Superior Vena Cava Syndrome after Repeated Insertion of Transvenous Pacemaker

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심박조음기의 개발로 많은 심혈관성 부정맥환자와 일부 반맥성 부정맥환자의 생명을 구할 수 있게 되었다. 심박조음기를 중심정맥을 통하여 심장으로 삽입하는 방법은 다른 방법에 비하여 비교적 쉽고 안전하며 합병증이 적은 것으로 알려지고 있어 현재는 거의 이 방법을 사용하고 있고 심박조음기도 기술개발로 점차 크기가 작아지고 풀리기능이 우수한 형태가 개발되고 있다. 그러나 심박조음기의 설치에 따른 여러 합병증이 생길 수 있는데 그중 드물게 상대정맥이 혈전으로 폐쇄되어 나타나는 상대정맥중후군이 발생할 수 있다. 심장과 대정맥내에 이물질이 있다면 혈전증의 가능성이 높다는 것은 널리 알려진 사실이다. 상대정맥내의 혈전증은 심박조음기의 전극을 따라서 발생하며 보통은 혈관내막의 섬유화와 혈착을 동반하는 것으로 알려지고 있다. 상대정맥혈전증의 치료방법으로는 항응고제, 혈전용해제의 사용과 풍선확장술, Forgaty 카테터에 의한 혈전체거술 및 수술 등이 있다.

저자 등은 반복적인 심박조음기설치 후 발생한 상대정맥중후군 1예를 수술로서 치험한 바 문헌고찰과 함께 보고하는 바이다.
Introduction

The use of transvenous permanent pacemakers have increased in patients with serious bradyarrhythmias and some tachyarrhythmias because of relative ease and safety. Many of the early limitations related to device size and longevity appear to have been resolved by the development of technology. But whenever a foreign body is introduced into the heart and central veins and left there permanently, the risk of thromboembolic complications exist\(^1\). There have already been reports of the superior vena cava syndrome resulting from thrombosis around the pacemaker electrode in the right ventricle and superior vena cava\(^2\). The patient reported here developed superior vena cava thrombosis after repeated insertion of transvenous pacemaker, and she was treated by surgical intervention.

Case report

The patient was a 57-year-old woman who was admitted with a 1-month history of progressive swelling of the face, neck, arms, and upper chest. Fourteen years earlier the patient had undergone implantation of a transvenous VVI pacing system in the right infraclavicular area for symptomatic sick sinus syndrome. Pacemaker revision with transvenous lead implantation was performed via the left subclavian vein 9 years earlier and the right generator was removed, but the right electrode was firmly adherent to the endocardium and could not be removed. Two years earlier the generator was replaced by a VVIR type and the electrode was not changed. Physical examination on admission revealed a body temperature of 36.7°C, blood pressure of 100/60 mmHg, pulse rate of 80 per minute, and respiratory rate of 20 per minute. The upper half of the body was swollen. A computed tomography scan of the chest with injection of contrast medium showed an enlarged superior vena cava and decreased attenuation, consistent with intraluminal clot. Through a right subclavian vein catheter, contrast medium was injected. Superior venacavography showed extensive clot formation and total occlusion of the superior vena cava with multiple collateral veins in the axilla and neck (Fig. 1). A urokinase was administered by a continuous infusion of 2000 units/hour for 3 days. But the edema did not subside. 2 days after discontinuation of a urokinase infusion, surgical interventions was performed. These included thrombectomy with a Fogarty catheter and superior vena cava reconstruction with pericardium. The electrodes in the superior vena cava and right ventricle was removed. Pacemaker revision with epicardial lead implantation was performed and the lead was positioned at the right ventricle. On the pathologic findings tissue obtained from the superior vena cava showed organized thrombus with calcification (Fig. 2). After surgery the edema subsided. The patient was discharged without taking anticoagulants.

Discussion

Thrombosis of the superior vena cava is an uncommon but potentially life-threatening complication of transvenous cardiac pacing. The pathogenesis of venous thrombosis after implantation of a transvenous permanent pacemaker has not been clearly determined. Thrombosis in the absence of coexistent stenosis tends to occur early, usually less than 1 year after implantation of a permanent pacemaker. Venous thrombosis that occurs more than 1 year after implantation of a permanent transvenous pacemaker is usually associated with underlying venous stenosis. Whenever the electrodes came in contact with the vascular endothelial lining the electrodes was incorporated into the vessel or endocardium and was overgrown by fibrous tissue and endothelium\(^3\). The development of the venous collaterals may further decrease flow in the stenotic venous segment. Consequently extension of thrombo-
Fig. 1. Superior venacavogram demonstrated large filling defect in the superior vena cava marked dilatation of the right internal jugular vein and subclavian vein and complete obstruction of the superior vena cava.
Multiple tortuous collateral vessel was noted.
Two pacemaker electrodes was found in the right ventricle.

Thrombosis and subsequent venous occlusion occur. The long-term residence of a permanent pacemaker electrodes in the central vein may also act as a continuing nidus for formation of thrombus. The presence of multiple transvenous pacemaker leads also increases the risk of thrombosis.

Symptomatic thrombosis of the central veins attributed to permanent transvenous pacemaker leads is uncommon; it affects from 1 to 3% of patients with permanent transvenous pacemaker electrodes. Most patients with chronic venous thrombosis remain asymptomatic because the gradual formation of a thrombus facilitates the development of an adequate venous collateral circulation. Symptomatic
pacemaker-induced venous thrombosis generally implies either acute venous thrombosis or extension of previously localized thrombus that occludes venous collaterals. Pacemaker-induced superior vena cava syndrome is rare and occurs from 1 month to 15 years after implantation of a pacemaker. Most cases are the result of thrombosis in the superior vena cava, but several cases involve fibrotic stenosis of the superior vena cava without thrombosis. In addition, propagation of thrombus from peripheral veins to the superior vena cava has also been noted.

The initial therapy for early electrode-induced superior vena cava syndrome was treated with bed rest and anticoagulation with heparin and subsequently with warfarin. Thrombolytic therapy has been used for initial management of electrode-induced acute thrombosis plus chronic thrombosis and for patients in whom anticoagulation fails. Successful thrombolysis usually occurs when thrombus has been present for less than 7 to 10 days. But in certain cases, thrombolytic therapy resulted in resolution of the clinical symptoms 3 weeks after onset. In instances in which superior vena cava obstruction induced by endocardial pacing electrodes, requires urgent relief, surgical decompression would be preferred. Thrombotic occlusion of the major venous tributaries may be relieved by thrombectomy with a Fogarty venous catheter, and patching or replacement of the diseased segment of the superior vena cava, and the stenotic area of the superior vena cava can be corrected by angioplasty.

Our case is the result of the thrombosis and there is no evidence of the fibrotic stenosis of the superior vena cava. In our patient surgical repair and thrombectomy with Fogarty catheter was performed because thrombotic segment failed to recanalize by thrombolytic therapy and symptoms was progres-
sive. Our pathologic findings demonstrated thrombosis and organization of thrombi with calcification around pacemaker.

Reference

3) Huang TY, Baba N: Cardiac pathology of transvenous pacemakers. Am Heart J 83: 469-474, 1972