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100+ CLINICAL CASES IN PEDIATRICS

R Arvind

6th Edition

JAYPEE

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CASE

Acute Rheumatic Fever

PRESENTING COMPLAINTS

A 7-year-old girl was brought with the complaint of:

- Sore throat since 15 days
- Pain in the joint since 10 days
- Palpitation since 7 days
- Fever since 5 days

History of Presenting Complaints

A 7-year-old girl came with history of pain in left knee joint. Her mother noted that there was swelling of the joint. The swelling was very painful and the child was not allowing to move her leg. She also complained of similar type of pain in the left shoulder. She also told that the pain in the left shoulder joint has disappeared by the time she noticed pain in left knee joint. There was also associated history of fever. Fever used to be of moderate to high degree. There was no history of chills and rigors. Fever used to be more in the evening and night. She also complained of abnormal awareness of the heartbeat. She used to feel much tired even for the routine work.

CASE AT A GLANCE

Basic Findings

Height	:	117 cm (50th centile)
Weight	:	19 kg (50th centile)
Temperature	:	38°C
Pulse rate	:	116 beats/min
Respiratory rate	:	24 breaths/min
Blood pressure	:	90/60 mm Hg

Positive Findings

History:
- Fleeting type of joint pain
- Fever
- Sore throat
- Palpitation

Examination:
- Febrile
- Arthritis
- Diastolic murmur
- Loud first heart sound

Investigation:
- *Hemoglobin:* 9 g/dL
- *ESR:* Raised
- *Chest X-ray:* Cardiomegaly
- *ASLO:* Raised
- *CRP:* Raised
- *ECG:* Prolonged PR interval

(ASLO: antistreptolysin O; CRP: C-reactive protein; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate)

Past History of the Patient

She was the only sibling of nonconsanguineous marriage. She was born at full term by normal vaginal delivery. Her birth weight was 3 kg. She cried immediately after the delivery. There was no significant postnatal event. She was exclusively on the breastfeeds for the first 3 months. Later weaning was started as per the advice of the family doctor and the

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child was on family food by 18 months. Her developmental milestones were normal. She had been completely immunized. Her performance at the school was good.

Her mother complained that her daughter used to develop repeated attacks of throat pain, for which she was seeking medical treatment. She had pain in the joints occasionally.

EXAMINATION

On examination, the girl was moderately built and moderately nourished. She was in agony with pain in the joint. Her anthropometric measurement included her height 117 cm (50th centile) and the weight 19 kg (50th centile).

She was febrile, i.e., 38°C, the pulse rate was 116 beats/min, and the respiratory rate was 24 breaths/min. Blood pressure recorded was 90/60 mm Hg.

There was pallor, no lymphadenopathy, and no cyanosis. There was pain and swelling in the left knee joint. There were limitations of the movements.

Cardiovascular system revealed the presence of diastolic thrill at the apex associated with the mid-diastolic murmur at the apex. The first heart sound was loud. Second heart sound was normal. Respiratory system revealed presence of crepitation at the base. Per abdomen examination revealed no significant abnormality.

INVESTIGATION

- *Hemoglobin:* 9 g/dL
- *Total leukocyte count (TLC):* 7,600 cells/cumm
- *Differential leukocyte count (DLC):* P_{72} L_{18} M_2 E_6 B_2
- *Erythrocyte sedimentation rate (ESR):* 28 mm in the first hour
- *X-ray chest:* Cardiomegaly
- *Antistreptolysin O (ASLO):* 400 Todd units
- *C-reactive protein (CRP):* 1,100 µg/dL (normal range: 67–1,000 µg/dL)
- *Throat culture and sensitivity:* Sterile
- *Blood culture and sensitivity:* Sterile
- *Electrocardiogram (ECG):* PR interval is prolonged, right ventricular hypertrophy seen

DISCUSSION

Rheumatic fever is a multisystem disease, triggered by group A beta-hemolytic *Streptococcus*. Its acute manifestations include arthritis, carditis, chorea, subcutaneous nodules, and erythema marginatum. It is recurrent in nature and results in chronic heart disease. Hence it is said, rheumatic fever licks the joints and bites the heart.

Acute rheumatic fever (ARF) is a non-suppurative sequela of pharyngeal infection with group A *Streptococcus* (GAS). Target organs of the resultant autoimmune process include the heart, joints, central nervous system, and subcutaneous tissues. Permanent cardiac damage is the most important consequence of this disease.

Acute rheumatic fever is an immunological disorder. This is initiated by group A beta-hemolytic streptococci. Antibodies produced against the streptococcal protein and sugar react against the connective tissue of the body as well as heart. This results in rheumatic fever.

