

Case Report

Cardiology

Giant Organized Left Atrial Thrombus in Rheumatic Mitral Stenosis: A Case Report

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Abstract

Rheumatic mitral stenosis, atrial fibrillation, and marked left atrial remodeling create a strong substrate for intracardiac thrombosis and systemic embolism. We report a 67-year-old woman with a previous ischemic stroke who had not received long-term anticoagulation or cardiological follow-up and presented with progressive exertional dyspnea, lower-limb edema, and signs of right-sided heart failure. Electrocardiography showed atrial fibrillation. Transthoracic and transesophageal echocardiography demonstrated rheumatic mitral stenosis with a mitral valve area of 1.5 cm² and a mean transmitral gradient of 8 mmHg, massive left atrial enlargement, dense spontaneous echo contrast with sludge, and multiple thrombi; the largest measured 60 × 40 mm. Therapeutic anticoagulation was initiated. Because of symptomatic valvular disease and the extensive thrombus burden, the patient underwent mitral valve replacement with left atrial thrombectomy. An organized 60 × 40 mm thrombus was removed, and postoperative recovery was uneventful. This case illustrates that thromboembolic risk in rheumatic mitral stenosis may be driven as much by atrial fibrillation, atrial remodeling, and blood stasis as by the transmitral gradient itself, and highlights the central role of transesophageal echocardiography in defining thrombus burden and guiding treatment.

Keywords: Rheumatic mitral stenosis, left atrial thrombus, atrial fibrillation, transesophageal echocardiography, spontaneous echo contrast, systemic embolism, mitral valve replacement, thrombectomy.

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INTRODUCTION

Rheumatic mitral stenosis (MS) promotes left atrial (LA) pressure overload, progressive atrial enlargement, atrial fibrillation (AF), and blood stasis, all of which favor thrombus formation and systemic embolism [1]. AF itself is a major independent risk factor for ischemic stroke [2]. Although large LA thrombi are usually associated with advanced rheumatic valve disease, massive thrombotic burden has also been reported in patients whose hemodynamic obstruction appears less striking, particularly when AF and marked atrial enlargement coexist [3,4]. We report a patient with rheumatic MS, AF, a previous ischemic stroke, dense spontaneous echo contrast, and multiple LA thrombi, including a giant organized thrombus requiring surgical removal.

CASE REPORT

Patient Information and Clinical Findings

A 67-year-old woman with a history of ischemic stroke two years earlier, without subsequent cardiological follow-up or long-term medical therapy, presented with progressively worsening exertional dyspnea over approximately two years. She had never received long-term anticoagulant therapy and had not undergone prior echocardiographic evaluation. She was admitted after clinical deterioration with worsening dyspnea and lower-limb edema. Physical examination showed signs of right-sided heart failure, and electrocardiography demonstrated atrial fibrillation.

Diagnostic Assessment

Transthoracic echocardiography showed marked LA enlargement with a large heterogeneous intracavitary mass. The mitral valve had rheumatic morphology, with a valve area of 1.5 cm² and a mean transmitral gradient of 8 mmHg (Figure 1).



Figure 1: Transthoracic echocardiography showing a giant left atrial thrombus within a markedly enlarged left atrium

Transesophageal echocardiography confirmed rheumatic mitral valve disease with leaflet thickening, commissural fusion, a characteristic hockey-stick configuration of the anterior leaflet, and restricted posterior leaflet motion. The LA was massively enlarged

and contained dense spontaneous echo contrast with sludge and multiple thrombi; the largest measured 60×40 mm (Figure 2). Moderate tricuspid regurgitation was present, with an estimated pulmonary artery systolic pressure of 40 mmHg.



Figure 2: Transesophageal echocardiography showing a giant thrombus occupying most of the left atrial cavity in the setting of dense spontaneous echo contrast and sludge

Therapeutic Intervention and Outcome

Therapeutic anticoagulation was initiated. Given the symptomatic rheumatic MS, the extensive and apparently organized LA thrombus burden, and the high embolic risk, the patient was referred for surgery. She

underwent mitral valve replacement combined with left atrial thrombectomy. An organized thrombus measuring 60×40 mm was successfully removed (Figure 3). The postoperative course was uneventful, with good clinical and hemodynamic recovery.

