CASE REPORT

ST-elevation myocardial infarction secondary to coronary artery spasm provoked by food

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SUMMARY

We describe a patient with recurrent episodes of inferior ST elevation, secondary to coronary artery spasm. Each episode appeared to be provoked by the ingestion of rice and accompanied by a troponin T rise. An inpatient coronary angiogram immediately following an episode of pain demonstrated a focal area of spasm affecting the right coronary artery, which resolved with intracoronary nitrate injection. Although these episodes were self-limiting, cardiac MRI confirmed an acute subendocardial infarct. An association between food substances and coronary artery spasm with subsequent infarction has not been documented previously. Following appropriate advice and titration of antispasmodic medication, the patient has been pain free.

BACKGROUND

Coronary artery spasm (CAS) should be considered in any patient with transient ST elevation on ECG and normal coronary angiography. CAS is now recognised to contribute to a clinically significant number of patients presenting with acute coronary syndrome (ACS). Traditionally, management of spasm has been challenging and this may be due to unrecognised precipitating factors. We report, for the first time, a possible association between CAS and rice. This case highlights the importance of a thorough history to formulate an effective management plan and proposes an alternative trigger of CAS.

CASE PRESENTATION

A 41-year-old man was admitted after an episode of syncope, followed by burning pain migrating from just below the sternum, up the chest and radiating to both shoulders. The patient had reported presyncopal symptoms including dizziness and generalised weakness before sudden loss of consciousness for approximately a minute, witnessed by a relative. These symptoms occurred shortly after eating lunch. There were no symptoms or signs suggestive of a seizure. He was subsequently admitted at his local district general hospital.

While he had never experienced an episode of syncope in the past, the patient did report similar episodes of pain for the past 7 years, occurring in waves often multiple times a day for up to 10 weeks. The pain would last for approximately 45 min each time, relieved by belching and defecation. He had noted that consumption of rice and lentils had often precipitated these episodes. Two years earlier he had presented to a different hospital with an acute severe episode of pain and a

subsequent coronary angiogram was unremarkable. Owing to the atypical nature of his pain, he had also previously been referred for a gastroenterology opinion and gastroscopy, neither of which had identified any abnormalities.

There was no other significant medical history and he led an active life. The only coronary risk factor noted was a 20-pack-year smoking history. There was no significant family history of cardiac disease or sudden cardiac death. Cardiopulmonary examination was normal.

During admission while pain free, the patient ate a portion of rice, which was rapidly followed by a further episode of pain with significant inferior lead ST elevation captured on ECG (figure 1). He was subsequently referred to our cardiothoracic centre for urgent percutaneous coronary intervention.

INVESTIGATIONS

On arrival at our catheter laboratory, the patient's pain and ST segment changes had settled and coronary angiography revealed entirely smooth unobstructed coronaries (figure 2). A troponin T level was significantly raised (176 ng/L; normal levels <14 ng/L). An initial departmental echocardiogram demonstrated good left ventricular systolic function with an ejection fraction of 55–60%, normal right heart function and no discernible valvular or regional wall motion abnormalities.

In the following days, while pain free, the patient reported again that he had noted an association between his atypical pain and the consumption of rice. In an effort to prove his point, he ordered and ate a portion of rice. Remarkably, the same pain was reproduced accompanied by significant ST elevation (figure 3). He was taken directly to the laboratory for a repeat coronary angiogram, however, on intubation of his coronary vessels, his ST segments had begun to settle. Angiography on this occasion demonstrated a focal area of resolving spasm in the middle segment of the right coronary artery (figure 4). This settled completely with intracoronary nitrate. A repeat troponin T level demonstrated a new rise (31 ng/L) having returned to normal limits (7 ng/L) after the initial event. In view of two separate raised troponin T levels, transient ST elevation and chest pain, a cardiac MRI was organised as an inpatient, which identified an acute subendocardial inferior infarct.

DIFFERENTIAL DIAGNOSIS

- CAS with possible food triggers
- Atherosclerotic plaque rupture event
- Embolic event

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Figure 1 Significant inferior lead ST elevation postingestion of rice.

OUTCOME AND FOLLOW-UP

Aspirin and clopidogrel were continued in view of an established infarct and the dose of nicorandil doubled to reduce spasm. The patient's medication will be reviewed following a repeat cardiac MRI in 6 months. When he was pain free for 5 days he was discharged. He has been advised to avoid foods that precipitate pain and at 3-month follow-up has been pain free and his ECG is within normal limits. The patient has

decided not to have an implantable loop recorder to investigate his episode of syncope.

DISCUSSION

A significant proportion of patients presenting with features of ACS, may have underlying CAS. Ong *et al*¹ identified that, of 488 consecutive patients undergoing coronary angiography for suspected organic coronary disease, 9% had no culprit lesion and spasm was demonstrated on intracoronary provocation testing with acetylcholine. Typically, coronary spasm occurs early morning at rest, with fewer episodes in the afternoon or evening and may be due to the diurnal variation of flow-mediated endothelial dependent vasodilation.² The aetiology of CAS appears complex with multiple triggers identified in the literature and numerous case reports suggesting novel associations.

