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Case report

ACUTE MYOCARDIAL INFARCTION ASSOCIATED WITH COCAIN USE IN YOUNG PATIENT

Doan Chi Thang[1], Huynh Thuc Bao[2], Hoang Anh Tien[3]
[1]Cardiology Department, Hue Central Hospital
[2]Cardiology Department pf Hoan My Hospital Da Nang
[3]Hue University of Medicine and Pharmacy

ABSTRACT

Background: *Acute myocardial infarction may occur following cocaine abuse. Cocaine-induced infarction is particularly common in younger patients aged 18 to 45 years old. Patients may or may not have evidence of coronary artery disease at the time of symptom presentation. We report the case of a 17 - year - old male patient who had an acute myocardial infarction after cocaine abuse and had underwent emergency coronary intervention. Our case highlights important treatment taken in dealing with this acute scenario.*

Case report: *Male, 17 years old, history of cocain use in 2 days, smoking 1 pack.year, no history of disease or appearance of symptoms related to cardiovascular before. The patient was admitted to the hospital in the context of intermittent shortness of breath with constant pain in the chest behind the sternum. Laboratory tests aim to diagnose acute myocardial infarction. The patient underwent emergency coronary angiography and intervention with 2 stents in the left anterior descending artery and the right coronary artery. After coronary intervention, the patient is awake, no chest pain, and vital signs are stable.*

Conclusion: *Acute myocardial infarction is rare, but is a serious consequence of cocain use. Clinicians should consider interrogating all young patients presented to the emergency department with acute chest pain for cocaine abuse. Also, appropriate addiction management in this patient population is considered important secondary prevention strategy that helps reduce the risk of future cardiovascular events.*

Keywords: *Myocardial infarction, cocain use, young patient.*

I. INTRODUCTION

More than two decades ago, experimental studies demonstrated that drugs administered to experimental animals lead to atrial and ventricular rhythm disturbances, myocardial ischemia with coronary artery spasm, as well as heart failure. directly reduces cellular function [1]. Similar effects also occurred in humans; in the mid-1980s, Isner and colleagues reported severe ventricular arrhythmias, myocardial infarction, and even temporary sudden death associated with drug use [2]. More worryingly, the occurrence of cardiac side effects occurs even when the drug is used medically as a local anesthetic. Drug use also leads to other heart - related disorders, such as cardiomyopathy, hypertension, aortic aneurysm, and noncardiac chest pain. The incidence of complications is much higher (25%) in younger patients aged 18 to 45 years 13 and especially in those with pre-existing cardiovascular risk factors [3].

In Vietnam, Illicit drug use is a serious issue, but information about their prevalence is not clear, mainly based on seizure data. Wastewater-based epidemiology is an alternative tool for obtaining such information, especially when stigma related to drug use can hinder survey approaches. Prevalence of cocaine use was low compare to other drugs [4]. In addition, there have been no reports in Vietnam

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Corresponding author: Doan Chi Thang. Email: thangdoanchi1981@gmail.com. Phone: 0905469595

about myocardial infarction in patients abusing cocaine. We report the case of a 17 - year - old male patient who had an acute myocardial infarction after cocaine abuse and had underwent emergency coronary intervention. Our case highlights important investigation about cocain use and treatment taken in dealing with this acute scenario

II. CASE PRESENTATION

Male, 17 years old, used drugs 2 days ago, smoked cigarettes for 2 years (1 pack.year), the patient and his family have no history of cardiovascular disease before. The patient was admitted to the hospital with intermittent difficulty breathing with constant pain in the chest area behind the sternum, no fever, no symptoms of cough or previous viral infection. His vital signs during examination: blood pressure: 130/90 mmHg, pulse: 85 beats/minute, respiratory rate 21 beats/minute, SpO2 95%. 12-lead electrocardiogram: ST elevation in lead III, aVF, aVR; ST elevation in V2-V4 (Figure 1)

