Acute Thrombosis of Double Major Coronary Arteries Associated with Amphetamine Abuse

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Drug-induced acute myocardial infarction is not a common phenomenon. The underlying mechanism in the majority of such patients has been related to coronary spasm, including in those with amphetamine abuse, in whom the coronary arteriogram was always found normal. We report a 30-year-old male amphetamine abuser with acute myocardial infarction owing to acute thrombosis of the left anterior descending coronary artery and left circumflex coronary artery. We postulate a relationship between the use of amphetamine and occurrence of acute thrombosis of multiple major coronary arteries.

Key Words: Amphetamine • Coronary • Thrombosis

INTRODUCTION

Amphetamines have been gaining popularity as a recreational drug worldwide over the past few decades. Acute myocardial infarction (AMI) owing to amphetamine abuse often occurs in young adults, in whom coronary spasm is thought to be the underlying mechanism. To our knowledge, there is no published registry of amphetamine-induced AMI with multiple coronary thrombosis. To highlight the occurrence of thrombosis in amphetamine-induced AMI, we describe an amphetamine abuser presenting with AMI owing to multivessel coronary thrombosis. The patient failed to survive despite early percutaneous coronary intervention (PCI).

CASE REPORT

A 30-year-old man was being arrested by policemen after intruding into a private apartment when acute chest pain occurred. He was brought by policemen to our emergency unit 2 hours after the onset of acute chest pain, which radiated to the back and was accompanied by nausea and vomiting but no shortness of breath. The patient had no history of hypertension, hyperlipidemia, diabetes mellitus, atrial fibrillation, or family history of coronary artery disease. He had smoked 2 packs of cigarettes daily for more than 10 years. Several hours before the onset of chest pain, the patient had “snorted some amphetamine.” He also admitted frequent use of amphetamine for more than 2 years.

On arrival, blood pressure was 96/69 mmHg, heart rate was 107 beats per minute, respirations were 20 breaths per minute, and temperature was 36.1 °C, with an oxygen saturation of 100% on room air. His lungs were clear, and his abdominal examination revealed nothing particular. An immediate electrocardiogram (ECG) (Figure 1A) showed sinus rhythm with diffuse ST elevation in the precordial leads (V2 through V6) and limb leads (I, II, III, and aVF) as well as the development of Q waves in leads V1~V5. Laboratory data showed total creatine kinase level of 2157 IU/L, creatine kinase-MB level of 82 U/L, and troponin-I level of 11.70 ng/ml. No thrombocytosis (platelet count: 368,000/uL) was found. A urine drug screen was positive for amphetamines but negative for cocaine. Bedside cardiac ultrasonography showed hypokinesia of the inferior, posterior and ante-
rior wall of the left ventricle. Under the impression of ST elevation AMI with cardiogenic shock, the patient was given aspirin and subcutaneous low-molecular-weight heparin. Glycoprotein IIbIIIa antagonist was not administered then because we had failed to gain approval from the patient himself.

Although chest pain had recurred, unfortunately, primary PCI could only be performed 8 hours after the onset of initial chest pain after obtaining permission from the prosecuting attorney. Coronary angiography revealed a thrombus-like filling defect (Figure 2A, bold arrow) in the mid-portion of the left anterior descending coronary artery (LAD) as well as a 90% stenosis in the proximal LAD, with only TIMI grade II flow (Figure 2A, arrowhead). The left circumflex coronary artery (LCx) was also totally occluded at the distal-portion (Figure 2A, arrow). Neither significant fixed stenotic lesion nor thrombus were found in the right coronary artery (Figure 2B). The LCx and LAD were considered to be the infarct-related arteries, due to their evident thrombi with sluggish flow.

A thrombectomy device (Thrombuster, Kaneka) was used sequentially through a 7-French guiding catheter, and gained a TIMI grade 3 flow in both vessels. After large amount of thrombus was aspirated, a $2.75 \times 32$-mm and a $3.0 \times 28$-mm bare-metal stents were implanted to the distal-portion of the LCx and the proximal LAD, respectively. A TIMI grade 3 antegrade flow and TIMI frame count of 35 counts were achieved without residual stenosis in both LCx (Figure 2C) and LAD (Figure 2D). The chest pain was attenuated and ECG showed resolution of ST eleva-
tions (Figure 1B). During the course of PCI, reperfusion-induced arrhythmias such as VT or VF had never developed, but persistent hypotension with severe metabolic acidosis leading to frequent asystole occurred and was immediately restored after cardiac massages.

