Acute Myocardial Infarction Caused by Coronary Embolism from *Aspergillus* Endocarditis

Guang-Won Seo, Sang-Hoon Seol, Tae-Hun No, Hui-Jeong Jeong, Tae-Jin Kim, Jae-Kyun Kim, Pil-Sang Song, Dong-Kie Kim, Ki-Hun Kim and Doo-Il Kim

Abstract

Although the primary cause of acute coronary syndrome is atherosclerotic disease, it is important to include other causes of acute myocardial infarction. This report describes the case of a 53-year-old man with acute myeloid leukemia, who was referred to our cardiology department for treatment of acute myocardial infarction with ST segment elevation on an electrocardiogram. Portable echocardiography showed large areas of vegetation on the anterior mitral leaflet, while coronary angiography demonstrated the total occlusion of the left anterior descending artery. A histologic examination of the embolectomy specimen confirmed the presence of *Aspergillus* fungal thrombi. This report highlights a rare case of fatal *Aspergillus* endocarditis with myocardial infarction due to embolism in an immunosuppressed patient.

Key words: myocardial infarction, *Aspergillus*, acute myeloid leukemia

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Introduction

Myocardial infarction due to *Aspergillus* embolism has been reported in several patients (1-4). Cardiac involvement of aspergillosis, including myocardial infarction, endocarditis, and pericarditis, is considerably rare, and treating cardiac aspergillosis is very difficult, even when accurately diagnosed. Cardiac aspergillosis is rarely diagnosed before death, and is usually fatal (5). We herein report a case of acute ST-elevation myocardial infarction (STEMI) caused by *Aspergillus* embolism in the coronary artery.

Case Report

A 53-year-old man presented with acute myeloid leukemia in 2012. Chemotherapy was initiated with the hyperfractionated cyclophosphamide, vincristine, Adriamycin, and dexamethasone (CVAD) regimen. After the second cycle of chemotherapy, there was no evidence of recurrence on a bone marrow examination or other laboratory data, as confirmed by a hematologist. On day 1, the patient suddenly complained of anterior chest pain with dyspnea. At the time of arrival to the emergency department, he went into cardiac and respiratory arrest. Cardiopulmonary resuscitation was attempted for 10 minutes, and return of spontaneous circulation (ROSC) was performed without consciousness. On a physical examination, the patient’s body temperature was 36.7°C and his respiratory rate was 40 breaths per minute. Coarse breathing sounds were noted. Mid-diastolic heart murmurs (Grade III/IV) were audible, and a chest X-ray showed moderate pulmonary edema without cardiomegaly. An electrocardiogram revealed ST elevation in leads V2 to V6 (Fig. 1), and the serum troponin I level was elevated at 629.3 ng/mL. Portable echocardiography showed an irregularly shaped, large (3×2 cm in size) mobile echogenic mass at the tip of the anterior mitral leaflet with chaotic motion (Fig. 2A) and hypokinetic regional wall motion of the LAD territory in addition to a decreased left ventricular systolic function. Color Doppler echocardiography revealed severe mitral regurgitation (MR) due to rupture of the anterior mitral leaflet during systole (Fig. 2B). Emergency coronary angiography showed total occlusion of the left anterior descending artery (Fig. 3A). Percutaneous coronary intervention (PCI) was performed with thrombectomy using a Thrombaster. The embolectomy specimen was white with an...
An electrocardiogram revealed ST elevation in leads V2 to V6.

Figure 2. A: Apical 4 chamber view of portable echocardiography showed irregular shaped large (3×2 cm sized) mobile echogenic mass at the tip of the anterior mitral leaflet with chaotic motion (arrow). B: Color Doppler echocardiography revealed severe mitral regurgitation due to rupture of anterior mitral leaflet during systole.

Figure 3. A: Coronary angiography showed total occlusion of the left anterior descending artery (arrow). B: Embolectomy specimen was white colored with irregular shape.

irregular shape (Fig. 3B). Although PCI was performed successfully, the patient’s condition was unstable after the procedure. On day 3, 2013, he developed sudden cardiac arrest and gradually progressed to multiorgan failure. Cardiopulmonary resuscitation was attempted, however, the patient died.
After the patient’s death, a histopathologic examination of the embolectomy specimen showed Grocott-Gomori methenamine-silver nitrate positive staining and dichotomously branching hyphae of *Aspergillus* (Fig. 4).

**Discussion**

*Aspergillus* is an omnipresent mold capable of causing several diseases in both healthy humans and immunosuppressed hosts. Disseminated aspergillosis can occur in immunosuppressed hosts. Cardiac aspergillosis, in particular, is one of the most serious complications of systemic aspergillosis. Various clinical symptoms have been reported, including myocardial infarction, myocarditis, endocarditis, pericarditis, pericardial tamponade, superior vena cava syndrome, aortitis, intracardiac mass formation and atrioventricular block (5).

*Aspergillus* endocarditis is a serious condition whose prevalence is increasing in the hospital population. The areas of vegetation observed in patients with *Aspergillus* endocarditis are large and highly mobile, and peripheral embolism is common in the early stage of the disease (6). *Aspergillus* endocarditis is associated with embolic phenomena more commonly than bacterial endocarditis. The organs most frequently involved are the brain, kidneys, spleen and lungs. Myocardial infarction due to *Aspergillus* embolism often complicates the differential diagnosis of common myocardial infarction. For hemodynamic reasons, embolic events associated with *Aspergillus* embolism are likely to engage the descending branch of the left coronary artery (1).

Cardiac aspergillosis is rarely diagnosed before death, and is usually fatal, even with early detection and treatment. A poor health status, recurrent and severe embolic episodes, and diagnostic delay are the primary factors responsible for the dismal outcomes (7). Our patient died from myocardial infarction with acute severe MR due to *Aspergillus* fungal occlusion of a coronary artery. If the diagnosis had been established before death, surgical valve reconstruction may have been considered, since medical treatment with amphotericin B does not result in sterilization of the cardiac valves.

The *Aspergillus* species is very rarely isolated from blood cultures. Many cases are diagnosed on surgical specimens or at autopsy. Regarding the early detection of *Aspergillus*, some reports have suggested that polymerase chain reaction and enzyme-linked immunosorbent assays are useful for diagnosing the disease (8-10). Making an early diagnosis using various diagnostic procedures and providing early therapy are very important in immunosuppressed patients. For this reason, the empirical use of amphotericin B should be initiated if an immunosuppressed patient has a persistent fever and antibiotics are ineffective (11).

*Aspergillus* endocarditis and myocardial infarction caused by embolism are rare, and there is limited experience with their diagnosis and treatment. Native valve *Aspergillus* endocarditis is uniformly fatal without immediate and extensive surgical intervention combined with prolonged antifungal therapy. We herein reported a case of fatal *Aspergillus* endocarditis associated with myocardial infarction caused by embolism in an immunosuppressed patient diagnosed on a histopathologic examination of the embolectomy specimen.

**The authors state that they have no Conflict of Interest (COI).**

**References**

5. Itoh M, Takahashi M, Mori M, et al. Myocardial infarction caused...


