The postpericardiotomy syndrome is a well-known complication of opening and manipulating the pericardium. The occurrence of this syndrome following transvenous pacemaker insertion is very rare, and only 5 cases have been reported to date. The present patient repeated this syndrome 3 times in a short period following 3 different interventional techniques: a temporary transvenous pacemaker, a permanent transvenous pacemaker and surgical pericardiotomy. (Jpn Circ J 2001; 65: 343–344)

**Key Words:** Complication; Pacemaker; Postpericardiotomy syndrome

### Case Report

A 71-year-old man was admitted to hospital on October 6, 1999 for sinus arrest of 4.6 s with dizziness on ambulatory 24-h Holter recording. His past history was unremarkable, and the 12-lead ECG on admission was normal. After admission, sinus arrest of 6.7 s with symptoms was found on continuous ECG monitoring. On October 7, a temporary transvenous pacemaker was inserted into the right ventricle without incident, and on October 15, a permanent DDD pacemaker (Marathon-DR Model No. 294-09E, Sulzer Intermedics, Angleton, Texas) was implanted. An atrial active fixation screw-in pacing lead (Model No. 5568-53 Medtronic Inc, Minneapolis, MN, USA) and a ventricular bipolar passive fixation pacing lead (Model No. 430-10 Sulzer Intermedics, Angleton, TX, USA) were introduced via left subclavian venous puncture. The ventricular electrode was positioned without difficulty, although satisfactory atrial sensing and pacing functions were only achieved after 3 different positions had been attempted. Cefotiam dihydrochloride was given as a prophylactic antibiotic. On October 18, the patient’s temperature rose to 37–39°C and laboratory analysis showed leukocytosis and elevated C-reactive protein. The patient had mild left anterior chest pain that lasted for 3 days. Three blood cultures were negative and the surgical wound was not infected. We replaced cefotiam dihydrochloride with cefozopran hydrochloride and amikacin sulfate, after which the inflammatory reaction improved temporarily. However, on October 30, fever, leukocytosis and elevation of C-reactive protein appeared again, and we detected moderate pericardial effusion on echocardiography. On November 1, he had tachypnea, tachycardia and pulsus paradoxus, and the chest X-ray...
revealed bilateral pleural effusion and enlargement of the heart. Echocardiogram showed a large anterior and posterior effusion with collapse of the right ventricle on diastole. The same day, we diagnosed cardiac tamponade and performed surgical pericardiotomy for drainage without complications, removing 800 ml of straw-colored fluid, which was exudative, but negative on culture and cytologic examination. After pericardiotomy, we gave amikacin sulfate, imipenem/ cilastatin sodium and indomethacin, following which the inflammatory reaction improved. However, on November 15, an increase in the inflammatory markers occurred once again (Fig 1). On giving 40 mg/day prednisolone, the inflammatory markers improved immediately and prednisolone was tapered to 5 mg/day without a relapse. He continued to receive prednisolone, the clinical course was good, and he was discharged on December 15. Paired serum viral titers did not show any significant rise. Anti-nuclear antibody and rheumatoid factor were negative. Complement C3 and C4 fractions were within normal limits. Anti-cardiac antibody was not detected.

Discussion

The postpericardiotomy syndrome is clinically diagnosed by exclusion.\(^1,7,9\) In the present patient, bacterial infection of the site of implantation of the pacemaker and infective endocarditis were excluded, because the wound was well healed and non-tender, and blood cultures were negative. The nature of the pericardial fluid also excluded myocardial perforation by the pacemaker electrodes. The clinical features, investigations and the response to steroids strongly supported the diagnosis of the postpericardiotomy syndrome.

It is interesting that the first elevation of inflammatory markers occurred 11 days after insertion of the temporary pacemaker, and the second elevation with cardiac tamponade occurred 15 days after implantation of the permanent pacemaker, and that the third time elevation of it occurred 14 days after surgical pericardiostomy for cardiac tamponade. Thus the clinical features indicating postpericardiotomy syndrome appeared after nearly identical intervals of approximately 2 weeks after placement of the temporary pacemaker, placement of the permanent pacemaker and surgical pericardiostomy. This syndrome may recur on occasion, but it is difficult to accept that the features of the present patient as recurrences, because recurrences of this syndrome tend to appear during 1 to 6 months after surgery.\(^1,10\) In short, in this case there is a strong possibility that the postpericardiotomy syndrome occurred 3 times following 3 kinds of interventional techniques.

In 5 previous cases of the postpericardiotomy syndrome following transvenous pacemaker insertion, temporary electrodes had been used in 2 cases\(^5,6\) and active fixation screw-in atrial leads had been used in 2 other cases.\(^5,6\) There was no description of whether a screw-in atrial lead had been used or not in the remaining case.\(^7\) Both temporary electrodes and an active fixation screw-in atrial lead were used in the present patient. The precise cause of the postpericardiotomy syndrome is not clear, but De Scheerder et al reported that an autoimmune reaction precipitated by myocardial or pericardial injury was involved.\(^9\) Therefore, although myocardial perforation was not detected, the possibility remains that a stiff temporary electrode in the temporary pacemaker and the screw-in atrial lead in the permanent pacemaker damaged the myocardium. In summary, we report a case of triple occurrence of postpericardiotomy syndrome in a short period following 3 different interventional techniques: a temporary transvenous pacemaker, a permanent transvenous pacemaker and surgical pericardiostomy.

References