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Acute Cocaine Myocarditis: A Mimic of ST Elevation Myocardial Infarction

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Abstract Cocaine remains one of the most commonly abused drugs in the United States and ingestion often presents with chest pain. The American Heart Association (AHA) published guidelines in 2008 on cocaine associated myocardial infarction (MI) to help assess and manage patients with obstructive coronary artery disease (CAD) in a timely manner. Cocaine may cause MI through increased platelet activation or through coronary vasoconstriction and spasm. However, cocaine induced myocarditis presenting as ST elevation myocardial infarction (STEMI) is uncommon. We report a case of a 35-year-old male with no significant medical history who presented with an 8-hour history of central chest pain. The patient admitted to ingesting cocaine within the last 8 hours and urine toxicology was positive for cocaine metabolites. EKG showed ST segment elevations in leads I and aVL and the patient was taken urgently for coronary angiography. Coronary angiography revealed no significant obstructive CAD. Transthoracic echocardiogram showed mildly reduced left ventricular ejection fraction (LVEF) and Cardiac MRI showed late gadolinium enhancement of the inferior and septal segments consistent with myocarditis. Cardiac MRI with contrast is a useful modality in differentiating ischemic from non- ischemic causes of STEMI after cardiac catheterization.

Keywords: *cocaine, myocarditis, cardiac MRI*

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1. Introduction

Cocaine remains one of the most commonly abused illicit drugs in the United States. The effects of cocaine on the heart are widespread and well documented in the literature. Cocaine likely induces myocardial ischemia and infarct through increased platelet activation leading to acute thrombosis, increasing myocardial oxygen demand through beta receptor stimulation and decreasing oxygen supply through coronary vasoconstriction [1]. The AHA published guidelines on cocaine associated MI in 2008 recommend timely percutaneous intervention if STEMI is suspected. However, acute cocaine induced myocarditis presenting as STEMI has infrequently been described in the literature.

2. Case Presentation

A 35-year-old male with no significant medical history presented to urgent care with an 8-hour history of retrosternal chest pain that he described as a pressure on his chest. He admitted to cocaine use in the 12 hours prior to presentation. The patient denied having any upper respiratory infection symptoms, cough or sick contacts in the last 2 weeks and denied starting any new medications. Electrocardiogram in the emergency room showed ST segment elevation of 2mm in leads I, aVL and 1 mm in leads V5 and V6 with ST segment depression in leads III and V1. Complete blood count including white cell count and differential was normal. Initial troponin I was 19.1 ng/ml. Urine toxicology screen was positive for cocaine metabolites. The patient was loaded with 325 milligrams of aspirin and 180 milligrams of ticagrelor and taken to the cardiac catheterization lab. Coronary angiography revealed no significant obstructive CAD. Echocardiography demonstrated mildly reduced left ventricular ejection fraction of 40% with moderate global hypokinesis. Cardiac MRI performed 24 hours after transthoracic echo revealed LVEF of 32% with global left ventricular hypokinesis, apical subendocardial edema, and delayed myocardial enhancement consistent with myocarditis. The patient was treated with beta-blockers and angiotensin converting enzymes inhibitors and discharged appropriately after adequate monitoring.

3. Discussion

Acute myocarditis should be considered in a young patient with cocaine use and normal coronary arteries on cardiac catheterization as a cause of ST elevation MI. Differentials to consider for acute MI in these patients are acute coronary spasm or thrombosis which lyses spontaneously. These patients should appropriately undergo

cardiac catheterization in a timely manner to rule out obstructive CAD. Acute myocarditis with cocaine use may be under reported. Reported autopsy data of patients with detectable cocaine levels at death by Virmani et al demonstrated increased mononuclear infiltrates suggestive of myocarditis compared with control groups [2]. It is unclear if these mononuclear infiltrates occur as a result of myocardium death or a primary hypersensitivity reaction [2]. Cocaine's direct negative inotropic effect on myocardium and an associated increase in catecholamine levels may have a direct toxic effect and induce myocyte apoptosis [3].

Gitter et al reported early repolarization abnormalities on EKG in about a third of their patient cohort with cocaine induced chest pain [4]. More than half of cocaine users had baseline abnormal EKGs [4]. About 42% of the patients had some manifestations of ST segment elevations but did not have positive biomarkers [4]. Only 2% of patients had changes concerning for an ST elevation MI [4]. Mohamad et al reported about 32% of patients met criteria for STEMI and found that the majority of patients presenting with cocaine associated MI had significant obstructive coronary artery disease [5]. EKG abnormalities in cocaine users are relatively frequent but offer limited diagnostic value.

