Case Report

Recurrent ventricular tachycardia from severe aortic stenosis improves after percutaneous aortic balloon valvuloplasty and transcatheter aortic valve replacement

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ABSTRACT

We report a case of recurrent ventricular tachycardia from severe aortic stenosis that improved after percutaneous aortic balloon valvuloplasty and transcatheter aortic valve replacement. The electrocardiographic features of the arrhythmia were compatible with ventricular tachycardia originating from the left ventricle. Myocardial ischemia and electrolyte abnormalities were ruled out. Clinicians should be aware that recurrent left ventricular tachycardia associated with severe aortic stenosis is a potentially reversible condition by transcatheter intervention.

<Learning objective: Calcific aortic stenosis is a common problem in the elderly. There is a high prevalence of ventricular arrhythmias in these patients. Transcatheter aortic valve replacement is a relatively new procedure for high-risk patients with severe aortic stenosis. We report a case that illustrates for the first time resolution of recurrent ventricular tachycardia after percutaneous aortic balloon valvuloplasty and transcatheter aortic valve replacement in an elderly patient with severe aortic stenosis.>

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Introduction

Calcific aortic stenosis (AS) is a common problem in the elderly. Symptomatic severe AS is associated with an increased risk of sudden cardiac death (8–34%) compared to adults with asymptomatic AS (up to 5%) [1]. Although the exact mechanism of sudden death is unknown, patients with AS have a high prevalence of ventricular arrhythmias, more commonly in those with higher left ventricular systolic stress and reduced systolic function [2,3]. Symptoms in AS are a clear indication for valve replacement, which has been proven to reduce mortality. Transcatheter aortic valve replacement (TAVR), a relatively new procedure for high-risk patients with severe AS, is effective in the short and medium term [4]. Malignant tachyarrhythmias have been reported to occur after TAVR and aortic valve interventions. We report a case that illustrates for the first time resolution of recurrent ventricular tachycardia (VT) after percutaneous aortic balloon valvuloplasty (PABV) and TAVR in a patient with severe AS.

Case report

An 87-year-old female with a history of single-vessel branch coronary artery disease and AS had coronary angiography performed 4 years previously for a non-ST-elevated myocardial infarction (peak troponin 75.15 ng/ml). She had 100% stenosis of the first diagonal artery successfully treated with balloon angioplasty (without stenting because of the small caliber of the artery). She remained stable from a cardiac standpoint for three years until recent echocardiography showed moderate to severe AS with preserved left ventricular systolic function [left ventricular ejection fraction (LVEF) 55%, peak aortic velocity 2.78 m/s, and peak/mean gradient 31 mmHg/17.5 mmHg, aortic valve area 0.99 cm², and dimensionless index of 0.29]. She later developed symptoms of dyspnea on exertion [New York Heart Association (NYHA) class II] of 6 months’ duration and frequent episodes of palpitations unrelated to exertion. She described these episodes as a sudden onset of regular rapid heart beat lasting several minutes, not associated with any other symptoms, and occurring several times a week. She denied syncope, lightheadedness on exertion, or other symptoms such as...
angina, paroxysmal nocturnal dyspnea, orthopnea, or pedal edema. Repeat echocardiogram revealed progression of AS with preserved LVEF, peak aortic velocity 3.12 m/s, peak/mean aortic valve gradients 39.1 mmHg/22.6 mmHg, aortic valve area 0.74 cm², indexed aortic valve area 0.46 cm²/m², and dimensionless index 0.22. Electrocardiography revealed multiple runs of non-sustained VT with right bundle branch block (RBBB) morphology (Fig. 1) for which the patient was then hospitalized.

Inpatient cardiac telemetry monitoring showed multiple frequent episodes of non-sustained VT with RBBB morphology. Some episodes were associated with asymptomatic hypotension. She complained of palpitations and denied any other symptoms. Extensive workup included coronary angiography, which revealed non-significant coronary artery disease. She was treated with metoprolol and amiodarone but continued to have VT episodes. The patient underwent an electrophysiology study; however, the VT was not inducible during that study. We recommended continuation of medical treatment for VT and to proceed with AVR because AS was thought to be the cause or major contributing factor of VT. Surgical AVR was offered to the patient but she was reluctant to proceed and decided to wait until she could meet criteria for TAVR.

