ST elevation myocardial infarction after coronary artery spasm with no clear trigger

Introduction

Coronary artery vasospasm is a mimic of coronary artery disease both in terms of clinical presentation and associated electrocardiogram changes. It is most strongly implicated in microvascular dysfunction, but in rarer instances it can provoke ST elevation myocardial infarction, ventricular dysrhythmias, cardiogenic shock and death. However, in most of these contexts there is an established precipitant such as cocaine use. This article presents a rare case of a patient who presented with profound, dynamic ST elevation secondary to coronary artery vasospasm and without a clear precipitant.

Discussion

Coronary artery vasospasm, otherwise known as Prinzmetal angina, is a condition in which smooth muscle vasculature tends to contract (Prinzmetal et al, 1959). The pathogenesis is distinctly different from coronary artery occlusion as a sequelae of atherosclerosis and acute plaque rupture, although clinical presentations can be remarkably similar. Two-thirds of patients with Prinzmetal angina have concurrent atherosclerosis which is often mild and not in correlation with the degree of symptoms (Nobuyoshi et al, 1992). Subjects are generally younger than those with classic coronary disease. There is also a reported association with tobacco smokers. Indeed the patient described in this case presentation was relatively young and a heavy smoker.

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In the context of dynamic electrocardiogram changes, aetiology is less attributable to microvascular dysfunction and most document exposure to an established precipitant such as cocaine (Benzaquen et al, 2001). A distinguishing feature of Prinzmetal angina, as seen in this case, was the prompt resolution of symptoms and electrocardiogram changes with vasodilator therapy. Vasospasm, however, can result in impaired blood flow to the territories supplied by the affected vessel. If significant, this can cause infarction with secondary troponin rise. Of further concern, this ischaemic burden can increase the propensity for dysrhythmias. One study suggests that

CASE REPORT

A 61-year-old man collapsed with syncope while walking. He complained of preceding central chest pain radiating to the left arm and neck. There was associated breathlessness, sweating and nausea. He recovered consciousness spontaneously. Risk factors included hypercholesterolaemia and a smoking history of 40 pack years. He had no prior history of cardiovascular disease and was not on any regular medications. On admission to the emergency department he was haemodynamically stable and cardiovascular examination was unremarkable. Initial 12-lead electrocardiogram trace showed new T-wave inversion in lead III. Chest X-ray showed mild cardiomegaly but no evidence of pulmonary oedema. Blood testing on admission, including full blood count and urea and electrolytes, returned normal.

A 12-hour troponin returned positive at 2282 ng/litre (normal <50 ng/litre). He was transferred to coronary care on telemetry and treated as having had a non-ST elevation myocardial infarction. Secondary prevention pharmacotherapy was commenced as per National Institute for Health and Care Excellence (2013) guidelines. This included dual antiplatelets, high-dose statin, bisoprolol, ramipril and glyceryl trinitrate spray. Subsequent angiography demonstrated entirely normal coronaries with preserved left ventricular function. Inpatient 24-hour ambulatory electrocardiogram monitoring was unremarkable with no evidence of dysrhythmia. He made an uneventful recovery and was discharged home 3 days later.

Four days post-discharge he presented with a further episode of central chest pain followed by transient syncope. Electrocardiogram tracing on admission confirmed anterolateral ST elevation (leads VI–V6) with reciprocal ST depression and ventricular bigeminy (*Figure 1*). Administration of a single puff of glyceryl trinitrate spray resulted in resolution of dynamic electrocardiogram changes (*Figure 2*) and alleviation of chest pain. He was commenced on an intravenous glyceryl trinitrate infusion.

On the following day, he experienced further chest pain with secondary syncope. Electrocardiogram tracing confirmed anterior ST elevation, with acute deterioration into pulseless electrical activity. Following adminstration of intravenous 1:10 000 adrenaline 1 mg and a 2-minute cycle of cardiopulmonary resuscitation as per Advanced Life Support guidelines his rhythm reverted to pulsed ventricular tachycardia. Amiodarone 300 mg intravenous was administered which resulted in reversion to normal sinus rhythm and restoration of cardiac output.

Repeat angiography was performed which excluded acute plaque rupture. However, there was luminal narrowing in the left anterior descending territory suggestive of coronary vasospasm. Echocardiography showed preserved left ventricular systolic function with no regional wall motion abnormalities or valvular disease. Blood electrolytes, including calcium and magnesium, were within normal ranges. A formal diagnosis of ischaemia-induced arrhythmia precipitated by coronary artery vasospasm was made. An implantable cardioverter defibrillator was inserted and pharmacological therapy including isosorbide mononitrate and amlodipine was initiated. No further dysrhythmias were noted during admission. He was discharged with planned review in the outpatient setting.

Figure 1. Electrocardiogram at the onset of chest pain confirming anterolateral ST elevation with reciprocal changes.