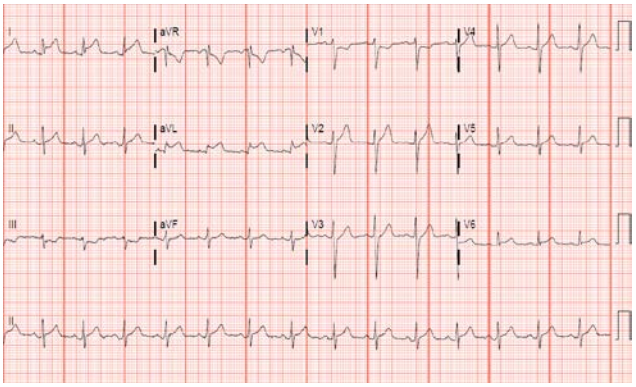

Figure 1. EKG showing: Sinus tachycardia with 2mm ST elevations in leads I and aVL

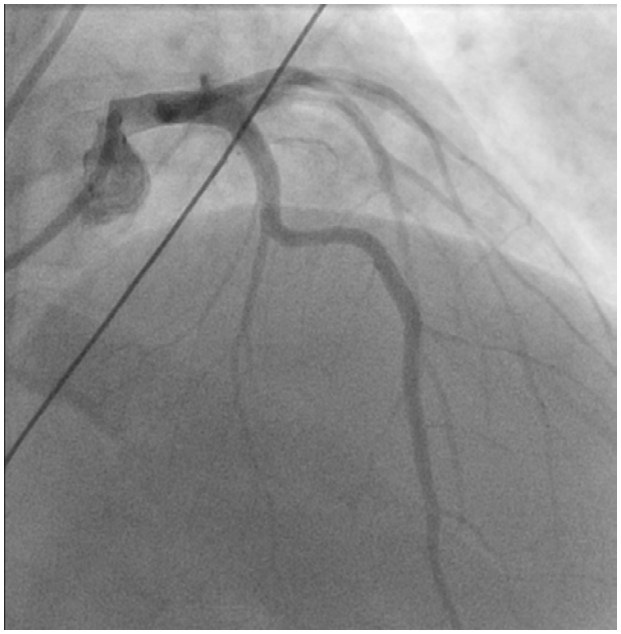

Figure 2. Coronary Angiogram demonstrating no obstructive coronary artery disease in the left main, left anterior descending and left circumflex coronary arteries

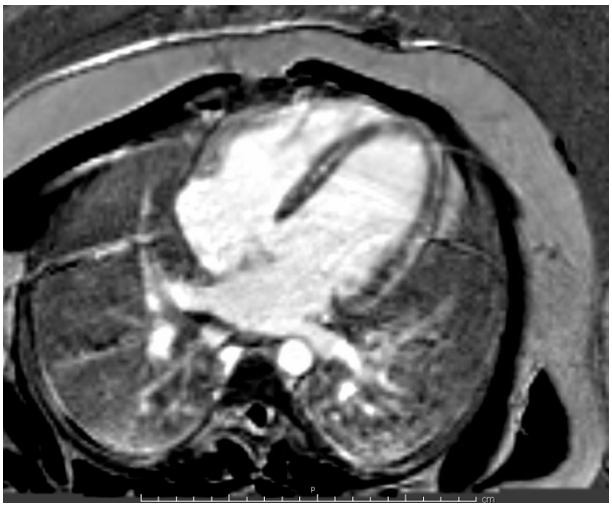

Figure 3. Delayed post contrast cardiac MR four chamber images showing myocardial enhancement in the septum and lateral wall

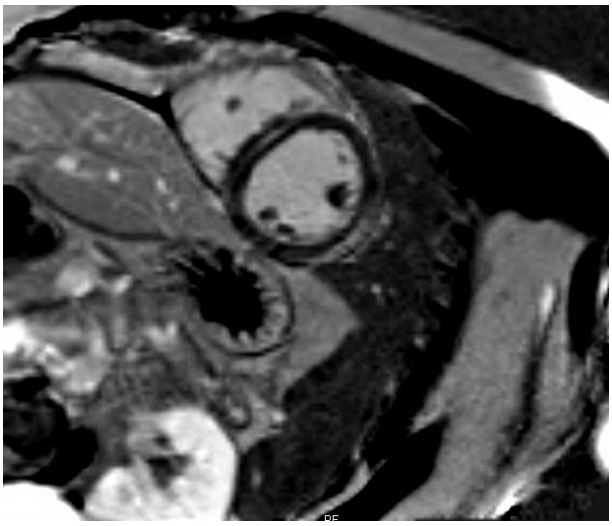

Figure 4. Delayed post contrast cardiac MR short axis images showing myocardial enhancement in the septum

Recent literature has described the echocardiographic characteristics of cocaine induced acute myocardial depression similar to Takotsubo cardiomyopathy which resolves after cessation of cocaine use [6]. This is characterized by transient left ventricular dilatation and depressed ejection fraction. Studies have shown that chronic cocaine users have an increased LV mass index, increased thickness of the posterior wall and may be at risk of developing concentric left ventricular hypertrophy [6].

Contrast cardiac MRI may be helpful in differentiating between ischemic and non-ischemic causes. Active myocarditis pathologically is characterized by intracellular and interstitial edema and in severe cases cell death and fibrosis [7]. Early gadolinium enhancement ratios can be used to detect distribution in the interstitial space suggestive of inflammation [7]. Late gadolinium enhancement on cardiac MRI suggests necrosis and fibrosis and correlates with a high specificity of myocarditis [7].

4. Conclusion

Subendocardial edema and delayed enhancement on Cardiac MRI in our patient suggests active myocarditis

with possible necrosis. A substantial portion of patients with cocaine induced chest pain and abnormal EKG will have obstructive CAD. Cardiac MRI is a useful modality in assessing cocaine associated STEMI in patients with no obstructive CAD on cardiac catheterization and may help differentiate ischemic from non-ischemic causes and offer valuable prognostic clues.

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