Predisposing factors include poor socio-economic condition leading to unhygienic living conditions and overcrowded households. Under and poor nutrition will alter the immune system.

The most common age involved is 5–15 years. Mitral valve disease and chorea are common in female. Aortic valve involvement is common in males. Rheumatic fever occurs during winter season.

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As many as two-thirds of patients with an acute episode of rheumatic fever a have history of an upper respiratory tract infection several weeks before, and the peak age and seasonal incidence of ARF closely parallel that of GAS pharyngitis. Patients with ARF almost always have serologic evidence of a recent GAS infection. Their antibody titers are usually considerably higher than those seen in patients with uncomplicated GAS infections.

Rheumatic fever follows an attack of streptococcal infection of upper respiratory tract and usually there will be laboratory evidence of recent streptococcal infection. The latent period of rheumatic fever is 1–3 weeks and that of chorea is 2–6 months. The major epidemiological risk factor for the development of ARF is group A β-hemolytic streptococcal infection of the upper respiratory tract. Almost all serotypes of streptococci are involved.

Not all serotypes of GAS can cause rheumatic fever. When some GAS strains (e.g., M type 4) cause acute pharyngitis in a very susceptible rheumatic population. Certain serotypes of GAS (M types 1, 3, 5, 6, 18, 19, 24, 27, and 29) are more frequently isolated from patients with ARF than are other serotypes.

The concept of rheumatogenicity is further supported by the observation that although serotypes of GAS frequently associated with skin infection can be isolated also from the upper respiratory tract, they rarely cause recurrences of rheumatoid factor (RF) in individuals with a previous history of RF or first episodes of RF.

Certain populations with increased genetic susceptibility such as polymorphisms of interferon gamma (IFN-γ), angiotensin-converting enzyme (ACE), FCN, FcgRIIA, toll-like receptor 2 (TLR-2), and human leukocyte antigen (HLA) can explain why rheumatic fever occurs specifically in certain individuals.

PATHOGENESIS

The cytotoxicity theory suggests that a GAS toxin is involved in the pathogenesis of ARF and rheumatic heart disease. However, a major problem with the cytotoxicity hypothesis is its inability to explain the substantial latent period (usually 10–21 days) between GAS pharyngitis and onset of acute RF.

In addition to the specific characteristics of the infecting strain of GAS, the risk of developing ARF is also dependent on various host factors. The incidence of both initial attacks and recurrences of ARF peaks in children aged 5–15 years, which is the age-group greatest at risk for GAS pharyngitis. Patients who have had an attack of ARF tend to have recurrences, and the clinical features of the recurrences tend to mimic those of the initial attack. In addition, there appears to be a genetic predisposition to ARF.

The streptococcal cell wall proteins as well as carbohydrates have the capacity to produce antibodies capable of reacting with human connective tissue resulting in rheumatic fever. It appears to be the result of host's unusual response at both the cellular and humoral level to the *Streptococcus*. Antibodies against the heart muscle, antiheart antibodies and the nervous system, antineuronal antibodies are found in high titers with carditis and chorea.

An immune-mediated pathogenesis for ARF and rheumatic heart disease has been suggested by its clinical similarity to other illnesses with an immunopathogenesis and by the latent period between the GAS infection and ARF. The antigenicity of several GAS cellular and extracellular epitopes and their immunologic cross-reactivity with

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cardiac antigenic epitopes also lends support to the hypothesis of molecular mimicry.

Common epitopes are shared between certain GAS components (e.g., M protein, cell membrane, group A cell wall carbohydrate, capsular hyaluronate) and specific mammalian tissues (e.g., heart valve, sarcomere, brain, joint). Additionally, the involvement of GAS superantigens such as pyrogenic exotoxins in the pathogenesis of ARF has been proposed.

A more recently proposed pathogenetic hypothesis is that the binding of an M protein N-terminus domain to a region of collagen type IV leads to an antibody response to the collagen, resulting in ground substance inflammation especially in subendothelial areas like cardiac valves and myocardium.

PATHOLOGY

Acute rheumatic fever occurs most often in the winter and spring seasons and in children ages 5–15 years. Much less commonly, it has been reported in the preschool age-group. Patients with ARF have a high likelihood of recurrence when reinfected with GAS; this tendency declines with age and age increased time since the last episode. Environmental factors such as nutrition, wading, and age all appear to influence the incidence of ARF, probably because the same factors influence the incidence of streptococcal pharyngitis.

