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Coronary reactivity testing in vasospastic angina leading to cardiac arrest and coronary dissection



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1. Case report

A 45-year-old woman was admitted to the ER of our hospital because of acute chest pain with ST segment elevation on ECG. She was then referred to the cath lab, where, despite the persistence of symptoms and ECG abnormalities, normal coronary arteries were documented (Fig. 1). Hyperventilation test was negative. On day 2, chest pain without ECG abnormalities was followed by cardiac arrest in VF. On day 6 after admission, coronary angiography with Ergonovine (Erg) test was performed (Fig. 2).

After administration of 16 mcg Erg, a severe spasm with total occlusion of the mid-segment of RCA occurred (Fig. 2, panel B). The spasm was resistant to high dosages of intracoronary nitrates, verapamil and nitroprusside. After 1 h of ischemia hemodynamic conditions deteriorated, requiring oro-tracheal intubation. Four episodes of VT–FV immediately treated with DC shock occurred (Fig. 2, panel C). Because of the persistence of hemodynamic instability, and prolonged ischemia exposure, an ExtraCorporeal Membrane Oxygenation System (ECMO) was implanted. In the meantime, the spasm started to vanish with evidence of spiral dissection starting from the middle segment of RCA to the distal

portion of IVP and branch PL, with luminal filling defects, contrast staining in the dissected false lumen and late “slow-flow” (Fig. 2, panel C). After ECMO positioning, three drug eluting stents were implanted with a final TIMI 3 result, with a residual dissection no-flow limiting and absence of staining contrast of the distal segment of PL and IVP (Fig. 2, panel D).

Two days later the patient was weaned from ECMO and high dosages of Diltiazem, Nifedipine and nitrates were started. The patient remained asymptomatic for the following days; no asymptomatic ST segment alteration was documented and an ICD was implanted on day 14. She was discharged on day 26 with Diltiazem 120 (3 times/day), Nifedipine 40 mg, and nitrates 20 bid. Her follow-up is negative.

2. Discussion

Coronary artery spasm can lead to myocardial infarction, left ventricular dysfunction, promote potentially life threatening arrhythmias and ultimately sudden cardiac death. A key aspect of coronary spasm is its temporarily transient nature which often leads to difficulties in diagnosis [1–5].

A diagnosis of coronary spasm is usually made once coronary spasm is suspected from history and investigations have been performed. These tests may be non-invasive (i.e. hyperventilation) or invasive like coronary reactivity testing (CRT) or may not be needed at all. Indeed, as reported in the Japanese Circulation Society Guidelines, coronary angiography can be avoided altogether if a patient meets all listed criteria for coronary spasm [5].

In our case, coronary artery spasm was the main diagnostic suspect, although during the urgent coronary angiography, despite the presence of symptoms and ECG abnormalities, coronary arteries (baseline and during hyperventilation) were normal. Notably during the following days the patient still complained symptoms without ST segment changes; cardiac arrest occurred after one of these episodes. In our case, 2 key points of interest arise.

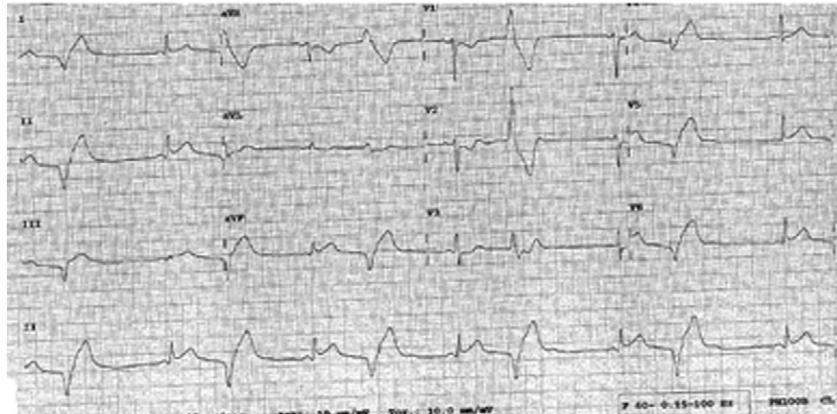
2.1. Safety of Ach/Erg test

In usual clinical practice, myocardial spasm provocation tests are not performed routinely because of the concern of adverse events during

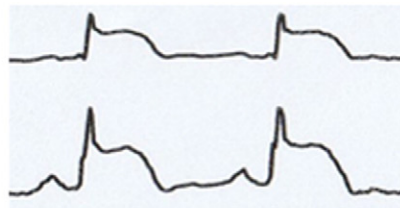
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Admission + cath lab



ECG
Cath lab



Angor

+++

Coronary
Angiography

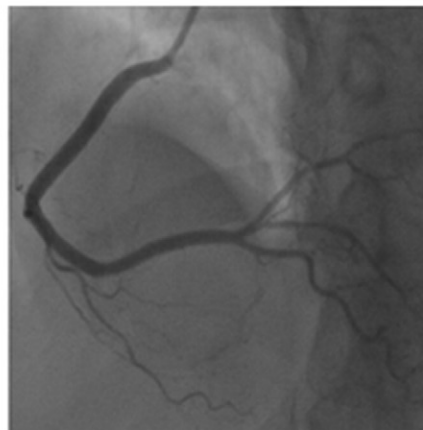


Fig. 1. Clinical scenario at presentation.

drug administration. We agree that probably the discharge therapy of our patient would have been the same also without Erg testing and ICD in secondary prevention would have been implanted anyway, but the decision to perform CRT and expose the patient to the risk of a provocative test was supported by the safety profile reported by the latest studies, and the need to acquire prognostic information [6–9].

In a retrospective study encompassing more than 20,000 patients that have undergone CRT (10,628 with Ach; 10,884 with Erg), Ach was associated with a higher rate of cardiac complication (0.9% vs 0.4%). However, major cardiac events (defibrillation, chest compression, IABP, ECMO) happened in only 0.7% of patients [6].

In another study on 921 patients with unobstructed coronary arteries, a similar complication rate was observed (only 1% had minor complications and no serious complications occurred [7]).

In another study of 1244 patients with variant angina, VT/VF and brady-arrhythmia development was reported in 3.2% (Erg) and 2.7% (Ach) of patients, respectively with an overall incidence of arrhythmic events of 6.8% [8]. Diffuse right coronary artery spasm had a significant correlation with provocation-related VT/VF, while a focal plus diffuse multivessel spasm had an important association with MACEs, whereas provocation-related arrhythmias did not [8].

Another study focused on 293 women with microvascular coronary dysfunction and no obstructive CAD, the risk of serious intra-procedural

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