SUMMARY

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The immune system is an extremely complicated one with a variety of roles in maintaining homeostasis and health. This function arises through the action of a number of subpopulation of the T and B cells and macrophages (Katz, 1982).

There are several defence mechanisms against infection. These include humoral mechanism which is concerned in production of immunoglobulins which combine with various antigens. The cell-mediated mechanism which result in production of lymphokines-from sensitized lymphocyte - which have different types of actions (Weir, 1983). The complement system passes through series of steps resulting in many factors which are important for opsonisation as well as lysis of the invading cells.

Rheumatic fever is multisystem disease of obscure aetiology. It occurs secondary to pharyngeal infection with group A beta-heamolytic streptococci. This disease manifests itself with polyarthritis, chorea erythema marginatum, subcutaneous nodules and carditis, these may occur singly or in combination (Markowitz et al., 1965). The worst sequelae of this disease is rheumatic heart

diseases which cause heart valves deformities.

Several studies showed that immunological process plays important role in the pathogenesis of rheumatic fever. It was found that certain types of immunoglobulins were elevated in the serum of rheumatic patients, especially in acute attack as compared to chronic state (Fahey, 1965). Also, many studies have suggested the presence of heart-reacting antibodies which react with the sarcolemmal and interfibrillar regions of mammalian heart tissue, these antibodies result from antigenic stimulation of streptococcal antigens (Zabriskie et al., 1970).

It has been suggested that complement components are decreased within the synovial fluid of the joints in patients who have polyarthritis, this may raises the possibility of the presence of complemental involvement in the developing rheumatic arthritis (Svartman et al., 1975).

The rheumatic patients also have high level of circulating immune complexes in their serum. This is due to presence of various antibodies against multiple cellular and extracellular streptococcal antigen (Yang et al., 1977).

The rheumatic patients exhibit exaggerated cellular reactivity to streptococcal membrane antigens. This hieghtened reactivity of lymphocyte is of particular interest, as this antigen cross-react with sarcolemma of mammalian heart tissue and walls of blood vessels.

Many studies showed that cell-mediated immune response to cardiac antigen, present only in cases who have rheumatic carditis. This suggests that it is heart specific and produced only when the heart is involved (Agarwal et al., 1980).

These previous data suggest the role of immune system in the pathogenesis of rheumatic fever, but the precise nature of this process is still not fully clear.