Sudden Cardiac Arrest as a Presentation of Malignant Coronary Vasospasm in a 69-year-old Man

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Introduction

Vasospastic angina (VSA) is a focal spasm of one or more coronary arteries, without clinically significant atherosclerosis or atherosclerotic plaque. These spasms, though responsible for inducing anginal symptoms and temporary ischemia, are rarely associated with ventricular arrhythmia and cardiac death (1). Here, we present a 69 y.o. male with a history of hypertension and hyperlipidemia who was hospitalized after a ventricular fibrillation/witnessed cardiac arrest.

Case Presentation

- Patient was previously admitted with complaints of angina, mild troponin elevation 0.05, transient ST-elevation in the anterior leads which prompted an urgent cardiac catheterization.
- Angiographically, the patient had 30% proximal left anterior descending artery stenosis, with no other areas of coronary disease.
- His dynamic ST changes were attributed to coronary vasospasm, patient was initiated on dual antiplatelet therapy with aspirin/clopidogrel and discharged home on diltiazem and isosorbide mononitrate as antianginal therapy.
- One month later, he was emergently brought to the emergency department with chest pain, and collapsed in the passenger seat.
- His initial rhythm was ventricular fibrillation. Emergent defibrillation resulted in ROSC in the ED. Targeted temperature management was initiated and he was transferred to CICU.
- EKG on admission had no significant ST-T changes (Figure 1).
- During rewarming, patient was again noted to have dramatic ST elevations in anterior leads noted on telemetry, and confirmed with a 12-lead EKG (Figure 2).
- Bedside echocardiogram at the time notable for wall motion abnormality in the anterior wall and apex, consistent with left anterior descending artery (LAD) territory.
- He was placed on IV nitroglycerin with resolution of his dynamic ST changes (Figure 3) and regional wall motion abnormalities on echocardiogram within minutes of initiation.
- He returned to the catheterization laboratory, with similar findings angiographically as previous. IVUS was performed which demonstrated 80% eccentric plaque in the proximal LAD with no evidence of rupture, and he received a drug eluting stent to the LAD (Figure 4).
- Attempts at weaning IV nitroglycerin resulted in ST changes, suggestive of vasoreactivity.
- Patient was subsequently extubated, initiated on PO diltiazem and isosorbide mononitrate, and had an ICD placed for secondary prevention of ventricular arrhythmias due to coronary vasospasm. He was discharged in stable condition without additional EKG changes or anginal symptoms.

Discussion

- As introduced, vasospastic angina is a distinct entity with focal spasm of epicardial coronary arteries (1).
- Precipitating factors for coronary vasospasm include hyperlipidemia, smoking, and physical/emotional stressors (3).
- Though uncommon to the classic definition, with the above risk factors, vasospastic angina can coincide with atherosclerosis, as in the case in our patient, which can create a diagnostic dilemma for clinicians (3).
- However, in our patient, he had transient ischemic ECG changes improving rapidly with IV nitroglycerin, lack of ongoing wall motion abnormalities on echocardiogram, and IVUS demonstrating absence of plaque rupture
- His presentation was out-of-proportion to the degree of atherosclerosis, and the rapidity of his symptom improvement suggests vasoreactivity as the culprit to his initial arrest.
- VSA is generally thought to carry a favorable prognosis. Rarely do patients with VSA present with ventricular arrhythmias, though spasm of the left anterior descending artery carries a high risk (4).
- Optimal medical treatment for coronary vasospasm includes calcium channel blockers and nitrates. However, patients continue to have an angina recurrence rate up to 30% (4).
- As in our case, despite optimal medical therapy, recurrent events can result in arrhythmia and sudden cardiac death, creating a challenge for clinicians.
- While optimal medical therapy offers symptom relief and may exhibit some cardioprotective effects from ventricular arrhythmias, refractory vasospasm can have devastating effects (5).
- Implantable cardioverter defibrillator (ICD) implantation is a proven, effective way to abort sudden cardiac arrhythmia due to coronary vasospasm, and currently carries a Class Ila recommendation for this cohort (5,6).
- For our patient’s survival benefit, ICD implantation was crucial in addition to his ongoing oral vasodilator therapies.

References