Pure Ventricular Septal Myocardial Infarction in a Young Man With Coronary Artery Ectasia

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SUMMARY

The case of a 30-year-old man with myocardial infarction localized in the interventricular septum is described. Coronary angiography performed on day 28 after the onset of symptoms revealed ectasia in the right and left coronary arteries, but no overt stenotic or occlusive lesions were present. Spasm was induced in the first septal branch of the left anterior descending artery by an acetylcholine provocation test, and single photon emission computed tomography myocardial perfusion imaging showed a reduced thallium-201 uptake localized in the interventricular septum. (Int Heart J 2006; 47: 131-137)

Key words: Coronary artery ectasia, Coronary vasospasm, Pure septal infarction

Pure septal infarction is a rare type of myocardial infarction,1) and is often difficult to diagnose from variations in the ECG patterns.2) On the other hand, there is a high incidence of vasospastic angina in the Japanese population and coronary vasospasm is thought to be heavily involved in the pathogenesis of acute myocardial infarction.3,4) Here, we report our experience of a rare case of pure septal infarction that may have been caused by coronary vasospasm provoked in the first septal branch of the ectatic left anterior descending artery in a 30-year-old man.

CASE REPORT

The patient was a 30-year-old man with hypercholesterolemia who was a heavy smoker, had no history of collagen disease or Kawasaki disease, and no family history of coronary artery disease. The plasma levels of total cholesterol and low-density lipoprotein cholesterol were 294 and 160 mg/dL, respectively, but he had no history of familial hypercholesterolemia. He presented to our hos-
hospital because of chest pain that had lasted for about 1 hour. On arrival, the symptoms had disappeared and an ECG on admission revealed ST elevation in V₁ and V₂ and ST depression in aVL, but none of these changes was marked. ECG performed about 5 hours after admission showed reduced R-wave amplitude in V₁ and V₂, and terminal T-wave inversion, in addition to the above changes. ECG 19 days after onset showed persistent ST elevation in V₁ and V₂, and terminal T-wave inversion, but ST depression in aVL returned to baseline. Septal Q waves normally present in V₅ and V₆, however, were absent from the time of hospital admission.

Figure 1. Time course of electrocardiography.
ECG on admission revealed ST elevation in V₁ and V₂ and ST depression in aVL, but none of these changes was marked. ECG performed about 5 hours after admission showed reduced R-wave amplitude in V₁ and V₂, and terminal T-wave inversion, in addition to the above changes. ECG 19 days after onset showed persistent ST elevation in V₁ and V₂, and terminal T-wave inversion, but ST depression in aVL returned to baseline. Septal Q waves normally present in V₅ and V₆, however, were absent from the time of hospital admission.

Figure 2. SPECT myocardial perfusion imaging at 25 days after admission.
A reduced TI-201 uptake is seen in the septal territory from the basal through middle segments of the left ventricle (A: short axis, B: sagittal axis).

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seen on either the chest X-ray or ECG. The WBC count, CK, and CK-MB levels peaked at 12,000/mm³, 1,250 IU/L, and 125 IU/L, respectively, and returned to within almost normal ranges at 24 hours after hospitalization. Viral testing of paired serum samples showed no significant changes. Single photon emission computed tomography (SPECT) myocardial perfusion imaging demonstrated a reduced thallium (TI)-201 uptake in the septal territory from the basal through middle segments of the left ventricle (Figure 2).

Coronary angiography was performed on day 28 after the onset of symptoms. No significant organic stenosis was found in either the right or left coronary arteries, but coronary artery ectasia was noted in the proximal and middle segments of the left anterior descending artery as well as the right coronary artery.

**Figure 3.** Acetylcholine provocation test and intravascular ultrasound image of coronary artery ectasia in left anterior descending artery. 

A: Intracoronary injection of acetylcholine (50 µg) provoked 99% spasm in the ventricular septal branch of the left anterior descending artery (left anterior oblique). Arrow indicates ventricular septal branch. B: Coronary spasm was resolved by intracoronary injection of isosorbide dinitrate. Coronary artery ectasia is present from the proximal through middle segments of the left anterior descending artery. IVUS demonstrates dilatation of the luminal diameter and the scattered presence of intimal thickening (Arrows indicate intimal thickening).
When an acetylcholine provocation test was performed, no spasm occurred in the right coronary artery, but spasm was provoked in the first septal branch of the left anterior ascending artery (Figure 3A). Intravascular ultrasound images of the ecstatic lesions showed dilatation with the maximum luminal diameter of 6.9 mm and the presence of intimal thickening in a number of places (Figure 3B a-d). Left ventriculography revealed wall motion abnormalities with dyskinesis localized in the interventricular septum (Figures 5A-D). Based on

(Figure 3B; Figure 4). When an acetylcholine provocation test was performed, no spasm occurred in the right coronary artery, but spasm was provoked in the first septal branch of the left anterior ascending artery (Figure 3A). Intravascular ultrasound images of the ecstatic lesions showed dilatation with the maximum luminal diameter of 6.9 mm and the presence of intimal thickening in a number of places (Figure 3B a-d). Left ventriculography revealed wall motion abnormalities with dyskinesis localized in the interventricular septum (Figures 5A-D). Based on

(Figure 4). Angiographic findings of left and right coronary arteries. A: Left anterior descending artery (right anterior oblique) B: Right coronary artery (left anterior oblique)

Figure 5. Left ventriculography. A, B: Right anterior oblique. C, D: Left anterior oblique. Wall motion of the interventricular septum shows dyskinesis in the systolic phase. Arrows indicate wall motion abnormality.