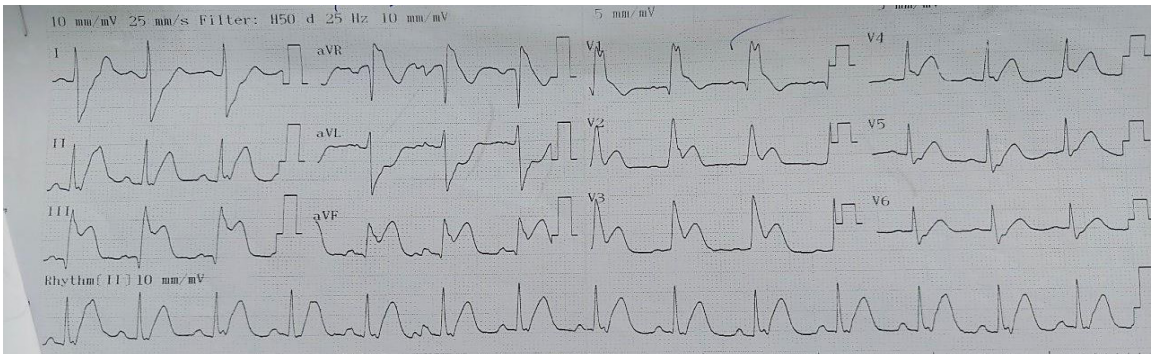
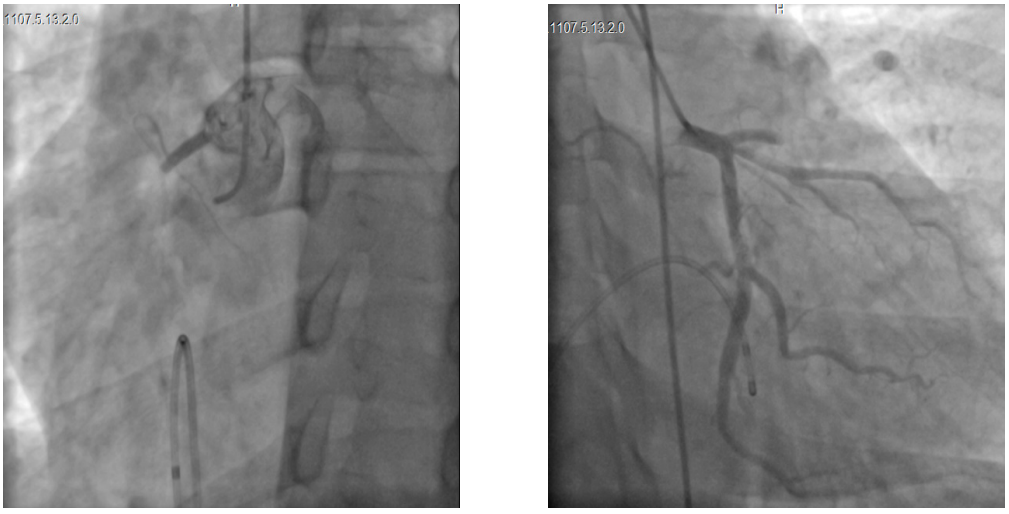


Figure 1: Electrocardiogram during hospital admission

Laboratory tests: Troponin I: 7.0 pg/ml; NT-proBNP: 141.3 pg/ml; Creatinine 99.9 µmol/l; Glucose 7.66mmol/l; SGOT 31 U/L; SGPT 18 U/L; Cholesterol 4.46 mmol/l; LDL-C 3.07 mmol/l; Triglyceride 3 mmol/l; Blood sodium 133 mEq/l; Blood potassium 3.3 mEq/l; Blood calcium 2.2 mEq/l; arterial blood gas showed pH: 7.42; pCO2 33 mmHg; pO2: 103.5mmHg; HCO3 18.8 mmol/l. At the emergency department, the patient was treated according to the protocol for acute myocardial infarction with anticoagulants, antiplatelet agents, statins and was consulted with the interventional cardiovascular team and decided to undergo angiography and intervention for patients. Angiography showed complete occlusion of left anterior descending artery and right coronary artery (Figure 2)



A B

Figure 2: (A)Complete occlusion of segment I of the left anterior descending artery large thrombotic burden. (B) Complete occlusion of the right coronary artery from segment I.

