Rheumatic Fever - How Relevant in India Today?

GS Sainani*, Anjana R Sainani**

INTRODUCTION

Rheumatic fever is an immunologically mediated connective tissue disease subsequent to throat infection with group-A beta haemolytic streptococci. It is characterized by an inflammatory process involving collagen fibrils and the ground substance of the connective tissue. The primary sites of affliction are heart, joints and central nervous system. The most important sequel of rheumatic fever is the rheumatic heart disease (RHD), which results in significant morbidity and mortality.

EPIDEMIOLOGY

The epidemiology of acute rheumatic fever (RF) is linked with that of group-A beta hemolytic streptococcal pharyngitis; both have a maximum incidence in the age group of 5-15 years. RF is rare before the age of 5 years in the West.1 In India, the average age at presentation has been reported by Padmavati to be between 10 and 14 years.2 However, the early development (under 5 years) of established rheumatic heart disease and rapid progression to disabling cardiac involvement poses a major problem in India and has been termed as “Juvenile Mitral Stenosis”.3 On occasions, it may occur in older age groups, as is seen in the epidemics occurring in closed populations like military recruits, crowded living conditions and those in contact with school children.4 In adults, it is mostly seen in the second and early third decade of life. It is more common in the winter season when the streptococcal pharyngitis is also on the rise. The incidence of rheumatic fever has been on a decline in developed countries (less than 5/100,000/yr). It still remains a major health problem in developing countries (the incidence being 27-100/100,000/yr).5 The prevalence of rheumatic heart disease in school children in various parts of the world is shown in Table 1.

The prevalence rate of rheumatic heart disease in India is around 6-11/1000 in school children.6 The prevalence of rheumatic heart disease in school children under twelve years in Mumbai is 15 per 10000 population.7 Rheumatic fever occurs in all races worldwide. Crowded living conditions and poverty are associated with a higher prevalence of the disease.

There has been resurgence and decline in rheumatic fever all over the globe. It is worth stressing that RF and RHD were unknown in India in the first 3 decades of the last century.8 Rogers,9 a professor of pathology in Calcutta Medical College did not find a single case of rheumatic carditis in Indians among the 4800 of post-mortem records of 37 years. It was only in 1935 that Kutumbiah10 reported it to account for 40% of heart cases in males and 52% in females. After World War II, several school surveys have been reported. Between 1940 and 1983, school surveys estimated the average prevalence of rheumatic heart disease to be between 1.8 and 11 per 1000 (average 6 per 1000) in school children. While from 1984 to 1995, the prevalence was reduced (1 to 3.9 per 1000). As regards acute rheumatic fever, the prevalence was 0.05 to 1.7 per 1000 (from 1940-1983) and 0.18-3.0 per 1000 (from 1984-1995).11 But recently the reports of school surveys reveal increase in prevalence of rheumatic heart disease.12

In 1999, Sharma et al13 from Delhi examined 191 children below 12 years of age with definite clinical features of rheumatic fever. As regards the age group, 60% children were between 9 and 12 years, 31.4% were between 5 and 9 years and only 7.9% were below 5 years. Whereas Mishra et al14 from Orissa examined children below 19 years (mean age 15.1 ± 4.4 years). The M:F ratio was 4:1. Mild mitral stenosis was diagnosed in 34.9% and severe mitral stenosis was diagnosed in 33%. In the year 2000, Lachhundani et al15 while surveying rural and urban school children of Kanpur district examined 3963 children. The prevalence of rheumatic heart disease was 4.54 per 1000 (urban 2.56 and rural 7.42) The M:F ratio was 1:0.8. The prevalence of rheumatic fever in this group was 0.75 per 1000 (rural 1.20 and urban 0.42).

