CASE REPORT

Coronary spasm in acute myocardial infarction

G Y H Lip, K K Ray, M F Shiu

Abstract

A 30 year old man had an acute anterolateral myocardial infarction following which he developed unstable angina requiring percutaneous transluminal coronary angioplasty. He subsequently developed further angina with recurrence of coronary artery lesions that were reversed by intracoronary nitrate. A diagnosis of prinzmetal (vasospastic) angina was made and this had been the apparent cause of his myocardial infarction. He was treated with a calcium antagonist and an oral long acting nitrate with resolution of symptoms. He remained well and symptom free, and was reviewed in the outpatient clinic six weeks after discharge without problems.

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Keywords: coronary spasm; acute myocardial infarction; syndrome X; prinzmetal's angina

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There have been numerous reports of angina or myocardial infarction despite normal coronary arteries. Many have been due to causes such as syndrome X, coronary artery emboli, or coronary vasospasm. Nevertheless, persisting coronary vasospasm responding only to intracoronary nitrates is unusual.

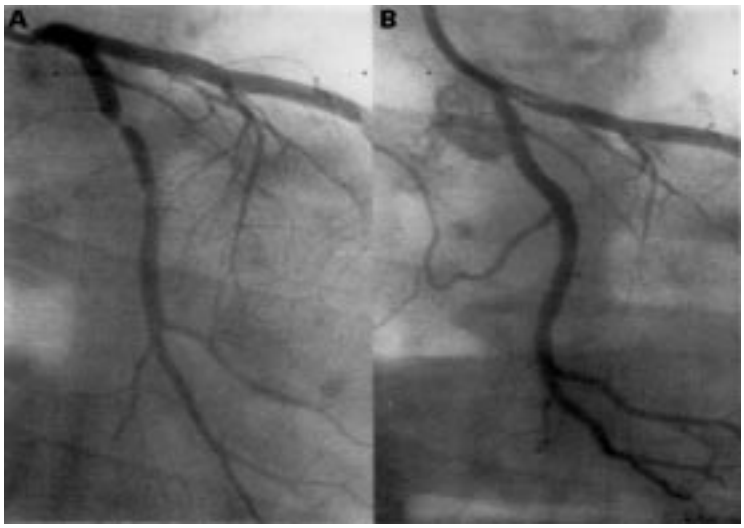

Figure 1 Coronary angiography showing a severe proximal circumflex artery stenosis (99%) (right anterior oblique projection) (A) before PTCA; (B) repeat left coronary angiography following successful PTCA.

We report a patient with coronary vasospasm in the setting of acute myocardial infarction who was being treated with systemic nitrates. Spasm was reversed only following administration of intracoronary nitrate.

Case report

A 30 year old man was admitted as an emergency with a two week history of left sided chest pain that occurred while walking his dog. Because of a history of constipation followed by loose stool, epigastric tenderness, bloating, and discomfort, he was initially treated for possible gastroenteritis and admitted to a surgical ward. He did not have a fever, his blood pressure was 115/75 mm Hg, his heart sounds were normal, and abdominal examination was unremarkable. There was no history of drug intake or substance abuse, including cocaine. Chest radiography was normal. Electrocardiography was also performed but not reviewed until the following morning, when it became apparent there were Q waves in leads V1, V2, and V3, with T wave inversion in leads I, aVL, and V4–6. Cardiac enzymes measured as an emergency showed an increase in creatine kinase to a peak of 1189 IU/l. The patient was transferred from the surgical ward to the coronary care unit for cardiac monitoring.

Later that evening he developed further severe chest pain associated with inferolateral ST elevation. There was little response to sublingual and systemic (intravenous) nitrates and the pain required relief with intravenous opiates. He was transferred to a tertiary referral centre for urgent cardiac catheterisation, which initially demonstrated a severe proximal circumflex artery stenosis (99%) and a proximal right coronary artery stenosis of 80–90% (fig 1, 2). His left main coronary artery and left arterial descending artery were normal.

We performed percutaneous transluminal coronary angioplasty (PTCA) to the circumflex lesion using a Voda left 3.5 guide catheter, Phantom wire, and 3.5 mm Viva balloon (Boston Scientific, Cambridge, Massachusetts, USA). After a single inflation, there was a good angiographic result. We therefore proceeded to perform PTCA to the right coronary artery lesion using a Judkins right 4.0 guide catheter and the same wire and balloon. Again, after a single inflation, there was an excellent angiographic result. Balloon inflations were associated with angina and ECG changes in the inferolateral leads. The patient remained well and

