CASE REPORT

Coronary Arterial Spasm in Single Coronary Artery

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SUMMARY A 48-year-old man developed severe chest pain and became unconscious. Coronary cineangiography revealed single coronary artery of the type L2b by Sharbaugh and White. Ergonovine, 0.2 mg i.v., produced coronary arterial spasm in the right coronary artery.

This case suggests that coronary arterial spasm might be a cause of sudden death in patients with single coronary artery. However, an association of single coronary artery and coronary arterial spasm might be coincidental.

SINGLE CORONARY ARTERY is a rare congenital anomaly1 that has been regarded as having little clinical significance.2-4 However, some investigators suggest that patients with single coronary artery may be predisposed to sudden death4-8 or myocardial infarction (MI).9-11 Several explanations for sudden death in this anomaly have been proposed: a compression of the aberrant artery between the pulmonary artery and the aorta;4-9 a flap-like closure of the ostium of the anomalous coronary artery;6 a congenital underdevelopment of the left coronary artery;10 or slow controlled ischemia.1,3,7

In addition, spasm of the aberrant coronary artery is also suggested as a cause of sudden death or MI.11 However, coronary artery spasm in the aberrant artery has not been demonstrated. We describe a patient with single coronary artery who had spasm of the aberrant coronary artery. An episode of syncope and near death was attributed to spasm.

Case Report

A 48-year-old man had been in good health with no history of chest pain until September 1980. One morning, he developed severe chest pain while he was walking 15 minutes after carrying 30 kg of baggage. When he was being transferred to our coronary care unit, he lost consciousness for several minutes and became cyanotic.

The admission ECG demonstrated a complete atroventricular block (QRS complex 0.10 second), ST-segment elevation in leads II, III, aVF and V1, and reciprocal ST-segment depression in leads I, aVL and V4-6 (fig. 1). He was alert and had a mild chest oppressive sensation. In 10 minutes, symptoms spontaneously subsided and electrocardiographic abnormalities disappeared. Careful physical examination revealed no abnormal findings. Chest x-ray was normal. Hematologic, serologic and chemical data, including enzymatic changes for myocardial necrosis, were all normal.

Because we suspected that the episode was caused by coronary arterial spasm, he was begun on a regimen of 40 mg of isosorbide dinitrate and 240 mg of diltiazem12-16 every 6 hours. In the next 14 days, he had no recurrence, and several 24-hour Holter tape monitorings showed no significant ST-T changes. He underwent repeated treadmill exercise tests and the grade of exercise was progressively increased until he could tolerate Bruce stage VI of exercise (maximum heart rate 149 beats/min) without any antianginal drug. He remained free of symptoms, with no significant ST-T changes in any lead.

A catheterization was performed 39 days after the episode. Pressure data were normal. Left ventriculography showed no asynergy and normal contraction. The coronary cineangiogram obtained by the Sones technique revealed that the coronary artery was originated from the left sinus of Valsalva, bifurcated and ran as in the heart with normally distributing coronary trees. The right coronary artery (RCA) passed through between the great arteries (type 2 single coronary artery by Smith17 and type L2b by Sharbaugh and White1 [fig. 2]). There was a segmental stenosis of 25% in the RCA. After 0.2 mg of i.v. ergonovine, he developed chest pain associated with ST-segment elevation in leads II, III, aVF and V1. The coronary cineangiogram showed a 90% segmental stenosis at the originally stenotic portion of the RCA (fig. 3). After sublingual nitroglycerin, the ergonovine-induced coronary arterial spasm was relieved and the electrocardiographic changes and chest pain disappeared.

He was discharged on a regimen of 240 mg of diltiazem and has had no recurrence of chest pain or syncope.

Discussion

Coronary arterial spasm occurs spontaneously without apparent relation to increased oxygen demand of the myocardium18-20 and can be induced by drugs

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spasm is similar to spontaneous episodes and coronary arterial spasm can almost always be present in patients in whom coronary arterial spasm is induced by ergonovine. Also, the ECG findings on admission, ST-segment elevation in leads II, III, aVF and V₁, were identical to those during ergonovine-induced coronary arterial spasm. The development of a complete atioventricular block during coronary arterial spasm has been reported, particularly when spasm occurs in the RCA.

We considered that the episode of near death of this patient was not caused by a compression of the aberrant artery between great vessels or a flap-like closure of the ostium during high coronary flow, because the episode was not reproducible even with severe exercise. The patient tolerated Bruce stage VI exercise without antianginal drugs. The coronary trees were well developed, and thus, slow, controlled ischemia is not likely to explain the episode.

This case demonstrates that coronary arterial spasm may occur in single coronary artery and may be a cause of sudden death or MI. If arterial spasm occurs at the common portion of the coronary artery, it would produce severe global myocardial ischemia. However, it is not known whether single coronary artery is associated with higher incidence of coronary arterial spasm. The association of single coronary artery and coronary arterial spasm in this patient might be coincidental.

Clinical features of this case suggest that chest pain and subsequent unconsciousness was probably caused by coronary arterial spasm and secondary severe dysrhythmia. Intravenous ergonovine induced a 90% stenosis in the RCA at the portion where there was originally a mild stenosis. Coronary arterial spasm often occurs at the site of preexisting organic stenosis. After ergonovine, the patient developed chest pain. Ergonovine-induced coronary arterial

FIGURE 1. ECGs at admission (A) and 10 minutes later (B). The ECG at admission demonstrated a complete atioventricular block, ST-segment elevation in leads II, III, aVF and V₁, and reciprocal ST-segment depression in leads I, aVL and V₄a. Such electrocardiographic abnormalities were not observed 10 minutes later.

...such as methacholine and ergonovine by manipulations of autonomic nervous system, by alkalosis and even by exercise. It is generally accepted that coronary arterial spasm is present when the patient has transient ST-segment elevation with angina at rest.

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**Figure 3.** Right coronary cineangiograms selectively performed after 0.2 mg of i.v. ergonovine (A) and 0.3 mg of sublingual nitroglycerin (B) using an Amplatz catheter (60° right anterior oblique projection). The selective right coronary arterial injection during chest pain induced by ergonovine showed a segmental stenosis of 90% in the right coronary artery at the originally stenotic portion (A, arrow). After sublingual nitroglycerin, the ergonovine-induced stenosis disappeared, associated with complete relief of chest pain (B).

**References**

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