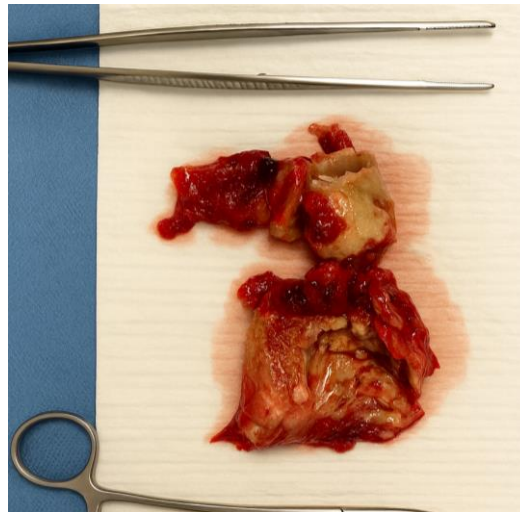


Figure 3: Operative specimen of the giant organized left atrial thrombus removed during mitral valve surgery

DISCUSSION

This case illustrates the complex relationship between the hemodynamic severity of rheumatic MS and its thromboembolic consequences. The valve area of 1.5 cm² lies at the threshold used in the 2025 ESC/EACTS recommendations for clinically severe rheumatic MS, while the mean gradient was only 8 mmHg. Because the transmitral gradient is influenced by flow and heart rate, thromboembolic risk should not be judged from the gradient alone [1]. In this patient, AF, massive LA enlargement, dense spontaneous echo contrast, a previous ischemic stroke, and absence of long-term anticoagulation created a particularly high-risk setting for extensive thrombosis.

Similar reports have described very large LA thrombi in rheumatic MS with AF and marked atrial enlargement, including cases in which the valve stenosis was described as moderate [3,4]. The present case adds a clear sequence from transthoracic detection to transesophageal characterization and surgical confirmation of a giant organized thrombus. Transesophageal echocardiography is particularly valuable in this setting. In a prospective surgical series of patients with rheumatic mitral valve disease, transesophageal echocardiography showed high diagnostic accuracy for LA thrombi and detected substantially more thrombi than transthoracic echocardiography [5].

Anticoagulation remains a cornerstone of management in rheumatic MS with AF, but persistence of LA or left atrial appendage thrombus despite therapeutic anticoagulation has been reported [6]. In our patient, anticoagulation was started at presentation, but prolonged medical therapy alone was not considered sufficient because of the massive thrombus burden, symptomatic valve disease, and previous embolic event. Current ESC/EACTS guidance lists LA thrombus as a contraindication to percutaneous mitral commissurotomy; surgery is therefore appropriate when

intervention is required and the percutaneous approach is unsuitable [1]. Mitral valve replacement and thrombectomy allowed treatment of both the valvular lesion and the embolic source in a single procedure.

The practical message is that rheumatic MS should not be considered low risk simply because the transmitral gradient is not high. Rhythm, LA size, spontaneous echo contrast, prior embolic events, and anticoagulation status are essential components of risk assessment. Earlier cardiological evaluation after the previous ischemic stroke might also have identified the underlying valvular disease and thromboembolic substrate before the thrombus burden became so extensive.

CONCLUSION

Rheumatic mitral stenosis complicated by atrial fibrillation and advanced left atrial remodeling can be associated with massive LA thrombosis even when the transmitral gradient is modest. Transesophageal echocardiography is essential for defining thrombus burden and guiding management. In symptomatic valvular disease with an extensive organized LA thrombus and high embolic risk, surgical mitral valve treatment combined with thrombectomy can provide definitive management.

Conflict of Interest: The authors declare no conflicts of interest.

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