

Infrequent concomitant mitral, pulmonary, and tricuspid valve prolapse associated with right ventricular failure: Correct diagnosis using multimodality imaging 🎧

A 34-year-old male patient was admitted to our hospital with the symptoms of increased shortness of breath, palpitation, and exercise intolerance. The patient had no past medical history. Electrocardiography revealed atrial tachycardia. A bedside transthoracic echocardiography (TTE) indicated a markedly redundant, elongated tricuspid valve leaflet that had prolapsed into the right atrium (Video 1). Tricuspid annular plane systolic excursion was normal; however, right ventricle (RV) end-systolic area was 30.8 cm² (Fig. 1), RV long-axis diameter was 74 mm, and RV basal diameter was 64 mm which are normal 8.6 cm², 67

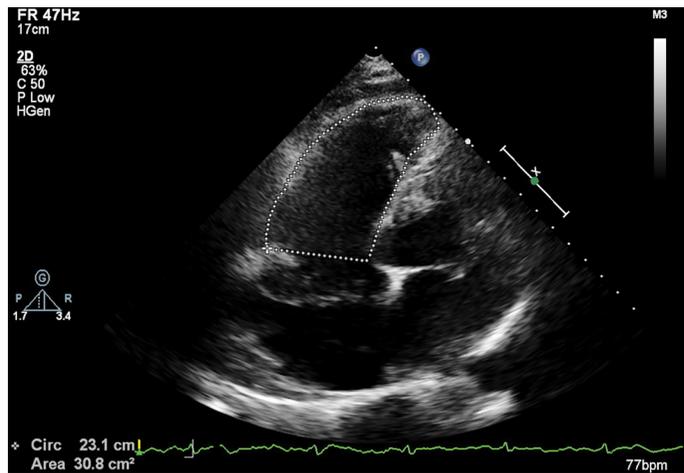


Figure 1. On transthoracic echocardiography (TTE), right ventricular (RV) end-systolic area was 30.8 cm²

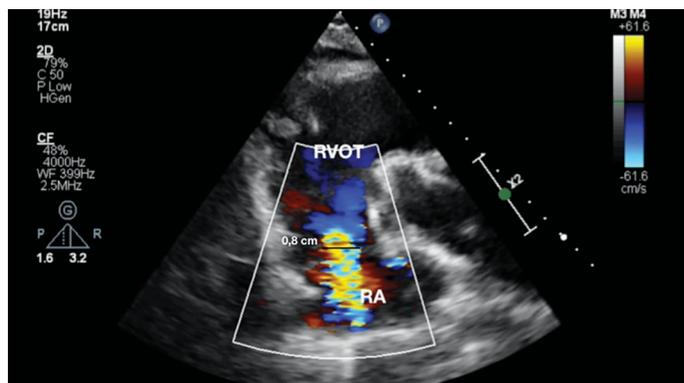


Figure 2. Tricuspid regurgitation vena contracta is seen 0.8 cm in parasternal short axis view

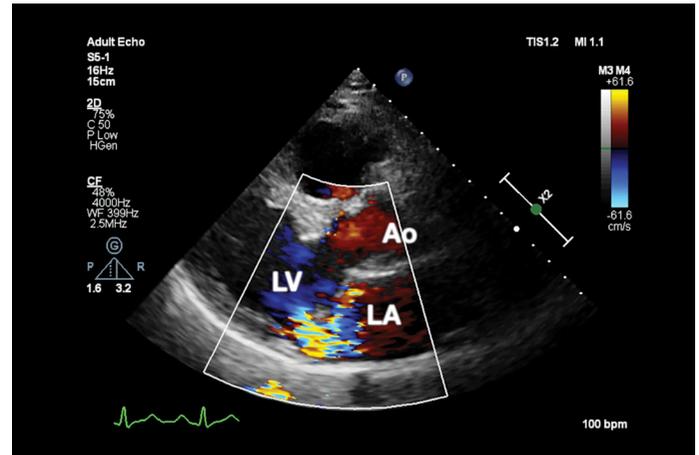


Figure 3. Parasternal long-axis TTE shows moderate non-holosystolic mitral regurgitation



Figure 4. Pulmonary valve prolapse is seen on magnetic resonance (MR) image

mm, 34 mm respectively. That means severe tricuspid regurgitation (Fig. 2), causing RV dilatation. Additionally, the patient had non-holosystolic, moderate mitral regurgitation (Fig. 3) related to multi-scallop Barlow's disease cm² (Video 2) and had mild pulmonary insufficiency related to pulmonary valve prolapse (PVP) (Video 3). Furthermore, aortic valve redundancy was present, causing eccentric mild aortic regurgitation. After the diagnosis, we performed cardiac magnetic resonance imaging (MRI) that clearly showed tricuspid valve prolapse, mitral valve prolapse, and PVP (Fig. 4, Videos 4, 5). Functional evaluation was also performed. Cardiac MRI findings confirmed severe tricuspid regurgitation, with the regurgitant volume being 66 mL; moderate mitral regurgitation, with the regurgitant volume

