Left Coronary Artery-Left Ventricular Fistula With Acute Myocardial Infarction, Representing the Coronary Steal Phenomenon: A Case Report

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Abstract

A 59-year-old man presented with a left anterior descending coronary artery to left ventricular fistula manifesting as myocardial infarction, representing the coronary steal phenomenon. Electrocardiography showed poor R progression in leads V_1 through V_3 . The biochemical markers of myocardial injury were elevated. Creatine kinase level was 509 IU/l, creatine kinase MB isoenzyme (CK-MB) 47 IU/l, cardiac troponin T 0.62 ng/ml, myosin light chain 6.1 ng/ml, and myoglobin 142 ng/ml. Thallium-201 myocardial perfusion imaging with dobutamine stress showed a dobutamine-induced perfusion deficit of the anteroseptal wall of the left ventricle with 0.1 mV ST-segment depression in II, III, aVF, V_5 , and V_6 . The mean left anterior descending blood flow measured with the Doppler guidewire was increased from 211 to 378 ml/min. Selective coronary arteriography showed dominant left coronary artery with the contrast medium streaming into the left ventricle via a maze of fine vessels from the distal left anterior descending coronary artery. No critical stenosis of the left anterior descending coronary artery was observed. Administration of acetylcholine 100 μ g into the left coronary artery did not induce vasoconstriction of that artery. The fistula terminating in the left ventricle was ligated surgically and the patient became free of chest pain. Thallium-201 myocardial perfusion imaging with dobutamine stress revealed no perfusion deficit of the anteroseptal wall of the left ventricle.

The presence of coronary steal phenomenon was detected by dobutamine stress myocardial imaging.

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Key Words

- Congenital heart disease Coronary vessels (coronary left ventricular fistula)
- Myocardial infarction (coronary steal phenomenon)
- Radionuclide imaging (dobutamine stress myocardial imaging)

INTRODUCTION

Coronary artery fistula is an unusual anomaly. In one series of 55,856 coronary angiographic examinations in adults, there were only 24 cases with coronary artery fistulae¹⁾. The most common types

of coronary fistulae drain into the right side of the heart, such as the right atrium, right ventricle, pulmonary artery, or coronary sinus. Fistulous communication from the left coronary artery to the left ventricle is extremely rare. Myocardial ischemia and infarction, which are thought to be due to a

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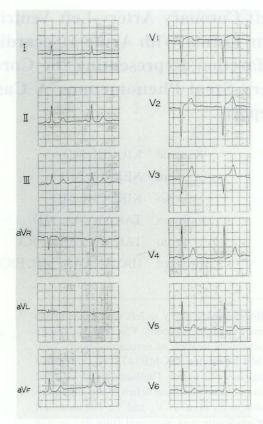
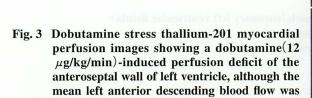


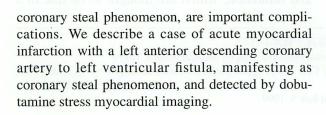
Fig. 1 Electrocardiogram on admission showing poor R progression in leads $V_1 - V_3$



elevated from 211 to 378 ml/min

Upper row panels: short. Lower row panels:

Vertical.



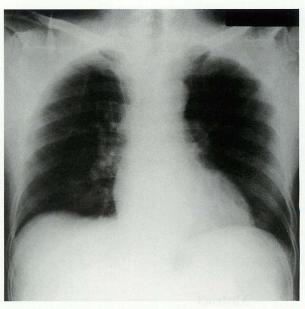
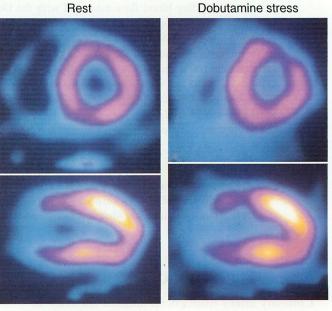


Fig. 2 Chest radiograph revealing a normal-sized heart with clear lung fields



CASE

A 59-year-old man was admitted to the Nippon Medical School Hospital with suspected myocardial infarction. He was well until 3 years before admission when he began to have recurrent

