CASE REPORT

Atherothrombotic out-of hospital cardiac arrest: watch out for a second train

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Accepted 23 January 2018

SUMMARY

A 63-year-old man presented with an out-of-hospital cardiac arrest. The coronary angiogram with intravascular imaging was performed and showed a mid-left anterior descending artery atherosclerotic cap rupture with thrombus burden treated with drug-eluting stent (DES) implantation. During the hospitalisation in the intensive care unit, the patient experienced recurrent ventricular fibrillations and asystole with transient ST-segment elevation in the inferior leads with normal coronary angiography. A methylergonovine provocative test was therefore performed and showed an occlusive right coronary artery (RCA) spasm. Due to recurrent RCA spasm, the patient was treated with DES implantation with favourable results at 3-month follow-up.

BACKGROUND

Out-of-hospital cardiac arrest (OHCA) is a major public health issue. The survival rate from OHCA has been increasing in association with the

widespread use of automatic external defibrillators and educational campaigns on the importance of bystander's cardiopulmonary resuscitation (CPR).¹ Particularly, among OHCA survivors, a known cause that can lead to sudden death by life-threatening ventricular arrhythmias is coronary artery spasm (CAS).² Often, at the acute phase, the interventional cardiologist will focus on thrombotic stenosis and plaque ruptures and not necessarily on CAS. In our case report, we present a patient who had OHCA due to two potential main causes: initial thrombotic plaque rupture and secondary diagnosed CAS.

CASE PRESENTATION

A 63-year-old man presented with OHCA, witnessed by his wife, who quickly began cardiopulmonary resuscitation (CPR). Medical history included active smoking, dyslipidaemia and peripheral artery disease. Paramedics arrived 6 min after cardiac arrest and resumed CPR. A semiautomatic

Figure 1 (A) Initial ECG obtained after resuscitation showing atrial flutter and anterior-leads ST-segment elevation. (B, C) First coronary angiogram showing mid-left anterior descending artery mild stenosis with heterogeneous aspect (B, arrow) and atherosclerotic plaques on left circumflex and right coronary arteries (C). (D) Optical coherence tomography imaging showing plaque rupture and thrombosis (arrow).

To cite: Petruescu L, Picard F, Cariou A, et al. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-223529

Figure 2 (A) ECG showing inferior-leads ST-segment elevation, obtained immediately after tube replacement. (B, C) Methylexgonovine administration for spasm test showing right coronary artery occlusive spasm (arrow), disappearing after nitroglycerin administration.

defibrillator was placed and delivered a single shock for ventricular fibrillation (VF). Return of spontaneous cardiac activity was obtained within 12 min. Total no-flow was less than 2 min and low-flow under 10 min. ECG showed atrial flutter with anterior-leads ST-segment elevation (figure 1A). The patient received 250 mg of aspirin, 4800 IU of unfractionated heparin and 500 mL of saline solution. He was put under mechanical ventilation and transferred to the nearest catheterisation laboratory.

Initial coronary angiogram (figure 1B, C) displayed mid-left anterior descending (LAD) artery mild stenosis with heterogeneous aspect suggesting acute thrombosis, as well as atherosclerotic plaques on the left circumflex (LCx) and the right coronary arteries (RCAs). Optical coherence tomography (OCT) performed on LAD, LCx and RCA demonstrated plaque rupture and non-occlusive thrombus in the mid-LAD (figure 1D). He was therefore treated with ad hoc LAD angioplasty with drug-eluting stent (DES) and transferred to the intensive care unit. Initial echocardiography demonstrated moderate hypokinesia of anteroapical segments (left ventricular ejection fraction: 40%) and trivial mitral regurgitation.

Forty-eight hours later, the patient had another VF episode, successfully treated by CPR and single-shock therapy. The pre-VF monitoring ECG did not show any ST-segment modification and the postresuscitation ECG showed positive T-waves and normal ST segment. Therefore, the patient immediately underwent a

new coronary angiogram, showing no stent thrombosis on the LAD and normal LCx and RCA.

