

중간정도 협착을 보이는 관상 동맥에서 발생한 급성심근경색

서울대학교 의과대학 보라매병원 ¹내과, ²영상의학과, ³서울대학교 의과대학 내과학교실

황도연^{1,3} · 김학령^{1,3} · 박찬순^{1,3} · 이현정^{1,3} · 진광남^{2,3} · 조주희^{1,3}

Acute Myocardial Infarction Occurring at a Preexisting Intermediate Coronary Artery Stenosis

Doyeon Hwang^{1,3}, Hack-Lyoung Kim^{1,3}, Chan-Soon Park^{1,3}, Hyun-Jung Lee^{1,3}, Kwang-Nam Jin^{2,3}, and Joo-Hee Zo^{1,3}

*Departments of ¹Internal Medicine and ²Radiology, Seoul National University Boramae Medical Center,
Seoul National University College of Medicine, Seoul;*

³Department of Internal Medicine, Seoul National University College of Medicine, Seoul, Korea

Acute myocardial infarction often evolves from a mild coronary lesion. Therefore, the evaluation and management of intermediate coronary stenosis are important to prevent cardiac events. However, the decision on how to treat these lesions is challenging. Here, we report acute myocardial infarction occurring at a preexisting intermediate coronary stenosis based on invasive coronary angiography performed 10 days before the event. (Korean J Med 2015;89:448-451)

Keywords: Coronary stenosis; Myocardial infarction

INTRODUCTION

Coronary artery disease (CAD) is a leading cause of mortality worldwide. Invasive coronary angiography (ICA) is the gold standard in the diagnosis of CAD. However, when ICA reveals an intermediate coronary stenosis, the decision on how to treat this lesion is challenging [1]. Importantly, many cases of acute myocardial infarction (AMI) are believed to occur in mild coronary stenosis. However, angiographic detection of vulnerable plaque in a lesion with mild luminal narrowing is difficult, and

little is known about the progression time from mild or intermediate stenosis to an occlusive lesion causing a coronary event. Here, we describe a patient who suffered AMI because of a lesion with intermediate coronary stenosis, as confirmed by ICA 10 days before the AMI.

CASE REPORT

A previously healthy 59-year-old man visited our outpatient clinic with a 2-week history of anterior chest pain. The chest

Received: 2014. 11. 18

Revised: 2015. 4. 29

Accepted: 2015. 5. 19

Correspondence to Hack-Lyoung Kim, M.D., Ph.D.

Division of Cardiology, Department of Internal Medicine, Seoul National University Boramae Medical Center, Seoul National University College of Medicine, 20 Boramae-ro 5-gil, Dongjak-gu, Seoul 07061, Korea
Tel: +82-2-870-3235, Fax: +82-2-831-0714, E-mail: khl2876@gmail.com

Copyright © 2015 The Korean Association of Internal Medicine

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted noncommercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

pain occurred regardless of his activity, was triggered by position change, persisted for 30 seconds, and was then relieved spontaneously. There were no accompanying symptoms like dyspnea, fever, cough, or chills. He had no history of hypertension, diabetes mellitus, or dyslipidemia. He was a 40-pack-year ex-smoker. His vital signs were stable (blood pressure, 100/60 mmHg; heart rate, 67 beats/minute; body temperature, 36.4°C), and laboratory test results were within normal ranges, including a fasting glucose of 96 mg/dL and a low-density lipoprotein (LDL) cholesterol of 98 mg/dL. Chest radiographs and electrocardiograms were normal. Transthoracic echocardiography showed good left ventricular systolic function without regional wall motion abnormalities. Coronary computed tomography angiography (CCTA) revealed mild discrete stenosis (30%) with calcified plaques in the proximal left anterior descending coronary artery (LAD) and mild discrete stenosis (30%) with non-calcified plaques in the proximal left circumflex coronary artery (LCX). The right coronary artery (RCA) was normal. There was no evidence of aortic dissection or pulmonary embolism on CCTA. Gastroduodenoscopy showed no noteworthy finding, except superficial gastritis. Based on these results, the chest pain was considered nonspecific and he was followed without medication.

During follow-up, his chest pain worsened slightly. During an exercise treadmill test, there was no chest pain, but there was 2-mm horizontal ST-segment depression in leads II, III, aVF, and V₄ to V₆ at stage 2 of the Bruce protocol (Fig. 1). ICA was performed based on the exercise treadmill test result and persis-

tent chest pain. There was an intermediate focal stenosis (stenosis diameter of 49% by quantitative coronary angiography) in the proximal LCX. The LAD looked normal, and the RCA was hypoplastic without stenosis (Fig. 2). Since the proximal LCX lesion was considered insignificant, no further testing or intervention was performed. After ICA, aspirin 100 mg was prescribed, and he was discharged from the hospital.

