CASE REPORT

Sudden cardiac death in asymptomatic aortic stenosis: is the valve to blame?

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SUMMARY

An active 68-year-old man with asymptomatic severe aortic stenosis and normal functional capacity on a conventional bicycle exercise test underwent a haemodynamic stress test with simultaneous invasive haemodynamic monitoring and echocardiography during supine bicycle testing as part of a research project. With exercise, the patient developed pulmonary venous hypertension and mild regional wall motion abnormalities on echocardiography. The patient terminated the test due to exhaustion. In the recovery period, he developed sustained ventricular tachycardia and became unconscious. No symptoms were present during exercise or prior to cardiac arrest. The following coronary angiogram revealed significant 2-vessel disease, and the patient subsequently underwent successful aortic valve replacement and coronary-artery bypass graft surgery.

BACKGROUND

Although it has been reported that severe aortic stenosis is associated with increased risk of sudden cardiac death in 0.4% of cases per year, the mechanism is not fully understood. Cardiac arrest may be the consequence of aortic stenosis, per se, as it may lead to alterations in cardiac haemodynamics, reduced coronary blood flow reserve and myocardial structure. However, it may also reflect not yet recognised underlying coronary artery disease.

CASE PRESENTATION

A 68-year-old man developed sustained ventricular tachycardia during a supine exercise test. He had been followed in the outpatient clinic for several years due to a severely calcified bicuspid aortic valve (mean gradient 55 mm Hg, aortic valve area 0.8 cm²) with a mildly dilated ascending aorta (figure 1). The patient was a former smoker but otherwise healthy. There was no history of sudden cardiac death or other known cardiomyopathies in the family. Three years earlier, a coronary angiogram, performed due to atypical chest symptoms, had revealed atheromatosis but no significant coronary artery disease. The patient did not receive any medication. Three years earlier, he was treated with a cholesterol-lowering statin. Treatment was stopped after 1 year due to side effects. The patient was physically active and was performing more than 10 h of road cycling on a weekly basis.

As part of a scientific investigation, the patient underwent a haemodynamic stress test with simultaneous invasive haemodynamic monitoring and

echocardiography. He exercised 15 min and reached a workload of 125 W, then stopped due to exhaustion. In the recovery period, the patient developed sustained monomorphic ventricular tachycardia at a rate of 260 bpm for about 40 s, with haemodynamic deterioration, and became unconscious. Heart rhythm spontaneously converted to sinus rhythm after a few chest compressions and consciousness returned before cardioversion was performed. The patient was admitted for further assessment of aortic stenosis valve severity and surgical evaluation, and underwent subsequently aortic valve replacement and revascularisation. He was discharged on the fifth postoperative day after an uneventful course.

INVESTIGATIONS

Routine blood tests were normal, including normal liver and kidney function.

Cholesterol levels, 5 months before the examination, had shown low-density lipoprotein 4.0 mmol/L (ref: <3 mmol/L), high-density lipoprotein 2.0 (ref: >1 mmol/L), plasma cholesterol 6.3 (ref: <5 mmol/L) and triglycerides 0.75 (ref: <2 mmol/L).

Cardiac MRI with gadolinium enhancement showed no signs of myocardial fibrosis (figure 2).

Lung function was normal, and no hypertension was present.

An echocardiogram showed a preserved ejection fraction (52% biplane Simpson method) and concentric remodelling of the left ventricle (LV) (LV mass index=124 g/m², relative wall thickness=0.48).

Longitudinal deformation was abnormal with global longitudinal strain value of -13.8%.

ECG pre-exercise revealed LV hypertrophy and during exercise the patient developed ST-segment depression. QRS duration was 107 ms (figure 3). An ECG 1 year before showed similar QRS duration.

ECG postexercise and immediately after consciousness was restored revealed pathological ST-segment depression in leads II, III, aVF and V3-V6 (figure 4).

High sensitivity troponin I measurements were elevated after 2 (175 ng/L), 6 (162 ng/L) and 24 h (25 ng/L), with a reference value <25 ng/L.

Invasive Swan-Ganz measurements showed normal pulmonary artery pressure at rest but a considerable increase during exercise, with a mean pulmonary artery pressure of 52 mm Hg and a pulmonary capillary wedge pressure (PCWP) of 29 mm Hg at maximal exercise (figure 5). Mixed

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Figure 1 Continuous-wave Doppler of highest aortic jet velocity signal (m/s).

venous oxygen saturation fell from 83% to 22%, and arterial oxygen saturation from 99% to 88% with maximal exercise.

Arterial systolic pressure showed a steady increase during the exercise test, increasing from 129 mm Hg at rest to 150 mm Hg at peak exercise.

A coronary angiogram showed a 75% left main stenosis and a 50% right coronary artery stenosis.

A 48 h Holter monitoring prior to the exercise test revealed 76 premature ventricular beats with a polymorphic ECG pattern and no episodes of ventricular tachycardia.

TREATMENT

During admission, the patient underwent aortic valve replacement and two coronary artery bypass grafts, a left internal mammary artery to left anterior descending and one saphenous vein graft to the right coronary artery.