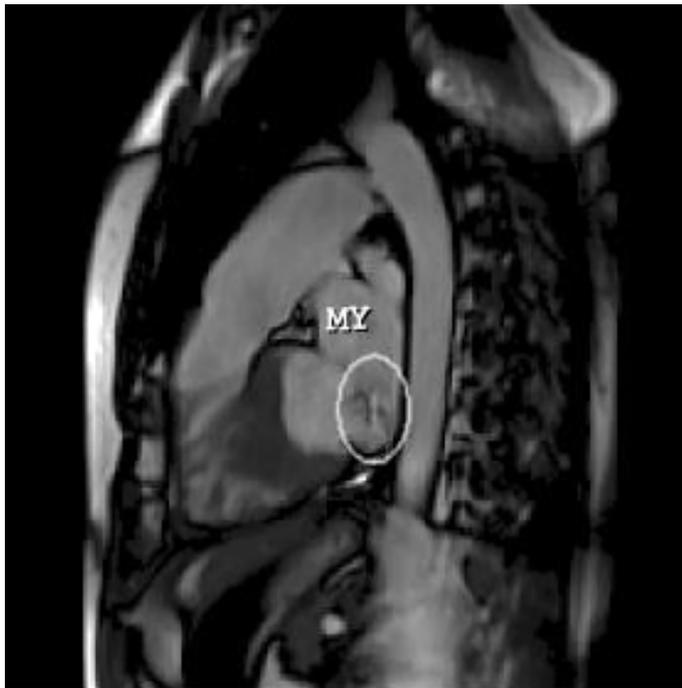


Figure 5. MR image showing mitral regurgitation

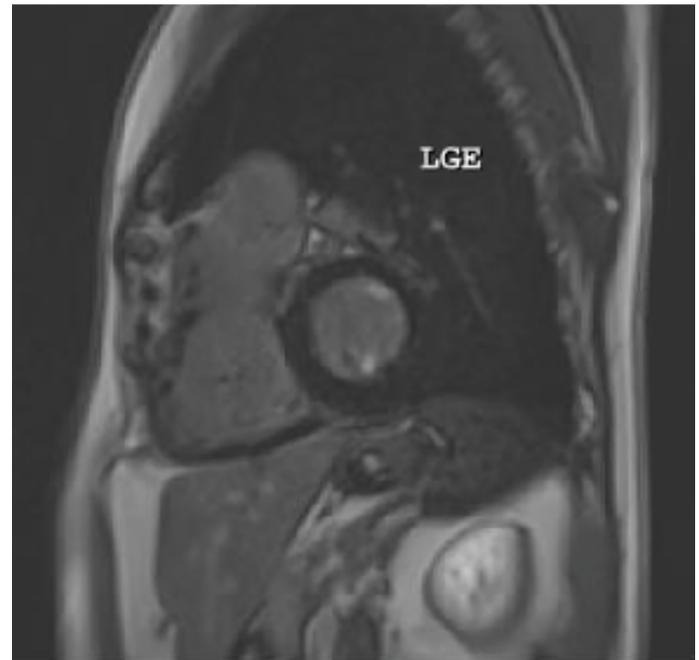


Figure 7. Late gadolinium enhancement fibrosis is seen in mitral papillary muscle

