

Isolated Left Coronary Ostial Stenosis —A Case Report—

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A patient with isolated left coronary ostial stenosis proved by coronary angiography is presented. Isolated left coronary ostial stenosis is a rare condition of unknown etiology.

Unique clinical and angiographic profiles are discussed in detail with a review of the literature, along with a suggestion of a natural history distinct from that usually seen in atherosclerotic coronary diseases.

Key Words: *Coronary ostial stenosis*

INTRODUCTION

As with left main coronary artery diseases, patients with coronary ostial stenosis are at high risk of premature death, which relates directly to the extent of the area of myocardium placed in jeopardy.

Isolated coronary ostial stenosis is rare. In a previously reported series¹⁻⁶⁾, the incidence of ostial stenosis has varied between 0.13 and 2.7%, and in the majority of cases there is a coexisting disease in the multiple coronary vessels. It has been assumed to be atherosclerotic in origin¹⁻⁵⁾ and excludes the coexisting homozygous familial hypercholesterolemia, otherwise normal coronary vessels in patients with syphilis⁶⁻¹⁰⁾ or other form of aortitis, congenital anomaly¹¹⁾ and iatrogenic ostial stenosis¹²⁻¹⁶⁾ as a complication of coronary angiography or after coronary perfusion at the time of cardiac surgery¹²⁻¹⁴⁾.

The clinical and angiographic profile of isolated coronary ostial stenosis is unique and at variance with that normally expected in patients with atherosclerotic coronary disease.

CASE REPORT

A 50-year-old postmenopausal woman was admitted to the hospital because of crescendo angina.

She was well except for tuberculous pleurisy experienced seven years earlier, when the onset of chronic stable angina pectoris took place, which, since then, responded to nitroglycerin. During the year or two before her admission, the angina became more frequent and occurred on minimal exertion. Pain radiating to both shoulders and upper arms frequently accompanied the angina episodes.

After admission, the angina was controlled by bed rest and intensive medical treatment with beta blocking and calcium channel blocking drugs and sublingual nitrates. There was no history of hypertension, cigarette smoking, use of alcohol, or birth control pills.

Upon admission, her temperature was 36.9°C, pulse per minute and respiration 16 per minute. The blood pressure was 130/90 mmHg.

On examination, the patient appeared acutely ill. However, the head and neck were normal; the jugular vein was not distended. The lungs were clear. The heart sounds were regular; no murmurs or pericardial friction rub was heard. The abdomen

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ISOLATED LEFT CORONARY OSTIAL STENOSIS

was normal; no organ or mass was palpated. There was no peripheral edema, clubbing or cyanosis. Neurologic examination was negative.

The urine was normal. The hematocrit was 37%; the white-cell count was 9,900 per cubic millimeter with 61% neutrophils, 6% band form, 35% lymphocytes and 2% basophils. The platelet count was 304,000 per cubic millimeter. The prothrombin time was 10.3 seconds with 100% of control; the

bleeding time was 2.00 minutes; the clotting time was 7.00 minutes. The SGOT was 15 u, the LDH 120 u and the CPK 41 u per liter. The total cholesterol was 250 mg, the triglyceride 99 mg, and the HDL-cholesterol 59 mg per deciliter. The serologic test for VDRL was negative.

The electrocardiogram was normal at rest and showed slight depression of the ST segments and inverted T waves in Leads I, aVL and V4-6 during an angina episode.