The data from Vellore on 43,544 consecutive patients of cardiovascular disorders over a 30 year period were analysed from 1960 to 1989 by Krishnaswami et al16. From a peak of 800 annual admissions of rheumatic valvular heart disease in late 1970s, it declined to 500 in 1989. Whereas cases of acute rheumatic fever (ARF) or rheumatic carditis of 85 admissions per year came down to 0 in late 1980s. Gupta et al17 reported after screening 10,263 school going children (6 to 16 years), none had ARF but 14 had valvular lesion.

To study the prevalence of rheumatic heart disease in school children of 46 countries, it has been reported by Achuuti et al18 that prevalence was higher in the decade of the 70s compared with the 80s when the study was repeated. The same downward trend in incidence was seen in Chile, Brazil and Cuba.

Table 1:

<table>
<thead>
<tr>
<th>Area</th>
<th>Prevalence Per 1000</th>
</tr>
</thead>
<tbody>
<tr>
<td>United States</td>
<td>0.6</td>
</tr>
<tr>
<td>Japan</td>
<td>0.7</td>
</tr>
<tr>
<td>India</td>
<td>6.0-11.0</td>
</tr>
<tr>
<td>Asia (other)</td>
<td>0.4-21.0</td>
</tr>
<tr>
<td>Africa</td>
<td>0.3-15.0</td>
</tr>
<tr>
<td>South America</td>
<td>1.0-17.0</td>
</tr>
</tbody>
</table>

*Emeritus Prof. of Med. (for life) Grant Medical College and JJ Group of Hosp., Mumbai; Hon. Physician, Jaslok Hospital & Research Centre, Mumbai; **Hon. Consultant, Armed Forces Medical Services, Mumbai.

www.japi.org © SUPPLEMENT TO JAPI • JUNE 2006 • VOL. 54
Decline in the incidence, severity and mortality of ARF has been reported in the Western countries over the period of last century. This decline has been attributed to improved nutrition of children and diminished exposure to the infecting organism. The reasons for decline of RF in western countries has been due to improved socio-economic status of the community. RF is considered as a social disease i.e. alteration in socio-economic state of a community will adversely or favourably affect the incidence of this disease.[1]

Its prevalence in low income developing countries as compared to affluent nations also gives credence to it.[15] The incidence of RF fell steeply in USA from 1970 onwards with extensive use of penicillin for prevention. In India, however, primary prevention has hardly been used. The third important factor that is responsible for the decline in RF is due to diminished streptococcal virulence and presence of fewer rheumatogenic sero types. Hence alteration in the virulence of streptococci seem to be a major factor in the decline or resurgence of the disease in the last century.[6]

**AETIOLOGY**

Though group-A beta haemolytic streptococci is the known aetiological agent, the exact pathogenic mechanisms are unknown. Only certain serotypes (e.g. M types 1,3,5,6,14,18,19,24,27,29) are known to cause rheumatic fever with a higher frequency than others and have been referred to as “rheumatogenic” strains.[19]

Evidence of an antecedent Group A streptococcal (GAS) infection is required for the confirmation of the initial diagnosis of acute rheumatic fever (RF). At the time of diagnosis of acute RF, only about 11% of patients have throat cultures positive for GAS. The paucity of positive cultures is due, in part to elimination of the organism by host defence mechanisms during the latent period between the onset of infection and the subsequent development of RF.[20]

Several rapid GAS antigen detection tests are available. Most of these tests have a high degree of specificity but a low sensitivity in a clinical setting. A negative test does not rule out the presence of group A streptococcal infection in the throat. A positive throat culture or rapid antigen test does not distinguish between a recent infection that can be associated with acute RF and chronic carrier of organism in throat.[20]

Because the presence of GAS in the throat may not reflect active infection, elevated or rising ASO titre provides more reliable evidence of a recent streptococcal infection than a positive culture or a positive rapid antigen test. The most commonly used antibody tests are the antistreptolysin O (ASO) and antideoxyribonuclease B (Anti-DNAase B). The ASO test is usually done first and if not elevated, the anti-DNAase B test is done. Elevated titres for both tests may persist for weeks or months. ASO titre rise and fall rapidly than anti-DNAase B. Other antibody tests which are occasionally done are anti hyaluronidase H (AH) and antistreptozyme (ASTZ). It must be stressed that elevated ASO titre (>250 Todd units (adults) and >333 Todd units (Children) are considered to be significant for diagnosis. ASO level may rise and fall irrespective of the course of rheumatic fever.[21]

