The Role of Immunological Response in the Development of Experimental Amyloidosis

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The pathogenetic mechanism of the murine experimental amyloidosis induced by repeated treatment with heterologous immune-complex (human gammaglobulin-anti-human gammaglobulin complex) was studied, and following results were obtained.

1) The mice sensitized with immune-complex had high levels of serum gammaglobulin as compared to non-sensitized mice.

2) Almost complete immunological tolerance to immune-complex was produced in the mice with immunosuppressive drug, 6-mercaptopurine, and incomplete tolerance with prednisolone or cyclophosphamide.

3) The incidence of amyloidosis was as follows; the mice treated with only immune-complex 1/12, immune-complex and prednisolone 2/13, immune-complex and 6-mercaptopurine 7/8, and immune-complex and cyclophosphamide 2/5. The mice which received only phosphate buffered saline or single immunosuppressive drug never developed amyloidosis.

4) The mice which developed amyloidosis had no antibody activities to the immune-complex.

5) There was no difference in severity of plasma cells responses in lung, lymphnode, and spleen between amyloidosis induced mice and non-induced ones.

6) Th perivascular, peribronchial, and finally interstitial massive plasma cells infiltration in the lung were characteristic in the mice repeatedly treated with immune-complex, whether amyloidosis developed or not.

These results suggest that amyloidosis develops under the condition of acquired humoral specific immunological tolerance to the amyloidogenic agent, and that plasma cells play, at least in part, a crucial role in the development of amyloidosis.