Case Report

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Simultaneous Thrombosis of the Left Anterior Descending Artery and the Right Coronary Artery in a 34-Year-Old Crystal Methamphetamine Abuser

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This case report underscores that crystal methamphetamine abuse is an important cause of multivessel coronary thrombosis and raises doubts about the therapeutic options. The patient was a 34-year-old smoker and crystal methamphetamine abuser with no significant medical history, who presented with retrosternal chest pain associated with cold sweats. Twelve-lead electrocardiogram revealed diffuse ST-segment elevation in I, II, AVL, AVF, and V 2–6 leads. He underwent urgent coronary angiography and it showed Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow in coronary arteries and presence of a thrombus in the left anterior descending artery (LAD) and the right coronary artery (RCA). The patient underwent medical therapy with antiplatelet agents and anticoagulants. Repeat coronary angiography after three months of dual therapy with warfarin and aspirin did not show any thrombus or any significant lesion in the RCA and the LAD having TIMI grade 3 flow. **(Korean Circ J 2015;45(2):158-160)**

KEY WORDS: Methamphetamine; Coronary thrombosis; Angiography.

Introduction

The use of methamphetamine is common; and in many countries, it is the most common drug that is abused. Hence, it is important to recognize the adverse effects of methamphetamine on the cardiovascular system.[1)2)]

Methamphetamine use may aggravate the underlying cardiac pathology, such as coronary atherosclerosis or cardiomyopathy, and thus can escalate the risk of an acute episode such as myocardial infarction or even unexpected cardiac death. Long-term methamphetamine users are at increased risk of cardiovascular damage, such as premature, accelerated coronary artery disease. Methamphetamine toxicity per se is more likely to have a fatal outcome with chronic use.[1)2)]

In spite of limited data on the distinct mechanism of methamphetamine-induced coronary thrombosis, acute and chronic cardiovascular complications of cocaine and crack, which is the most addictive type of cocaine, have been well demonstrated. Increased myocardial oxygen demand, coronary vasoconstriction, and coronary thrombosis are believed to be the three major mechanisms of cocaine- and crack-related myocardial ischemia and infarction, which could be helpful in clarifying the role of methamphetamine in coronary artery thrombosis.[3)]

Case

A 34-year-old smoker and crystal methamphetamine abuser with no significant medical history, presented to the emergency department with retrosternal chest pain associated with cold sweats 12 hours after intranasal crystal methamphetamine use. His initial blood pressure, pulse rate, and respiratory rate were 135/85 mm Hg,

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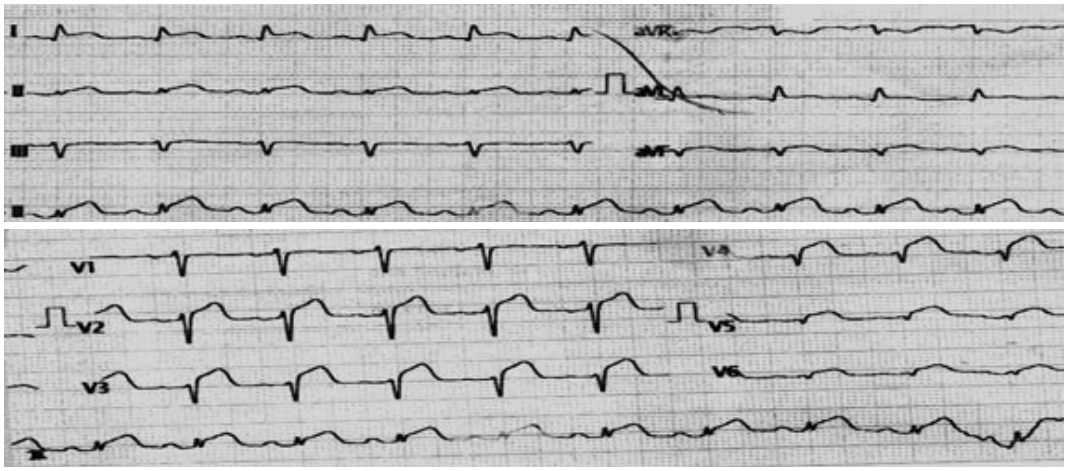

Fig. 1. Twelve-lead electrocardiogram revealed ST-segment elevation in I, II, AVL, AVF, and V 2–6 leads.

