CASE REPORT

Coronary artery spasm: An often overlooked diagnosis

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ABSTRACT

Coronary vasospasm can lead to myocardial injury and even sudden cardiac death. It has generally been overlooked as a diagnosis since atherosclerosis is a more common cause of acute coronary syndromes and because of the dilemma involved in its diagnosis. A middle-aged man with a history of smoking and cocaine use presented to the emergency department with left-sided arm/chest discomfort and diaphoresis. The electrocardiogram showed anterior ST elevation and hyper-acute T waves, which completely resolved shortly after sublingual nitroglycerin was administered. Subsequent angiogram revealed a 70% focal stenosis in the mid-left anterior descending artery. Coronary vasospasm occurs more commonly in arteries with underlying atheromatous disease, although normal vessels are not excluded. Cigarette smoking and cocaine use are among the major culprits that have been implicated as risk factors for the occurrence of coronary vasospasm. Eventually, the patient had percutaneous coronary intervention of his left-anterior descending artery and remained asymptomatic.

Key words: Angina, coronary, prinzmental, vasospasm

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INTRODUCTION

A syndrome that closely resembled acute myocardial infarction from thrombosis was first described by Prinzmetal *et al.,* in 1959. It was described as a "variant angina," which typically occurred at rest and was associated with transient acute ischaemic electrocardiogram (EKG) changes.[1] It was later discovered to occur from spasm of coronary arteries that resulted in occlusion of the arteries.[2]

Coronary artery spasm is often missed because of its infrequent incidence and the dilemma involved in its diagnosis;[3] it is an uncommon cause of acute coronary syndrome and ventricular arrhythmias.

We report a case of myocardial injury from coronary vasospasm occurring in a middle-aged man with a history of active cigarette smoking and of previous cocaine use.

CASE REPORT

A 49-year-old African-American man presented to the emergency department with sudden onset of left arm discomfort and to a much lesser extent, left-sided chest discomfort, which started about 30 minutes before presentation. This was associated with diaphoresis and it occurred at rest. Of note he had 2 similar events in the preceding month but did not seek medical intervention as symptoms resolved within 10 minutes of onset. He was an active smoker with a 35-pack-year history and also had a history of cocaine use in the past. Cardiovascular examination was unremarkable and the vital signs recorded were: blood pressure of 145/95 mmHg, a heart rate of 69 bpm, respiratory rate of 16 cpm, temperature of 98.3° F, and oxygen saturation of 98% on room air. The EKG on admission showed normal sinus rhythm, left ventricular hypertrophy and no acute ischaemic changes. He received aspirin 325 mg orally. His symptoms resolved shortly after presentation. The troponin I level was <0.015.

The patient was to be discharged but again developed left-sided arm and chest discomfort with diaphoresis while walking in the emergency department. Vital signs at this time were, a blood pressure of 153/103 mmHg, heart rate of 62 bpm, respiratory rate of 17 cpm, and oxygen saturation of 100% on room air. An EKG done immediately showed ST elevation and hyper-acute T waves in leads V2, V3, and V4 [Figure 1]. He was given nitroglycerin 0.4 mg sublingually twice and intravenous morphine 4 mg and his discomfort resolved. The troponin I level drawn at that time returned as 0.477. The EKG was repeated (35minutes after the preceding EKG), and it showed a resolution of ST elevation and hyper-acute T waves post treatment [Figure 2].

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The patient was subsequently admitted to the coronary care unit where he received clopidogrel, lovenox, simvastatin, lisinopril, and isosorbide mononitrate. Beta-blocker therapy was withheld pending urine toxicology result; his resting heart rate was between 50 bpm and 60 bpm. An echocardiogram done later revealed moderate to severely reduced left-ventricular function with an ejection fraction of 35% and severe anterior wall hypokinesis. Urine toxicology was negative for cocaine.

