Hiroshima J. Med. Sci.
Vol.37, No.4, 171～173, December, 1988
HIJM 37-30

Left Ventricular Aneurysm with Mural Thrombus Occurring in a Patient with Patent Coronary Arteries

Taijiro SUEDA, Yuichiro MATSUURA, Hiroshi ISHIHARA, Yoshiharu HAMANAKA, Yasushi KAWAUE, Keiichi KANEHIRO and Takayuki NOMIMURA

The First Department of Surgery, Hiroshima University School of Medicine, 1-2-3, Kasumi, Minami-ku, Hiroshima 734, Japan

ABSTRACT

A patient who developed a left ventricular aneurysm without stenosis of his coronary arteries is presented. A 52-year-old man with a history of severe chest pain 12 years previously, was admitted to our hospital because of right femoral arterial thrombosis. ECG on admission showed QS pattern and ST segment elevation in leads V_1—V_4. Left ventriculography revealed that the anterior and lateral walls of the left ventricle were akinetic and an aneurysm had developed at the apex. However, the left anterior descending artery was patent without stenosis. Resection of the left ventricular aneurysm and thrombus was performed successfully under cardiopulmonary bypass. The cause of myocardial infarction without a coronary lesion is unknown, but it is assumed that coronary spasm and hypercoagulability may have had some role in the onset of myocardial infarction in this patient.

Key words: *Left ventricular aneurysm, Mural thrombus, Coronary artery without stenosis, Coronary spasm*

Total coronary arterial occlusion develops within a few hours of the onset of symptoms in patients with myocardial infarctions. Since recent thrombolytic therapy has proved effective for recanalization of occluded coronary arteries in 60 to 80% of patients with acute myocardial infarction[1], coronary arterial thrombosis must play a major role in the onset of this disease. Coronary atherosclerotic change is a precursor of myocardial ischemia, however 3 to 4% of patients with acute myocardial infarction have no stenosis of the coronary artery supplying blood to the area of infarction[3]. Coronary spasm is one cause of acute coronary occlusion, but the relationship between coronary spasm and infarction is not understood fully.

In the present report, we describe a patient who developed a left ventricular aneurysm with mural thrombus without coronary arterial stenosis who was treated successfully by surgery.

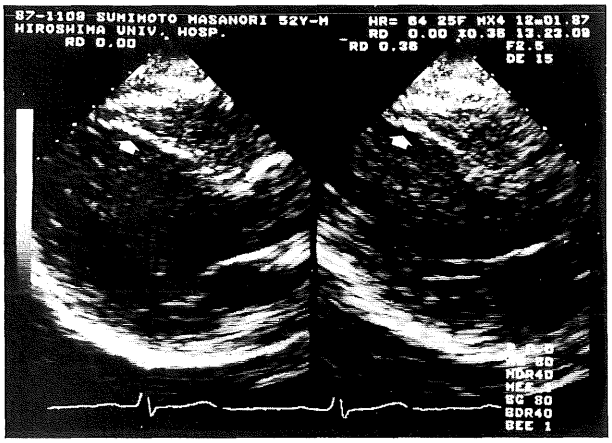
CASE REPORT

A 52-year-old man visited our emergency department with right femoral arterial thrombosis. He had had an episode of severe chest pain 12 years earlier and a transient cerebral ischemic attack one year previously. He didn't have complained anginal attack after the first episode of severe chest pain, but he had sometimes experienced chest discomfort on effort.

On physical examination, the right femoral arterial pulse was absent. The heart sounds was normal and no heart murmur was detected. ECG on admission showed QS Pattern and ST elevation in the precordial leads V_1—V_4. Echocardiogram (B-mode) showed a dilated left ventricle with dyskinesis of antero-septal wall and akinesis of lateral wall of the left ventricle. A mural thrombus (diameter 20 × 20 mm) also was found at the base of the left ventricular aneurysm. Left ventricular contractility was severely depressed (Fig. 1).

After emergency femoral arterial thrombectomy under local anesthesia, ischemia of right lower leg was relieved. The thrombus seemed to originate from the mural thrombus in the left ventricle. Coronary cineangiography was performed 5 days after thrombectomy and showed a patent anterior descending branch of the left coronary artery, supplying blood to the infarcted myocardium (Fig. 2-a). Other branches also were intact and without stenosis. Collateral flow to the infarcted area was not found, either. Coronary cineangiogram after sublingual nitroglycerin administration revealed slight dilatation of all three coronary vessels and no antheroscherosis in any branch (Fig. 2-b). An ergonovine test was not performed, since coronary spasm as the etiology of the myocardial infarction had not been ruled out.