**PATHOGENESIS**

The association between group-A beta haemolytic streptococcal upper respiratory tract infection and the subsequent development of acute rheumatic fever is well established. The exact pathogenic mechanisms are unknown largely due to lack of an animal model, though two basic mechanisms are implicated:

1. A toxic effect of the extra-cellular toxin of group-A beta haemolytic streptococci on target organs like myocardium, valves, synovium and brain
2. An abnormal immune response of the host to the streptococcal antigens

The group-A Streptococcus is a complex micro-organism producing a large number of somatic and extra-cellular antigens. Certain human tissues bear antigenic similarities to these and hence antibodies produced against the streptococcal antigens cross-react with the tissue antigens to produce an autoimmune response. The levels of these antibodies decline when the target tissue is experimentally removed from the body. The data supporting the cross reactivity theory of rheumatic fever includes the following:

1. Group-specific polysaccharide of the group-A beta hemolytic streptococcal wall is antigenically similar to the glycoprotein found in human and bovine cardiac valves
2. The somatic antigens of the streptococcal cell wall and cell membrane are similar to the human myocardial sarcolemma

| Table 2 |
|------------------|------------------|
| Major criteria   | Minor criteria   |
| Carditis         | Fever            |
| Polyarthritis, migratory | Arthralgia |
| Erythema marginatum | Elevated acute phase reactants (ESR,CRP) |
| Chorea           | Prolonged PR interval in ECG |
| Subcutaneous nodules | CRP=C reactive protein, ESR=Erythrocyte sedimentation rate, ECG=Electrocardiogram |
| Plus Evidence of preceding group-A streptococcal infection (culture, rapid antigen, and antibody rise/elevation) |

Two major or one major and two minor criteria plus evidence of preceding streptococcal infection indicate a high probability of rheumatic fever. In the three special categories listed below, the diagnosis of rheumatic fever is acceptable without two major or one major and two minor criteria. However, only for a and b can the requirement for evidence of a preceding streptococcal infection be ignored.

a. Chorea, if other causes have been excluded
b. Insidious or late-onset carditis with no other explanation
c. Rheumatic recurrence: in patients with documented rheumatic heart disease or, prior rheumatic fever, the presence of one major criterion, or of fever, arthralgia or elevated acute phase reactants suggests a presumptive diagnosis of recurrence. Evidence of previous streptococcal infection is needed.

3. The M protein of group-A beta haemolytic streptococci cross-reacts with human tissues as it shares certain common amino acid sequences. This M protein is the virulence factor that confers on the organisms the ability to resist phagocytosis and also the type specific immunity that is conferred against the specific M protein type.
4. In Sydenham chorea, antibodies directed against antigens found in group-A streptococcal cell membrane cross-react with tissues in the caudate nucleus of the brain.

Despite the knowledge of the inciting agent, it is not very well understood why only certain individuals are susceptible to development of rheumatic fever subsequent to streptococcal pharyngitis. The absence of acute rheumatic fever in very young children suggests that repeated exposures of the host to group-A Streptococcus is essential for precipitating the illness. The immunological system of the host including both cell-mediated and humoral, is an important factor for the susceptibility to acute rheumatic fever, but the exact mechanisms are unknown. Also, there are certain genetic influences which play a role since only about 3% of individuals develop acute rheumatic fever following acute streptococcal pharyngitis. There is also a higher concordance amongst monozygotic twins for the development of acute rheumatic fever. Recently, a B-lymphocyte alloantigen has been implicated in the determination of susceptibility to acute rheumatic fever in 70-90% of rheumatic patients. Certain HLA types viz. HLA-DR 1, 2, 3 and 4 haplotypes have been implicated in certain ethnic groups.