Because of recurrent episodes of VT suspected to be secondary to AS and her reluctance to proceed with surgical AVR, we considered the option of PABV as a therapeutic trial to evaluate her VT response as well as a bridge to surgical AVR or TAVR.

She underwent successful PABV in 2012; the peak aortic valve gradient decreased from 38 to 12 mmHg, the mean aortic valve gradient decreased from 22 to 12 mmHg, and the aortic valve area increased from 0.64 cm² to 0.84 cm². Her symptoms of dyspnea on exertion (NYHA class II) and palpitations resolved completely after PABV. Furthermore, no more episodes of VT were observed on telemetry monitoring. She was asymptomatic for 6 months and then presented with recurrent dyspnea on exertion (NYHA class II) and worsening aortic valve gradients. She was reevaluated for surgical AVR but considered high risk due to advanced age and comorbidities. She underwent TAVR with a 23-mm Edwards SAPIEN valve (Edwards Lifesciences Corp., Irvine, CA, USA) via a transfemoral approach with excellent results. Post-TAVR her dyspnea on exertion resolved, she has not experienced palpitations, and has not had any documented episodes of VT either by telemetry monitoring during her 5-day inpatient stay after TAVR or during outpatient 30-day continuous telemetry monitoring (Fig. 2).

**Fig. 1.** Electrocardiogram obtained during echocardiogram encounter, while patient complained of palpitations, showing a run of non-sustained ventricular tachycardia with right bundle branch block morphology and inferior axis, representing an origin from the left ventricle.

**Fig. 2.** Electrocardiogram obtained post-transcatheter aortic valve replacement; no further episodes of non-sustained ventricular tachycardia with right bundle branch block morphology were observed after the procedure was successfully performed.
Discussion

This case illustrates that PABV and TAVR served to resolve recurrent non-sustained VT episodes refractory to medical therapy in a patient with severe AS. The electrocardiographic features of the arrhythmia were compatible with an origin from the left ventricle, with RBBB morphology, and inferior axis. The ventricular tachyarrhythmia was not secondary to epicardial coronary artery disease-induced ischemia or an electrolyte imbalance. The suspected cause of VT was secondary to severe aortic stenosis causing left ventricular hypertrophy, increased left ventricular end-diastolic pressure, increased wall stress, and subsequent decreased coronary perfusion possibly leading to subendocardial ischemia. The electrocardiograms in Figs. 1 and 2 reveal how diffuse T wave inversions, likely related to the above mentioned mechanisms, improved after intervention. Once the aortic valve gradient improved after PABV and later resolved after TAVR, the VT resolved as well, suggesting that the physiology of severe AS was its main cause or contributing factor.

Patients with severe AS who have not undergone surgical AVR have been reported to suffer from frequent and complex ventricular arrhythmias associated with a reduced LVEF and elevated systolic wall stress, among multiple different arrhythmogenic factors [2,3]. For those who have undergone surgical valve replacement for AS, a strong relationship has been observed between complex ventricular arrhythmias and left ventricular performance [5]. A trend toward decreased frequency and complexity of ventricular arrhythmias has been observed when valve replacement is followed by a marked improvement of left ventricular systolic function [6]. One study showed a 43% reduction in the left ventricular mass within the first 2 years post-surgery, contributing to a decreased vulnerability to ventricular arrhythmias [7]. No cohort studies have yet evaluated the effect of TAVR on ventricular arrhythmias after resolution of severe AS, although the physiological effects on the left ventricular systolic function and filling pressures are similar to those experienced in surgical valve replacement. Our report documents that recurrent ventricular tachycardia originating from the left ventricle associated with severe aortic stenosis is a potentially reversible condition by transcatheter intervention.

Conflict of interest

The authors declare no conflict of interest.

References