Antigenic differences among GAS serotypes are related to the bacterial M protein, found within its cell wall. Recent data demonstrated a shift in prevalence from "rheumatogenic" to "nonrheumatogenic" M types over the past 40 years that parallels, the decrease in the incidence of ARF over this period. The period of 10 days to 3 weeks between streptococcal pharyngitis and ARF is consistent with a cellular and humoral immune response. Cross-reactivity or streptococcal antigens and human cardiac, synovial, and brain antigens also support an immune mechanism of ARF.

Pathologic changes are found throughout the body in connective tissue and around small blood vessels. The pathognomonic, lesson of rheumatic fever is the Aschoff body, a painless nodular connective tissue lesion consisting of fibrinoid changes and a collection of lymphocytes, plasma cells, and histiocytes. Within the heart, the endocardium and myocardium are most often affected; the pericardium involved by extension or as serositis. Active valvulitis results in variable degrees of valve insufficiency, with chronic changes possibly leading to valvar stenosis. The mitral and aortic valves are affected most commonly, the tricuspid less frequently, and the pulmonary valve rarely.

Pathologic changes in the joints consist of joint effusion, exudation with edema of synovial membranes, focal necrosis in the joint capsule, and edema and inflammation in periarticular tissue. These changes are completely reversible. Subcutaneous nodules seen during the acute phase of the disease histologically resemble Aschoff bodies.

The pathological process is described in two stages:

1. Exudative stage
2. Proliferative stage

Exudative stage: In this acute phase, there is fibrinoid necrosis of connective tissues with edema of collagen fibers, resulting in two major clinical signs.

1. *Arthritis:* Without residuals
2. *Pancarditis:* It is a life-threatening condition

Proliferative stage: It is a more prolonged process, resulting in scarring of myocardium and endocardium. During acute RF, Aschoff bodies involve all the layers to produce pancarditis. The hallmark of the proliferative

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phase is the formation of Aschoff bodies. These are granulomas due to injury to collagen. Other important event is deposition of gamma globulins in heart. Lesion in pericardium is a fibrinous inflammation and macroscopically shows a "bread and butter" appearance. Endocarditis affects the valvular or mural endocardium. When the valvular endocardium heals, it results in scarring and deformity of valves. The valves damaged usually are mitral, aortic, tricuspid, and pulmonary.

CLINICAL FEATURES (FIG. 1)

The classical clinical picture of rheumatic fever includes streptococcal sore throat with fever. Rheumatic fever is diagnosed depending upon presence of criteria, which may be major or minor. Major criteria include carditis, subcutaneous nodules, chorea, and erythema marginatum. The minor criteria include fever, arthralgia, ASLO titer value, positive throat culture, acute phase reactions, and prolonged PR interval.

Diagnosis requires that an individual have either two major criteria or one major criterion plus two minor criteria along with evidence of streptococcal infection. Exceptions are chorea or indolent carditis, which each may by itself indicate rheumatic fever.

The essential criteria include evidence of recent streptococcal infections such as raised ASLO titer and positive throat culture.

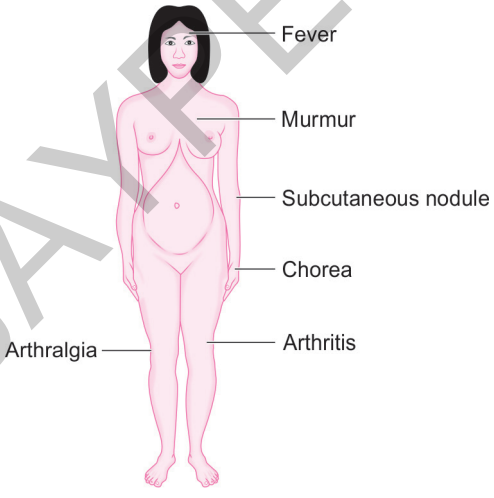

Fig. 1: Clinical features.

ESSENTIAL DIAGNOSTIC POINTS

- Migratory polyarthritis
- Carditis
- Sydenham's chorea
- Evidence streptococcal infection such as scarlet fever, positive throat culture, ASLO titer
- Acute phase reactants are elevated
- EEG changes

(ASLO: antistreptolysin O; EEG: electroencephalogram)

CLINICAL MANIFESTATIONS AND DIAGNOSIS

There is no single specific clinical manifestation or specific laboratory test that establishes the diagnosis. There are a number of selective clinical findings (Jones criteria) that make diagnosis of RF highly probable. The revised Jones criteria are described in **Table 1**.

TABLE 1: Revised Jones criteria.

