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Large Intra-Coronary Thrombus Induced by Cocaine Use

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Abstract **Case Report**

Cocaine is a highly addictive substance and represents a significant public health concern. The incidence of cocaine-associated myocardial infarction is low (4.7%). Coronary spasm is the presumed etiology as most patients have normal coronary arteries. We report a case of a 31-years old patient with known history of cocaine abuse that presented an acute thrombosis of the left anterior descending by a large intra-coronary thrombus. There are no specific guidelines about treating thrombus formation in coronary arteries due to cocaine use. Thereby, an optimal anti-thrombotic alone remains an effective alternative, As reported in our case, with a complete thrombus resolution.

Keywords: Cocaine associated myocardial infarction; CAMI; Cocaine; Coronary thrombosis; Stent thrombosis, myocardial infarction.

INTRODUCTION

Cocaine is a powerful addictive stimulating drug made from the leaves of the coca plant. It is estimated that >14 million people regularly consume cocaine worldwide. The prevalence of cocaine use in Morocco is between 1.2-2% [1].

It is a pro-thrombotic and vasoconstrictive substance responsible for cocaine-associated myocardial infarction (CAMI). We present a case of a large intra-coronary thrombus with normal coronary arteries treated by optimal antithrombotic therapy alone. In the absence of guidelines for the management of this phenomenon, we reviewed medical literature for an evidence-based therapeutic strategy.

CASE PRESENTATION

A 31-years old woman with a history of substance abuse (cannabis and occasional cocaine use) presented with a crushing retrosternal chest pain starting 4h prior to admission, radiating to the left arm. Relevant past medical history of a borderline personality disorder under venlafaxine and no known family history of coronary artery disease. She had used cocaine one day before. Upon arrival, the physical examination was unremarkable. Admission electrocardiogram showed an extensive anterior ST-elevation with ST-depression in inferior leads without Q wave (Figure 1).

The patient was taken emergently to the catheterization lab after receiving a loading dose of aspirin 300mg, clopidogrel 600mg and enoxaparin0.6 mg. Coronary angiogram found no evidence of atherosclerosis but a large coronary thrombus in the proximal portion of left anterior descending artery (LAD), obstructing flow (TIMI0) extending to the proximal circumflex artery and ramus intermedius (RI) with distal occlusion (Figure 2A). Thrombus aspiration could not be attempted to prevent further clot embolization downstream. A decision was taken to start tirofiban for 48hours on top of maintaining anticoagulation by enoxaparin.

The patient was sent to cardiac care unit for further medical optimization with the introduction of Betablockers and statin. Post-intervention ECG showed a resolution of ST elevation. Transthoracic echocardiogram demonstrated evidence of extensive hypokinesis of anterior, anteroseptal and anterolateral walls with a mildly reduced ejection fraction (48%). Lab exams found an iron deficiency anemia, an hyperfibrinogenemia, no abnormalities of serum lipids, or blood glucose.

A control angiography was performed 48H later, confirming a restored flow but persistent thrombus in LAD (Figure 2B). During hospitalization, the patient had an uneventful course. At discharge she was prescribed aspirin, clopidogrel, enoxaparin 0.6 Bid for 10

days, carvedilol 6,25 Bid, rosuvastatin, and iron. After one month follow-up, a repeat coronary angiogram revealed complete resolution of LAD thrombus under anti-platelets therapy only (Figure 3).