A history of smoking, particularly in male patients, appears to be the most important coronary risk factor for spasm.^{3–4} Endothelial dysfunction, impaired coronary artery smooth muscle contraction and congenital myocardial bridging, have all been proposed as contributing factors.^{5–6} More recently, mutation analysis has uncovered an increasing cohort of genetic alterations, which may predispose to endothelial dysfunction and changes to vasomotor tone. Endothelial nitric oxide synthase (ENOS), an enzyme important in the regulation of vasomotor properties of coronary arteries through production of nitric oxide, has often been identified as a potential target for research.⁷ Last year, Glueck *et al*⁸ demonstrated an increased allele frequency of an ENOS T-786C polymorphism in patients with angina secondary to CAS. This group of patients reported an improvement of their symptoms in response to L-arginine therapy (the amino acid from which nitric oxide is derived).⁸ With the ongoing growth and understanding of genetics in the role of CAS, similar associations are likely to be found in the future, developing our understanding of the underlying pathophysiology and providing potential for novel therapies.

Another form of CAS is Kounis syndrome, precipitated by histamine release from mast cell activation secondary to an

Figure 2 Image from the first coronary angiogram demonstrating an unobstructed right coronary artery. No focal disease. The patient's pain and ST segments had settled prior to arriving at the catheter laboratory.

Figure 3 Electrocardiogram demonstrating inferior lead ST elevation, while experiencing a further episode of chest pain after eating a further portion of rice.

acute allergic reaction, which can occur in the context of unobstructed coronary arteries (type 1) or known coronary artery disease (type 2).⁹ There have been multiple case reports in the literature describing this phenomenon, often due to medication and associated with symptoms of anaphylaxis, chest pain, evidence of ECG changes and a troponin rise.¹⁰

Our patient appeared to have CAS with ST elevation, precipitated by food. We are unable to find similar case reports, however, there is evidence in the literature for the existence of a cardio-oesophageal neural reflex, which due to alterations in

sympathetic or vagal tone, could account for his presentation. Early work by Rasmussen *et al*¹¹ demonstrated a small group of patients with known coronary spasm who had prolonged oesophageal contractions on provocation with reproduction of their typical chest pain and subsequent ECG changes. Similarly, Chauhan *et al*¹² showed oesophageal stimulation with hydrochloric acid can decrease coronary flow and again reproduce a patient's typical pain associated with CAS. There was no effect, however, in a patient group post cardiac transplant suggesting the presence of a neural reflex between the oesophagus and coronary vessels mediating a reduction in coronary flow. It also provided some evidence for the multifactorial aetiology of coronary spasm as hydrochloric acid stimulation was ineffective in reproducing chest pain or reducing coronary flow in a subgroup of patients with known CAS. Owing to the presence of decreased coronary flow in the absence of a change in epicardial coronary diameter, it was postulated that the reflex may be secondary to a local release of factors promoting microvascular constriction.¹²

Manfrini used oesophageal manometry to demonstrate frequent abnormal oesophageal spasm in patients with CAS preceding episodes of pain and ECG changes indicative of myocardial ischaemia.¹³ This timeline suggests oesophageal spasm in patients with an abnormal neural reflex can induce myocardial ischaemia and chest pain, which may be the underlying mechanism in our case. More research is required to explore this reflex and the implications it may have on the identification and management of such patients.

CAS has a good prognosis with survival figures at 1-year, 2-year and 5-year mortality reported as 95%, 92% and 89%, respectively.¹⁴ It may, however, account for up to 4.7% of sudden cardiac deaths as reported by Hill *et al*¹⁵, most likely due to ventricular arrhythmias secondary to myocardial ischaemia. Risk factors include a history of previous syncopal episodes and documentation of multivessel spasm during coronary angiography.^{14 15} These groups of patients are more likely to have recurrent significant cardiac events and 'medication resistant' angina.⁴ These findings could be used to stratify patients

Figure 4 Image from the second coronary angiogram as the chest pain was resolving, demonstrating a focal area of resolving spasm affecting the right coronary artery (see arrow); the most likely cause of this patient's infarction.

according to risk and identify those who may benefit from intensive follow-up.

Patients with CAS who have sustained an out of hospital cardiac arrest may benefit from secondary prevention with an implantable cardiac defibrillator (ICD).¹⁶ There are currently no data, however, on the prognostic implications of ICDs in this group of patients or on whether other patients with CAS would benefit. Furthermore, there is little evaluation of the long-term outcome or management of patients with CAS who present with ventricular tachycardia without continuation to arrest. Holter monitoring or implantable loop recorders could provide further value to risk stratify patients.

The management of CAS includes lifestyle advice, smoking cessation and medical therapy. Calcium channel blockers are the most established and the additive benefit of long-acting nitrates has been recognised.¹⁷ Dihydropyridine-type calcium channel blockers over diltiazem or verapamil may have greater protective effects against endothelial dysfunction in patients with CAS due to upregulation of the NO-cGMP system.¹⁸ Studies have, however, demonstrated that 50% of patients with CAS at 3-year follow-up report persistent angina, and therefore represent a complex and difficult to treat cohort.^{19 20} This may be due to unidentified triggers as suggested in our case report.

Learning points

- ▶ Coronary artery spasm is an important differential to consider in patients presenting with ST elevation with a normal coronary angiogram.
- ▶ A detailed history is required to make the diagnosis and to consider precipitating factors, which may have an impact in preventing further episodes.
- ▶ Referral to other specialties may be necessary for further assessment, particularly in cases of refractory pain.
- ▶ Patients with recurrent syncope may need ECG Holter monitoring to rule out ventricular arrhythmias and may benefit from implantable loop event recorders.

Contributors WY wrote the discussion and case history. RB assisted with writing the case history. AK performed the coronary angiogram, reviewed the manuscript and gave advice regarding its contents. WH reviewed the manuscript and gave advice regarding its contents.

Competing interests None.

Patient consent Obtained.

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