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Silent Vessels, Loud Murmurs: A Case of Takayasu Arteritis with Multivalvular Regurgitation and Severe PAH

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Abstract

Takayasu arteritis (TA) is a rare, chronic large-vessel vasculitis predominantly affecting young women. We present a 42-year-old female with exertional dyspnea, asymmetrical pulses, and multiple cardiac murmurs. Echocardiography revealed moderate mitral and aortic regurgitation with severe pulmonary arterial hypertension (PAH). CT angiography confirmed the occlusion of major aortic branches, consistent with TA. She was managed with corticosteroids, immunosuppressants, and antiplatelet therapy. This case underscores the need for high clinical suspicion and multidisciplinary evaluation in TA with cardiac involvement.

Keywords: Cardiac manifestations, Echocardiography, Stenosis, Takayasu arteritis

Introduction

Takayasu arteritis (TA) is a rare, chronic, large-vessel vasculitis that primarily affects the aorta and its major branches. It most commonly affects women under the age of 40 and has a higher prevalence in Asian populations [1]. TA is characterised by granulomatous inflammation of the arterial wall, resulting in stenosis, occlusion, aneurysm formation, or vessel dilation. Although it is a large-vessel disease, Takayasu arteritis often has significant cardiac involvement, which contributes to morbidity and worsens prognosis [1,2].

Cardiac manifestations are reported in up to 70% of patients and include systemic hypertension, aortic regurgitation, congestive heart failure, pulmonary hypertension, and coronary artery involvement. Among valvular abnormalities, aortic regurgitation is the most frequently reported, typically caused by annular dilatation or inflammatory damage to the aortic root [2.] Less commonly, mitral regurgitation may also occur due to inflammation-related structural distortion of the mitral valve apparatus or secondary to left ventricular dilatation. In a large retrospective study, mitral regurgitation was observed in nearly 39% of TA patients with cardiac involvement, highlighting its clinical significance [2,3].

Clinically, mitral regurgitation in TA may present with dyspnea, orthopnea, fatigue, and a holosystolic murmur at the apex, often mimicking other causes such as rheumatic heart disease. Recognising this rare valvular involvement is essential for accurate diagnosis, especially when combined with systemic features and vascular findings.

Early detection of mitral valve involvement, particularly mitral regurgitation, is crucial for guiding appropriate immunosuppressive therapy and facilitating timely surgical referral when necessary [4].

Case Description

A 42-year-old female presented with complaints of central chest pain for the past 10 days. The pain was sudden in onset, localised over the sternum, non-radiating, and was not influenced by exertion, posture, or respiration. She also reported progressive shortness of breath on exertion, particularly noticeable when climbing stairs, becoming breathless after approximately 10 steps, with symptomatic relief upon resting. Additionally, the patient complained of generalised myalgia, more pronounced in the lower limbs, upper back, and neck, which developed gradually and was alleviated with rest.

She was a known case of pulmonary arterial hypertension (PAH). She had been on regular medications, including torsemide and spironolactone 10 mg OD and Sildenafil 25 mg BD and Riocigaut (0.5, BD), for symptom control and pulmonary pressure management.

On examination, a striking asymmetry in peripheral pulses was noted—absent on the left side, while the right radial artery exhibited a collapsing, high-volume pulse. Blood pressure measurements revealed a significant inter-arm systolic difference of 70 mmHg, with the left arm at 76/48 mmHg and the right arm at 142/52 mmHg. Cardiac auscultation uncovered multiple murmurs: a diastolic decrescendo murmur with a soft A2 at the aortic area, a mid-diastolic murmur with a loud P2 at the pulmonary area, a holosystolic murmur with soft S1 at the tricuspid area, and the mitral area, both a mid-diastolic rumbling murmur (without opening snap) and a holosystolic murmur with soft S1 were appreciated. Additionally, a carotid bruit was audible on the left side.

Lung auscultation revealed bilateral inspiratory and expiratory crepitations across all lung fields, which decreased on coughing. There was no hepatosplenomegaly, pedal edema, or raised jugular venous pressure (JVP). Vitals were stable, with a respiratory rate of 16 breaths/min, a pulse rate of 80 beats/min, an SpO_2 of 98%, and she was afebrile. Arterial blood gas analysis was within normal limits.

Transthoracic and transesophageal echocardiographic evaluations revealed a normal left ventricular volume and preserved systolic function with an ejection fraction of 60%. Notably, there was evidence of severe pulmonary arterial hypertension (PAH) and mild concentric left ventricular hypertrophy. Valvular assessment demonstrated moderate mitral regurgitation (MR) and moderate aortic regurgitation (AR), with a pressure half-time (PHT) of 317 ms. Additionally, moderate to severe tricuspid regurgitation (TR) was present, with an estimated right ventricular systolic pressure (RVSP) of 75 mmHg, supporting the diagnosis of severe PAH.

