

## EXTENSIVE MYOCARDIAL ABSCESS COMPLICATED BY MYOCARDIAL INFARCTION

Yoshio NAKAZAWA, Yutaka ISHIBASHI, Yoshio MATSUNO, Hirotohi TODA, Hideki YOSHIGANE, Shigefumi MORIOKA, Katsutoshi MORIYAMA and Takayuki HARADA\*

Fourth Department of Internal Medicine and First Department of Pathology\*, Shimane Medical University, Izumo 693, Japan

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A 65-year-old woman had an extensive myocardial abscess due to *Streptococcus pyogenes* complicated by an acute myocardial infarction. Fifteen other cases of isolated bacterial myocardial abscess following myocardial infarction have been reported. In these cases the abscesses were small and few in number. Our case had extensive and multiple myocardial abscesses in autopsy. Myocardial abscess was not diagnosed clinically. No primary focus of infection was found.

Myocardial abscesses are uncommon and the formation of abscesses with an acute myocardial infarction is even more uncommon. Their presence is usually obscured by an associated overwhelming generalized sepsis and is not discovered until autopsy.

Fifteen cases of bacterial abscesses in areas of acute myocardial infarction and one case in the area of myocardial scar have been reported to our knowledge. This report concerns a patient with *Streptococcus pyogenes* myocardial abscess followed by acute myocardial infarction.

### CASE REPORT

A 65-year-old woman was admitted to a hospital with dyspnea and fever. She had a history of transient left hemiplegia, mitral regurgitation and congestive heart failure. Two days prior to admission she complained of chills, fever and dyspnea. One day prior to admission she noticed a transient left hemiparesis. She had a temperature of 37.1°C, blood pressure of 142/82 mmHg, an irregular pulse of 72 beats per minute, respirations of 42 per minute, and peripheral edema. No rale was audible. There was a grade 3/6 holosystolic murmur loudest at the apex.

The white blood cell count was 14,600/mm<sup>3</sup> and the blood urea nitrogen was 75 mg/100 ml. The electrocardiogram showed atrial fibrillation, 2 mm to 10 mm ST segment elevations from V<sub>1</sub> through V<sub>4</sub>, and ST segment depression in II, III, and aV<sub>F</sub>. A x-ray film of the chest showed an enlarged heart. While she was breathing oxygen containing air, her arterial blood pH was 7.20, PO<sub>2</sub> 97 mmHg, and PCO<sub>2</sub> 30 mmHg.

Amikacin sulfate and cefotiam were given. Deslanoside, diuretics, and oxygen were started for deteriorating heart failure. Cardiac enzymes were compatible with a recent myocardial infarction. The maximum creatine phosphokinase was 584 IU/L, the maximum lactic dehydrogenase(LDH) 1706 IU/L, the maximum serum glutamic oxalacetic transaminase

(SGOT) was 177 IU/L. Blood taken on admission yielded *Streptococcus pyogenes*. The spiking fever continued. On the sixth hospital day she had chest pain. Demonstrated ST segment elevations in I, aV<sub>L</sub>, and V<sub>5,6</sub> again and idioventricular rhythm appeared. She was transferred to our hospital.

At admission to our hospital the patient appeared pale. The pulse was 40 per minute and irregular, and blood pressure 140/50 mmHg. Rales were audible over both lung fields. There was a grade 3/6 holosystolic murmur loudest at the apex with radiation to the axilla. There were hepatomegaly and peripheral edema.

The white cell count was 14,300/mm<sup>3</sup>, with 90 per cent neutrophils, 3 per cent band forms, 4 per cent lymphocytes, and 3 per cent monocytes. Creatinine 3.8 mg/100 ml, and blood urea nitrogen 160 mg/100 ml. SGOT was 29 IU/L, LDH 879 IU/L and creatine kinase 202 IU/L. An electrocardiogram demonstrated atrial fibrillation with atrioventricular block; poor R wave progression was present in leads V<sub>1</sub> through V<sub>4</sub>; abnormal Q waves were present in I, aV<sub>L</sub> and V<sub>5,6</sub>.

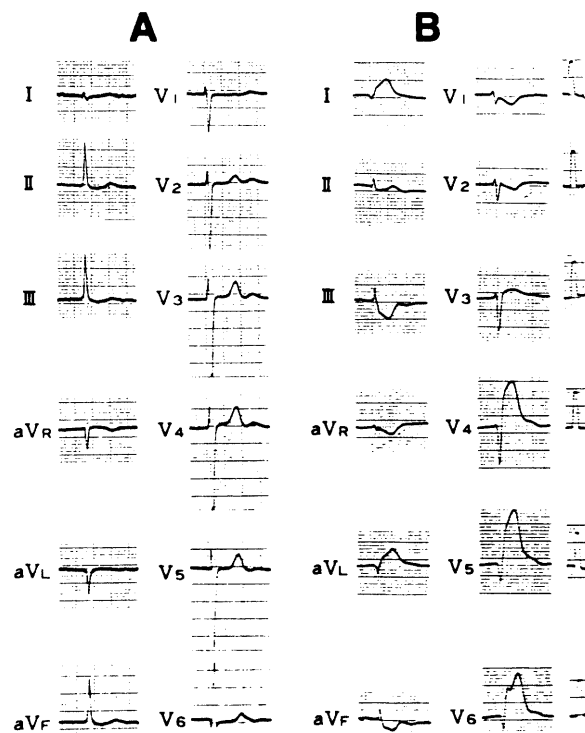


Figure 1. A; Electrocardiogram taken three days before first admission. This electrocardiogram was taken by chance. B; Electrocardiogram taken on first admission.

