

Case Report

A case of infected left ventricular thrombus as a complication of myocardial infarction

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Abstract

A 68-year-old man was admitted with high fever and dyspnea. Under the diagnoses of pneumonia and heart failure, antibiotic and diuretic therapies were performed, but elevated CRP of more than 6 mg/dl persisted. An apical type of hypertrophic cardiomyopathy-like structure was observed in echocardiography. On the 18th hospital day, he died suddenly of fatal arrhythmia, and infected apical left ventricular (LV) thrombus complicated by old inferior and acute anterior myocardial infarction was found in the autopsy. In this case, the LV mural thrombus was adhered around the apical portion of the myocardium and presented inflammatory change, which might have caused the fatal arrhythmias.

(Key words: Infection, left ventricular thrombus, myocardial infarction)

Case Presentation

A 68-year-old man was sent to our hospital with high fever and dyspnea. On the day of admission, he presented resting and exertional dyspnea. Bacterial pneumonia was suspected and the patient was immediately admitted to our hospital. Upon admission, his oxygen saturation in arterial blood was 91% (room air) and the laboratory data showed increased leukocyte counts 13550/ μ l (neutrophils 83%), total bilirubin 2.1 mg/dl, AST 181 U/l, ALT 40 U/l, ALP 145 U/l, LDH 622 U/l, C-reactive protein (CRP) 8.23 mg/dl, hemoglobin A1c 6.9%, serum glucose 229 mg/dl, and fibrin degradation product (FDP) 47 μ g/ml. An electrocardiogram showed negative T waves from leads V4 to V6, and chest x-ray showed mild infiltration in the right lung and enlarged cardiac shadow.

Under an initial diagnosis of pneumonia and heart failure, antibiotic (imipenem/cilastatine 1g per day and per oral administration of clarithromycin 400 mg/day) and diuretic therapies were performed. On the 2nd hospital day, the inflammatory markers increased to leukocytes 13810/ μ l (neutrophils 78.1%) and CRP 23 mg/dl. The following day, the condition of the patient deteriorated and he was intubated emergently. The electrocardiogram at the time of worsening showed QS pattern from V1 to V5 and ST depression from V4 to V6 (**Fig.1**). Echocardiography revealed hypertrophy of the apical wall, and the LV wall motion was generally reduced, especially at the apical portion. The LV ejection fraction was 43% by the Teichholz method, and moderate mitral and tricuspid regurgitations were observed.

On the 8th hospital day, ventricular fibrillation appeared suddenly after the patient accidentally extu-

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bated himself, and cardiopulmonary resuscitation was performed. Considering the possibility of aspiration pneumonia, the antibiotics were changed to cefozopran (CZOP) 3g and clindamycin (CLDM) 1200 mg. After that, the condition of the patient improved, but a moderate level of CRP (6~8 mg/dl) remained despite the improvement of his general conditions. At the 17th hospital day, ventricular tachycardia and ventricular fibrillation started suddenly. We tried to stop these effects by DC counter shock and i.v. use of amiodarone, but they were not controllable. Cardiopulmonary resuscitation was performed for 90 minutes, but he died. Informed consent was obtained for an autopsy, which was performed on the next morning.

The results of the autopsy were as follows: acute myocardial infarction (MI) at the antero-septal wall; old MI at the inferior wall; a ball-like thrombus that adhered all around the surface of the apical portion of the LV myocardium (**Fig.2**), and that showed organic and inflammatory changes (**Fig.3**); arteriosclerosis and nephrosclerosis; and pulmonary congestion and mild pneumonia.

Figure 1 : Electrocardiogram on June 25th.

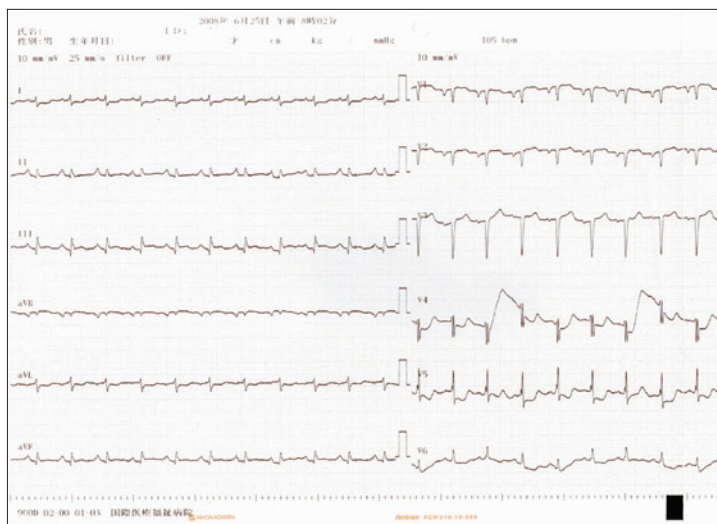


Figure 2 : Cut surface of the LV showing myocardial infarction and thrombus in the ventricle. The left panel shows a fresh specimen and the right shows a fixed specimen with formalin. The arrows indicate ball-like thrombus adhered all around the surface of the apical portion of the LV myocardium.

