Accentuating systolic aortic regurgitation during premature ventricular systole

A 47-year old male patient with chest pain during deep inspiration was admitted to our hospital. He had no dyspnea or history of heart disease. His blood pressure was 90/60 mm Hg, and he had infrequent extra beats during examination. The patient was unaware regarding these beats, and his physical examination was otherwise normal. Electrocardiography showed a sinus rhythm with ventricular premature systoles that probably originated from the left ventricle (LV) outflow tract (Fig. 1). Transthoracic echocardiography revealed normal LV diameters and systolic functions with mild mitral and barely visible aortic regurgitations (ARs). The valves appeared normal. An accentuation in AR simultaneous with mitral regurgitation was noticed during the systolic phase of premature beats, which did not continue during the diastolic phase of these beats (Video 1 and Fig. 2).

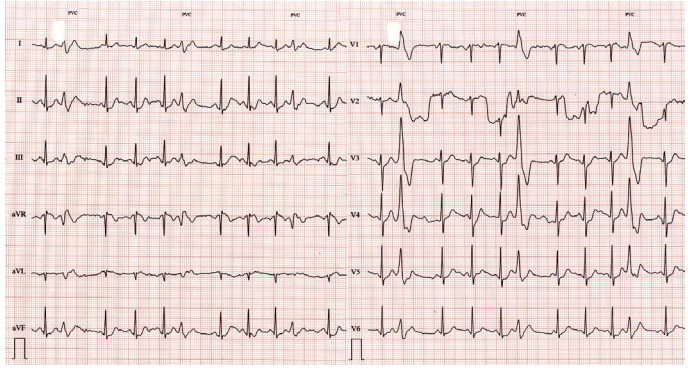

Figure 1. Electrocardiogram showing sinus rhythm with premature ventricular beats that probably originated from the left ventricle outflow tract
PVCs - premature ventricular contractions

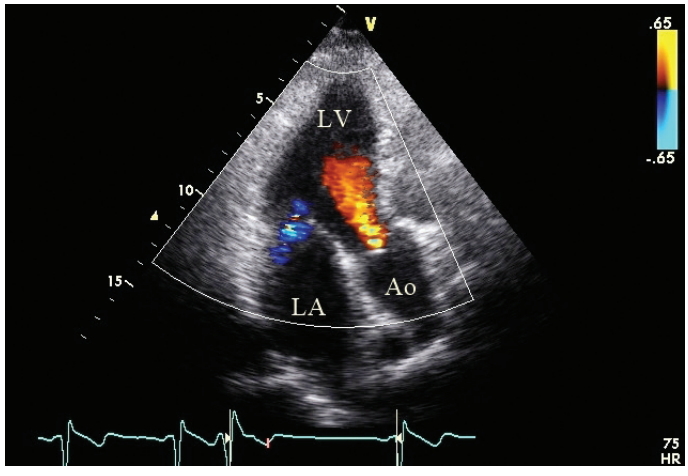

Figure 2. Color Doppler echocardiography showing simultaneous aortic and mitral regurgitation flows during the systolic phase of the premature ventricular beat
Ao - aorta; LA - left atrium; LV - left ventricle

A continuous wave Doppler evaluation also confirmed this phenomenon with the absence of a forward flow through the aortic valve and accentuated reverse flow during premature ventricular systole compared with sinus-derived beats (Fig. 3).

We speculated that the premature contraction that originated in close proximity to the LV outflow and aortic cusps may distort the coaptation of the aortic valve, creating an enlarged regurgitant orifice that accentuates regurgitation. Emphasizing on the probable origin of the premature beat, a contraction propagating reversely (from the LV outflow to the rest of the myocardium) may also induce a "milking" movement, drawing blood from the aorta into the outflow tract. Besides, aortic valve exerts conformational changes in size, shape, and stiffness throughout the cardiac cycle, and a premature systole in proximity may deceive solely the valve without generating an adequate intraventricular pressure.

The aortic valve may show adaptive changes in histological and mechanical properties in response to hemodynamic aberrations such as systolic regurgitation. In this context, tachycardia/arrhythmia-induced cardiomyopathy is a well-known entity, and further studies on this topic may beget the emergence of the concept "tachycardia/arrhythmia-induced valvulopathy."

Video 1. Color Doppler echocardiography of the apical 5-chamber view.

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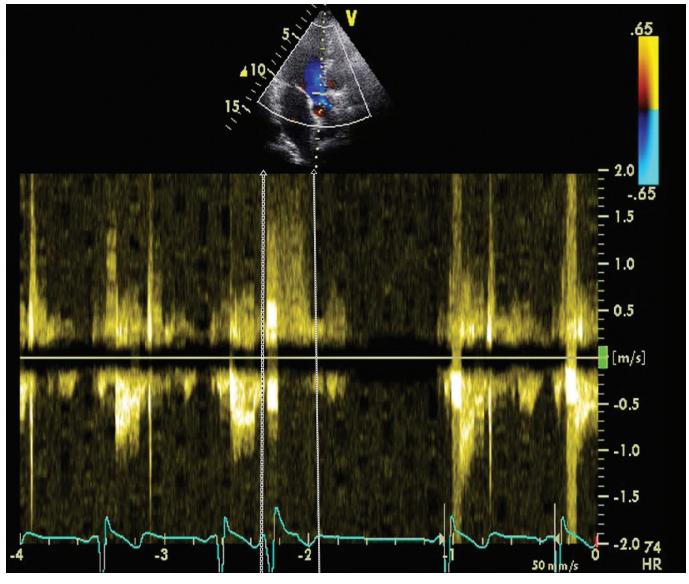

