

Acute Coronary Syndrome Caused by Coronary Artery Dissection Mimicking Acute Plaque Rupture

Saibal Kar, MD, Prediman K. Shah, MD, FACC, FACP, FCCP

Division of Cardiology, Cedars-Sinai Medical Center, Los Angeles, CA

When a middle-aged, nonpregnant female patient with no coronary risk factors presents with chest pain, what are the red flags for unusual causes? This case report provides important diagnostic clues as well as progressive therapeutic steps to solving a potentially life-threatening problem. [Rev Cardiovasc Med. 2001;2(4):215–219]

© 2001 MedReviews, LLC

Key words: Aneurysm, dissecting • Angioplasty • Coronary artery bypass • Myocardial infarction • Stents

Acute rupture of an atherosclerotic plaque with superimposed thrombosis is the most common pathophysiologic mechanism of acute coronary syndromes.¹ On rare occasions, a similar clinical presentation results from an acute coronary dissection of a nonatherosclerotic coronary artery. Spontaneous coronary dissection, even though rare, is an important cause of acute myocardial infarction (MI) in young and middle-aged women, especially during pregnancy or the postpartum period.² We describe the case of a middle-aged, nonpregnant woman who had no known risk factors for coronary atherosclerosis and who presented with an acute coronary syndrome, which was shown, eventually, to be caused by a spontaneous coronary artery dissection. An initial attempt at stent placement in the culprit vessel led to an iatrogenic retrograde progression of dissection, requiring urgent coronary artery bypass graft (CABG) surgery. Clinical presentation, pathology, and management of this form of nonatherosclerotic coronary artery disease are also discussed.



Figure 1. ECG at the time of the patient's presentation in the emergency department.

Case Report

A 43-year-old, previously healthy white woman presented to a local emergency department (ED) following abrupt-onset, severe anterior chest discomfort after sexual intercourse. The severe chest discomfort lasted for 30 minutes and radiated to the left arm and to the neck; it was associated with nausea and emesis. Assessment of the patient's coronary risk factors revealed that she was normotensive, nondiabetic, premenopausal, and a nonsmoker. She had a normal lipid profile and no family history of premature atherosclerosis. She also denied illicit drug or oral contraceptive use. In the ED, she was given 2 sublingual nitroglycerin tablets, which relieved the continuing chest discomfort within several minutes.

At presentation, the patient's temperature was 37.8°C (100.04°F), pulse was 90 beats per minute and regular,

and blood pressure was 110/80 mm Hg. The patient's oxygen saturation was 95% at rest while breathing room air.

Physical examination revealed an anxious lady in some distress. The lungs were clear. First and second heart sounds were normal, without audible murmurs or rub. There were no associated findings, such as cyanosis, jugular venous distention, calf tenderness, or inequality of pulses or blood pressures in her arms. An ECG showed normal sinus rhythm, with no associated ST-segment elevation or depression (Figure 1). The chest radiograph did not show mediastinal widening or pneumothorax. The patient was treated with aspirin and heparin and was admitted to the coronary care unit for further evaluation and treatment. Subsequent testing revealed an elevated troponin level of 30 ng/mL and ST-segment elevation in leads II, III, aVF, and V₄ to V₆ (Figure 2).

Urgent coronary angiography was performed, which revealed a small filling defect (thrombus) in the distal segment of a large, first obtuse marginal (OM1) branch of the left circumflex artery, along with a proximal small dissection and long segment of narrowing. The patient underwent successful percutaneous transluminal coronary angioplasty (PTCA) of the distal segment of the culprit vessel with restoration of normal blood flow in the vessel. No mechanical intervention was performed on the proximal dissection. The rest of the coronary anatomy showed no luminal irregularities.

Following PTCA, the patient experienced episodes of chest pain, which prompted a second angiography the next day. This angiogram confirmed that the culprit vessel was still open. Her persistent symptoms were presumed to be caused by vasospasm, and she was treated with nitrates and