Thoracotomy confirmed that the left ventricular aneurysm was located in the apex of the left ventricle and a large segment of antero-lateral wall of



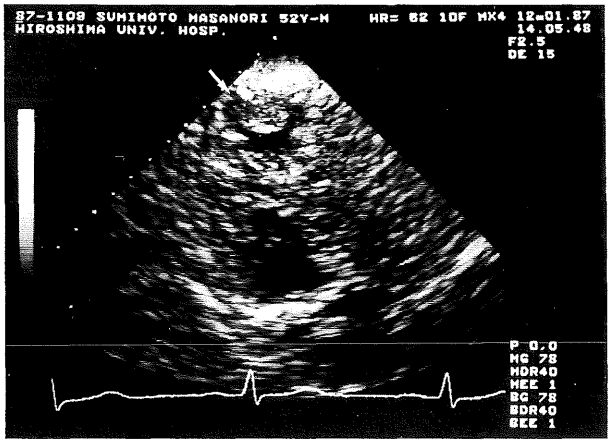


Fig. 1. Echocardiogram of left ventricular aneurysm: contractility of left ventricle was severely depressed and ventricular wall motion of antero-septal portion was dyskinetic (⇧). Short axis view revealed mural thrombus (diameter 20 × 20 mm) at the base of left ventricle (⇧).

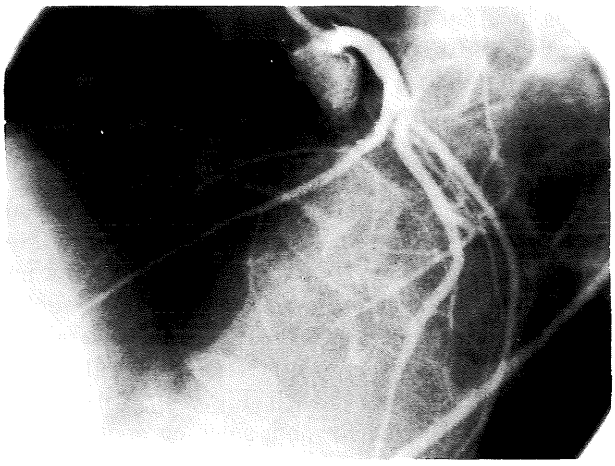


Fig. 2-a. Left coronary arterial cineangiography (hepato-clavicular position): CAG showed a patent anterior descending branch of the left coronary artery without stenosis.

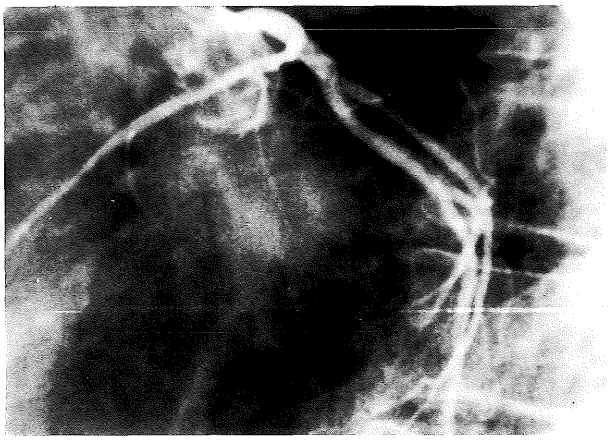


Fig. 2-b. Left coronary arterial cineangiography (After sublingual nitroglycerin administration) CAG revealed slight dilation of left anterior descending branch but no prominent stenosis.

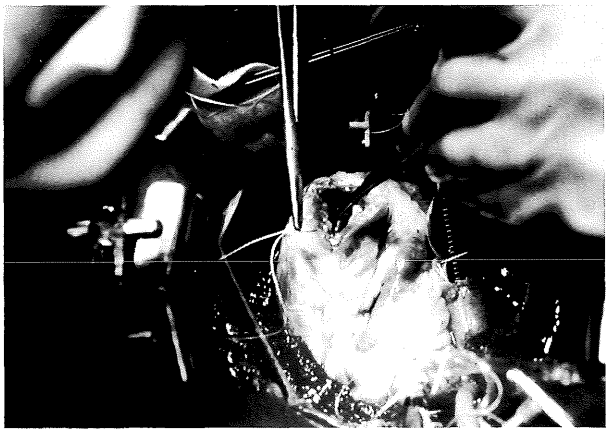


Fig. 3. Operative finding of the left ventricular aneurysm. The aneurysm was located anterior and apical portion of the left ventricle and mural thrombus (30 × 20 mm in size) was adhered to the aneurysmal wall.

the left ventricle was akinetic. The size of aneurysm was 6 × 8 cm in diameter and the resected size of the aneurysm was 3 × 6 cm. The organized thrombus (3 × 2 cm in diameter) was adhered to the apical wall of the aneurysm. Aneurysmectomy and thrombectomy were performed successfully under extracorporeal circulation with moderate hypothermia (Fig. 3). Heart action remained hypokinetic after amputation of the aneurysm, but cardiopulmonary bypass could be discontinued with the aid of dopamine administration (5μg/kg/min).

