A young patient with acute myocardial infarction due to bonsai treated with glycoprotein IIbIIIa inhibitor

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Introduction

Acute myocardial infarction (AMI) often occurs after an atherogenic plaque rupture, but substance use is one of the rare causes of myocardial infarction (MI). Recently, substance abuse such as cannabis and cocaine has significantly increased in Europe and our country. A few cases of MI related to the use of bonsai, a type of synthetic cannabinoid, have been described in the literature. However, the management and treatment of these cases is still controversial. Therefore, we aimed to present the case of a patient with an anterior MI due to bonsai use who received medical treatment and was followed-up.

Case Report

A 34-year-old male patient was admitted to our emergency department with a typical chest pain that started 3 hours ago. The patient had no history of chronic disease. Upon admission, the patient was observed to be agitated and tachypneic, and no other remarkable findings were observed in the physical examination. He had a smoking history of 15 packs/year. In addition, he was found to have a history of intense bonsai use by inhalation approximately 24 hours before arriving at the hospital. In the electrocardiogram (ECG), a widespread ST segment elevation in the anterior leads was detected (Fig. 1). The patient was taken to the angiography laboratory for primary coronary intervention (PCI). Before the procedure, 300 mg of acetylsalicylic acid (ASA) and 180 mg of ticagrelor were administered orally to the patient. In the coronary angiogram (CAG), intense thrombus images were observed in the proximal and distal left anterior descending artery (LAD) (Fig. 2, Video 1). The circumflex artery (CxA) and right coronary artery (RCA) were completely normal. Treatment with glycoprotein IIbIIIa inhibitor (tirofiban) infusion was planned, considering that the thrombus did not show a specific localization and was widespread, and the patient’s complaints regressed. After an intracoronary tirofiban bolus was administered, the patient was taken to the coronary intensive care unit to continue the 24-hour infusion. Ejection fraction (EF) was 60% and the valve structure and functions were normal in the echocardiography (Video 2a, 2b). In the patient’s follow-up, regression of the ST segment elevation in the anterior leads on the ECG was observed. In the control coronary angiogram performed approximately 36 hours later, the thrombus was observed to have regressed significantly (Fig. 3, Video 3). During his hospitalization, low molecular weight heparin (LMWH) was administered in addition to dual antiplatelet therapy. The patient was discharged in good health on the 4th day of hospitalization.

Discussion

In this case, we present a patient who had an anterior MI after bonsai use and was treated medically.

AMI is one of the most important causes of mortality and morbidity worldwide. Underlying plaque rupture is frequently involved in the pathology of AMI. However, causes such as coronary trauma, vasculitis, coronary embolism, drug or sub-
Coronary artery stenosis due to angiopathy and plaque rupture, slow coronary blood flow or thrombus formation due to the prothrombotic effect, coronary artery dissection due to the hemodynamic effect, increased sympathetic activity, endothelial dysfunction, and coronary vasospasm constitute the important part of these hypotheses (16).

Currently, primary PCI therapy in patients with cocaine-associated AMI is more frequently used than fibrinolytic and other medical therapies. Fibrinolytic therapy should be considered when primary PCI therapy is not possible (17). The treatment of bonsai-induced MI is controversial. Although the PCI method was used in the treatment of two reported cases, thrombus aspiration or medical therapy is also considered as a treatment option because there is no underlying atherogenic plaque in these patients. In our case, because it was thought that the use of bonsai could cause intense intracoronary thrombus and related MI, medical treatment was primarily performed.

Antiplatelet and antithrombin agents seem reasonable, even if primary PCI or to a lesser extent thrombolytic therapy was used in the treatment of AMI due to cocaine use. There are no studies about the role of aspirin, gIIb/IIIa inhibitor, clopidogrel, and LMWH in the treatment of these patients. However, few cases have been reported showing that tirofiban therapy may be beneficial in these patients. In a case presented by Frangogiannis et al. (18), only tirofiban was administered to a patient with cocaine-associated AMI after heparin treatment, and the control CAG performed seven days later found that the thrombus regressed completely. In another case of cocaine-related AMI reported by Doshi et al. (19), resolution of the thrombus was achieved with tirofiban and bivalirudine after CAG.

Conclusion

AMI associated with bonsai and similar substances has increased in recent years due to the easy availability and cheap price of these substances. It should be considered in the differential diagnosis of patients with chest pain and substance abuse, as substance use-related AMI may occur especially in young individuals. The treatment and management of AMI in these patients is still controversial. Comprehensive studies are needed to determine the effect of bonsai use on the cardiovascular system.

Informed consent: A written informed consent was acquired from the patient.

Video 1. First coronary angiography showed a thrombus in the proximal and distal LAD

Video 2a. Transthoracic echocardiography (apical 4-chamber view) showed a normal left ventricular ejection fraction

Video 2b. Transthoracic echocardiography (apical 2-chamber view) showed a normal left ventricular ejection fraction

Video 3. Control coronary angiography showed a reduction in the proximal thrombus and no thrombus in distal LAD
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