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CASE REPORT

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Diagnostic Role of Incidental Premature Contractions During Doppler Echocardiography

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ABSTRACT

Cardiac sonographers often perceive premature beats as a limiting factor during echocardiography because they alter filling and contractility, and loops recorded during or after a premature contraction are often discarded. Here we present 2 cases in which the incidental occurrence of premature beats on Doppler echocardiography contributed to the diagnosis. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:822–825) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

INTRODUCTION

Cardiac sonographers often perceive premature beats occurring during echocardiography as a limiting factor because of the associated alterations in hemodynamics, filling, and contractility. Here we present 2 cases in which the incidental occurrence of premature beats was instrumental in improving the diagnostic accuracy of the Doppler echocardiographic examination.

LEARNING OBJECTIVES

- To recognize the diagnostic and prognostic value of incidental premature beats occurring during Doppler echocardiography examinations.
- To understand that post-extrasystolic potentiation and prolonged diastolic filling are the main underlying mechanisms.

PATIENT 1

A 78-year-old patient with a history of arterial hypertension, chronic obstructive lung disease, and prostate carcinoma was admitted for pulmonary edema. Echocardiography performed after hemodynamic stabilization showed calcified aortic stenosis (AS) with a mean transaortic pressure gradient of 31 mm Hg and a maximal velocity of 3.5 m/s (**Figure 1A** and first beat of **Figures 1B and 1C**). Using the continuity equation, the aortic valve area was calculated as 0.8 cm^2. The left ventricular (LV) ejection fraction (LVEF) was 55%, and the indexed stroke volume was 30 mL/m^2, consistent with paradoxical low-flow, low-gradient AS. The patient had several premature ventricular complexes (PVCs) during echocardiography. These PVCs were associated with a post-extrasystolic increase in transaortic velocities, with the mean pressure gradient and the maximal velocity being 43 mm Hg and 4.4 m/s, respectively

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(third beat of Figures 1B and 1C). Combining the post-extrasystolic beat continuous wave and LV outflow tract (LVOT) pulsed wave Doppler velocities (the coupling was similar during both recordings), the calculated aortic valve area was 0.97 cm^2, consistent with true severe AS. The calcium score of the aortic valve was 6,224 AU. The patient refused transcatheter aortic valve replacement and eventually died 3 months later of a further episode of pulmonary edema.

PATIENT 2

A 55-year-old asymptomatic woman with a history of rheumatic heart disease and symptomatic rheumatic mitral valve stenosis presented for routine echocardiography 6 months after percutaneous mitral commissurotomy. She had diastolic doming of the anterior mitral valve leaflet and a thickened and restricted posterior leaflet (Figures 2A to 2C); there was no mitral regurgitation. The mean transmitral pressure gradient was 7 mm Hg during sinus beats, and the pressure half-time (PHT) was consistent with a 2.3-cm^2 mitral valve area (MVA), but the diastolic period was short. Following a premature supraventricular contraction (PSVC), the diastolic filling time was longer, and the PHT-derived MVA was 1.5 cm^2. MVA was 1.6 cm^2 by planimetry of the mitral valve orifice in the short axis (Figures 2A to 2C).

DISCUSSION

PATIENT 1. In this patient with low-flow, low-gradient AS, post-extrasystolic potentiation increased the mean pressure gradient and maximal velocity to levels consistent with true severe AS. The high calcium score further supported this diagnosis. In patients with reduced LVEF and low-flow, low-gradient AS, dobutamine stress echocardiography is useful to distinguish between "true severe" severe and "pseudosevere" AS, and as such is recommended by current guidelines.[1] However, this technique is time-consuming and has several complications. Because post-extrasystolic peak velocity and mean pressure gradient have been shown to correlate well with dobutamine stress maximal velocity and mean pressure gradient, post-extrasystolic potentiation has the potential to identify true severe low flow, low gradient AS,[2] especially given that PVCs are highly prevalent in patients with AS.[3] In patients with preserved LVEF (paradoxical low-flow, low-gradient AS), the role of dobutamine stress echocardiography is more controversial. However, post-extrasystolic potentiation may also prove useful in this setting because it is associated with increased stroke volume. In a recent study including patients with various ranges of ejection fraction, Dehghani et al[4] suggested that an inability to generate ≥40 mm Hg of mean transaortic pressure gradient post-PVC was highly specific for nonsevere AS.

