

A Case of Acute Myocardial Infarction Caused by Coronary Thrombus Associated with a Myocardial Bridge and Slow Coronary Flow

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ABSTRACT

Acute myocardial infarction is generally caused by the rupture or erosion of an atheromatous plaque and thrombosis. Acute myocardial infarction associated with a myocardial bridge or slow coronary flow is rare. We experienced a case of acute myocardial infarction, caused by a coronary thrombus in association with a myocardial bridge and slow coronary flow. A 33-year-old man presented with the sudden onset of chest pain. A diagnostic coronary angiography revealed an intraluminal contrast-filling defect, proximal to the myocardial bridge in the left anterior descending artery, with TIMI-2 flow. After an intracoronary injection of 150,000 units of urokinase and an intravenous injection of abciximab, the patient's chest pain subsided, with the follow-up coronary angiography showing the disappearance of the coronary thrombus. (*Korean Circulation J* 2005;35:639-642)

KEY WORDS : Myocardial infarction ; Thrombus ; Myocardial bridging ; Coronary flow.

Introduction

Acute myocardial infarction is generally caused by the rupture or erosion of an atheromatous plaque and thrombosis, but can also be caused, on rare occasions, by severe aortic stenosis, severe anemia, hyperthyroidism, congenital heart disease, coronary spasm, coronary embolism, aortitis, trauma, myocardial bridge or slow coronary flow, without any significant coronary atherosclerosis.¹⁻³⁾ There have been previous reports of acute myocardial infarction associated with a myocardial bridge or slow coronary flow,³⁻⁵⁾ but very few of acute myocardial infarction caused by a coronary thrombus associated with the combined effects of a myocardial bridge and slow coronary flow. The clinical significance of a myocardial bridge or slow coronary flow is not clear. In this report we describe the case of a 33-year-old man with acute myocardial infarction, caused by a coronary thrombus associated with a myocardial bridge and slow coronary flow.

Case

A 33-year-old man was admitted to our hospital due to resting chest pain of 30 minutes duration. He had no previous specific medical or family history. His coronary risk factors included tobacco use of 10 years duration. On admission, his blood pressure was 170/120 mmHg, pulse rate 78/min and body temperature 36.2°C. The physical examination revealed regular heart beats without gallop or murmur, clear breathing sound, no hepatomegaly and no pretibial pitting edema. The initial laboratory findings included a hemoglobin level of 16.1 g/dL, a white blood cell count of 9,000/ μ L and a platelet count of 274,000/ μ L. The blood chemistry results were glucose 121 mg/dL, BUN 14.3 mg/dL, creatinine 1.1 mg/dL, AST 41 IU/L and ALT 66 IU/L. The cardiac biomarkers included a creatinine kinase (CPK) level of 134 IU/L, with an MB fraction of 5.2 ng/mL, a lactate dehydrogenase (LDH) level of 410 IU/L and a negative troponin I test. His chest X-ray showed no abnormality. Electrocardiography (ECG) revealed regular sinus rhythm, right axis deviation and an elevated ST segment in leads V1 to V4 (Fig. 1). After a diagnosis of hyperacute myocardial infarction of the anteroapical wall, aspirin, clopidogrel, ramipril, atenolol, heparin and nitrate (4 mg/hr i.v.) were administered. Echocardiography showed akinesia of the anterior wall, without intracardiac

Received : March 14, 2005

Accepted : May 3, 2005

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thrombus, and a slightly decreased ejection fraction of 50%. A diagnostic coronary angiography was performed 1 hour and 30 minutes after the onset of chest pain. The coronary angiography was remarkable for a myocardial bridge in the middle left anterior descending artery (LAD), with an intracoronary contrast-filling defect, proximal to the myocardial bridge, which was suggestive of an intracoronary thrombus (Fig. 2A). We also noted a significantly slow antegrade flow in the LAD, with TIMI-2 flow (TIMI frame count of 59), in the left circumflex artery (TIMI frame count of 53) and right coronary artery (TIMI frame count of 49). 150,000 units of urokinase were injected intracoronarily and 20 mg of abciximab intravenously by bolus, followed by an infusion of 10 μ g/min for 12 hours. After coronary angiography, the cardiac biomarkers were CPK 2946 IU/L, with an MB fraction of 68.7 ng/mL, LDH 1273 IU/L and a positive troponin I test. Coagulation tests revealed no abnormality (the D-dimer level was 31 μ g/dL, the antithrombin III was 104.4%, the fibrinogen level was 278.6 mg/dL, the fibrinogen degradation product test was negative, the protein C activity was 99% and protein S activity was 82%). Subsequently, the patient's