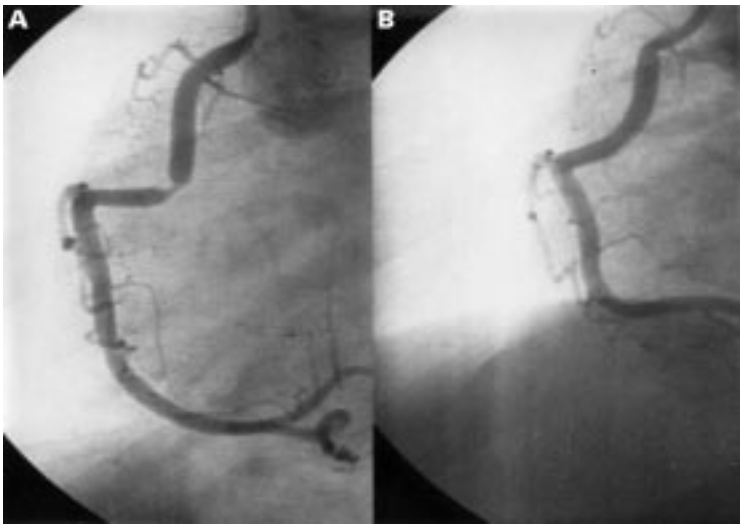

Figure 2 (A) Coronary angiography showing a proximal right coronary artery stenosis of 80–90% (left anterior oblique projection). (B) Repeat coronary angiography following successful PTCA.

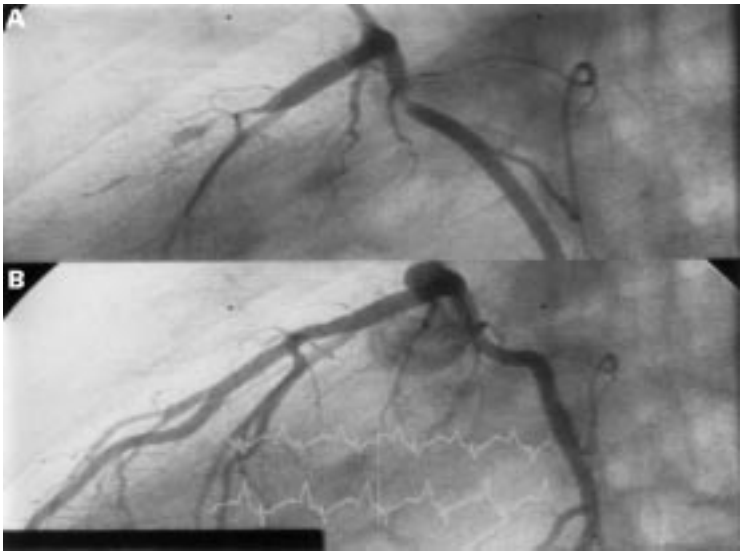

Figure 3 (A) Repeat coronary angiography demonstrating a completely occluded left anterior descending artery from the mid-segment, and a tight stenosis in the proximal circumflex artery at the site of the previous PTCA (left lateral projection). (B) Completely healthy and normal left coronary system after intracoronary nitrate.

pain free the following day, and his arterial sheath was removed. In view of a history of asthma he was not started on β blockers.

Later that evening he complained of more chest pain with little relief from sublingual nitrates. A repeat ECG showed ST elevation in the inferior and lateral leads. Repeat coronary angiography demonstrated a completely occluded left arterial descending artery from the mid-segment, and a tight stenosis in the proximal circumflex artery at the site of the previous PTCA (fig 3). His right coronary artery appeared normal. Despite sublingual and systemic nitrates, we gave him intracoronary nitrate (Tridil 300 μg; Du Pont, Stevenage, Herts, UK) into the left coronary artery. After 30 seconds, there was a completely healthy and normal left coronary system. There was no evidence of coronary bridging on dynamic images. A diagnosis of prinzmetal (vasospastic) angina was made and this had been the apparent cause of his myocardial infarction. He was initially treated with sustained release verapamil 240 mg daily but developed further chest pain with minor ECG changes that resolved with intravenous nitrate (0.05% Isoket; Schwartz, Chesham, Bucks, UK). He was therefore given an oral long acting nitrate (Imdur 60 mg daily; Astra, Watford, Herts, UK) with resolution of symptoms. Echocardiography demonstrated mild hypokinesia of the lateral wall of the left ventricle. The patient completed stage 3 of a modified Bruce protocol exercise test with no chest pain or ECG changes. A 24 hour Holter monitor with ST segment analysis demonstrated no significant ECG changes. He remained well and symptom free, and was reviewed in the outpatient clinic six weeks after discharge without problems.

