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Severe Coronary Vasospasm During an Acute Myocardial Infarction with Cardiogenic Shock

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Abstract

Coronary artery vasospasm can cause a transient, abrupt, marked decrease in the diameter of an epicardial coronary artery. Various mechanisms have been reported, including vasoconstrictor substances, pharmacologic stimuli, and neurohumoral effects. Spasm usually develops at the site of subcritical or critical stenosis, but it may also occur in angiographically normal arteries, particularly in Asian patients. There appears to be a higher prevalence of coronary spasm in patients with acute coronary syndrome (20% to 38%) than in those with stable angina (< 6%). We report a case of coronary artery spasm of the non-infarct-related arteries during an acute myocardial infarction with cardiogenic shock. This possibility should be kept in mind so that it can be properly managed if present. (J Intern Med Taiwan 2005; 16: 129-133)

Key Words ： Acute myocardial infarction, Cardiogenic shock, Coronary artery vasospasm

Introduction

Coronary artery vasospasm is defined as total or near-total occlusion of a vessel that is reversible, or more specifically, it is a significant (> 50%) transient narrowing in either normal or diseased arterial segment reversible with isosorbide dinitrate[1]. Coronary vasospasm of infarct-related arteries during an acute myocardial infarction (AMI) has been postulated to occur at the site of intraplaque or mural thrombosis because of local vascular hyperreactivity to stimuli of constriction such as thromboxane A2, serotonin, and thrombin[2]. Severe spasm of a non-infarct-related coronary artery, however, has rarely been reported.

Case Report

An 85-year-old male who had history of hypertension, cerebrovascular accident, and coronary

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artery disease (CAD) for 2 years. He was treated with balloon angioplasty for lesions in the diagonal branch of the left anterior descending (LAD) artery and the middle segment of the left circumflex (LCX) artery, and a stent was deployed in the LCX at that time. The patient was a smoker. He has been asymptomatic since then till the evening before admission, when he complained of recurrent chest discomfort and drowsiness while at rest. On arrival in the emergency room next morning, his blood pressure was 143/70 mmHg, pulse 73 beats/min, respiratory rate 20/min, and temperature 36.3 ℃. No abnormalities were found on physical examination of the heart, lungs, or abdomen. A chest x-ray showed clear lung fields. Blood tests showed a BUN of 16 mg/dl, white blood cell count 11900/mm^3, hemoglobin 14.2 mg/dl, magnesium 2.7 mg/dl, creatine kinase (CK) 15 u/l, CK-MB 9.8 u/l, and troponin-I < 0.10 ng/dl.

20 minutes post arrival, he subsequently developed bradycardia and hypotension with BP 64/26 mmHg. An electrocardiogram (ECG) showed ST segment elevation in leads II, III, and AVF and ST depression in leads I, AVL, V1-V5, and complete AV block with junctional escape rhythm at a rate of 55 beats/min was noted, there was also Q wave in leads V1-V3 (Fig. 1). Right precordial ECG showed no ST elevation. He was put on transcutaneous pacemaker (TCP), followed by intravenous fluid challenge and dopamine infusion to maintain his blood pressure. He was admitted to the coronary care unit to wait for catheterization laboratory vacancy for a scheduled primary angioplasty.

On the way to catheterization laboratory one hour post admission, patient developed several episodes of ventricular tachycardia which were successfully converted by electric shocks. Because of hemodynamic instability and the duration of chest pain was less than 12 hours, primary percutaneous coronary intervention (PCI) was performed quickly after coronary care unit admission, with temporary pacemaker implanted for rhythm backup. Coronary

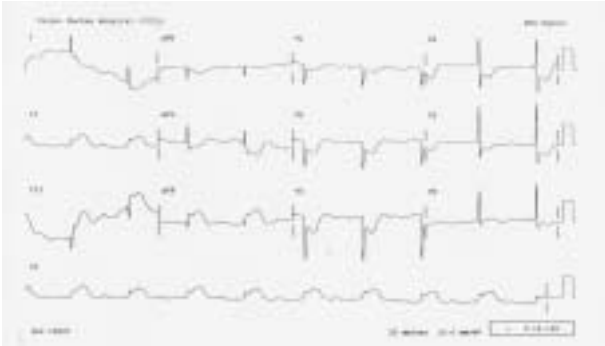

Fig. 1. ECG demonstrating 3-to-5 mm ST-segment elevations in leads II, III, and AVF, ST segment depression in leads I, AVL, and V1-5, and complete AV block with junctional escape rhythm at 55 beats/min. The Q wave in leads V1-3 was also seen.

