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Left Main Coronary Artery Stenosis in a Patient with Chronic Cocaine Use: A Case Report

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Abstract: ***Cardiovascular disease is the leading cause of death in the world. Atherosclerosis of epicardial coronary arteries is the most characteristic lesion, however, involvement of the left main coronary artery (LMCA) is described in 4-6% of cases. Illicit drug use is a key modifiable cardiovascular risk factor. We present the case of a 41-year-old male with persistent precordial pain without troponin elevation and a positive stress test showing ischemia in the lower face. Coronary angiography reported high-risk left coronary artery ostium stenosis managed with coronary angioplasty with remission of symptoms.***

Keywords: Cocaine, Atherosclerosis, Coronary, Cardiovascular health

1. Introduction

Cardiovascular disease is the main cause of death around the world, representing more than 30%, and its incidence is increasing[1,2]. The main alteration is atherosclerosis of the epicardial coronary arteries, both in its chronic and acute presentation[3]. It is normally the culmination of a long and complex process where the occlusion of the coronary artery leads to myocardial ischemia and infarction[4]. On the other hand, arteriosclerotic lesion of the left main coronary artery (LMCA) is reported in 4-6% of patients undergoing coronary angiography and is often accompanied by involvement of the other epicardial vessels; advanced disease carries a greater risk of infarction associated with a greater number of unstable plaques [5].

Despite substantial improvements in the prevention and treatment of atherosclerotic cardiovascular disease, ASCVD for its acronym Atherosclerotic Cardiovascular Disease and according to cardiovascular risk estimation studies such as Framingham, ASCVD Risk Estimator and the SCORE system that have variables such as age, smoking, systolic blood pressure, and HDL cholesterol; the consumption of recreational substances as well as illicit drugs is one of the key modifiable risk factors [2,6]. The pathophysiological mechanisms behind this association include a general hyperadrenergic state with increased oxidative stress, endothelial dysfunction, and direct toxic effects[7]. The clinical case of a patient with a high-risk cardiovascular event associated with chronic cocaine use without other important antecedents is presented.

Clinical Case

A 41-year-old male with no history of chronic diseases, mild Covid-19 disease in January 2021 with outpatient management, smoking with a smoking index of 4, and inhalation of cocaine 3 times a week (equivalent to 2 grams) for 20 years.

He began his 1-month illness with angina pectoris and dyspnea, which was progressive with small efforts (brushing teeth, walking and talking), for which a chest x-ray was taken (Figure 1). In April 2021, he presented angina at rest plus vegetative, associated with inferior ischemia in the electrocardiogram (ECG), a stress test was indicated, which was early clinical and electrocardiographic positive (Figure 2).

An echocardiogram was performed, which showed akinesia of the inferior, middle, and basal wall, and inferior apical hypokinesia with an ejection fraction of 50%.

In May, he presented a new picture of angina at rest without elevation of ischemia markers (troponin I 0.05 ng/mL, myoglobin 30 ng/mL, CKMB 1 ng/mL, BNP 21.2 pg/mL, LDH 110 U/L, triglycerides 127 mg/dL, total cholesterol 100 mg/dL, HDL-C 31mg/dL) and ECG changes in the lower face. Coronary angiography was performed on May 18, documenting 90% ostial stenosis of the left main coronary artery with balanced dominance. A drug-eluting stent was implanted without complications (Figure 3 and 4). Discharging the patient at 48 hours asymptomatic.

2. Discussion

Cocaine abuse represents a considerable threat to the integrity of the cardiovascular system associated with the increased risk of developing hypertension, coronary spasms, arrhythmias, cardiomyopathy, atherosclerosis, and coronary artery disease [6,7]. According to Mahtta D, et al [6], the use of all forms of recreational substances is independently associated with a 1.5- to 2-fold increased likelihood of premature ASCVD, while illicit drugs represent a 3-fold increased risk. The fatal intravenous dose for adults is 1 gram for direct myocardial toxicity. The average dose of abuse by inhaled oral route is estimated between 8.7 mg and 14 mg, but it can be around 200 mg and the lethal dose of cocaine is between 0.5 grams and 1.5 grams (each line has from 15mg to 25mg)[8]. The mechanism behind cocaine-induced myocardial ischemia includes an increase in

myocardial oxygen demand as a result of increased inotropic and chronotropic effect, which is inappropriately accompanied by coronary vasoconstriction and a prothrombotic state[8]. Accelerated atherosclerosis is common in young chronic users, a process associated with a greater number of mast cells that suggests an increase in local inflammatory damage [9]. Qureshi et al [10]., in the study of the Third National Health and Nutrition Examination Survey (NHANES III and NHANES 2005-2006) indicates that the use of cocaine on more than 10 occasions in life was associated with a high prevalence of myocardial infarction in people aged 18-45 years in age adjusted models (OR = 4.60, 95% CI: 1.12-18.88, p value = 0.035). The association was attenuated in multivariate adjustment models (OR=3.84 95% CI: 0.98 to 15.07, p value=0.054) ; that is, 1 out of every 4 heart attacks in young people is related to the frequent use of cocaine [8,10,11].

