Recurrent Forearm Thrombophlebitis after Transvenous Permanent Pacemaker Insertion

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SUMMARY

This report describes a 69-year-old Japanese woman with bilateral forearm thrombophlebitis that developed soon after transvenous permanent pacemaker insertion. Intravenous administration of urokinase and heparin rapidly resolved fever, painful forearm swelling and inflammatory findings. Digital subtraction venograms revealed a brachiocephalic vein thrombosis. A ten-month treatment with warfarin and ticlopidine resulted in the complete restoration of venous flow. Although thrombophlebitis associated with transvenous permanent pacemaker insertion has a relatively low incidence (0.3%), prompt diagnosis and treatment with anticoagulant and/or thrombolytic agents are important for the late clinical outcome in cases with deep venous thrombosis. (*Jpn Heart J* 34: 809–813, 1993)

Key Words:

Thrombophlebitis Thrombosis Pacemaker insertion

ALTHOUGH venographic studies have revealed that venous stenosis and thrombosis after transvenous permanent pacemaker insertion are a more common complication than previously thought,^{1)–3)} symptomatic acute venous thrombosis is uncommon.⁴⁾ Its clinical findings include edema, pain, cyanotic discoloration of the affected arm, and venous prominence due to venous congestion. Since superficial thrombophlebitis has been rarely reported^{5),6)} as a complication of the insertion of transvenous permanent pacemaker leads, its incidence, clinical course, and treatment have been unclear. This report describes a rare case in which bilateral forearm thrombophlebitis and deep venous thrombosis occurred simultaneously and were successfully treated soon after transvenous permanent pacemaker insertion.

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CASE REPORT

A 69-year-old Japanese woman was referred to our hospital in January 1992 because of near syncope associated with atrioventricular block. Physical examination revealed a regular heart rate of 70/minute. The blood pressure was 128/78 mmHg. Laboratory studies revealed a normal urinalysis, normal blood cell counts and normal serum lipid levels. Serological tests were negative for C-reactive protein (CRP). The erythrocyte sedimentation rate (ESR) was 24 mm/hour. The cardiothoracic ratio on chest X-ray was 57%. The resting electrocardiogram (ECG) was within normal limits. A 24-hour Holter ECG revealed a transient advanced atrioventricular block. The electrophysiological study revealed a prolonged HV interval (65 msec) and atrioventricular effective refractory period (790 msec). On February 13, she underwent elective insertion of

Fig. 1. This figure shows the clinical course. From top to bottom, are the date, body temperature (BT), clinical signs, laboratory findings, and treatment. PM=pacemaker implantation (1; initial implantation, 2; reoperation for correction of the malposition of the atrial pacemaker lead.); iv DSA=intravenous digital subtraction angiography; CRP=C-reactive protein; ESR=erythrocyte sedimentation rate; WBC=white blood cell; RBC=red blood cell; Hb=hemoglobin; Ht=hematocrit; Plat=platelet; TB-test=thrombotest; DIV=drip intravenous injection; SC=subcutaneous administration.

Fig. 2. Digital subtraction venograms (A, B) revealed a brachiocephalic vein thrombosis (indicated by large arrows in B) with well-developed collaterals via the anterior jugular veins. A and B were taken following the injection of iopamidol (nonionic contrast medium) from the left and the bilateral cephalic veins, respectively. A ten-month treatment with warfarin and ticlopidine resulted in the complete restoration of venous flow, and the thrombus and collaterals disappeared as shown in C. Small arrows indicate the direction of venous flow. "A" and "V" in B show the atrial and the ventricular leads, respectively.

transvenous permanent bipolar endocardial electrodes to both the right atrium (Pacemaker model 1012T) and right ventricle (ELA model T84F) via the left subclavian vein. These procedures were performed without complication, but reoperation was required the next day because of dislocation of the atrial lead. On February 15, fever was noted and on February 18, she complained of pain on the back of the left hand and thereafter a painful reddish swelling developed from the left hand to the elbow with active inflammatory findings such as leukocytosis and CRP elevation. Although elevation of the affected arm, and administration of a nonsteroidal anti-inflammatory agent and antibiotics were not effective, intravenous administration of heparin (12,000 IU/day) and urokinase (120,000 IU/day) for 7 days improved these symptoms. Thereafter, an antiplatelet agent (ticlopidine 400 mg/day) was administered. On March 2, recurrence of thrombophlebitis in both forearms was noticed. Oral administration of warfarin and subcutaneous injection of heparin (20,000 IU/day) without antibiotics re-

solved the clinical symptoms. On March 13, digital subtraction venography revealed thrombus formation in the left brachiocephalic vein and collateral formation via the anterior jugular vein (Fig. 2A,B). The values of antithrombin III, protein C, protein S, and tissue plasminogen activator were within normal limits. The clinical course is summarized in Figure 1.

She was discharged on March 23 and there has been no recurrence under anticoagulation therapy with warfarin and ticlopidine. On December 25, digital subtraction venograms revealed the disappearance of both the thrombus and collateral vessels as shown in Figure 2C. Thereafter, warfarin therapy was discontinued, with no recurrence of symptoms.

DISCUSSION

From 1977 to 1992, 330 patients received transvenous permanent pacemaker insertion at our hospital, and forearm thrombophlebitis occurred in only one case (0.3%). Thus, the incidence of forearm thrombophlebitis seems to be relatively low as a complication of transvenous permanent pacemaker insertion. In cases of simultaneous occurrence of superficial thrombophlebitis and deep vein thrombosis, it has been proposed that the two sites of thrombosis might form independently⁷⁾ due to a hypercoagulable state. Because intravenous catheters have been known as a cause of secondary hypercoagulable states⁸⁾, intravenous pacemaker leads per se might be the cause. The precise mechanism of coagulation abnormalities could not be clarified, but our case provided an important clinical suggestion that the use of long-term warfarin therapy until the complete resolution of deep venous thrombus might be essential for the prevention of recurrence of forearm thrombophlebitis.

Thrombolytic therapy has been used for the treatment of pacemaker lead-induced acute venous thrombosis and for patients in whom anticoagulation using heparin failed.⁴⁾ Although complete restoration of venous flow has been reported following streptokinase therapy with a loading dose of 250,000 units followed by a continuous infusion of 100,000 units per hour for three days, gross hematuria occurred.⁴⁾ In our case, the relatively low dose of urokinase of 120,000 units per day did not result in active bleeding, but its clinical effectiveness was questionable. Since thrombolytic therapy has potential risks such as life-threatening bleeding and pulmonary embolism, a standard regimen should be established.

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