Cocaine Induced Myocardial Infarction

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INTRODUCTION

Cocaine use is commonly associated with acute cardiovascular effects rather than chronic effects while chest pain represents the most common symptom [1,2]. In those patients, exclusion of acute myocardial infarction or ischemia is vital in adition to other critical conditions like aortic dissectionand myocarditis [3]. Inhibition of norepinephrine reuptake into the synaptic cleft by sympathetic neurons is the major mechanism for cocaine related cardiovascular effects resulting in potentiation of the response to sympathetic stimulation increasing myocardial oxygen demand and decreased myocardial perfusion leading commonly to chest pain which is often accompanied by anxiety, dyspnea, palpitations, and nausea however it is very difficult to be differentiated from other causes of chest pain and based on that all young patients with an acute myocardial infarction (AMI) or ischemiacocaine use should be excluded[8,9]. Cocaine can constrict the large as well as the small coronary vessels mediated primarily through stimulation of the alpha-adrenergic receptors[10-13]. Cocaine is structurally similar to acetylcholine, and stimulation of cholinergic receptors in small vessels that lack endothelium or in large vessels with dysfunctional endothelium is a putative mechanism of cocaine-induced vasoconstriction and ischemia [14]. Cocaine induced coronary thrombus formation mainly due to platelets activation, stimulation of platelet aggregability, and potentiation of thromboxane production [4-6]. All patients with chest pain following cocaine ingestion should promptly receive a 12-lead electrocardiogram (ECG) and in case of

ECG evidence of acute ischemia patients require admission to the hospital and should be evaluated and managed according to the standard protocol for patients with acute myocardial infarction with one exception is that drugs with beta receptor blocking properties should generally be avoided in cocaine users(15,16). While the most common acute condition is myocardial ischemia, only approximately 6% of patients with chest pain and recent cocaine use develop a myocardial infarction (MI). Most cocaine-associated MI occur in the absence of significant atherosclerotic coronary artery disease.The evaluation of a patient with chest pain following cocaine ingestion is similar to that of nonusers with chest pain and includes complete history and physical examination, 12-lead ECG, Serial serum biomarkers (troponin T or troponin I) in adition to radiologicalevaluation based on the clinical presentation, ECG findings, and cardiac biomarkers. Coronary computed tomography angiography (CCTA) may be needed in select patients to document large vessel coronary pathology with equivalent diagnostic value to invasive coronary angiography. However, if an acute coronary syndrome is highly suspected, invasive coronary angiography, whichcan provide the option to revascularize affected vessels if needed, is the preferred approach.Additionally, toxicology assays for cocaine or its metabolites in urine may be useful if exposure to cocaine is suspected or requires confirmation. Recent cocaine ingestion confirmed by testing the urine for cocaine and its metabolites, rise of cardiac biomarkers, along with supportive evidence in the form of typical symptoms,

suggestive ECG changes, or imaging evidence of new loss of viable myocardium or new regional wall motion abnormality represents classic presentation of acute myocardial infarction due to cocaine use.Based on American Heart Association scientific statement on the management of cocaine-associated chest pain and MI and the guidelines for the management of patients with unstable angina/NSTEMI [7,15,16] patients with cocaine-related myocardial infarction are mainly managed in a manner similar to other patients with these diagnoses. One notable exception is the use of beta blockers which are not recommended in the early phases of acute coronary syndromes (ACS) in patients with recent cocaine use prior to elimination of the cocaine from the patient's body based on concerns of coronary artery vasoconstriction and systemic hypertension, which can result from unopposed alpha-adrenergic stimulation. Aspirin is an important part of the early treatment of chest pain associated with cocaine use, particularly given the propensity of cocaine to induce thrombus formation via the activation of platelets, stimulation of platelet aggregability, and potentiation of thromboxane production. Unless there is also a high suspicion for acute aortic dissection, aspirin 325 mg (nonenteric coated) should be given to the patient to chew and swallow. Nitroglycerin and calcium channel blockers — Sublingual or intravenous (IV) nitroglycerin (NTG) and/or oral calcium channel blockers are safe and effective medications for patients with ischemic chest discomfort and ST-segment elevation or depression.Calcium channel blockers are used as adjunctive therapy in patients with ongoing or recurrent symptoms of ischemia despite optimal therapy with NTG. Patients with recent cocaine ingestion should receive adequate doses of benzodiazepines, as needed, for sedation and control of blood pressure and heart rate. Early invasive strategy in some patients with NSTEMI, early reperfusion

strategies in patients with STEMI and the appropriate use of antithrombotic drugs are considered in cocaine induced MI [7].

Case report:

We present a 30 years old male patient with cocaine inducedSTEMIwith a past medical history of smoking and recent intake of 2 gram cocaine by snorting method(sniffing the powder into the nasal passages)from 14 hours presented with an acute onset of retro-sternal chest pain radiating to left arm and lasting 10 hours since its onset. Upon arrival, his blood pressure was 105/7, pulse 65, respiratory rate 17, and oxygen saturation of 97 percent on room air. His electrocardiogram (**Figure1)** demonstrated normal sinus, normal left axis, 1-2 mm ST-elevation in precordial leads V1-V3. Initial labs demonstrated an elevated total CK level(499 U/L) and elevated cardiac myoglobin(93 ug/L), normal initial troponin T of less than 5 ng/l with rising titre after 2 and 8 hours to 46 and 2135 ng/l, NT-proBNP 24 ng/l, total cholesterol 4.8 mmol/L, triglycerides 1.2 mmol/L, white blood cell count 12.81 per uL with relative neutrophilia 8.48 per uL, hemoglobin 13.6 g/dL, platelets 229 per cmm, sodium 140 mEq/L, potassium 3.2 mEq/L, creatinine 103 mmol/L with estimated GFR 84 ml/min and alcohol blood levelof less than 2.2 mmol/L. Emergent cardiac catheterization was performed. Access was obtained through the right radial artery and a 6 French (Fr) arterial sheath was inserted. Diagnostic coronary angiography demonstrated a hazythrombus like mass in the proximal portion of the left anterior descending coronary artery**(Figure2).** A 6 Fr, EBU 3.5 guide catheter was used to engage the ostium of the left coronary artery and ASAHI Sion Blue(Terumo) was advanced in the LAD artery and we decided to perform OCT using C7 DRAGONFLY™ (St. Jude Medical)that confirmed the presence of thrombus in proximal LAD(**Figure3**). We decided to perform direct proximal LAD stenting using 1 Resolute Integrity Stent4.5x30

mm(Medtronic) and we repeated OCT after stent implantation that showed mild stent apposition**(Figure4)**. Stent after dilatation using Quantum high pressure ballon(Boston Scientific) 4.5x15 mm with excellent final angiographic
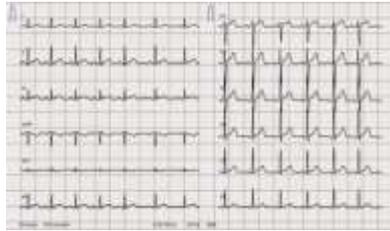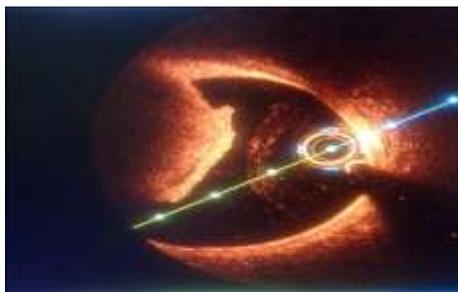

Figure 112 lead ECG) showing normal sinus, normal left axis, 1-2 mm ST-elevation in precordial leads V1-V3.

Figure 2 Coronary angiography RAO 15/CR 30 view showing hazy thrombus like mass in proximal LAD.

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results**((Figure5).** Patient discharged on DAPT Aspirin 100 mg/d and Prasugrel 10 mg/d in addition to Rosuvastatin 20 mg/d and Amlodipine 10 mg/d, patient is doing well after 6 months follow up.

Figure 3 OCT image using C7 DRAGONFLY™(St. Jude Medical) confirming the presence of thrombus in proximal LAD

Figure 4 OCT image using C7 DRAGONFLY™(St. Jude Medical) showing mild stent apposition in proximal LAD.
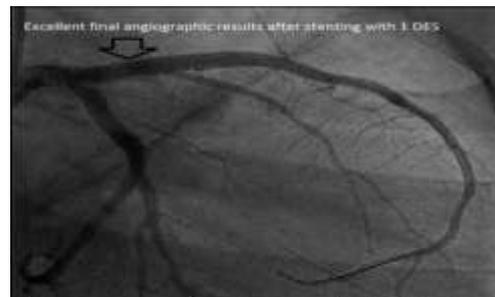

Figure 5 Coronary angiography RAO 25/Caudal 33 view showing excellent final angiographic results after stenting with 1 DES.

differentiated from other causes of MI mandating the need of high index of

suspicion especially in young patients presenting with acute myocardial infarction or acute aortic dissection, The evaluation and management of a patient with suspected myocardial infarction or

Discussion

Cocaine is a recreational drug with sympathomimetic effects in additional to being a sodium channel blocker, Cocaine can induce acute MI through vasoconstriction, atheroma rupture and/ or dissection. Chest pain in patients with cocaine-induced MI is difficult to be

ischemia following cocaine use is similar to that of nonusers with one exception thatbeta-blocker use is best avoided in the acute management of cocaine-induce acute myocardial infarction due to concerns of unopposed alpha effect. Management includes complete history and physical examination, 12-lead ECG, Serial serum cardiac biomarkers in adition to

radiological evaluation based on the clinical presentation, ECG findings, and cardiac biomarkers, coronary computed tomography angiography (CCTA) has equivalent diagnostic value to invasive coronary angiography. However, if an acute coronary syndrome is highly suspected, invasive coronary angiography is preferred as it can provide the option to revascularize affected vessels if needed. Toxicology assays for cocaine or its metabolites in urine may be useful if exposure to cocaine is suspected or requires confirmation. Cocaine use cessation is extremely important excellent prognosis for future ischemic cardiac events.

Conclusion

Cocaine induced MIdiagnosisrequires high index of suspicion especially in young patients presenting with chest pain. Management of Cocaine induced MIis similar to that of nonusers with one exception thatbeta-blocker use is best avoided in the acute management of cocaine-induce acute myocardial. Invasive coronary angiography is preferred in case of suspected cocaine induced MI after exclusion of other fatal diagnoses like acute aortic dissection as it provides the option of early culprit artery revascularization. We presented two case reports with STEMI in young patients after recent cocaine intake and both of them referred to invasivecoronary angiography and treated with stenting with excellent final angiographic results.

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