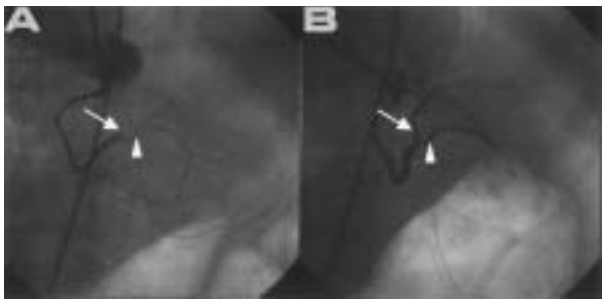

Fig. 2. A:Coronary angiography in the right anterior oblique cranial projection. There are severe lesions in the PDA (90%) and PLV (100%) branches of the RCA.
B:Repeat coronary angiography following successful PCI in the right anterior oblique cranial projection with estimated residual lesions in the PDA (4%) and PLV (10%) branches of the RCA.

angiography (CAG) showed severe stenosis (90%) of the posterior descending artery (PDA) branch of the right coronary artery (RCA) and total occlusion of the posterior left ventricular (PLV) branch (Fig. 2A). The proximal portions of the LAD and LCX were severely obstructed (Fig. 3A). CAG was repeated again after 500 μg bolus of intracoronary isosorbide dinitrate to rule out coronary vasospasm, which revealed the same results. PCI was attempted at first only for the infarct-related artery (RCA), with good result (Fig. 2B).

An intra-aortic balloon pump and a Swan-Ganz catheter were inserted for hemodynamic support and monitor, and medications adjustment. He was suc-

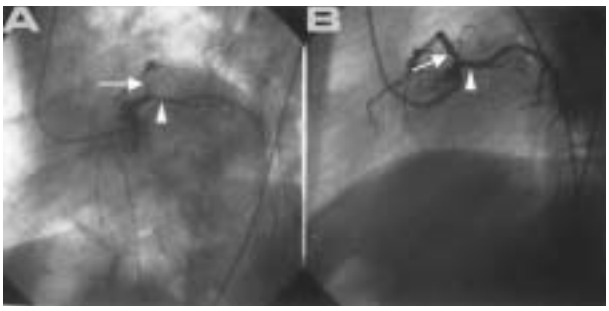

Fig. 3. A:Coronary angiography in the left anterior oblique caudal projection. There is severe stenosis in the proximal portion of the LAD (75%, white arrow) and LCX (85%, white arrow head). B:Repeat coronary angiography five days later after resolved of cardiogenic shock in the left anterior oblique caudal projection. There is an insignificant lesion of the proximal LAD (30%, white arrow) and a patent proximal LCX (white arrow head).

cessfully weaned off mechanical and inotropic agent five days later. Then CAG was repeated showing the previously severe lesions in the LAD turned out to be insignificant and the LCX was totally patent (Fig. 3B). We therefore speculated that the previously severe lesions in both LAD and LCX could be due to vasospasm at the time of an acute infarction complicated with cardiogenic shock. Calcium channel blocker was prescribed subsequently. He remained well and symptom-free in subsequent follow up at the outpatient clinic.

Discussion

Coronary vasospasm typically occurs in a segment of an epicardial conduit artery. It is appears to be more frequent in patients with acute coronary syndrome (20% to 38%) than in those with stable angina (< 6%)[2,3]. Coronary vasospasm can superimposed on angiographically advanced atherosclerotic coronary artery disease in about 60% of cases[3,4], and on angiographically normal coronary arteries, particularly in Asian patients[5].

