

Sustained monomorphic left ventricular outflow tract tachycardia early after aortic valve replacement

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Abstract

Sustained monomorphic ventricular tachycardia (VT) after valve surgery is uncommon. Cases of focal VT or bundle-branch re-entry after aortic valve surgery have been reported. We present the case of a 60 year-old patient with an incessant outflow tract VT early after aortic valve replacement. We suggest the disease process affecting the valve and adjacent area, and/or the surgical procedure, might somehow relate to VT substrate adjacent to the aortic annulus. (Cardiol J 2012; 19, 3: 320–322)

Key words: ventricular tachycardia, aortic valve replacement, left ventricular outflow tract, scar-related-re-entry

Introduction

Catheter ablation is an established therapy for ventricular tachycardia (VT) in patients with structural heart disease [1]. Sustained monomorphic VT after valve surgery is uncommon. Cases of focal VT or bundle-branch re-entry after aortic valve surgery have been reported [2, 3]. We present the case of a 60 year-old patient with incessant VT early after aortic valve replacement (AVR) and aortocoronary bypass surgery.

Case report

A 60 year-old patient was referred to our hospital with sustained monomorphic VT one month after biologic AVR (Medtronic Hancock 29 mm) and triple aortocoronary bypass surgery due to severe aortic valve stenosis and three vessel coronary artery disease. The patient had no history of myocardial infarction (MI). The VT was noticed during car-

diac rehabilitation. The patient presented with shortness of breath and sweating. Initial attempts to terminate the VT by amiodarone and external electrical cardioversion were unsuccessful. Thereafter, ajmalin was given, which temporarily halted the VT. However, frequent premature ventricular beats with the same morphology as the clinical VT were still present. Echocardiography showed normal left ventricular ejection fraction (LVEF 65%) without wall motion abnormality during sinus rhythm. Coronary angiography was performed which excluded new coronary artery stenosis and/or graft stenosis/closure.

In the electrophysiological study, the clinical VT (Fig. 1, cycle length: 380 ms) was easily induced with programmed ventricular stimulation from the right ventricular (RV) apex and terminated with overdrive pacing. Based on the VT morphology, activation mapping was begun in the RV outflow tract using CARTO-3[®] mapping system (Biosense Webster, Inc, Diamond Bar, California, USA). However,

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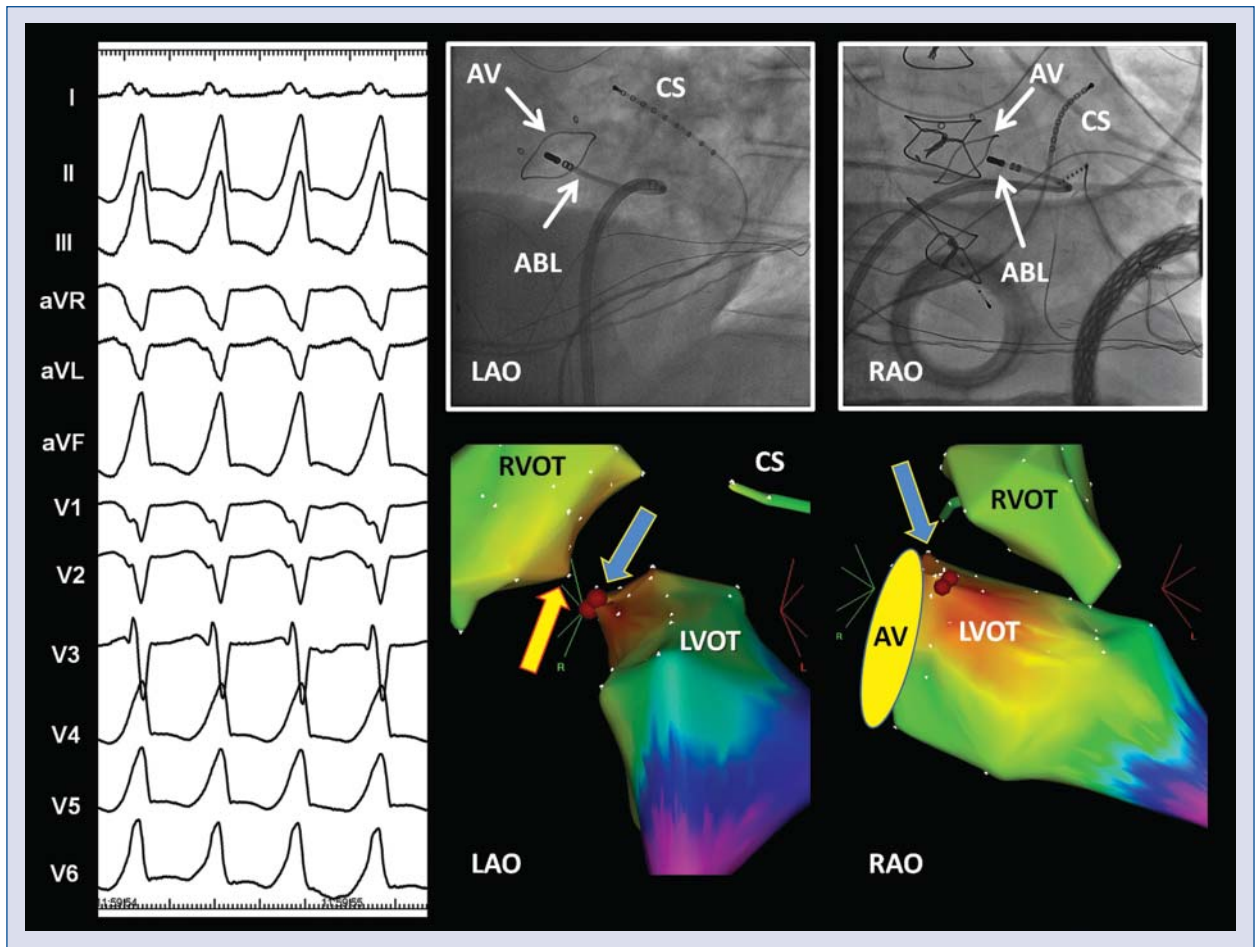


