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Case Report

Coronary Artery Spasm Induced Intimal Dissection in a Patient after Percutaneous Transluminal Coronary Angioplasty

Michael Chich-Kuang Chang, Andrew Ying-Siu Lee, Tien-Jen Chen, Wen-Fung Chang
Division of Cardiology, Jen Ai Hospital, Tali, Taichung, Taiwan

Abstract: There is as yet no convincing evidence that coronary spasm predisposes to intimal dissection. We report a case of acute dissection of the left circumflex coronary artery which occurred following coronary spasm as a result of angioplasty of the left anterior descending coronary artery.

Key Words: Coronary dissection; Coronary spasm.

Introduction

Coronary spasm and intimal dissection are common, well-known complications of percutaneous transluminal coronary angioplasty (PTCA) with life-threatening consequences.[1] It has been shown that coronary spasm may be superimposed on an area of dissection after balloon angioplasty of organic coronary lesions.[2,3] However, the reverse coexistence, that is, intimal dissection secondary to coronary spasm, has never been reported.

We describe a patient in whom acute dissection of the left circumflex coronary artery (LCX) occurred following severe multivessel spasms of all coronary arteries during the procedure of PTCA of the left anterior descending coronary artery (LAD), which resulted in transient complete occlusion with accompanying signs of transmural ischemia.

Case Report

A 60-year-old man with a history of two-vessel disease was admitted for coronary angiography because of increasing exertional and rest angina for several months' duration. The blood pressure was 108/60 mm Hg, heart rate 84 beats/min, respiration 24/min and the temperature was 37 °C. The lungs were clear bilaterally. The heart rhythm was regular and a grade 2 to 3/6 systolic murmur was heard over the left sternal border. The remainder of the physical examination was normal. The electrocardiogram and chest X-ray were normal. Complete blood count and blood chemistry profile were within normal limits.

The patient underwent cardiac catheterization by the Judkins' technique. The coronary arteriograms revealed a tortuous right coronary artery (RCA) with a symmetrical 90% stenosis in the mid-RCA, a 70% eccentric stenosis in the mid-LCX and an 80% eccentric stenosis in the mid-LAD arteries (Fig. 1, top). The patient subsequently underwent PTCA. An 8-F four-bend Judkins guiding catheter was used to cannulate the arteries and a 0.014 flexible steerable guide wire was used to cross the lesions. Dilatations were performed with Tsurugi 1.5 × 20mm and Worldpass 2.0 × 20mm balloon catheters. Angiography performed after balloon deflations showed initial successful dilatation of the RCA and LCX lesions, and the target stenoses were reduced to less than 25% (Fig. 1, second row).

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Correspondence: Dr. Andrew Ying-Siu Lee
Divison of Cardiology, Jen Ai General Hospital, Tali, Taichung, Taiwan, R.O.C.

However, when subsequent dilatations of the LAD were performed, multivessel spasms were observed in all coronary vessels, with complete occlusion of the LAD (Fig. 1, third row). The patient then complained of severe chest pain and had marked electrocardiographic ST segment elevation. Systemic hypotension also developed. After multiple intracoronary injections of nitroglycerin, verapamil and adenosine, and repeated balloon dilatations of the LAD, chest pain reduced gradually and the ST segment changes became stabilized. Subsequent angiography showed partial resolution of the LAD lesion with a residual stenosis of 50%. Surprisingly, clear evidence of intimal dissection was also observed in the LCX near the atherosclerotic lesion, which was not present before the insult of multivessel spasms (Fig. 1, bottom). Later, the chest pain, ST segment elevation and hypotension kept recurring, necessitating intravenous infusions of urokinase, heparin, nitroglycerin and dopamine. Moreover, the coronary dissection was treated with prolonged balloon inflations which successfully restored the patency and distal flow of the artery. Two days later, the patient became stable and asymptomatic, and was discharged apparently well.