The postoperative course was excellent without complications and neither heart failure nor arrhythmias were encountered. Pathological examination revealed that the resected specimen consisted of the organized thrombus and the degenerative myocardial tissue of the left ventricle, especially fibrotic degeneration of the endocardium. The patient was

discharged from the hospital on the 19th postoperative day, and a postoperative echocardiogram showed no thrombus in apex of the left ventricle. He has experienced no further episodes of arterial thrombosis and chest discomfort under medication with a ca^{++} antogonist and antiplatelet agent.

DISCUSSION

Although the pathophysiology of myocardial infarction has yet been explained fully, there is no doubt that complete coronary occlusion is the basic pathology in the very early hours of infarction[1]. The coronary arteries supplying blood to the area of infarction usually have stenosis or occlusion. However, it has been shown that 3 to 4% of patients with myocardial infaction have no stenosis in the coronary artery supplying the area of infaction[3]. Obstructive thrombi are found in 80 to 97% of patients with acute infarction[2], in addition, most patients (75 to 98%) have coronary atherosclerotic changes, such as intimal destruction, laceration, ulcer formation and bleeding into the intima.

What is the cause of complete obstruction of a coronary artery without organic stenosis? The role of coronary spasm in anginal attacks already has been proved[5], but it is unknown whether coronary spasm has a direct relation to infarction. In patients with variant angina, infarction is relatively rare (0.4%)[4], considering the frequency of episodes of spasm. Even among paients with ergonovine-evoked spasm, infarction occurs rarely. Some reports give a low incidence (below 10%) of recanalization after intracoronary nitroglycerin in these patients with acute infarction[6]. Therefore the event which initiates myocardial infarction must consist not only of spasm but also of blood coagulation and acceleration of coagulation. Of course, a stenotic coronary lesion which could not be detected by coronary cineangiography may have been present.

Several hours of continuous coronary occlusion is necessary for the myocardium to infarct and a ventricular aneurysm to form. Hyperlipidemia and gout could increase blood coagulability and result in coronary thrombosis. Regardless, left ventricular aneurysms without coronary artery disease are rare, and the pathogenesis of coronary occlusion is unclear.

We consider that one of the etiology of infarction in this patient might be coronary spasm and hypercoagulability due to hyperlipidermia, and hemoconcentration is attributable to the onsent of infarction, too.

(Received August 1, 1988)
(Accepted November 15, 1988)

REFERENCES

1. **Dewood, M., Spores, J., Hensley, G.R., Simpson, C.S., Euguster, G.S., Surtherland, K.I., Grunwald, R.P. and Shield, J.P.** 1983. Coronary arteriografic findings in acute transmural myocardial infarction. Circulation **68 suppl. 1**: 39—49.
2. **DeWood, M., Spore, J., Notshe, R., Mousei, L., Burroughs, R., Golden, M. and Lang, H.** 1980. Prevalence of total coronary occlution during the early hours of transmural myocardial infarction. N. Eng. J. Med. **303**: 897—903.
3. **Maseri, A., Chierchia, S. and Davies, G.** 1986. Pathophysiology of coronary occlution in acute infarction. Circulation **73**: 233—239.
4. **Maseri, A., Severi, S., DeNes, D.M., Chierchia, S., Marzilli, M., Ballestra, A.M. and Parodi, O.** 1978. "Variant" angina: one aspect of a continuous vasospastic myocardial ischemia. Am. J. Cardiol. **42**: 1019—1023.
5. **Oliva, P.B., Potts, D.E. and Pluss, R.M.** 1978. Coronary arterial spasm in Printzmetal angina. N. Eng. J. Med. **288**: 745—751.
6. **Rentrop, P., Blanke, H., Karsch, K.R., Kaiser, H., Kostering, H. and Leitz, K.** 1981. Selective intracoronary thrombolysis in acute myocardial infarction and unstable angina pectoris. Circulation **63**: 307—312.