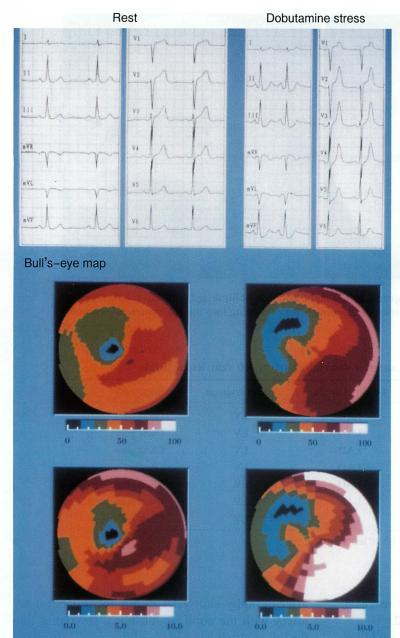


Fig. 4 Dobutamine ($12 \mu g/kg/min$) stress technetium-99 tetrofosmin scintigrams showing development of anteroseptal hypokinesis, *i.e.*, depressed regional ejection fraction (*middle row*) and wall motion (*lower row*) with 0.1 mV ST-segment depression in the II, III, aVF, V₅, and V₆ (*upper row*).

episodes of chest pain. This chest pain usually occurred with exertion and was relieved after about 3 min of rest. On July 8, 1998, the chest pain occurred on exertion, and was not relieved by sublingual nitroglycerin. He had a history of diabetes mellitus and hypertension, for which he had been treated with nifedipine (30 mg/day) and voglibose (0.6 mg/day). On admission, physical examination revealed blood pressure of 176/104 mmHg and heart rate of 49 beats/min. There was no heart murmur, gallop, or pericardial friction rub. Abdominal

examination showed no organomegaly or ascites. No lymphadenopathy was noted.

Laboratory data showed that the total white cell count was elevated at 9,600/mm³. The erythrocyte sedimentation rate was raised at 44 mm in the first hour. The biochemical markers of myocardial injury were all elevated, *i.e.*, creatine kinase level was 509 IU/*l*, creatine kinase MB isoenzyme 47 IU/*l*, cardiac troponin T 0.62 ng/m*l*, myosin light chain 6.1 ng/m*l*, and myoglobin 142 ng/m*l*. Electrocardiography showed poor R progression in

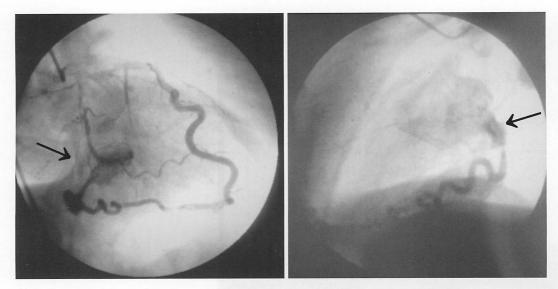


Fig. 5 Selective left coronary angiograms in the right anterior oblique (*left*) and lateral (*right*) views showing the enlarged left anterior descending artery draining into the left ventricle (*arrows*)

Case No. Authors Origin of fistula Site of drainage Symptoms 1 Vlodaver et al, 19753) LAD LV Chest pain 2 Sastri et al, 19754) LAD LV Chest pain 3 Midell et al, 19775) LAD LV Chest pain Arani et al, 19786) LV Chest pain, dyspnea 4 LAD 5 Cheng, 19821) LV Chest pain LAD Takiya et al, 19897) LAD LV Chest pain 6 LAD LV Chest pain 7 Present case

Table 1 Cases of left anterior descending artery-left ventricular fistula

LAD=left anterior descending artery; LV=left ventricle.

leads $V_1 - V_3(\mathbf{Fig. 1})$. Chest radiography revealed a normal heart with clear lung fields (**Fig. 2**).

