

# Rheumatic Fever: A Comprehensive Review

## Abstract

Rheumatic fever (RF) is a complex autoimmune inflammatory disorder triggered by inadequately treated group A Streptococcus (GAS) infections, primarily streptococcal pharyngitis. This review provides a comprehensive overview of RF, spanning its pathophysiology, clinical manifestations, diagnosis, treatment, and prevention strategies. RF's pathogenesis involves molecular mimicry between GAS antigens and host tissues, leading to immune-mediated damage primarily affecting the heart, joints, skin, and central nervous system. Clinically, RF presents with fever, migratory polyarthritides, carditis, subcutaneous nodules, and Sydenham's chorea. Carditis, characterized by valvulitis, is the most severe complication, with repeated episodes potentially resulting in rheumatic heart disease (RHD) and cardiac dysfunction. Accurate diagnosis relies on clinical criteria established by the Jones criteria, complemented by laboratory tests and echocardiography to assess cardiac involvement. Treatment goals include prompt antibiotic therapy to eradicate the streptococcal infection, symptom management with anti-inflammatory agents, and prevention of recurrences. Long-term antibiotic prophylaxis is imperative for individuals with a history of RF or RHD. In advanced RHD cases, cardiac surgery or valve replacement may be necessary. Preventing RF necessitates early diagnosis and treatment of streptococcal infections, reinforced by public health measures and improved healthcare access. Education campaigns promoting adherence to antibiotic regimens and awareness of RF's consequences are essential in reducing the global burden of this preventable but persistent disease.

**Keywords:** Rheumatic fever • Streptococcal infection • Adolescents

## Introduction

Rheumatic fever (RF) is an autoimmune inflammatory condition that can result from inadequately treated streptococcal infections, particularly in children and adolescents. This Editorial aims to provide a comprehensive overview of RF, encompassing its pathophysiology, clinical manifestations, diagnosis, treatment, and prevention strategies. Diagnosis involves a meticulous evaluation, combining clinical criteria and laboratory tests. Early detection is crucial to initiate timely treatment and prevent cardiac complications. Treatment focuses on eradicating the streptococcal infection, managing symptoms, and preventing recurrences. Antibiotics, anti-inflammatory drugs, and, in severe cases, corticosteroids play vital roles [12].

Prevention is at the heart of RF control. Swift identification and treatment of streptococcal

throat infections can prevent initial RF episodes. Long-term antibiotic prophylaxis is essential for individuals with a history of RF or rheumatic heart disease (RHD) [2]. Public health efforts, such as improved hygiene and healthcare access, are integral in reducing RF's global burden. Ultimately, RF's persistence emphasizes the need for continued research, education, and awareness campaigns. With a concerted global effort to implement preventive measures, increase access to healthcare, and bolster public health infrastructure, we can envision a future where RF and its devastating consequences are relegated to the annals of medical history, allowing children and communities to thrive without the specter of this preventable disease looming over their lives [3].

## Pathophysiology

RF arises as a delayed immune response to

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group A *Streptococcus* (GAS) infections, primarily streptococcal pharyngitis. Molecular mimicry between GAS antigens and host tissues triggers an autoimmune reaction. Immune-mediated damage predominantly targets the heart, joints, skin, and central nervous system. The formation of cross-reactive antibodies, especially against cardiac antigens like myosin and valvular tissue, plays a central role in the pathogenesis of RF [4, 5].

#### Clinical manifestations

RF's clinical presentation varies widely but commonly includes fever, migratory polyarthritis, carditis, subcutaneous nodules, and Sydenham's chorea. Carditis, which manifests as valvulitis, poses the most severe and long-term complication. Rheumatic heart disease (RHD) develops as a consequence of repeated RF episodes, potentially leading to valve deformities and cardiac dysfunction. Early diagnosis is crucial to initiate prompt treatment and prevent irreversible cardiac damage [6].

#### Diagnosis

Diagnosing RF involves a combination of clinical criteria established by the Jones criteria, supported by laboratory tests such as throat cultures, anti-streptolysin O (ASO) titers, and C-reactive protein (CRP) levels. Echocardiography helps assess cardiac involvement. Physicians must consider both major and minor criteria to make an accurate diagnosis, as RF can mimic other diseases. A thorough evaluation and collaboration between clinicians and cardiologists are essential [7].