In one study, the basal myocardial blood flow per gram of perfusable tissue was comparatively lower in the infarct regions than in regions remote from the infarct[6]. Response to vasodilators was significantly impaired not only infarct-related arteries but also in remote regions perfused by angiographically normal coronary arteries. The most likely explanations included impairment of endothelium-dependent dilatation, generalized increase in neurohumoral sympathetic activity, or persistent release of vasoconstrictor by the coronary thrombus[6]. Spontaneous normal coronary artery spasm after remote coronary stretching, as in the LAD and LCX vasospasm after RCA stent deployment, has been reported[7].

In our case, focal coronary spasm was initially noted in arteries which were subsequently seen to be normal and near-normal. The patient did not have multivessels multifocal critical stenosis to cause shock. There are several other explanations for this: firstly, a higher incidence of exaggerated segmental response to constrictor stimuli has been observed in ACS, in both normal and abnormal coronary arteries[2,3]; secondly, we were administering dopamine and norepinephrine, both of which have been reported to trigger a coronary vasospastic response[8,9]; thirdly, it has been reported that vasospasm appears to occur in artery segments with at least minimal atherosclerotic change not detected by coronary angiography but detected by more sensitive techniques such as intravascular ultrasonography[10]. The patient did have 30% stenosis of the LAD, and there was mild calcification in the proximal portion of the LAD and LCX; and finally, it has been suggested that there may be increased vasoconstrictive sensitivity secondary to endothelial dysfunction in non-infarct-related arteries during an acute infarction[11].

The question arises regarding the patient's vasospasm not responding to a 500 μg bolus intracoronary isosorbide dinitrate given during the initial PCI. It can be due to low coronary perfusion and stagnant blood flow that there lead to inadequate concentration of nitrates at the site of spasm. Other possibilities include the presence of powerful stimuli from the thrombus or simply a limited response to the drug in the involved vessels[6,12].

Coronary artery vasospasm may lead to ventricular arrhythmia[13] and further myocardial dysfunction. In our patient, several episodes of ventricular tachycardia could have been triggered due to myocardial infarction or injury caused by the coronary spasm. Coronary vasospasm of the LAD and LCX not only reduces perfusion to the myocardium they supply but may also decrease collateral flow to the RCA, further impairing myocardial function[6]. This patient's cardiogenic shock may therefore have been due to a combination of bradycardia and myocardial dysfunction secondary to severe coronary vasospasm of left coronary arteries and thrombotic atherosclerosis of RCA.

First-line treatment for coronary vasospasm is the removal of precipitating factors. In many cases, vasospasm responds to nitrates and calcium channel blockers[14]. The latter have been reported to be useful in reducing the risk of cardiac arrest and sudden death[14]. Stent placement may be an adjunct in the management of carefully selected patients with focal epicardial coronary artery spasm refractory to medical therapy[4]. However, meticulous management of a patient's known atherosclerotic coronary artery disease is also important because of the association between regression of atherosclerosis and cessation of vasospastic activity seen in some studies[2,15]. Therefore, attention should be paid to risk factors modification as well as use of appropriate medications. In our patient this would include cessation of smoking, control of hypertension, and avoidance of beta-blocker usage.

This case report illustrates the important role of coronary artery vasospasm in mimicking focal coronary atherosclerosis. We must exclude coronary vasospasm by intracoronary nitrate or Calcium channel blocker injection before coronary angiography routinely. When severe stenosis appears at the site of the non-infarct-related arteries in a patient with an AMI especially during the hemodynamic unstable conditions, vasospasm should be included in the differential diagnosis.

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嚴重冠狀動脈的痙攣
發生於急性心肌梗塞併心因性休克

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摘　要

冠狀動脈血管痙攣能造成一個暫時的，突然的，顯著的心外冠狀動脈內徑的減少。各種機轉曾被報告過，包括促血管收縮物質，藥物的刺激及神經荷爾蒙的影響。痙攣通常發生在次嚴重或嚴重狹窄處，但它也會發生在血管攝影正常的血管，尤其是亞洲的病人。在急性冠狀動脈症候群的病人(二十到三十八個百分比)比穩定性心絞痛的病人(小於六個百分比)有較高比率會有冠狀動脈血管痙攣。我們報告一個在急性心肌梗塞併發心因性休克時在非梗塞冠狀動脈的痙攣。我們必須謹記這種可能性並做適當正確的處理。