Severe Spontaneous Multiple Coronary Artery Spasm during Coronary Angiography

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Abstract

Acute coronary syndrome (ACS) is a critical disease especially in case of acute ST segment elevation myocardial infarction (STEMI). Acute STEMI typically shows ST segment elevation on electrocardiography (ECG), elevation of serum cardiac enzyme and abnormal regional wall motion on echocardiogram. However, coronary artery spasm(CAS), subarachnoid hemorrhage, pheochromocytoma, electroconvulsive treatment, and apical ballooning syndrome[1-3] can also result in similar clinical scenarios. Here, we report a case of a 65-year-old male who was admitted to our hospital because of severe chest pain associated with cold sweating and syncope. Coronary angiography (CAG) revealed spontaneous total occlusion of the left anterior descending artery (LAD) and right coronary artery (RCA) accompanied with severe chest pain, ST segment elevation, and hypotension. The severe coronary spasms were relieved via intracoronary administration of isosorbide dinitrate. After discharge, the patient was treated with verapamil, oral long-acting nitrate, and nicorandil. No chest pain with cold sweating or syncope occurred at OPD follow-up. (J Intern Med Taiwan 2010; 21: 359-365)

Key Words： **Coronary artery spasm, Acute ST segment elevation myocardial infarction, Coronary angiography**

Introduction

CAS is a well-documented etiology of variant angina characterized by spontaneous episodes of angina in association with ST segment elevation on electrocardiogram[4]. CAS is usually focal and typically occurs at a single site, although spasm in more than one site and diffuse spasm have been described[5,6]. Further, CAS generally occurs only transiently when a patient is at rest[4]. It has also been suggested that CAS is a cause of angina pectoris, myocardial infarction, syncope, malignant arrhythmia, and sudden death in patients who otherwise have normal or near normal coronary arteries. Spontaneous, multivessel coronary spasm with coronary artery total occlusions during CAG is less common. This case describes a 65-year-old male who presented with severe chest pain associated with cold sweating and syncope. CAG

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revealed spontaneous total occlusion of the LAD and RCA accompanied with severe chest pain, ST segment elevation, and hypotension.

Case Report

A 65-year-old male presented to Cheng-Hsin hospital because he had an episode of severe chest pain associated with cold sweating and loss of consciousness for about 10 seconds at rest. When he regained consciousness, the chest pain was relieved. The patient was asymptomatic on presentation and a resting 12-lead electrocardiogram (ECG) was normal. Initial assessment suggested acute coronary syndrome. His coronary risk factors were hypertension, diabetes mellitus, and more than 40 years of cigarette smoking. The patient was being treated for diabetes and hypertension under metoprolol-XR 50mg qd, losartan 50mg qd, metformin 500mg bid and pentoxifyllione 400mg qd.

On the first day of hospitalization, 300mg clopidogrel and 300mg aspirin were given. A treadmill exercise test using the standard Bruce test protocol revealed significant flat ST depression in the precordial leads during stage 2 of the test (Figure 1). Diagnostic cardiac catheterization with a right radial artery approach was performed on the second day of hospitalization. Administration of 5000IU of intravenous heparin was given before CAG. Left coronary angiography was performed using a 5 French, 3.5 cm left Junkins catheter and right coronary angiography was performed using 5 French, 4 cm right Junkins catheter. About 6-8 mL of Iopamidol (a low-osmolar nonionic contrast) was injected into coronary artery for each CAG. The initial left coronary angiography revealed insignificant stenoses in the proximal segment of the left anterior descending artery (LAD); however, subsequent imaging demonstrated severe coronary spasm with complete obliteration of the LAD. At this point during the angiography, the patient experienced chest pain, ST elevation in lead I, and

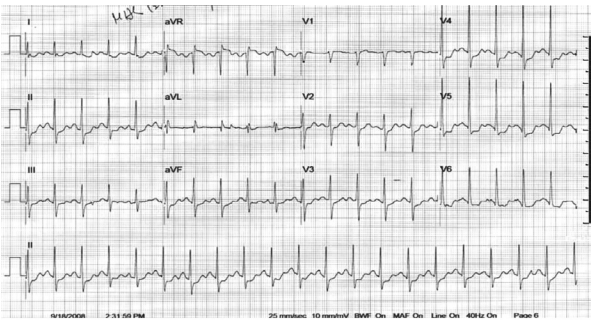

Fig. 1. The exercise treadmill test showed V4-V6 ST segment 2 mm horizontal depression at 5 minutes and 18 seconds of the test.

