

Iatrogenic perforation of the aortic valve cusp as a complication of percutaneous coronary intervention

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DOI: 10.33963/KPa2023.0085

Received:

January 25, 2023

Accepted:

March 19, 2023

Early publication date:

March 30, 2023

A 64-year-old obese man, former smoker, was hospitalized for *de novo* diagnosis of failure. The patient reported increased fatigue and episodes of chest pain and palpitations during physical activity in the past 1–2 years (New York Heart Association [NYHA] classification, class II, Canadian Cardiovascular Society [CCS] classification, class II). Echocardiography revealed decreased left ventricular ejection fraction (LVEF) of 25%–30%, akinesia of the basal and middle segments of the inferior wall, and hypokinesia of the remaining LV segments, without significant valve pathologies.

Left coronary artery angiography revealed significant proximal left anterior descending artery (LAD) stenosis. Catheterization of the right coronary artery (RCA) required multiple diagnostic catheters (JL-3.5/6F, JR-4.0/6F, 3DRC/6F, AL 2.0/6F, AL 1.0/6F, AR 1.0/6F), and the RCA injection was subselective. Significant proximal stenosis of the RCA was suspected. RCA intubation was achieved with a DRC/6F catheter, and, finally, instantaneous wave-free pressure ratio (iFR)/fractional flow reserve (FFR) measurements ruled out the significance of RCA stenosis. The LAD was successfully treated with drug-eluting stent (DES) implantation. The patient did not report any symptoms, and no significant cardiac murmur was observed. Medical therapy was optimized, and the patient was discharged on the following day with a scheduled admission in 3 months for re-evaluation and implantable cardioverter-defibrillator (ICD) qualification.

On the next elective admission, the patient reported a significant reduction in previously reported symptoms (NYHA I, CCS I). Diastolic

murmur 2/6 was audible and blood pressure was 115/50 mm Hg. There was no fever or other signs of inflammation. C-reactive protein (CRP) was normal. As previously, global left ventricular hypokinesia was visualized on transthoracic echocardiography, however, with an increase in LVEF up to 40% with a simultaneous increase in LV end-diastolic dimension (LVEDD) from 60 to 66 mm (LV end-diastolic volume [LVEDV] from 260 to 310 ml). Moreover, the presence of a previously absent mobile 8 mm subvalvular structure in the left ventricular outflow tract (LVOT) was noted (Figure 1A). It was confirmed by 3D transesophageal echocardiography showing moderate/severe aortic regurgitation due to iatrogenic perforation of the non-coronary cusp (Figure 1B–D). The patient was again presented to Heart Team and qualified for surgical valve replacement, which was successfully performed (Figure 1E, F). The damaged valve was removed and a SJM 25A mechanical aortic valve was implanted. The postoperative course and wound healing were uneventful. The patient was discharged from the hospital in good general condition.

Iatrogenic aortic valve perforation represents a very rare complication of percutaneous coronary intervention. Its approximate prevalence is 0.0001% and only 16 similar cases have been reported in the literature [1]. It must be taken into account when a forceful technique has to be used to intubate the coronary artery. Clinical presentation is mostly acute, and it results from the laceration or perforation of a cusp by the catheter [1]. In our case, the course was almost asymptomatic but