Discussion

Myocardial infarction is predominantly caused by atherosclerotic vascular disease. However, in a few patients selective coronary angiography shows normal coronary arteries with no evidence of atherosclerotic lesions.[1] These patients often have unusual causes of myocardial infarction, such as coronary artery emboli, coronary artery thrombosis, syndrome X, or coronary artery vasospasm. Horimoto *et al* reported a case of coronary spasm implicated as a cause of both coronary thrombosis and myocardial infarction.[2] This raises the possibility that some young subjects may be particularly thrombus prone and this initiates spasm; some evidence for this comes from the demonstration that coronary spasm may be associated with thrombogenicity.[3] Coronary spasm has also been implicated in prinzmetal's variant angina, ventricular arrhythmia, sudden death, and acute coronary reocclusion or restenosis following PTCA.[4] Finally, coronary spasm and variant angina may be associated with other vasospastic disorders, such as migraine headaches and Raynaud's phenomenon.

In many cases, coronary artery spasm responds to nitrates or calcium antagonists.[4–6] The latter have been reported to be useful in reducing the risk of cardiac arrest and sudden death in patients with variant angina.[4 5] In our patient, substantial amounts of sublingual nitrate were administered before the first coronary angiography and he had been started on intravenous nitrate infusion. Despite this, he continued to experience severe chest pain and the initial selective coronary angiography suggested severe lesions in the right coronary artery and circumflex branch of the left coronary artery, leading to what initially appeared to be a successful two vessel PTCA. At the second cardiac catheterisation, the recurrence of the stenosis at the circumflex artery and the mid-left anterior descending artery occlusion, with the latter occurring where there was no previously documented arterial lesion, led to the suspicion of coronary vasospasm. His subsequent response to intracoronary nitrates and clinical treatment with oral verapamil and nitrates are further support of this diagnosis. Our case has many similarities

to an Italian report of a 48 year old man who underwent coronary angiography five months after an anterior myocardial infarction, which showed normal coronary arteries. This was followed by sudden spasm of both left anterior descending and circumflex arteries at the end of the procedure, which was reversed by intracoronary nitrates.[7]

While administration of intracoronary nitrate before coronary angiography is not standard practice in the UK, it is commonly done in mainland Europe. Intracoronary administration provides a high local concentration of nitrates that works on endothelial derived relaxing factor[8] and this may have accounted for the prompt reversal of the initial angiographic appearances at the second cardiac catheterisation. There was no associated precipitating cause of spasm in our patient. It has been postulated that coronary artery spasm may occur in the absence of any inciting chemical or spasmogen, perhaps reflecting an abnormality in an intrinsic tone regulation involving pacemaker cell discharge, intercellular conduction through gap junctions, and cycling of extracellular and bound calcium.[9] There was no history to suggest vigorous exercise as a precipitant, hyperventilation, blunt chest trauma, drug intake or substance abuse in our patient. These factors may be associated with coronary spasm and variant angina; in particular, there is evidence that cocaine abuse may be associated with myocardial infarction in young patients with normal coronary arteries, perhaps owing to induction of spasm.[10] Contrary to the suggestion that recurring coronary artery spasm may lead to the development of fixed atherosclerotic coronary obstructions, this has not been confirmed in a recent study,[11] suggesting that the long term outlook is satisfactory in such patients if appropriately treated with calcium antagonists and nitrates. Indeed, there is generally an excellent long term outcome in such patients, with 89–97% overall survival at five years.[12] Long term prognosis depends on severity of disease, the degree of spasmic stenosis, and number of vessels involved.[13] Patients with variant angina and concomitant obstructive coronary artery disease or arrhythmias have a poorer prognosis.[14]

Why coronary spasm develops at focal sites, as seen in our patient, is uncertain, although the possibility of focal abnormalities at those sites, such as an underlying plaque seen by detailed imaging such as intravascular ultrasound, cannot be discounted. For example, increased atherosclerotic plaques have been noted proximal to coronary artery segments affected by myocardial bridging, caused by increased wall shear stress.[15] Coronary spasm does occur in both normal and diseased vessels, typically within 1 cm of an atherosclerotic plaque in a diseased vessel. However, selective coronary angiography showed smooth and normal epicardial coronary arteries in our patient, whose young age also makes atherosclerotic disease less likely. The value of lipid lowering treatment in such patients is undetermined, although risk factor modification such as cessation of smoking and lowering of hyperlipidaemia should be encouraged. Finally, there was no evidence of myocardial bridging in our patient, which has been reported as a rare cause of coronary thrombosis and myocardial infarction.[16 17] Nitrate use would have been detrimental in myocardial bridging as, by reducing the intrinsic coronary wall tension, their use may worsen symptoms.

In summary, we present a patient with myocardial infarction who subsequently developed unstable angina. Initial coronary angiography had suggested coronary stenoses that did not respond to sublingual or intravenous nitrate, and "successful" angioplasty was performed. The abnormalities at repeat coronary angiography were immediately reversed following administration of intracoronary nitrates.

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