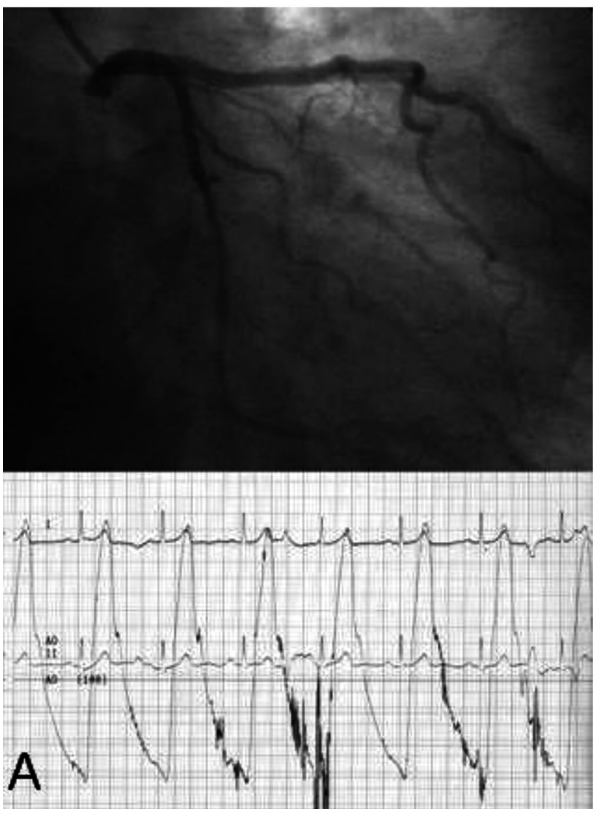

Fig. 2. (A) Coronary angiography revealed mild stenosis (30%) of the proximal segment of the LAD, 50% stenosis of the distal segment of the LAD, and 50 % stenosis of the circumflex middle segment. Baseline blood pressure was 150/70 mm Hg.

the patient's blood pressure decreased to 112/60 mm Hg from 150/70 mm Hg (Figures 2A and 2B).

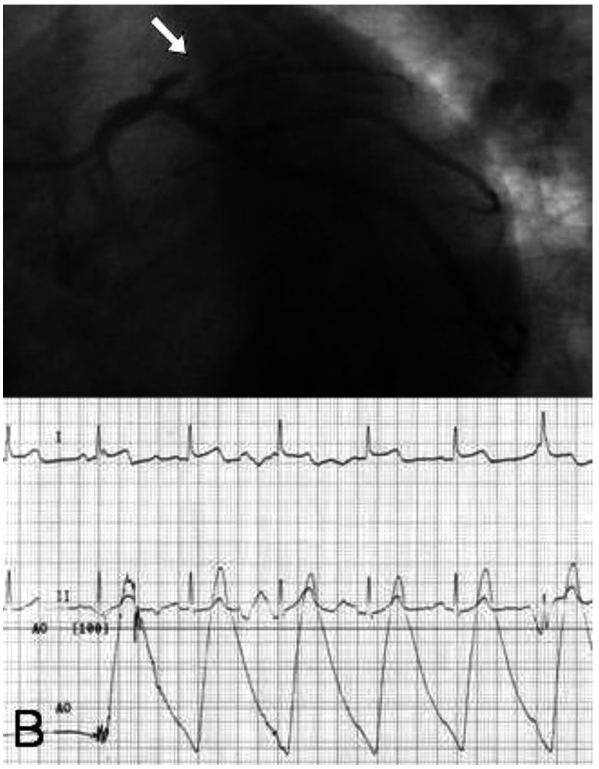

Fig. 2. (B) Coronary angiography revealed total occlusion of the proximal segment of the LAD (arrow). Systemic blood pressure dropped to 112/60 mm Hg.