X-ray films of the chest showed pulmonary congestion and cardiomegaly. A specimen of arterial blood, drawn with breathing room air, disclosed that pH was 7.46, PO<sub>2</sub> 60 mmHg, PCO<sub>2</sub> 27 mmHg.

Antibiotic therapy was changed to cefoperazone. Digitalis was discontinued due to bradycardia. On account of congestive heart failure, dopamine and nifedipine were begun. Furosemide, isosorbide dinitrate and oxygen were continued, and a temporary pacing catheter was inserted due to high degree atrioventricular block. Subsequently the dyspnea gradually improved and premature ventricular contractions disappeared. Blood cultures were negative. On the second hospital day she passed large tarry stools. On the fourth hospital day ventricular fibrillation suddenly occurred. Resuscitative efforts were unsuccessful.

Autopsy, excluding the head, was performed. The aortic valve had an organized vegetation and thrombi.

Many thrombi were present in the atrial wall, left auricle and mitral valve. A thrombus with large abscess was located in the right auricle. There was myocardial necrosis and abscess formation with both

leukocytes and cocci in the myocardium of both ventricles. Abscesses extended to the epicardial surface and purulent epicarditis was seen. There were multiple abscesses in the midportion of the left ventricle, apex and outflow tract of right ventricle. Gram positive cocci were seen in myocardial abscesses and myocardial thrombi. Recent and old thrombi were present in the mid portion of left anterior descending coronary artery and diagonal branch. Hemorrhages were seen in the anterior wall of the left ventricle. Recent infarction and clumps of bacteria, the same as those seen in the heart, were observed in the spleen. The lung and adrenal gland also had microthrombi. A gastric ulcer and duodenal ulcer were seen. The peritonitis around the duodenum due to duodenal perforation extended to the gallbladder.

## DISCUSSION

There are few patients with myocardial abscesses complicating acute myocardial infarction. Fifteen cases have been reported(1-13). Twelve of the fifteen cases were documented unexpectedly at autopsy. Three cases were diagnosed at surgery. Concerning myocardial abscess complicating old myocardial infarction, Weisz reported an isolated myocardial abscess due to *Bacteroides fragilis* developed in the scar of myocardial infarction(14). The majority of reported cases had small abscesses and they were few in number.

But our patient had extensive and multiple myocardial abscesses. Electrocardiographic findings in our case might suggest extensive myocardial damage. But we judged that those findings signified myocardial infarction because of the following reasons; first, the changes in electrocardiogram and cardiac enzymes were compatible with a recent myocardial infarction, although electrocardiogram three days before first admission showed results within normal limits. Second, there was a fresh thrombus in the left anterior descending artery at autopsy. Third, hemorrhages were seen in the anterior wall of the left ventricle.

It is difficult to decide whether myocardial infarction followed myocardial abscess or myocardial abscess followed myocardial infarction. But we thought that myocardial infarction followed myocardial abscess. Because bacteremia and infectious symptoms such as chill and fever preceded electrocardiographic and enzymatic changes. Besides she had multiple infarction in the spleen, the lung, and the kidney. It seems that mural thrombi over the abscesses developed and multiple embolism occurred. If it is true that myocardial infarction followed myocardial abscess, this case is very rare. We could find no other report of myocardial abscess, this case is very rare. We could find no other report of myocardial abscess followed by myocardial infarction.

In summary, a case of acute myocardial infarction with complicated diffuse and extensive abscesses of the myocardium was reported. The infecting organism was *Streptococcus pyogenes*. No definite course of infection was found.

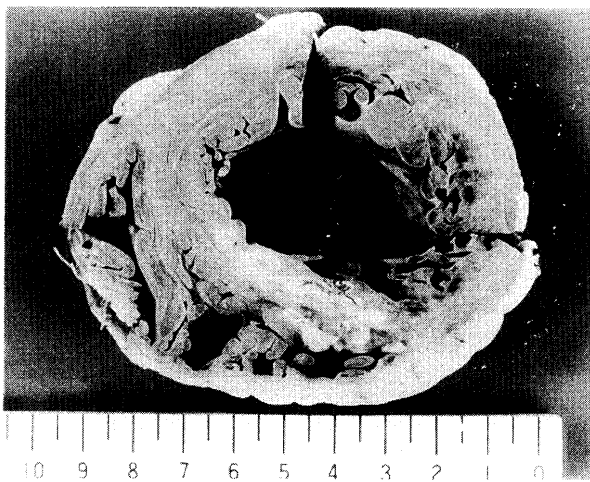


Figure 2. Transverse section of myocardium, showing myocardial abscesses in ventricular septum and hemorrhage in anterior, lateral wall and papillary muscle.

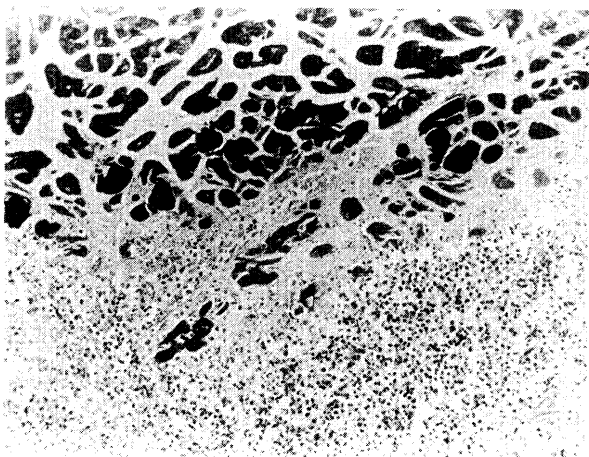


Figure 3. Myocardial abscess of anterior wall. Dark masses at top are clumps of organisms. Myocardium diffusely infiltrated with inflammatory cells.

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