iled information on cardiology books, visit our website www.ejaypee.com, for detailed information on cardiology books, visit our website www.ejaypee.com

Clinical Cardiology

Made Easy®



Ramesh R Rau

JAYPEE

Contents

1. Ischemic Heart Disease **1**
- *Angina Pectoris 1*
- *Acute Coronary Syndromes 11*
- *Acute Myocardial Infarction 15*

2. Hypertension: The Silent Killer **29**
- *Epidemiology 29*
- *Definition 29*
- *Etiology 30*
- *Symptoms 31*
- *Signs 32*
- *Measurement of Blood Pressure 32*
- *Laboratory Evaluation of Hypertension 33*
- *Secondary Hypertension 36*
- *Treatment 37*

3. Heart Failure **46**
- *Epidemiology 46*
- *Causes 46*
- *Pathophysiology 47*
- *Neurohormonal Response to Low Cardiac Output 49*
- *Symptoms 50*
- *Signs 50*
- *Investigations 53*
- *Treatment of Heart Failure 54*

4. Rheumatic Fever **60**
- *Incidence 60*
- *Genetics 60*
- *Etiopathogenesis 61*
- *Pathology 61*
- *Clinical Features 62*
- *Investigations 64*
- *Management 65*

5. Valvular Heart Disease **68**
- *Clinical Pattern of Valvular Involvement 68*

6. Cardiac Arrhythmias **93**
- *How is Heart Rate Controlled? 93*
- *Disorders of Impulse Formation 95*
- *Disorders of Impulse Conduction 110*

7. Myocardial Diseases **116**
- *Hypertrophic Cardiomyopathy 116*
- *Management 120*
- *Dilated Cardiomyopathy 122*
- *Alcoholic Cardiomyopathy 127*
- *Restrictive Cardiomyopathy 127*

8. Congenital Heart Disease **131**
- *Atrial Septal Defect 132*
- *Ventricular Septal Defect 136*
- *Patent Ductus Arteriosus 140*
- *Tetralogy of Fallot 143*
- *Transposition of Great Arteries (Complete Transposition, D-transposition) 147*
- *Corrected Transposition (L-transposition) 149*
- *Total Anomalous Pulmonary Venous Connection (TAPVC) 150*
- *Ebstein's Anomaly 151*
- *Eisenmenger Syndrome 153*

9. Pregnancy and Heart Disease **155**
- *Hemodynamics of Normal Pregnancy 155*
- *Congenital Heart Disease 159*
- *Systemic Hypertension During Pregnancy 161*

10. Pericardial Disease **162**
- *Pericarditis 162*
- *Cardiac Tamponade 166*
- *Differential Diagnosis 169*
- *Prognosis 169*
- *Treatment 170*
- *Chronic Constrictive Pericarditis 172*

11. Endocarditis **176**
- *Changing Pattern 177*
- *Pathogenesis and Pathology 178*
- *Clinical Features 180*
- *Diagnosis 183*
- *Differential Diagnosis 183*
- *Treatment 184*
- *Prophylaxis 188*

12. Pulmonary Artery Diseases **190**
- *Pulmonary Hypertension 190*
- *Chronic Cor Pulmonale 195*
- *Pulmonary Embolism 203*

13. Miscellaneous Disorders **228**
- *Thoracic Aortic Aneurysm 228*

14. ECG analysis **241**
- *Normal ECG 241*

Index **251**

Chapter
4

Rheumatic Fever

Incidence

Rheumatic fever (RF) is rare these days in developed countries, although complacency in its control there has produced a resurgence in many parts of USA and the UK. Indian statistics are not complete; it is estimated that there are more than 1 million patients with rheumatic heart disease in India. Conservative estimates put the incidence of acute RF at 50,000 new cases per year. A special feature of this disease in India is the early onset of symptomatic valvular disease in children; a few of these can be truly malignant in their clinical course (Juvenile rheumatic heart disease).

The disease is common in childhood and adolescence, and is rare in infants and in old age and attacks the poor, especially those living in damp, dirty, and unhygienic surroundings. Its incidence parallels that of streptococcal tonsillitis. Although rare in adults, it presents with more severe joint disease in them. The saying is "it licks the joint but bites the heart in children and licks the heart and bites the joint in older patients"; however this may not always be correct. Figure 4.1 gives the natural history of RF.

