Case Report

Angina Pectoris Induced by Microcoronary Artery Spasm

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Abstract: A case of 62-year-old female with repeated chest pain by microcoronary artery spasm is reported. Coronary arteriography revealed no significant stenosis in either right or left coronary arteries. Acetylcholine provocation test of the right coronary artery induced chest pain in the absence of coronary artery spasm on arteriogram. The electrocardiogram taken during the chest pain showed ST segment elevation in leads II and III and aVF. These findings suggest microcoronary artery spasm, which can be a cause of myocardial ischemia.

Key words: angina pectoris, acetylcholine provocation test, microcoronary artery, coronary artery spasm

Introduction

Coronary arteriography sometimes reveals no significant stenotic change in patients with repeated attacks of angina pectoris-like chest pain and ischemic ST-T changes on electrocardiogram. This has been suggested to be due to coronary artery spasm1) or microcoronary artery stenosis2). We encountered a patient in whom coronary arteriography showed no significant stenosis, and acetylcholine provocation test induced chest pain with the appearance of ischemic ST-T changes on the electrocardiogram but did not show significant stenosis on coronary arteriogram. This case suggests small coronary artery spasm as a cause of angina.

Case Report

A 62-year-old female had suffered from chest pain at rest every morning which lasted for 5 to 20 minutes for 1 month. Chest pain was improved after sublingual administration of nitroglycerin. She was admitted to hospital because of repeated attacks of chest pain suspicious of angina pectoris. There were no electrocardiographic records during attacks. She had histories of extrauterine pregnancy, uterine myoma, gall stones or urticaria induced by NSAIDs, and no family history of ischemic heart disease. On admission, her blood pressure was 130/68 mmHg, pulse rate 54/min. regular, and there were no abnormal physical findings except urticaria on the extremities. Chest roentgenograms were normal, and electrocardiogram (Fig. 1) showed the sinus rhythm, normal axis, and no ischemic ST-T changes. Laboratory examinations revealed no abnormalities. Exercise thallium scintigraphy (Fig. 2)

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Fig. 1. Electrocardiogram on admission: heart rate 68/min., sinus rhythm, normal axis, counter-clockwise axis rotation is observed in the transitional zone, but no ischemic ST-T changes are evident.

Fig. 2. Exercise thallium scintigram
A Tl accumulation image immediately after exercise is shown, but comparison between pre-and post-exercise images showed no ischemic change. Panel A is the myocardial vertical long axis image and panel B is the horizontal long axis image. Panels C and D are the short axis images of myocardial perfusion, and show base and apex, respectively.
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Fig. 3. Right coronary arteriogram: LAO 60° (upper panel) 
Left coronary arteriogram: RAO 25° caudal 25° (lower panel) No significant stenosis is observed.

was started but was discontinued due to fatigue of the lower limbs 9 minutes after initiation of bicycle ergometer exercise (75 W). No chest pain, no ischemic ST-T changes on electrocardiogram and no transient defect of T1 accumulation images were observed during the examination. Cardiac catheter examination was performed. The hemodynamics findings were normal (PCWP 10 mmHg, CI 3.77). Left ventriculography revealed normal left ventricular wall motions. Right and left coronary arteriograms showed no significant stenosis (Fig. 3). The acetylcholine provocation test was performed to induce coronary artery spasm. One minute after administration of acetylcholine (50 μg) into the right coronary artery (RCA), chest pain developed simultaneously with ST elevation in II, III,
Fig. 4. 12 lead electrocardiogram: ST elevation was observed in II, III, and aVF after acetylcholine provocation.

and aVF on electrocardiogram (Fig. 4). Right coronary arteriography was performed during the attack of chest pain, but no coronary artery spasm was observed with prolonged contrast enhancement (about 2-fold increased) due to decreased blood flow velocity (Fig. 5). Spasm of RCA ostium was also ruled out by coronary arteriograms. Before and after acetylcholine injection, the rate-pressure product (systolic atrial pressure × heart rate), which is an index of myocardial oxygen demand did not change markedly (8400 vs 9028). After nitroglycerin injection into the RCA, the chest pain and ST elevation on electrocardiogram rapidly improved. She has been treated with a calcium antagonist on an outpatient basis without recurrence of chest pain.

Discussion

In about 10% of the patients with chest pain, coronary arteriography shows neither significant stenosis in the coronary artery nor other heart diseases. The detailed mechanism of this phenomenon is unclear but is considered to be coronary artery spasm or ischemia due to microcoronary artery stenosis. Injection of acetylcholine into the coronary artery is useful for inducing spasm and is used in the diagnosis of vasospastic angina. In this patient, the acetylcholine provocation test induced reproducible chest pain and ST segment elevation on electrocardiogram, and coronary arteriography showed a prolongation of contrast enhancement without spasm in the epicardial coronary artery. The possibility cannot be excluded that attacks of chest pain and the chest pain during examination in this patient are due to ischemia caused by coronary artery spasm. However, the absence of macrocoronary spasm and the prolongation of contrast enhancement on coronary
arteriograms during chest pain suggest microcoronary artery spasm. Furthermore, there was no marked change in the pressure-rate product which indicates that the chest pain did not occur as a result of an increase in demand for myocardial oxygen, but the decrease in blood flow velocity also suggested microcoronary artery spasm. Several authors\textsuperscript{5-7} have reported patients in whom ergonovine or acetylcholine provocation tests induced no macrocoronary artery spasm but induced ST elevation or depression on electrocardiogram simultaneously.
with chest pain, suggesting microcoronary artery vasospasm. In this patient, the acetylcholine provocation test induced ST elevation on electrocardiogram in the absence of macrocoronary artery spasm. This finding indicates that microcoronary artery spasm can induce wall-penetrating severe myocardial ischemia. The other possible mechanism of this phenomenon is occlusion with micro thrombi and micro air embolism. However, these possibilities can be excluded in this patient since both chest pain and ST elevation rapidly improved after coronary nitroglycerin injection. The putative metabolites of nitroglycerin such as nitric oxide and S-nitrosocysteine produced dilatation of all size classes of vessels. The pathogenesis of coronary artery spasm in humans is unclear. However, arteriosclerotic or inflammatory changes in the vascular smooth muscle and vascular endothelial cells may underlie hypersensitive constriction of microcoronary arteries. This patient may have damage to endothelial cells and smooth muscle cells of microvessels. The differences in the sensitivity to acetylcholine between macro- and microcoronary arteries are unclear, however, acetylcholine receptors may vary in quantity and quality in different blood vessels.

We report a patient with chest pain and ischemic changes on electrocardiogram in whom coronary arteriography showed normal findings, and microcoronary artery spasm is likely to be the cause of this condition.

References