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 recived 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-

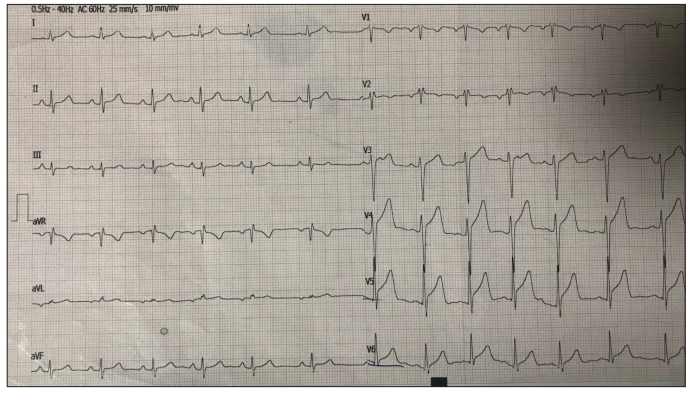

Figure 1. Electrocardiography perfomed on admission in the emergency department

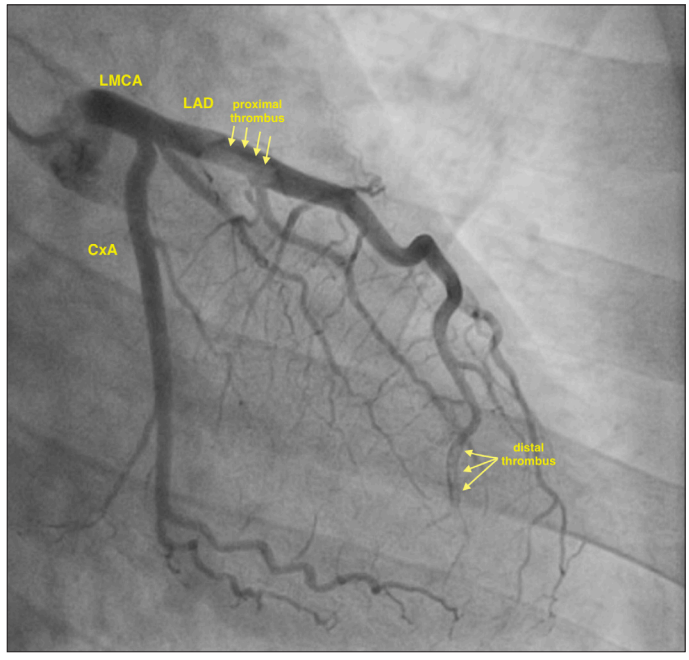

Figure 2. First coronary angiography showed a thrombus in the proximal and distal LAD

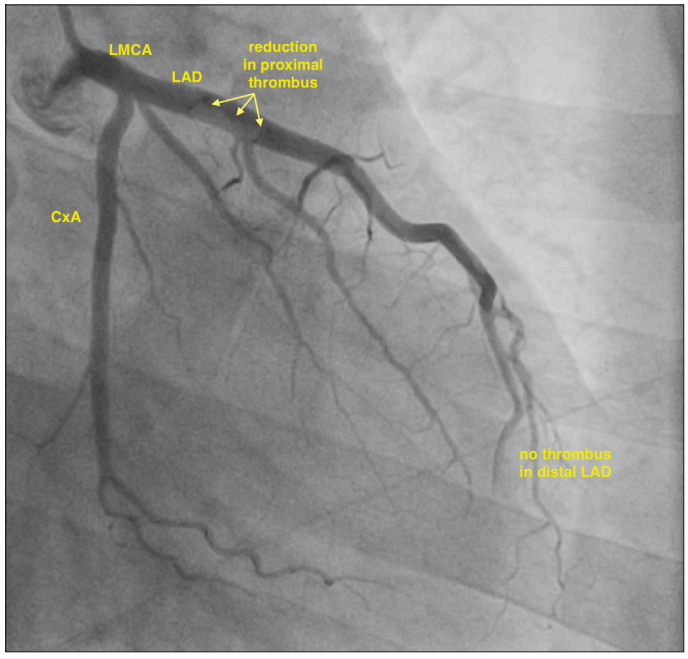

Figure 3. Control coronary angiography showed a reduction in the proximal thrombus and no thrombus in the distal LAD

stance use, toxins, sepsis, coronary artery anomaly, and aortic dissection can also lead to AMI (1). Therefore, substance use should be considered in patients presenting with MI, especially those without cardiovascular risk factors.

The illegal use of bonsai and other synthetic substances is increasing (2). Although the psychoactive properties of these substances have been emphasized to date, it has been shown that these substances increase the number of emergency department admissions for cardiac reasons such as MI, sudden cardiac death, and coronary vasospasm (3-5). Myocardial infarction (MI) and coronary pathologies can be observed after cocaine use (6, 7). However, there are few reports on MI cases due to cannabinoids. Currently, the reported cases were associated with the use of marijuana, a natural cannabinoid (8-10).