**Pathology**

The hallmark of acute rheumatic fever is an exudative and proliferative inflammatory reaction involving the collagen and connective tissue primarily of the heart, joints, brain and skin. Apart from the presence of a generalized vasculitis, the basic change is in the form of fibrinoid degeneration of the collagen characterized by the presence of Aschoff cells, which are modified fibrohistiocytic cells. Aschoff nodules are pathognomonic of rheumatic carditis but are documented only in 30-40% of biopsies from patients with rheumatic fever. Valvulitis is the main lesion accounting for the principal clinical manifestations. Valvulitis is characterized by edema, cellular infiltration of the valves and the chordae tendinae causing verrucae formation and hyaline degeneration with subsequent regurgitant valves. There is eventual fibrosis and calcification leading to stenotic valves.

**Clinical Features**

The American Heart Association (AHA) recommends the revised Jones criteria as a guide for diagnosis of acute rheumatic fever which has been approved by WHO study group for the diagnosis of the initial attack of acute rheumatic fever (Table 2).

**Major criteria**

- Carditis: The carditis of acute rheumatic fever is a pancarditis with involvement of pericardium, epicardium, myocardium and endocardium. Carditis occurs in 40-60% of the cases of rheumatic fever. Valvular insufficiency is the commonest defect. It most often involves the mitral valve. The Carrey-Cooms murmur of acute rheumatic fever is a sign of active mitral valvulitis. It is a soft, high pitched early diastolic murmur, varying from day to day. Isolated aortic valvular involvement is rare, while tricuspid and pulmonary valvular involvement is unusual. Pericarditis, pericardial effusion, arrhythmias (1st and 3rd degree heart blocks) are other features of rheumatic carditis. Pericarditis is manifested by characteristic chest pain, pericardial rub, typical ECG changes or pericardial fluid on echocardiography. Myocarditis manifests itself as disproportionate tachycardia, soft heart sounds, cardiomegaly or congestive cardiac failure. Congestive cardiac failure is the usual complication. Rheumatic heart disease is the only residual sequel of acute rheumatic fever.

- Polyarthritis: It occurs in almost 75% of the cases. It presents as red, swollen, warm and tender joints and is typically migratory in nature. Each joint is involved for not more than a week. Elbows, knees, ankles and wrist joints are most commonly involved, while fingers, toes or spine are rare sites of affection. Resolution of joint symptoms usually occurs after 6 weeks. There is polymorphonuclear leukocytosis in the synovial effusion. These changes do not produce chronic joint disease or deformity.

- Chorea: It is a late onset manifestation, occurring late as 3 months following throat infection. There are choreo-athetoid movements sometimes associated with emotional lability. At times, chorea may be the only symptom of acute rheumatic fever. It is seen in almost 20% of the patients, and lasts for weeks to months and rarely recurs. It is more common in boys than in girls.

- Erythema marginatum: Though unique, it is a very infrequent clinical finding seen in less than 10% of the patients. It presents as an evanescent, erythematous, non-tender, non-pruritic macular rash over the trunk. The macules have a pale centre and serpiginous borders. Patients with erythema marginatum usually have chronic carditis.

- Subcutaneous nodules: These are present as non-tender, firm pea-sized nodules seen on the extensor surfaces of the joints like knees, elbows and spine, and seen in less than 3% of the patients. There is usually chronic carditis in these patients.

**Minor criteria**

- Fever: It ranges from 101-102 degrees F.

- Arthralgia: It is diagnosed only in the absence of underlying arthritis.

**Evidence of group:** A streptococcal infections: It requires evidence of preceding streptococcal infection as confirmed by a positive throat culture, a history of scarlet fever, or elevated streptococcal antibodies such as antistreptolysin-O (ASO), antideoxyribonuclease-B (anti-DNAse-B) or antihyaluronidase (AH)

**Differential Diagnosis**

The diagnosis of acute rheumatic fever is based on a constellation of non-specific signs and symptoms. Hence, a variety of clinical entities need to be excluded.