The coronary spasm was promptly relieved with intra-coronary administration of isosorbide dinitrate 100ug. Right coronary angiography of the right coronary artery (RCA) showed a mild (<30%) stenosis in the proximal segment (Figure 2C). At the end of the procedure at disengagement of the diagnostic catheter, the patient suddenly complained of severe chest pain, profound cold sweating, ST segment elevation in the inferior leads, and marked bradycardia. The systolic blood pressure decreased to 76 mm Hg and subsequent angiography revealed totally obliteration of the proximal segment of the RCA (Figure 2D). Intra-coronary isosorbide dinitrate 100ug was administered immediately despite continued hypotension. Successful reversal of the severe spasm was achieved. Post-procedurally, the patient was treated with verapamil, a long-acting nitrate, and nicorandil. The patient

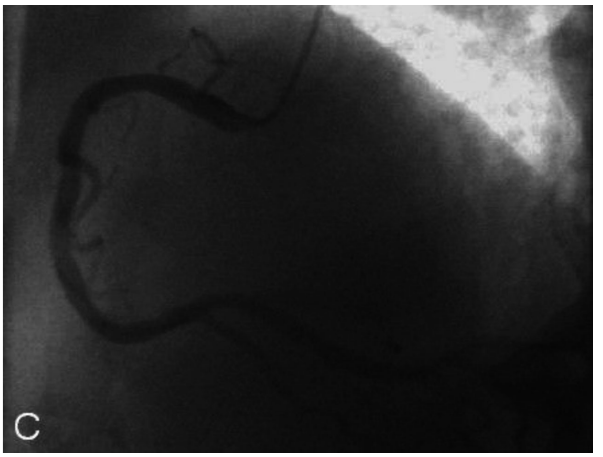

Fig. 2. (C) Right coronary arteriography revealed mild stenosis (30%) in the proximal segment of the RCA.

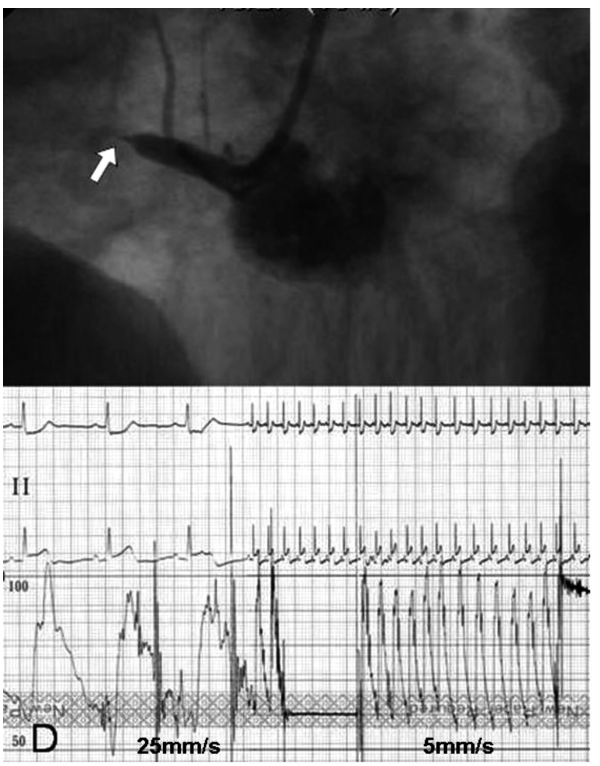

Fig. 2. (D) Coronary angiography revealed total occlusion of the proximal segment of the RCA (arrow). Systemic blood pressure decreased to < 100 mm Hg.

was discharged in stable condition. No chest pain with cold sweating or syncope occurred after OPD follow-up.

Discussion

CAS is a well documented etiology of variant angina, which causes myocardial ischemia, angina, syncope[7], life-threatening ventricular arrhythmia, and sudden cardiac death. Variant angina, also called Prinzmetal's angina, was first described by Prinzmetal et al. in 1959[4]. Patients with CAS and no or only mild coronary obstruction tend to experience a more benign course than patients with associated severe obstructive lesions. The reported incidence of coronary arterial spasm during coronary angiography varies between 0.26% and 2.93%[8-11]. The incidence of spontaneous multivessel CAS during CAG remains unknown, but is widely considered to be rare. Atherosclerotic disease affecting large coronary arteries alters the vasomotor tone and reactivity of the affected vessels and an intimate association of spasm with sites of organic stenosis is suggested. MacAlpin reported that 88% of spasm cases causing ischemia were localized to the site of an organic stenosis[12].

Friedman et al.[13] reported angiographic criteria to distinguish between the spasm of variant angina and catheter-induced spasm. Catheter-induced spasm is usually asymptomatic, almost invariably in the RCA, occurs at the catheter tip, is smooth, concentric, and less than 2 mm in length along the vessel. In contrast, spontaneous spasm occurs in any coronary artery, at a distance ranging from 1-4 cm from the catheter tip, is usually irregular and eccentric, and is associated with angina, ST segment elevation, hypotension, and dysrhythmia.