chest pain gradually improved. Three days after admission, the ST segment had normalized, and the Q wave appeared on his ECG. One week after admission, a follow-up coronary angiography revealed the disappearance of the intracoronary contrast-filling defect (Fig. 2B), with persistence of the myocardial bridge (Fig. 3A, B) and slow flow. Intravascular ultrasound (IVUS) was performed, which verified the systolic compression of the LAD, with no evidence of atherosclerosis (Fig. 3C, D). Twelve days after admission, the patient was discharged without symptom, and has been followed up at the out-patient department.

Discussion

A myocardial bridge is characterized by temporary systolic compression of the intramyocardial segment of an epicardial coronary artery, and was first described by Reyman in 1737.⁶⁾ The prevalence of this malady has varied from 5 to 85% in different studies.⁷⁻⁹⁾ Its occurrence is higher in patients with hypertrophic cardiomyopathy. The middle portion of the LAD is most commonly involved,¹⁰⁾ but the myocardial bridge usually has a benign

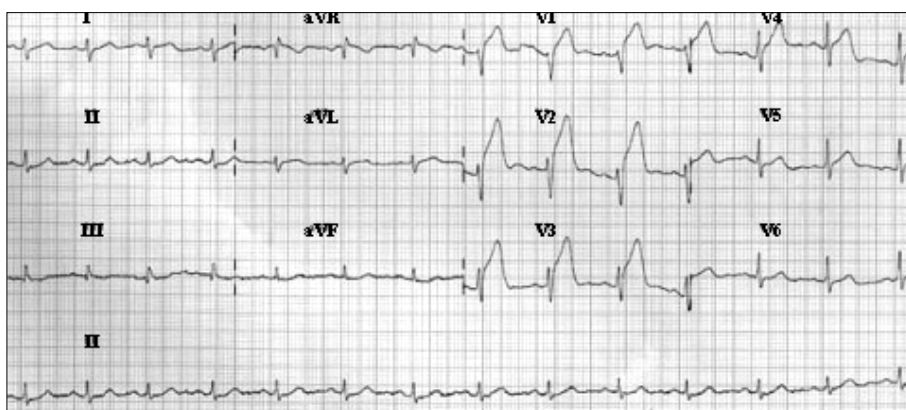


Fig. 1. Electrocardiography on admission shows regular sinus rhythm and ST segment elevation in precordial leads V1 to V4.

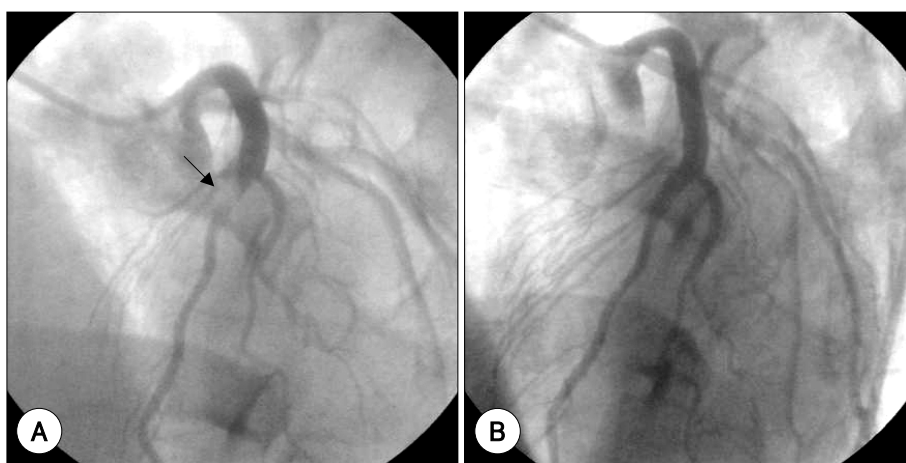


Fig. 2. A: angiography of the left coronary artery on admission shows a large contrast-filling defect (arrow) in the middle left anterior descending artery (LAD). B: angiography of the left coronary artery, seven days after thrombolytic therapy, shows the disappearance of the contrast-filling defect in the LAD.