Figure 3. Continuous wave Doppler echocardiography image showing the flow through the aortic valve. The forward flow ceases during premature ventricular systole, and the reverse flow accentuates but does not continue during the whole diastole (premature ventricular systole between vertical arrows). The diastolic reverse flow is barely observed during a normal sinus beat

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Real-time three-dimensional echocardiography imaging of the main pulmonary artery pseudoaneurysm

A 20-year-old man with complaints of fever and chest pain visited the emergency department of our hospital. He was previously diagnosed with patent ductus arteriosus (PDA) in his early childhood, which was not treated. The patient had no history of catheterization or trauma. On arrival, his body temperature was 37.1°C, blood pressure was 109/56 mm Hg, pulse rate was 120 beats/min, and oxygen saturation was 97% on air. Bedside transthoracic echocardiography revealed a ductal diameter between the left pulmonary artery and aorta, which measured 0.7 cm, and a large mass adjacent to the dilated main pulmonary artery, with arterial blood flowing into it (Fig. 1a, b; Videos 1, 2). Real-time three-dimensional echocardiography provided valuable data regarding the shape of the pseudoaneurysm and its association with the pulmonary artery; the saccular outpouching was observed at the left of the pulmonary trunk, and the maximum width measured was 1.9 cm (Fig. 2a, b, Videos 3, 4). Chest computed tomography complemented echocardiography (Fig. 3). A diagnosis of PDA and main pulmonary artery pseudoaneurysm (PAP) was made. The patient expired 2 days after progressive decline in blood pressure.

PAP is an uncommon disease entity, most of which is caused by trauma, infection, and connective tissue disorders. PAP observed in the pulmonary artery trunk is rare because it shows a strong predilection for peripheral pulmonary arteries. Pseudoaneurysms do not involve all layers of the arterial wall, and the adventitia or adherent fibrous tissue contains the extravasated blood from vessel ruptures. We assume that in our case, the untreated large PDA played a role in PAP development.

Video 1, 2. Two-dimensional transthoracic echocardiography of the pseudoaneurysm.

Video 3, 4. Three-dimensional echocardiography of the pseudoaneurysm.

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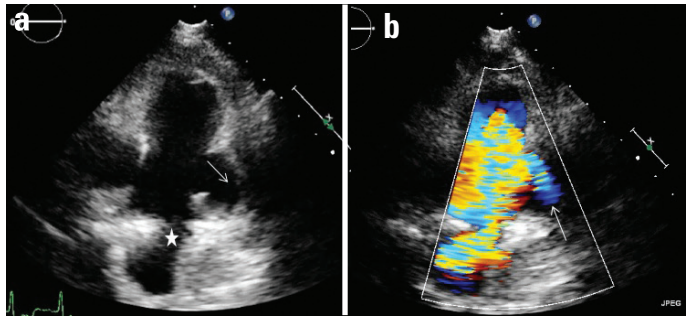

Figure 1. (a) Two-dimensional transthoracic echocardiography showed the patent ductus arteriosus (asterisk) and the pseudoaneurysm (arrow). (b) Color Doppler echocardiography demonstrated the blood flow from the pulmonary artery into the pseudoaneurysm (arrow)

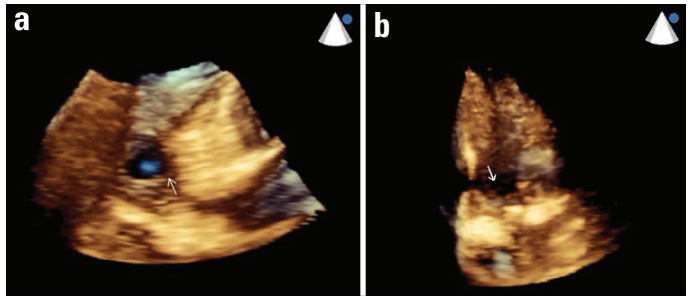

Figure 2. (a) The cross-section of three-dimensional echocardiography view of the pseudoaneurysm (arrow) from the pulmonary arterial perspective. (b) The longitudinal section of the pulmonary artery and the orifice of the pseudoaneurysm (arrow)

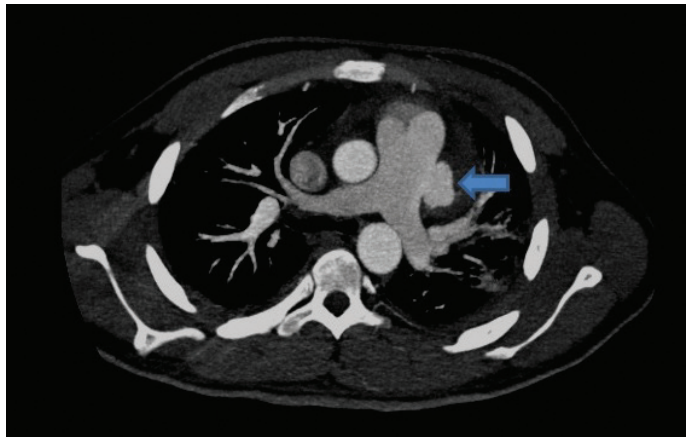

Figure 3. Computed tomography confirmed the association between the pseudoaneurysm (arrow) and the main pulmonary artery

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