---Case Report---

A Case of Coronary Embolism in Mitral Steno-insufficiency: Clinical and Pathological Survey

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This is a report of a case of coronary and cerebral embolism occurred in a patient with mitral steno-insufficiency. Clinical diagnosis of coronary and cerebral embolism was made by evidences of left hemiplegia and myocardial infarction shown by electrocardiogram. Clinical diagnosis was confirmed at autopsy and postmortem coronary angiography disclosed the location of the coronary embolus.

Pathologically, the diagnosis of coronary embolism was first made by Virchow1, clinically by Hamman2. Up to the present, approximately one hundred cases of coronary embolism have been presented in the world literatures. Oakley et al.3 reported that coronary embolism was neither uncommon, nor usually fatal and that it is most important cause of angina pectoris in a patient with mitral stenosis, but others reported that it was rare disease. There are very limited numbers of cases diagnosed clinically with pathological confirmation. Only a few cases of them have been reported in Japanese literatures.

We observed a patient with mitral steno-insufficiency who presented during his admission left hemiplegia and myocardial infarction following cerebral and coronary embolism. Clinical diagnosis of coronary and cerebral were confirmed at autopsy.

Case Report

Twenty-six-year-old man was admitted to the Kobe University Hospital because of palpitation and dyspnea. At 14 years of age, he was found to have a cardiac disease, but had not any treatment because of no complaint. At the age of 23, he first noted of palpitation, dyspnea and edema in face after a severe exertion and when he had a common cold. In Jan. and Aug. 1954, he had an episode of cough with bloody sputum and edema in face. Those symptoms were relieved in a few days. Since Nov. 24th, 1964, he had noted of decrease in urine volume resulting in edema in face. There was palpitation, dyspnea after slight exertion. He was admitted to Kobe University Hospital on Dec. 2nd, 1964.

At the time of admission his face was puffy, but with no distress. The blood pressure was 135/80mmHg. The pulse was 68 per min., re-

(Received for Publication, January 18, 1968)
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angular and good in tension. The heart was enlarged to both sides. A systolic murmur and diastolic rumble were audible over the apex. The second pulmonic sound was accentuated. The lung were clear. The liver was 2 finger-breadth below the right costal margin palpable. There was pitting edema on the lower extremities. The deep tendon reflexes were all normal and no pathologic reflexes were elicited. Urine volume through hospitalization was from 1050 to 2000 ml per day. Leucocyte count on admission was 5500. Sedimentation rate was 3 mm per one hour. The serologic tests for syphilis was negative. The C-reactive protein test was negative. The Anti-Streptysin 0-titer was 166 Todd Unit. The rheumatoid arthritis test was positive. A chest-film revealed a cardiac enlargement to both sides. An electrocardiogram showed right axis deviation, horizontal heart position, auricular fibrillation with ventricular premature beat and right ventricular hypertrophy (Fig. 1).

He was diagnosed as mitral steno-insufficiency with auricular fibrillation. At 6 A.M. on the 6th hospital day, the patient fell down and went into unconsciousness. Physical examination revealed comatous man with pale face and left hemiparesis. An electrocardiogram taken immediately after the stroke showed abnormal Q waves and elevated ST segments in leads II, III, aVF, depressed ST segments in leads I, aVL, V₅₋₆ (Fig. 2). Laboratory data after the attack, showed leucocyte count 17,000 (8500 before the attack), S-GOT 31 Karmmen unit (25 unit before the attack) and S-GPT 39 Karmmen unit (25 unit before the attack). Clinical diagnosis was made of coronary and cerebral embolism. The patient fell into coma gradually. Immediately after the spell, the blood pressure was 125/80 mmHg, the pulse 72 per min. The patient did not respond to the cardiovascular agents and the anticoagulant.

![Electrocardiogram](image)

**Fig. 1.** Electrocardiogram on admission.

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At 10 p.m. on the day, a pulmonary edema occurred and died 16 hours after the onset of the episode.

Postmortem coronary angiogram with 76 per cent Urographin confirmed that there were two occlusions in the right coronary artery; the one extending as long as about 3 cm in the posterior descending branch, the another of a saddle-like shape in the acute marginal branch (Fig. 3, 4).

The autopsy findings were as follows; the heart weight 520 g. The myocardium of the left ventricle was 14 mm in thickness. Mitral valve leaflets were markedly thickened with adhesion with each other. A cicatrical and calcification alteration of the mitral valve were observed. Mitral orifice was 13 mm in diameter. Ulceration measuring 20 mm in diameter was observed on the auricular surface of the valve and covered with dusty brownish yellow thrombus (Fig. 5). The chorda tendinae were slightly thickened and shortened. The wall of the right ventricle measured up 7 mm in thickness. The coronary arteries showed no atherosclerotic changes on gross finding. There were two dark-ed thrombi in the right coronary artery and the sites of occlusion were coincided with the findings of the postmortem coronary angiogram (Fig. 6, 7). Sections through the myocardium of the posterior wall of the both ventricles revealed haemorrhages resulting in necrotic changes. Microscopic examination of the coronary arteries disclosed some fibrous thickening of intima, but no sclerotic changes were detected in the occluded areas. There was no fibrotic connection between the intima and the thrombus. Bleeding and disappearance of muscle fibers were noted on the microscopic examination. Necrotic changes were observed into advanced areas of the bleeding. In cerebrum, a softening was revealed in the right internal capsule, putamen and caudate nucleus. On histologic examination of the involved areas, there were the ischemic changes of the nerve cells, and the degeneration, spongy changes of