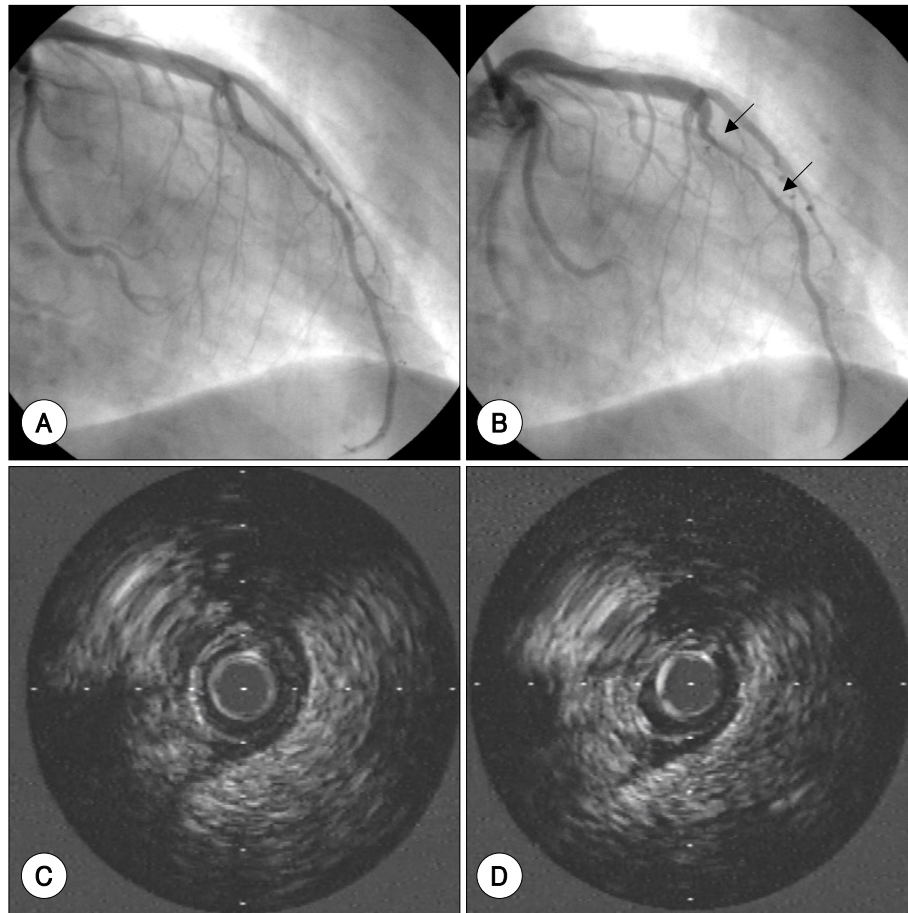


Fig. 3. A and B, angiography of the left coronary artery shows systolic compression of the middle left anterior descending artery (LAD) (between the arrows). A: diastole, B: systole, C: diastole, D: systole. C and D: intravascular ultrasound images show systolic compression of the LAD, which is suggestive of a myocardial bridge.

prognosis, and can result in myocardial infarction, arrhythmias, left ventricular dysfunction and sudden cardiac death in some cases.¹⁰⁻¹²⁾ With this condition, the coronary diameter is significantly reduced not only during systole, but also during diastole, and the coronary flow reserve distal to the bridge is reduced.¹³⁾ However, the pathophysiology of myocardial ischemia due to a myocardial bridge remains unclear.

The slow coronary flow phenomenon is an angiographic finding, characterized by delayed distal vessel opacification, but without significant coronary stenosis, and was first described by Tambe in 1972.¹⁴⁾ Its incidence is 7% in patients with typical angina.¹⁵⁾ Patients with slow coronary flow are often men and current smokers, and usually present with an acute coronary syndrome.³⁾ The suggested pathophysiology is associated with microvascular dysfunction, a dynamic increase of microvascular tone,¹⁶⁾ endothelial dysfunction¹⁷⁾ and diffuse intimal thickening,¹⁸⁾ but the exact mechanism remains unclear. Slow coronary flow may respond to oral dipyridamole or sublingual nifedipine due to a decrease in the microvascular resistance,¹⁹⁾²⁰⁾ but no definite therapy has been established.