In a study conducted by Hollander et al. (11), AMI was found in 14 of 246 patients who presented to the emergency department for chest pain that started after cocaine use. In a case series of 10 patients published by Sharma et al. (12), an invasive treatment (PCI and thrombectomy) was performed in all patients with cocaine-related myocardial infarction and the outcomes were favorable. In the review study published by Patel et al. (13), approximately half of 62 patients with AMI after using marijuana, a natural cannabinoid, were followed-up medically. Thrombectomy and primary PCI treatment was performed in the remaining patients (13). In a case report published by Ayhan et al. (14), primary PCI treatment was performed on a patient with AMI due to bonsai use and success was achieved. In another bonsai-related AMI case published by Köklü et al. (15), in addition to the primary PCI treatment, tirofiban, which is a g2b3a inh, was administered intravenously, and the treatment was successful.

The mechanism of cannabinoid-related acute coronary syndrome is still unclear, although different hypotheses have been proposed. 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 observed in the control CAG by administering LMWH, tirofiban, and bivaluridine 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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References

1. Mechanic OJ, Grossman SA. Acute Myocardial Infarction. 2020 Nov 20. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020.
2. Hall W, Degenhardt L. Prevalence and correlates of cannabis use in developed and developing countries. Curr Opin Psychiatry 2007; 20: 393-7. [Crossref]
3. Evren C. Türkiye Psikiyatri Derneği, Sentetik Esrar Türevleri (Bonzai) ile ilgili Basın Açıklaması 04.09.2014. Bağımlılık Dergisi 2014; 15: 57-9. [Article in Turkish]
4. Hurst D, Loeffler G, McLay R. Psychosis associated with synthetic cannabinoid agonists: a case series. Am J Psychiatry 2011; 168: 1119. [Crossref]
5. Every-Palmer S. Warning: legal synthetic cannabinoid-receptor agonists such as JWH-018 may precipitate psychosis in vulnerable individuals. Addiction 2010; 105: 1859-60. [Crossref]
6. Ifedili I, Bob-Manuel T, Kadire SR, Heard B, John LA, Zambetti B, et al. Cocaine Positivity in ST-Elevation Myocardial Infarction: A True or False Association. Perm J 2019; 23: 18-048. [Crossref]
7. Finkel JB, Marhefka GD. Rethinking cocaine-associated chest pain and acute coronary syndromes. Mayo Clin Proc 2011; 86: 1198-207. [Crossref]
8. Jouanjus E, Lapeyre-Mestre M, Micallef J; French Association of the Regional Abuse and Dependence Monitoring Centres (CEIP-A) Working Group on Cannabis Complications*. Cannabis use: signal of increasing risk of serious cardiovascular disorders. J Am Heart Assoc 2014; 3: e000638. [Crossref]
9. Casier I, Vanduynhoven P, Haine S, Vrints C, Jorens PG. Is recent cannabis use associated with acute coronary syndromes? An illustrative case series. Acta Cardiol 2014; 69: 131-6. [Crossref]
10. Draz EI, Oreby MM, Elsheikh EA, Khedr LA, Atlam SA. Marijuana use in acute coronary syndromes. Am J Drug Alcohol Abuse 2017; 43: 576-82. [Crossref]
11. Hollander JE, Hoffman RS, Gennis P, Fairweather P, DiSano MJ, Schumb DA, et al. Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group. Acad Emerg Med 1994; 1: 330-9. [Crossref]
12. Sharma AK, Hamwi SM, Garg N, Castagna MT, Suddath W, Ellahham S, et al. Percutaneous interventions in patients with cocaine-associated myocardial infarction: a case series and review. Catheter Cardiovasc Interv 2002; 56: 346-52. [Crossref]
13. Patel RS, Kamil SH, Bachu R, Adikey A, Ravat V, Kaur M, et al. Marijuana use and acute myocardial infarction: A systematic review of published cases in the literature. Trends Cardiovasc Med 2020; 30: 298-307. [Crossref]
14. Ayhan H, Aslan AN, Süygün H, Durmaz T. Bonsai induced acute myocardial infarction. Turk Kardiyol Dern Ars 2014; 42: 560-3. [Article in Turkish] [Crossref]
15. Köklü E, Yüksel İÖ, Bayar N, Üreyen ÇM, Arslan Ş. A new cause of silent myocardial infarction: Bonsai. Anatol J Cardiol 2015; 15: 69-70. [Crossref]
16. Goyal H, Awad HH, Ghali JK. Role of cannabis in cardiovascular disorders. J Thorac Dis 2017; 9: 2079-92. [Crossref]
17. McCord J, Jneid H, Hollander JE, de Lemos JA, Cercek B, Hsue P, et al.; American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Management of cocaine-associated chest pain and myocardial infarction: a scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. Circulation 2008; 117: 1897-907. [Crossref]
18. Frangogiannis NG, Farmer JA, Lakkis NM. Tirofiban for cocaine-induced coronary artery thrombosis: a novel therapeutic approach. Circulation 1999; 100: 1939. [Crossref]
19. Doshi SN, Marmur JD. Resolution of intracoronary thrombus with direct thrombin inhibition in a cocaine abuser. Heart 2004; 90: 501. [Crossref]

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