During the procedure, the patient's condition worsened with the appearance of ventricular fibrillation on monitoring, followed by third-degree AV block, sporadic ventricular rhythm, and invasive blood pressure that could not be measured. After receiving intensive cardiopulmonary resuscitation for nearly 15 minutes, the patient had a weak pulse and low blood pressure of 70/40mmHg. When the patient is in shock, the sooner the coronary artery is revascularized the better the chance of saving the patient's life. The option of thrombolysis and vasodilators is no longer the preferred choice in this patient. The intervention team decided to intervene with balloon dilatation and place a drug-eluting stent for the patient to restore flow in the fastest time (Figure 3).

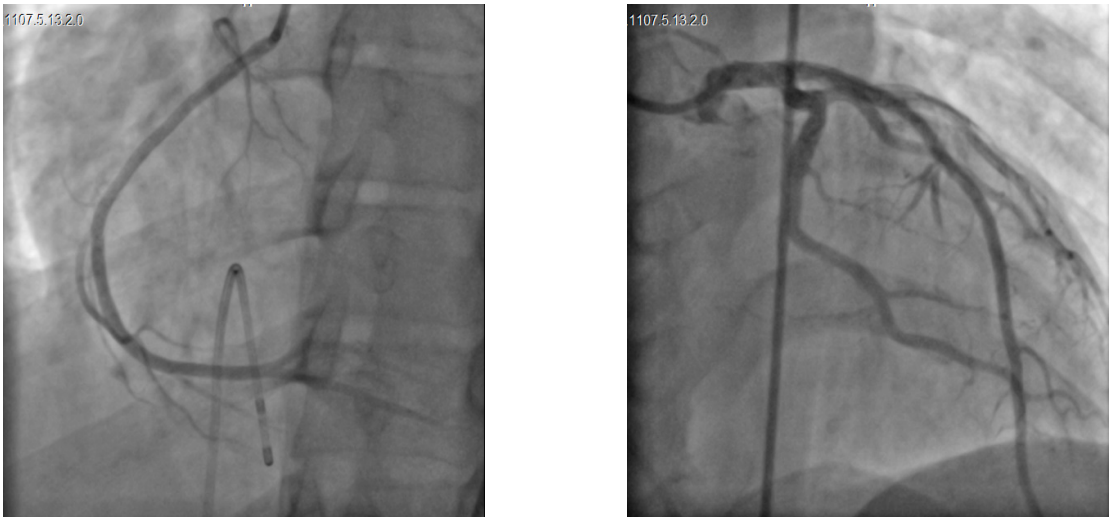


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Figure 3: The patient had 2 drug-eluting stents placed in the left anterior descending artery (A) and the right coronary artery (B)

The patient received balloon dilation and a 3.5 x 33 mm Eucalimus-eluting stent was placed at the occlusion site. After intervention, downstream flow was good, TIMI III. The stent is dilated using a high-pressure balloon. The patient's hemodynamics gradually stabilized. Intervention team evaluates right coronary artery intervention. After passing the wire through the lesion, the coronary artery must re-open but cannot completely recover after administering local vasodilators. The right coronary artery was intervened with a 2.75 x 43 mm Eucalimus - eluting stent. Dilation with a 3.0 x 15 mm high-pressure balloon. Take a good flow check, TIMI III. The patient had 2 drug-eluting stents placed in the anterior interventricular artery and right coronary artery. After the intervention, the patient was awake, no longer felt chest pain, no difficulty breathing, the temporary pacemaker conducted well, blood pressure was 90/ 50mmHg, vasopressor dose gradually reduced, electrocardiogram and Troponin I showed improvement (Figure 4 and 5). The patient was cared for and treated at the cardiovascular resuscitation department for 4 days, was discharged from the hospital for outpatient treatment and had regular follow-up examinations, his health was stable, improvement of left ventricular function (Figure 6), without any signs of chest pain or clinical manifestations of heart failure.