**I. Polyarthritis due to other causes:**

a. Septic arthritis - Blood cultures should be taken to identify the organism.

b. Gonococcal arthritis - Therapeutic trial of penicillin may help in diagnosis of gonococcal infection.

c. Tuberculosis arthritis which is usually monoarticular.

d. Viral infections like rubella and hepatitis B

e. Juvenile idiopathic - It is characterised by a persistent type of arthritis with an eventual development of typical deformities.

f. Serum sickness - Penicillin administration can give rise to polyarthritis
g. Subacute bacterial endocarditis (SABE) in a pre-existing rheumatic heart disease which may be confused as recurrence of acute rheumatic fever. Raised ASO levels are helpful in excluding SABE.

h. Also one should consider and rule out the possibility of Henoch-Schonlein purpura, the inflammatory enteropathies such as ulcerative colitis and regional enteritis and haematologic diseases particularly sickle cell anaemia, haemophilia, and leukaemia

II. Rash: Other diseases where initial rash may be seen are SLE and Lyme disease. Lyme disease has presented diagnostic difficulties in children with arthritis in the endemic geographic areas. It most often has onset in the summer months and the characteristic rash, erythema chronicum migrans, occurs in half of the children coincident with the tick bite. Arthritis is delayed until 1 to 2 months after onset and is often a monoarthritis or oligoarthritis, occasionally polyarthritis occurs.\textsuperscript{20}

It is mandatory to withhold the administration of either steroids or salicylates till diagnosis is confirmed.

**LABORATORY DIAGNOSIS**

1. **Throat culture:** At the time of acute rheumatic fever, only 11% of the patients have a positive throat culture for group-A beta haemolytic streptococci.\textsuperscript{27} Certain rapid antigen detection kits are available which are specific but sensitivity is low.

2. **Streptococcal antibody test:** ASO titre is raised in 80% of the patients. Some important practical points about ASO test need to be noted. ASO titres vary with age, geographical area and other fevers influencing the frequency of streptococcal infection. A raised ASO titre merely indicates a recent streptococcal infection. The ASO titres are raised to their maximum 2-3 weeks after the streptococcal infection and rapidly fall in the next few months up to six months. Acute polyarthritis of rheumatic fever occurs at or close to the peak of the antibody response. Therefore, arthritis associated with a normal ASO titre is not in favour of the diagnosis of rheumatic fever.\textsuperscript{28} Anti-DNAase B and AH levels are important indicators of recent streptococcal infection. A high level or a rising level is significant.

3. **Acute phase reactants:** ESR, CRP are raised in almost all patients of carditis and arthritis and, sometimes in patients with chorea. ESR is useful in following the course of disease since the levels decline as rheumatic activity subsides.

4. **ECG:** Prolonged PR interval, though seen in all cases with carditis, is a non-specific finding. ECG may also show tachycardia, AV block, QRS-T changes suggestive of myocarditis.

5. **Chest radiography:** It is useful in assessing cardiac size. Pericarditis, pulmonary oedema and increased pulmonary vascularity are other findings which may be seen.

6. **Echocardiography:** It may show endocardial, myocardial and/or pericardial involvement. It is useful to diagnose valve affection.