Throughout PCI, the patient was actually handcuffed to the table. He had been very uncooperative and was struggling hard at all times trying to free of the handcuffs so that he could escape. As a result, intraaortic balloon pump and extracorporeal membrane oxygenation devices were not considered in this patient for safety reasons. Unfortunately, despite successful primary PCI and aggressive inotropic agents, the patient died of cardiogenic shock in the coronary care unit.

**DISCUSSION**

AMI at a young age is unusual and is mainly owing to congenital coronary anomaly, coagulopathy, premature atherosclerosis or coronary artery spasm or is drug-induced. AMI caused by thrombus-filled coronary vessels after the use of amphetamine is rare as compared with AMI caused by other illegal drugs (for example, cocaine). In our case, we clearly documented the existence of thrombus in multiple coronary arteries during emergency coronary angiography. AMI with multiple acute obstruction of major coronary vessels is a rare clinical and angiographic finding. To our knowledge, however, this is the first reported case of multivessel acute thrombosis after amphetamine-induced myocardial infarction. There were few other cases reported in detail in the literature; one was secondary to cocaine use; others usually occurred in patients with essential thrombocythemia.

Several mechanisms have been proposed for amphetamine-induced myocardial ischemia though induction of coronary arterial spasm was the likely mechanism of amphetamine-related acute MI. Previous studies have shown that cocaine can stimulate the release of catecholamines from presynaptic nerve terminals, and block the reuptake of these catecholamines, resulting in a hyperadrenergic state. Also, cocaine decreases concentrations of protein C and antithrombin III, activate platelets, and potentiate thromboxane production, thereby facilitating thrombotic coronary occlusion. However, the contribution to thrombus formation associated with amphetamine has not been well documented. Speculation is based on the effects of amphetamine being similar to those of cocaine. Therefore, our observations suggest the hypothesis that high catecholamine levels after exposure to amphetamine may trigger AMI by enhancement of platelet activation and thrombin generation, and might be responsible for the thrombotic occlusion at the site of multiple “vulnerable” plaques. A different explanation is likely in our patient, i.e. acute occlusion of one large culprit vessel may have triggered subsequent pump failure, thereby compromising flow in another coronary vessel where a severe stenosis was already present, and causing acute “secondary” thrombosis. The observations in our patient with cardiogenic shock, although compatible with the hypothesis of a “low-flow”-driven thrombosis of a second vessel, cannot provide any definite answer as to whether the shock was a cause for, or an effect of, acute occlusion of multiple coronary vessels.

The ECG change of extraordinary diffuse ST eleva-
tion in our patient suggests an extensive myocardial infarction, in which acute multivessel obstruction should be considered. However, a large infarction usually kills the patient before he can come to medical attention or is associated with severe circulatory impairment, as presented in our patient who failed to survive soon after AMI.

Medical treatment for amphetamine-induced myocardial infarction deviates from the standard treatment in certain aspects. In amphetamine abusers presenting with chest pain, oxygen, aspirin, coronary vasodilators and benzodiazepines can be given initially. β-blockers were avoided because of the concern of unopposed α activation, which could result in severe hypertension.

In conclusion, our case report demonstrates that acute occlusion of multiple coronary vessels by thrombosis can occur in amphetamine abusers. Further study of the effects of amphetamine on vascular biology is required to clarify the mechanisms underlying the acute coronary thrombosis by amphetamine.

REFERENCES

在一個安非他命濫用者發生雙冠狀動脈急性血栓的病例報告

藍偉仁  葉宏一  侯嘉殷  周友三
台北市 馬偕紀念醫院 心臟內科

藥物引起的急性心肌梗塞並不是一個常見的現象。而且大部分病例中，其發生的機轉與冠
狀動脈痙攣有關，包括安非他命濫用者，其冠狀動脈攝影通常都是正常。我們報告一個30
歲男性安非他命濫用者發生左前降枝及左迴旋枝的急性冠狀動脈血栓造成急性心肌梗塞。
我們推論安非他命的使用和發生多重冠狀動脈急性血栓的形成存在著因果關係。

關鍵詞：安非他命、冠狀動脈、血栓。