CASE REPORT

Coronary spasm: a case of transient ST elevation and syncopal ventricular tachycardia without angina

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We report the case of a 60-year-old male with recurrent pre-syncope, referred with a provisional diagnosis of carotid sinus syndrome on the basis of a 4 s asystolic pause following carotid sinus massage. On repeat Holter monitoring there was ST-segment elevation followed by episodes of polymorphic ventricular tachycardia during a mild episode of pre-syncope. Coronary angiography showed mild right coronary artery irregularity without significant stenosis. An automatic cardioverter defibrillator was implanted and high dose combined vasodilator therapy was commenced. At follow-up 18 months after implantation, the device has recorded no episode of tachycardia and the patient reports no recurrence of symptoms.

KEYWORDS
Coronary spasm; Sudden death; Ventricular tachycardia; Ventricular fibrillation; Syncope; Carotid sinus syndrome

Introduction
Prinzmetal or variant angina is an unusual syndrome of cardiac pain secondary to myocardial ischemia that occurs almost exclusively at rest, is not usually precipitated by physical exertion or emotional stress, and is associated with ST-segment elevation.1 The syndrome is associated with ventricular tachycardia and fibrillation.2 Patients with variant angina differ from those with typical angina in that traditional risk factors for atherosclerosis are often absent.

Case report
A 60-year-old man underwent investigation of recurrent episodes of pre-syncope and one episode of syncope. Clinical examination and initial investigations were normal. The 12-lead ECG showed mild first degree AV block and right bundle branch block. Echocardiogram, chest X-ray and routine biochemical and haematological analyses were normal. No symptoms occurred during the initial Holter recording which was normal.

Carotid sinus massage, performed at a local referral centre, produced sinus bradycardia and transient atrioventricular block, with a pause of 4 s. Pre-syncope similar to the patient’s spontaneous symptoms occurred in association with this pause. The patient was referred to us with view to pacemaker implantation.

While awaiting admission to our centre, a repeat 24 h ambulatory ECG was performed by the referring cardiologist. The only available Holter monitor was a 12-lead system. On this occasion, episodes of mild but otherwise typical symptoms occurred during the period of monitoring. These symptoms corresponded to episodes of fast polymorphic ventricular tachycardia (Figure 1). Each episode was preceded by ST-segment elevation in leads II, III, and aVF with reciprocal ST-segment depression in the anterior leads (Figure 2). All episodes of ventricular tachycardia occurred during the resolution of a period of ST-segment elevation of at least 30 s duration.

Coronary angiography demonstrated an angiographically normal left coronary system, with irregularity without significant stenosis throughout the proximal part of a dominant right coronary artery. Combined vasodilator therapy with diltiazem and nitrates was commenced, and the patient underwent implantation of a dual-chamber defibrillator. At follow-up 18 months after implantation, the device has recorded no episode of tachycardia and the patient reports no recurrence of symptoms.

Discussion
The symptoms of variant angina are similar to those of classical angina but occur without provocation and may be accompanied by syncope. When this is present, syncope is typically associated with inferior ST-segment elevation and either AV block or ventricular tachyarrhythmias.3-5
Figure 1  A brief episode of polymorphic ventricular tachycardia associated with mild pre-syncope.

Figure 2  ST elevation in the inferior leads with reciprocal ST depression in the anterior leads preceded each episode of polymorphic ventricular tachycardia.
Ventricular arrhythmias tend to occur during the reperfusion phase of the vasospastic episode rather than at the time of maximum ischemia. The treatment of vasospastic angina is based on non-specific vasodilators, particularly the non-dihydropyrridine calcium channel antagonists and the nitrates. Coronary stenting is helpful in patients with discrete proximal fixed obstructive lesions and may be indicated in patients with clinically severe, angiographically documented spasm refractory to aggressive pharmacologic management.

Our patient showed none of the recognised indications for coronary stenting, but posed a particular challenge because of the aggressive nature of the ventricular arrhythmias documented and because of the lack of anginal symptoms. The absence of such symptoms denied us any reliable guide to the efficacy of vasodilator therapy in reducing the risk of sudden death. Although unselected patients with variant angina have a reasonable prognosis with 89–97% survival at 5 years, the risk of sudden death is high in patients who exhibit serious arrhythmias during episodes of chest pain.

The positive response to carotid sinus massage in our patient, and the resulting decision to implant a pacemaker illustrate the hazards of reliance on this test. But for the occurrence of an event during repetition of an ambulatory ECG, the patient would have undergone implantation of a dual chamber pacemaker and no other therapy. Asystole for more than 5 s can occur in response to carotid sinus massage in healthy persons. Implantation of a pacemaker solely on the basis of this test exposes some patients to the hazards and inconvenience of an unnecessary procedure. As our case illustrates, it may also expose some to the greater danger of missing an important diagnosis.

Conflict of interest: None declared

References