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Ventricular Fibrillation following Coronary Vasospasm

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Abstract

Vasospastic angina (VSA) is a common cause of acute coronary syndrome (ACS) and carries a good long-term prognosis when medically treated [1]. Ventricular arrhythmias are an infrequent presentation. We present a case of ventricular fibrillation caused by coronary vasospasm and discuss the rationale behind subsequent treatment decisions.

Keywords: Ventricular Tachycardia; Acute Coronary Syndrome; Secondary Prevention

Abbreviations: VSA: Vaso Spastic Angina; ACS: Acute Coronary Syndrome; ICD: Implantable Cardioverter Defibrillator; LAD: Left Anterior Descending; LCx: Left Circumflex; VA: Ventricular Arrhythmia; MI: Myocardial Infarction; ECG: Electro Cardiogram; RHC: Right Heart Catheterization

History of Presentation

A 36-year-old female presented with crushing substernal chest pain and diaphoresis. She had been admitted four times within the previous month for sudden onset chest discomfort. During the first hospitalization she was found to have a minimally elevated troponin and an exercise stress test that was negative for ischemia in the setting of submaximal exercise. During a subsequent admission she experienced syncope 2-3 minutes after the onset of symptoms and her electrocardiogram (ECG) was notable for ST elevation in lead aVR and diffuse ST depressions (Figure 1A). She had non-obstructive coronary artery disease on coronary angiography and a negative V/Q scan. A provisional diagnosis of vasospastic angina (VSA) was made, and the patient was started on nifedipine and isosorbide mononitrate, the latter of which was discontinued secondary to an urticarial rash. She presented again to the hospital within hours of discharge with recurrent chest pain. Her physical exam was notable for an elevated blood pressure, a grade II/VI holosystolic murmur at the apex, bibasilar crackles, and cool/clammy extremities. Serial ECGs revealed predominantly inferior and lateral ST depressions as well as ST elevation in aVR (Figure 1B). A bedside echocardiogram demonstrated new inferolateral hypokinesis and severe mitral regurgitation. Shortly after arrival, the patient had a ventricular fibrillation arrest and was successfully defibrillated with a single shock.

Past Medical History

The patient has a history of hypertension, hyperlipidemia, chronic kidney disease, and multiple sclerosis.

Differential Diagnosis

The primary working diagnosis was coronary vasospasm causing myocardial ischemia and ventricular fibrillation. Other possibilities included acute coronary syndrome (ACS) from plaque rupture (though this was far less likely given her recent normal coronary angiogram), an in situ coronary thrombus due to an undiagnosed vasculitis, a thromboembolic phenomenon, or myocarditis.

Investigations

The patient was taken for emergent coronary angiography, which revealed diffuse vasospasm of her left anterior descending (LAD) and left circumflex (LCx) arteries, including a focal 60% LCx lesion (Figure 1C). These abnormalities resolved after administration of intracoronary nitroglycerin (Figure 1D). Right heart catheterization (RHC)exhibited markedly elevated bilateral filling pressures, severely reduced cardiac index, and markedly increased systemic vascular resistance. A cardiac MRI noted delayed enhancement within the inferolateral wall,

consistent with myocardial infarction and superimposed edema. A comprehensive rheumatologic laboratory evaluation revealed a positive ANA (1: 1280) in a speckled pattern with negative antiphospholid antibodies. It was felt that her positive ANA titer was due to multiple sclerosis.

