STUDY THE PREVALENCE OF RHEUMATIC HEART DISEASE IN SOME GOVERNMENTAL PRIMARY SCHOOLS

THESIS SUBMITTED FOR PARTIAL FULFILLMENT OF MASTER DEGREE IN CARDIOLOGY

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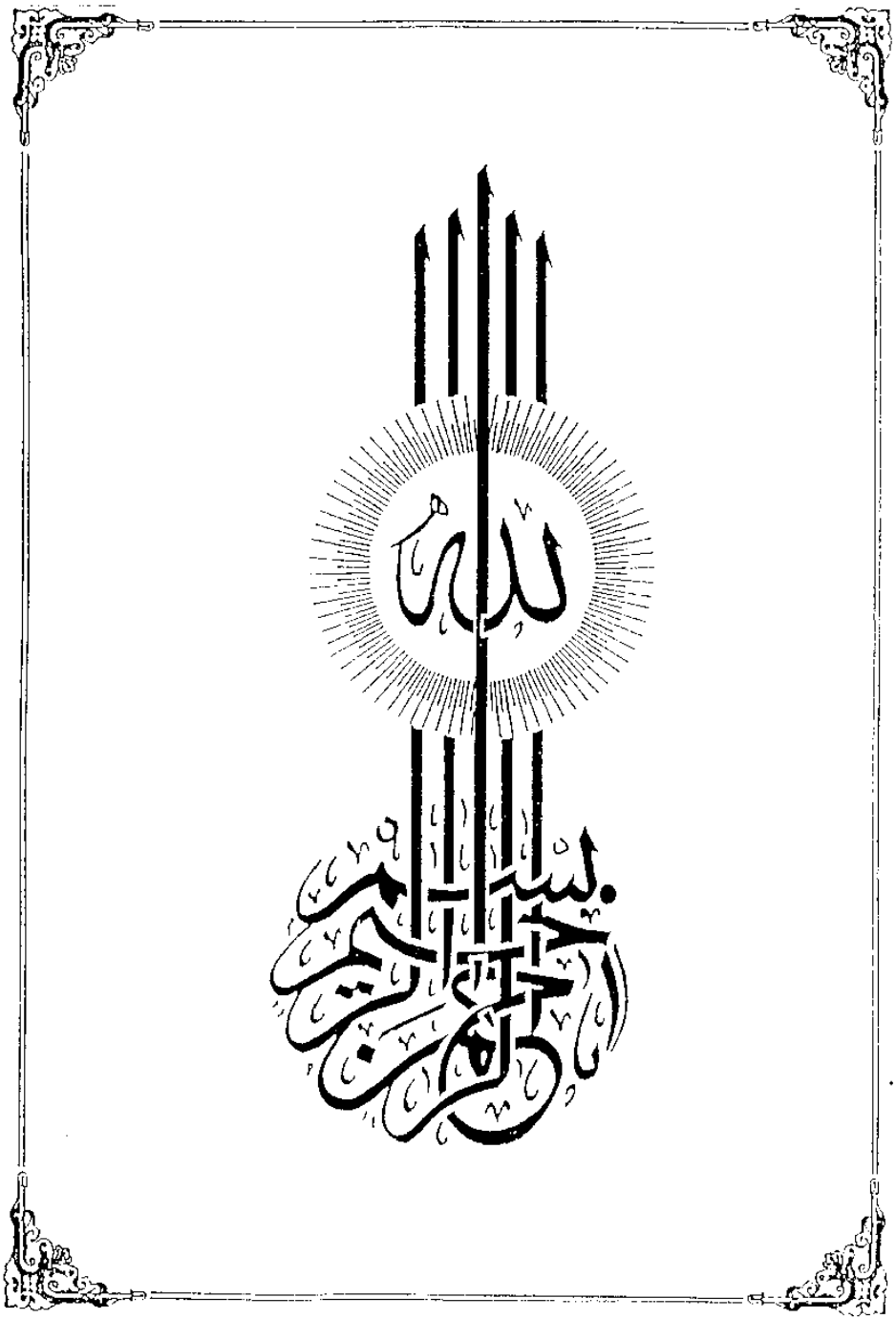
TO MY PARENTS



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INTRODUCTION

INTRODUCTION :

Rheumatic fever is the chief cause of serious valvular heart disease. Acute rheumatic fever and subsequent rheumatic heart disease remain one of the important cardiovascular problems in the developing countries.

Rheumatic heart disease still constitutes the leading cause of death from heart disease in the 5 - 24 years old age group in these countries.

Although preventable, this disease occurs because of poor economic conditions, substandard housing, over crowding, the deceptive self limited nature of streptococcal pharyngitis and its improper treatment. It occurs most frequently between 5 and 15 years of age.

The incidence of rheumatic fever and associated mortality rate have been decreasing for several years in countries where housing and economic conditions have been steadily improving. The rate of decline has also probably been accelerated by the wide use of antimicrobial therapy.

AIM OF THE WORK :

To study the prevalence of rheumatic heart disease in some governmental primary schools.

REVIEW OF LITERATURE

BRIEF HISTORY OF RHEUMATIC FEVER

Guillaume de Baillou in france (1538 – 1616),also Known as "Ballonius" first clearly distinguished acute arthritis from gout, he described it under the name of " acute articular rheumatism"[5].

Thomas Sydenham in England (1624 – 1689) described chorea, but failed to associate this entity with other manifestations of acute rheumatic fever.