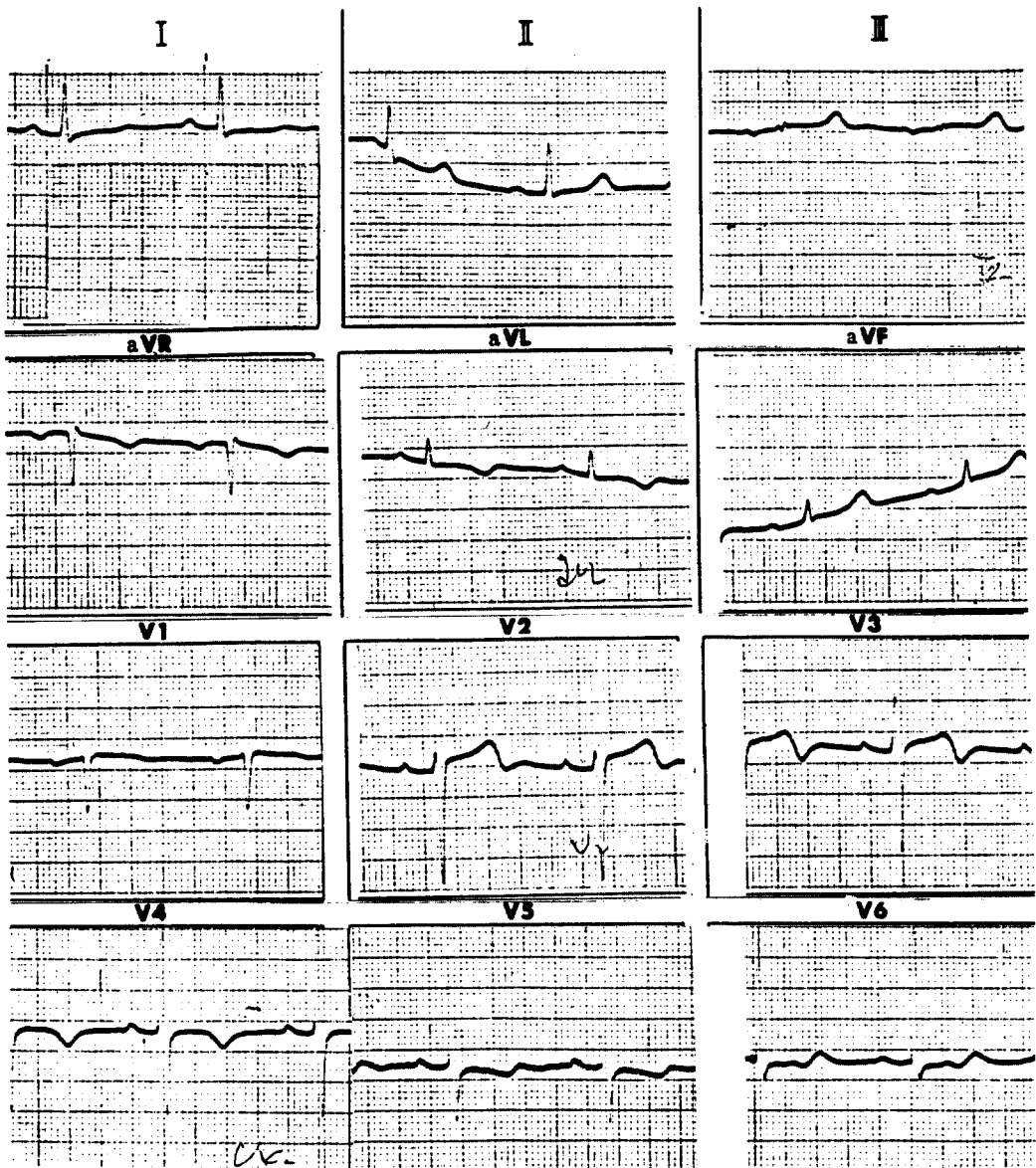


Fig. 1. An electrocardiogram shows slight depression of ST segments and inverted T waves in Lead I, aVL and V4-6 during an angina episode.

during the angina episodes (Fig. 1). A chest X-ray showed that the lungs were clear and the heart size was normal; the pulmonary vascular pattern was normal.

A cardiac catheterization was performed through the right femoral vein and artery. The pulmonary artery pressure was 50/20 mmHg and PCWP 20 mmHg; the aortic pressure was 165/85 mmHg and the LVEDP 15 mmHg; the cardiac output using the Fick oxygen method and the ejection fraction were 7.831 liter per minute and 0.75, respectively.

On left coronary angiography, a profound decrease in distal coronary pressure after coronary engagement with angina and the appearance of ST-T wave changes in monitoring, the electrocardiogram were noticed (Fig. 2).

There was no abnormalities on the left coronary angiography on the routine LAO and RAO projection. But a shallow LAO projection with a mild craniocaudal tilt of the left coronary angiography demonstrated a critical stenosis of the left main coronary ostium (Fig. 3). There were no abnormalities on the right coronary angiography.

The patient underwent coronary bypass graft surgery and has been doing well without occurrence of angina since the surgery.

DISCUSSION

Isolated coronary ostial stenosis appears to be a rare lesion and in one series⁴⁾ occurred with an incidence of 0.2% in a population of patients with coronary heart disease defined by coronary

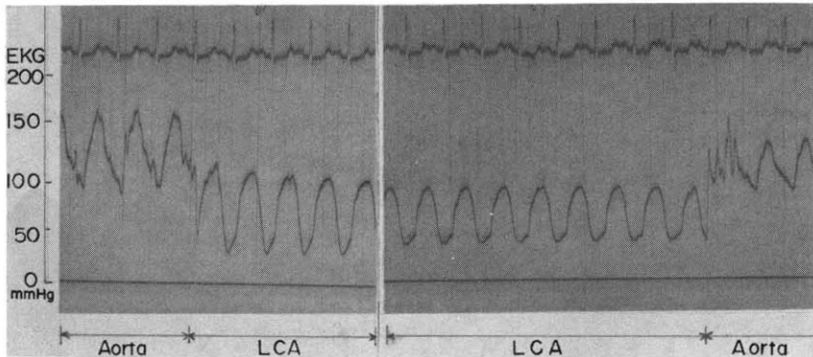


Fig. 2. A profound decrease in distal coronary pressure after left coronary engagement is seen, with the appearance of ST-T wave changes in monitoring the electrocardiogram.

