CASE REPORT

ST-segment elevation and ventricular tachycardia due to multivessel coronary artery spasm without significant coronary artery stenosis

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Abstract - We present a 74-year-old male with persisting acute chest pain, inferior wall ST elevation on ECG and polymorphic ventricular tachycardia, who had simultaneous coronary spasm of both the right coronary artery and the left anterior descending artery in the absence of significant coronary artery stenosis. We also discuss the relevant literature.

Keywords - Coronary spasm; variant angina; Prinzmetal; ventricular tachycardia; ST segment elevation acute myocardial infarction

Case report
A 74-year-old white male presented with intense crushing chest pain which had started one hour previously (i.e. at 7:30 A.M.) and did not resolve on sublingual nitroglycerin. Apart from hypertension and smoking his medical history was unremarkable. He had not adhered to the prescribed antihypertensive medication which included a calcium antagonist.

At presentation, his blood pressure was 100/60 mmHg. Physical examination revealed no abnormalities of the heart and lungs. The ECG showed an atrioventricular-nodal escape rhythm, with ST elevation in the inferior leads and in lead V4R, together with reciprocal ST depression in the precordial leads (Figure 1), thus suggesting acute inferior wall myocardial infarction with right ventricular involvement due to occlusion of the proximal right coronary artery (RCA).

Several minutes after arrival, rapid polymorphic ventricular tachycardia occurred with subsequent loss of consciousness. This episode was terminated by electrical cardioversion.

Immediate coronary angiography revealed a non-significant stenosis in the proximal RCA, as well as two significant stenoses in the proximal left anterior descending artery (LAD) (Figure 2). However, these stenoses resolved following an intracoronary nitroglycerin infusion, leaving only wall irregularities of the proximal LAD. Also, the chest pain and ST segment elevation disappeared. We concluded that both this episode of chest pain with ECG abnormalities suggesting acute occlusion of the right coronary artery, and the polymorphic ventricular tachycardia were caused by transmural ischaemia due to coronary spasm.

Discussion
Prevalence
Coronary spasm causing myocardial ischaemia is the underlying mechanism of Prinzmetal or variant angina. In addition, coronary spasm plays a role in the symptoms of atherosclerotic coronary artery disease including stable angina pectoris, acute coronary syndromes and sudden cardiac death [1]. The prevalence of coronary spasm in acute chest pain patients has been estimated at 15 to 20% [2], and occurs most commonly in elderly men and post-menopausal women [1]. Reports suggest that its prevalence has decreased in Europe and North-America, possibly due to the lower prevalence of smoking and increased use of calcium antagonists [1]. Also, spasm may be less often diagnosed as provocation tests are less often performed [1]. The prevalence of coronary spasm is higher in Japanese subjects, which may be related to genetic factors and the high prevalence of smoking [1].

Pathophysiology
Coronary spasm usually occurs at rest, particularly in the early morning, possibly due to circadian variations in autonomic nervous system activity and hormone levels [1]. Additional important precipitating factors include cold, alcohol, smoking, cocaine, and magnesium deficiency [1,3]. Coronary spasm itself often induces spasm in other coronary segments [1].

Figure 1. The ECG on arrival at the emergency department suggests acute inferior wall myocardial infarction.
Electrocardiographically spasm may present with ST segment elevation, usually with reciprocal ST segment depression. ST segment elevation may occur in different perfusion territories during different attacks, and, more rarely, simultaneously in multiple perfusion territories during one episode [1].

Coronary spasm may also cause arrhythmias, including supraventricular arrhythmias, atrioventricular conduction disturbances, ventricular tachycardia and, more rarely, ventricular fibrillation [4-6]. Patients with multi-vessel spasm seem to be at increased risk of lethal arrhythmias [1].

Several mechanisms have been proposed for the pathogenesis of coronary spasm. Firstly, endothelial nitric oxide (NO) production is deficient, which may explain the coronary-spasm-relieving effect of nitrates [1]. Secondly, chronic low-grade inflammation may be involved as indicated by elevated high-sensitivity C-reactive protein levels in subjects with coronary spasm [1]. Thirdly, coronary smooth muscle hypercontractility seems important, as calcium antagonists suppress coronary spasm [1]. Statins, which are thought to prevent calcium-independent hypercontractility, may be beneficial as demonstrated in a recent trial in which the addition of fluvastatin to regular calcium antagonist treatment suppressed coronary spasm [1].

**Diagnosis**

A definite diagnosis of coronary spasm depends on angiographic demonstration of lumen narrowing during chest pain and/or ECG changes that resolve spontaneously or upon intracoronary infusion of nitroglycerin. Alternatively, a provocation test can be performed with intracoronary administration of either ergonovine or acetyl choline [1].

**Treatment**

Sublingual nitrates can relieve an attack of coronary spasm. For the prevention of coronary spasm calcium antagonists are essential [1]. Patients with multivessel coronary spasm are recommended to continue calcium antagonists in maximum tolerated doses indefinitely to avoid lethal arrhythmias [1]. If necessary, long-acting nitrates can be added [1]. Additional drugs such as statins, may be of benefit [7]. Patients should refrain from smoking and alcohol. Insufficient data on the value of implantable cardioverter defibrillators (ICDs) exist [8,9]. Percutaneous coronary intervention for non-significant coronary atherosclerosis does not help to prevent spasm [10].

**Follow-up on our patient**

Following the coronary angiography, our patient was started on a calcium antagonist and a long-acting nitrate. Furthermore, he was prescribed an angiotensin-converting enzyme inhibitor for better blood pressure control, while acetylsalicylic acid and a statin were added because of the non-significant atherosclerotic lesions in the LAD. The maximum troponin-T level was 0.07 mg/l, with a normal CK level. He subsequently remained free of chest pain and arrhythmias. As current evidence on the benefit of ICD on coronary spasm is inconclusive, we did not choose to implant an ICD.

**Conclusion**

This case illustrates that patients with chest pain and ST elevation on ECG do not always have acute thrombotic coronary artery occlusion. In patients with acute coronary syndromes, stable angina pectoris or ventricular tachyarrhythmias and normal coronary arteries on angiography, a provocation test with ergonovine for example, can be considered in the search for possible coronary spasms.

**Conflict of interest statement: none (for all authors)**
References