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Case Report

Acute Myocardial Infarction as a Sequel of Cocaine Abuse

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Abstract

Cocaine is considered the second most frequently used illicit drug and the most frequent cause of drug-related deaths in the world. Worldwide, the majority of cocaine users are younger people between the ages of 16 and 26 years old; and 11% of the general population is thought to have used it at some point in time. Cocaine users primarily administer cocaine orally, intranasally, intravenously, or by smoking it. Cocaine abuse leads to serious complications that involve all body systems, especially the cardiovascular system. In this study, we reported a case of acute myocardial infarction in a young patient without known chronic diseases. Twenty years old male patient presented with acute epigastric pain and repeated vomiting. He has a two-year history of cocaine abuse. All routine investigations were rapidly done; however, the patient collapsed and cardiopulmonary resuscitation (CPR) was performed. Clinical outcome: the patient, unfortunately, died after all trials to save his life and was diagnosed with severe myocardial infarction.

Keywords: Acute coronary syndrome, Cocaine abuser, Coronary spasm, Platelet aggregation,plaques.

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Case Report: Twenty four years old male arrived at the Emergency Department of Sebha Medical Center on April 15, 2007 at 01:00 p.m. complaining of sudden severe epigastric pain half an hour before he arrived at the hospital. The pain associated with vomiting contains food particles three times with no hematemesis or melena. The bowel motion was normal, the patient has no history of peptic ulcer or gastritis. The patient had two years history of heavy cocaine smoking and

alcohol addiction, he does not has any known chronic medical condition or history of trauma; however, he had only a family history of hypertension. On examination: the pain score was 10, he was alert, mild dyspneic and tachypneic, and his heart rate was 105 beats/min. The abdomen was soft and tender at the epigastric region. The other vital signs and examinations were normal.

The case was treated as acute gastritis; Ranitidine and Malox were given and the patient was sent for chest and abdominal X-Ray. The ECG was normal at this time; however, when the patient returned from the X-Ray, the Glasgow Coma Scale (GCS) dropped from 15 to 11, the blood pressure dropped from 120/77 to 80/55) and the heart rate declined from 105 to 50 beats/minute. The patient was transferred to the intensive care unit (ICU). In the ICU the ECG showed ST elevation at inferior leads (II, III, avf) as shown in Figure 1. Blood investigations were done and were as follows: WBC: 10 x10^3, HB:16.4gm/dl., platelets: 300x10^3, ESR: 20mm/h, blood sugar, 103 mg/dl., urea 44, creatinine: 1.1 mg/dl, serum electrolytes were within normal ranges, amylase: 44 u/l., troponin: 0.7 ng/ml, creatinine kinase MB: 11 ng/ml, s-cholesterol: 65mg/dl and triglycerides: 50 mg/dl.

The echocardiography was not done, and illicit drug tests were not available at that time. During the preparation for the intubation, the patient collapsed and the cardiac monitor showed ventricular fibrillation (VF). Immediate CPR, DC shock and adrenaline, three times refractory VF, amiodarone 150mg, 300mg was given, but the patient developed asystole. CPR and the adrenaline continued for 25 minutes with no response. The patient died after 2 hours after arriving at the ICU.

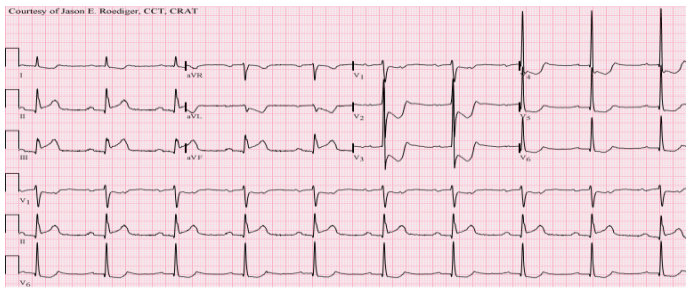

Figure 1: ECG of the patient

Discussion: Acute coronary syndrome may occur in cocaine abusers, it has been reported that myocardial infarction induced by cocaine abuse is common in young patients between 16 to 40 years old without any angiographic evidence of coronary artery disease [2]. It has also been reported that coronary artery occlusion can be due to coronary artery spasm in addition to platelet activation, which can play an important role in the acceleration of

coronary artery disease [2, 3]. The pathophysiology of myocardial infarction is usually associated with a progression of atherosclerotic coronary artery disease, with or without plaque rupture as shown in Figure 2.