OUTCOME AND FOLLOW-UP

At follow-up after 30 days, the patient had no cardiac-related symptoms. An echocardiogram showed a well-positioned aortic valve with no dysfunction and a preserved ejection fraction (>50%).

DISCUSSION

This case represents a witnessed episode of 'sudden cardiac death' due to ventricular tachycardia as part of exercise testing in an otherwise low-risk patient with severe aortic stenosis.

Current guidelines favour exercise testing, which is considered safe in the asymptomatic patient, as part of assessment of symptom status and adding prognostic information.

The asymptomatic patient with severe aortic stenosis is generally believed to have a good prognosis, although several studies have suggested a small risk of sudden cardiac death. In general, the risk is estimated to be approximately 0.4% per year.¹

Several mechanisms focusing on the interplay between haemodynamic burden and risk of myocardial ischaemia may predispose to sudden cardiac death in asymptomatic aortic stenosis. Although speculative, this could also possibly explain the episode of ventricular tachycardia (VT) in this case. The patient had developed significant coronary artery disease, despite being asymptomatic and with no obstruction on a recent coronary angiography. At rest, we observed that central haemodynamics were normal. However, with exercise, an abrupt increase in filling pressure and thus wall stress was noted. This, together with the decrease in arterial oxygen saturation, extensive decrease in mixed venous oxygen saturation and two vessel coronary artery disease, caused myocardial ischaemia, which is the most obvious reason for ventricular tachycardia. Fractional flow reserve (FFR) was not performed on the two coronary lesions. FFR would have provided interesting information on whether the two lesions were attributable to myocardial ischaemia. Troponin I levels were elevated as a sign of myocardial injury. A bedside echocardiogram was performed shortly after the patient regained consciousness and ejection fraction was preserved. No

Figure 2 MRI of the heart. Late gadolinium enhancement technique showing no signs of myocardial fibrosis. Long-axis and short-axis views of the left ventricle.

Figure 3 Resting ECG, 25 mm/s. Sinus rhythm, heart rate 75 bpm. QRS duration, 107 ms. Left ventricular hypertrophy pattern.

Figure 4 ECG postexercise, 25 mm/s. Sinus rhythm, heart rate 84 bpm. Pathological ST-segment depression in leads II, III, aVF and V3–V6.

ST segment elevation and no other signs of transmural ischaemia were observed. Thus, the combination of increased haemodynamic burden and coronary artery disease most likely explains the myocardial impairment and dysfunction. The remarkably low risk profile and high physical activity of the present case emphasise the difficulties in risk stratifying these patients. Although focal myocardial fibrosis, especially midwall fibrosis, detected by cardiac MRI (cMRI), has been associated with increased mortality and risk of sudden cardiac death,² no signs of myocardial fibrosis were detected on cMRI in the present

case. T1 mapping and myocardial perfusion sequences were not performed. Fibrosis can serve as a substrate for arrhythmia, but this case cannot link a pattern of diffuse fibrosis to sudden cardiac death. Moderate concentric remodelling was present, and progression rate of valve stenosis was slow. We did, however, find a broadening of the QRS interval in the ECG. Recent data from the Simvastatin and Ezetimibe in Aortic Stenosis SEAS trial suggest that the ECG contains important prognostic information in predicting sudden cardiac death.³ In SEAS QRS, prolongation to 100–119 ms was associated with a

Figure 5 Rises in pulmonary pressures during exercise. Mean pulmonary artery pressure (PAPM) and pulmonary capillary wedge pressure (PCWP). Increments of 25 W were applied for every 3 min of exercise until exhaustion. Pressures in mm Hg.

Learning points

- ▶ The risk of sudden cardiac death in asymptomatic aortic stenosis is approximately 0.4% per year.
- ▶ Risk of sudden cardiac death in aortic stenosis is likely to be a complex interplay between several potential risk factors, including myocardial fibrosis and myocardial ischaemia.
- ▶ Surveillance of QRS duration should be included as part of risk assessment in asymptomatic aortic stenosis.

fivefold increase in risk of sudden cardiac death among patients versus QRS duration <85 ms. The link between QRS broadening and ventricular arrhythmia remains speculative although in the present case this was more likely a result of myocardial ischaemia than of myocardial fibrosis.

A reduction in QT dispersion has been observed after aortic valve replacement in symptomatic patients.⁴ The predictive value for sudden cardiac death in patients with asymptomatic severe aortic stenosis is unclear. There is a considerable overlap of values between patients with different cardiac diseases, and even between healthy subjects and patients. QT-dispersion is of limited value for risk stratification of sudden cardiac death in severe asymptomatic aortic stenosis.

All of the above findings/mechanisms seem likely to play a role in modifying the risk of sudden cardiac death in patients with severe aortic stenosis. However, none of the potential risk factors seem obligatory prerequisites for sudden cardiac death in aortic stenosis. Further prospective and randomised studies are warranted in order to determine the complex interplay and significance in sudden cardiac death, and to improve risk stratification in these patients.

Competing interests None declared.

Patient consent Obtained.

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