PATIENT 2. In this patient, who was evaluated 1-year following mitral percutaneous commissurotomy, the MVA obtained from the PHT method was much closer to that obtained with planimetry when evaluated during the PSVC (1.5 cm^2 and 1.6 cm^2, respectively) than when measured in sinus rhythm with a short diastolic period (2.3 cm^2). Following mitral percutaneous commissurotomy, an

ABBREVIATIONS AND ACRONYMS

AS = aortic stenosis

LV = left ventricular

LVEF = left ventricular ejection fraction

LVOT = left ventricular outflow tract

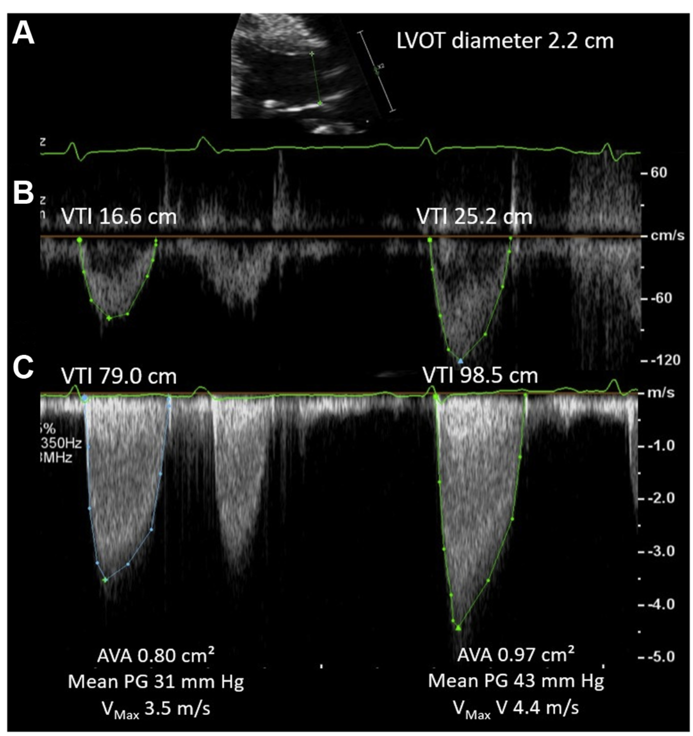
MVA = mitral valve area

PHT = pressure half-time

PVC = premature ventricular complex

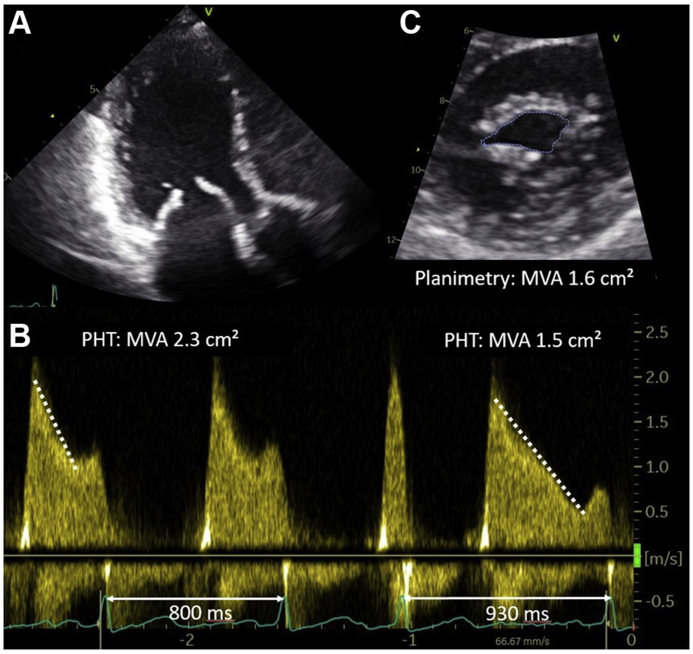
PSVC = premature supraventricular complex