Ten days later, he visited our emergency department with severe anterior chest pain at rest that persisted for more than 30 minutes. His blood pressure was 114/69 mmHg and pulse rate was 62 per minute. An electrocardiogram showed markedly elevated ST-segments in leads II, III, and aVF, with reciprocal ST-segment depression in leads V₁ to V₄ (Fig. 3). Emergency ICA revealed total thrombotic occlusion in the proximal LCX (Fig. 4A). First, a thrombectomy was performed using aspiration

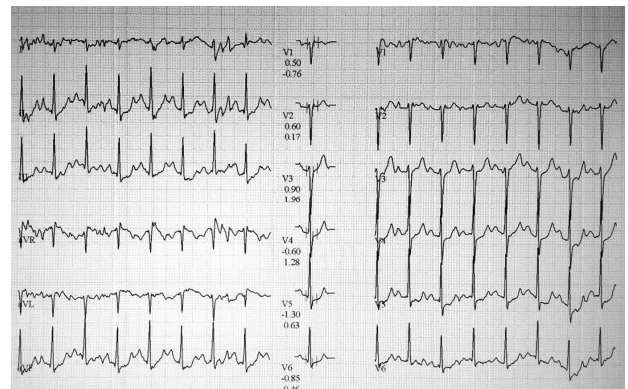


Figure 1. Electrocardiographic changes at the Bruce protocol stage 2 of the treadmill exercise test.

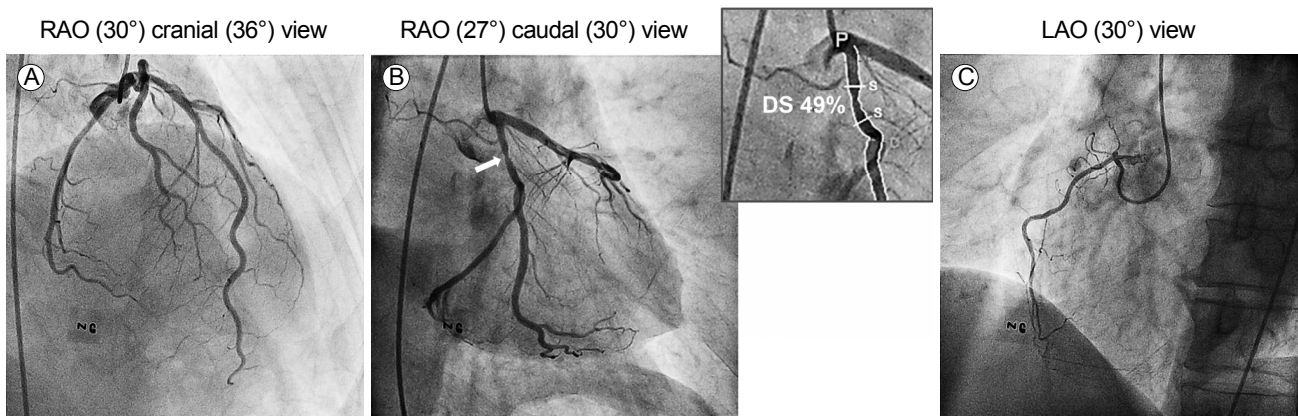


Figure 2. Invasive coronary angiography showing (A) no significant luminal stenosis in the left anterior descending artery, (B) intermediate stenosis in the proximal left circumflex artery (white arrow), and (C) a hypoplastic right coronary artery without stenosis. RAO, right anterior oblique; LAO, left anterior oblique; NG, nitroglycerin; DS, diameter stenosis; P, proximal; S, stenosis; D, distal.

