IMMUNOLOGICAL ASPECTS OF HERPESVIRUS VARICELLA

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Summary

Sera of 20 patients suffering from varicella and zoster were examined immunoelectrophoretically. IgM was found to be slightly decreased in sera of varicella patients examined on the first day of appearance of the cutaneous eruptions. Sera of both zoster and varicella patients examined after appearance of skin lesions showed marked reduction in IgM. All the cases revealed normal immunoelectrophoretic picture after the disappearance of the eruption. The role played by IgM. in viral immunity is discussed.

Although varicella and zoster differ in their clinical manifestations yet the accumulated laboratory evidences now makes it almost certain that they are different responses to infection with the same virus (1). Zoster is considered to be the clinical response to a second infection or reactivation of the varicella virus in partly immune individuals (2).

Viruses being potent antigens are capable of producing various types of antibodies. The role played by these antibodies in viral immunity is not well understood.

The aim of this work is to study the changes in immunoglobulins in sera of patients suffering from varicella and zoster by immunoelectrophoretic technique in an attempt to throw some light on the possible role played by these immunoglobulins in viral immunity.

Material and Methods

In this study the sera of three groups of patients suffering from varicella and zoster were examined immunoelectrophoretically. The first group comprised 10 cases of varicella examined on the first day of appearance of the cutaneous eruption and re-examined on the third day. Their average age was 7 years. The second group included 10 cases of zoster. Their average age was 21 years. Their sera were examined within a period varying from 2 to 4 days from the appearance of the eruptions. The third group comprised cases of group I and II re-examined when the cutaneous lesions completely subsided.This was obtained in a period varying from 7 to 15 days from the appearance of the skin lesions.

Blood sera were collected from all the patients and examined immunoelectrophoretically according to the micro method devised by Scheidegger

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(3) and the double migration of Blanc (4). The antihuman serum (Polyvalent) Vomkaninchen Biotest Serum Institute was used in this study. Sera of normal individuals of the same age groups were used as control in every case.

Results

The immunoelectrophoretic analysis of sera of patients of group I showed slight decrease of IgM (Fig. I). On the third day the sera of these patients showed marked diminution of IgM. (Fig. II) Sera of group II showed also marked decrease of IgM. (Figs. III and IV). Sera of group III showed normal immunoelectrophoretic picture (Fig. V) Results are shown in the following table.

Discussion

Viral immunity passes through two different phases, namely viraemic and intracellular. During the viraemic phase the role played by antibodies against viruses is one of neutralization, while in the intracellular stage where the viruses are protected by the cell membrane the antibodies appear to be ineffective in prevention of infection and the recovery depends on non-immunological factors as the febrile response, local acidity (5) low oxygen tension produced by the inflammatory response (6 and 7) and the production of interferon (8).

The marked decrease of IgM obsreved in sera of patients of varicella and

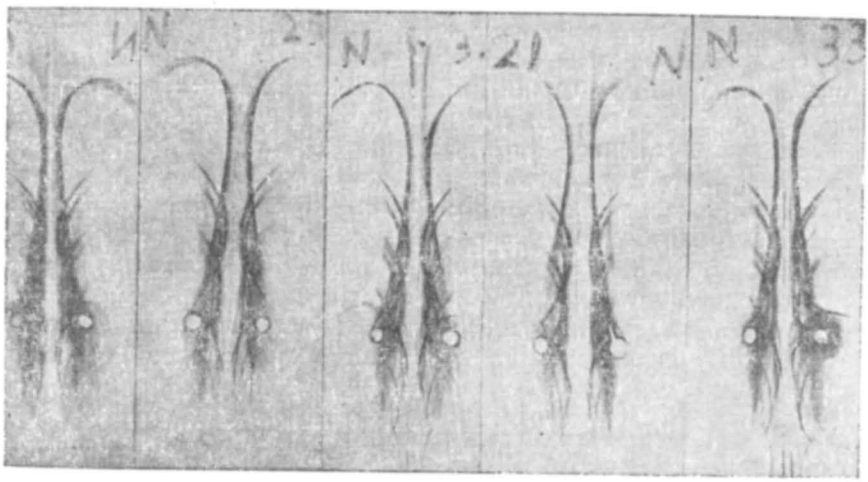

Fig. I Fig. 2 Fig. 3 Fig. 4 Fig. 5

Table shows number of cases, day of analysis and I. E. P. findings

Cases examined	Disease	Day of analysis	I. E. P.
10	Varicella	1st day	Slight decrease of Ig. M
Gr. I			
10	Varicella	3rd day	Marked decrease of Ig. M
Gr. II			
10	Zoster	2-7th day	Marked decrease of Ig. M
Gr. III			
10	Varicella	7th day	Normal
10	Zoster	15th day	Normal

Zoster can be explained on the following two assumptions. It is well known that IgM (19S) is the earliest antibody produced when an animal is immunized with any standard antigen (9). It has been shown that Ig.M has a structure basically similar to that of other immunoglobulins but instead of having two H and two L chains, each molecule has ten H and probably ten L chains (9). In any case it is clear that each molecule of Ig.M is multivalent capable to combine firmly by means of at least five combining groups to any antigen which has a repeating pattern of antigenic determinants close together on its surface. An example of these antigens are viruses whose outside consists of a close packed arrangement of capsids. By analogy with Ig. G it is expected that while each pair of H & L chains of Ig. G comprises one combining site, in case of Ig. M each pair would have 10 identical sites. The decrease of Ig.M in our patients might be due to the great affinity of Ig.M in combining with viruses compared with other immunoglobulins From its intravascular position (10), it is supposed that Ig.M is considered a powerful weapon in the prevention of infection during the viraemic phase and enhancing the uptake of micro-organisms by phagocytosis. It is thus clear that the recognition of the virus being a foreign material and its removal by phagocytosis is primarily the basic function of Ig. M fraction in particular.

The second suggestion for the diminution of Ig.M in sera of patients suffering from varicella and zoster can be explained on the fact that in infection with viruses there is a great accumulation of foreign antigens in the body. These antigens will stimulate the corresponding immunocytes to proliferate producing mature plasma cells. These cells are endothelial cells capable of producing A, M and G.

Although most M antibody is produced by plasma cells there is a direct evidence that some plasma cells at a certain stage of immunization are producing both G and M while at a later stage the great majority of plasma cells produce only G. The implication is that a proportion of cells initially producing M antibody subsequently switch to the production of G. This is proved experimentally by exposing animals to preformed G antigen of the some specifity resulting in reduction of M antibody producing cells (10).

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