FIGURE 1 A Patient With Low-Flow, Low-Gradient Aortic Stenosis (Patient 1)



(A) Left ventricular outflow tract (LVOT) diameter is 2.2 m/s. **(B)** With a resting heart rate of 72 beats/min, the velocity time integral (VTI) of the left ventricular outflow tract pulsed wave Doppler flow is 16.6 cm, thus enabling calculation of an indexed stroke volume of 30.0 mL/m^2. **(C)** The velocity time integral across the aortic valve by continuous wave Doppler is 79.0 cm, the mean pressure gradient (PG) is 31 mm Hg, and the maximal velocity across the aortic valve (Vmax) is 3.5 m/s. Using the continuity equation, the aortic valve area (AVA) is 0.80 cm^2. **(B)** During the third beat, which occurs after a premature ventricular beat, the left ventricular outflow tract velocity time integral is 25.2 cm, the transaortic velocity time integral is 98.5 cm, the mean pressure gradient is 43 mm Hg, the maximal velocity across the aortic valve is 4.4 m/s, and the calculated aortic valve area is 0.97 cm^2.

FIGURE 2 A Patient With Rheumatic Mitral Stenosis (Patient 2)



(A) A 3-chamber view showing diastolic doming of the anterior mitral leaflet and restricted posterior mitral valve leaflet motion. **(B)** During the first 2 diastoles recorded in sinus rhythm (heart rate, 75 beats/min), continuous wave Doppler imaging of the mitral valve inflow detected a pressure half-time (PHT) consistent with a mitral valve area (MVA) of 2.3 cm^2. **(C)** After a premature supraventricular beat, the PHT-derived-MVA was 1.5 cm^2, a value close to the 1.6 cm^2 obtained with planimetry (1.6 cm^2).

MVA <1.75 cm^2 predicts poor late functional results.[5] In patients with rheumatic mitral valve stenosis, the PHT method has received a Level 1 recommendation by the European Association of Echocardiography and the American Society of Echocardiography for assessment of MVA,[6] but the method is not recommended during short diastolic periods. Moreover, the deceleration slope may be bimodal, with a more rapid early diastolic decline in mitral flow velocity, which may contribute to overestimation of the MVA.[6] PSVCs offer the opportunity to unmask the deceleration slope of the transmitral velocities.

By providing a longer diastolic period, PSVCs may improve the accuracy of the PHT method at faster heart rates. Indeed, the PHT method has been shown to overestimate Gorlin-derived MVA at heart rates >80 beats/min.[7] In our patient, the resting heart rate was 75 beats/min (RR interval, 800 ms), which highlights the potential diagnostic role of PSVCs even at slower resting heart rates. Although the accuracy of the PHT method has not been tested specifically after PSVC, it has been well validated in patients with atrial fibrillation.[8] However, one should remain cautious when using PVC for this purpose because PVCs markedly impair relaxation and diastolic filling.[9]

Incidental premature beats may also be relevant in other clinical situations. For example, they may unmask latent dynamic LVOT obstruction.[10] In such cases, the diastolic filling time, end-diastolic volume, and hence the stroke volume are increased during the compensatory pause following the PVC, by the Frank-Starling mechanism. The increased contractility promotes LVOT obstruction, decreased pulse pressure, and increased LV pressure, a phenomenon known as the Brockenbrough-Braunwald-Morrow sign. This finding may influence patient management, with avoidance of vasodilator agents, diuretic agents, and inotropic agents. Post-extrasystolic potentiation can also be used in the assessment of myocardial viability in ischemic heart disease.[11]

CONCLUSIONS

Cardiac loops recorded during or after a premature contraction are often discarded from echocardiographic recordings and analyses. However, in selected cases, looking at the effects of premature contractions can improve diagnostic and prognostic assessment. Post-extrasystolic potentiation and prolonged diastolic filling are the main mechanisms underlying this added diagnostic value.

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KEY WORDS aortic valve, Doppler ultrasound, echocardiography, mitral valve, supraventricular arrhythmias, ventricular arrythmias