Figure 1. Left: 12-lead surface ECG during ventricular tachycardia. Upper panel on the right shows right and left anterior oblique fluoroscopic projections of the successful ablation site. Lower panel on the right shows activation mapping in the left ventricular outflow tract (LVOT) in the same projections. Yellow and blue arrows show the earliest activation sites in the right and left outflow tracts, respectively; ABL — ablation catheter; AV — prosthetic aortic valve; CS — coronary sinus; LAO — left anterior oblique; RVOT — right ventricular outflow tract.

the earliest site was only 10 ms earlier than the QRS complex (Fig. 1, right, yellow arrow). A transeptal approach was used to access the LV outflow tract. Substrate mapping revealed a very small scar area in the LV outflow tract, inferior to the right coronary cusp. Activation mapping showed the earliest activation in the same region (Fig. 1, -40 ms). Ablation at this site terminated the VT and rendered it non-inducible. The patient received an implantable cardioverter-defibrillator at another center. During the follow-up period of five months, there was no recurrence of VT.

Discussion

Sustained monomorphic VT in patients with ischemic heart disease is almost always due to macro-re-entry in the border zone of a ventricular scar

due to an old MI, which differs from the focal VT in the setting of a structurally normal heart. In a review of 496 patients referred within five years for electrophysiological study due to recurrent monomorphic VT, Eckart et al. [4] showed that only a small percentage of them (4%, 20 patients) had monomorphic VT after aortic or mitral valve surgery in the absence of MI. In 16 (80%) patients, monomorphic VT occurred 5–15 years after the valve surgery. The mechanisms of VT in these patients were scar-related re-entry in 14 cases and bundle-branch re-entry in the other two. Those patients with bundle-branch re-entry had marked systolic LV dysfunction [3, 4]. In four (20%) patients the VT occurred within a month of cardiac surgery. Only in one (5%) patient was the underlying mechanism of the VT due to re-entry [4].

Scar-related re-entry usually occurs long after valve surgery [4]. The reproducible induction of VT

with programmed ventricular stimulation and termination through ventricular overdrive pacing supports re-entry as the underlying mechanism of the clinical VT. Patients with scar-related macrore-entry usually have low-voltage regions, often with low-amplitude, fractionated electrograms. A very small scar area below the right coronary cusp could be seen in the voltage mapping of the outflow tract. Therefore we hypothesized that the mechanism of the observed VT was most probably due to microre-entry close to the prosthetic valve. We speculated that the disease process affecting the valve and the adjacent myocardium, and/or the surgical procedure, might somehow explain the observed VT substrate [4].

Conclusions

Early occurrence of monomorphic VT after valve surgery in the absence of previous MI is extremely rare. In addition to the above mentioned mechanisms, scar-related microre-entry should be

considered as a potential mechanism of VT early after valve surgery. The disease process affecting the valve and adjacent area, and/or the surgical procedure, might somehow relate to the VT substrate adjacent to the aortic annulus [4].

Conflict of interest: none declared

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