#### Treatment

The primary goals of RF treatment are to eradicate the streptococcal infection with antibiotics, manage symptoms, and prevent recurrences. Penicillin or other appropriate antibiotics are administered to clear the infection. Anti-inflammatory agents, such as nonsteroidal anti-inflammatory drugs (NSAIDs), are prescribed to alleviate joint pain and inflammation. In severe cases of carditis, corticosteroids may be necessary. Patients with RHD require long-term antibiotic prophylaxis to prevent further GAS infections and RF recurrences. Cardiac surgery or valve replacement may

be necessary for advanced RHD cases [8].

#### Prevention

Preventing RF involves promptly diagnosing and treating streptococcal throat infections to prevent initial RF episodes [9]. Public health measures, including improved hygiene and access to healthcare, are crucial. Long-term antibiotic prophylaxis for individuals with a history of RF or RHD is vital to prevent recurrences. Education and awareness campaigns are essential to promote adherence to antibiotic regimens and prevent RHD, a condition that remains a significant global health burden [10].

#### Conclusion

Rheumatic fever remains a preventable but persistent health concern, particularly in resource-limited settings. Timely diagnosis and treatment of streptococcal infections, adherence to antibiotic prophylaxis, and improved public health infrastructure are critical in reducing the incidence of RF and its devastating complications, ultimately paving the way for a healthier future, especially for vulnerable populations. In conclusion, rheumatic fever (RF) remains a challenging and preventable autoimmune condition with potentially severe and long-lasting consequences, primarily affecting children and adolescents. This review highlights the critical aspects of RF, including its pathophysiology, clinical manifestations, diagnosis, treatment, and prevention.

RF's pathogenesis is rooted in molecular mimicry between group A *Streptococcus* (GAS) antigens and host tissues, leading to an autoimmune response. Clinical presentations vary, encompassing fever, joint inflammation, carditis, skin manifestations, and neurological involvement. Carditis, with its potential for chronic heart valve damage, stands as a significant concern.

#### Acknowledgment

None

#### Conflict of Interest

None

**References**

1. Andrew NH, Sladden N, Kearney DJ *et al.* An analysis of IgG4-related disease (IgG4-RD) among idiopathic orbital inflammations and benign lymphoid hyperplasias using two consensus-based diagnostic criteria for IgG4-RD. *Br J Ophthalmol.* 99, 376-381 (2015).
2. Kobayashi S, Yoshida M, Kitahara T *et al.* Autoimmune pancreatitis as the initial presentation of systemic lupus erythematosus. *Lupus.* 16, 133-136 (2007).
3. Zaarour M, Weerasinghe C, Eter A *et al.* An Overlapping Case of Lupus Nephritis and IgG4-Related Kidney Disease. *J Clin Med Res.* 7, 575-581 (2015).
4. Wada Y, Matsuo K, Ito Y *et al.* A Case of Concurrent IgG4-Related Kidney Disease and Lupus Nephritis, in: IgG4-Related Kidney Disease. 303-311 (2016).
5. Chang LC, Wu CH, Hsu CL *et al.* IgG4-Related Lung Disease in a Patient with Systemic Lupus Erythematosus: A Case Report. *Vol.* 33, 6 (2008).
6. Deshpande V, Zen Y, Chan JK *et al.* Consensus statement on the pathology of IgG4-related disease. *Mod Pathol Off JUS Can Acad Pathol Inc.* 25, 1181-1192 (2012).
7. Khosroshahi A, Wallace ZS, Crowe JL *et al.* Second International Symposium on IgG4-Related Disease, International Consensus Guidance Statement on the Management and Treatment of IgG4-Related Disease. *Arthritis Rheumatol Hoboken NJ.* 67, 1688-1699 (2015).
8. Lin W, Lu S, Chen H *et al.* Clinical characteristics of immunoglobulin G4-related disease: a prospective study of 118 Chinese patients. *Rheumatology.* 54, 1982-1990 (2015).
9. Vidarsson G, Dekkers G, Rispens T. IgG Subclasses and Allotypes: From Structure to Effector Functions. *Front Immunol.* 5, 520 (2014).
10. Chiu ML, Goulet DR, Teplyakov A *et al.* Antibody Structure and Function: The Basis for Engineering Therapeutics. *Antibodies* 8, 55 (2019).