or plasmatic components of the blood. Further application of the antibody stimulating antigens by transfusions may lead to subliminal and subclinical antigen antibody reactions. Complement contained in the recipient's own blood or given with the transfusion may promote the formation of IA reactive Ag-AB-C'-complexes able to react with the indicator red blood cells. In this respect it is possible that such mechanisms are responsible to a shortened life-span of the red blood cells as well as to certain unexplained transfusion reactions not caused by blood group incompatibility.

With respect to incompatible transfusions in dogs followed by anemia, leukopenia and thrombopenia Swisher and Young (1954) and Swisher (1956) proved these correlations by demonstration of IA-reactive complexes in the blood of the transfused dogs and by accelerated clearance of these complexes from the circulation. Brody and Finch (1961) showed that complexes of human erythrocytes, blood group specific antibodies and complement are IA-reactive and that guinea-pig platelets can be attached. We found in recent investigations (Sachs (1967), Sachs and Friedburg (1967)) that such group specific complexes as well as complexes of white blood cells, leucocyte antibodies and complement in vitro are IA-reactive not only with non primate platelets but also with human erythrocytes.

In order to test whether IA-reactive complexes occur in vivo we investigated blood of patients with unclarified transfusion reactions.

The blood of 43 cases of post-transfusion reaction was investigated. The patients had received at least 5 and at most 42 transfusions. In no case the reactions were caused by irregular or regular group specific antibodies. The samples were taken with ACD solution during or right after the reaction occured. They were examined as follows:

1. In order to recognize plasmatic antigen antibody complexes to 0.5 ml of the separated plasma of each sample 0.1 ml of guinea-pig serum as a source of complement (C'Hs0 °~ 160 Hem U/ml) was added and the reaction tube was incubated for 60 min. at 37°C. Then twofold dilutions with buffered (barbiturate buffer pH 7.3) saline containing Ca and Mg (BCMS) were performed.

2. The cell sediment of each sample was washed thrice with saline (BCMS) and resuspended to a concentration of about 10⁹ cells/ml. To 1 ml of the suspension 0.2 ml of guinea-pig serum was added. The mixture was incubated for 30 min. at 0°C and rewashed thrice with saline (BCMS). Now the sediment was resuspended in EDTA-saline (10 mM EDTA in BCMS) and incubated once more for 2 h at 37°C. After repeated sufficient washing with buffered saline (at least three times) the last sediment was completely hemolysed with 0.8 ml of distilled water and adjusted to physiological concentration with concentrated saline. Finally twofold dilutions were prepared.

The addition of guinea-pig complement to the plasmas and to the cell sediments was necessary because otherwise no reaction took place.

3. To 1 ml of every dilution step of the plasmas and of the hemolysed cell sediments 0.1 ml of a suspension of human O-erythrocytes (10⁸ cells/ml in saline) were added. After shaking the reaction tubes were incubated for 1 h at 37°C and inspected macroscopically and microscopically.