INTERNATIONAL MULTI DISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
eISSN 2394-6334 Impact factor: 8.854 Volume 13, issue 05 (2026)

RHEUMATIC HEART DISEASE IN CHILDREN AND AORTIC VALVE CHANGES: PATHOPHYSIOLOGICAL AND CLINICAL PERSPECTIVES

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https://doi.org/10.5281/zenodo.20443397

Abstract: Rheumatic heart disease (RHD) remains a significant cause of acquired cardiovascular morbidity in children, particularly in low- and middle-income regions. It develops as a chronic sequela of acute rheumatic fever following untreated or inadequately treated group A streptococcal infection. Although mitral valve involvement is most common, aortic valve pathology is frequently observed and contributes to progressive hemodynamic deterioration. This article reviews the pathophysiological mechanisms, echocardiographic features, and clinical implications of aortic valve changes in pediatric rheumatic heart disease. Emphasis is placed on early diagnosis, Doppler echocardiographic assessment, and long-term management strategies aimed at preventing irreversible valvular damage.

Keywords: rheumatic heart disease, children, aortic valve, aortic regurgitation, echocardiography, valvular heart disease.

Introduction. Rheumatic heart disease is a chronic inflammatory condition resulting from an autoimmune response to Streptococcus pyogenes infection. In children, it remains a preventable yet persistent cause of valvular heart disease. The disease primarily affects the endocardium, leading to progressive valve thickening, fibrosis, and deformity.

While the mitral valve is most frequently involved, the aortic valve is affected in a substantial proportion of cases. Aortic involvement often appears in combination with mitral valve disease and is associated with worse clinical outcomes. Early identification of aortic valve changes in pediatric patients is essential for preventing long-term complications such as heart failure and pulmonary hypertension.

Pathophysiology of Aortic Valve Involvement

The development of rheumatic aortic valve disease is primarily initiated by an abnormal autoimmune response following infection with group A β-hemolytic streptococcus. A key mechanism underlying this process is molecular mimicry, where streptococcal antigens share structural similarities with human cardiac proteins, particularly myosin and valvular endothelial components. This immunological cross-reactivity triggers a cascade of inflammatory reactions that progressively damage cardiac structures.

The early stage of disease is characterized by endothelial activation and immune cell infiltration of the valvular tissue. This is followed by the release of pro-inflammatory cytokines, leading to chronic inflammation and progressive structural remodeling of the valve. As the inflammatory process continues, the aortic valve leaflets undergo thickening due to fibroblast

proliferation and collagen deposition. The commissures become fibrotic and rigid, resulting in reduced leaflet mobility and impaired coaptation.

Unlike degenerative aortic valve disease observed in older adults, which is primarily driven by calcification and mechanical wear, rheumatic involvement in children is predominantly inflammatory and fibrotic in nature. Recurrent episodes of rheumatic fever further intensify valvular injury, accelerating the progression toward chronic aortic regurgitation. Over time, leaflet retraction and deformation lead to progressive valve incompetence and significant hemodynamic burden on the left ventricle.

Echocardiographic Features

Echocardiography remains the cornerstone diagnostic modality for identifying and monitoring rheumatic aortic valve disease in pediatric patients. It allows real-time evaluation of both structural abnormalities and functional impairment of the aortic valve.

From a morphological perspective, typical findings include progressive thickening of the aortic valve leaflets, which is often most prominent at the free edges. The cusps may exhibit restricted mobility due to fibrotic changes, resulting in incomplete opening during systole and incomplete closure during diastole. Although commissural fusion is more characteristic of mitral valve involvement, it may also be present in advanced aortic valve disease. In chronic and severe cases, dilation of the aortic root may develop as a secondary consequence of long-standing volume overload and altered hemodynamic stress.

Doppler echocardiography provides critical functional information. The hallmark finding is a diastolic regurgitant jet directed from the aorta back into the left ventricle. The severity of regurgitation is assessed by jet width, vena contracta, and regurgitant volume. In moderate to severe disease, there is a noticeable increase in regurgitant flow, along with a reduction in pressure half-time, reflecting rapid equilibration of diastolic pressures between the aorta and left ventricle. Color Doppler imaging plays a particularly important role in the early detection of mild aortic regurgitation, often identifying hemodynamically significant lesions before clinical symptoms become apparent.