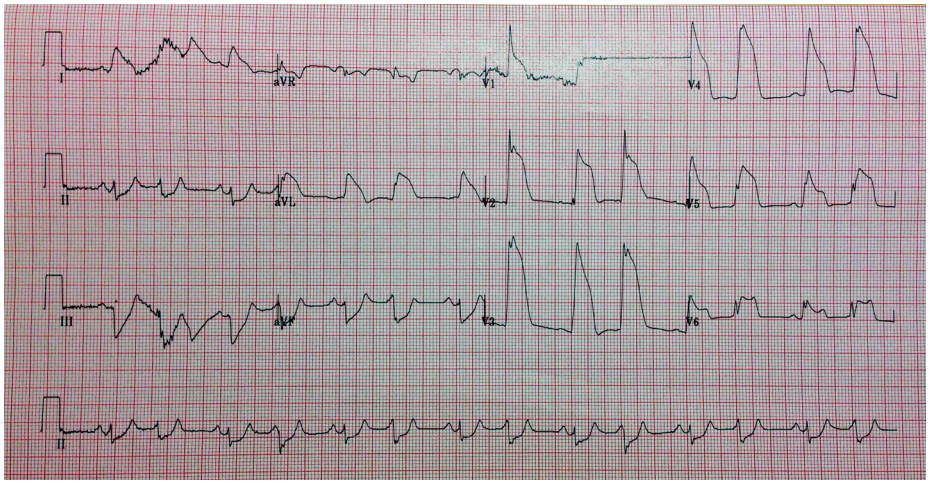
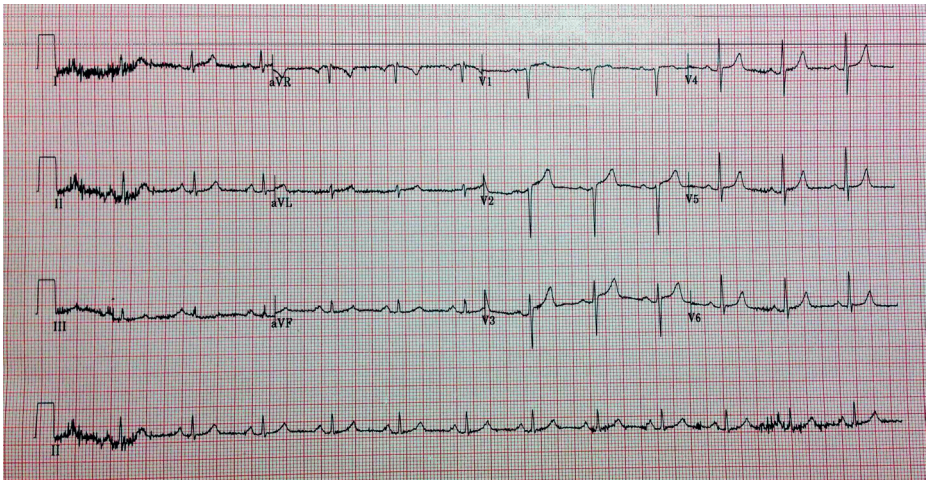


Figure 2. Electrocardiogram immediately after resolution of chest pain with a single dose of glyceryl trinitrate spray.



7% of out of hospital cardiac arrests are secondary to vasospasm (Chevalier et al, 1998).

Diagnosis can be challenging as attacks are unpredictable, transient and not typically inducible with exercise. Findings on angiography remain the gold standard in making a diagnosis (Chen and Pinto, 2003). Several provocative tests have been developed in order to induce vasospasm. The most commonly used agents include ergonovine and acetylcholine (Bertrand et al, 1982). Both these agents appear to have an acceptable safety profile with no documented reports of irreversible complications (Sueda et al, 2004). In this patient's case, angiographic findings after injection of contrast were strongly suggestive of vasospasm so further provocation testing was not performed. In routine practice, however, intracoronary administration of a nitrate or calcium-channel antagonist is beneficial to demonstrate normalization of vasculature.

For preventative therapy, dihydropyridine calcium-channel blockers with or without concomitant long-acting nitrates are established as effective treatment options (JCS Joint Working Group, 2008). Doses should be gradually increased according to tolerance and not acutely withdrawn because of the risk of lethal arrhythmias and sudden cardiac death in these patients. For those who develop significant dysrhythmias in the context of vasospasm, further management is determined by the underlying rhythm. This patient was noted to be in pulsed ventricular tachycardia immediately post-cardiopulmonary resuscitation. Therefore insertion of an implantable cardioverter defibrillator alongside standard medical therapy was appropriate based on current evidence (Matsue et al, 2012). **BJHM**

LEARNING POINTS

- Coronary artery vasospasm is a known mimic of atherosclerotic disease.
- This case describes the occurrence of profound, dynamic electrocardiogram changes of ST elevation in the context of vasospasm, but in the absence of a clear precipitant.
- Diagnosis can be challenging, although provocative testing during angiography is helpful to guide decision making.
- Those with associated ventricular dysrhythmias may require an implantable cardioverter defibrillator pre-discharge to reduce the risk of sudden cardiac death.

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