The troponin I peaked at 0.716 overnight and declined to 0.177 by the following morning. A diagnostic angiography done that morning revealed a 70% focal stenosis in the mid-left anterior descending artery, whose appearance was consistent with an atheromatous plaque [Figure 3]. Metoprolol was added on discharge for the left-ventricular dysfunction.

DISCUSSION

This case depicts a typical course of myocardial injury from coronary artery vasospasm. Patients with coronary vasospasm can present with atypical myocardial injury symptoms and cause a transient reduction in blood supply to the myocardium, which usually leads to transient EKG acute ischaemic ST or T wave changes.[2]

In a patient with or without atherosclerosis who presents with chest pain and EKG changes suggesting acute myocardial infarction a response to nitroglycerin can be used to distinguish the diagnosis of coronary vasospasm from a true acute myocardial infarction. If the chest pain diminishes and EKG changes revert to normal or baseline with nitroglycerin administration, the diagnosis can be said to be coronary spasm, but if the chest pain does not diminish and Q waves begin to appear, the diagnosis becomes acute myocardial infarction.[2,4] As an inference, angina symptoms occurring at rest and EKG changes that resolve with resolution of angina symptoms typically after nitroglycerin administration can be the classical presentation of myocardial injury from coronary artery spasm.

Persistent coronary vasospasm that is left untreated or is unresponsive to nitroglycerin can directly lead to acute myocardial infarction and sudden cardiac death.[2] Further, via an indirect route, coronary artery vasospasm complicating an atherosclerotic coronary artery can lead to thrombus formation, which can cause an acute myocardial infarction and even sudden cardiac death although spontaneous coronary reperfusion occurs in the early stages of the myocardial infarction.[5] Coronary vasospasm has been postulated as a common precipitant of acute myocardial infarction in patients with atherosclerosis. This conclusion was arrived at while trying to answer the question of 'what accounts for the lower incidence of total obstruction in patients studied 6-24 h after the onset of chest pain than in patients studied in the first 6 h'.[2] Coronary vasospasm occurs more commonly at the sites of atherosclerosis because of increased vascular reactivity to

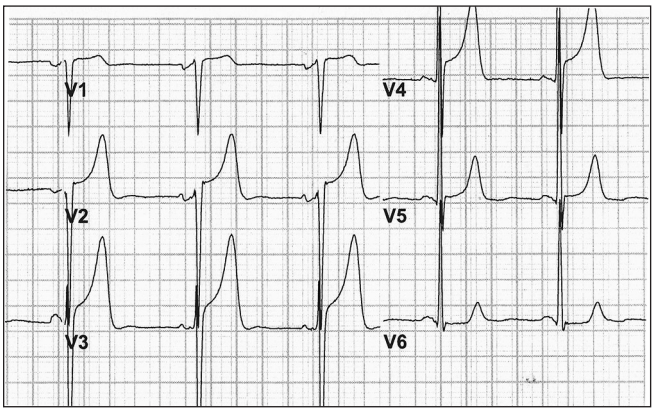

Figure 1: EKG showing Leads V1-V6 in the patient. The EKG was taken at the onset of chest pain in the emergency department and it shows marked ST elevation and hyper-acute T waves in V2, V3, and V4

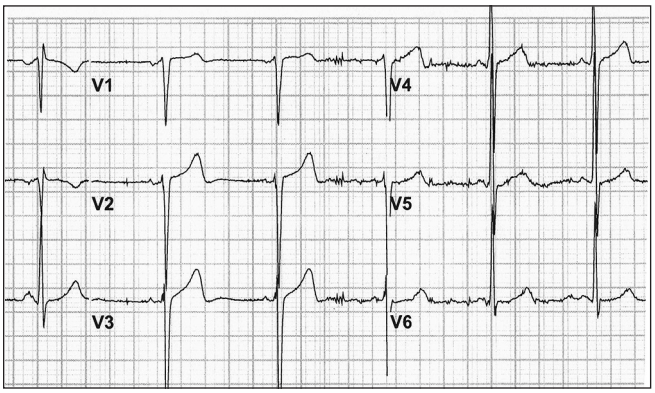

Figure 2: EKG showing Leads V1-V6 in the patient. The EKG was done 35 minutes after the previous EKG, shown in Fig. 1 (this was about 20 minutes after the 1st of the 2 doses of sublingual nitroglycerin given) and it shows resolution of initial EKG changes