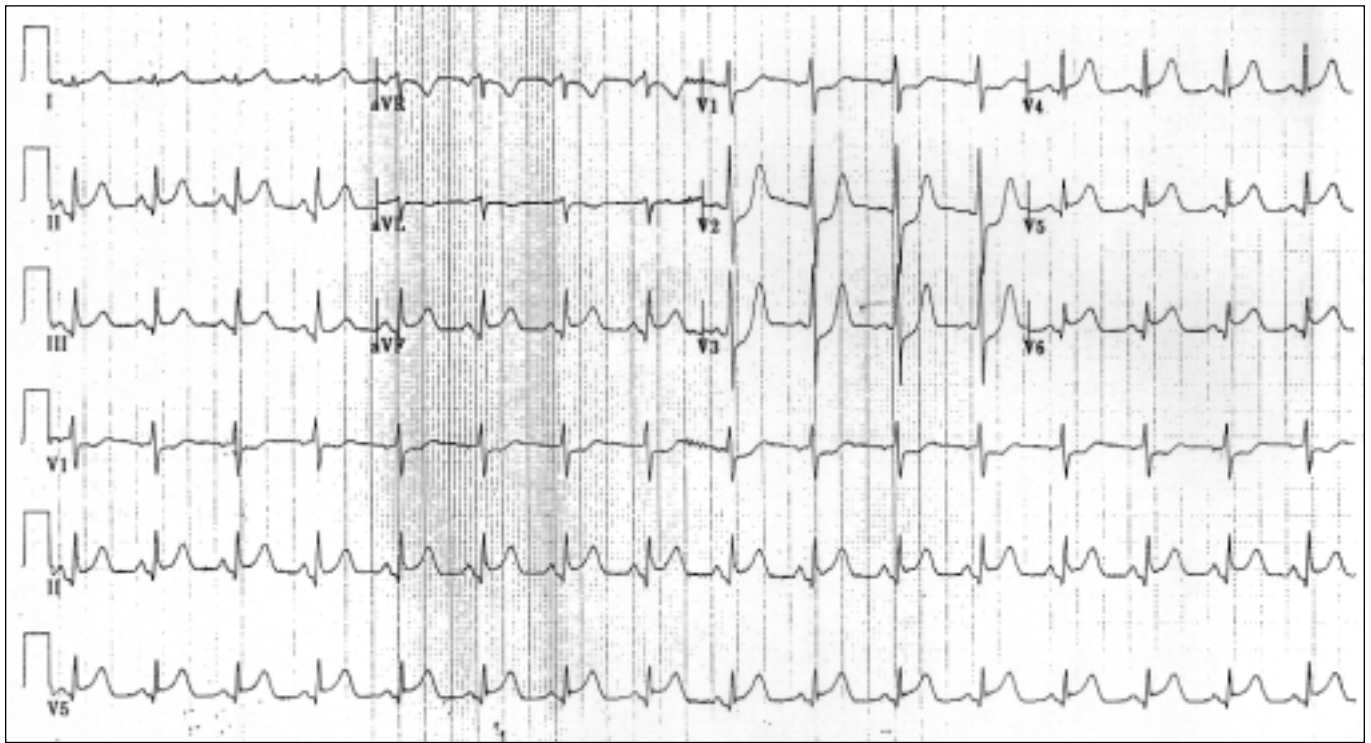


Figure 2. ECG several hours later; electrocardiography performed because of recurrence of the patient's chest discomfort.

calcium channel blockers, without significant relief. At this point, she was transferred to our hospital for further care.

The patient continued to experience intermittent chest discomfort. A careful review of the angiograms suggested that the dissection in the proximal part of the OM1 branch of the circumflex artery was the culprit lesion and that intervention would be needed to help reduce her recurrent ischemic symptoms. A third angiographic procedure was performed, and a 2.5×23 -mm stent was deployed at the above-described site (Figure 3). She was finally discharged symptom-free 4 days after the last coronary intervention.

A week following discharge, the woman presented with recurrence of severe angina. A radionuclide imaging test revealed a large area of ischemia in the territory of the OM1 branch of the left circumflex artery. Angiography performed the same day showed a greater than 90%

stenosis of the OM1 branch just proximal to the stented segment, along with TIMI (Thrombolysis In Myocardial Infarction) grade 1 flow in the artery (Figure 4A), possibly caused by proximal dissection. A 2.5×12 -mm stent was deployed successfully in the proximal part of the culprit vessel. Following deployment, angiography showed there was proximal extension of the dissection into the left main coronary artery without any compromise of blood flow to its branches (Figure 4B). Because of both the propensity of the patient's coronary arteries to dissect and the long-term benefits of coronary artery surgery over stenting of the left main coronary artery, she required urgent CABG surgery.

During surgery, it was found that the OM1 branch of the left circumflex artery was dissected beyond the stent, suggesting a primary dissection as a mechanism of the patient's initial presentation. The left anterior descending artery and the OM1

and OM2 branches of the circumflex artery were grafted using 3 arterial grafts. The patient had an uneventful postoperative course. At 1-year follow-up, she is doing well and is free of angina. Results of a stress test showed no ischemia.

Based on the clinical, angiographic, and operative findings, it seems most likely that her initial clinical presentation was caused by spontaneous dissection of the OM1 branch of the left circumflex artery with a resulting flow-limiting state; in addition, there was probable secondary luminal thrombus formation and distal embolization. This resulted in an inferoposterolateral MI of moderate size. The long segment of narrowing was a result of the intramural hematoma, causing compromise of the lumen.