A Doppler ultrasound of the arterial system in the left upper limb demonstrated echogenic material in the axillary and proximal brachial arteries, with monophasic flow in the distal brachial, radial, and ulnar arteries, suggestive of significant arterial obstruction. Carotid Doppler imaging revealed increased intima-media thickness bilaterally—2.2 mm on the left and 1.9 mm on the right—in the common carotid arteries.

To further delineate the vascular involvement, a CT angiography of the neck (fig.1) was performed, which revealed 30% luminal narrowing of the common carotid artery, complete occlusion of the left subclavian artery, non-opacification of the left axillary artery, and involvement of the VI segment of the vertebral artery. These findings were consistent with large-vessel vasculitis, highly suggestive of Takayasu arteritis.

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Management was initiated with oral corticosteroids (Omnacortil 20 mg in the morning and 10 mg in the evening) to control inflammation. Immunosuppressive therapy was escalated with the addition of azathioprine 100 mg once daily and methotrexate 7.5 mg twice weekly. Aspirin, in addition to PAH medication was also included in the regimen for vascular protection.

The patient was advised to have regular follow-ups every 4–6 weeks, with monitoring of clinical symptoms, and echocardiography every 6 months to track the progression of pulmonary hypertension and valvular regurgitation. A repeat CT angiography was planned at 6 months to assess vascular stability and determine candidacy for revascularisation of the occluded left subclavian artery. Steroids were to be gradually tapered under supervision, while long-term immunosuppressive therapy was continued. The patient remained under coordinated care of rheumatology, cardiology, and CTVS teams for integrated disease monitoring and management. Furthermore,

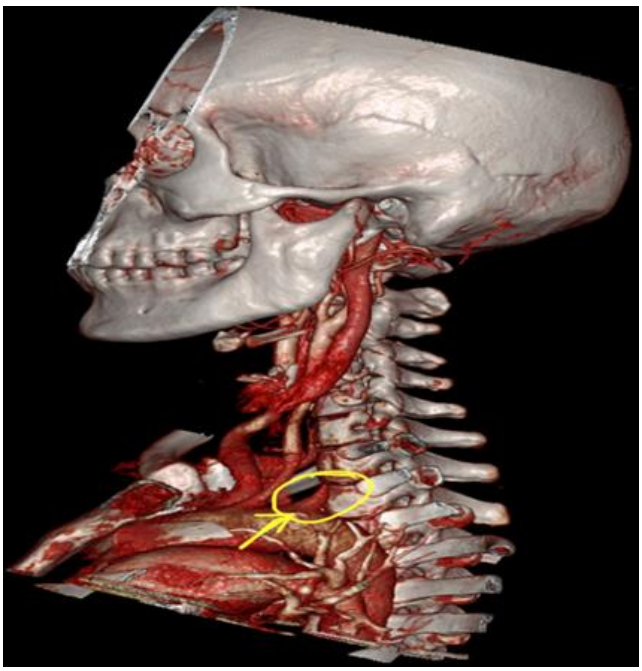

Figure 1: Three dimensional volume- rendered helical CT- angiogram of neck showing the aortic arch and major branch vessels. irregular narrowing of V1 segment of the left subclavian artery is visualised (yellow arrow) as seen in takayusu arteritis.

Discussion

Takayasu arteritis (TA) is a chronic, idiopathic large-vessel vasculitis that predominantly affects the aorta and its major branches. While vascular involvement remains its hallmark, cardiac manifestations often dictate prognosis. Our patient highlights the complexity of TA when it extends beyond vascular inflammation to involve multiple cardiac valves and the pulmonary vasculature [1].

Multivalvular regurgitation is uncommon in TA, with isolated aortic regurgitation being the most frequently reported lesion. In this case, coexistent severe mitral and aortic regurgitation, compounded by pulmonary hypertension, reflects an advanced stage of disease and illustrates the spectrum of cardiovascular sequelae. Such overlap not only worsens functional status but also creates significant challenges in management, especially in younger patients. [2,3,4]

The diagnostic process can be particularly challenging. Symptoms such as dyspnea, fatigue, or palpitations may initially be attributed to primary valvular disease or pulmonary hypertension of other etiologies. Recognition of the underlying vasculitic process requires a high index of suspicion, especially in young females with systemic complaints and absent or asymmetric pulses. Our case underscores the importance of comprehensive vascular imaging (CTA/MRA) in patients with unexplained multivalvular lesions [5,6,7].

Therapeutically, management must address both immunosuppression to control vascular inflammation and cardiac interventions to relieve hemodynamic burden. Valve surgery in TA is associated with high rates of restenosis or graft complications, especially if performed during the active phase of disease. Therefore, careful timing of surgery in relation to disease activity, guided

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by inflammatory markers and imaging, remains essential [8,9,10]. Multidisciplinary care involving cardiology, rheumatology, and cardiothoracic surgery is key [11].

In summary, this case reinforces the need for early recognition of cardiac manifestations such as mitral regurgitation and PAH in Takayasu arteritis. A comprehensive evaluation using echocardiography and vascular imaging is essential for diagnosis and monitoring. Timely initiation of immunosuppressive therapy and coordinated multidisciplinary care are vital to prevent complications and improve long-term prognosis.

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