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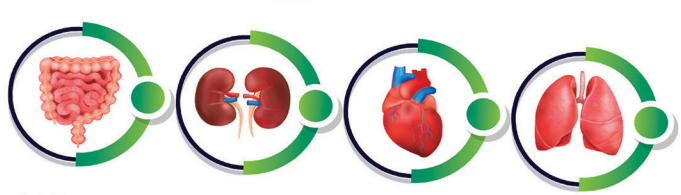

Editor-in-Chief
Jyotirmoy Pal

Editor
Nandini Chatterjee

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Editor-in-Chief

Milind Y Nadkar MD FICP FACP
President-Elect
Association of Physicians of India
Professor and Head
Department of Medicine and
Chief of Rheumatology Services
Seth GS Medical College and KEM Hospital
Mumbai, Maharashtra, India

Foreword

KK Pareek

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2

CHAPTER

Rheumatic Fever: What's New in Diagnosis and Management?

Monika Maheshwari, Vishwas Galav

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ABSTRACT

Acute rheumatic fever (ARF) is an immune-mediated disease caused by group A *Streptococcus* (GAS) infection, causing pharyngitis in children and adults. Repeated or severe ARF can lead to rheumatic heart disease (RHD), causing high morbidity and mortality. ARF and RHD are prevalent in regions with low income and limited healthcare access. The pathogenesis of ARF and RHD is poorly understood, and no immune-modulating therapies exist. Pro-inflammatory cytokines and chemokines, such as interleukin 6 (IL-6), IL-8/CXCL8, and tumor necrosis factor alpha (TNF-α), are implicated in ARF and RHD pathogenesis.

INTRODUCTION

Acute rheumatic fever (ARF) is an immune-mediated disease that occurs as a late sequelae to group A *Streptococcus* (GAS) infection. It accounts for 15–30% cases of pharyngitis among children and 10% cases among adults. Repeated or severe ARF leads to rheumatic heart disease (RHD), with high morbidity and mortality. ARF and its complication RHD cause a large burden of morbidity and mortality with an estimate 40.5 million people living with RHD and causing 3.06 lakhs deaths annually. In many high-income countries, improvements in living conditions and the introduction of antimicrobial drugs to treat GAS pharyngitis have nearly eliminated ARF. High prevalence rates of ARF and RHD continue to be reported from several geographic regions around the world with low income and limited access to healthcare such as Southeast Asia, the Western Pacific, and Africa.

The pathogenesis of ARF and RHD is poorly understood, and there are currently no immune-modulating therapies available to stop the progression of ARF to RHD. Cytokines and chemokines are immune-signaling molecules that are critical to the development of autoimmune diseases. Several studies point to a role for pro-inflammatory cytokines and chemokines in ARF and RHD pathogenesis, in particular interleukin (IL)-6, IL-8/CXCL8, and tumor necrosis factor alpha (TNF-α), which are elevated in circulation in both ARF and RHD patients. Histological studies also implicate T helper 1 (Th1)- and Th17-associated proinflammatory cytokines, chemokine CXCL9, and the fibrosis-associated cytokine Transforming growth factor beta (TGF-β) in progressive cycles of inflammatory damage and fibrotic repair. All of this suggests immune molecules contribute to both the acute inflammatory disease stage of ARF as well as cardiac remodeling and valve dysfunction in RHD.

DIAGNOSIS OF RHEUMATIC FEVER

The Jones criteria have been used for guidance in the diagnosis of ARF since 1944 and were modified by the American Heart Association (AHA) in 1992. The prominent role played by echocardiography in the diagnosis of heart disorders which have evolved over the past two decades which lead to identification of newer entity called 'subclinical carditis'. All these changes lead to further revision of Jones criteria in 2015 for the diagnosis rheumatic fever (RF). The 2020 Australian guideline for prevention, diagnosis, and management of ARF and RHD takes into consideration the 2015 revised Jones criteria, but it is more specific for Australian population.

