Acute Myocardial Infarction Caused by Simultaneous Occlusion of the Right Coronary Artery and the Left Anterior Descending Coronary Artery Probably Due to Coronary Spasm: A Case Report

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Abstract
A 56-year-old man had an attack of chest pain associated with ST-segment elevation in both the inferolateral and anteroseptal leads on electrocardiography. Emergency coronary angiography showed thrombus in the mid right coronary artery and total occlusion in the distal left anterior descending coronary artery. Intravenous heparin infusion and antiplatelet therapy were given without other coronary intervention. After 1 week, repeat coronary angiography showed neither significant stenosis nor thrombus in the coronary arteries. Severe coronary artery spasm in the left coronary artery was induced by the provocation test with intracoronary injection of 50 μg acetylcholine. He had an uneventful hospital course. This unique case demonstrated intracoronary thrombus formation in the right coronary artery and left anterior descending coronary artery simultaneously due to suspected coronary spasm.

Key Words
Myocardial infarction, pathophysiology
Coronary vasospasm
Angiography

INTRODUCTION
The incidence of acute myocardial infarction with simultaneous occlusion of two or three coronary arteries is not common1-3. The role of coronary spasm in the onset of myocardial infarction has often been considered4-8. We describe a case of a thrombus in the mid right coronary artery (RCA) and total occlusion in the distal left anterior descending coronary artery (LAD) manifesting as
myocardial infarction, which verified the cause as coexistence of coronary spasm and thrombosis.

**CASE REPORT**

A 56-year-old man was in reasonably good health except for heavy smoking (40 cigarettes/day). He was admitted to our hospital due to severe chest pain 3 hr after the onset. He experienced no chest pain before the myocardial infarction. He had no history of hypercoagulable disorders including hematological and autoimmune diseases. Electrocardiography (ECG) on admission showed ST-segment elevation in both the inferolateral and anteroseptal leads (Fig. 1 - left). ECG 1 hr later showed disappearance of ST-segment elevation and T-wave inversion (Fig. 1 - right). Echocardiography showed inferoposterior and apical hypokinesis without thrombus in the left atrium or ventricle. Heart rate was regular at 68 beats/min, and blood pressure was 126/76 mmHg. Laboratory findings were as follows: white blood cell count 9,820/μl, platelet count 217,000/μl, fasting blood sugar 98 mg/dl, hemoglobin A1c 5.7%, total cholesterol 188 mg/dl, high-density lipoprotein-cholesterol 38 mg/dl, triglycerides 148 mg/dl, uric acid 7.4 mg/dl, and creatine kinase 182 IU/l. Emergency coronary angiography revealed thrombus in the mid RCA (Fig. 2 - left) and total obstruction in the distal LAD (Fig. 2 - right).

He was transferred to the coronary care unit where intravenous heparin (500 U/hr), aspirin (200 mg/day), and ticlopidine (200 mg/day) were given, but no other coronary revascularization therapy. The hospital course was uneventful. Serum creatine kinase peaked at 1,624 IU/l. After 1 week, repeat coronary angiography showed neither significant stenosis nor thrombus in the RCA (Fig. 3 - A) or LAD (Fig. 3 - B). The coronary spasm provoca-
**Fig. 2** Emergency coronary angiograms

Thrombus was present in the mid right coronary artery (left, white arrow) and total obstruction in the distal left anterior descending coronary artery (right, black arrow).

**Fig. 3** Repeat coronary angiograms

No significant stenosis or thrombus was detected in the right coronary artery (A) or left anterior descending coronary artery (B). Severe coronary spasm of the left anterior descending coronary artery and left circumflex artery was induced by intracoronary injection of 50 μg acetylcholine (C).
tion test caused severe coronary spasm of the LAD and left circumflex artery by an intracoronary injection of 50 μg acetylcholine (Fig. 3· C). The left ventricular ejection fraction was 63%, with mild hypokinesis of the apical wall. The patient was entirely pain free and was discharged without a cardiac episode on the 10th day.

DISCUSSION
The incidence of myocardial infarction in patients with normal or almost normal coronary arteries ranges from 1 to 13%[7], but is very rare with simultaneous occlusion of two or three main branches. The pathogenetic mechanism of myocardial infarction in patients with normal coronary arteries remains unknown. In this patient, one coronary artery was completely blocked and another was incompletely occluded by thrombus. ECG or echocardiography on admission and follow-up showed two coronary arteries could be totally occluded.

Several factors, such as multivessel spasm, state of hypercoagulability at the onset of myocardial infarction, and decreased coronary pressure are thought to be involved in the mechanism of simultaneous occlusion of multiple coronary arteries. Increased coronary vasomotor tone was found in acute myocardial infarction patients with spontaneous coronary recanalization[8]. Rupture of the atheromatous plaque with subsequent platelet aggregation, prolonged occlusive coronary spasm superimposed on atherosclerotic lesion, or both, have been proposed as triggering mechanisms for thrombus formation. In addition, cigarette smoking and alcohol are promoters of the mechanism of enhanced platelet aggregation and adhesion that can be expected to increase the thrombotic risk to normal coronary arteries[9].

In this patient, the coronary artery was angiographically normal but vasospasm could be provoked. Although chest pain before the myocardial infarction was absent, coronary spasm may have caused formation of the intracoronary thrombus. The occurrence of coronary spasm of the coronary arteries strongly suggests that coronary spasm induced thrombus formation and led to myocardial infarction. However, hypotension and poor coronary perfusion, caused by single vessel occlusion, might trigger thrombus formation in another vessel. This concurrent thrombosis is uncommon, but may represent an important pathophysiologic syndrome related to hypercoagulability, hypervasoactivity in multiple vessels, or perhaps coincident plaque events such as fissuring or rupture. Intravascular ultrasonography may have revealed thrombi or unstable plaques that are difficult to image using conventional angiographic techniques in our case.

CONCLUSIONS
This case shows that simultaneous occlusion of the right coronary artery and the left anterior descending coronary artery may be caused by coronary artery spasm.

要約
冠挙縮によると思われる右冠動脈と左前下行枝の同時期の閉塞が認められた急性心筋梗塞の1例
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症例は56歳、男性である。胸痛を主訴に来院し、心電図上、前胸部誘導および下壁誘導にST上昇が認められた。緊急冠動脈造影検査では、右冠動脈の中間部に血栓像と左前下行枝遠位部に完全閉塞所見を示し、ヘパリンの投与および抗血小板療法のみで治療を行った。1週間後の追跡冠動脈造影検査では、明らかな冠動脈硬化所見や閉塞所見は認められなかった。同時にアセチルコリンを用いた冠挙縮誘発試験を行った。左冠動脈へアセチルコリン50 μgの投与を行った時点で著明な冠挙縮が誘発された。入院経過は良好であった。本症例は冠挙縮を誘因として形成された血栓によって右冠動脈と左前下行枝の2枝に同時閉塞を生じたと考えられた。
References