Major criteria	*Minor criteria*
• Carditis • Polyarthritis migratory • Erythema marginatum • Chorea	• Fever • Arthralgia • Previous history of rheumatic fever • Elevated acute phase • Phase reactants (ESR, CRP) • Prolonged PR interval on an ECG
Subcutaneous nodules	
Plus (essential criteria)	
Evidence of a preceding group A streptococcal infection (culture, rapid rise in antigen–antibody titer)	

(CRP: C-reactive protein; ECG: electrocardiogram; ESR: erythrocyte sedimentation rate)

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The presence of two major criteria or one major and two minor criteria indicates a high probability of the presence of rheumatic fever. Evidence of preceding streptococcal infection greatly strengthens the possibility of ARF. Its absence should Sydenham's make the diagnosis doubtful, except of Sydenham's chorea or long-standing carditis.

The *Jones criteria*, as revised in 2015, is now intended for diagnosis of the initial attack of ARF and recurrent attacks. There are five major and four minor criteria and a requirement of evidence of recent GAS infection.

Diagnosis of a first attack or recurrent attack of ARF can be established when a patient fulfills two major or one major and two minor criteria and has evidence of preceding GAS infection. Diagnosis of recurrent ARF can also be made only in the moderate/high-risk population by presence of five minor criteria with evidence of preceding GAS infection. In the 2015 Jones criteria revision, a major change from previous versions expands the definition of the major criterion carditis to include subclinical evidence [e.g., in the absence of a murmur, echocardiographic evidence of mitral regurgitation (MR) meeting specific criteria to distinguish physiologic from pathologic MR].

Migratory Polyarthritis

In rheumatic fever, 60–70% suffer from acute arthritis. Arthritis, the early manifestation of rheumatic fever lasts for 3–7 days and leaves no residual damage. It is polyarthritis involving the large joints such as knee, ankles, and elbows. Acute arthritis affecting the major joints is the characteristic involvement. The joints show signs of acute inflammation grade-IV tenderness and limitation of movements. The arthritis is migratory and fleeting and leaves behind no residual changes.

Arthritis occurs in approximately 75% of patients with ARF and typically involves larger joints, particularly the knees, ankles, wrists, and elbows. Involvement of the spine, small joints of the hands and feet, or hips is uncommon. Rheumatic joints are classically hot, red, swollen, and exquisitely tender, with even the friction of bed clothes being uncomfortable. The pain can precede and can appear to be disproportionate to the objective findings. The joint involvement is characteristically migratory in nature, i.e., a severely inflamed joint can become normal within 1–3 days without treatment, even as one or more other large joints become involved. Severe arthritis can persist for several weeks in untreated patients. Polyarthritis is the most common of the major criteria and lasts <4–6 weeks when untreated. Characteristic is a dramatic response to salicylates and nonsteroidal anti-inflammatory drugs (NSAIDs); early use in febrile children may confound diagnosis.

Carditis

Carditis occurs in approximately 50–60% of all cases of ARF. Recurrent attacks of ARF in patients who had carditis with their initial attack are associated with high rates of carditis with increasing severity of cardiac disease. The major consequence of acute rheumatic carditis is chronic, progressive valvular disease, particularly valvular stenosis, which can require valve replacement. In developing countries, 60–80% cases of rheumatic fever have clinical features of carditis and in 40% is seen during the first attack of rheumatic fever.

A major change in the Jones criteria is the acceptance of subclinical carditis (defined as without a murmur of valvulitis but with echocardiographic evidence of valvulitis) or clinical carditis (with a valvulitis murmur) as fulfilling the major criterion of carditis in all populations.

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Echocardiography is an important tool to confirm cardiac involvement in ARF.

The following are the echocardiographic criteria developed in 2012:

- *Doppler criteria:*
  - Pathological mitral regurgitation—four criteria (all must be met)
  - Visible at least in two projections
  - Regurgitation jet length ≥2 cm at least in one projection
  - Regurgitation peak velocity >3 m/s; regurgitation pansystolic
  - Pathological aortic regurgitation—four criteria (all must be met)
  - Visible at least in two projections:
    - Regurgitation jet length ≥1 cm at least in one projection
    - Regurgitation peak velocity >3 m/s
    - Regurgitation pandiastolic
- *Morphological criteria:*
  - In acute mitral valve involvement:
    - Dilatation of mitral annulus
    - Elongation of chordae tendinae
    - Rupture of chordae tendinae with acute mitral regurgitation
    - Prolapse of anterior (less often posterior) leaflet
    - Nodular lesions on leaflets
  - In chronic mitral valve involvement (invisible in acute involvement):
    - Thickening of leaflets
    - Thickening of chordae tendinae with their fusion
    - Limited mobility of leaflets
- *Calcifications:*
  - Lesions in acute and chronic aortic valve involvement:
  - Symmetrical or focal thickening of leaflets
  - Disturbed leaflet coaptation (leaflet closing during systole)
  - Limited mobility of leaflets
  - Prolapse of leaflets

Acute rheumatic carditis usually presents as tachycardia and cardiac murmurs, with or without evidence of myocardial or pericardial involvement. Moderate-to-severe rheumatic carditis can result in cardiomegaly and heart failure with hepatomegaly and peripheral and pulmonary edema. Echocardiographic findings are not diagnostic but include pericardial effusion, decreased ventricular contractility, and aortic and/or mitral regurgitation.

