A Case of Variant Angina with Normal Coronary Arteriogram Who Developed Acute Myocardial Infarction and Cerebral Embolism

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SUMMARY

We report a case of a 44-year-old male with variant angina who developed acute anterior myocardial infarction 2 weeks following demonstration of normal left coronary artery. This experience provides inferential evidence that myocardial ischemia resulting from coronary spasm may progress into myocardial infarction.

Additional Indexing Words: Variant angina, Coronary spasm, Myocardial infarction

PRINZMETAL’S variant angina pectoris, as originally described, was thought to be caused by a significant major coronary artery stenosis. However, subsequent reports in the literature have emphasized that in some patients who conformed to the clinical syndrome described by Prinzmetal, no significant coronary stenosis was found by coronary cineangiography. The prognosis of variant angina pectoris with significant coronary atherosclerosis appeared to be grave, with a high incidence of myocardial infarction. While, the prognosis in those without significant coronary obstructive lesions remains obscure.

We report a case of variant angina with angiographically normal coronary arteries who developed myocardial infarction and cerebral embolism.

CASE REPORT

A 44-year-old male patient had been in excellent health until February 1977, when he first experienced a boring, pressure-type substernal discomfort. The symptom occurred usually at rest and lasted for several minutes. On January 5, 1978, he had an episode of substernal chest pain occurring at rest after walking. He reported to a local hospital and was examined. His electrocardiogram showed
marked S-T elevations in leads I, aVL, and V1-6 (Fig. 1). He was transferred to our hospital for further evaluation on January 26, 1978.

The family history was negative for coronary artery disease.

The patient has smoked 2 packages of cigarettes daily for 20 years.

Physical examination revealed the blood pressure of 110/60 mmHg and the pulse rate of 66/min. The heart sounds were normal and no murmurs were audible. The rest of physical examinations was normal.

His laboratory evaluation showed no abnormalities except for an increase in serum cholesterol. The tests for coagulation and fibrolysis were normal.

The chest X-ray films were normal. His resting electrocardiogram showed no remarkable abnormalities (Fig. 2).

Treadmill stress test was performed with the Bruce protocol. He could walk 12 min without chest pain, pulse rate reaching to 162/min. The ECG revealed no S-T segment changes (Fig. 3).

Thallium-201 myocardial perfusion imaging was performed during exercise. Left ventricular scintigrams obtained in anterior, 30° and 60° left anterior oblique views showed no definite new perfusion defects (Fig. 4). These findings indicated that this patient had no significant coronary narrowings.1)

Retrograde left ventriculography and selective coronary arteriography were performed using the Amplatz percutaneous transfemoral technique. Left ven-
Tricular end-diastolic pressure was normal; ventriculogram revealed normal contraction (ejection fraction was 76%) (Fig. 5). Coronary arteriography revealed that the left anterior descending artery had no significant coronary narrowings and other major coronary arteries were almost normal (Fig. 6).

In the hospital, the patient had no symptoms and was discharged on February 23, 1978. But chest discomfort recurred at rest 4 days after discharge. He used oral Nifedipine, 10 mg 4 times daily. On March 1, 1978, he had the severe chest pain associated with nausea and diaphoresis occurring at rest after walking to his office. The electrocardiogram at this time showed marked S-T elevation and Q waves in leads I, aVL and V_{1-6}, indicating that acute myocardial infarction oc-
curred in the same location as the ischemic area of previous anginal attack. Serial ECGs and enzyme studies confirmed the diagnosis of acute anterior wall myocardial infarction (Fig. 7).

Furthermore, cerebral embolism producing unconsciousness and hemiplegia occurred 10 days later. The left carotid angiograms revealed a large embolus plugged the left internal carotid artery at the level of supraclinoid portion (Fig. 8). However, fortunately, this patient was well recovered and discharged later.