Clinical Manifestations

In pediatric patients, rheumatic aortic valve involvement often remains clinically silent during the early stages of disease. As valvular dysfunction progresses, subtle symptoms begin to emerge, reflecting increasing hemodynamic burden on the cardiovascular system.

Common clinical features include persistent fatigue, decreased physical endurance, and reduced tolerance to physical activity. As left ventricular volume overload worsens, children may develop palpitations due to compensatory tachycardia and increased myocardial workload. Dyspnea on exertion becomes more prominent as pulmonary circulation is affected by rising left ventricular filling pressures.

On physical examination, the most characteristic finding is a high-pitched, blowing early diastolic murmur best heard along the left sternal border. In more advanced cases, additional signs such as a widened pulse pressure, bounding peripheral pulses, and visible cardiac hyperdynamic activity may be present, reflecting significant aortic regurgitation and increased stroke volume.

Hemodynamic Consequences

Chronic aortic regurgitation results in persistent volume overload of the left ventricle, which must accommodate both the normal pulmonary venous return and the regurgitated blood from the aorta during diastole. Initially, the heart compensates through adaptive mechanisms, including eccentric left ventricular hypertrophy and chamber dilation. These changes help maintain stroke volume and preserve cardiac output despite the increased volume load.

However, with ongoing disease progression, these compensatory mechanisms become maladaptive. Continuous ventricular dilation leads to increased wall stress, myocardial oxygen demand, and eventual contractile dysfunction. As left ventricular compliance decreases, end-diastolic pressure rises, resulting in backward transmission of pressure into the pulmonary circulation.

Clinically, this progression manifests as pulmonary congestion, exertional dyspnea, and eventually signs of congestive heart failure in advanced stages. If left untreated, chronic volume overload leads to irreversible myocardial remodeling and significant deterioration in long-term cardiac function.

Management Strategies. The management of rheumatic aortic valve disease in children requires a comprehensive approach that includes prevention of disease progression, medical stabilization, and surgical intervention when necessary.

1. Secondary prevention. A cornerstone of management is the prevention of recurrent rheumatic fever episodes, which significantly contribute to progressive valvular damage. Long-term intramuscular benzathine penicillin prophylaxis is recommended to eliminate recurrent streptococcal infections. Patient adherence to prophylactic therapy is essential, as each recurrence of rheumatic fever can substantially worsen valve pathology.

2. Medical management. Pharmacological treatment is primarily aimed at controlling symptoms and reducing hemodynamic stress on the left ventricle. Diuretics are used in patients with signs of volume overload and heart failure to reduce pulmonary congestion and improve clinical status. In selected cases, vasodilator therapy may be beneficial by decreasing afterload and improving forward cardiac output. Regular echocardiographic monitoring is essential to evaluate ventricular function, assess progression of regurgitation, and guide treatment decisions over time.

3. Surgical intervention. Surgical management is reserved for patients with severe aortic regurgitation who exhibit refractory symptoms, progressive left ventricular dilation, or declining systolic function despite optimal medical therapy. Depending on the severity and anatomical condition of the valve, surgical options include valve repair or valve replacement. Early referral for surgical evaluation is critical in preventing irreversible myocardial damage and improving long-term outcomes.

Discussion. Aortic valve involvement in pediatric rheumatic heart disease represents a marker of advanced disease and worse prognosis. Compared to isolated mitral disease, combined aortic and mitral involvement leads to faster hemodynamic deterioration. Early echocardiographic screening in at-risk populations is essential for timely diagnosis.

Recent studies emphasize the importance of subclinical carditis detection using Doppler echocardiography, which allows initiation of secondary prophylaxis before irreversible structural damage occurs.

Conclusion. Rheumatic heart disease continues to be a major pediatric cardiovascular problem, with aortic valve involvement significantly contributing to disease severity. Echocardiography remains the gold standard for early detection and follow-up. Strengthening prevention strategies, early diagnosis, and continuous monitoring are essential to reduce long-term complications and improve outcomes in children.

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