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).

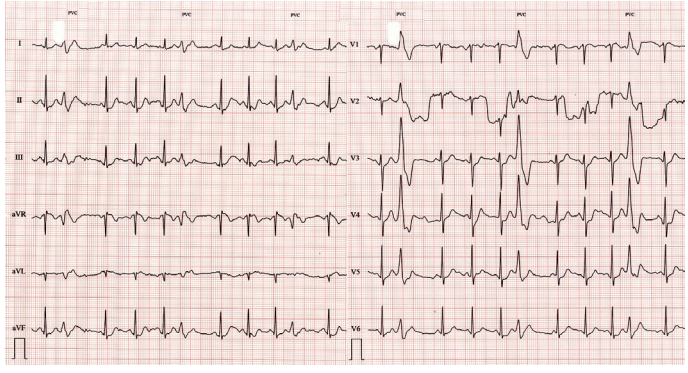

Figure 1. Electrocardiogram showing sinus rhythm with premature ventricular beats that probably originated from the left ventricle outflow tract
PVCs - premature ventricular contractions

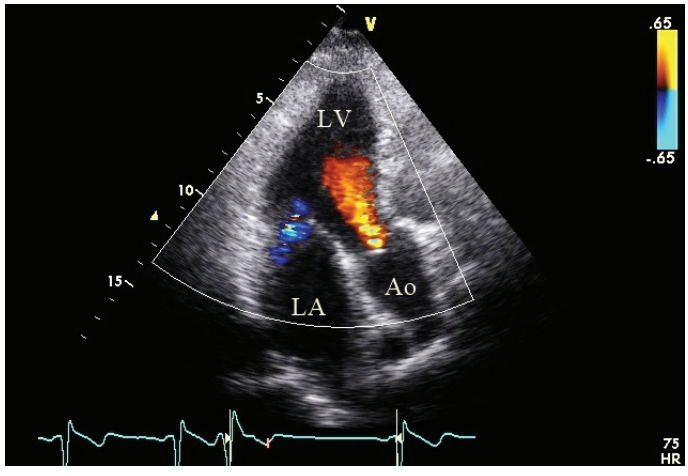

Figure 2. Color Doppler echocardiography showing simultaneous aortic and mitral regurgitation flows during the systolic phase of the premature ventricular beat
Ao - aorta; LA - left atrium; LV - left ventricle

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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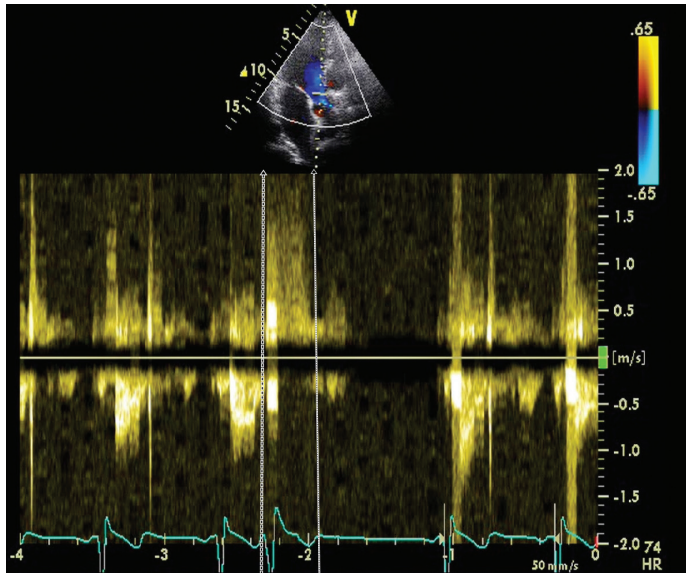

Figure 3. Continuous wave Doppler echocardiography image showing the flow through the aortic valve. The forward flow ceases during premature ventricular systole, and the reverse flow accentuates but does not continue during the whole diastole (premature ventricular systole between vertical arrows). The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

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