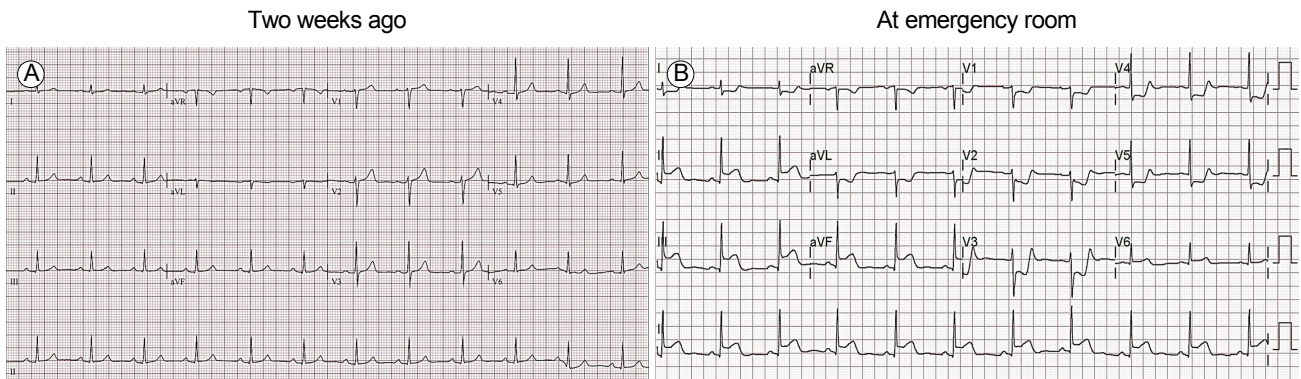


Figure 3. Electrocardiographic changes from normal findings before presentation (A) to acute myocardial infarction in the emergency department (B).

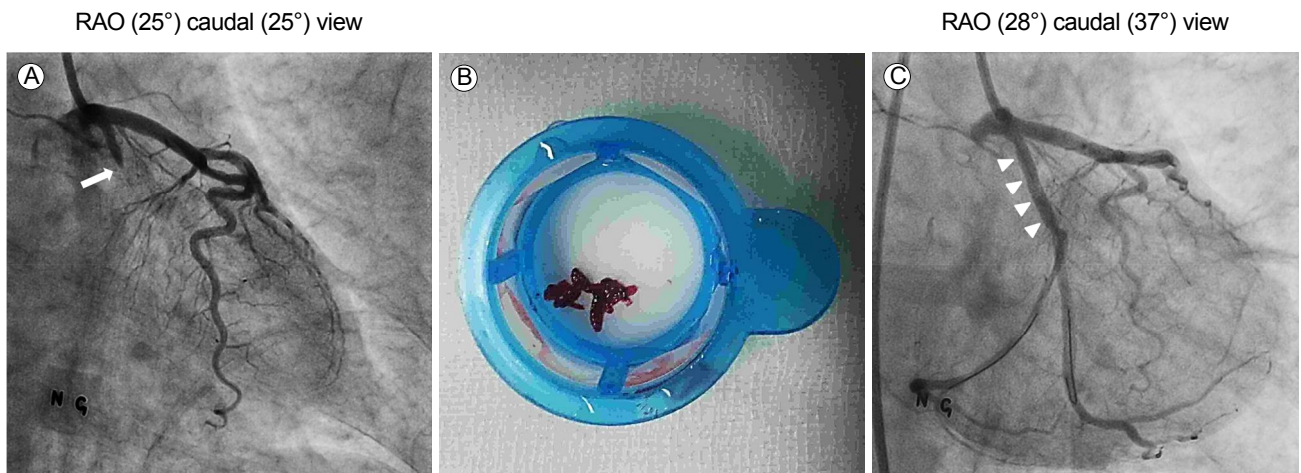


Figure 4. Percutaneous coronary intervention procedure. Angiograms showing (A) a totally occluded proximal portion of the left circumflex artery, (B) aspirated thrombi, and (C) the successfully implanted stent (white arrowheads). RAO, right anterior oblique; NG, nitroglycerin.

catheters, and much thrombi was aspirated (Fig. 4B). Then, a drug-eluting stent (3.0 × 18 mm, Resolute Integrity; Medtronic, Minneapolis, MN, USA) was implanted (Fig. 4C). He was stable after the procedure and discharged from the hospital 5 days later. Subsequently, his chest pain has not recurred.

DISCUSSION

ICA is the gold standard for evaluating coronary status. It provides information about the presence, location, and severity of CAD with excellent spatial and temporal resolution, and offers immediate therapeutic intervention. However, it is difficult to evaluate to decide on revascularization in an intermediate coronary lesion, defined as 40% to 70% stenosis of the coronary lu-

menal diameter [1]. In particular, the anatomical assessment of luminal narrowing by ICA provides no insight into the vessel wall characteristics, including plaque deposition and morphology. Since AMI sometimes originates from a mild or intermediate coronary lesion, it is important to predict which coronary angiographic lesion is likely to cause AMI [2]. In order to overcome these limitations of ICA, the fractional flow reserve (FFR) or intravascular ultrasound (IVUS) during intervention has been used to predict whether a lesion will cause symptoms and to decide on how to manage the lesion [3]. In our case, the atypical nature of the chest pain and insignificant CCTA results made us overlook an intermediate stenosis in the proximal LCX on ICA, although the exercise treadmill test was positive. If FFR or IVUS was performed at the initial ICA, hemodynamic significance or

unstable plaque characteristics may have been revealed. Therefore, it is possible that AMI could be prevented by more appropriate management.

