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CLINICAL SPOTLIGHT

Endovascular Treatment in a Patient with Left Main Coronary and Pulmonary Arterial Stenoses as an Initial Manifestation of Takayasu's Arteritis

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Takayasu's arteritis is a chronic inflammatory disorder that mainly involves medium to large sized arteries. Although it affects coronary and pulmonary arteries occasionally, physicians should consider the possibility of involvement of coronary or pulmonary arteries in patients with Takayasu's arteritis with chest pain or exertional dyspnoea. We report a case of Takayasu's arteritis who presented with exertional dyspnoea and generalised oedema due to severe bilateral pulmonary and left main coronary arterial stenoses. The patient was successfully treated by a one-stage percutaneous transluminal balloon angioplasty and stent implantation of the involved left main coronary and pulmonary arteries. The endovascular treatment may be one of the treatment options for the stenotic vascular lesions in patients with Takayasu's arteritis.

Keywords Takayasu's arteritis • Pulmonary artery stenosis • Coronary artery stenosis • Balloon angioplasty • Stent • Endovascular procedure

Introduction

Takayasu's arteritis (TA) is a chronic vasculitis of unknown aetiology, predominantly affecting the aorta and its main branches [1]. Although subclavian arteries are the most commonly involved arteries, pulmonary or coronary arteries can be involved occasionally which results in pulmonary hypertension or angina pectoris [1–7]. Here, we present a case of TA who had exertional dyspnoea which was caused by the involvements of left main coronary artery (LMCA) and both pulmonary arteries (PAs). The patient was successfully treated with one-stage percutaneous intervention of LMCA and PAs after medical stabilisation of TA.

Case Report

A 25 year-old woman attended with a three-month history of exertional dyspnoea and generalised oedema. The blood pressure was 126/84 mmHg at left and 94/66 mmHg at right arm.

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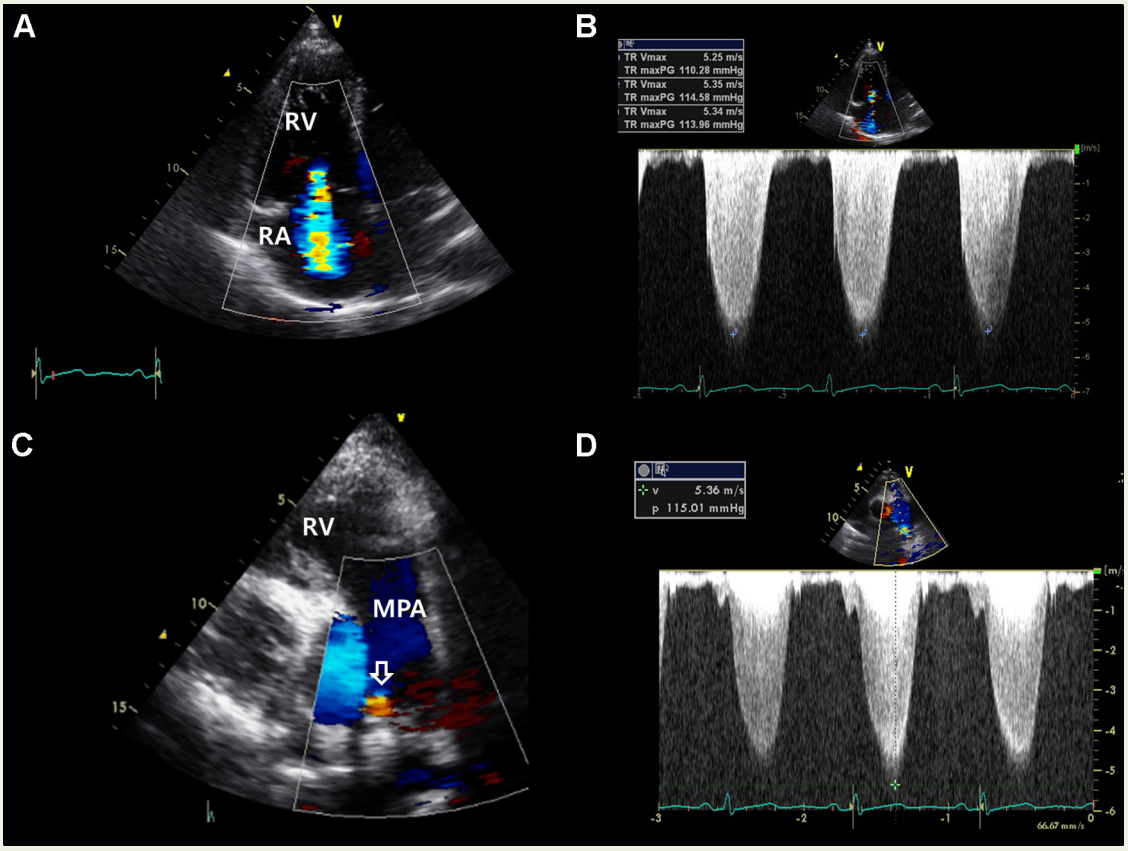

