Rupture of a Smoldering Mycotic Aneurysm of the Thoracic Aorta into the Lung

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A 65-year-old man was diagnosed with meningitis and bacteremia, as Streptococcus pneumoniae was isolated from spinal fluid and blood cultures. After three weeks of antibiotic therapy, computed tomography revealed a ruptured aneurysm of the descending thoracic aorta. The aneurysm had appeared during the first episode of meningitis but had remained silent for two years. The patient underwent in situ Dacron graft replacement and his postoperative course was uneventful with no infectious complication. (Ann Thorac Cardiovasc Surg 2002; 8: 177–9)

Key words: mycotic aortic aneurysm, meningitis, in situ graft replacement

Introduction

Mycotic aortic aneurysms (MAAs) almost always require emergency surgery because they tend to grow rapidly and subsequently rupture. We encountered a rare case of mycotic thoracic aortic aneurysm, that had formed during treatment for the first episode of meningitis, but had remained silent for two years. That aneurysm ruptured and extended into the left lung during antibiotic treatment for the second episode of meningitis and sepsis.

Case

A 65-year-old man was admitted to a local hospital with complaints of high fever and loss of consciousness. Blood analysis showed a white cell count of 20,800/µl and a C-reactive protein level of 32.3 mg/dl. The cell count in the spinal fluid had increased to 3,528/3, and Streptococcus pneumoniae was isolated from spinal fluid and blood cultures. Meningitis and bacteremia were diagnosed.

The patient had a past history of meningitis two years prior to the current illness. At that time, an abnormal shadow was identified at the left hilus on chest X-ray (CXR) after three weeks of antibiotic therapy. Computed tomography (CT) and magnetic resonance imaging (MRI) demonstrated an aortic saccular aneurysm of 1.5 cm in diameter surrounded by thickened soft tissue in the descending aorta (Fig. 1). An MAA was strongly suspected. The results of blood and spinal fluid cultures were negative. The patient did not receive further treatment because the inflammation had been resolved.

Although the patient received piperacillin sodium of 12 g per day for three weeks, he suffered persistent infection and had massive hemoptysis following hemosputum. CXR showed atelectasis and a pneumonia shadow in the left lung. CT revealed a ruptured aneurysm in the descending thoracic aorta that had extended into the left lung (Fig. 2). The patient was transferred to our hospital and underwent emergency surgery.

We performed complete aneurysmal excision and in situ graft replacement using a woven Dacron prosthesis under partial cardiopulmonary bypass, followed by wedge resection of the damaged lung tightly adherent to the aneurysm. The patient’s postoperative course was uneventful with no infectious complication, and he is currently doing well after a three-year postoperative follow-up.

Histological examination of surgical specimens revealed neutrophilic infiltration, atherosclerotic change and disappearance of elastic fibers in the media in the aortic wall. The resected lung had a cavity occupied by throm-
bus that might have temporarily sealed the fistulous communication. In the lung parenchyma, marked neutrophilic infiltration and necrosis with abscess were observed. No microorganisms were found, and cultures of these specimens were negative, probably secondary to prolonged antibiotic therapy.

**Discussion**

MAAs are almost always lethal without surgical intervention because of either acute hemorrhage following aneurysmal rupture or uncontrolled systemic sepsis.\(^1\,\text{,}\,^2\) They can occur due to hematogenous dissemination of microorganisms, direct involvement of the intima, or ex-
tension from a nearby septic focus. Pasic et al. described a mycotic aneurysm as indicating infectious erosive arteritis with a false aneurysm being caused by infection of the aortic wall or a manifestation of infection in a preexisting aneurysm. In our case, CXR had demonstrated a new abnormal shadow during the first episode of meningitis. An MAA was strongly suspected based on CT and MRI findings, however inflammatory signs had subsided. During the second episode of infection, the aneurysm grew rapidly and subsequently ruptured. We could not verify that either the preexisting aneurysm or meningitis was a primary infection because the signs consequent to each infection appeared simultaneously. Chan et al. reported that the interval between onset of febrile illness and aortic surgery ranged from two weeks to one year in 22 cases of MAAs. The expansion and rupture of aneurysms occurred in those patients regardless of whether infection was controlled. An MAA remaining silent for two years, as in our case, is rare.

An intimal disruption such as atherosclerotic plaques may be a site of bacterial lodgment, and histological specimens have often demonstrated neutrophilic infiltration and atherosclerotic change in the same aortic wall, as in our case.

For the treatment of MAAs, local debridement of the aneurysmal wall and complete resection of infected tissue, including damaged lung, are essential to minimize postoperative infection. In situ graft reconstruction is reasonable especially for the thoracic aorta and we consider no need for wrapping the graft with an omental pedicle when there is no abscess formation around the aneurysm and the margins of the arterial segment are normal.

Once an MAA has formed, it grows rapidly and then may rupture in general. But it is possible to remain silent and cause acute hemorrhage following rupture by infection to the aneurysm some years later. Prompt diagnosis and emergency surgery with complete resection of infected tissue and in situ graft reconstruction are required for a ruptured MAA.

References