Mitral regurgitation is characterized typically by a high-pitched apical holosystolic murmur radiating to the axilla. In patients with significant Initial regurgitation, this may be associated with an apical mid-diastolic murmur of relative mitral stenosis. Aortic insufficiency is characterized by a high-pitched decrescendo diastolic murmur at the left sternal border.

It is pancarditis involving pericardium, myocardium, and endocardium. Carditis occurs within 2 weeks of the onset of the fever. It is an early manifestation of the rheumatic fever so that by the time a patient seeks help, he/she had already had carditis.

Pericarditis may appear suddenly and may be associated with precordial pain and a friction rub. More often, however, patients with pericarditis are asymptomatic. Pericarditis seldom appears without endocarditis and myocarditis, the combination being termed pancarditis. Death may occur during the acute phase of carditis; permanent cardiac damage may result in long-term disability, usually because of mitral or aortic valvar insufficiency and/or stenosis. Pericarditis results in pericardial pain. The pain may be quite severe. Friction rub may be heard on auscultation. ECG shows ST and T wave changes. A patient of rheumatic pericarditis always has additional mitral or mitral and aortic regurgitation murmur.

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Myocarditis presents with cardiac enlargement, soft heart sound, and congestive cardiac failure (CCF). Carey Coombs murmur is a delayed diastolic mitral murmur heard during the course of ARF. S_3 gallop may be present. Gallop rhythm is an abnormal array of heart sounds in which third and fourth heart sounds may also be present, and usually indicates active disease of the heart. Arrhythmias are present.

Endocarditis (valvulitis) is represented by pansystolic murmur of mitral regurgitation. Tricuspid valvulitis results in tricuspid regurgitation. The severity of the valvular endocarditis causes acute or later chronic hemodynamic overload. Acute hemodynamic overload is the main reason of morbidity and mortality of the rheumatic fever. The most frequent murmur is an apical regurgitant systolic murmur of mitral regurgitation. With severe mitral regurgitation, the third heart sound may be followed or replaced by a low-pitched mid-diastolic rumble. The early diastolic murmur of aortic regurgitation is the second most common murmur in ARF and generally occurs only in patients who also have mitral regurgitation.

Cardiomegaly may be evident on radiograph. Severe myocarditis may result in congestive heart failure with signs including jugular venous distention, hepatomegaly, and pulmonary edema with rales. Prolongation of the PR demonstrated on an electrocardiogram interval is common but does not indicate carditis.

Clinical Features of Carditis

- *Murmurs:*
  - Significant apical systolic murmurs (musical quality of grade III)
  - Apical mid-diastolic murmur due to edema of valve (Carey Coombs murmur)
  - Basal diastolic murmur
- Enlarged heart
- Congestive heart failure
- Presence of pericardial rub

Others include:

- Tachycardia (sleeping)
- Heart sounds (decreased or muffled)
- Gallop, arrhythmias, changing murmurs
- Subcutaneous nodules
- Persistent dry cough, ECG changes, laboratory findings, raised ESR, and presence of CRP
- Valves affected in frequency of occurrence are mitral 70%, mitral and aortic (7%), and tricuspid and pulmonary valves are rarely affected

Subcutaneous Nodules

They appear as early as 6 weeks after the onset of fever. These lesions are very infrequent and are present in 1–10% of patients with rheumatic fever. Nodules are better seen than felt. They are firm, nontender, pea-sized nodules seen over the extensor aspects of forearm, elbows, wrist, knees, ankles, spine, scalp, and along tendons. The presence of rheumatic nodules indicates presence of activity in 100% of cases and underlying carditis in 70% of cases. Subcutaneous nodules are a rare finding (<1% of patients with ARF) and consist of firm nodules approximately 1 cm in diameter along the extensor surfaces of tendons near bony prominences. There is a correlation between the presence of these nodules and significant rheumatic heart disease.

Chorea

Chorea occasionally is unilateral (hemichorea). The latent period from acute GAS infection to chorea is usually substantially longer than for arthritis or carditis and can be months. Onset can be insidious, with symptoms being

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