Fig. 2. Electrocardiogram after the attack of left hemiparesis.
Fig. 3. Postmortem coronary angiography with 76% Urographin. (Vento-dorsal projection)

Fig. 4. Diagram of postmortem coronary angiography. There are two sites of occlusion in the right coronary artery. (arrows)

Fig. 5. Auricular surface of mitral valve with ulceration and brown-yellow thrombus.
the glia cells. No sclerotic changes were observed in the basilar artery. Old infarctions in the both kidney were detected. No other noteworthy finding were observed than congestive changes in the other organs.

**Comment**

Coronary embolism is a rare disease. Wenger and Bauer\(^4\) reported that in the years through 1929 to 1957 there have been, at the Mount Sinai Hospital, eleven well documented cases of coronary artery embolism which were confirmed by postmortem examination, representing an incidence of 0.06 per cent in 17,469 consecutive autopsy cases. Since Cheng's report\(^5\), a number of those of coronary embolism associated with mitral valve disease have been published in the world literatures\(^6\)\(^-\)\(^9\). In the Japanese literature, up to the present time, only two cases were reported clinically and pathologically confirmed; one with mitral valve disease by Miyahara\(^8\), the other with bacterial endocarditis by Tanabe et al.\(^1\)\(^1\).

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The rarity of embolization to the coronary arteries has been attributed to (1) the difference between the caliber of the aorta and that of the coronary, (2) the situation of the coronary vessels at root of the aorta, (3) the right angle emergence of the coronary arteries, (4) the bulk and swiftness of the blood current in this portion of the aorta, and (5) the fact that the major part of coronary filling occurs during diastole.

A review of literatures by Wenger and Bauer\(^4\) showed that bacterial endocarditis was the most common underlying disease, the incidence being 47 out of 74 cases (63.5\%). On the other hand, embolus due to intracardiac thrombus without endocarditis is considerably rare, and the incidence is 10.9 per cent of coronary embolism. Such underlying disease as a laetic aortitis, aortic atherosclerosis and paradoxical thrombus were all rare.

Embolic occlusion of the left coronary artery is more frequent (about 80 per cent) than of the right coronary artery\(^5\)\(^,\)\(^1\(^2\)\(^\). But Wood\(^1\(^8\)\) and Stuckey\(^1\(^4\)\) found that ischemic changes in the
electrocardiogram after effort in mitral stenosis were usually greatest in posterior leads and therefore Oakley et al. pointed out with interest that six out of eight myocardial infarcts in Wenger and Bauer’s cases of coronary embolism were posterior. An electrocardiographic changes of our case showed also posterior myocardial infarction and autopsy revealed coronary embolus situated right coronary artery.

Cheng et al. and Stuart et al. pointed out the convenience of thickening of embolism in cases of myocardial infarction in young people without a history of angina pectoris and in the presence of atrial fibrillation and mitral stenosis. It is difficult to differentiate a coronary thrombosis and embolism clinically, but the clinical diagnosis of coronary embolism in this case was strongly suggested by the following: (1) no history of angina pectoris beforehand and sudden onset, (2) concomitant incidence of coronary occlusion with cerebral attack, (3) young male, and (4) the presence of mitral valve disease, probably due to rheumatic origin. Karsner said that the definite diagnosis of a coronary embolus was impossible unless the source of the embolus was clearly indicated and the arterial wall at the site of the occlusion was healthy. The clinical and pathological data in our case are consistent with it and diagnosis of coronary embolus was convincingly established. Wood reported that mitral valve disease with atrial fibrillation was frequently associated with systemic embolization (6–14%) and that cerebral embolism was the most frequent one. In our case, cerebral and coronary embolism occurred at the same time, therefore the symptoms of coronary embolism were masked by the unconsciousness following cerebral embolism. However, an electrocardiographic changes suggested coronary embolism.

Location of the coronary embolus in this case was disclosed by means of postmortem coronary angiography. Rodriguez et al. reported that a postmortem angiographic study disclosed 3.5 times more coronary occlusion than were found in control routine autopsies. They concluded that postmortem coronary angiography was useful.

**Summary**

This is a report of a case of coronary and cerebral embolism in a patient with mitral steno-insufficiency. Clinical diagnosis of coronary and cerebral embolism was made by the evidence of left hemiparesis and myocardial infarction. The source of the embolus was thought to be mitral valvular thrombus. The antemortem diagnosis was confirmed by autopsy.

**REFERENCES**