2015 AHA-REVISED JONES CRITERIA FOR DIAGNOSIS OF ARF

For all patient population with evidence of preceding GAS infection **(Table 1)**

Diagnosis: Initial ARF—2 major manifestations *or* 1 major plus 2 minor

Diagnosis: Recurrent ARF—2 major *or* 1 major and 2 minor *or* 3 minor

To differentiate between low-risk and high-risk population the following criteria may be utilized:

- If a person comes from an environment or group where low rates of ARF or RHD are known to occur, it is reasonable to assume that they are at low risk for ARF (class IIa; level of evidence C).
- It is reasonable that low risk be defined as having an all-age prevalence of RHD of ≤ 1 per 1,000 population per year or an ARF incidence of < 2 per 100,000 school-aged children (typically 5–14 years old) each year where trustworthy epidemiological data are available (class IIa; level of evidence C).
- Depending on their reference population, children who are not obviously from a low-risk demographic are at moderate to high risk (class I; level of evidence C).

TABLE 1: American Heart Association (AHA)-revised Jones criteria 2015 for diagnosis of acute rheumatic fever (ARF).

Low-risk populations	Moderate- and high-risk populations
Major criteria	
Carditis (clinical or subclinical)	Carditis (clinical or subclinical)
• Arthritis (polyarthritis only) • Chorea	Arthritis (including polyarthritis, monoarthritis, or polyarthralgia)
Erythema marginatum	Chorea
Subcutaneous nodules	• Erythema marginatum • Subcutaneous nodules
Minor criteria	
Polyarthralgia	Monoarthralgia
Fever (≥ 38.5°C)	Fever (≥ 38°C)
ESR ≥ 60 mm in the first hour and/or CRP ≥ 3.0 mg/dL	ESR ≥ 30 mm in the first hour and/or CRP ≥ 3.0 mg/dL
Prolonged PR interval, after accounting for age variability (unless carditis is a major criterion)	Prolonged PR interval, after accounting for age variability (unless carditis is a major criterion)

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

ADVANCES IN UNDERSTANDING OF RF/RHD PATHOGENESIS

The precise mechanisms that underlie this intricate process are still not fully understood. According to what we know, the immune responses ARF and RHD are brought on by GAS pharyngitis. But more recently, RHD has been linked to other superficial streptococcal diseases such pyoderma and impetigo. Additionally, there is growing evidence that suggests the pathophysiology of RHD may involve group C and group G *Streptococcus*.

Molecular mimicry is the most widely accepted theory regarding the pathogenesis of RF/RHD; however, more recent research points to a more intricate series of events, including the possibility of collagen-associated neoantigens and additional mechanisms that may contribute to pathology, such as epitope spreading and T cell receptor (TCR) degeneration. Furthermore, some natural killer (NK) cells, CD4- CD8- T cells, and CD4+ T cells may also have a significant role in the destruction of tissue in RHD.

ADVANCES IN UNDERSTANDING OF THE GENETIC PREDISPOSITION TO ARF

Acute rheumatic fever occurs in about 3–6% in GAS-endemic populations, and the genetic risk for RHD is thought to be complex. This theory has been validated by historical research on RHD twins, and more recently, genetic investigation has provided fresh insights into the etiology and propensity of RHD.

Human leukocyte antigen (HLA) molecules, which are encoded by genes on chromosome 6, have been linked to a predisposition to develop RF/RHD through genetic connections in RHD. Class II *HLA* genes have been extensively documented, with most of them being associated with HLA-DR7 and HLA-DR4.

There is evidence linking polymorphisms in the *IL-2*, *IL-4*, *IL-6*, and *IL-10* genes to clinical illness, and IL-4 has been proven to have value in differentiating between latent and clinical RHD. Higher levels of IL-10 and IL-4 indicated unfavorable clinical outcomes, while IL-4, IL-8, and IL-1RA also appeared to predict the transition from latent to clinical disease. In people with advanced RHD, co-regulated production of IL-6 and TNF-α is associated with severe valvular dysfunction.

Genome-wide association studies (GWAS) have been the most significant development in the field of RHD genetics. When single-nucleotide polymorphism (SNP) distributions in patients and selected controls are compared, GWAS can evaluate a large number of gene variants in complicated disorders.

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Group A carbohydrate, N-acetyl-β-D-glucosamine-specific immunoglobulin G2 (IgG2) may be an important autoantibody in initial stages of the pathogenesis of streptococcal sequelae, emerging as a future candidate for a biomarker in early disease stages.

ADVANCES IN MANAGEMENT

Primordial Prevention

A number of factors, including ventilation, wetness, and home quality, are linked to a 1.7–3.6-fold greater risk of infection. These factors include household crowding. First-degree relatives of people with clinical RHD may be more likely to have RHD because of their similar socioeconomic circumstances. Primordial prevention approach is being tried in Australia.

