Effectiveness of an Antiplatelet Agent for Coronary Artery Ectasia Associated with Silent Myocardial Ischemia

Kazuhiro Nagata, MD, Tatsuya Kawasaki, MD, Akio Okamoto, MD, Akira Okano, MD, Satoshi Yoneyama, MD, Kazuki Ito, MD, and Shuji Katoh, MD

SUMMARY
A 74-year-old Japanese male was referred to our hospital because of an abnormal electrocardiogram. The electrocardiogram revealed tall P waves in leads II, III, and aVF. Echocardiography disclosed hypokinesis extending from the anteroseptal region to the apex. Iodine-123 15-(p-iodophenyl)-3-(R,S)-methylpentadecanoic acid (123I-BMIPP) scintigraphy revealed reduced uptake from the anteroseptal region to the apex. Coronary arteriography demonstrated diffuse dilatation of the right and left coronary arteries without organic stenosis, and left ventriculography showed hypokinesis at the same area. Furthermore, the coronary flow reserve in the left anterior descending artery was decreased. He was treated with an antiplatelet agent. Ten months later, the left ventriculography, 123I-BMIPP scintigraphy findings and coronary flow reserve were normalized. These findings demonstrate that antiplatelet therapy may be useful in the preservation of left ventricular function in patients with coronary artery ectasia. (Jpn Heart J 2001; 42: 249-254)

Key words: Coronary artery ectasia, Silent myocardial ischemia, Antiplatelet agent, 123I-BMIPP

CORONARY artery ectasia was initially described by Morgagni in 1761 and then again by Bougon in 1812. This condition involves localized or diffuse coronary artery dilatation with or without coronary artery stenosis. Clinically, coronary artery ectasia is reported to show an association with atherosclerotic disease.1,2) It has also been suggested that coronary ectasia alone may be a cause of angina pectoris and myocardial infarction.3,4) The present case report describes a rare case of coronary artery ectasia associated with silent myocardial ischemia. In this case, treatment with an antiplatelet agent led to improvements in left ventricular wall motion, the findings on myo-
cardiac scintigraphy performed using iodine-123 \((\text{p-iodophenyl})-3-(\text{R,S})\)-methylpentadecanoic acid (\(^{123}\text{I}\)-BMIPP), and the coronary flow reserve.

**CASE REPORT**

The patient was a 74-year-old Japanese man with a history of interstitial pneumonitis and pulmonary tuberculosis. He was referred to our hospital because of an abnormal electrocardiogram without chest symptoms. He had no coronary risk factors. On physical examination, there were no abnormal findings. The blood pressure was 120 / 88 mmHg and the pulse rate was 78 / min.

Laboratory tests showed normal values. There was interstitial fibrosis on the chest roentgenogram. The electrocardiogram featured tall P waves in leads II, III, and aVF. Echocardiography showed hypokinesis extending from the anteroseptal region to the apex of the left ventricle. Although stress \(^{99}\text{mTc}\)-tetrofosmin imaging was normal (Figure 1), \(^{123}\text{I}\)-BMIPP scintigraphy disclosed reduced uptake from the anteroseptal region to the apex (Figure 2, left). Coronary arteriography demonstrated diffuse dilatation of the right and left coronary arteries without organic stenosis (Figure 3). A provocation test for coronary artery spasm was negative. Left ventriculography showed hypokinesis extending from the anteroseptal region to the apex, and the ejection fraction was 48%. Furthermore, the coronary flow reserve (the ratio of peak hyperemic to resting coro-
Figure 2. $^{123}$I-BMIPP reduced uptake extending from the anteroseptal to apical regions on admission. Ten months later, $^{123}$I-BMIPP shows normal findings on both the initial and delayed images.

Figure 3. Coronary arteriography demonstrates diffuse dilatation of the right and left coronary arteries without organic stenosis.
nary flow velocity) was measured by placing a Doppler guide wire in the left anterior descending artery and administering intracoronary adenosine triphosphate disodium (50 µg). It was found to be decreased (Figure 4, top). Based on these findings, a diagnosis of three-vessel coronary artery ectasia associated with left ventricular dysfunction was made. He was treated with an antiplatelet agent (ticlopidine 200 mg / day). Ten months later, 123I-BMIPP scintigraphy findings had normalized for both the initial and delayed images (Figure 2, right). At that time, the left ventriculography showed improvement of the ejection fraction (58%) despite no change in the appearance of the right and left coronary arteries. The coronary flow reserve was also normalized (Figure 4, bottom). At 1.5-years follow-up, this patient was doing well with an antiplatelet agent.

**DISCUSSION**

Coronary artery ectasia is defined as localized or diffuse dilatation of the coronary arteries exceeding the diameter of the adjacent normal segments or the diameter of the largest coronary vessel by 1.5 times. 5) The incidence of coronary
Coronary artery ectasia is reported to range from 0.3% to 4.9%, and it is most frequently found in the right coronary artery. The incidence of combined right and left coronary artery involvements, like this case, ranges from 21% to 50% of all coronary artery ectasia.

The prognosis in patients with coronary arterial ectasia is controversial. Slow or turbulent blood flow in a dilated vessel might be expected to lead to thrombosis of the ectatic segment or embolization of the distal coronary artery. Indeed, Markis, et al. studied 30 patients with coronary arterial ectasia and demonstrated a poor prognosis due to sudden rupture of a coronary artery or myocardial infarction. The mortality rate of coronary artery ectasia patients who were treated medically was 13% over three years, which was similar to the five-year mortality rate of 16% obtained in the European Coronary Surgery Study. On the other hand, Hartnell, et al. studied 47 patients and showed that coronary arterial ectasia itself did not affect the outcome. They revealed that the incidence of myocardial infarction increased only in patients with both coronary arterial ectasia and significant coronary stenosis.

In our patient, there was no significant coronary stenosis although abnormalities in the ventricular wall motion, scintigraphy and the coronary flow reserve were present. These abnormal findings were improved by long-term therapy with ticlopidine. Ticlopidine is an antiplatelet drug that acts by interfering with the binding of fibrinogen and Von Willebrand factor to platelets. It prolongs bleeding time and blocks the platelet release reaction. Ticlopidine also has a potent antithrombotic activity. The coronary vasodilatation induced by ticlopidine is entirely related to its direct action on vascular smooth muscle. A number of randomized, double blind, placebo-controlled trials have demonstrated that ticlopidine is effective in preventing thromboembolic events and cardiovascular death in patients with cardiovascular disease. Furthermore, it has been recommended that coronary artery ectasia should be treated with anticoagulants or antiplatelet agents.

In summary, prolonged administration of an antiplatelet drug resulted in improvements in the left ventricular wall motion, scintigraphy findings and the coronary flow reserve in the present case. These findings demonstrate that antiplatelet therapy may be useful for the preservation of left ventricular function in patients with coronary arterial ectasia.

References