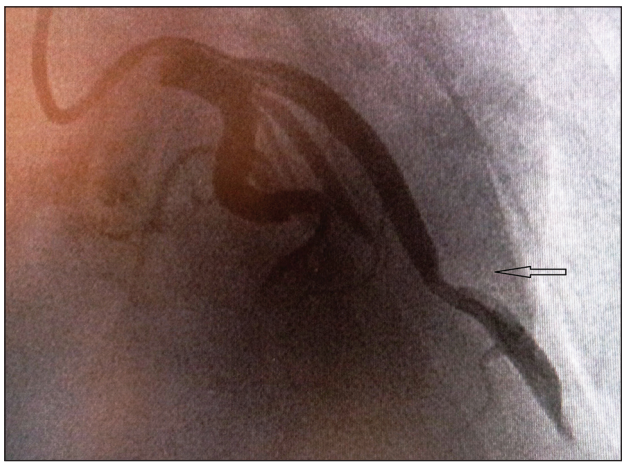

Figure 3: Coronary arteriogram showing area of 70% occlusion (arrow pointing towards the area)

vasoconstrictors such as thromboxane A2, thrombin, and serotonin at such sites.[6]

Cigarette smoking in our patient was a major risk factor for both atherosclerosis and spasm. Endothelium-mediated vasodilation has been found to be impaired in smokers so they would consequently have an increased susceptibility to vasospastic agents such as cocaine.[7] The patient in this case report also has a history of cocaine use although urine toxicology done was negative for cocaine. Cocaine has been linked to vasoconstriction and the already constricted coronary arteries make cocaine users more susceptible to clinically significant vasospasm. Cocaine has also been linked with having a direct vasospastic effect via an unknown mechanism.[7]

The definitive diagnosis of coronary vasospasm usually involves inducing the spasm under controlled conditions (usually in the cardiac catherisation lab) with a pharmacological agent, most commonly ergonovine.[6] However, provocative testing via the intracoronary route is infrequently used in clinical practice because of possible complications such as an irreversible spasm, although the incidence of these complications is low, as long as intracoronary nitrates are available to reverse the induced spasms.[8] Ergonovine echocardiography has also been postulated as a safe alternative to angiography. However, deaths have been reported from provocative testing and as such it has not been widely accepted as a safe method. These issues often make the definitive diagnosis a dilemma.[3] Historically, hyperventilation and the cold pressor test were used to identify spasm patients.[6]

Eliminating possible precipitating factors (cocaine use and smoking in our patient) should and always remain the first-line of therapy for any medical condition. Long-acting nitrates and calcium-channel blockers are the mainstay drugs of choice in preventing future coronary vasospastic episodes. Other drugs that have been tried with limited efficacy and have been reserved for refractory cases include endothelin antagonists such as bosentan.[9] Placement of stents can be considered in patients with coronary vasospasm refractory to medical therapy or in other carefully selected patients.[6]

Our patient had recurrence of symptoms on medical therapy and got percutaneous coronary intervention with bare-metal stent of his left-anterior descending artery. He was followed up in the out-patient clinic and he remained asymptomatic.

CONCLUSION

Coronary vasospasm should be considered in patients with a suggestive history and risk factors, particularly with dynamic electrocardiographic changes and clinical response to nitrates. Percutaneous coronary intervention with stenting may be done for medically refractory cases, as spasm most often occurs superimposed on coronary plaques.

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