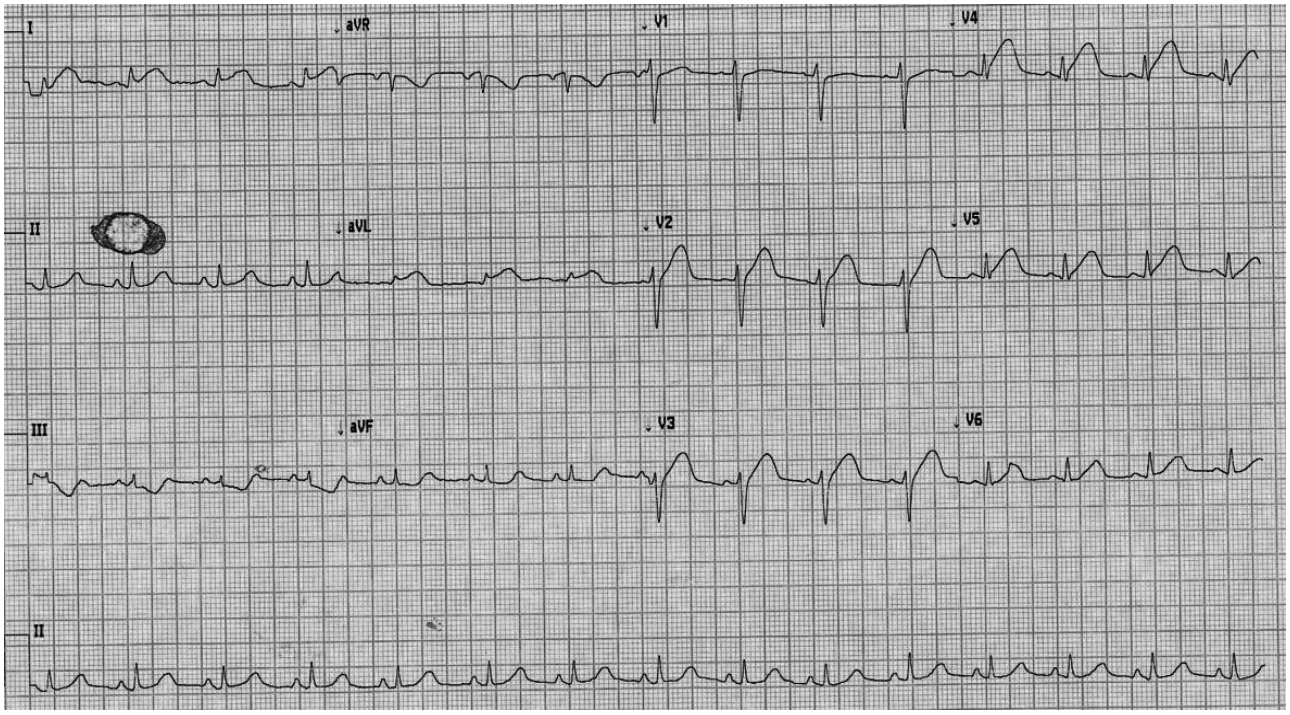

Figure 1: Admission ECG showing an extended anterior ST elevation

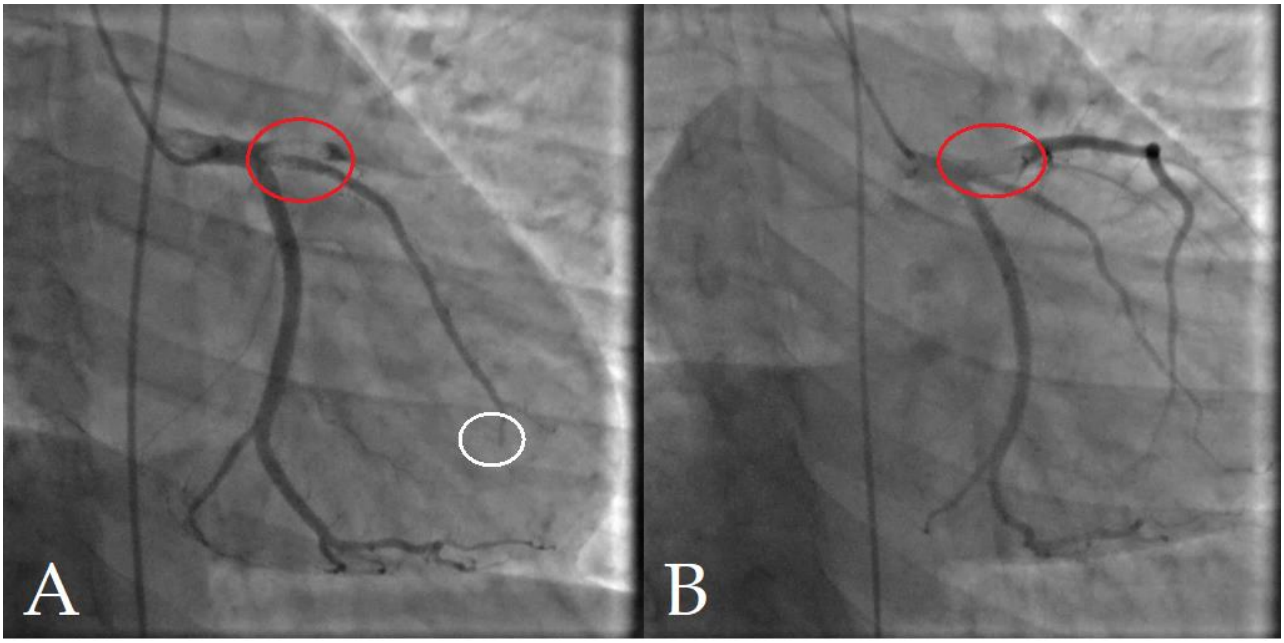

Figure 2: A) Initial angiography showing a large LAD thrombus (red circle) extended to circumflex and ramus intermedius arteries with occlusion of distal RI (white circle); B) 48h Control angiography revealing a restored flow but persistent thrombus (red circle)