Animal studies have shown that atherosclerotic changes could be more extensive with cocaine administration, and therefore, coronary artery disease can be induced by cocaine. Moreover, coronary artery disease was confirmed in 42% of cocaine users who underwent cardiac catheterization [1]. In addition, inhibition of catecholamine uptake in patients who might have aortic dissection may increase the risk of atherosclerotic and thereby will increase blood pressure [2]. The calcification has also been observed more in cocaine users [3]. However, previous studies showed that the risk factors such as hypertension, hyperlipidemia and diabetic mellitus in cocaine abusers might rapidly progress the acute coronary diseases [1].

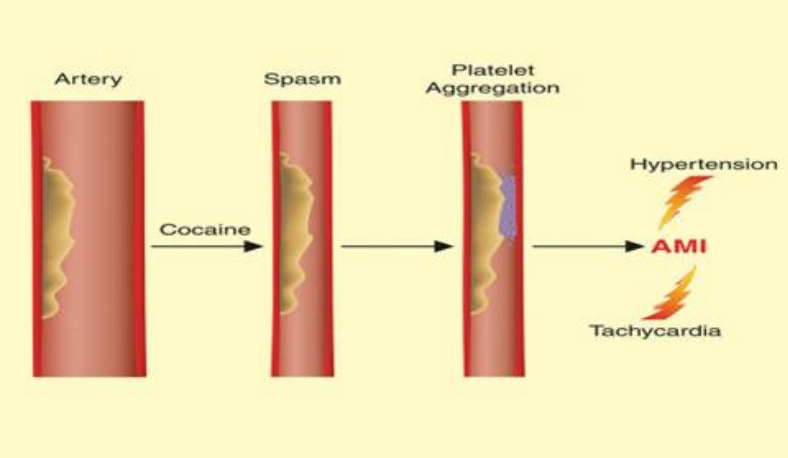

Figure 2: Diagram illustrates the mechanism of cocaine-induced myocardial infarction.

In cocaine-induced acute myocardial infarction, the rupture of plaque was less common despite extensive coronary atherosclerosis [4]. A good history and proper clinical examination of the patient along with appropriate investigation including cardiac enzymes (troponin and creatinine kinase) and classic electrocardiographic findings are important for early diagnosis and management, especially the elevated troponin, which is more specific than creatinine kinase in this situation. Other investigations such as blood tests may be helpful for drug-level screening when cocaine abuse is suspected.

Since the cocaine level can remain in the urine for up to 8 days after use, cocaine metabolites such as benzoylecgonine and ecgonine can be found and used in cocaine detection. The hair of a cocaine abuser can also be used to detect the presence of cocaine since it can last for a very long time in the patient's hair [4]. Furthermore, the two-dimensional echocardiogram can show regional wall motion abnormality.

There is no specific treatment for cocaine-induced acute myocardial infarction. Primary percutaneous coronary intervention (PCI) is the preferred method for ST-elevation myocardial infarction (STEMI). Unfortunately, Intracerebral haemorrhage and aortic dissection can be a complication in patients with cocaine intoxication and thrombolysis can be fatal in this situation [7].

The good effect of Glycoprotein IIb/IIIa (GP IIb/IIIa) inhibitors has been reported to have a positive effect; in addition, the use of antiplatelet with GpIIb/IIIa inhibitor is recommended along with conservative treatment for intrinsic fibrinolysis [6]. If hemodynamic deterioration happened then angiography should be repeated to assess the patency of the vessels [5]. Psychosocial and drug compliance assessments should be routinely done for all cocaine-induced acute myocardial infarctions.

Conclusion

: Identification of patients with acute coronary syndrome or chest pain who have a history of cocaine abuse especially young patients under 40 years old who does not have multiple risk factors for coronary artery disease is important. This importance is not only for statistical and epidemiological purposes but also because their treatment differs in specific ways from the treatment of major coronary events. Furthermore, to avoid the recurrence of cocaine-related MI, patients should be educated about the complications of cocaine abuse.

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