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THE ALARMING DANGER OF ACUTE RHEUMATIC FEVER CHILDREN

Bhuvnesh Chhabra
Samarkand State Medical University,
6th Year Student at Medical Faculty
E-mail: bhuvneshnishu@gmail.com

Simran Kumari
Scientific Supervisor, Assistant of the Pediatrics Department
Samarkand State Medical University
E-mail: Beauty.num3@gmail.com

Abstract
In the modern era of fast-moving world, lies the under rated plethora of so many infections and disorders which might seem so common yet with numerous complications and poorer outcomes. One such common disease which is prevailing in children is the acute rheumatic fever. With the word rheumatic, it is alarming itself because Rheumatology as such is a new Science which has a lot of research going on and so many areas which are touched upon less. The major setback lies upon the prognostic factors of the disease as such.

INTRODUCTION

Acute rheumatic fever is a multisystem disorder which is immune mediated. Post 10 days- 5 weeks of untreated streptococcal infection (pharyngitis). It is a non suppurative inflammation of joints, nervous system, skin, subcutaneous tissue and the heart. Following are the rheumatic strains of untreated strep infection: M1, M3, M5, M6, M4, M18, M19, etc. It is seen in genetically predisposed patients. Peak incidence of the disease is 5-15 years, as sore throat is very common in this age period. 2-3% of the streptococcal pharyngitis leads to acute RF. In terms of immunological aspects it is a Type-2 Hypersensitivity reaction.

Molecular mimicry of Streptococcal pyogenes with the Human tissue:

Structural component of S. pyogenes	**Human tissue with which it cross reacts**
Hyaluronic acid	Synovial fluid
Cell wall M protein	Myocardium
Group a carbohydrate	Cardiac valves
Cytoplasmic membrane antigens	Vascular intima
Peptidoglycan	Skin antigens

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Pathogenesis:

1. Molecular mimicry : Most accepted theory and predominant cause of immunological damage.
2. Immune complex formation (Granulomatous reaction around the sites of immune complexes.
3. Tissue injury mediated by toxic effects of hemolysin/ endotoxin (streptolysin O and streptolysin S).
4. Alteration of self-antigens (M protein binds to a region in collagen type 4 which modifies self-antigen leading to antibody formation especially in the sub endothelial matrix of endothelium and perivascular connective tissue).

Manifestations of rheumatic fever:

A) JOINTS: It is the earliest and most common manifestation. Fibrinous exudates are deposited over synovial membrane with a lot of serious effusions in the joints. There is no joint destruction. Children complain of arthralgia. Multiple joints can be involved at the same time. No residual damage/ deformity. Large joints are mostly affected- Knees, ankles, elbows, wrist. It usually lasts for 4-5 weeks. More intense the arthralgia, lesser the carditis.

B) NERVOUS SYTSEM: Affected in 3-15% of the patients. Due to the formation of autoantibodies against gangliosides there is inflammation in the basal ganglia. Characteristic Saint Vitus dance/ Syndenham Chorea (involuntary jerks + uncontrollable movements of face and extremities + muscle weakness + emotional liability)

C) SKIN: Observed in 2-3% of the patients. Erythema Marginatum (Non pruritic, non-painful, erythematous eruption/ rash seen mainly on trunk and proximal limbs. It starts as a maculopapular rash which expands with a central clearing. It resembles smoke rings. It appears and disappears intermittently for weeks to months. It has spontaneous resolution.

D) SUBCUTANEOUS TISSUE: Formation of painless subcutaneous nodules over the extensors, bony prominences and tendons is seen in 2-3% patients. It lasts for few weeks. Also has spontaneous resolution.

E) HEART: Manifestation seen in 40-50% patients. 2-3 weeks after joint manifestations. Severity of carditis decides the prognosis. Pericarditis (strands of fibrinous exudates between parietal and visceral layers of pericardium leading bread and butter appearance of pericardium. Sharp localized pain in substernal area. On auscultation: scratchy sound of pericardial rub is heard. On Echocardiography: Effusion + cardiac enlargement is seen). Myocarditis (Inflammatory perivascular granulomas in myocardium are observed. Biopsy shows Aschoff bodies which are swollen collagen fibers with infiltrating lymphocytes, plasma cells, neutrophils and histiocytes. This leads to reduced contractility leading to reduced pumping and thus manifesting Congestive heart failure). Endocarditis/valvulitis (It is the most common manifestation of heart in ARF. Leads to long term disability: Chronic rheumatic heart disease. It can lead to thickening of valves and shortening of chordae tendinae + valve commissure fusion and ultimately leading to stenosis or regurgitation).

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PURPOSE OF THE STUDY:

The following study was performed to analyze the hemodynamic features of ARF in patients with predisposed environmental factors and notice the prevalence of ARF in children and analyzing the prognosis.

METHODS AND MATERIALS:

The study involved young children with an average age of 5-15 years. A total of 100 children (60 males and 40 female children) suffering from suspected ARF and pharyngitis of different degrees were examined. The examination was carried out by questionnaire. Subjective and objective investigation of patients followed by Echocardiography was performed. Patients firstly underwent normal examination including, Height, Weight, Chest and BMI (Body Mass Index) to the formula.

$$BMI = \frac{\text{Weight (Kg)}}{\text{Height } (m^2)}$$

It is the ratio of body weight (in kilograms) to height (in meters) squared. Patients and their guardians were asked about their history and they were carefully examined with auscultation and biopsy of the skin lesions and echocardiography to know the revolving risk of any future complications.

RESULTS:

As per our study we observed that out of 100 children with ARF, around 50 patients had heart manifestations out of which, 30 showed the presence of valvulitis (MR>MS>AR>AS), 15 had myocarditis and 3 had pericarditis and patients were very serious with pancarditis manifestation. 75 out of 100 patients presented with joint manifestations majorly affecting knee and ankle joints which was migrating arthritis. 18 patients manifested as syndenham chorea while only 4 patients showed the presence of erythema marginatum. With least incidence of Subcutaneous nodules, i.e., only 2 patients we concluded our study and wrote down the famous JONES criteria.

Diagnostic Crietria:-

A) Major criteria (JONES)
- J- Joint involvement
- O- Carditis
- N- Nodules (subcutaneous)
- E- Erythema marginatum
- S – Syndenham Chorea

B) Minor criteria (JONES CAFÉ PAL)

C- CRP ↑
A- Arthralgia
F- Fever
E- Elevated ESR
P- Prolonged PR interval

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A- Anamnesis of rheumatism
L- Leukocytosis

Diagnosis:
Throat culture - negative often.
Elevated ASO titre.
2 major criteria or 1 major + 2 minor criteria.
Revised Jones Criteria:
Low risk population- Incidence of RF in school aged chidren <2 per one hundred thousands per year.
Moderate or high risk population- all others.
Treatment:

- Symptomatic treatment- Aspirin and steroids given till ESR normalizes.
- Heart failure- Reduced fluid intake, Digitalis, Diuretics, O_2 supplementation
- Antibiotics- Single dose off benzathine penicillin (IM) 1.2 million units based on weight, if allergic to penicillin, erythromycin.

CONCLUSION:

For the prophylaxis: Rheumatic patients are at high risk of developing recurrent ARF after streptococcal pharyngitis. Require continuous prophylaxis:
Benzathine penicillin at 3 weekly intervals deep IM; 1.2MIU/ 6LU.
Regular screening of the children with recurrent sore throat should be performed in order to avoid serious complication of the acute rheumatic fever.

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