Recognition of pacemaker lead infection by transoesophageal echocardiography

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Abstract
Transoesophageal echocardiography identified a pacemaker lead infection in a patient in whom transthoracic echocardiography was negative and there were no specific signs of skin inflammation over the pulse generator.

Infection of a permanent pacemaker system is defined as one or more of the following: (a) local inflammation and abscess formation in the pulse generator pocket; (b) erosion through the skin of part of the pacing system that is followed by secondary infection; (c) fever associated with positive blood cultures in a patient without a focus of infection elsewhere. Diagnosis can sometimes be difficult, especially when the pacemaker leads are infected without local inflammation of the pulse generator pocket and without erosion of part of the pacing system. Transthoracic echocardiography can sometimes be helpful but image resolution is often inadequate.

We found that transoesophageal echocardiography was useful in the diagnosis of infection of the pacemaker leads in one of our patients.

Case report
In March 1986 a 47 year old man was admitted for the first time to another hospital for surgical correction of an atrial septal defect. He was readmitted in June 1986 because of syncope caused by an atrioventricular block, Mobitz type II, and a dual chamber pacemaker was implanted. Four days after implantation of the pacemaker system, discrete inflammation appeared near the pulse generator and treatment with local disinfection and oral antibiotics (tetracyclines) was started. Forty eight hours later the inflammation had disappeared.

In November 1988 the patient was treated for septicemia caused by Staphylococcus capitis, bronchopneumonia, and spondylodiscitis of L2 and L3. The patient was treated with oxacillin for six weeks. Slight tenderness was noted over the pulse generator pocket. After the normal antibiotic treatment, there was normal healing of the pulse generator pocket and no inflammation. Computed tomography showed the beginning of organisation of the spondylodiscitis lesion.

The patient presented in March 1989 with tenderness over the pulse generator. Examination showed discrete inflammation without evidence of abscess formation. The pulse generator was removed but the pacemaker leads were left in place. A new pacemaker system with new atrial and ventricular leads was implanted on the contralateral (left) side.

The patient was readmitted in July 1989 with fever and chills. Both the old and new pacemaker pockets showed normal healing without inflammation. Leucocytosis (white cell count 20 x 10^9/l with 87% neutrophils) and considerable evidence of inflammation (erythrocyte sedimentation rate was 106 mm/h, fibrinogen was 600 mg/dl) were detected. Three blood cultures were positive for Staphylococcus capitis. Transthoracic echocardiography showed no evidence of infection of the pacemaker leads. The patient was treated with antibiotics but two days after intravenous administration was changed to oral treatment, fever reappeared and again three blood cultures grew Staphylococcus capitis.

The patient was referred to our hospital for transoesophageal echocardiography. Four pacemaker leads were detected in the superior vena cava and right atrium. We also saw what appeared to be a large (2 cm - 1 cm), mobile and lobulated vegetation (figure) alongside one or more of the leads. Because of this we decided to remove all the parts of the pacemaker.

The right atrium was opened to remove the four electrodes which were cut as high as possible in the superior vena cava. We found a large vegetation on an atrial lead. This lay along the wall between the tricuspid valve and inferior vena cava. The patches used to correct the atrial septum defect and the tricuspid valve were intact. A new pacemaker system was inserted: a pulse generator was implanted in the epigastric region and two electrodes were implanted on the epicardium, one at the base of the right atrium and the other on the right ventricle. The right pectoral leads and the left pectoral leads and left pulse generator (surrounded by an old haematoma) were removed.

At follow up four months later the patient was well, free from fever, and had no signs or symptoms of infection or inflammation without antibiotic treatment.

Discussion
Pacemaker infection is unusual. The reported incidence ranges from 0% to 12.6%; with current pacemaker procedures the infection rate ranges from 0.8% to 3%. The number of patients at risk of infection will increase as permanent pacemakers become more widely used.

This potentially lethal condition sometimes has a slow course and causes little pain. When there are no clear signs of infection and the
Transoesophageal echocardiogram ((A) M mode and (B) cross sectional) showing pacemaker lead with vegetation in the right atrium.

Pacing system has not eroded the skin, diagnosis of pacemaker system infection can be very difficult. This is often the case when intermittent treatment with antibiotics is given—as in our patient.

The leads often become infected in cases of pacemaker infection: pacing electrodes from 88% of patients operated for pacemaker infection gave positive cultures. This, however, is difficult to demonstrate preoperatively. Transthoracic echocardiography is not sufficiently sensitive. We found that transoesophageal echocardiography was helpful in our patient. We found only one report on the diagnosis of hidden pacemaker lead sepsis by transoesophageal echocardiography. Larger studies will be needed to assess the sensitivity and specificity of transoesophageal echocardiography in the diagnosis of pacemaker lead infection.

When there is a strong suspicion of pacemaker infection we recommend the replacement of the pulse generator and leads. Treatment with antibiotics alone is not sufficient and can mask the progression of the infection.

4 Buch J, Mortensen SA. Late infections of pacemaker units due to silicon rubber insulation boots. _PACE_ 1985;4:494.