Multivalent M protein-based vaccines, M protein vaccines with conserved C-repeat epitopes, cell wall carbohydrate vaccines, and non-M protein multi-component vaccines are possible options for vaccination against GAS, but none have shown encouraging results so far.

Primary Prevention

Nucleic acid amplification tests (NAATs) like illumigene assay for GAS pharyngitis have been explored and their sensitivity and specificity are estimated to be in excess of 99%, but their use is restricted by high cost.

Electrochemical detection which uses DNA is an affordable and effective method for diagnosing GAS; its results are available in 30 minutes and specificity is nearly 100%.

The Strepic® device (under development), has been especially developed as an effective, affordable, and commercially feasible autofluorescence-based diagnostic test.

Treatment of Group A Strep Pharyngitis

Benzathine Penicillin G is the gold standard for treatment of group A streptococcal infection. Streptococcal carriers are found in many communities, therefore, treatment is recommended only for acute streptococcal infection.

Secondary Prevention

The modified Jones criteria 2015 addressed two significant features: (1) distinguished criteria between low-risk and moderate-to-high risk populations based on ARF incidence or RHD prevalence; and (2) recommending echocardiography to suspected cases of ARF and incorporating subclinical carditis as evidenced by echocardiography as a major criteria.

The GOAL study demonstrated that subsequent antibiotic prophylaxis lowers the risk of disease progression at 2 years in children with latent RHD aged 5–17. This shows that echo screening and active case detection is an effective method for the management of RHD **(Table 2)**.

TABLE 2: Drug treatment of acute rheumatic fever.

Indication	Drug options listed in order of preference	Comment
Eradication of inciting streptococcal infection	• Benzathine benzylpenicillin G 1,200,000 units (child < 20 kg: 600,000 units, ≥ 20 kg: 1,200,000 units) intramuscularly, single dose, OR • Phenoxymethylpenicillin 500 mg (child: 15 mg/kg up to 500 mg) orally, every 12 hours for 10 days, OR • For patients with penicillin hypersensitivity (nonsevere): cefalexin 1 g (child: 25 mg/kg up to 1 g) orally, every 12 hours for 10 days, OR • For patients with immediate penicillin hypersensitivity: azithromycin 500 mg (child: 12 mg/kg up to 500 mg) orally, daily for 5 days	• Streptococcal infection may not be evident by the time acute rheumatic fever manifests (e.g., cultures often negative), but eradication therapy for possible persisting streptococci is recommended • Intramuscular penicillin is preferred as streptococcal eradication therapy due to better adherence and its subsequent ongoing use in secondary prophylaxis • Between 3 and 30% of group A *Streptococcus* isolates internationally are resistant to macrolide antibiotics (e.g., azithromycin)
Initial analgesia while awaiting diagnostic confirmation: • Mild-to-moderate pain • Severe pain	• Paracetamol 1,000 mg (in children: 15 mg/kg) orally, every 4 hours as needed up to a maximum of 60 mg/kg/day or 4,000 mg/day	• Initial analgesia is preferred during diagnostic uncertainty to avoid the masking effect that anti-inflammatory use can have on migratory joint symptoms, fever, and concentrations of inflammatory markers