**DISCUSSION**

A variant form of angina pectoris characterized by recurrent angina at rest with transient elevation of S-T segment, was described by Prinzmetal
et al. On the basis of postmortem examinations, he considered this syndrome to be due to atherosclerosis of a single major coronary artery. After Prinzmetal’s original description, coronary angiography has come into general use, and subsequent reports support his concept of the pathologic changes associated with this syndrome. A group of patients with variant
angina and normal coronary arteriogram have been discussed in recent reports.\textsuperscript{8)\textendash{}10} A spasm of a major coronary artery was postulated as a mechanism and the angina might develop without manifest atherosclerotic coronary disease. Angina pectoris in the presence of significant coronary artery disease has been associated with a increased incidence of morbidity and sudden death. The prognosis of these patients has been considered to be grave. The patients having usual angina pectoris with normal coronary arteriogram have been reported to follow a benign course.\textsuperscript{11),12} However, the prognosis in variant angina pectoris with normal coronary arteriogram remains obscure. The clinical course in the case reported here suggests that the fate of patient with variant angina pectoris and normal coronary arteries may not be favorable. Until a large series of patients are studied, a caution is needed to avoid making a clinical presumption that the prognosis in patients with angina pectoris and normal coronary arteries is always benign.

In most cases of myocardial infarction, the ischemia is caused by coronary occlusion. The most common cause of coronary occlusion is coronary thrombosis which is almost always a complication of coronary atherosclerosis. Rarely coronary occlusion is due to other causes, including coronary embolism and coronary spasm.

Recently, much attention has been payed to the patients with myocardial infarction and angiographically normal coronary arteries.\textsuperscript{13)\textendash{}15} Three major possible pathogenesis of myocardial infarction without arteriographic obstruction have been discussed; 1) myocardial infarction is produced by coronary...
arterial spasm, 2) thromboembolism causes myocardial infarction and disappears (e.g., lysis or recanalization of coronary thrombosis), 3) the significant lesions as a cause of the infarction are present but missed by the coronary arteriogram.

Arnett and Roberts\(^{16}\) showed that none of the 22 patients with variant angina and angiographically normal coronary arteries had an acute myocardial infarction, and suggested that myocardial infarction was never caused by coronary artery spasm. Rosenblatt\(^{17}\) also reported that myocardial infarction did not occur in patients with variant angina due to spasm of normal coronary arteries. Therefore, they speculated that the most reasonable explanation for the occurrence of myocardial infarction and normal coronary arteriogram was acute coronary embolism with subsequent clot lysis, or recanalization.

On the other hand, evidence in support of coronary artery spasm as a possible contributing factor to acute myocardial infarction has accumulated.\(^{18}\) The report\(^{19}\) of a patient who had acute inferior wall myocardial infarction resulted from a spasm of the left circumflex artery during selective coronary arteriography suggested that spasm could be responsible for acute myocardial infarction. Johnson et al\(^{20}\) described a case of variant angina with recurrent myocardial infarction, angiographically normal coronary arteries and an episode of coronary artery spasm induced by intravenous ergonovine; coronary spasm was suggested to have caused myocardial infarction and variant angina attacks.

In our patient, 2 weeks following the demonstration of normal left coronary artery, myocardial infarction occurred in the same location as that of the ischemia during attack of variant angina. This experience suggests that coronary artery spasm was responsible for the myocardial infarction. An embolic cause of myocardial infarction can be ruled out because of the absence of any identifiable source of emboli and of the low probability that emboli would plug the same vessel perfusing the territory of the previous transient ischemic attacks.

Arterial embolization constitutes a significant complication of myocardial infarction. However, cerebral embolism has been shown to be less frequent complication than had been suspected. Of 1000 cases of myocardial infarction studied by Russek et al,\(^{21}\) cerebral embolism occurred in only one patient. Our patient developed cerebral embolism 10 days after the onset of myocardial infarction. This embolism seemed to have been originated from the mural thrombi which formed on the endocardium of the infarcted left ventricle.
REFERENCES