---

**Table 3: Guidelines for bed test**

<table>
<thead>
<tr>
<th>Carditis status</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>No carditis</td>
<td>Bed rest for 2 weeks and gradual ambulation over 2 weeks even if no salicylates</td>
</tr>
<tr>
<td>Carditis with no cardiac enlargement</td>
<td>Bed rest for 4 weeks and gradual ambulation over 4 weeks</td>
</tr>
<tr>
<td>Carditis with cardiac enlargement</td>
<td>Bed rest for 6 weeks and gradual ambulation over 6 weeks</td>
</tr>
<tr>
<td>Carditis with heart failure</td>
<td>Strict bed rest as long as heart failure is present and gradual ambulation over 3 months</td>
</tr>
</tbody>
</table>

**MANAGEMENT**

It is considered under two headings i) Treatment and ii) Prevention

**TREATMENT**

1. **Treatment of group-A streptococcal infection**

   All patients with acute rheumatic fever should be treated for streptococcal infection at the time of diagnosis irrespective of isolation of the organism. Oral penicillin for 10 days or a single intramuscular injection of benzathine penicillin in a dose of 12,00,000 units can be given. Patients allergic to penicillin should be given erythromycin for 10 days.

2. **Treatment of clinical manifestations**

   Arthritis: Salicylates give prompt relief, however they must be withheld till complete definitive diagnosis is established as they may obscure the clinical findings, in place of which codeine or similar drugs can be administered. Aspirin in the dose of 100 mg/kg/day to attain a serum level of 20 mg% is required. The dose should be reduced gradually based on clinical and laboratory response (ESR, CRP). Original dose may be given again in the event of a relapse. A lack of response to full dose as aspirin, within 5 days warrants re-evaluation of diagnosis of RF.

   Carditis: In the absence of congestive cardiac failure, salicylates are beneficial, however corticosteroids are required if cardiac failure or severe carditis is present. Corticosteroids do not alter the subsequent development of rheumatic heart disease. Prednisolone in a dose of 1-2 mg/kg/day is given. Salicylates should be added during the last 4 weeks of corticosteroids when the dose of corticosteroid is being tapered, and then continued for another 3-4 weeks in order to prevent rheumatic rebound. The duration of salicylate therapy will depend upon the patient’s response. There is no evidence that non-steroidal anti-inflammatory drugs are more effective than steroids. Bed rest is indicated during an acute attack of rheumatic fever. The period of rest varies from 3 to 6 weeks, depending on severity of the carditis. Guidelines for bed rest are given in Table 3.

   The WHO expert committee (1988)\textsuperscript{28} states that acetylsalicylic acid (aspirin) is useful for the treatment of arthritis and fever. Corticosteroids have similar effects but in general should be reserved for treatment of serious or life threatening carditis. Both agents will suppress acute
Table 4: Prophylaxis of Rheumatic Fever

<table>
<thead>
<tr>
<th>Prophylaxis</th>
<th>Drug</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary prophylaxis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intramuscular</td>
<td>Benzathine penicillin G</td>
<td>12,00,000 U (600,000 U, if weight less than 27 kgs) once daily</td>
</tr>
<tr>
<td>Oral</td>
<td>Penicillin V</td>
<td>500mg bid daily for 10 days</td>
</tr>
<tr>
<td>Erythromycin</td>
<td></td>
<td>250mg qid daily for 10 days</td>
</tr>
<tr>
<td>Others (Clindamycin, nafcillin, ampicillin, cephalaxin)</td>
<td>Dose varies</td>
<td></td>
</tr>
<tr>
<td><strong>Secondary prophylaxis</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intramuscular</td>
<td>Benzathine penicillin G</td>
<td>12,00,000 U every 3-4 weeks</td>
</tr>
<tr>
<td>Oral</td>
<td>Penicillin V</td>
<td>250mg bid daily</td>
</tr>
<tr>
<td>Sulfadiazine</td>
<td></td>
<td>1 gm od (0.5gm od in children)</td>
</tr>
<tr>
<td>Erythromycin stearate</td>
<td></td>
<td>250mg bid daily</td>
</tr>
</tbody>
</table>