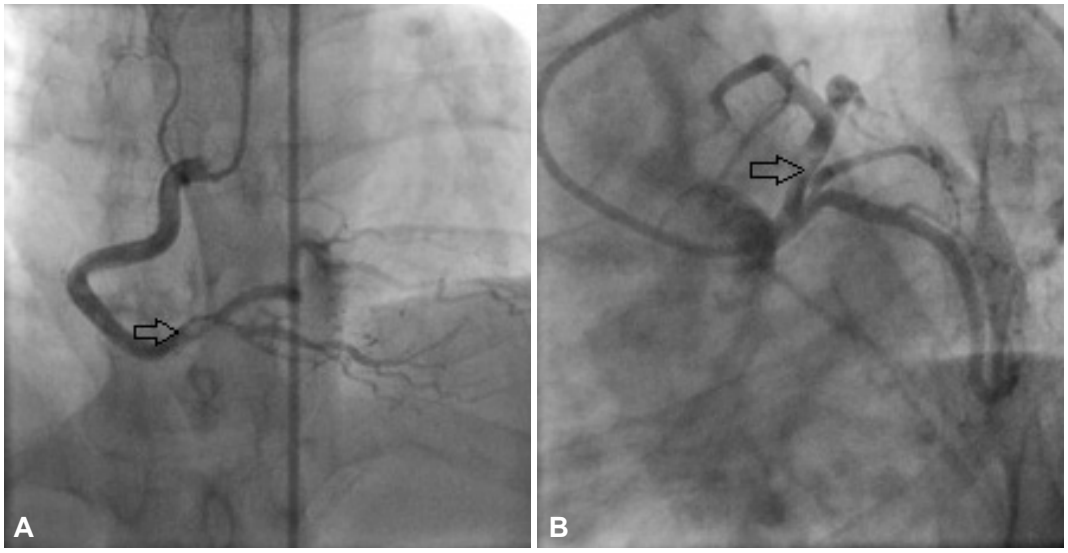

Fig. 2. Initial cardiac catheterization showed a thrombus in the distal portion of the right coronary artery (arrow) just before bifurcation (A) just before bifurcation and a large thrombus in the proximal portion of the left anterior descending artery (arrow) (B) having Thrombolysis in Myocardial Infarction grade 3 flow

92 bpm, and 22 breaths per min, respectively. Cardiac and respiratory examination was unremarkable. Twelve-lead electrocardiogram revealed ST-segment elevation in I, II, AVL, AVF, and V 2–6 leads (Fig. 1). In addition, elevated cardiac markers (myocardial bound creatine kinase/creatine kinase, 398/964 U/L and troponin-I level, 33 ng/mL) were noted.

Patient was transferred to our cath lab for urgent cardiac catheterization. Cardiac catheterization showed a large thrombus in the proximal portion of the left anterior descending artery (LAD) having Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow and another thrombus in the distal portion of the right coronary artery (RCA) just before bifurcation (Fig. 2). The patient had 10% of the initial pain; two boluses of intracoronary eptifibatide (10 minutes apart) were immediately injected and continued intravenously (2 microgram/kg/min) for 48 hours along with intravenous heparin, oral aspirin (81 mg daily) and clopidogrel (75 mg daily) due to the TIMI grade 3 flow in both the LAD and the RCA.

Also, the echocardiography findings revealed an ejection fraction of 45%, apex and mid anteroseptal wall akinesia, and a large apical clot (1.3×1.4 cm).

The laboratory tests including factor V Leiden and prothrombin gene, protein C, protein S, and antithrombin III, antiphospholipid antibodies, and his medical and family history were not indicative of a hypercoagulable state.

After two weeks, the patient was discharged on dual therapy (ASA 81 mg daily and warfarin 5 mg daily) to achieve a target international normalized ratio of 2–3. Clopidogrel was discontinued due to the recent history of GI bleeding.