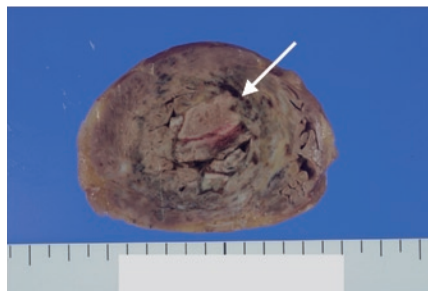
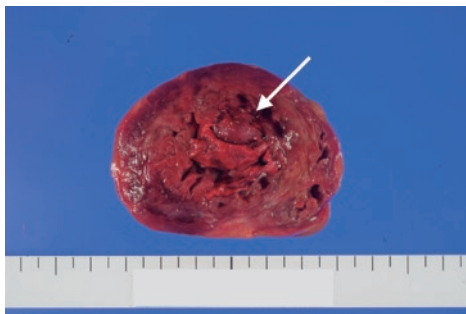
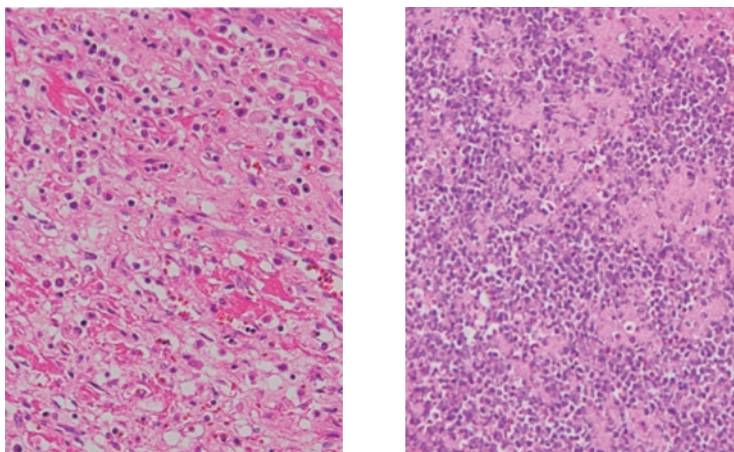


Figure 3 : Inflammation of organizing thrombus and altered leukocytes in the thrombus

The left panel shows inflammation of the organizing thrombus, and the right panel shows altered leukocytes in the thrombus; both panels provide evidence that the thrombus was infected.

**Discussion**

We reported a case of infected LV thrombus as a complication of acute anterior and old inferior MI. There have been 10 case reports describing infected LV thrombus. In 8 of these cases, bacteria were found in blood culture and resected thrombus, and most of them were gram-negative rods such as salmonella, *E. coli*, pseudomonas, Proteus, or Klebsiella. In cases of infected LV mural thrombus, a very high rate of mortality has been reported because an accurate diagnosis is sometimes difficult, and it is hard to control infection. Because conventional antibiotic therapy is not very effective, surgical resection is the best therapy as soon as the condition is diagnosed^[1-3].

In the present case, the diagnosis of MI was difficult because of the lack of information of previous MI, the poor presentation of symptoms, and the atypical ECG change (ST segment depression in the leads from V4 to V6). The quality of the echocardiography was poor because it was obtained using a portable machine under mechanical ventilation. The initial presentation of this patient (vomiting, high fever and dyspnea) further made it difficult to obtain a correct diagnosis.

We suspected some type of thrombus formation at the time the abnormally high FDP value was detected. However, we could not detect large LV thrombus by echocardiography. LV thrombus is not a particularly rare complication of MI. In the GISSI-3 database of 8326 patients^[4], LV thrombus was present in 5.1% of patients overall, and in 11.5% of those with an anterior wall infarction. However, there has been no report of a ball-like thrombus adhering all around the surface of the LV myocardium. Due to its special characteristics that LV cavity was connected by thrombus like a hinge, and the LV cavity could not be enlarged despite the cardiac cause of death in the autopsy (**Fig.2**). Retrospectively, the atypical electrocardiogram finding could have been due to the ball-like thrombus adhering to the apical portion of the myocardium which mimics apical-type hypertrophic cardiomyopathy. Careful cardiac ultrasonography should have been repeated in such a case.

In the present case, inflammation of the thrombus might have made the patient susceptible to VT and Vf. The origin of infection is not known, but based on the pathological report, thrombus could have been

formed in 2 weeks. No infective organism was detected from the culture and pathological sample; this absence of a clear source of infection was also seen in a previous report^[5], but CZOP 3g+CLDM 1200 mg were partially effective in the clinical course. Therefore, gram-negative organisms could have been the cause, as reported in many previous reports^[1-3, 6].

Conclusions

We reported a case of infected LV mural thrombus as a complication of acute and old MI. In this case, the LV mural thrombus was adhered around the apical portion of the myocardium and presented inflammatory change, which might have caused the fatal arrhythmia.

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心筋梗塞の合併症としての心内血栓が感染した一症例

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要 約

症例は68歳男性、高熱と呼吸困難感を主訴に入院した。肺炎と心不全の診断で抗生剤と利尿剤で治療を開始した。一時軽快傾向となったが、CRP値6以上が持続していた。心エコーでは、び慢性壁運動低下と心尖部付近の心筋肥大がみられた。第18病日に致死的不整脈を発症し、CPRを行ったが、突然死した。病理解剖

の結果、下壁の陳旧性心筋梗塞と前壁の急性心筋梗塞に伴う心尖部の感染性血栓がみつかった。本症例では、左室の壁に血栓が心筋の心尖部に全周性に癒着しており、感染による炎症性変化を来しており、致死的不整脈につながったと考えられた。

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