Phase reactants during use but there is no evidence that either will shorten the period of rheumatic activity or limit valve damage. When required for symptoms, the doses should be reduced gradually as soon as the symptoms permit. That subsequent development of RHD does not depend upon whether aspirin, steroids or ACTH was used during the attack of acute RF was proved by the UK US cooperative study (1965). Hence as has been emphasized by several authorities, to prevent RHD in a patient who has had acute RF, the emphasis should be on secondary prophylaxis. The duration of treatment in rheumatic fever without carditis varies from patient to patient and is usually up to few weeks in tapering doses. According to Braunwald, the initial dose of acetyl salicylic acid should be continued until a satisfactory clinical response is obtained i.e. until there is complete relief of symptoms and signs of arthritis and temperature have returned to normal range. Thereafter doses may be reduced to 2/3 of the initial doses and may be maintained until all laboratory manifestations of inflammatory disease has returned to normal. For the remainder of the course of therapy, the dose may be reduced to half the initial daily dose. Should clinical and laboratory evidence of relapse occur, it is advisable to go back to higher doses.

Sydenham chorea: In mild cases, diazepam is sufficient, however in severe cases, haloperidol may be required.

Skin manifestations: These do not require any medical therapy.

3. Treatment of the complications

Congestive cardiac failure is managed by the conventional methods.

Prevention of Rheumatic Fever (Table 4)

The Indian Council of Medical Research (ICMR) studies on primary, secondary and seasonal prophylaxis have shown the efficacy of these methods. As regards primary prevention, it is very difficult as it has to be given for all cases of sore throat in whom streptococci are isolated but since isolation of streptococci is almost impossible before treatment, primary prevention is very difficult and not in practice. Secondary prophylaxis is the cornerstone of prevention of rheumatic fever.

In 1966 when prevalence of rheumatic fever was significant, Government of India had included rheumatic fever in the fourth five year plan. However in subsequent five year plans, it was dropped. The ICMR has carried out six, nationwide research projects on rheumatic fever and rheumatic heart disease between 1966 and 1990 but due to lack of enthusiasm of governments (central and state), these programmes have not become popular. Public education is being carried out though videos in English and vernacular languages by ICMR and All India Heart Foundation, New Delhi. These are available on national net work as well. Because of this public health education, tablets and injections of penicillin are being given freely to school children. Unfortunately there is ban on administering penicillin injections in government hospitals in some Indian states. Still lot is desired to be done in some of the troubled and under developed states such as Bihar, UP, MP, North Eastern regions and Jammu and Kashmir state.

Krishnaswami et al in their follow up of 2340 adult patients above 26 years of age with rheumatic valvular disease seen since 1987, discontinued inj. benzathine penicillin (once in 3 weeks) and all the patients were followed for an evidence of rheumatic fever for 1-9 years period (mean 3 years). Recurrence of rheumatic fever was recorded in 5 patients (0.21%) showing that recurrence of rheumatic fever in patients with RHD over 26 years of age is very low. Hence long term chemo-prophylaxis to patients above 26 years age, should be decided on individual basis.

Finally, rheumatic fever (RF) vaccine, ICMR is working actively to produce RF vaccine. ICMR has initiated Jal Vigyan Mode Project at Chandigarh and Vellore where the development of RF vaccine is in progress. Vaccine is being prepared using Indian strains of A streptococci. Also ICMR has started RF registries for secondary prophylaxis at those centres and has plans to extend these registries to other parts of the country.

It is now 100 years that we have encountered RF/RHD and this period is a long time for a disease of infectious origin to continue. Although RF has almost disappeared from western countries but it may recur again as it occurred in USA in 1987. It still continues to be a problem in Asia, Africa and South America. Whether in the new millennium, the disease will vanish because of less virulence of streptococci and the possibility of RF vaccine in the near future is yet to be seen. The main question is whether RF and RHD in India has declined significantly that we can ignore this problem. The answer is no. Since the coronary artery disease, hypertension have come to India in epidemic proportion, the hospitals (private, public) and cardiologists are concentrating on management of these diseases. Hence RHD is receiving less publicity and attention. The bottom line is that we should detect RHD early and should carry out secondary prophylaxis. If a safe RF vaccine becomes available, it will be a boon for control of the disease.

REFERENCES