Older studies have reported cocaine use rates among young myocardial infarction patients as high as 25%, while others suggest 10%[11]. For its part, the characteristics of patients with ischemic heart disease associated with cocaine are a consumption of 150 mg to 2 g, and serum concentrations of 0.01-1.02 mg/L, of chronic frequency, for recreation or first- time use and the time of presentation of the ischemic event is often within a few minutes up to 5-15 hours1[12].

Left main coronary artery disease portends a higher prognostic risk as a result of the large myocardial territory involved, ranging from 75% to 100% depending on the predominance of the left coronary circulation. Current clinical practice guidelines, including the American College of Cardiology, the American Heart Association, and the European Society of Cardiology, recommend revascularization for all patients with stenosis greater than 50%, regardless of symptomatic status or associated ischemia burden[13].

Therefore, in a scenario of chest pain in young patients with a history of illicit substance use, it should be evaluated with clinical history, physical examination and vital signs, followed by an electrocardiogram and cardiac troponins. Those who present electrocardiographic changes of ischemia or persistent symptoms should be referred for coronary angiography with possible angioplasty and stent implantation[9,14].

Regarding the study carried out by Arora S, et a1[15], in 3,735 patients on the impact of non-invasive and invasive management and revascularization for patients with non-ST elevation acute coronary syndrome associated with cocaine use, the latter was associated with a lower rate of adverse cardiovascular events, but a higher number of emergent revascularization in patients with noncompliance with medical management.

3. Conclusion

Although the worldwide epidemic of metabolic syndrome has been blamed for the rising incidence of premature ASCVD, the risk attributable to cocaine use may increase the occurrence of myocardial infarction, particularly among younger populations. Emphasizing the need for large studies to quantify its burden on public health.

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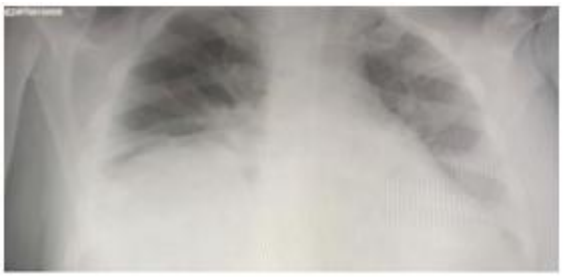

Figure 1: Chest X-ray with minimal bilateral pleural effusion, flow cephalization, right diaphragmatic elevation and peripheral clouding.

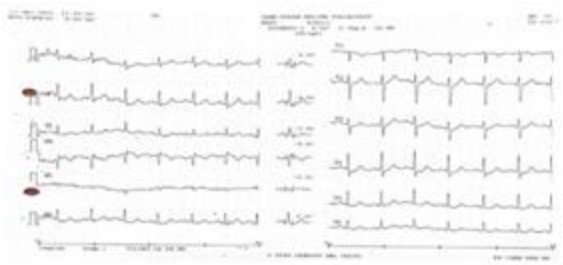

Figure 2: Bruce protocol stress test suspended at 2.25 min of the 2nd stage due to change in register and angina: negative ST segment elevation in DII, AVF and V3-V5, plus ST segment elevation in AVR.

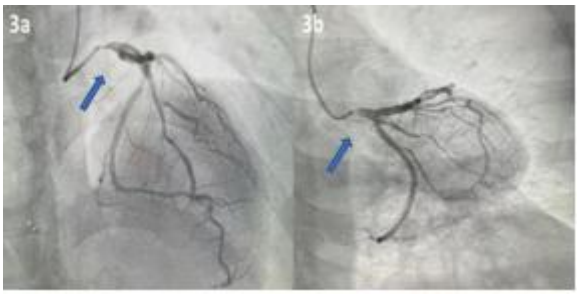

Figure 3: a-b). Coronary angiography with critical stenosis of 90% of the ostium of the left main coronary artery (arrows), TIMI III anterograde flow. AVF and V3-V5, plus ST segment elevation in AVR

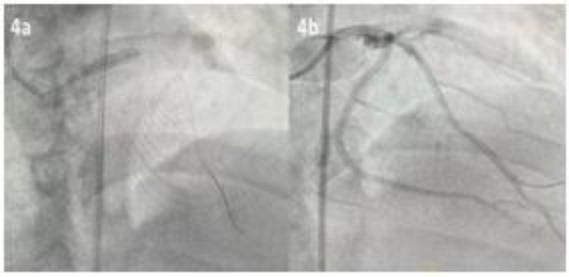

Figure 4: a-b). A 4.0 x 15 mm medicated stent was placed towards the left anterior descending coronary artery, confirming good position.

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