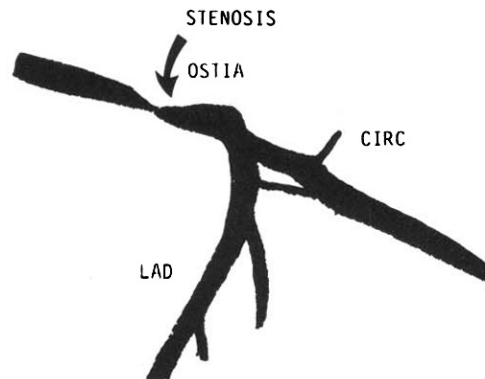
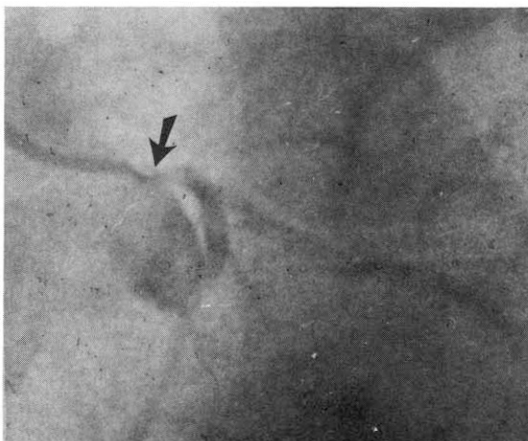


Fig. 3. The shallow LAO projection with mild craniocaudal tilt of the left coronary angiography demonstrates the critical stenosis of the left main coronary ostium.

ISOLATED LEFT CORONARY OSTIAL STENOSIS

angiography²⁻⁹). It occurs predominantly in women, usually before menopause (mean age 51 years).

Despite previous reports¹⁷ which have suggested a more frequent involvement of a right coronary ostium, recent analysis shows no significant differences in the distribution among left, right and bilateral ostial stenosis.

The etiology of isolated coronary ostial stenosis is entirely unknown. Among adults it has been assumed that atherosclerosis, particularly early atheroma, is the most likely cause of this lesion. Other investigations have suggested a congenital arterial hypoplasia complicated by progressive thickening of the aortic intima by advancing age¹⁷ or an inflammatory basis¹⁸. Histologic studies of nondiseased coronary ostia have suggested the presence of a circumferential sphincter-like muscle in the right coronary orifice of many patients, and it has been proposed that this may offer resistance to blood flow¹⁹. Occurring predominantly in women, usually before menopause, it is unknown whether humoral factors are important.

It is unclear whether the apparent rarity of this condition represents the true state of affairs or whether it is in part a reflection of the failure to report or recognize these cases at angiography. It is possible, using a preformed catheter, that the ostial lesion may be crossed during coronary engagement and remain undetected during subsequent cineangiography.

Thompson⁴ has suggested that a series of events should alert the cardiologist to the possibility of an ostial stenosis, including: 1) difficulty in cannulation of the coronary ostium; 2) a profound decrease in distal coronary pressure after coronary engagement with or without angina or the appearance of ST segment change in the monitoring electrocardiogram; and 3) failure to observe return of contrast medium into the sinus of Valsalva after intracoronary injection.

The use of an Amplatz catheter, approaching the coronary ostium from below, may help to prevent catheter impaction in this circumstance. Nonselective injection into the sinus of Valsalva usually provides inadequate definition of coronary ostium⁷ and, as reported previously, the left coronary ostium is best visualized in the shallow LAO projection (15 to 25°) with approximately 20° of craniocaudal tilt. Once a left ostial lesion has been demonstrated, only a limited number of other views are advisable, including an RAO projection.

Instant video playback facilities are essential in this regard. Nitroglycerin administered immediately prior to coronary angiography reduces the possibility of spontaneous as well as catheter induced spasm^{7,20}.

Clinically, the observed severe symptoms of NYHA functional class III and IV is significant clue, and strongly positive tests in the early stage, frequent in the presence of left main coronary ostial stenosis, should alert the clinician to the possibility of left main coronary artery disease. Suspecting the presence of left main coronary ostial stenosis prior to catheterization is important in reducing the risk of coronary angiography^{8,21-23} and improving the chances of correct diagnosis.

Angiography entails some risk because the catheter may readily occlude the stenotic ostium, resulting in a fall in pressure at the catheter tip, chest pain, dyspnea, diaphoresis, and a fall in systemic pressure. Frequently, the catheter and ostium are not viewed in a plane suitable for demonstration of the ostial stenosis. Unless the angiographer is aware of the possibility of ostial occlusion by the catheter, this may occur and lead to hypotension, arrhythmia, and cardiac arrest. When the junction of the catheter and ostium are viewed in the proper obliquity, the stenosis of the left main coronary artery can be observed. Although the ostial stenosis can be appreciated visually, the diagnosis is frequently made by pressure changes and by failure of the contrast medium to be flushed from the coronary artery until the occluding catheter is removed from the ostium.

By virtue of the extensive areas of myocardium placed in jeopardy, patients with coronary ostial stenosis, particularly of the left coronary artery, are at high risk of myocardial infarction and premature death. Whether the hemodynamic consequences of this lesion are different from those of left main coronary artery disease is entirely unknown. There is little doubt that coronary bypass graft is indicated for symptomatic left ostial stenosis, as left main coronary disease. Optimal therapy consists of coronary bypass to the LAD and one or more branches of the circumflex. Coronary bypass surgery does not carry a higher mortality, and further longitudinal studies will be required to determine the influence on long-term prognosis.

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