Repeat cardiac catheterization after three months of dual therapy with warfarin and aspirin did not show any thrombus or any significant

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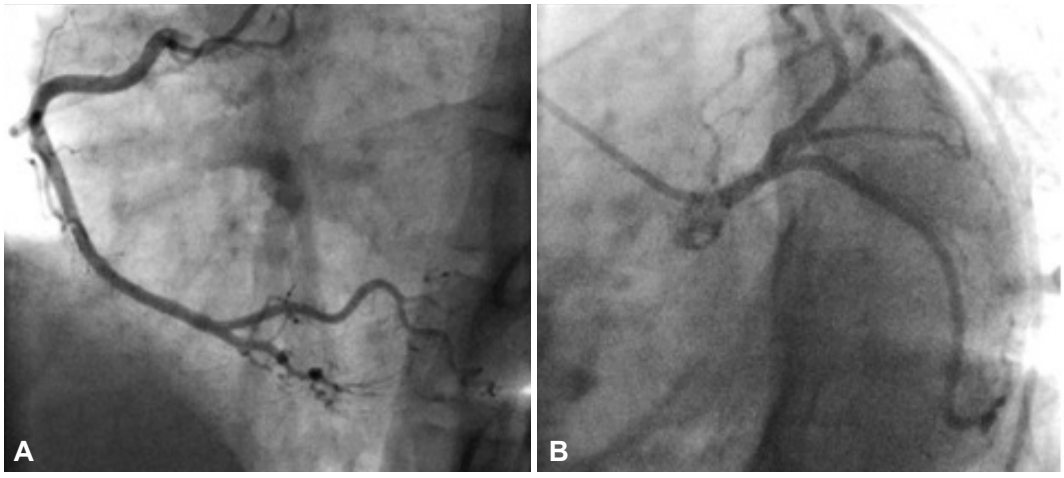

Fig. 3. Repeat cardiac catheterization after three months of dual therapy with warfarin and aspirin did not show any thrombus or any significant lesion in the right coronary artery (A) and the left anterior descending artery (B) having Thrombolysis in Myocardial Infarction grade 3 flow.

lesion in the RCA and the LAD having TIMI grade 3 flow (Fig. 3). Moreover, repeated echocardiography did not demonstrate any apical clot. The patient was symptom-free; warfarin was discontinued and he was planned to undergo a routine cardiac follow-up.

Discussion

Although the accurate mechanisms of methamphetamine-induced cardiac damage have not been described, they are assumed to be similar to those involved in cocaine-induced cardiac damage.

Since methamphetamine- and cocaine-associated coronary thrombosis may not be associated with severe occlusive or flow-limiting atherosclerotic heart disease, perhaps the most favorable therapeutic strategy would be to focus on the privileged use of aspiration thrombectomy, balloon angioplasty, and/or medical therapy as well as on the use of anti-coagulants and platelet inhibitors.

It should be noted that there is little experience of fibrinolytic therapy in the setting of crystal methamphetamine- and cocaine-associated myocardial infarction, and it should be regarded as the last option. In addition, fibrinolytic agents can cause major bleeding complications.[4-9)]

Our patient had not undergone thrombolytic therapy or percutaneous coronary intervention (PCI) because of the acceptable TIMI flow grade and the potential risk of the no-reflow phenomenon.

This case report underscores that crystal methamphetamine abuse is an important cause of multivessel coronary thrombosis, and it is a rare condition due to simultaneous thrombosis of two major coronary arteries and it raises doubts about the medical therapy with antiplatelet agents and anticoagulants as effective therapeutic options for diminution of the thrombotic mass in crystal methamphetamine users presenting with acute ST-segment elevation myocardial infarction. Moreover, diffuse ST-segment elevation could be suggestive of other diagnoses including acute pericarditis.

However, emergency PCI and stenting may be considered in this setting, particularly if there is hemodynamic compromise or persistent chest pain. Also, stent thrombosis as a notable complication and its high incidence in crack and crystal methamphetamine abusers should always be considered.

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