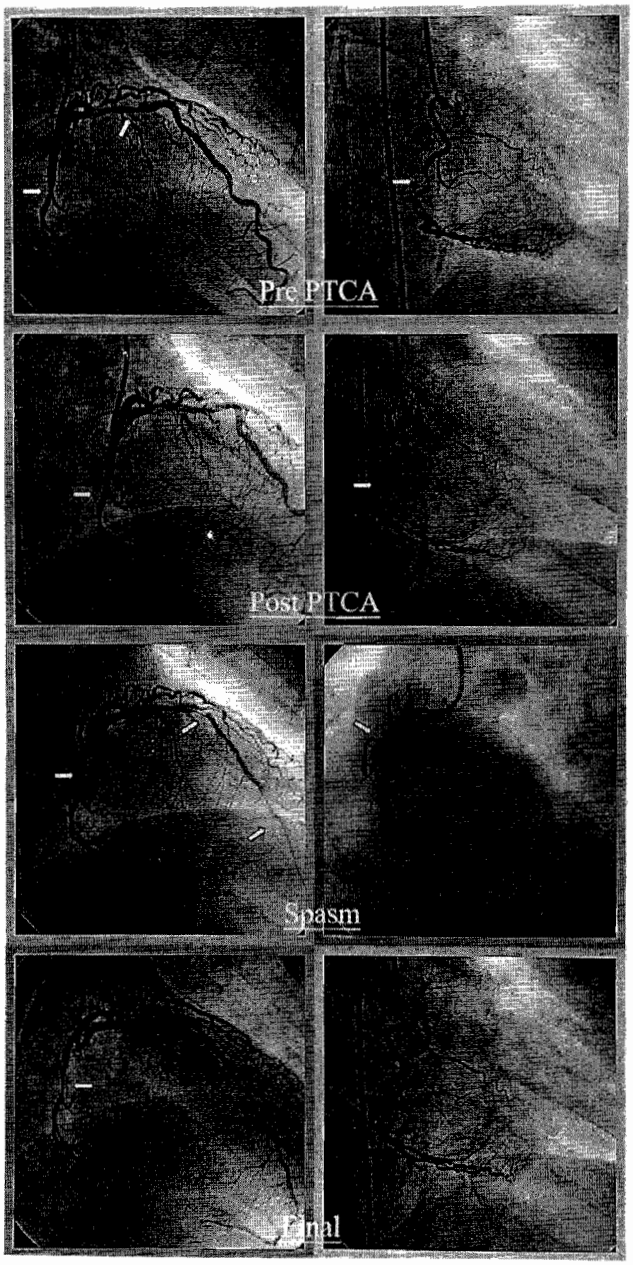

Fig. 1. Top: Cine frames of the coronary arteries showing 90, 70 and 80% middle lesions in the RCA, LCX and LAD, respectively. Second row: Successful PTCA of the RCA and LCX. Third row: Multivessel spasms in all coronary vessels due to PTCA of the LAD. Bottom: Subsequent cine frames after intracoronary nitroglycerin and repeated balloon dilatations documenting the relief of the total obstruction in the LAD, and showing a new intimal dissection of the LCX.

Discussion

During coronary arteriography, it has been reported that ergonovine administration[4,5] or prior coronary dissection[4] might predispose to coronary spasm. Recently, the coexistence of coronary dissection and spasm has also been observed in patients with acute myocardial infarction. Coronary dissection in the RCA with coronary spasm in the LAD,[6] as well as coronary dissection in the LCX with coronary spasm in RCA,[7] have been reported. Therefore, although there is as yet no convincing evidence that coronary spasm is a significant factor in the causation of coronary dissection, the possibility should be kept in mind. Although small intimal injury or dissection occurs frequently following angioplasty (incidence of 60-80%), in our patient, however, there was no evidence of dissection in the LCX prior to performance of PTCA of the LAD. Following angioplasty of the LAD, however, severe multivessel spasms of all coronary arteries were observed which resulted in sudden, severe coronary obstruction. The failure of coronary

obstruction to be relieved promptly with nitroglycerin raised the possibility of coronary dissection, which was found in the formerly well-looking LCX. The course and serial arteriograms of our patient therefore give direct evidence that coronary spasm predisposes to intimal dissection. It is speculated that when the coronary artery is markedly constricted, the intima may become wrinkled or develop ridges,[8] which may predispose to subsequent dissection. The fact that only the LCX was dissected following coronary spasm might be due to a preexisting more serious intimal injury after angioplasty of that artery than of the others. Moreover, since the coexistence of coronary dissection and spasm has been reported in acute myocardial infarction patients prior to the performance of PTCA,[6,7] whether or not coronary spasm per se may induce coronary dissection in patients with intact coronary arteries remains to be elucidated.

The coexistence of coronary spasm and dissection is of clinical importance, since both of these complications occur with reasonable frequency during PTCA. Both conditions can, during the first few minutes after they happen, present a clinical and electrocardiographic picture of sudden, severe coronary obstruction leading to life-threatening consequences. Both complications can predispose to or be superimposed on one another, which may contribute importantly to the clinical picture. Both of these entities can adversely affect the outcome of coronary angioplasty and need to be treated differently and quickly. Meticulous adherence to good technique of coronary angioplasty is the only protection against these potentially dire complications.

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冠狀動脈痙攣引發血管剝裂 — 一病例報告

張之光 李應紹 陳天珍 張文芳

台中縣大里市 仁愛綜合醫院心臟內科

摘 要: 目前文獻上尚未證實冠狀動脈痙攣會引發血管剝裂，現報告一病例，病人於接受氣球擴張術時發生冠狀動脈痙攣，繼而引發血管剝裂。

關鍵詞: 冠狀動脈痙攣；血管剝裂。

聯絡人：李應紹醫師，412 台中縣大里市東榮路 483 號，仁愛綜合醫院心臟內科

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