Five new VF episodes occurred within the following three days and were attributed to postischaemic electrical storm despite beta-blocker treatment and normal potassium levels. During a ventilation tube change, inferior-leads modification suddenly occurred (figure 2A), followed by bradycardia and asystole, treated by 2 mg of epinephrine.

INVESTIGATIONS

A third angiography was performed showing again full patency and no thrombosis of the LAD stent, with normal LCx and RCA. Therefore, a provocative test (PT) was performed using methylexgonovine injection. The PT was abnormal with RCA occlusive spasm associated with severe bradycardia (figure 2B, C). RCA occlusive spasm slowly resolved after 3 mg of intracoronary nitroglycerine, which was mandatory to avoid another episode of asystole. The patient was then treated by antispastic medical therapy including a combination of calcium-channel blocker (amlodipine and diltiazem), a transdermal nitroglycerin patch and an oral anticholinergic drug.

TREATMENT

A final angiography with PT test was performed to evaluate optimal medical therapy (ie, association of two calcium-channel

blockers, nitrates, statins and anticholinergic drugs) 22 days later, showing refractory spasm under medical treatment despite no clinical sign of myocardial ischaemia. This refractory spasm resolved after 2 mg of intracoronary nitrates. Hence, decision was made to perform RCA angioplasty with DES implantation and cardiac defibrillator implantation. At discharge, echocardiography was normal with complete wall abnormalities recovery.

OUTCOME AND FOLLOW-UP

At a 9-month follow-up, the patient was symptom-free.

DISCUSSION

Cardiac aetiologies of OHCA are still debated. We report a clinical case where two mechanisms could be demonstrated in the same patient. First, we observed by OCT plaque rupture with thrombus; and second, we had the occurrence of asystole due to RCA spasm. One must realise that these two potential causes of coronary artery disease might have been responsible for the patient's cardiac arrest. Nevertheless, in our case, we cannot determine if the recurrent VF phenomenon was due to coronary spasm or could be imputable to electrical storm. Also, it remains unclear whether the initial coronary angiogram finding of cap rupture and thrombus on mid-LAD was initially a spastic site or not as methylergonovine testing was not initially performed. However, this stenotic thrombotic lesion could be related to a transient spastic phenomenon. Recently, Shin *et al*³ described

OCT imaging findings in 69 patients with a total of 80 spasm sites. In their series, atherosclerotic plaque was seen on OCT in 79 of the 80 spasm sites. Fibrous cap disruption was detected at three sites (4%), and OCT-defined erosion was observed at 21 spasm sites (26%). Nevertheless, coronary artery provocation testing should not be advised at the acute phase of an OHCA and the finding of thrombotic plaque rupture treated with angioplasty and cap sealing with stent implantation sounds reasonable. However, in patients surviving OHCA without coronary artery disease, testing for CAS is important as it is not only very feasible but also proved itself useful to detect underlying causes and thus establish appropriate medical therapy.⁴ However, whether methylergonovine-induced spasm test should be performed on every cardiopulmonary arrest without obvious cause remains unclear but appears to be reasonable as it is simple, quick, feasible and can be performed a couple of days after initial cardiac arrest.

Contributors All authors treated the patient during his ICU and cardiology hospitalisation. FP and LP drafted the case report. OV and AC performed literature review, participated in writing and revised the case report critically for important intellectual content. All authors have approved the manuscript and agree with its submission. All authors agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Funding This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- Often, at the acute phase, the interventional cardiologist will focus on thrombotic stenosis and plaque ruptures and not necessarily on coronary artery spasm.
- Different causes can provoke cardiac arrest in the same patient, and coronary artery spasm should be tested in case of recurrent myocardial ischaemic episodes despite initial revascularisation.
- In patients surviving out-of-hospital cardiac arrest without coronary artery disease, testing for coronary artery spasm is important as it is not only very feasible but also proved itself useful to detect underlying causes and thus establish appropriate medical therapy.

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