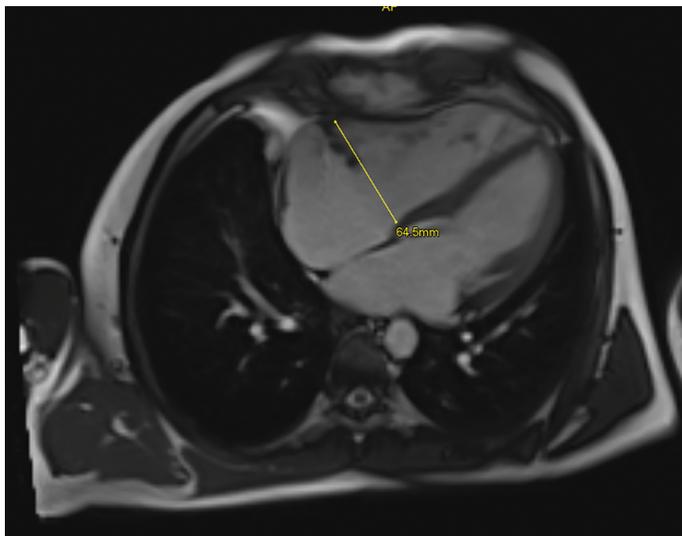


Figure 6. MR images show that RV basal diameter had increased 6 months later

being 34 mL (Fig. 5); and mild pulmonary insufficiency. RV ejection fraction (EF) was 53.6%, end-systolic volume was 167 mL, and end-diastolic volume was 354 mL; furthermore, RV basal diameter was 64 mm (Fig. 6, Video 5). Additionally, MRI demonstrated late gadolinium enhancement (Fig. 7) in the mitral papillary muscle which predicts more severe mitral regurgitation, RV dilatation, reduced RV EF, and adverse outcomes of mitral valve surgery. This would be important at follow-up. Six months later, a follow-up MRI showed that RV basal diameter had increased to 69.3 mm (Fig. 8) and RV EF had decreased to 51%. Thus, we decided to proceed with surgery because of the increasing RV failure and dilatation. The surgeon first performed posterior



Figure 8. MR images show that RV basal diameter had increased 6 months later

leaflet resection, followed by tricuspid bicuspidization and implantation of a 36 no. rigid ring to the annulus. Postoperative echocardiography revealed mild tricuspid regurgitation. We conclude that follow-up MRI is valuable for RV function monitoring and determining the correct operation timing. There are few studies in the literature that have reported diagnosing this type of disease via multimodality imaging.

Video 1. Parasternal right ventricular (RV) in-flow view depicts tricuspid valve prolapse.

Video 2. Parasternal long axis transthoracic echocardiography (TTE) depicts mitral valve prolapse (MVP).

Video 3. PSAX TTE video image shows pulmonary valve prolapse (PVP)

Video 4. Magnetic resonance imaging demonstrates PVP.

Video 5. Apical four-chamber MRI video shows MVP, RV dilatation and RV systolic dysfunction

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Right atrial appendage aneurysm: Does it have to be resected?

Here we show the case of a 51-year-old woman with right atrial appendage aneurysm (RAAA) that was detected on physical examination 10 years ago. No murmur was found in the cardiac auscultation area, and the patient did not experience any clinical symptoms, such as palpitations, heart fatigue, and shortness of breath. Imaging findings from the most recent follow-up were as follows: A 56×84-mm RAAA was detected and further assessed on echocardiogram and computed tomography image (Fig. 1a–1d, blue arrow). No thrombosis was detected in RAAA and right atrium, and compared with previous imaging findings, RAAA showed no obvious expansion and growth. In patients having isolated RAAA, with no clinical manifestations, no arrhythmia, and no thromboembolism, should the atrial appendage be resected? RAAA is a rare structural malformation of unknown etiology in congenital heart disease; patients with RAAA may be asymptomatic or exhibit symptoms associated with atrial arrhythmias (e.g., atrial flutter, atrial fibrillation, focal atrial tachycardia, and supraventricular tachycardia) or thromboembolism. In most of the cases of RAAA reported in the literature, the lesions were surgically removed because the patients suffered

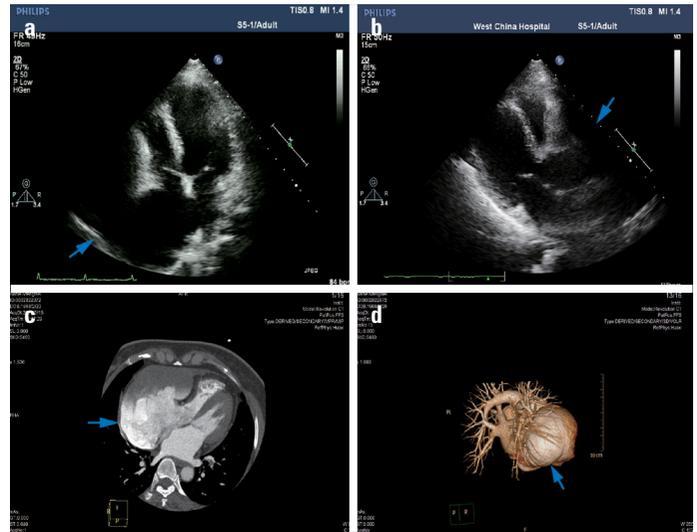


Figure 1. A 56×84-mm right atrial appendage aneurysm was detected and further assessed on echocardiogram and computed tomography image (Fig. 1a-1d, blue arrow)

from the above symptoms or had the condition combined with other congenital heart diseases, such as atrial septal defect and patent foramen ovale. Regarding the surgical indications for asymptomatic patients, the size and the annual growth rate of RAAA must be considered. Furthermore, long-term monitoring of atrial size and annual growth rate of RAAA, airway compression, arrhythmias, and thrombosis is strongly recommended.

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