Syncope and chest pain at rest in aortic stenosis

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Introduction

Angina pectoris occurs in 30–40% of patients with aortic stenosis, despite a normal coronary circulation.[1,2] This along with syncope, classically occurs during exercise. There are a number of suggested pathophysiological mechanisms for these symptoms, all of which lead to an imbalance between myocardial oxygen supply and demand.[3] We report an 81-year-old patient who had several episodes of chest pain occurring at rest, leading to syncope resulting in electro-mechanical disassociation (EMD) cardiac arrest. The electrocardiogram (ECG) during these episodes showed profound ST depression, leading to the hypothesis that the underlying pathophysiology was due to myocardial ischaemia caused by the aortic stenosis alone.

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Case report

An 81-year-old woman was admitted as an emergency with an episode of typical cardiac chest pain and a period of 'unresponsiveness'. A diagnosis of severe aortic stenosis had been made three years previously (echocardiographic peak gradient of 79 mmHg) and she was under regular review by a cardiologist, although she had previously been asymptomatic from the lesion. Clinical examination on admission revealed a murmur consistent with aortic stenosis and signs of congestive cardiac failure. ECG revealed left ventricular hypertrophy with strain, but no evidence of an infarct (figure 1). Serial creatine kinase (CK) measurements were within normal limits. Chest X-ray showed pulmonary oedema. Initial treatment was with diuretics, nitrates and heparin.

Several hours later the patient had a further episode of chest pain followed by syncope, with loss of cardiac output. An ECG during this episode showed profound ST depression in leads II, III, aVF, V5 and V6 with ST elevation in leads V2–V4 and aVL (figure 2).

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Figure 1. Admission ECG showing left ventricular hypertrophy with strain but no evidence of infarct

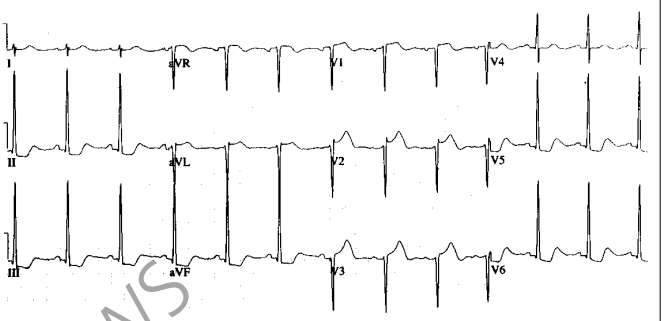
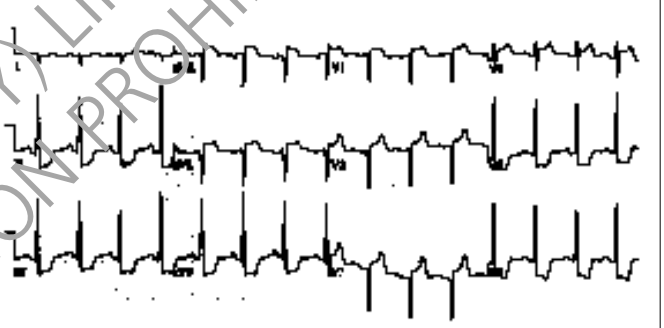


Figure 2. ECG taken several hours later showing profound ST depression



Resuscitation with adrenaline and chest compression was successful and the ECG changes resolved over the following 40 minutes. Over the next 24 hours, there were several episodes of chest pain, again leading to syncope. A coronary angiogram was performed which showed normal coronary arteries. The patient then underwent emergency aortic valve replacement with a xenograft and remains symptom free two years following the procedure.

Discussion

Angina is a common symptom in patients with aortic stenosis and normal coronary arteries and is usually associated with advanced disease and a poor prognosis.[4] There have been several proposed mechanisms for the occurrence of angina. It is generally thought there is an imbalance in myocardial oxygen

supply and demand – in the presence of severe myocardial hypertrophy with an increased vascular resistance, a reduction in subendocardial blood flow occurs. This is reflected by ECG changes of ST depression.

Not all patients with aortic stenosis develop angina. Julius *et al.*[3] performed a retrospective analysis of haemodynamic factors in 61 patients with angina and aortic stenosis. Patients studied had no significant coronary artery disease. The study demonstrated that patients who developed angina appeared to have smaller coronary arteries and a lower coronary flow reserve than those patients with aortic stenosis and no angina. Inadequate left ventricular hypertrophy (small left ventricular muscle mass that is inappropriate for associated high wall stress) with high systolic and diastolic wall stresses were also found in angina patients. This suggested that myocardial ischaemia may be due to hypoperfusion of the myocardium under high flow and high demand situations.

The accepted mechanism for syncope in these patients is a vasodepressor response from stimulation of left ventricular baroreceptors, resulting in reflex hypotension and bradycardia.[5] Several other theories exist, e.g. hypersensitive carotid sinus complete atrioventricular block and ventricular arrhythmias. Myocardial ischaemia is probably overlooked as an important substrate by which the syncope can be precipitated.[6] A review of several cases of syncope by Baltazar *et al.* showed that ischaemia ST changes or symptoms of angina were evident before the occurrence of syncope, hypothesising that myocardial ischaemia may trigger left ventricular baroreceptors, accompanying the vasodepressor reflex.

Both symptoms are normally associated with exercise and, along with breathlessness, form the classic triad of exertional symptoms in aortic stenosis. Wilmshurst *et al.*[7] even went as far to state: "When patients with aortic stenosis experience syncope unrelated to exertion, the symptom is usually unrelated to the valve disease and another cause should be sought". This was based upon data from consecutive patients who all had syncope, occurring on exertion or at rest, undergoing aortic valve replacement. Valve replacement alleviated the symptom in the group with exertional syncope, but approximately two-thirds of the group with syncope at rest had recurrence of their symptoms.

Our patients experienced both chest pain and syncope at rest; after valve replacement the symptoms disappeared. This would suggest the stenosis of the aortic valve was the underlying cause of these symptoms. The mechanism for these episodes is consistent with myocardial ischaemia due to ECG showing profound ST depression during these episodes.

Key messages

- Angina pectoris occurs in 30–40% of patients with aortic stenosis
- Myocardial ischaemia can cause syncope in aortic stenosis
- Valve replacement can alleviate syncope in such patients

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