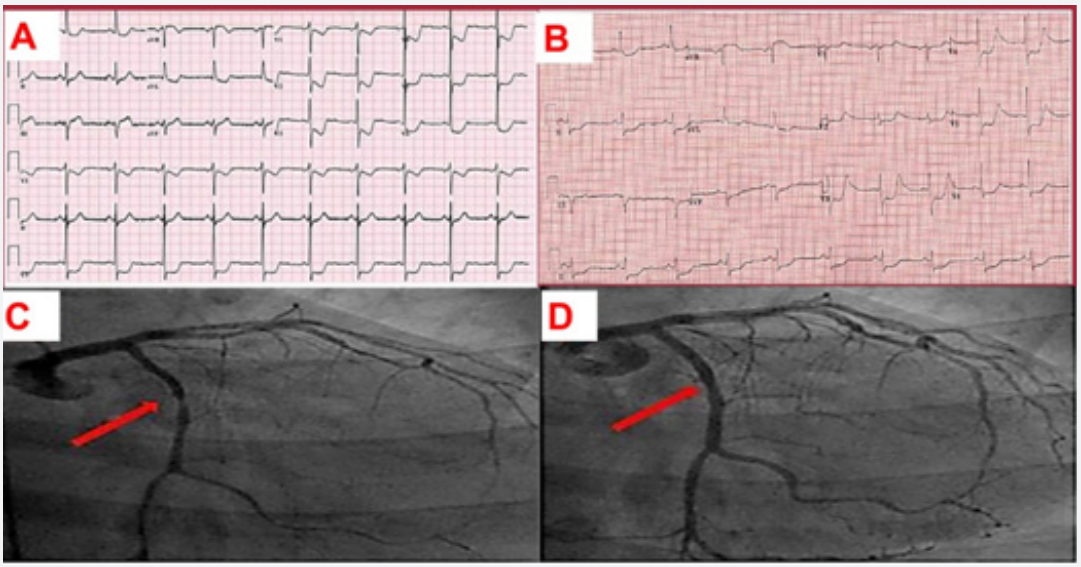

Figure 1: Admission electrocardiogram (A), demonstrating diffuse ST depressions and isolated ST elevation in a VR. Coronary angiogram performed during initial admission demonstrated mild coronary artery disease. Electrocardiogram performed during subsequent admission (B), revealing ST depressions predominantly in inferior and lateral leads and ST elevation in aVR. Coronary angiogram performed at that time revealed a focal 60% left circumflex lesion and diffuse vasospasm of the left anterior descending (C), which resolved with administration of intracoronary nitroglycerin (D).

Management

The patient was deemed to be in low output heart failure and was placed on intravenous milrinone, intravenous nitroglycerin, and oral afterload reducing agents. Over the course of the hospitalization, she weaned from the intravenous medications, and was discharged on nifedipine, diltiazem, and isosorbide dinitrate (with close monitoring for an allergic reaction). A subcutaneous ICD was implanted prior to discharge for secondary prevention of ventricular arrhythmias.

Discussion

Despite higher initial in-hospital mortality rates, inpatient survivors of myocardial infarction (MI) complicated by ventricular arrhythmias (VA) do not appear to have a worse long-term prognosis compared to those that do not have VA [2]. Current guidelines recommend ICD implantation for secondary prevention following an MI only in patients who are unable to be revascularized.

VSA is a common cause of acute coronary syndromes (ACS), particularly in younger patients, and carries a good long-term prognosis when appropriately treated [1]. Yasue et al demonstrated a ten-year survival rate of 93% in 245 VSA patients. Simultaneous ST elevations in the anterior and inferior leads were the strongest negative prognostic marker. Survival was improved in those treated with calcium channel blockers and those who refrained from smoking and alcohol usage [3].

In contrast, VA are an uncommon manifestation of VSA. Data are limited regarding the characteristics of VSA patients who develop VA [4]. Takagi et al identified 1429 patients with VSA over 10 years, 35 of whom suffered VA. Patients that experienced VA had a poorer survival-free rate of major adverse cardiac events than those that did not (72% vs. 92%, respectively) [5]. Limited data suggest that VSA patients who have an episode of VA are at high risk of recurrence. Sueda et al performed a meta-analysis totaling 137 VSA patients who received an ICD for secondary prevention and found that 25% received an appropriate shock within an average follow up time of 41 months. Of note, there were high rates of single agent calcium channel blocker therapy, and low rates of double or triple calcium channel blocker therapy among patients in this study. The authors suggest that more aggressive calcium channel blocker therapy may have reduced the rates of recurrent VA but were otherwise unable to identify any other clinically significant predictors of recurrent VA [6].

In summary, based on limited data showing a high rate of VA recurrence in VSA, as well as no clearly identifiable clinical characteristics to differentiate those at highest risk of VA

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recurrence, the decision was made to refer this patient for ICD implantation. This is in line with current guidelines which state that in patients resuscitated from sudden cardiac death due to coronary artery spasm, an ICD in addition to medical therapy may be reasonable if meaningful survival of greater than 1 year is expected (IIB recommendation) [7].

Follow-Up

The patient was subsequently lost to follow-up until she returned to the hospital nearly one year later due to an episode of syncope and an ICD shock for ventricular fibrillation in the setting of intermittent medical adherence. She was discharged on higher doses of coronary vasodilators.

Conclusion

This case highlights how secondary prevention of ventricular arrhythmias in vasospastic angina differs from that of successfully revascularized MI. A low threshold for ICD implantation should be considered in those who present with ventricular arrhythmias in the setting of coronary vasospasm. Larger prospective studies are needed to further refine this approach.

Learning Objectives

a) Recognize the presentation of sudden cardiac death secondary to vasospastic angina.

b) Understand how the approach to secondary prevention for ventricular arrhythmias in this population differs from those with atherosclerotic myocardial infarctions.

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