Genetics

Genetic factors have been implicated in the pathogenesis and it is not infrequent to find multiple cases in one family. The present data support the hypothesis of a rheumatic fever gene within or near the HLA complex. Patients with RF may have an abnormal immunological response both at the humoral and cellular levels to streptococcal antigens cross-reactive with mammalian tissue. This abnormal response is likely to be genetically programmed.

JAYPEE BROTHERS

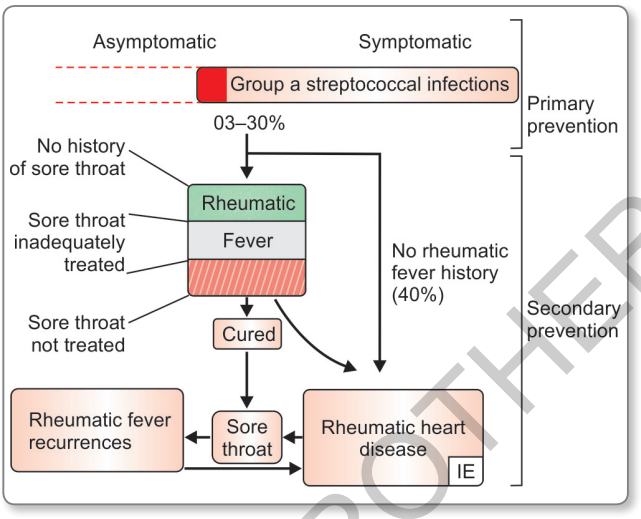

Fig. 4.1: Natural history of rheumatic fever

Etiopathogenesis

There is, as yet, no proof of an infective agent causing RF, though it is nearly always preceded by streptococcal sore throat, the latent interval being 10–20 days.

Rheumatic fever is the result of an abnormal immune response to the products of hemolytic streptococcal infection in a sensitized individual. It is possible that some of the antibodies to antigens present on the streptococcal cell wall may cross-react with myofibrils in the heart, thereby setting up pancarditis.

Pathology

Fulminating attacks which end fatally show only non-specific changes like the Arthus phenomenon, seen in many other acute infections. Edema, fragmentation of collagen, leukocyte infiltration, hyperemia and capillary hemorrhage are seen in synovial membranes of the larger joints, pericardium, myocardium, endocardium, the pleura and the lungs.

JAYPEE BROTHERS

The classical rheumatic lesion, however, is proliferative. This occurs later, the best example being the Aschoff nodule. The subcutaneous nodule is another example. A necrotic collagenous center is surrounded by large multinucleated reticuloendothelial cells, along with lymphocytes and plasma cells. This is seen in the myocardium near blood vessels. Fibroblastic proliferation is usual. Rarely panarteritis of many viscera may be seen.

A true valvulitis occurs in the heart. In the acute stage the valve is edematous, and soon shows signs of damage to the area of maximum apposition just below its free edge. Small pink nodules may be seen at this stage. The inflamed valves later get fibrosed and fusion occurs. This repair process might damage the valves, papillary muscles, chordae tendineae, and the valve rings. The mitral valve is most damaged, with the aortic valve next in order; the other valves also get damaged occasionally.

Clinical Features

Rheumatic fever is a systemic disease with varied clinical presentations (Fig. 4.2). Although subclinical attacks may not have significant joint involvement, clinical presentation without joint involvement is very rare.

Arthritis: This is usually polyarthritis, sometimes flitting from joint to joint (migratory), affecting the larger joints more than the smaller ones. Swelling, redness and tenderness are the common findings and occasionally joint effusions. Vague pain in the joints may not be a feature of this disease. Joint manifestations are more prominent in adults. History of sore throat 2–4 weeks prior to the joint involvement may be obtained in some people.

Skin lesions: The classical erythema marginatum—large erythematous lesions with prominent margins slightly raised—are seen more often in white people and is rare in Indians. The red margins take longer to fade and so the lesions sometimes look like red rings.