Raymond Vieussens (1641 – 1715) published pathologic descriptions of mitral stenosis and aortic insufficiency[5].

Deformities of the heart valves, described by Morgagni in Italy in 1761, were noted to crop up frequently in the autopsies of patients with histories of acute articular rheumatism[77].

It remained ,however, for William Charles Wells in 1812 to emphasize the association of rheumatism and carditis and to provide the first clear description of subcutaneous nodules, but the clinical description of rheumatic heart disease had to await the invention of the stethoscope by Laennec in 1819[5].

Jean Baptiste in 1836 and Walter B. Cheadle in 1889 published extensive studies of rheumatic arthritis and carditis that have come to be regarded as classic works in this field. Cheadle had described the full rheumatic fever syndrome: carditis, polyarthritis, and chorea, as well as subcutaneous nodules and erythema marginatum. Thus,

rheumatic fever, as we know it today, resulted from the fitting together of entities originally thought to be unconnected[77].

J.K. Fowler pointed out the association of sore throat and rheumatic fever in 1880[5].

By the beginning of this century Aschoff had described the specific myocardial lesion that bears his name and the connection between a history of a previous sore throat and rheumatic fever was strongly suspected.

The introduction of Rebecca Lancefield's grouping system for beta hemolytic streptococci allowed clarification of the epidemiology of the disease by a number of investigators, Collis in England and Coburn in the United States 1931.Subsequent immunological studies more firmly established the relation between group A streptococcal pharyngitis and rheumatic fever. However, the most convincing evidence that streptococcal infections actually cause rheumatic fever came from Coburn and Moore, who in 1939 showed that recurrences of rheumatic fever could be prevented by continual anti-streptococcal medication, and a decade later from Massell and from Wannamaker et al.who demonstrated that adequate treatment of streptococcal pharyngitis with penicillin could prevent first attacks of rheumatic fever[77].

Thus, rheumatic fever emerged as a clinical syndrome with a known single etiology group A beta-hemolytic streptococcal infections of the throat.

Finally, the widespread introduction of antibiotic agents after world war II resulted in the development of strategies for primary and secondary prevention of rheumatic fever[5].

EPIDEMIOLOGY OF RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE

DEFINITION :

Rheumatic fever is an inflammatory disease which occur as a delayed sequel to pharyngeal infection with group A streptococci. It involves principally the heart, joints, central nervous system, skin, and subcutaneous tissues[70].

Although rheumatic fever may occur at any age, it is extremely rare in infancy, it appears most commonly between the ages of 5 and 15 years, when streptococcal infection is most frequent and intense. Similarly the geographic distribution, incidence, and severity of rheumatic fever are, in general, a reflection of the frequency and severity of streptococcal pharyngitis.

FACTORS DETERMINING THE ATTACK RATE OF RHEUMATIC FEVER:

One factor is the severity of antecedent streptococcal infection . In patients with frank, exudative streptococcal pharyngitis caused by certain pharyngeal strains of virulent group A streptococci, rheumatic fever followed at a fairly predictable rate(approximately 3 percent) regardless of the age, race or ethenic group studied and regardless of the year or season in which the study was made[69].

When streptococcal pharyngitis is sporadic and mild or due to strains of lesser rheumatic potential, the attack rate of rheumatic fever may be very much lower[67].

In patients who had previous attacks of rheumatic fever, the attack rate is increased to as high as 5 to 50 percent[74].

The magnitude of the immune response to the antecedent streptococcal infection and the duration of convalescent carriage of organism also affect the attack rate[57]. Weak ASO response is associated with acute rheumatic fever attack rates considerably less than one per cent whereas strong responses are associated with rates well in excess of 5 per cent

If the infecting organism in the pharynx is not eradicated during convalescence, treatment of streptococcal pharyngitis failed to reduce the attack rate of rheumatic fever.

Variations in the "rheumatogenicity" of group A streptococcal strains have a great effect. Some serotypes of group A streptococci that have rheumatogenic Potential(including M types 1, 3, 5, 6, 14, 18, 19 24, 27, and 29) appear to have been more frequently (but not exclusively) associated with cases of rheumatic fever[72].

Like streptococcal sore throat, acute rheumatic fever occur most commonly in the young school age child and very rarely in early infancy. It is estimated that 40 percent of streptococcal infections in pediatric populations occur

in children 2 to 6 years of age , suggesting that repeated streptococcal infections and sensitization of the host are prerequisite to the development of rheumatic fever[5].

No true difference in sex, race, or ethenic group susceptibility have been established . Crowded living conditions account for whatever apparent increased susceptibility has been reported[69].

The relationship of rheumatic fever to the intensity and severity of streptococcal disease is the same in tropics as in the temperate climates. Also the frequency of the clinical manifestations of rheumatic fever are the same in the tropics as in the United States[63].

Genetic influences affecting rheumatic fever susceptibility have been searched for extensively. One of the most fascinating is the recently discovered marker reported to be present on B-lymphocytes of a majority of individuals with rheumatic fever.[35]

THE CHANGING PATTERN OF RHEUMATIC FEVER

The current confusion concerning the epidemiology of rheumatic fever stems from the dramatic decline in incidence and prevalence of the disease despite the fact that group A streptococcal pharyngitis still appears to be common among populations in which rheumatic fever has become rare[6].

Reasons for this decline are uncertain, but are