The woman did not have a body habitus suggestive of a connective tissue disorder, such as Marfan syndrome. Also, at the time of presentation, she was not pregnant or

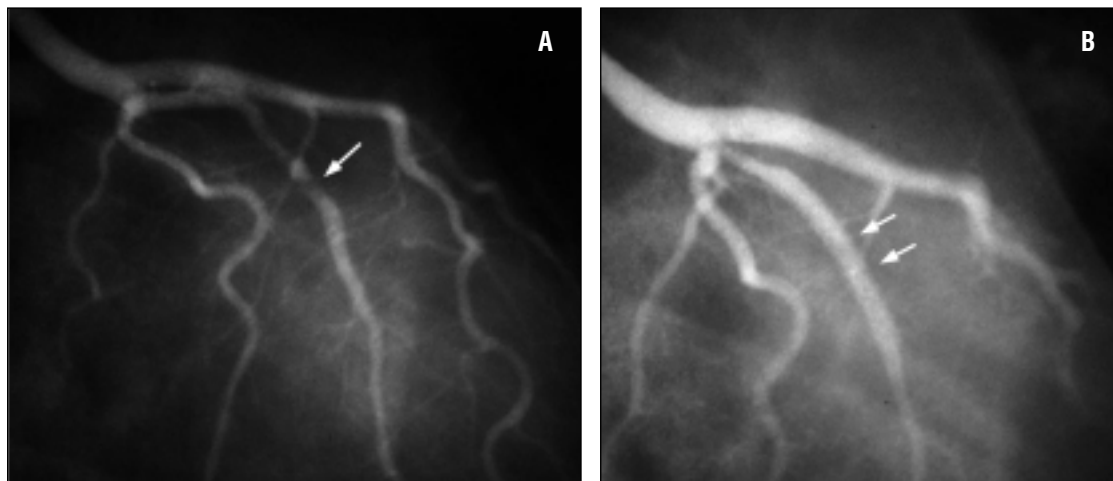


Figure 3. (A) Angiogram showing a spontaneous dissection of the proximal segment of the first obtuse marginal (OM1) branch of the left circumflex artery (arrow). (B) Angiogram following successful deployment of the stent at the lesion (arrows).

postpartum, nor was she taking oral contraceptives—all known risk factors for spontaneous coronary artery dissections.

Discussion

Most acute coronary dissections are secondary to aortic dissections, Marfan syndrome, CABG surgery, or cardiac catheterization/coronary interventions, and only rarely cause acute MI.²⁻⁴ Primary or spontaneous coronary dissections are an important (though rare) cause of acute coronary syndrome, especially in young and middle-aged women. In our patient, a spontaneous dissec-

tion was the possible cause of the initial presentation. In addition, the PTCA-induced proximal extension of the dissection resulted in a significant left main coronary artery dissection that required urgent CABG.

Eighty percent of spontaneous coronary dissections occur in women.² Most happen during pregnancy or the postpartum period or are related to oral contraceptive use.^{5,6} Gender differences do exist, however. Left coronary dissections are more common in women; right coronary dissections, in men.^{7,8} At autopsy, extensive intramural dissections have been observed. The

plane of dissection is usually in the outer third of the media or between the adventitia and media.⁸ The exact pathophysiologic mechanism of these dissections is still unclear. It has been postulated that hormones (especially progesterone) can cause weakening of the media; the stress of labor or of strenuous activity may lead to dissection of a weakened vessel. Several reports have demonstrated inflammatory cell infiltration in the walls of the affected vessels, suggesting a role for a cytokine-mediated process. It remains unclear whether these inflammatory changes are the cause or effect of the dissection.⁹