JAYPEE BROTHERS

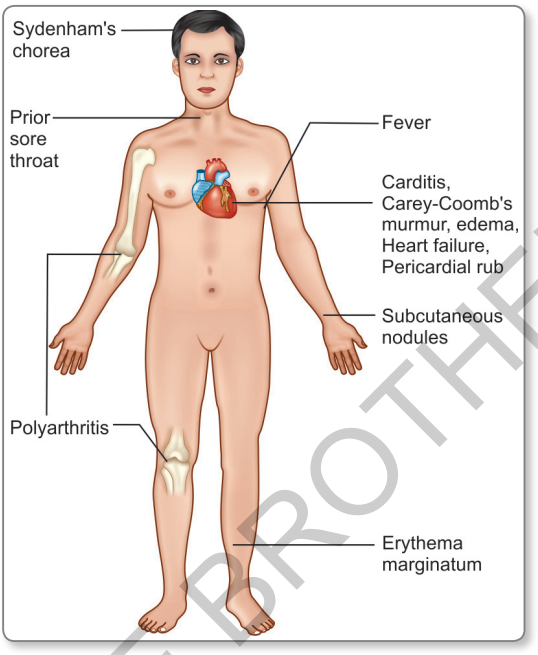

Fig. 4.2: Clinical features of the rheumatic fever

Subcutaneous nodules: These are painless, roundish, firm lumps overlaid by normal looking skin (Fig. 4.3). They range from a few millimeters to 1.5 cm in diameter, and are localized over bony prominences like the elbow, shin and spine. If carefully looked for, these are not as rare as reported. They sometimes last longer than a month.

Carditis: This is the single most important lesion. It presents with breathlessness, chest pain of pericardial type, and palpitations due to tachycardia. Cardiac enlargement with heart murmurs is not uncommon. The mitral valve being the most common valve involved, a soft middiastolic murmur due to thickening of the mitral valve, the Carey-Coombs murmur, is the classical picture; in practice, a soft systolic murmur is more common. Pericardial rub may be heard at some stage in the disease.

JAYPEE BROTHERS



Fig. 4.3: Subcutaneous nodules

Severe carditis with mitral and aortic valve leaks may result in heart failure; heart failure may also be due to myocarditis. The latter may present with heart blocks and syncope. A fulminant course is not uncommon in the tropics where the disease progresses to heart failure and death in a very short time.

Rheumatic chorea: Described first by Sydenham, this clumsy movement of the limb muscles in a child is rarely a feature of acute RF. It is usually seen after a lapse of 4–6 weeks, during the cold stage. Jerky, involuntary movements are the feature and they usually subside after sometime. The child may be thought of as being restless or inattentive in school.

Fever: This is usually a feature during the acute phase. Pleurisy, pneumonia, and occasionally pleural effusion may also be seen but are rare.

Modified Duckett-Jones criteria: The clinical features of this disease have been grouped into major and minor criteria by Duckett-Jones; these have been revised by the American Heart Association. A diagnosis of rheumatic fever is likely when two major criteria are present or when one major criterion is seen along with two minor ones, along with evidence of past streptococcal infection (Table 4.1).

Investigations

Routine blood counts and erythrocyte sedimentation rate (ESR) are nonspecific but are useful for follow up. Throat cultures for

JAYPEE BROTHERS

Table 4.1: Jones criteria (revised by American heart association, 1984)

Major		Minor
	Clinical	
Carditis		Fever
Polyarthritis		Arthralgia
Chorea		Postrheumatic fever or rheumatic heart disease
Erythema marginatum		
Subcutaneous nodules		
	Laboratory	
		Elevated ESR
		CRP positive
		Leukocytosis
		Prolonged PR interval
	Plus	

Supporting evidence of preceding streptococcal infection (increased ASO or other streptococcal antibodies; positive throat culture for group A streptococcus; recent scarlet fever).

streptococci are helpful. Anti-streptolysin O (ASO) titers are useful if there is a rising titer or, in its absence, levels above 200 units. In many patients this may be normal.

ECG evidence of carditis is not infrequent. Prolonged PR interval, varying grades of heart block and occasionally repolarization changes (ST-T changes) may suggest pericardial or myocardial involvement. Echocardiography may help to diagnose cardiomegaly and/or pericardial effusion. Valve damage may also be seen (Table 4.2).