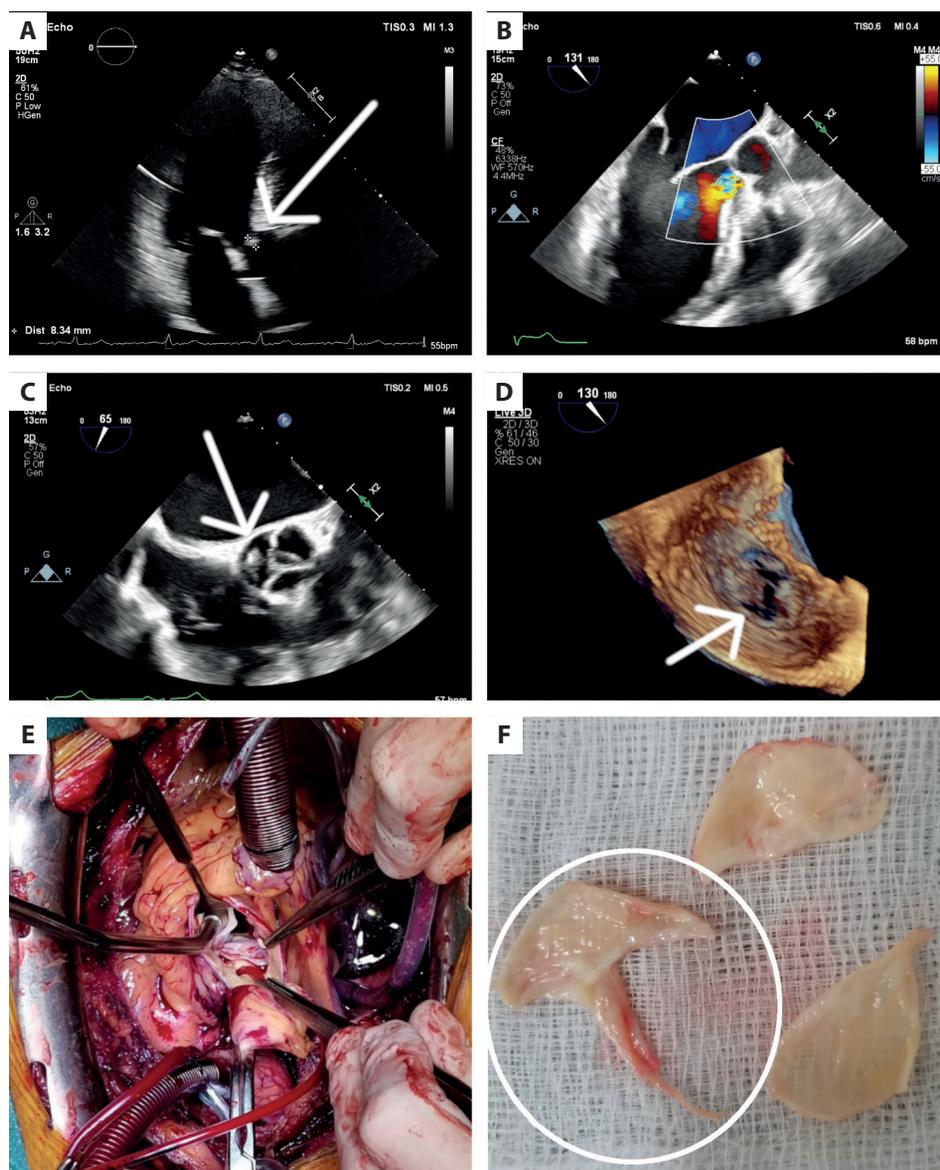


Figure 1. A. 2D trans-thoracic echocardiogram, apical 3-chamber view — an 8 mm long structure in the left ventricular outflow tract (arrow). B. 2D transesophageal echocardiogram (TEE), ME LAX view — severe aortic regurgitation. C, D. 2D, and 3D TEE, ME aortic valve SAX view — perforation of the non-coronary aortic cusp (arrows). E, F. Cardiac surgery with aortic valve replacement. The perforated cusp is marked with a circle

led to progressive dilatation of the left ventricle. The tear of the leaflet may have progressed during the 3 months of follow-up. It was important to differentiate the mechanical cause from infective endocarditis [2]. According to the so far largest systematic review, aortic valve replacement is the most common treatment [1]. In accordance with the current guidelines, the Heart Team should decide about qualifying patients for an appropriate treatment method for that rare complication [3–5].

Article information

Conflict of interest: None declared.

Funding: None.

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