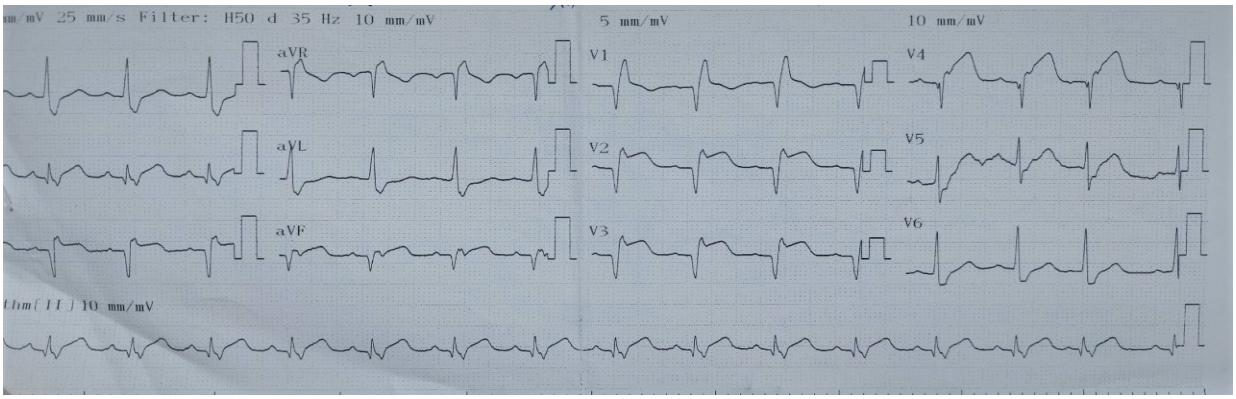

Figure 4: Electrocardiogram 24 hours after intervention

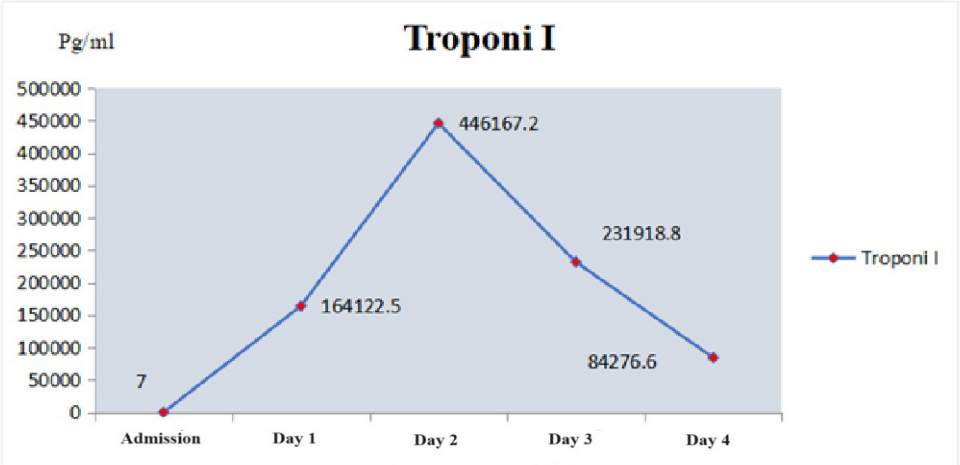

Figure 5: Troponin I level progression

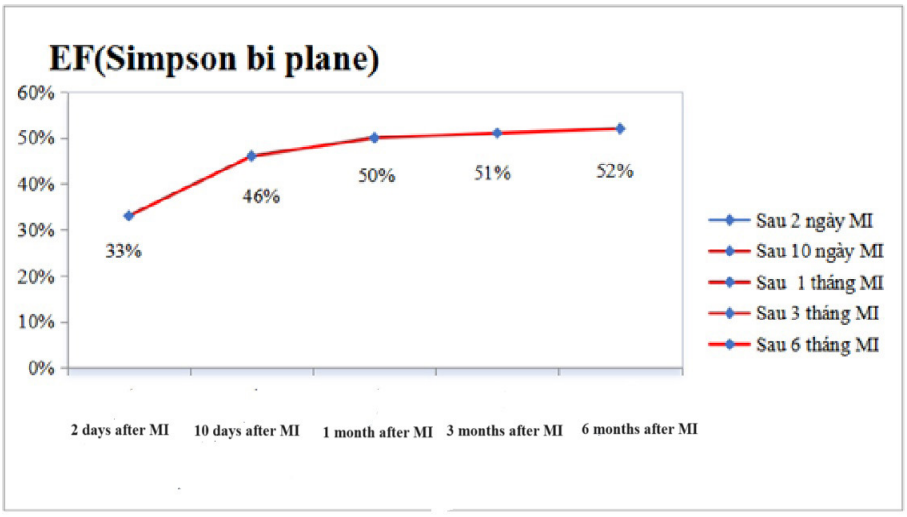

Figure 6: EF follow-up after MI

III. DISCUSSION

Cocaine use is the major cardiovascular risk factor. The effects of drugs on the cardiovascular system are diverse, with several mechanisms that can facilitate myocardial ischemia and lead to acute myocardial infarction. The primary mechanism involves increased circulating norepinephrine secondary to reuptake inhibition. This leads to

increased cardiovascular sympathetic response, increasing myocardial oxygen demand and reducing myocardial perfusion secondary to coronary vasospasm. Drugs accelerate atherosclerosis through the development of vascular endothelial defects leading to increased peroxidase and low-density lipoprotein permeability, and thrombi are formed due to increased activation and platelet aggregation. The importance of this case report lies in knowing that the mechanism of myocardial infarction in this group of patients is not only secondary to coronary vasospasm, but that drugs also damage the vascular endothelium, causing increases platelet aggregation and impairs normal fibrinolytic pathways. Thus, acute myocardial infarction is not solely the result of the sympathetic response, and therefore the use of Aspirin, P2Y2 receptor antagonists, glycoprotein IIb/IIIa inhibitors, heparin and statins is clearly justified. pathophysiologically in this group of patients [5].

If a drug user presents with an ST-elevation myocardial infarction within 6 hours of the onset of chest pain, immediate thrombolytic therapy or emergency percutaneous coronary intervention should be performed as soon as possible. Emergency intervention is always a top priority if the medical facility has a qualified cardiovascular intervention team available. In cases where the medical facility does not have a cathlab, the patient should receive intravenous thrombolysis immediately [6, 7]. In a retrospective study of drug users, thrombolytic therapy was found to be safe, but patients must meet exclusion criteria and be assured of no contraindications before initiating therapy. It is necessary to carefully examine and do the necessary tests to assess the risk of bleeding, especially cerebral hemorrhage, and at the same time rule out the possibility of aortic dissection and carotid artery dissection, which are common in patients who abuse drugs [8, 9].