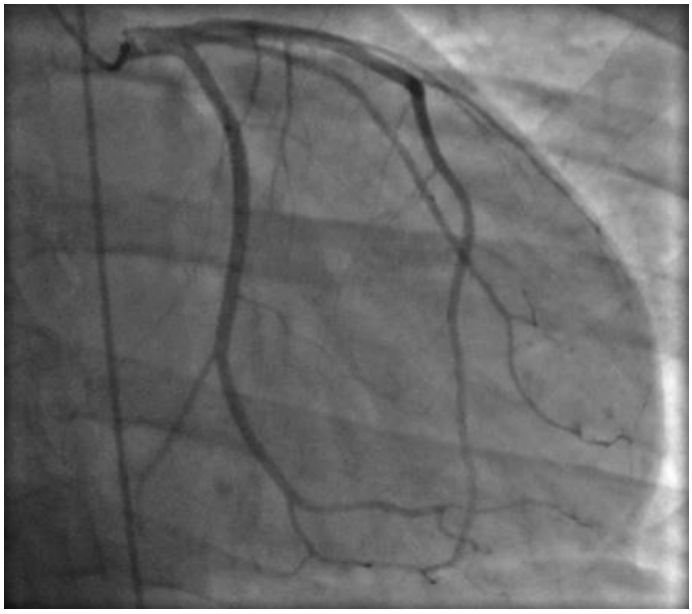

Figure 3: 1 month control angiography with complete resolution of LAD thrombus

DISCUSSION

The overall CAMI incidence is low (4.7%) with a lower rate (1.1%) in patients with no or 1 risk factor. The risk is 24-times higher within the first hour and can persist even days after exposure due to cocaine metabolites. The majority of patients presenting with cocaine-associated chest pain are young, male, and regular tobacco smokers, and many report previous occurrences of chest pain in association with cocaine use [2].

The pathophysiology of CAMI is multifactorial. Cocaine blocks the uptake of monoamines (dopamine, norepinephrine, epinephrine) leading to an excessive amount of these substances at postsynaptic receptor sites. The cardiovascular effect is an increased heart rate, blood pressure and myocardial contractility, thus increasing myocardial oxygen demand. At the same time, supply is decreased by an alpha-adrenergic mediated coronary vasoconstriction in epicardial arteries. A more potent effect is present in diseased segments [3].

Patients with normal coronary arteries may present with myocardial infarction in the setting of cocaine use, likely due to subsequent thrombosis, even in the absence of coronary artery spasm [4]. Cocaine and its metabolites lead to excessive platelet activation resulting in arterial thrombosis in the absence of atherosclerosis. It acts as an endothelial agonist, promoting platelet-mediated thrombosis with a significant increase in VonWillebrand factor (VWF) concentration up to 40%. The vasoconstriction results in elevated shear-stress promoting VWF secretion and flow reduction, thereby causing thrombus formation. Cocaine also induces endothelial dysfunction by endothelin production, reduction of nitric oxide production and tissue factor. This pro-thrombotic state results in the thrombus formation, playing a major role in CAMI [5].

In our case, cannabis cannot be responsible for the intra-coronary thrombus. Recent studies found no association between synthetic cannabinoids or tetrahydrocannabinol and thrombus formation, although it may be involved in atherogenesis and plaque rupture [6].

In our review of the literature, CAMI management lacks proper evidence-based guidelines. Hence, anti-thrombotic therapy, percutaneous coronary intervention (PCI), even urgent coronary artery bypass grafting have been used on individual clinical cases [7]. When addressing a ST-elevation myocardial infarction in the general population, PCI is paramount to treat artery thrombosis. Thus, stent thrombosis occurred 10-times more in cocaine users compared to the normal population (7.6% vs 0.6%) [8]. This may also be explained by their poor medical compliance, increased thrombogenicity and repeated cocaine use. Fibrinolytics could be used in selected patients, but it is associated with major bleeding complications, including intracranial hemorrhage and lacks a documented efficacy [9].

Since CAMI is more related to thrombus formation and vasospasm rather than atherosclerotic plaque rupture, perhaps the optimal strategy should be limited to aspiration thrombectomy, and antithrombotic therapy. A well conducted anti-thrombotic treatment could be sufficient in managing these situations, as reported in our case.

Initial and long-term management of these patients is challenging. The systematic use of beta-blockers is prohibited in CAMI as highlighted in a joint scientific statement from AHA/ACC, particularly in

patients demonstrating signs of acute cocaine intoxication. It may be reasonable only in patients who received coronary vasodilators (nitroglycerine , calcium channel blockers) [10]. Due to the theoretical risk of unopposed alpha-adrenergic stimulation resulting in excessive vasoconstriction.

The continuation of antithrombotic treatment for at least 12 months is to be considered as recommended in all acute coronary syndromes. But Cocaine use cessation is paramount, and addiction control remains a must in these patients.

CONCLUSIONS

Young patients presenting myocardial infarction should be screened for substance use. As CAMI management remains challenging in the absence of a clear consensus. Optimal antithrombotic treatment or direct stenting must be assessed individually depending on the patient's cardiovascular risk and coronary artery anatomy. This case highlights that medical therapy alone provides an effective alternative for the resolution of the occlusive thrombus in CAMI.

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List of abbreviations:
CAMI: Cocaine-associated myocardial infarction
LAD: Left anterior descending artery
RI: Ramus intermedius
VWF: Von Willebrand factor
PCI: Percutaneous coronary intervention

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