Mycotic Pulmonary Artery Aneurysm: Complication of Aspergillus Endocarditis

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Aspergillus endocarditis is an uncommon condition associated with immunosuppression, drug addiction, indwelling catheters, and, recently, cardiac surgery [1, 2]. Complications of Aspergillus endocarditis include septic emboli to the brain, liver, spleen, and kidney as well as mycotic aortic aneurysms and pulmonary infarction [1, 2]. We report a case of Aspergillus endocarditis with the unusual occurrence of a mycotic aneurysm of the pulmonary artery which ultimately was responsible for the patient’s death.

Case Report

A 12-year-old girl with Tetralogy of Fallot had a 4 month history of intermittent fever. At age 3 years, she underwent closure of a ventricular sepal defect and repair of the pulmonic outflow tract. At age 11, revision of the outflow tract was undertaken using a synthetic patch and insertion of a 27 mm porcine valve in the pulmonary position. A partial tricuspid annuloplasty was also done using sutures with Teflon felt pledgets. Initially she did well, but 4 and 5 months later developed left lower lobe pneumonias responsive to antibiotics.

Six months after cardiac surgery, she had a fever of 38.7°C and intermittent back pain. Physical examination revealed a pale, chronically ill child with a dry cough and a harsh, grade 5/6 holosystolic murmur with a loud porcine valve click. A diffuse precordial heave was noted. Laboratory data included a white blood cell count of 9800, and a hemoglobin of 7.1 g/dL. Coagulation studies including fibrin split products were elevated. A platelet count was normal on admission but fell to below 50,000. Chest radiography (fig. 1) demonstrated an enlarged heart with the porcine valve in place and a rounded density in the superior mediastinal aspect of the left lower lobe.

A presumptive diagnosis of endocarditis was made. The working differential diagnosis of the lung mass included a lung abscess, round pneumonia, or mycotic aneurysm. Multiple blood cultures were obtained of which only one grew coagulase negative Staphylococcus, while all the others were sterile. Two-dimensional echocardiography demonstrated a bulky vegetation on the tricuspid valve (fig. 2). The patient developed hemoglobinuria and was thought to have a disseminated intravascular coagulopathy as well as traumatic hemolysis. A percutaneous lung biopsy of the left lower lung density with a 22 gauge needle was attempted, but only blood was obtained. She acutely developed signs of tricuspid valve obstruction and was taken to the operating room for tricuspid valve replacement. A 2 x 3 cm vegetation was found attached to a pledget used in the previous annuloplasty. Hyphae were seen on a touch preparation and later identified as Aspergillus. The tricuspid valve was replaced. On postoperative day 2 she suddenly developed gross bleeding from the endotracheal tube and died despite vigorous resuscitative attempts.

At autopsy a smooth, ovoid, yellow, 5 x 5 x 7 mm vegetation was attached to the prosthetic pulmonic valve. A similar vegetation, 7 x 7 x 10 mm, was found in the right main pulmonary artery. Microscopic examination of the vegetations demonstrated branching septate hyphae consistent with Aspergillus. On examination of the left lung, a 3 x 4 cm pulmonary artery aneurysm was found in the proximal part of the branch to the posterior basal segment of the left lower lobe. The lumen was filled with organizing thrombus and contained a 1-cm-diam yellow mass containing fungal hyphae (fig. 3). Microscopic sections of the aneurysm wall showed replacement of the intima by thrombus, necrosis of the media, and disruption of the elastic laminae. The serosal surface and the vasa vasorum were not involved. The aneurysm had ruptured into the posterior basal bronchus and the entire tracheobronchial tree was filled with fresh blood clot.

Discussion

Infectious endocarditis is an uncommon complication of prosthetic valve surgery [3]. Although most infectious agents are bacterial, fungal endocarditis is being reported more frequently than in the past [2, 3]. Newman and Cordell [4] reported the first case of Aspergillus endocarditis involving a prosthetic valve in 1964. Intravascular objects such as central venous and ventriculatoatrial catheters are also well known sources of fungal endocarditis [2]. It has been demonstrated that the presence of an intracardiac foreign body is necessary to initiate intracardiac infection with Aspergillus in rabbits; although once initiated, the infection can persist in the absence of the foreign body [5].

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Although *Aspergillus* is a ubiquitous organism, it is an uncommon pathogen. Why patients who have had open heart surgery are predisposed to developing endocarditis with this organism is unclear. Some authors have been able to trace *Aspergillus* contaminants to operating room ventilators and heart-lung machines. Other isolated cases remain unexplained. In our case, the infection seems to have arisen on a surgical pledget. Among seven cases reviewed by Newman and Cordell [4], the time between surgery and onset of endocarditis ranged from 12 days to 23 months. The reason for the long delay in onset of symptoms is unknown. Prior broad-spectrum antibiotic therapy has been implicated as a predisposing factor [6].

