Ischemia in the Territory of the First Major Septal Perforator Branch Anomalously Originating From the First Diagonal Branch Leads to a Transient Leftward Shift of the QRS Axis in the Frontal Plane

— A Case Report —

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A patient with non-Q wave myocardial infarction had severe luminal narrowing in the first major septal perforator branch, which arose anomalously from the first diagonal branch. In this case, an exercise ECG showed a transient leftward QRS axis shift. (*Circ J* 2003; 67: 885–888)

Key Words: Anomalous origin; First major septal perforator branch; Left anterior descending coronary artery; Leftward QRS axis shift

Origination of the first major septal perforator branch from a site other than the left anterior descending coronary artery (LAD) is a relatively rare and benign congenital variation¹. Coronary angiography reveals anomalous vessels originating directly from the aorta and from the proximal portion of the right coronary artery as collateral circulation to the LAD, particularly in patients with proximal LAD disease^{2,3}. Anomalous vessels also arise from the proximal circumflex coronary artery, diagonal branch, or obtuse marginal branch⁴.

A transient leftward QRS axis shift on the electrocardiogram (ECG) during anginal attacks is a specific predictor of proximal LAD disease^{5–8}. Conduction disturbance caused by ischemia of the anterior fascicle of the left bundle branch, which is supplied by the first major septal perforator branch of the LAD⁹, is thought to be one reason for the axis shift.

Here, we describe a patient with a non-Q wave myocardial infarction whose coronary arteriogram documented severe luminal narrowing in the first major septal perforator branch arising anomalously from the first diagonal branch. Exercise ECG showed a transient leftward QRS axis shift.

Case Report

A 61-year-old man was referred because of anterior chest pain that started 4 h before admission. His medical history included stable angina pectoris on exertion, hyper-

tension, and gout. The patient's physical examination on admission was unremarkable, except for a high systemic blood pressure of 162/108 mmHg. A radiograph of the chest taken in the supine position showed a cardiothoracic ratio of 62% and no pulmonary congestion. The ECG demonstrated ST-segment elevation in leads V_{1–4} and ST-segment depression in leads I, aV_L, V₅, and V₆. Diphasic or negative T waves in leads I and aV_L, and in leads V_{2–6} were also found.

The patient was treated with isosorbide dinitrate (40 mg/day), bisoprolol (2.5 mg/day), and aspirin (162 mg/day). Heparin (10,000 U/day) was also administered intravenously. Creatine kinase, myosin light chain I, and troponin T peaked at 472 IU/L, 9.8 ng/ml, and 2.38 ng/ml, respectively. On the 21st hospital day, he underwent cardiac catheterization. The coronary arteriogram documented severe luminal narrowing in the proximal LAD, first diagonal branch, which originated from the site of just LAD lesion, and first septal perforator branch originating from the first diagonal branch. The left ventriculogram disclosed hypokinesis in the anterolateral, septal, and apical regions with an ejection fraction of 40%.

On the 36th hospital day, percutaneous coronary angioplasty (PCI) of the proximal LAD and first diagonal branch was performed. An 8F Judkins-type guide catheter was inserted into the left coronary artery, and an intracoronary injection of isosorbide dinitrate was administered before PCI. After baseline angiography, two 0.014-inch guide wires were advanced through the stenotic lesions in the proximal LAD and the first diagonal branch. First, a 3.5×20-mm balloon catheter was placed within the proximal LAD lesion, and the balloon was inflated for 1 min at 10 atmospheres, the pressure at which the balloon indentation disappeared. Second, a 2.0×20-mm non-compliant balloon catheter was placed within the stenosis in the first diagonal branch, and the balloon was inflated at a pressure

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Fig 1. ECG before (Left) and during (Right) coronary angioplasty of the proximal left anterior descending coronary artery and first diagonal branch.

Fig 2. Right (left panel) and left (right panel) anterior oblique views of the follow-up left coronary angiogram performed 6 months after coronary angioplasty show restenosis in the first diagonal branch (white arrows). The ostium of the first septal perforator branch, which arises anomalously from the first diagonal branch, is also markedly stenotic (arrowheads). Black arrows indicate the left anterior descending coronary artery.

of 12 atm for 1 min. Third, using the 'kissing balloon' technique with the same balloon catheters, the lesions in the proximal LAD and first diagonal branch were dilated simultaneously. Subsequently, a coronary stent 3.5 mm in diameter and 20 mm in length was deployed in the proximal LAD. Post-dilatation using a 3.5×20-mm high-pressure balloon catheter was performed at the maximum inflation pressure of 15 atm. An immediate postangioplasty angiogram showed residual luminal narrowing of 0% in the proximal LAD, and 40% in the first diagonal branch. The heart rate and QRS axis in the frontal plane before PCI were 56 beats/min and 0°, respectively. During the first balloon inflation, we observed ST-segment elevation in leads I, aVL, and V4–6, and reciprocal ST-segment depression in leads II, III, and aVF. The heart rate and QRS axis just after the 1-min balloon inflation were 77 beats/min and –67°, respectively (Fig 1). Thus, the difference between QRS axis before and after the balloon inflation was 67°.

On a follow-up coronary angiogram performed 6 months after PCI, restenosis in the first diagonal branch was confirmed, but the proximal LAD was free from restenosis (Fig 2). Severe luminal narrowing in the first septal perforator branch was also seen. Three days after the follow-up coronary angiography, exercise testing using a bicycle ergometer was performed. At peak exercise intensity, the patient's heart rate and double product reached 105 beats/min

and 20,500, respectively. The QRS axis in the frontal plane before and after the exercise tests was +58° and –3°, respectively, and the difference between the QRS axis before and after the tests was 61°. Thus, the ECG showed a significant leftward QRS axis shift. However, no other ischemic changes, such as ST-segment deviation, were seen (Fig 3). Repeat PCI of the first diagonal branch was not done because the patient refused the procedure.