Thallium-201 myocardial perfusion imaging with dobutamine stress showed a dobutamine (12 μ g/kg/min) -induced perfusion deficit of the anteroseptal wall of left ventricle (Fig. 3). The mean left anterior descending blood flow measured with the Doppler guidewire was elevated from 211 to 378 ml/min. Dobutamine (12 μ g/kg/min) stress technetium-99 tetrofosmin scintigraphy showed development of anteroseptal hypokinesis with 0.1mV ST-segment depression in leads II, III, aVF, V_5 , and V_6 (Fig. 4). Cardiac catheterization showed normal right and left heart pressures and no evidence of an intracardiac shunt. Left ventriculography revealed the left ventricle was normal in size as well as in contractile pattern. Selective coronary

arteriography showed a dominant left coronary artery with the contrast substance streaming into the left ventricle via a maze of fine vessels from the distal left anterior descending coronary artery (**Fig. 5**). No critical stenosis of the left anterior descending coronary artery was observed and administration of $100\,\mu\mathrm{g}$ of acetylcholine into the left coronary artery did not induce vasoconstriction of that artery.

The fistula terminating in the left ventricle was ligated surgically and the patient became free of chest pain. Cardiac catheterization performed post-operatively revealed no further evidence of a fistula. Furthermore, thallium-201 myocardial perfusion imaging with dobutamine stress revealed no perfusion deficit of the anteroseptal wall of the left ventricle.

DISCUSSION

Fistulous communication from the left coronary artery to the left ventricle is extremely rare. Fistulous communication from the left coronary artery to the left ventricle was found in only one case (0.6%) of a series of 163 cases of primary congenital coronary artery fistula². Only 6 cases of left anterior descending artery-left ventricular fistulae have been documented (**Table 1**) $^{1,3-7}$).

Myocardial ischemia and infarction, which are thought to be due to a coronary steal phenomenon, are important complications. A steal phenomenon, in which blood is shunted away from the distal coronary circulation by the fistulous connection, may cause reduced perfusion of the myocardium. In our patient, the coronary steal was manifested by the ST-segment depression on electrocardiogram following dobutamine infusion accompanied by chest pain, a reduction in regional wall motion with anteroseptal hypokinesis following dobutamine, and reversible perfusion deficit in the anteroseptal wall on thallium and technetium scintigraphy, although the mean left anterior descending blood flow was increased from 211 to 378 ml/min. Thus, we demonstrated the perfusion defect by dobutamine stress thallium scintigraphy, even though the

total coronary blood flow was increased during dobutamine infusion. Perfusion defects in stress thallium studies have been reported by Cheng¹⁾ and Ahmed et al.8 Kiso et al.9 treated a patient with right coronary-left ventricular fistula in whom the intra-operative mean right coronary blood flow was recorded at 315 ml/min and dropped to 35 ml/min after closure of the fistula, suggesting that a large amount of blood, which might correspond to more than 20% of the cardiac output, was stolen by the fistula. This apparently large amount of coronary blood flow that emptied directly into the left ventricular cavity could cause coronary steal, and subsequently myocardial ischemia and minor myocardial damage, since after surgical closure of the left coronary artery-left ventricular fistula, thallium-201 myocardial perfusion imaging with dobutamine stress procedure revealed no perfusion deficit of the anteroseptal wall of left ventricle and the patient became free of chest pain. In our case, coronary steal was demonstrated in a patient with acute myocardial infarction.

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·要 約-

冠動脈左室瘻による冠盗流現象が関与した急性心筋梗塞の1例

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症例は持続する胸痛を主訴に入院した59歳、男性、入院時、血圧176/104 mmHg、脈拍49/min、整、白血球9,600/mm³、CK509 IU/l、CK-MB 47 IU/l、トロポニンT $0.62 \, \mathrm{ng/ml}$ 、ミオシン軽鎖 $6.1 \, \mathrm{ng/ml}$ 、ミオグロビン $142 \, \mathrm{ng/ml}$. 心電図では $V_1 - V_3$ で poor R progression を認めた。 冠動脈造影では左冠動脈(前下行枝) 左室瘻を認めたが、有意狭窄およびアセチルコリンによる冠動脈の攣縮は認められなかった。ドブタミン負荷シンチグラムにおいて左冠動脈(前下行枝) の血流増加とともに前壁での灌流欠損を認めた。また、負荷時、胸痛を伴う心電図変化も認めた。後日、外科的に冠動脈瘻を結紮した。その後は胸痛発作なく、ドブタミン負荷シンチグラムにおいても灌流欠損は認められなくなった。

以上より,本症例の急性心筋梗塞の発症には,冠動脈左室瘻による冠盗流現象の関与が示唆された.

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