

Coronary spasm as a cause of sudden death induced by malignant ventricular arrhythmia

JOSEPH JOHN, GERRY C KAYE

Introduction

Coronary artery spasm is an uncommon presentation of angina and may be associated with other vasospastic diseases such as Raynaud's disease. It is widely accepted that local imbalance of production and removal of nitric oxide (NO) and other endothelium-derived factors is generally responsible for the arterial spasm in variant angina.^{1,3} Very rarely, diffuse spasm can herald ventricular arrhythmias due to sudden reduction in perfusion.^{4,5}

Br J Cardiol 2005;**12**:230-1

Case report

A 53-year-old woman was admitted to our coronary care unit with a history of persistent episodic retrosternal chest pain of several hours' duration during the previous night. She was an ex-smoker and had no history of diabetes or hypertension nor did she have any significant family history of ischaemic heart disease. She had a previous history of Raynaud's disease and had been treated with calcium channel blockers for several years.

She also gave a history of intermittent chest pain of several years' duration, occurring mostly at rest; indeed, she had a few admissions to our acute assessment unit with chest pain. On each occasion, resting ECGs and serum troponin T were found to be normal. Six months prior to the present admission she had undergone a diagnostic coronary angiogram. During the first injection of dye into the left coronary tree, left anterior descending artery and left circumflex artery, all appeared initially normal, but then spastic changes were demonstrated along the course of the arteries (figure 1). These changes were associated with severe chest pain and hypotension. Intracoronary nitrates reversed the spasm, with resolution of symptoms (figure 2). She was subsequently discharged home with high-dose calcium channel blockers and oral nitrates.

Following this latest admission, she was pain-free for the first 12 hours but complained of intermittent chest pains associated with global ST-segment depression suggestive of ischaemia. A nitrate infusion was commenced, which resulted in symptomatic

Figure 1. Angiogram showing spastic changes along the course of the coronary arteries

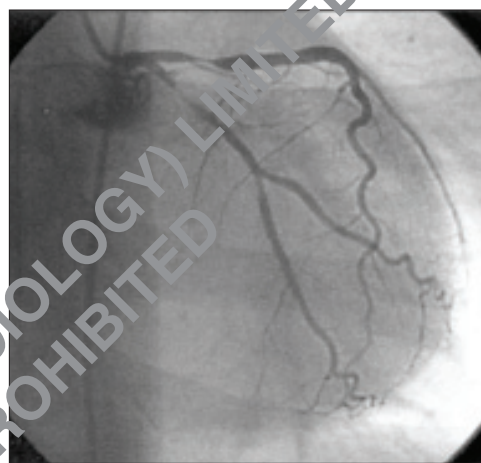
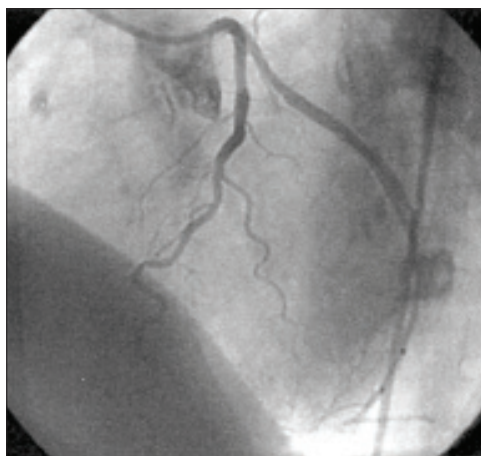


Figure 2. Angiogram showing reversion of the spasm after intracoronary nitrates



Castle Hill Hospital, Cottingham, Hull, HU16 5JQ.
Joseph John, Specialist Registrar
Gerry C Kaye, Consultant Cardiologist
Correspondence to: Dr J John
(e-mail: Josephjohn99@hotmail.com)

improvement and resolution of electrocardiogram (ECG) changes. A few hours later, she had an unheralded syncopal attack with ventricular tachycardia, which reverted back to sinus rhythm spontaneously. However, she continued to have intermittent episodes of pain with brief episodes of pulseless electrical

activity (PEA) and sustained ventricular tachycardia resistant to treatment. A few hours later she developed global ST-segment elevation on the ECG with tonic clonic seizures and haemodynamic collapse. Cardiopulmonary resuscitation was unsuccessful. It was clear, from the background history and investigations, that she had suffered global spasm of the coronary arteries leading to sudden-onset malignant ventricular arrhythmias.

Discussion

In 1959 Prinzmetal and associates described an unusual syndrome of cardiac pain secondary to myocardial ischaemia that occurs almost exclusively at rest, is associated with electrocardiographic ST-segment elevations, and that is not usually precipitated by physical exertion or emotional stress. Variant angina pectoris has been demonstrated convincingly to be due to coronary artery spasm. The latter causes a transient, abrupt, marked reduction in the diameter of an epicardial coronary artery, resulting in myocardial ischaemia.^{2,6}

The diagnosis of variant angina depends on the detection of ST-segment elevation with pain. In some patients, episodes of ST-segment depression follow episodes of ST-segment elevation and are associated with T-wave changes. Many patients exhibit multiple episodes of asymptomatic ST-segment elevation (silent ischaemia). Transient conduction disturbances may occur during episodes of ischaemia. Ventricular ectopic activity is more frequent during longer episodes of ischaemia. Spasm of a proximal coronary artery with resultant transmural ischaemia has been convincingly documented arteriographically and is the diagnostic hallmark of Prinzmetal's angina. Spasm is most common in the right coronary artery, and it may occur at one or more sites in one artery or in multiple arteries simultaneously.^{2,6}

Management of coronary spasm can be difficult in some patients. They usually respond well to nitrates, and long-acting nitrates are useful in preventing attacks. Response to beta block-



Key messages

- Coronary artery spasm is uncommon as a cause of angina
- It is more prevalent in women and usually has a benign course
- Multiple artery spasm can result in acute severe ischaemia
- Malignant ventricular arrhythmia and death are rare

ers in patients with Prinzmetal's angina is variable. Calcium channel blockers are effective in preventing the coronary artery spasm of variant angina and should ordinarily be prescribed in maximally tolerated doses.

Conflict of interest

None declared.

References

1. Kim Lamping. Enhanced contractile mechanisms in vasospasm. Is endothelial dysfunction the whole story? *Circulation* 2002;**105**:1520-2.
2. Ogawa H, Yasue H, Okumura K *et al*. Platelet-derived growth factor is released into the coronary circulation after coronary spasm. *Coron Artery Dis* 1993;**4**:437-42.
3. Itakano H, Nakamura T, Satou T *et al*. Regional myocardial sympathetic dysinnervation in patients with coronary vasospasm. *Am J Cardiol* 1995;**75**:324-9.
4. Cohen M. Variant angina pectoris. In: Fuster V, Ross R, Topol EJ (eds). *Atherosclerosis and coronary artery disease*. Philadelphia: Lippincott-Raven, 1996: 1367-76.
5. Mayer S, Hillis LD. Prinzmetal's variant angina. *Clin Cardiol* 1998;**21**:243-6.
6. Cox ID, Kaski JC, Clague JR. Endothelial dysfunction in the absence of coronary atheroma causing Prinzmetal's angina. *Heart* 1997;**77**:584.