Management

Supportive therapy: Bed rest is important and activity should be curtailed until the ESR comes to normal. Heart failure when

JAYPEE BROTHERS

Table 4.2: Work-up in a case of acute rheumatic fever

Blood	CBC (leukocytosis), ESR (raised), C-reactive protein (raised)
	ASO titer (raised > 200 units)
Throat	Throat swab for β-hemolytic streptococci.
Chest X-ray	Cardiomegaly
ECG	Increased PR interval (I degree heart block, rarely II degree heart block) If pericarditis—Low voltage, T wave inversion
2D-echo	For valve abnormality and cardiomegaly

Table 4.3: Differential diagnosis

1. Rheumatoid arthritis: Small joints are involved, not migratory; older age group is affected, females more often; deformities of joints are seen, small muscle wasting may be seen, and the serology is diagnostic.
2. Osteoarthritis: Older age, single joint involvement, and effusion are more common; Heberden's nodes may be seen in the fingers; serology is negative.
3. Gouty arthritis: Usually the great toe is affected but it could be polyarthritic; tophi may be seen. Older men are affected, uric acid levels are high and the clinical presentation is very typical.
4. Systemic lupus erythematosus: The clinical picture and laboratory tests should distinguish it.
5. Lyme disease: Usually seen in the West, it presents with annular erythematous skin rashes, knee joint pain, fever, heart blocks and similar serological features.
6. Fibrositis: Muscular rheumatism is a vague syndrome of adults where minor aches and pains mostly reflect their mild depressive illness. The clinical picture is typical and the joint involvement is only an arthralgia.
7. Septic arthritis: In children this should be easy to distinguish as the clinical presentation is more dramatic with signs of systemic infection, and the joint fluid will grow the organism.
8. TB joints: Joint signs are classical, and systemic manifestations may be seen. A single joint is involved except in the spine.
9. Adverse drug reaction: Arthralgia is a common feature of allergy seen with many drugs. Multiple joints may be affected and the skin lesions and history will be diagnostic.

JAYPEE BROTHERS

present needs attention. If the fulminant disease progresses to valve damage, rarely surgery may be needed on an urgent basis. Heart blocks are usually transient and rarely need pacing.

Salicylates: Aspirin in large doses (60 mg/kg body weight in six divided doses) is the drug of choice. In adults double this dose can be given. This should be continued till the ESR comes down and then tapered off. Nausea, tinnitus, vomiting, deafness, and rarely tachypnea and acidosis may be the side effects. Reye's syndrome precludes its use in small children.

Steroids: These are more useful in symptom relief especially in severe cases, although there is no long-term advantage compared to aspirin. Prednisolone in doses of 0.25 mg/Kg body weight in divided doses is given until the ESR comes to normal and then tapered off over 3–4 weeks.

Antibiotics: To treat the streptococcal infection an initial dose of 1,200,000 units of benzathine penicillin should be given, initially once a week for three weeks and then monthly for the Ist year. Long-term prophylaxis is still debated. Benzathine penicillin 1,200,000 units monthly or three weekly, or oral phenoxymethyl penicillin 500 mg daily may be continued until the age of 40 years. Benzathine penicillin once in 2 weeks may give better results than the monthly dose. In community studies long-term penicillin has been shown to reduce the incidence of streptococcal reinfection but in individuals there is no proof of that. In penicillin-sensitive individuals erythromycin or sulfadiazine are preferred (Table 4.3).

Long-term Care

These patients need follow-up on a regular basis to rule out cardiac valvular involvement, preferably by echocardiography. When present, its management with drugs or surgery may be required. Carditis is seen usually within weeks of the onset but may rarely be seen for the first time after the 4th week. Recurrence of RF is very common and must be prevented by antibiotic prophylaxis and also by change in the environment of the patient.

JAYPEE BROTHERS

detailed information on cardiology books, visit our website www.ejaypee.com, for detailed information on cardiology books, visit our website www.ejay

Clinical Cardiology
Made Easy®

There are so many books on cardiology. Why one more? This one is an attempt to present the fundamentals of cardiology in simple terms which a student of medicine can grasp. There is good coverage for a busy doctor to revise any topic or seek more information but in a concise way about any topic. Thus, it is a good addition to any doctor's personal library.



Ramesh R Rau MD FIAMS CCP was born in Mysuru, Karnataka, India but had his schooling in Ahmedabad, Gujarat, India. Presently, he is Consultant Physician and Perfusionist, SAL Hospital, Ahmedabad. He secured 3rd rank in Gujarat state SSC Board and 2nd in pre-university examination in Gujarat University. He studied medicine at the Government Medical College, Vadodara, Gujarat. He completed his postgraduation in medicine there and started practice. He learnt cardiopulmonary perfusion with heart lung machine and became the first perfusionist of Gujarat. This association with open heart surgery created an interest in cardiology. He has 34 years of experience in medicine and heart surgery.

Shelving Recommendation
CARDIOLOGY

ISBN 978-93-5152-662-9
9 789351 526629

Buy from ejaypee
JAYPEE BROTHERS