The mechanisms of CAS remain uncertain but are considered to be multifactorial. Many researchers believe that coronary spasm that develops during catheterization is partly spontaneous and partly catheter-induced. Predisposing factors for catheter-induced coronary spasm include use of a larger catheter size, a small left main coronary artery (LMCA) diameter, a large catheter/LMCA ratio, catheter contact with the vessel wall, and vessel bulging[14]. In the case described herein, the distance from the catheter tip to the LAD vasospastic segment was more than 1 cm and when the LAD was totally occluded, the patient was symptomatic. The ECG showed ST segment elevation and the blood pressure dropped after right coronary angiography and disengaging the catheter. Subsequent angiography showed total occlusion of the proximal segment of RCA. Together, the information suggests that the CAS occurred spontaneously.

Rare cases of CAS induced by contrast media had been reported[15]. Clinical presentations of allergic reaction include bronchospasm, cyanosis, severe hypotension, skin rash, rhinorrhea, conjunctivitis, facial edema and pruritus. Ionic contrast media is more allergenic than nonionic contrast media (3% versus <1%)[16]. In our patient, no above allergic reaction was noticed during or after CAG. CAS happened minutes after contrast injection, rather than happened immediately during each injection, makes contrast media-related CAS in doubt.

In the study of Bory et al.[17], a low incidence of sudden death (3.6%) and myocardial infarction (6.5%) was reported after a median follow-up of 89 months. This study included a large population of 277 patients with vasospastic angina and normal or nearly normal coronary arteries (<50% stenosis) with calcium channel blockers therapy. Multivariate statistical analyses showed that the only predictors of major coronary events (i.e., death, myocardial infarction, or angina requiring repeat CAG) were systemic hypertension or the finding of minor parietal irregularities on the initial CAG[17].

Cardiac arrest and sudden death are important risks of variant angina even in the absence of severe organic coronary stenosis. MacAlpin[18] found that the risk of one of these events in variant angina was increased 1.5-fold in the absence of high-grade

coronary stenosis and increased three-fold by history of angina-liked syncopal attacks. Sudden death could occur due to coronary spasm without pain. Multivessel coronary artery spasm is associated with a high risk of a life-threatening cardiac event[19,20]. Patients who experience an arrhythmia complication, especially ventricular fibrillation and sudden death, during an episode of spasm could also have a poor prognosis.

Patient with Prinzmetal variant angina should be strongly encouraged to stop smoking. The mainstay of therapy is calcium antagonist alone or in combination with long-acting nitrates. There are several important differences between the optimal management of variant angina and classic angina. For example, beta-blockers, effective in the management of typical angina pectoris, do not relieve pain and, in fact, could precipitate and intensify pain in patients with variant angina. Calcium antagonists have proven to be extremely effective in preventing the coronary spasm in variant angina and should routinely be prescribed at maximally tolerated doses on a long-term basis. In patients with refractory variant angina, Prazosin (a selective alpha adrenoreceptor blocker) and nicorandil may also have value in patients with refractory variant angina[21,22]. Estradiol supplementation has also been tested with some promise in women[23]. Percutaneous coronary intervention and, occasionally, coronary artery bypass grafting may be helpful in patients with CAS associated with discrete, proximal fixed obstructive lesions[24], but are contraindicated in patients with isolated coronary artery spasm without accompanying fixed obstructive disease.

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嚴重多條冠狀動脈自發性痙攣發生於冠狀動脈攝影

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摘 要

急性冠心症是一種嚴重之疾病，尤其是急性ST段上升心肌梗塞。急性ST段上升心肌梗塞典型呈現心電圖ST段上升、血清心肌酵素酶上升及心臟超音波有局部心肌活動異常。臨床上有一些情形類似急性ST段上升心肌梗塞，包括冠狀動脈痙攣、蜘蛛網膜下出血、嗜鉻細胞瘤、電休克治療及心尖球型綜合症。在此我們提出一位65歲男性，因嚴重胸悶並冒冷汗及昏厥住院，冠狀動脈攝影時發現左前降支及右冠狀動脈自發性全阻塞，並有胸悶、ST段上升及低血壓情形，經冠狀動脈內注射isosorbide dinitrate後緩解，出院後病患接受口服藥物verapamil、長效型nitrate及nicorandil治療，門診追蹤期間未再發生胸悶、冒冷汗或昏厥。