Aspirin, P2Y2 receptor antagonists, heparin, and statins should be administered promptly at the recommended dose [10]. Although it is recommended that all patients with myocardial infarction should be treated with beta-blockers early to reduce infarct size, reduce arrhythmias and improve mortality. However, in drug users, blocking beta receptors may counteract alpha effects, causing paradoxical increases in blood pressure, which may increase myocardial oxygen demand and infarct size. Therefore, beta - blockers should be avoided in the early stages of treatment while the drug's effects are still present, but started before discharge when the drug's effects have subsided.

Discontinuation of drug use is the primary goal of secondary prevention strategies, because recurrence of chest pain is high in patients who continue to use drugs. Therefore, implementing detoxification programs that incorporate smoking cessation, reduction of alcohol use, control of comorbidities such as hypertension and diabetes, and aggressive lipid-lowering treatment, along with rehabilitation cardiovascular function, are useful combined treatment strategies in these patients.

IV. CONCLUSION

Cocaine is one of illicit drugs and has a lot of negative effects towards the heart. It can cause more severe and acute cardiovascular complications such as acute myocardial infaction. Although the management of cocaine-abusing patients who present to an emergency room with acute chest pain remains difficult because of conflicting results regarding the incidence of myocardial infarction , we hope that this case will contribute to raising public awareness about cocaine use and its impact to our health. Also, appropriate addiction management in this patient population is considered important secondary prevention strategy that helps reduce the risk of future cardiovascular events.

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