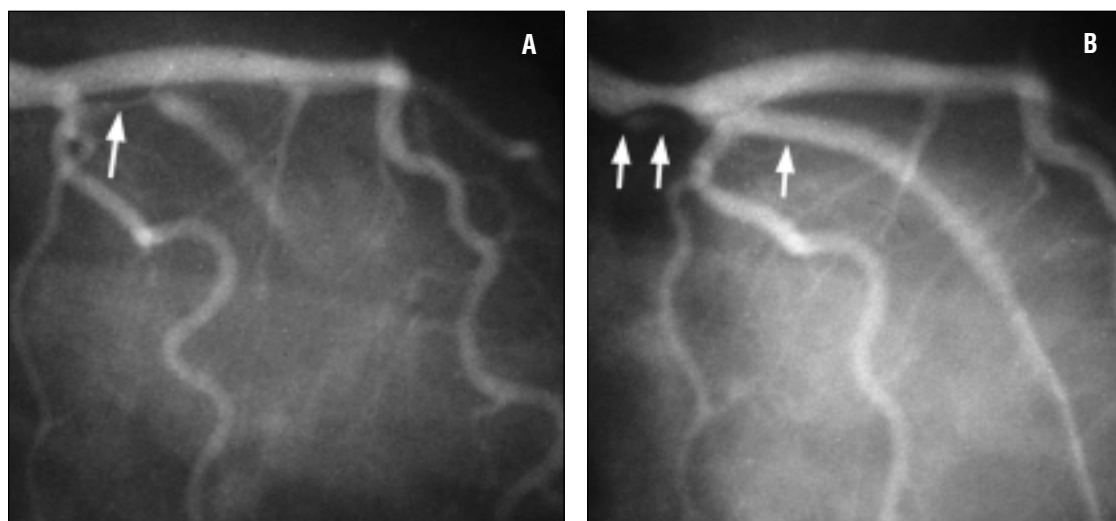


Figure 4. Angiograms taken 1 week following stent placement. (A) Angiogram showing severe stenosis (arrow) of the first obtuse marginal (OM1) branch of the left circumflex artery just proximal to the stent. (B) Angiogram showing a retrograde dissection of the left main artery (arrows) following deployment of the new stent at the proximal part of the OM1 vessel (arrow).

Our patient, however, was not in a peripartum period and was not taking oral contraceptives.

Most dissections have been described in young or middle-aged individuals with minimal or no cardiac risk factors. The clinical presentation of spontaneous dissections depends on the extent of dissection and the vessel involved. Sudden cardiac death is reported to occur in 50% of the cases, especially in those with left main coronary artery dissections.^{2,10} Other patients may present with angina or acute MI.

Once clinical suspicion is raised, a diagnosis can be established by coronary angiography. A high index of suspicion for such a condition should be entertained in young individuals who present with an acute coronary syndrome, especially if they are women without any conventional risk factors. The prognosis of patients with this condition depends on early diagnosis and performance of early revascularization procedures. Most studies support the use of CABG as the treatment of choice for patients with multivessel or left main coronary artery dissections.^{7,11}

Successful angioplasty and stenting of spontaneous dissections in both single-vessel and multivessel

Main Points

- Acute dissection of the coronary artery is a rare cause of acute coronary syndrome.
- Most cases occur in younger individuals with no cardiac risk factors.
- Sudden cardiac death can occur in a significant number of these patients.
- Early angiography is necessary to establish the diagnosis.
- Most patients are treated successfully with such revascularization procedures as percutaneous transluminal coronary angioplasty with stent deployment or coronary artery bypass grafting.

disease have been reported,^{12,13} although detecting the true lumen may be challenging.¹⁴ In our patient, the dissection progressed retrograde following stenting. ■

References

1. Falk E, Shah PK, Fuster V. Coronary plaque disruption. *Circulation*. 1995;92:657-671.
2. Basso C, Morgagni GL, Thiene G. Spontaneous coronary artery dissection: a neglected cause of acute myocardial ischaemia and sudden death. *Heart*. 1996;75:451-454.
3. Ciraulo DA, Chesne RB. Coronary arterial dissection: an unrecognized cause of myocardial infarction, with subsequent coronary arterial patency. *Chest*. 1978;73:677-679.
4. Smith JC. Dissecting aneurysm of coronary arteries. *Arch Pathol Lab Med*. 1975;99:117-121.
5. Bac DJ, Lotgering FK, Verkaaik AP, Deckers JW. Spontaneous coronary artery dissection during pregnancy and post partum. *Eur Heart J*. 1995; 16:136-138.
6. Azam MN, Roberts DH, Logan WF. Spontaneous coronary artery dissection associated with oral contraceptive use. *Int J Cardiol*. 1995;48:195-198.
7. Cohen DE, Strimike CL. A case of multiple spontaneous coronary artery dissections. *Catheter Cardiovasc Interv*. 2000;49:318-320.
8. Kearney P, Singh H, Hutter J, et al. Spontaneous coronary artery dissection: a report of three cases and review of the literature. *Postgrad Med J*. 1993;69:940-945.
9. Robinowitz M, Virmani R, McAllister HA Jr. Spontaneous coronary artery dissection and eosinophilic inflammation: a cause and effect relationship? *Am J Med*. 1982;72:923-928.
10. Adkins GF, Steele RH. Left coronary artery dissection: an unusual presentation. *Br Heart J*. 1986; 55:411-414.
11. Atay Y, Yagdi T, Turkoglu C, et al. Spontaneous dissection of the left main coronary artery: a case report and review of the literature. *J Card Surg*. 1996;11:371-375.
12. Togni M, Amann FW, Follath F. Spontaneous multivessel coronary artery dissection in a pregnant woman treated successfully with stent implantation. *Am J Med*. 1999;107:407-408.
13. Hanratty CG, McKeown PP, O'Keeffe DB. Coronary stenting in the setting of spontaneous coronary artery dissection. *Int J Cardiol*. 1998;67:197-199.
14. Klustein MW, Tzivoni D, Bitran D, et al. Treatment of spontaneous coronary artery dissection: report of three cases. *Cathet Cardiovasc Diagn*. 1997;40:372-376.