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Indication	Drug options listed in order of preference	Comment
	• Tramadol immediate-release 50–100 mg (in children: 1–2 mg/kg) orally, every 4 hours as needed up to a maximum of 400 mg/day	• Tramadol (or codeine) is usually avoided in children < 12 years of age due to variable metabolism. Use only when strong analgesia is essential and cautious monitoring is available
Symptomatic management of arthritis/arthralgia after confirmation of acute rheumatic fever diagnosis	• Naproxen immediate-release 250–500 mg (in children: 10–20 mg/kg/day) orally twice daily, up to a maximum of 1,250 mg daily, OR • Ibuprofen 200–400 mg (in children: 5–10 mg/kg) orally three times daily, up to a maximum of 2,400 mg daily, OR • Aspirin 50–60 mg/kg/day orally, in 4–5 divided doses in adults and children; dose can be escalated up to a maximum of 80–100 mg/kg/day in 4–5 divided doses	• Naproxen may be safer than aspirin and convenient due to twice-daily dosing and the availability of oral suspension • Ibuprofen is well-tolerated and readily available, but there are less data and experience with its use for acute rheumatic fever than those associated with naproxen • The dose of NSAIDs needed for acute rheumatic fever is generally higher than the dose recommended for other conditions; therefore, it may be appropriate to start at the higher dose range • Due to the rare possibility of Reye's syndrome in children, aspirin may need to be discontinued during intercurrent acute viral illness; thus, influenza vaccination is strongly recommended to reduce the likelihood of this case
Symptomatic management of moderate to severe chorea	• Carbamazepine 3.5–10 mg/kg per dose orally twice daily • Sodium valproate 7.5–10 mg/kg per dose orally twice daily	Treatment of Sydenham chorea should be considered if movements interfere substantially with normal activities
Symptomatic management of very severe chorea or chorea paralytica	In addition to an anticonvulsant drug, consider adding a corticosteroid: • Prednisolone 1–2 mg/kg up to a maximum of 80 mg orally once daily	
Symptomatic management of carditis	Pediatric dosing: • Furosemide (frusemide) 1–2 mg/kg orally as a single dose, then 0.5–1 mg/kg (to a maximum of 6 mg/kg) orally every 6–24 hours • Spironolactone 1–3 mg/kg (initially) up to 100 mg orally in divided doses daily; round dose to a multiple of 6.25 mg (a quarter of a 25 mg tablet)	Treatment of heart failure may be required for severe, acute carditis. Seek advice from a specialist cardiologist
	Enalapril 0.1 mg/kg orally in 1 or 2 divided doses daily, increased gradually over 2 weeks to a maximum of 1 mg/kg orally in 1 or 2 divided doses daily; alternative ACE inhibitors: captopril, lisinopril	The choice of ACE inhibitor will vary depending on the clinical situation. Seek advice from a specialist cardiologist
	Adult dosing: • Furosemide (frusemide) 20–40 mg orally or intravenously as a single dose followed by 20–40 mg orally or intravenously every 8–12 hours. Ongoing dose adjustment is based on clinical progression and renal function • Spironolactone may be added for patients with limited or no response to loop diuretic; 12.5–200 mg orally once daily with dose escalation based on clinical and electrolyte responses	The management of acute carditis follows the same principles as those for the management of acute heart failure. This table provides a guide to the initial management of acute heart failure due to acute carditis in adults. Seeking advice from a specialist cardiologist early is strongly recommended

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Indication	Drug options listed in order of preference	Comment
	• Nitrate therapy may be added for patients with limited or no response to diuretic therapy and systolic blood pressure greater than 90 mmHg. Intravenous or topical glyceryl trinitrate may be used • ACE inhibitor therapy with perindopril or ramipril is recommended in patients with moderate or severe left ventricular systolic dysfunction, unless contraindicated	
Disease-modifying (immunomodulatory) treatments	Prednisolone 1–2 mg/kg up to a maximum of 80 mg orally, once daily	Considered for use in selected cases of severe carditis, despite meta-analyses in which the overall benefit was not evident
Secondary prophylaxis	• Benzathine benzylpenicillin G by deep intramuscular injection 1,200,000 units (≥20 kg) or 600,000 units (<20 kg), OR • Phenoxymethylpenicillin (penicillin V) 250 mg orally twice daily, OR • For patients with penicillin hypersensitivity (nonsevere) or immediate penicillin hypersensitivity: erythromycin 250 mg orally twice daily	• Every 28 days • Every 21 days for selected groups • Intramuscular penicillin is preferred due to greater effectiveness in head-to-head trial and better adherence

(ACE: angiotensin-converting enzyme; NSAIDs: nonsteroidal anti-inflammatory drugs)

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Milind Y Nadkar MD(MEDICINE) FICP FACP is Professor and Head, Department of Medicine at Seth GS Medical College and KEM Hospital, Mumbai, Maharashtra, India. His areas of interest are Rheumatology, Infectious Diseases, and Tropical Medicine. He is President-Elect, Association of Physicians of India, and Chairman, Scientific Committee for APICON 2024 being held at New Delhi. He was formerly Honorary General Secretary and Honorary Treasurer of API. He was Editor-in-Chief of Journal of the Association of Physicians of India (JAPI) for over 7 years from 2014 to 2021. He has a very long association with JAPI, with almost 25 years of experience on the Editorial Board of JAPI. He is also associated with API Textbook of Medicine from 7th edition onward and is the editor of the book's latest 12th edition. He has over 200 publications in various national and international journals and books. He is also associated with Golwalla's Medicine for Students and Davidson's Principles and Practice of Medicine. He has edited many Medicine Updates over the years. He is also associated with many guidelines and monographs published by the API.

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