Discussion

Bream et al⁴ observed an anomalous origination of the septal perforator branch from the left coronary system other than the LAD in 18 (2.25%) of 800 patients studied at coronary angiography; in 12 of these, the vessel arose from the proximal first diagonal branch, as reported here.

In the present patient, a transient leftward QRS axis shift was seen both during exercise testing and PCI of the proximal LAD and first diagonal branch. We previously reported that a transient leftward QRS axis shift during exercise testing or PCI is a specific marker of proximal LAD disease⁸ and moreover, in some cases, an axis shift without ST-segment deviation is the only ischemic change seen on exercise ECG. One of the reasons for a transient leftward QRS axis shift is thought to be a conduction disturbance caused by ischemia of the anterior fascicle of the left bundle branch, which is supplied by the first major septal perforator branch.⁹ Although the first septal perforator branch arose from the first diagonal branch in this case, it appeared to have the same course and distribution as a normal vessel originating from the LAD. Thus, in the present patient, blood to the anterior fascicle of the left bundle branch was likely supplied from the first septal perforator branch, which originated anomalously from the first diagonal branch.

On the other hand, Azuma et al¹⁰ reported a case of vasospastic angina in which the culprit vessel was the first major septal perforator branch. During an attack, QRS axis deviation to the left as well as right bundle branch block and an increase in R wave amplitude in the precordial leads occurred together with ST-segment elevation in leads V1–3 and they concluded that the ECG changes were induced by ischemia of the right bundle branch, and the anterior and septal subdivisions of the left bundle branch. Moreover, Knight et al¹¹ have described a procedure that provides a

Fig 3. ECG before (Left) and during (Right) exercise testing using a bicycle ergometer. Exercise-induced ST-segment deviation was not observed.

unique clinical setting in which to observe the electrophysiological consequences of occlusion of the first major septal perforator branch. They demonstrated an anterior divisional block of -30° after alcohol injection into the first major septal perforator branch in 3 of 18 patients with hypertrophic obstructive cardiomyopathy, although the difference in QRS axis before and after the procedure, which is an important indicator for detecting proximal LAD disease⁸ was not detailed in these reports.^{1,12} Furthermore, both Knight et al¹¹ and Faber et al¹³ reported that this procedure seems to affect the right bundle branch most frequently, rather than the anterior fascicle of the left bundle branch. However, in our previous study, no patients with proximal LAD disease had complete or incomplete right bundle branch block during the treadmill tests or during PCI of the proximal LAD.⁸

The present ECG finding during PCI was definitely secondary to an ischemia-related phenomenon. It can be difficult to decide whether an exercise-induced leftward shift of the QRS axis is secondary to a heart-rate-related or an ischemia-related phenomenon. In this patient, the post-exercise QRS axis in the frontal plane was -3° , which did not fulfill the criterion for a left anterior divisional block;¹⁴ however, the difference in the QRS axis before and after the exercise test was 61° , which is extremely abnormal. In our previous study⁸ a tachycardia-related leftward QRS axis shift of 61° was seldom seen, so the patient's ECG finding during the exercise test was secondary to an ischemia-related phenomenon rather than a heart-rate-related phenomenon.

These ECG findings during PCI result not only from ischemia in the territory of the first diagonal branch, including a first major septal perforator branch that originates from the first diagonal branch, but also from ischemia in the territory of the LAD. However, the transient leftward QRS axis shift was likely caused by ischemia in the territory of the first diagonal branch or by ischemia in the territory of a first major septal perforator branch that originated from the first diagonal branch, rather than by ischemia in the territory of the LAD, because the ECG findings were seen even during the exercise test, when myocardial ischemia did not develop in the region perfused by the LAD. Incidentally, during follow-up coronary angiogra-

phy, severe organic stenosis was located not only in the first major septal perforator branch originating from the first diagonal branch, but also in the first diagonal branch itself. Therefore, it could not be determined whether the transient leftward QRS axis shift developed as a consequence of ischemia in the territory of the former vessel. However, this ECG finding has not been reported to result from ischemia in the territory of the diagonal branches.

The degree of leftward QRS axis shift was greater during PCI than during the exercise test. Moreover, the post-PCI QRS axis in the frontal plane was -67° , which fulfilled the criterion for left anterior divisional block. Acute coronary occlusion by balloon inflation during PCI produced ST-segment elevation, as shown in Fig 1, whereas the exercise test using a bicycle ergometer did not produce significant ST-segment deviation, as shown in Fig 3. Thus, more severe myocardial ischemia developed during the PCI procedure than during the exercise test, leading to the greater leftward QRS axis shift during PCI. Another difference in the ECG findings was the QRS axis before balloon dilatation or exercise. Before PCI, the QRS axis in the frontal plane was 0° , whereas before the exercise test it was $+58^\circ$. Before PCI, diphasic or negative T waves were seen in leads aVL and V1-5, reflecting myocardial damage secondary to myocardial infarction or myocardial hibernation involving the anterior fascicle of the left bundle branch, resulting in the greater degree of leftward QRS axis shift during PCI than during the exercise test.⁸

In conclusion, we have described a patient with non-Q wave myocardial infarction whose coronary arteriogram documented severe luminal narrowing in the first major septal perforator branch originating from the first diagonal branch. His exercise ECG showed a transient leftward QRS axis shift, which was probably caused by ischemia in the territory of the first major septal perforator branch.

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