Detection of Pacemaker Lead Infection by Fluorodeoxyglucose Positron Emission Tomography

Takashi Miura MD, Osamu Kinoshita MD, Mitsuaki Horigome MD, Hiroki Kasai MD, Kazunori Aizawa MD, Tohru Yoshioka MD, Takeshi Tomita MD, Setsuo Kumazaki MD, Hiroshi Tsutsui MD, Jun Koyama MD, Yoshikazu Yazaki MD, Uichi Ikeda MD

The Division of Cardiovascular Medicine, Shinshu University School of Medicine

An 80-year-old man was implanted with a DDD pacemaker to treat his sick sinus syndrome in 1990. Eleven years later, he had a pocket infection and cutaneous inflammation. Blood cultures were negative, and $^{67}$Ga scintigraphy revealed uptake in the left subclavian region. However, intense abnormal fluorodeoxyglucose (FDG) uptake along the pacemaker leads was detected with positron emission tomography (PET). Thoracotomy was performed, vegetations were removed from the right atrial wall and the tricuspid leaflet, encapsulating fibrous tissue was incised, and the lead was removed from the right ventricle.

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Introduction

It has been reported that approximately 1% of patients with endocardial pacemakers developed septicemia.\(^1,2\) Infections have occurred early after implantation in most cases, and these leads can be intravenously retracted.\(^3\) Persistent septicemia is a rare occurrence late after the implantation of permanent pacemakers. In these cases, the electrode cannot be withdrawn from the vasculature because it has become firmly enclosed by fibrous tissue along its course from the vein tract to the right ventricle.\(^4\) It has often been difficult to discriminate whether an infection spreads over the leads or only the pocket.\(^5\) We report a case in which positron emission tomography with fluorodeoxyglucose (FDG-PET) was successfully used to find an infected pacemaker lead 15 years after implantation.

Case Report

In 1990, an 80-year-old man was implanted with a DDD pacemaker into the left subclavian vein because of sick sinus syndrome. Eleven years later, he had a pocket infection. Prompt removal of the generator and debridement of the degenerated tissue was performed, and a new pacing system on the right side of the chest was implanted. In November 2004 he was admitted to our hospital because of cutaneous inflammation on the left side. Blood cultures were negative, transthoracic echocardiogram showed no thrombus and vegetation. CT scan revealed no specific signs of pacemaker lead infection. $^{67}$Ga scintigraphy revealed uptake only on the left subclavian region. He was treated with debridement of the pocket at the left side, and cutting and suturing the leads. Antibiotics were continued for 6 month.
In August 2005, he was readmitted to our hospital, because of failure to bring his infection under control. Fever as high as 37.5°C recurred, C reactive protein was 1.77 and the white blood cell count was 3670 per cubic millimeter. Our impression was that he had bacteremia, most likely caused by endocarditis, involving the endocardial pacemaker electrodes. A blood culture at this time was also negative. Transthoracic echocardiogram did not show abnormal high-density mass echoes or vegetations.

After 5 hours fasting, patient was injected 236 MBq of $^{18}$F-FDG intravenously. The acquisition protocol consisting of a set of whole-body emission scans (2 min/bed position) and post-injection transmission scans (1.5 min/bed position) starting 60 minutes after injection. Total acquisition time for whole body imaging was 21 minutes over 7 bed positions. Attenuation corrected PET image set was obtained by GE Advance NXi PET system (Milwaukee, U.S.) using two dimensional acquisition mode and reconstructed with ordered subsets expectation maximization algorithm with segmented attenuation correction. Reconstructed transaxial images were displayed on a $128 \times 128$ matrix and a $4.3 \times 4.3 \times 4.25$ mm. Intense abnormal FDG uptake along the pacemaker leads was recorded with PET (Figure 1).

On the basis of a clearly positive FDG-PET investigation, a thoracotomy was performed. The lead was tightly attached to the entrance of the superior vena cava and edge of the leaflet of the tricuspid valve. Vegetation was removed from the right atrial wall and the tricuspid leaflet. Encapsulating fibrous tissue was incised, and all leads which implanted from the left and right subclavian vein, were removed from the right ventricle and atrium, the pacemaker generator at the right side was also removed, and a new epicardial lead and pacemaker were implanted. The explanted pacemaker leads (Figure 2) was sent for culture, which confirmed the presence of *Staphylococcus epidermidis*.

### Discussion

Recent reports show an incidence of pacemaker infection of 1%.1,2) Most infections occur within 4 weeks after implantation and are limited to the pacemaker pocket. The early infections are mainly due to peri-operative bacterial infiltration. *Staphylococcus aureus* are commonly seen early after implantation. The patients have a high incidence of sepsis, and are managed with drugs effective against *Staphylococcus aureus* while the results of cultures are awaited. The total removal of the entire pacemaker system is indicated, and leads can be retracted easily from the subclavian vein, if implantation was performed recently.

Late infections occurs either after initial implantation in mechanical skin necrosis around the implanted pacemaker generator, or in patients who had many previous implants at the same site. The most common infecting bacteria are *Staphylococcus epidermidis*.

AB Lewis6) reported that conservative therapy was not effective and removal of the entire infected pacing system successfully eradicated infections in all 74 patients. The pacing system should be completely removed and replaced by an epicardial system or a new endocardial system on the opposite side of the chest.

Procedure of lead removal of chronically implanted transvenous lead systems have improved since the market release of the transvenous tools in the early 1990s. Most leads can be safely removed by using a lead removal kit. HJ Smith7) reported that this technique allowed the successful extraction of up to

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**Figure 1** FDG-PET shows abnormal uptake along the pacemaker leads extending to right atrium.

**Figure 2** Removed leads were encapsulated with fibrous tissue and thrombus.
nearly 98% of intravascular leads. However, these devices can not be utilized in our country now. In addition, intravascular techniques for extraction of permanent pacemaker leads might carry the risk of cardiac perforation, rupture, tamponade, pulmonary embolism and even bacterial endocarditis. ME Lee\(^8\) reported avulsion of a tricuspid valve leaflet during traction on an infected, entrapped endocardial pacemaker electrode. Determining whether infection has spread over the leads or only as far as the pocket is often difficult in patients with negative blood cultures. Transthoracic or transesophageal echocardiography is a readily available and accurate method for diagnosis of bacterial endocarditis. \(^{67}\)Ga scintigraphy has been used for localizing infection or abscess. About 90% of circulating \(^{67}\)Ga is in the plasma, and nearly all of it is bound to transferrin. Increased blood flow and increased vascular membrane permeability result in increased delivery and accumulation of transferrin bound \(^{67}\)Ga at inflammatory foci. On the other hand, deoxyglucose combines with glucose transporter in the inflammatory cell and malignant cell. \(^{18}\)F-FDG uptake increases in malignant tumor, inflammatory tissue, or central nervous system. PET is becoming an established imaging tool in the field of clinical oncology, neurology or cardiology. Recently Fukuchi, et al.\(^9\) reported that aortic graft infection could be detected by FDG-PET, because of the high glucose uptake of peri-prosthetic skeptical inflammatory tissues.

In the presented case here, intense positive FDG uptake along the pacemaker leads recorded with PET, and was helpful for detecting the width of the infected area. Because of the high glucose uptake of inflammatory cells, FDG scanning is an appropriate tool for use in detecting inflammation or to evaluate infectious area in patients suspected of pacemaker lead infection.

References