Aspergillus Infection in Large Thrombus of a Permanent Ventricular Pacing Lead

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Fungal infection of intracardiac pacing wire is very rare. We experienced a case of patient with functioning transvenous pacemaker lead, inserted 3 years previously, which was completely encased in a large thrombus infected with aspergillus. The lung biopsy also confirmed aspergillus infection.

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Kew word: 1. Aspergillosis
2. Thrombus
3. Pacemaker, artificial

Case

Though uncommon, infection associated with these permanently indwelling intravenous prostheses may occur with little local evidence of inflammation. The incidence of infection after pacemaker transvenous implantation is 1~3 percents, the most common presentation being the abscess of the generator site. Pacemaker-related fungal infection is extremely rare. This is the first reported case of Aspergillus flavus transvenous pacemaker lead infection, associated with a huge thrombi in right atrial and ventricular chambers.

A 30-year-old man was admitted to our hospital with a exertional dyspnea during three months. About 3 years ago, he was diagnosed as complete atrophic ventricular block and atrial paralysis, and a permanent transvenous pacing system(DDD) was established in the right subclavian vein. He had no pulmonary tuberculosis infection and diabetes history.

This patient received oral diuretics for several days, and his orthopnea improved soon, but NYHA class III heart failure continued despite medical therapy. On ECG, his pacing was satisfactory. He had no fever but WBC count(18,500/mm³) and eosinophil count(9.5 × 10³/L) increased on peripheral blood. Ig E-PRISt was 1,300 IU/L. Peripheral blood culture and urine culture showed no growth. Serologic test revealed no immunocompromised state. Echocardiography revealed a large and shaggy mass extended across tri cuspids valve into the right ventricle and involving the transvenous pacemaker leads(Fig. 1), and also showed severe tricuspid and mitral regurgitation.

The operation was performed via a median sternotomy using moderately hypothermic cardiopulmonary bypass. After the pericardiotomy, we found severe cardiomegaly and distended right atrium without normal contraction. Epicardial electrophysiologic study was done and we
confirmed atrial paralysis and marked the site of epicardial lead on the right ventricular free wall. Cardiopulmonary bypass was established with aortic and bicaval cannulas. The right atrium was opened, we found a large infected thrombus which was strongly attached to the end of the pacemaker lead (Fig. 2). But there was no thrombi at atrial pacemaker lead. We carefully removed the pacemaker leads with a large thrombi (8 × 3 cm). Tricuspid valve had annular dilatation and granulations on posterior leaflet due to ventricular pacemaker lead. After the left atriotomy, we found mitral annular dilatation without any valvular destruction. Mitral annuloplasty was performed with modified Daviler method and tricuspid annuloplasty with modified De-Vega method. Heart beat was restored without defibrillation and 2 epicardial pacemaker electrodes were inserted on previous epicardial mapping site. Postoperative transesophageal echocardiography confirmed no intraatrial and ventricular thrombi, and also showed trivial tricuspid regurgitation and mitral regurgitation.

The presence of mycelial hyphae in the histologic section of the thrombus was confirmed (Fig. 3). Peripheral blood and sputum cultures and other immunologic studies were done, but their results revealed no evidence of aspergillus infection. Postoperative plain chest radiogram showed pneumonic patch density in right upper lobe, just
beneath the subcutaneous pocket for the previous pacemaker generator. Chest computed tomogram showed disseminated aspergillosis patterns in right upper lobe and both lower lobes(Fig. 4). He received systemic administration of amphotericin B. At postoperative 7th day, hemoptysis developed abruptly, and improved with anticoagulation therapy.

Open lung biopsy was performed at right upper lung field. The result of histologic study was aspergillosis of lung. After 40 days of the amphotericin B therapy, the infiltration of right upper lung was disappeared.

**Discussion**

Since 1965, the implantation of a permanent transvenous pacemaker is a well-accepted procedure for the treatment of cardiac conduction disturbance and arrhythmias. This procedure is relatively easy and safe, but complications associated with retained pacemaker leads can occur. The development of a large thrombi in the right atrium and ventricle in the presence of a permanent pacemaker electrode has been described in the literature as rare, yet serious complication.

Huang and Abe found in examinations of nine postmortem cases, organized thrombus related to the electrode catheter in the form of a fibrous band closely attached to the tricuspid valve, the chordae tendinae, and around the tip of the pacing catheter. Hendler reported a case of a right atrial thrombus in the presence of a permanent pacemaker electrode with polycythemia vera. To the best of our knowledge, only one case of significant tricuspid regurgitation as a complication of a thrombus related to a permanent electrode has been described. Our patient had no coagulopathy but his right atrium appeared very poor contraction. The right atrial paralysis seemed to cause blood stasis in right atrium, and induced thrombus formation with electrode. So, we recommend that the long-term anticoagulant therapy should be considered in selected cases, and suggest that the epicardial pacemaker electrodes might be considered in patients at high risk of thrombosis.

Pacemaker-related fungal infection is extremely rare. Disseminated Petriellidium boydii infection and pacemaker endocarditis has been described in a 62-year-old woman on chronic high dose steroid therapy for mixed connective tissue disease. Davis and associates reported a 71-year-old man with diabetes who developed heart failure, fever and leukocytosis ten months after placement of a transvenous pacemaker for complete heart block associated with syncope. After the pacemaker was implanted, he was treated with broad-spectrum antibiotics for recurrent urinary tract infections and transurethral resection of the prostate. He died because of refractory congestive heart failure and necropsy disclosed a large vegetation arising in the right atrium and occluding the tricuspid valve; this contained dimorphic fungi which proved to be Candida. Moorman and associates presented aspergillus infection in a large thrombus of right ventricle and myocardium of 80-year-old woman who died infective endocarditis with negative blood culture. The occurrence of embolization is not clear and septic pulmonary emboli from an infected pacemaker have been reported.

Aspergillus pneumonia and disseminated aspergillosis occur in immunosuppressed patients, especially those with neutropenia. Pulmonary infection is acquired by inhalation of the organisms, which are omnipresent in air. Aspergillus endocarditis, on the other hand affects patients with prosthetic cardiac valves who are not usually immunosuppressed. Infection could arise in several ways; airborne organisms may settle into the operative field during valve replacement; they may be introduced when intravascular catheters are placed; or they may be enter the bloodstream from the lungs. In our patient, the source of the infection is not known. The able route of infection is the bloodstream from the lungs, because the immediate postoperative chest radiogram showed pneumonic patch density in right upper lobe, just beneath the subcutaneous pocket for the previous pacemaker generator.

In previous reports of fungal pacemaker lead infections, meaningful risk factors is above 60 years of age, broad spectrum antibiotics therapy, diabetes, and steroid therapy. These underlying illness presumably predisposed the patients by setting the stage for fungemia during which small thrombi associated with the pacemaker leads were seeded by circulating organisms. But our patient had none of the above probable factors. His causing factor is huge intracardiac thrombosis due to blood stasis from right atrial paralysis. He had not received anticoagulants after the implantation of the transvenous pacemaker.

Finally, it must be emphasized that the presence of blood stasis due to significant tricuspid regurgitation or paralysis of right atrium, indicated a long-term anticoagulant therapy and serial echocardiogram. Moreover, Zager et al. suggested that epicardial pacemaker electrodes might be considered in patients at high risk of thrombosis. The treatment of choice would be surgical removal of the infected pacemaker system and associated vegetations combined with systemic administration of amphotericin B.
References

1. Huang TY, Babe N. Cardiac pathology of transvenous pacemakers. Am Heart J 1972;83:469-74