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SERUM CHOLESTEROL AND TRIGLYCERIDE LEVELS IN CHILDREN WITH RHEUMATIC HEART DISEASE WITH AND WITHOUT CONGESTIVE HEART FAILURE BEFORE AND AFTER TREATMENT

THESIS

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بسم الله الرحمن الرحيم

قالوا سبحانك

لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم

« البقرة ٣٢ »

صدق الله العظيم

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Introduction

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Rheumatic Fever

Acute rheumatic fever (ARF) is a non suppurative complication of group A streptococcal upper respiratory infection, and it is a systemic disease involving more frequently the joints and the heart and less frequently the central nervous system, skin and subcutaneous tissues *(Markowitz, 1987)* [1].

Epidemiology:

The decreasing incidence of Rheumatic fever and Rheumatic heart disease (RHD) in the so-called western world (U.S, Western Europe and Japan) has become an axiom which meets the consensus of the pediatrician, the cardiologist and the epidemiologist . The number of hospitalized cases of rheumatic fever is declining, the disease is becoming milder, and the identification of non rheumatic heart diseases mimicking rheumatic heart disease has become more frequent *(Disciascio & Taranta, 1980)* [2] . .

Rheumatic fever sill forms a major community health Problem in Egypt *(Eissa, et al. 1988)* [3] . This is because severe cases with heart failure & multiple valvular lesions are frequent especially in the Delta region *(Eissa,et al. 1985)* [4]

The highest reported mortality rate in the world for RHD [27.5/100.000] and one of the highest for acute rheumatic

fever [1.0/100.000] are in Egypt, whose capital lodges ¼ of the total country population in conditions of extreme crowding and where rheumatic fever is frequent *(Shiokawa & Yamada, 1977)* [5].

In the Egyptian countryside, rheumatic fever is expected to be comparatively rare, despite the prevailing poverty . But this is not true as rheumatic fever comes next to bilharziasis as important commonly health problem affecting children of the school age allover our Egypt*(Shoheib, 1988)*[6].

Rheumatic fever occurs most commonly, like Streptococcal sore throat or tonsillitis, in school age children between the age of 5 and 15 years . Rheumatic fever is uncommon in those less than 5 years of age and one study it was 0.1/1000 *(Rosenthal,et al. 1968) (Shoheib, 1995)* [7,8].

Etiology & Pathogenesis:

The etiology and pathogenesis of rheumatic fever are still controversial , however, the role played by infection with group A, β heamolytic streptococci is well established *(Shulmen, et al.1974)* [9].

Initial pharyngeal streptococcal infection is usually followed two to three weeks later by the first clinical sign of rheumatic fever. The interval between pharyngeal infection and the onset of rheumatic activity appears to be critical for the development of entire pathological process . However, The precise relationship between initiating group

of pharyngeal streptococcal infection and subsequent acute rheumatic fever has never been conclusively defined *(Ginsburg, 1985)* [10].

The etiologic agent or organism "Group A, β hemolytic streptococcus)

Streptococcus pyogenes is a gram + ve coccus . Going from the out side in, we may or may not find a capsule of hyaluronic acid [more frequent in epidemic strains] *(Stollernman, et al. 1965)* [11].

The streptococcal cell wall consists of three layers: (a) the outer layer formed by protein antigens, (b) the middle layer containing group-specific carbohydrates, and (c) the inner or mucopeptide layer which is a rigid peptidoglycan skeleton *(Kashani, 1981)* [12].

The outer layer formed by the protein antigens which are three types (M, T and R proteins). The M protein is used to identify streptococcal type as there are about 80 M types of group A, β hemolytic streptococci each leading to formation of type-specific antibody *(Fyler, 1992)* [13].

The middle layer of the streptococcal cell wall of β hemolytic streptococci contains group specific polysaccharide, upon which they are divided into

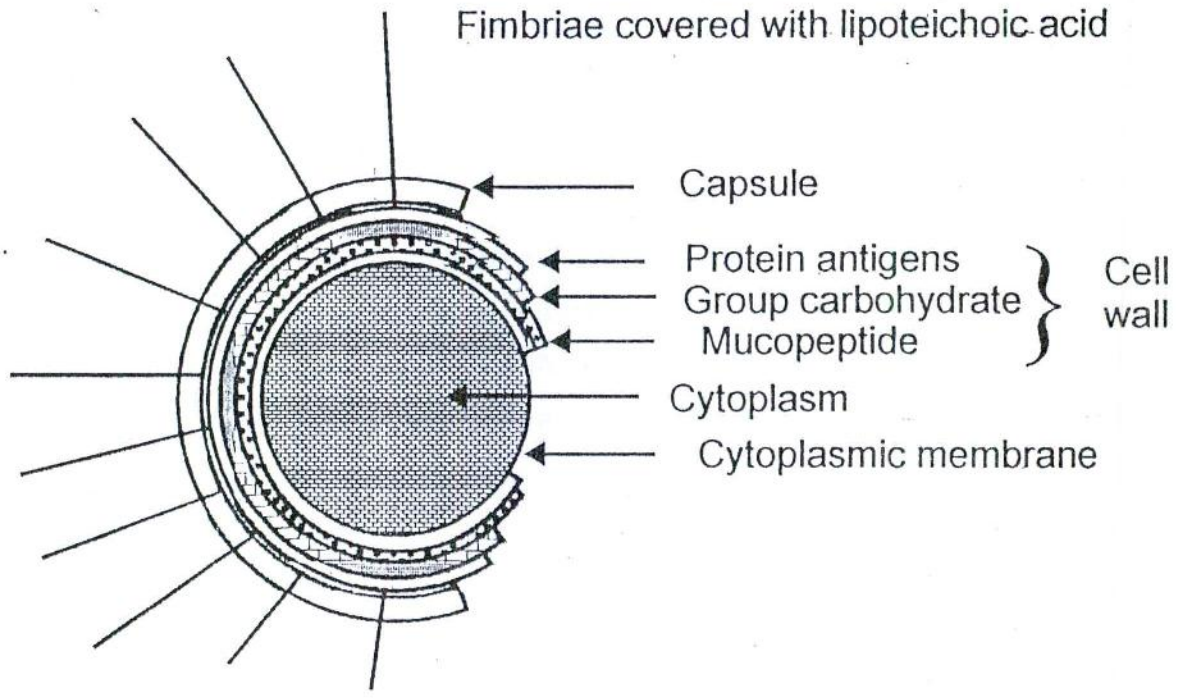

Fig. (1) : Antigenic structure of group A

2-Autoimmunity theory :

The basis for this concept is that several streptococcal antigens cross-react immunologically with human tissue antigen *(Tagg, et al. 1972)* [22] .

As a result of this antigenic mimicry the immune response to streptococci may mistake the host's antigen as foreign . This error may be the mechanism causing damage in RF . Recently developed techniques have allowed identification of actual amino acid sequences in group A-streptococcal M-proteins that are identical to and cross react with specific human tissue and M-protein remains a prime candidate *(Dale, et al. 1985)* [23] .

The group A carbohydrate component is still another candidate since it has been reported that this antigen cross react with a glycol protein in human heart valves*(Goldstein, et al. 1967)* [24] .

3-Circulating immune-complex (C.I.C) Hypothesis:

Another hypothesis is thc circulating immune complex by which streptococcal toxins secreted induce the formation of antibodies in the reticuloendothelial system . The streptococcal toxin reacts with already formed antibodies and these combine with the serum complement to form immune complex that circulated in the blood, when the concentration of these complexes reach certain threshold it reacts with the tissues of the susceptible child *(Shaat, 1991)* [25] .