A B

C D

Figure 4. Angiographic findings of left and right coronary arteries. A: Left anterior descending artery (right anterior oblique) B: Right coronary artery (left anterior oblique)
these findings, a diagnosis of myocardial infarction possibly associated with coronary vasospasm of the septal branch was made.

**DISCUSSION**

Although the incidence of coronary artery ectasia reported in the past varies, it is generally estimated to be from 0.3% to 4.7% of patients with coronary artery disease.\(^4\) Atherosclerosis is involved in the pathogenesis of coronary artery ectasia, and reports have indicated that it is an unusual manifestation of atherosclerosis including dysplasia of the tunica media.\(^5\)-\(^7\) On the other hand, it has been also reported to be associated with syphilis,\(^8\) congenital heart disease,\(^9\) polyarteritis nodosa,\(^10\) scleroderma,\(^11\) Ehlers-Danlos syndrome,\(^12\) bacterial infections,\(^13\) familial hypercholesterolemia,\(^14\) and hypertension.\(^15\) As with the intravascular ultrasound images of the present case, the intravascular ultrasound images in two other reports on coronary artery ectasia indicate the presence of intimal thickening, suggesting an association with atherosclerosis.\(^14,16\)

A correlation between coronary artery ectasia and coronary vasospasm has also been reported\(^17,18\) and a review article by Sorrell, et al recommended that patients with coronary artery ectasia should be treated with calcium channel blockers to prevent coronary vasospasm, in addition to warfarin and aspirin to prevent thrombus formation.\(^19\) The majority of cases of coronary artery spasm occur within or around the ectatic site of the epicardial artery,\(^17\) and coronary vasospasm occurring in an area far beyond the ectatic site, as in our case, is thought to be rare.

The incidence of vasospastic angina is higher in Japanese patients than in Caucasian patients, and coronary artery spasm plays an important role as a trigger of acute coronary syndrome. Pristipino, et al employed the acetylcholine provocation test in patients within 14 days after myocardial infarction and showed that Japanese patients had a higher incidence of coronary spasm than Caucasians in not only infarct-related coronary arteries but also noninfarct-related coronary arteries.\(^3\) Akiyama, et al demonstrated that coronary vasoconstrictor response was increased in the acute phase of myocardial infarction, and coronary spasm was heavily involved in its onset.\(^4\) The higher incidence of vasospastic angina in Japanese versus Caucasians may be associated with differences in coronary risk factors, genetic predisposition, autonomic balance, and the severity of endothelial dysfunction between these populations.\(^20-22\) There have also been reports of a number of myocardial infarction cases directly caused by coronary spasm.\(^23,24\)

In clinical practice, cases of vasospastic angina that can be diagnosed and treated are usually caused by spasm occurring in the epicardial arteries that run along the epicardium. In fact, coronary vasospasm occurring in the ventricular
septal branch, in which the myocardial perfusion area is relatively small, is unlikely to become a clinical problem. To date, Azuma, et al have reported a case of coronary vasospasm occurring only in the septal branch,\textsuperscript{25} while our database research indicates that several cases of myocardial infarction localized in the interventricular septal region have been reported.\textsuperscript{26-29} However, ours is a rare case of myocardial infarction apparently caused by spasm at the angiographically normal first septal branch of the ectatic left anterior descending artery in a young patient.

There are variations in the ECG patterns for pure septal infarction, and its diagnosis based on the standard 12-lead ECG is recognized to be difficult.\textsuperscript{1} ECG patterns reported are ST depression in limb leads, ST elevation in V\textsubscript{1}, V\textsubscript{2}, and V\textsubscript{3}, ST depression in V\textsubscript{5} and V\textsubscript{6}, and the absence of a septal Q wave in V\textsubscript{5} and V\textsubscript{6}.\textsuperscript{29} but all of these are minor changes and can be easily missed. The present case exhibited ST elevation in V\textsubscript{1} and V\textsubscript{2}, followed by terminal T-wave inversion, ST depression in aVL, and the absence of a septal Q wave.

In summary, we experienced a case of pure septal myocardial infarction possibly due to coronary vasospasm in a 30-year patient with coronary artery ectasia.

\textbf{REFERENCES}

8. Denham SW. Syphilitic aneurysm of the left coronary artery. AMA Arch Pathol 1951; 51: 661-5.