A Case of Rest Angina due to Microvascular Spasm

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A 66-year-old woman underwent elective cardiac catheterization for investigation of periodic attacks of chest pain at rest. During the examination, a chest pain attack occurred unexpectedly, resulting in ST elevation in the precordial leads on electrocardiography. Immediate coronary arteriography demonstrated no organic stenosis but markedly delayed contrast medium perfusion in the mid to distal portion of the left anterior descending artery. These phenomena spontaneously disappeared about 3 min later, and the patient was diagnosed as having angina pectoris due to microvascular spasm. The demonstration of angina pectoris due to microvascular spasm by coronary arteriography during a spontaneous attack is very rare.

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Key Words: Microvascular spasm; Rest angina

Some patients with angina-like chest pain and angiographically normal coronary arteries show electrocardiographic ST-segment depression following exercise or atrial pacing, and are diagnosed as having microvascular angina or 'syndrome X'. Such patients have a reduced ability to decrease coronary resistance and increase coronary flow in response to atrial pacing or pharmacologic vasodilator stimuli!

A few reports have described patients with coronary microvascular spasm with ST-segment elevation after coronary angioplasty for acute coronary syndrome. This disorder is thought to be related to an endogenous vasoconstrictor released by a nonobstructive thrombus^{2,3}

We describe a rare case of rest angina due to microvascular spasm accompanied by ST-segment elevation that was demonstrated by coronary arteriography during a spontaneous attack of angina.

Case Report

The patient was a 66-year-old Japanese woman who was admitted to our hospital with a history of periodic attacks of chest pain during the night over a 7-year period. Treatment with nitrates and calcium channel blockers had reduced the frequency of the attacks.

Physical examination on admission showed a blood pressure of 134/80 mmHg, a heart rate of 80/min, and no abnormalities of the chest or abdomen. Chest radiography revealed a slight thickening of the right pleura. Electrocardiographic findings were normal. Treadmill exercise testing did not provoke chest pain or ST-T change.

Elective cardiac catheterization was performed after interruption of the patient's usual nitrate and calcium channel blocker treatment. The baseline coronary arteriogram revealed no organic stenosis of either the right or left coronary artery. The administration of acetylcholine chloride, 50–150 μ g, into the right coronary artery provoked a 50% narrowing in the mid-portion without chest pain or change in the ST-T wave. About 10 min after the administration of acetylcholine chloride into the right coronary artery, during preparation for left coronary arteriography, the patient experienced severe chest pain, which was similar to that experienced before admission. ST-segment elevation was then seen in V_{3–5} (Fig 1). Immediate coronary arteriography demonstrated a marked delay in perfusion of contrast medium in the mid to distal portion of the left anterior descending artery (Fig 2) but no narrowing of the epicardial coronary artery. It took 5.9 sec for contrast medium to fill the left anterior

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descending artery from the proximal to the distal portion, but only 1.9 sec to fill the left circumflex artery and diagonal branch from the proximal portion of the anterior descending artery. The chest pain, ST elevation and delayed perfusion of the left anterior descending artery disappeared spontaneously about 3 min later. The administration of acetylcholine chloride into the left coronary artery demonstrated no vasospasm of the epicardial artery and no delay in perfusion of the contrast medium. Left ventriculography demonstrated normal systolic function.

Fig 1. Electrocardiograms obtained at baseline (left) and when the patient experienced chest pain (right) during cardiac catheterization. Elevation of the ST-segment in V3-V5 was observed during the attack of chest pain.

Coronary arteriographic and the electrocardiographic findings during chest pain in our patient were the same as those observed when coronary microvascular spasm was provoked by the administration of leukotrienes C₄ and D₄ into the coronary artery of pig⁴. Therefore, we believed that the only possible explanation was microvascular spasm in the area supplied by the left anterior descending artery that was severe enough to induce transmural ischemia, although microvascular spasm was not demonstrated by microscopy. The patient was diagnosed as having angina pectoris due to microvascular spasm; the administration of nitrates and calcium channel blockers was resumed.

Discussion

Two previous reports on microvascular spasm after angioplasty in patients with acute coronary syndrome describe the occurrence of this phenomenon distal to the angioplasty site, and have suggested that it is provoked by vasoconstrictor mediators released by a nonobstructive thrombus^{2,3}. Sekiya et al⁵ report a rare case of acetylcholine-induced myocardial ischemia without epicardial coronary artery spasm. They consider that the myocardial ischemia was caused by microvascular spasm induced by acetylcholine, but did not observe slow progression of contrast medium in coronary artery. Hattori et al⁶ report a case demonstrating slow progression of contrast medium in the coronary artery with chest pain and ST elevation after the administration of ergonovine and glyceryl trinitrate, which is similar to our case angiographically. They believe that this phenomenon was caused by diffuse arteriolar constriction. Leukotrienes C₄ and D₄, which are released from leukocytes and macro-

Fig 2. Arteriograms of the left coronary artery during an attack of chest pain. Perfusion of contrast medium in the mid to distal left anterior descending artery was markedly delayed, but perfusion to the distal left anterior descending artery was complete with no spasm of the epicardial artery. It took 5.9 sec for contrast medium to fill the left anterior descending artery, but only 1.9 sec to fill the left circumflex artery and the diagonal branch from the proximal portion of the anterior descending artery.

phages, reportedly induce coronary microvascular spasm with ST-segment elevation in the swine coronary artery model⁴. It has been indicated that norepinephrine induces microvascular vasoconstriction via the α -receptor⁷. In our patient, no intracoronary thrombus formation or injury to the coronary artery was observed. As the onset of anginal pain occurred more than 10 min after the administration of acetylcholine into the right coronary artery, it was unlikely that acetylcholine provoked a microvascular spasm in the area supplied by the left anterior descending artery. Our patient experienced arthritic knee joint pain during cardiac catheterization. Such pain may activate sympathetic tone and induce microvascular spasm via the α -receptor.

Coronary microvascular spasm can reportedly lead to necrotic fibrosis of the myocytes in an experimental model of dilated cardiomyopathy⁸. However, our patient had a normal systolic function of the left ventricle, despite a 7-year history of chest pain. Whether the microvascular spasm was responsible for all the episodes of chest pain in this patient is not clear.

Although angina due to microvascular spasm, as in the present patient, is very rare, both spasm of the epicardial artery and microvascular spasm have to be considered in the differential diagnosis of patients with angina pectoris accompanied by transient ST-segment elevation and without organic stenosis of the coronary arteries.

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