Figure 1 Transthoracic echocardiography shows significant tricuspid valve regurgitant jet reflecting the markedly elevated pulmonary artery pressure (A). Continuous wave Doppler flow of tricuspid valve regurgitation jet velocity is about 5.3 m/sec corresponding to estimated right ventricle systolic pressure more than 100 mmHg (B). Parasternal short axis with colour Doppler mapping demonstrates flow acceleration at both pulmonary arteries (arrow, C). Continuous wave Doppler flow of right pulmonary artery shows peak systolic velocity is about 5.4 m/sec (D). RV; right ventricle, RPA; right pulmonary artery.

The echocardiogram demonstrated severe dilatation of RV with thickened RV walls and moderate tricuspid regurgitation (TR) with calculated RV systolic pressure of about 130 mmHg (TR Vmax = 5.3 m/sec, Fig. 1A and B). There were narrowed proximal portions of bilateral PAs and peak flow through the right PA was measured up to 5.4m/sec (Fig. 1C and D). The computerised tomography revealed stenosis of the proximal portion of bilateral PAs without evidence of pulmonary thromboembolism (Fig. 2A, B and C). Mild peri-aortic soft tissue thickening was also noted at the proximal descending thoracic aorta (Fig. 2D). The pulmonary angiography demonstrated severe stenosis of the proximal portions of bilateral PAs with peak systolic pressure of main PA of about 100 mmHg (mean 65 mmHg) and the peak pressure gradient across the stenotic right PA of about 80 mmHg. The coronary angiography showed severe stenosis from its ostium to bifurcation of the LMCA (Fig. 3A). Laboratory parameters revealed increased erythrocyte sedimentation rate (ESR, 36 mm/h) and C-reactive protein concentration (CRP, 1.3 mg/dL). The autoantibodies were all negative.

The patient was diagnosed as TA and was medicated with high dose prednisolone, azathioprine, aspirin, statin, beta-blocker, nitrate and calcium antagonist for two months. Although inflammatory markers including ESR and CRP were normalised, her symptoms were not improved. The follow-up echocardiography revealed aggravated systolic LV and RV function with similar degree of pressure overload of RV. We recommended surgery but she refused. So, we decided to perform percutaneous intervention.

After engaging the left coronary artery with a 7 Fr EBU 3.5 guiding catheter, intravascular ultrasound study with virtual histology (VH-IVUS, Volcano s5 imaging system, Volcano corp., Rancho Cordova, CA, USA) showed huge catheter encircling soft tissue plaque from the ostium of the LMCA with obscured external elastic membrane (Fig. 3B). The main pathology was huge fibrous plaque of media and adventitial wall (Fig. 3C). The obstructed LMCA lesion was successfully treated with an everolimus-eluting Xience PRIME stent (4.0 × 18 mm, Abbott Vascular, Santa Clara, CA, USA) (Fig. 3D). Subsequent intervention of both PAs was performed at the same procedure. Preprocedural VH-IVUS for right PA revealed huge fibrous plaque of media and adventitial wall (Fig. 3E). The stenosed right PA was treated with balloon dilatation (10.0 × 40 mm Powerflex, Cordis