Recurrence ventricular fibrillation caused by coronary artery spasm leading to implantable cardioverter defibrillator implantation


Department of Cardiology, VU University Medical Centre, De Boelelaan 1117, 1081 HV, Amsterdam, The Netherlands

*Corresponding author. Tel: +31204444444. E-mail address: t.hendriks@vumc.nl

Coronary artery spasm has been known to induce ischaemia and ventricular arrhythmias. We present a case of recurrent ventricular fibrillation caused by spasm-associated transmural myocardial ischaemia. During an intra-coronary acetylcholine provocation test, severe coronary spasm could be induced. The patient was treated with a hybrid approach of medication and an implantable defibrillator.

Case report

A 58-year-old woman presented with chest pain at rest and electrocardiogram (ECG) changes. She had no history of cardiovascular disease. Risk factors included hypertension and hypercholesterolaemia. Her medication consisted of diltiazem. After admission, she complained of chest pain with signs of transmural ischaemia with ST elevations in leads aVR.

![Monitored strip during coronary artery spasm (top) and coronary angiography before (A) and after (B) acetylcholine provocation test, showing spasm (bottom).](https://academic.oup.com/europace/article/10/12/1456/465095)
aVL, and V1–V3 and with reciprocal ST changes in leads II, III, aVF, and V4–V6. Within minutes, her rhythm deteriorated into ventricular fibrillation (VF) treated with successful defibrillation. At this time, the diagnosis was acute coronary syndrome and a coronary angiography (CAG) was performed. The right coronary artery was normal, the left descending artery (LAD) showed a proximal hazy lesion, and the circumflex artery (RCX) showed a proximal lesion of 40%. All coronary arteries showed normal TIMI 3 flow. Echocardiography showed a normal left ventricular function and blood laboratory tests were normal. During admission, she had several additional episodes of angina with ECG changes, all responding to nitrates. One of these episodes resulted in VF, which was defibrillated successfully. A second CAG was performed and revealed no additional lesions. At this time, coronary artery spasm (CAS) was suspected. Acetylcholine provocation test induced a spasm of the proximal RCX, resulting in transmural ischaemia with ECG changes similar to the ECG on admission. Intravascular ultrasound showed a moderate eccentric plaque in both the LAD and RCX. Flow measurements indicated no significant lesions in the RCX (Fractional flow reserve = 0.85). She was diagnosed of ischaemia-induced VF mediated by CAS. An implantable cardioverter defibrillator (ICD) was implanted and diltiazem was replaced by nifedipine. After discharge, the patient had an uneventful 6 months follow-up without ICD detected ventricular arrhythmias.

Discussion

In 1959, Prinzmetal et al.1 published the first observation on a variant form of angina pectoris. Since then, Prinzmetal angina refers to decreased blood flow secondary to spontaneous spasm of the arterial wall usually without significant atherosclerotic plaques. Provocative testing with an alpha receptor stimulator is optional in the diagnosis of CAS. In a substantial number of patients, Prinzmetal angina is associated with haemodynamically important arrhythmia and is usually treated with calcium channel blockers. Controversy exists about the optimal treatment of patients with CAS, specifically in the presence of life threatening arrhythmia.2 Additional coronary intervention in patients with CAS is controversial. Mohri et al. studied a large group of 117 patients with possible CAS and reported that in 25% of these patients no spasm of a large epicardial coronary artery could be induced, despite the presence of angina such as chest pain, ischaemic ECG changes, or both. They refer to this as microvascular spasm.3 Thus, the vasoconstrictive segment could not be located in a substantial number of patients. Moreover, spontaneous vasospasm may be located in a different segment when compared with acetylcholine-induced vasospasm; thus limiting the effect of a coronary intervention. In the presence of CAS-induced potential lethal arrhythmias, a combination of calcium blocking agents and ICD implantation appears the most appropriate option.

Conflict of interest: none declared.

References