CASE REPORT

Coronary artery spasm leading to life threatening arrhythmias

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Abstract

A 39 year old woman sustained life threatening arrhythmias associated with coronary artery spasm. On both occasions she was attending hospital outpatient clinics and was successfully resuscitated. Electrocardiography performed during further episodes of pain suggested that spasm could occur in either the right or left coronary artery.

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A 39 year old woman with a history of mild chronic renal failure and poorly controlled hypertension collapsed while attending a gynaecology outpatient clinic. Immediately before this event she had complained of central chest pain and lightheadedness, she had then vomited and collapsed. She had no pulse or respiratory effort. Cardiopulmonary resuscitation was started, and adrenaline and atropine were administered, with restoration of cardiac output and respiration before cardiac monitoring was available.

On admission to the coronary care unit she was confused and uncooperative. Her pulse was 120 beats/min and blood pressure 140/70 mmHg. Her routine medication included quinapril, atenolol, bendrofluazide, and norethisterone for menorrhagia; there had been no recent alteration to her medication. She was a heavy smoker and had in the past consumed large quantities of alcohol. Several years previously she had been diagnosed as having Raynaud's phenomenon. Serum electrolytes including magnesium were within normal limits, and renal function was mildly impaired. Serial electrocardiograms (ECG) and cardiac enzymes showed no evidence of myocardial infarction. Echocardiography showed normal cardiac structure and left ventricular contraction. A tilt test, ventilation perfusion scan, and Doppler ultrasound of the leg veins were all negative. Inpatient, 24 hour ambulatory ECG monitoring was performed, which revealed no evidence of ST segment depression or arrhythmia. She made an uneventful recovery and was discharged home.

Three weeks later while attending the renal clinic she again complained of central chest tightness, shortness of breath, and sweating. She collapsed and again had no pulse or respiratory effort. Cardiopulmonary resuscitation was started and the patient intubated. An ECG tracing showed sinus bradycardia with broad bizarre QRS complexes and ST elevation in the inferolateral leads (fig 1). Adrenaline and atropine were administered, which led to spontaneous respiration and restoration of cardiac output. A 12-lead ECG performed after recovery showed only sinus tachycardia.

Twenty four hours after readmission to the coronary care unit, the patient complained of further chest pain, which was associated with profound ST elevation in the anterior leads (fig 2) and self terminating episodes of broad complex tachycardia. The pain and ECG changes resolved within 30 minutes of intravenous nitrate administration. The following day coronary angiography was performed via the right femoral artery. The left coronary system was normal. After two injections in the right coronary artery the patient complained of chest pain, and the ECG showed bradycardia with broad QRS complexes and ST elevation in the inferior leads, similar to the pattern in fig 1. A further injection into the right coronary

Figure 1 Electrocardiogram taken at time of collapse demonstrating sinus bradycardia, broad QRS complexes, and ST elevation in the inferior leads.

Figure 2 Electrocardiogram taken during chest pain demonstrating widespread ST elevation in the anterior chest leads

artery confirmed ostial spasm; therefore, intra-coronary nitrates were given before the catheter was disengaged. The patient's symptoms and ECG changes rapidly settled and further injections confirmed that the right coronary was large and normal.

The patient was started on calcium channel antagonists and her β blocker discontinued. An exercise tolerance test was performed and she completed 10 minutes of the Bruce protocol, achieving 90% of target heart rate with no chest pain or ECG changes. She made an uneventful recovery and was discharged home. Follow up at three months found her to be pain free with no episodes of collapse.

Discussion

Coronary artery spasm leading to myocardial ischaemia, infarction or life threatening arrhythmias was first characterised in 1959 by Prinzmetal.¹ Variant angina is associated with cigarette smoking² and the use of cocaine.³ In some subjects the basal coronary artery tone is often increased. The coronary arteries can appear normal at angiography, but in some cases the site of spasm is adjacent to atheromatous plaques. Subjects with variant angina are generally younger than those with classic angina. Episodes of pain are often severe and tend to occur between midnight and early morning, there is often no precipitating factor. It has occasionally been reported in subjects who also suffer from migraine or Raynaud's phenomenon, and some patients find emotional stress can lead to spasm. The patient in the present case was a heavy smoker with Raynaud's syndrome who reported she was anxious about visiting outpatient clinics on both occasions.

Coronary artery spasm is a relatively rare cause of out of hospital cardiac arrest, although accurate figures are difficult to ascertain. In the

original account of variant angina, Prinzmetal described a patient with polymorphic ventricular tachycardia¹; in a more recent study of 356 survivors of out of hospital cardiac arrest Myebug *et al* found five subjects with coronary spasm associated with myocardial ischaemia and ventricular arrhythmias.⁴ Furthermore, in a study of silent ischaemia associated with coronary artery spasm, Egashira *et al* found that spontaneous arrhythmias occurred during 9% of silent ischaemic episodes detected by ambulatory monitoring.⁵

The precise nature of the arrhythmia precipitated by coronary artery spasm and resulting in out of hospital cardiac arrest is unknown. However, in a study by Igarashi *et al* cardiac rhythm was documented in eight patients: asystole was present in five, polymorphic ventricular tachycardia in one, ventricular fibrillation in one, and idioventricular rhythm in the remaining subject.⁶

Our patient had ST elevation in the inferolateral leads at the time of her second collapse, suggesting spasm of the right coronary artery, and anterior ST elevation during a further episode of chest pain implying spasm in the left coronary system. The inferolateral ST elevation was associated with a bradycardia, and the anterior ST elevation with a self terminating broad complex tachyarrhythmia. At the time of angiography, spasm of the right coronary artery only was demonstrated precipitating a bradycardia.

In patients with normal coronary arteries and variant angina, β blockade may prolong or precipitate episodes of coronary spasm. Calcium channel antagonists are very effective at preventing coronary spasm and thus the associated life threatening arrhythmias.⁷ Our patient had been on atenolol for some time before her first admission and this was replaced by a calcium channel antagonist after the diagnosis was confirmed; she has experienced no further difficulties.

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