In this case, it is thought that the acute myocardial infarction was caused by an intracoronary thrombus associated with the combined effects of the myocardial bridge and the slow coronary flow. In case of a recurrence of acute coronary syndrome associated with a myocardial bridge, stent implantation can be considered as an optional treatment for the myocardial bridge.

REFERENCES

- 1) Antman EM, Braunwald E. *Acute myocardial infarction*. Braunwald E, Zipes DP, Libby P, editors. *Heart Disease: a textbook of cardiovascular medicine*. Philadelphia: Saunders company; 2001. p.1115-23.
- 2) Berry JF, von Mering GO, Schmalfuss C, Hill JA, Kerensky RA. *Systolic compression of the left anterior descending coronary artery: a case series, review of the literature, and therapeutic options including stenting*. *Catheter Cardiovasc Interv* 2002;56:58-63.
- 3) Beltrame JF, Limaye SB, Horowitz JD. *The coronary slow flow phenomenon: a new coronary microvascular disorder*. *Cardiology* 2002;97:197-202.
- 4) Bauters C, Chmait A, Tricot O, Lamblin N, van Belle E, Lablanche JM. *Images in cardiovascular medicine: coronary thrombosis and myocardial bridging*. *Circulation* 2002;105:130.
- 5) Lee JH, Bae Y, Lee HS, et al. *A case of myocardial infarction in a patient with myocardial bridge and atrial fibrillation*. *Korean*

- Circ J* 2004;34:319-22.
- 6) Reyman HC. *Disertatio de vasis cordis propriis*. Med Diss Univ Goettingen 1737;7:1-32.
 - 7) Edward JC, Burnsides C, Lansing AI, Swarm RL. *Arteriosclerosis in the intramural and extramural portions of coronary arteries in the human heart*. *Circulation* 1956;13:235-41.
 - 8) Polacek P, Kralove H. *Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions*. *Am Heart J* 1961;61:44-52.
 - 9) Cho KH, Rhee I, Jung JY, et al. *Clinical study of myocardial bridge*. *Korean Circ J* 2001;31:311-6.
 - 10) Mohlenkamp S, Hort W, Ge J, Erbel R. *Update on myocardial bridging*. *Circulation* 2002;106:2616-22.
 - 11) Endo M, Lee YH, Hayashi J, Wada J. *Angiographic evidence of myocardial squeezing accompanying tachyarrhythmia as a possible cause of myocardial infarction*. *Chest* 1978;73:431-3.
 - 12) Gow RM. *Myocardial bridging: does it cause sudden death?* *Card Electrophysiol Rev* 2002;6:112-4.
 - 13) Schwarz ER, Klues HG, von Dahl J, Klein I, Krebs W, Hanrath P. *Functional, angiographic, and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication*. *J Am Coll Cardiol* 1996;27:1637-45.
 - 14) Tambe AA, Demany MA, Zimmermann HA, Mascarenhas E. *Angina pectoris and slow velocity of dye in coronary arteries: a new angiographic finding*. *Am Heart J* 1972;84:66-71.
 - 15) Mangieri E, Macchiarelli G, Ciavolella M, et al. *Slow coronary flow: clinical and histopathological features in patients with otherwise normal epicardial coronary arteries*. *Cathet Cardiovasc Diagn* 1996;37:375-81.
 - 16) Beltrame JF, Limaye SB, Wuttke RD, Horowitz JD. *Coronary hemodynamic and metabolic studies of the coronary slow flow phenomenon*. *Am Heart J* 2003;146:84-90.
 - 17) Sezgin AT, Sigirci A, Barutcu I, et al. *Vascular endothelial function in patients with slow coronary flow*. *Coron Artery Dis* 2003;14:155-61.
 - 18) Cin VG, Pekdemir H, Camsar A, et al. *Diffuse intimal thickening of coronary arteries in slow coronary flow*. *Jpn Heart J* 2003;44:907-19.
 - 19) Kurtoglu N, Akcay A, Dindar I. *Usefulness of oral dipyridamole therapy for angiographic slow coronary flow*. *Am J Cardiol* 2001;87:777-9.
 - 20) Kapoor A, Goel PK, Gupta S. *Slow coronary flow: a cause for angina with ST segment elevation and normal coronary arteries*. *Int J Cardiol* 1998;67:257-61.