Large aneurysm of left coronary sinus of Valsalva presenting with effort-related ventricular fibrillation

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INTRODUCTION

Sinus of Valsalva aneurysms (SVAs) of the right coronary sinus account for 94% of cases, in comparison to the less-common involvement of the non-coronary sinus (5%) and left coronary sinus (1%) [1, 2]. The clinical presentation varies, depending on which sinus is predominantly affected and on associated complications such as rupture or dissection. Extremely rare SVAs have been reported, involving two or three sinuses of Valsalva and required aortic valve replacement and myocardial revascularization [3]. Acquired aneurysms are usually caused by infective, traumatic and degenerative aetiologies. Congenital SVAs are extremely rare, with predominance in the Asian population [4].

CASE REPORT

A 46-year old man displayed symptoms of ventricular fibrillation (VF) whilst running at his local gym. He was successfully resuscitated after 5 minutes of cardio-pulmonary resuscitation and a single direct current shock, and made a full recovery. He was otherwise asymptomatic. There was a family history of ischaemic heart disease but none of acute aortic syndrome or connective tissue disorder. He was a current smoker but had no other comorbidities.

Physical examination revealed a pansystolic murmur at the left sternal border. Laboratory data were normal. The chest X-ray showed a discrete convexity on the left heart border, immediately below the subaortic fossa (Fig. 1a). Transthoracic echocardiography indicated a tri-leaflet aortic valve with good systolic excursion and trivial aortic regurgitation, associated with aneurysmal sinuses of Valsalva (4.4 cm max) extending posterolaterally beneath the right ventricular outflow tract. Computed tomography (CT) coronary angiography showed an aneurysm of the right sinus of Valsalva, measuring 23 mm at its maximum diameter, and an aneurysm of the left sinus of Valsalva measuring 45 mm at its maximum diameter and containing thrombus. The dominant right coronary artery passed over the right sinus aneurysm without evidence of obstruction. The left main stem (LMS) and proximal segments of the left anterior descending (LAD) and circumflex (Cx) arteries were draped over the main body of the left coronary sinus aneurysm, and the proximal LAD appeared stretched and moderately narrowed (Fig. 1b–c).

The patient was then referred for surgical correction. The intraoperative transoesophageal echocardiogram (TOE) confirmed the presence of severe asymmetrical aortic root dilatation and mild (central) aortic regurgitation. The lateral extent of the left coronary sinus was acting as a space-occupying lesion beneath the main pulmonary artery and with extrinsic compression of the proximal LAD. The aortotomy revealed a tricuspid aortic valve that was structurally normal, apart from some hypertrophy of the noduli of Arantii. The left coronary sinus of Valsalva was in direct communication with a large false aneurysm containing organised clots, and the lower margin of this cavity was formed essentially by the annulus of the left coronary cusp of the aortic valve. The right coronary sinus was also aneurysmal but to a lesser extent (Fig. 2). A valve-sparing aortic root replacement (Yacoub remodelling procedure) was performed using 26 mm Gelweave graft, the proximal end of which was scalloped to create three neo-sinuses. The aortic valve was skeletonized and anastomosed to the graft using continuous 4-0 Prolene, and the upper end of the three commissures anchored to the graft using Teflon-buttressed 2-0 Ethibond sutures. The coronary buttons were then implanted in the usual fashion, as

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was the anastomosis between the distal end of the graft and the native aorta.

The final TOE appearance was entirely satisfactory, demonstrating only a trace of aortic regurgitation and baseline biventricular function.

The patient made an uneventful recovery and he was discharged on the 6th postoperative day, medicated with aspirin, amlodipine and losartan.

The patient was reviewed in the clinic 9 months after surgery and was asymptomatic, with an excellent exercise tolerance. He did not suffer any further episodes of VF. Transthoracic echocardiography showed good aortic valve function with trivial regurgitation and normal left ventricular function. The aortic dimensions were 2.7 cm at the level of the sinus of Valsalva, 2.7 cm at the proximal ascending aorta and 2.0 cm at the level of the aortic arch.

**COMMENTS**

Sinus of Valsalva aneurysms are extremely rare. Patients are usually asymptomatic and present only when rupture or dissection occurs—or very rarely when secondary compression of neighbouring structures occurs [4]. The aortic root is sometimes involved in retrograde dissection of the aorta but dissection that is limited to the sinus of Valsalva is rare [5]. Compression of the coronary artery by SVAs has rarely been reported to cause myocardial ischaemia [1–3]. We speculate that our patient developed critical ischaemia and VF during exercise, due to a combination of extrinsic compression of the LAD by the SVA, combined with increased metabolic demand by the myocardium.

**Conflict of interest:** none declared.

**REFERENCES**