The failure to administer optimal medical therapy to patients with intermediate coronary stenosis is an important issue. With advances in medical therapy, current medical treatment is very effective and has outcomes comparable to those of drug-eluting stents [3,4]. Statins are powerful cholesterol-lowering agents with pleiotropic effects. Statin therapy reduces cholesterol levels, induces the regression of coronary atherosclerosis, and stabilizes coronary plaques, improving patient outcomes [5]. Inhibitors of the renin-angiotensin system and beta-blockers also reduce atherosclerosis and its complications, such as myocardial infarction [6,7]. In our case, however, the use of these cardioprotective drugs was difficult because of the patient's low blood pressure and low LDL cholesterol levels.

Several noninvasive cardiac tests have been used to evaluate CAD. However, a single test often has low diagnostic value. Further tests are frequently needed to improve diagnostic accuracy. Although CCTA has been considered an accurate diagnostic tool for detecting CAD, it provides no insight into the hemodynamic significance of luminal stenosis [8]. In our case, although CCTA revealed a non-calcified coronary plaque in the proximal LCX, the lesion did not receive attention due to the mild luminal narrowing. However, the exercise treadmill test result suggested myocardial ischemia. Our case shows the limitation of a single test and emphasizes the importance of additional tests, especially when the initial test results are inconclusive or do not match the clinical findings.

It is possible that vasospasm caused plaque rupture and thrombosis [9] in the LCX lesion. However, the clinical presentation of the chest pain was incompatible with variant angina in our case. Consequently, we did not perform a confirmatory provocation test during the initial ICA.

In conclusion, our case shows that AMI can occur after plaque rupture at a preexisting intermediate stenosis. Additional

noninvasive diagnostic tests should be considered, especially in patients with ambiguous results of the initial test. In addition, FFR or IVUS evaluations during ICA, as well as subsequent optimal medical therapies, should be considered in patients with intermediate coronary stenosis.

중심 단어: 관동맥 협착, 급성심근경색

REFERENCES

1. Tobis J, Azarbal B, Slavin L. Assessment of intermediate severity coronary lesions in the catheterization laboratory. *J Am Coll Cardiol* 2007;49:839-848.
2. Bentzon JF, Otsuka F, Virmani R, Falk E. Mechanisms of plaque formation and rupture. *Circ Res* 2014;114:1852-1866.
3. Hong YJ, Choi YH, Park SY, et al. Clinical outcomes in patients with intermediate coronary stenoses: MINIATURE Investigators (Korea Multicenter Trial on Long-Term Clinical Outcome According to the Plaque Burden and Treatment Strategy in Lesions with Minimum Lumen Area Less Than 4 mm²) Using Intravascular Ultrasound). *Korean Circ J* 2014;44:148-155.
4. De Bruyne B, Pijls NH, Kalesan B, et al. Fractional flow reserve-guided PCI versus medical therapy in stable coronary disease. *N Engl J Med* 2012;367:991-1001.
5. Rosa GM, Carbone F, Parodi A, et al. Update on the efficacy of statin treatment in acute coronary syndromes. *Eur J Clin Invest* 2014;44:501-515.
6. Liang C, Xiaonan L, Xiaojun C, et al. Effect of metoprolol on vulnerable plaque in rabbits by changing shear stress around plaque and reducing inflammation. *Eur J Pharmacol* 2009;613:79-85.
7. Hotchi J, Hoshiga M, Takeda Y, et al. Plaque-stabilizing effect of angiotensin-converting enzyme inhibitor and/or angiotensin receptor blocker in a rabbit plaque model. *J Atheroscler Thromb* 2013;20:257-266.
8. Patel MR, Peterson ED, Dai D, et al. Low diagnostic yield of elective coronary angiography. *N Engl J Med* 2010;362:886-895.
9. Wang LX, Lü SZ, Zhang WJ, Song XT, Chen H, Zhang LJ. Coronary spasm, a pathogenic trigger of vulnerable plaque rupture. *Chin Med J (Engl)* 2011;124:4071-4078.