*Aspergillus* tends to be an invasive organism and in the myocardium tends to involve contiguous areas [2] as well as distant areas via embolization, as in our case [1]. This property is not unique to *Aspergillus*. *Candida* and *Histoplasma* are other invasive fungi [7]. Walsh et al. [2] suggest that the organism’s ability to elaborate oxalic acid, which is toxic to tissue, may in part account for this feature. Hemorrhagic pulmonary emboli, brain abscesses, cerebral artery aneurysms, and renal, hepatic, splenic, and gastrointestinal infarcts and abscesses have been reported [1, 2]; however, abrupt embolic occlusion of peripheral arteries does not commonly occur in *Aspergillus* endocarditis. Mycotic aneurysms of the aorta caused by *Aspergillus* have been described with and without endocarditis [2, 8]. Airborne lung infection with *Aspergillus* can spread to adjacent pulmonary arteries causing invasion and thrombosis.

While mycotic aneurysms of the aorta are well known, mycotic pulmonary artery aneurysms due to any organism are rare. Causative organisms include *Staphylococcus*, *Streptococcus*, *Klebsiella*, *Propionibacterium*, *Candida*, and *Treponema pallidum* [9, 10]. A strong correlation has been found between pulmonary artery aneurysms and patent ductus arteriosus, which accounted for 21%–66% in different series [11, 12]. In one series, pulmonary artery aneurysms involved the main pulmonary artery in 88% of cases, the right pulmonary artery in 8%, and the left pulmonary artery in 3% [11].

Histologically, fungal mycotic aneurysms show thinning

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Fig. 1.—Large, round, soft-tissue density situated medially in left hemithorax just posterior to hilum. Metallic ring is porcine pulmonary valve.

Fig. 2.—Two-dimensional echocardiogram (apical four-chamber sector scan). Bulky, highly echogenic vegetation (arrow) on septal leaflet of tricuspid valve. RA = right atrium; RV = right ventricle; LV = left ventricle.
and necrosis of the media, disruption of the elastic laminae, and fibrous replacement of smooth muscle [8]. The aneurysms may contain thrombus and fungal hyphae which can extend into the vessel wall especially at points of branching. A granulomatous response to the organisms may be present with giant cell formation. Several pathogenic mechanisms have been advanced to explain the development of mycotic aneurysms: (1) embolism of the vasa vasorum; (2) the lodgment of septic emboli within the vessel with subsequent direct vessel wall invasion; and (3) erosion of the vessel wall from an adjacent process in the lungs, either by direct extension or by lymphatics [6]. The second seems most likely in our case.

The antemortem diagnosis of fungal endocarditis with mycotic pulmonary artery aneurysm is difficult. Symptoms such as cough, dyspnea, chest or back pain, and cyanosis are nonspecific. Massive hemoptysis is often the terminal event. Chest radiographs may show round fusiform masses in the lungs that mimic pneumonia, edema, or metastatic lesions [9]. Echocardiography is quite useful for the early diagnosis of endocarditis [13]. Areas of valve thickening and nodule formation are especially well seen using the real-time two-dimensional technique. Disease in peripheral pulmonary artery branches, however, is not visualized. Angiographically, mycotic aneurysms of the pulmonary arteries conform well to the chest radiograph findings. The aneurysms opacify slowly but remain filled longer than surrounding vessels, thereby suggesting slow or turbulent flow within them [9].

Culturing Aspergillus is difficult because the organism does not sporulate on blood. Peripheral hyphae counts are very low and may be reduced by the filtering effect of lungs and capillary beds [3]. Even when cultured, it may be dismissed as a contaminant [1]. Early diagnosis is “difficult but crucial” [2] and Aspergillus infection should be strongly considered when symptoms persist in the face of negative blood cultures and lack of response to antibiotics. Candida and Histoplasma blood cultures are usually positive, but other elusive causative agents include Haemophilus, rickettsiae, mycobacteria, and viruses [7]. Surgical removal of infected tissue and specific antifungal agents are the current modes of therapy, but mortality remains high [2, 3].

In summary, Aspergillus endocarditis has become a well recognized, although unusual, complication of open heart surgery. It is difficult to diagnose and treat. The hallmark of this organism is its invasive nature. Delayed onset of symptoms, prolonged clinical course, and bizarre complications are not uncommon. Our case is remarkable for the presence of a mycotic pulmonary artery aneurysm due to Aspergillus. As more intracardiac procedures involving insertion of prosthetic materials are performed, similar complications may be expected and can only be diagnosed and treated if those involved in the care of these patients have a high index of suspicion.

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REFERENCES


Fig. 3.—A, Gross specimen of pulmonary artery aneurysm cut in cross section. Thick exterior wall (arrows) and polyoid mass (m) of organized clot and hyphae situated eccentrically in lumen attached to one wall. B, Photomicrograph of thrombus within pulmonary artery aneurysm. Hyphae of Aspergillus flavus. Grocott stain. x 320.