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Case Report

Right Atrial Pacemaker Lead Thrombosis Incidentally Detected by Transesophageal Echocardiography

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A B S T R A C T

In a 62-year-old man with permanent atrial fibrillation and recurrent stroke, a large right atrial thrombus attached to a permanent pacemaker lead was incidentally identified by transesophageal echocardiography. Surgical treatment, recommended because of the large dimensions of the mass, was refused by the patient, and thrombus was successfully dissolved by anticoagulant treatment. Pathogenesis of pacemaker lead thrombosis, clinical implications, diagnostic and therapeutic options are discussed.

Key words: *pacemaker, stroke; thrombosis; transesophageal echocardiography*

Introduction

Pacemaker lead induced venous thrombosis is a well-known phenomenon that occurs in approximately 30–45% of patients after implantation of a permanent transvenous pacemaker[1]. However, development of right-heart thrombosis in the presence of permanent pacemaker leads has been rarely described. We report a case of a large right atrial thrombus attached to a pacemaker lead incidentally diagnosed by transesophageal echocardiography (TEE).

Patient and Methods

A 62-year old man had an echocardiographic examination done four weeks after the recurrence of an ischemic stroke. He had been treated because of arterial hypertension, ischemic cardiomyopathy, and permanent atrial fibrillation. A single-chamber pacemaker (Medtronic SPECTRAX S) with steroid eluting unipolar transvenous lead with silicone insulation was implanted, because of symptomatic bradyarrhythmia, thirteen years ago. CT of the brain confirmed several smaller ischemic cerebral insults in the left parietal, right occipital and temporal region. The patient was receiving aspirin 100 mg daily, as well as antihypertensive therapy. Clinical and laboratory parameters (including coagulation parameters) were within normal limits. The electrocardiogram confirmed atrial fibrillation and normal function of the pacemaker in the VVI mode. Transthoracic echocardiogram (Vivid 3, General Electric, Milwaukee, WI, USA) with a 2.5 to 3.5 MHz ultrasound transducer demonstrated increased left atrial diameter (LA = 46 mm) as well as a reduced left ventricular systolic function (LVEF = 45%). A mobile mass was seen in the right atrium, but its echocardiographic evaluation was difficult because of the patient's obesity. TEE using a 5-MHz multiplane imaging transducer, which was performed owing to poor transthoracic echocardiographic images and inability to exclude an intracardiac embolic source showed a large thrombus (25 × 21 mm) attached to the atrial segment of the pacemaker lead (Figure 1). The thrombotic mass did not propagate through tricuspide orifice, and there were no sign of acute or chronic right ventricular inflow or outflow obstruction. A spontaneous echocontrast in the left atrium and left atrial appendage, as well as reduced emptying flow velocity of left atrial appendage (0.22 m/s) was detected. The superior vena cava was free of thrombus. No other intracardiac masses were found. TEE examination with bubble contrast study (0.9% saline) and Valsalva maneuvre did not show intracardiac shunt on the atrial and/or ventricular level. Other intracardiac masses, atrial or ventricular septal defect were not found. Three-dimensional ultrasonography with Doppler study

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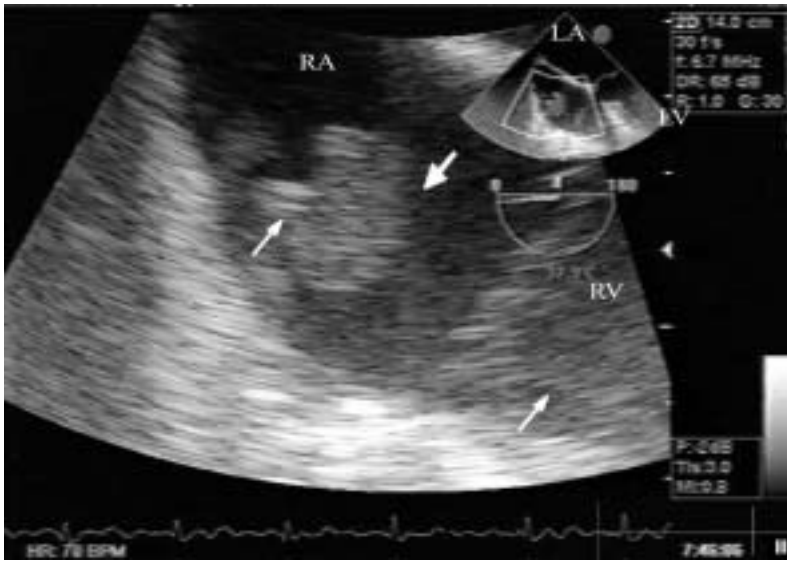

Fig. 1. Transesophageal echocardiographic image: large thrombus (thick arrow) attached to the atrial portion of the pacemaker lead (thin arrow). LA = left atrium; RA = right atrium; LV = left ventricle; RV = right ventricle.

of the carotid arteries that is a reliable diagnostic method in evaluating atherosclerotic lesions of carotid arteries [2], as well as a TEE imaging of the thoracic aorta did not show plaques that could be potential sources of embolization. Lung scintigraphy by technetium 99 did not reveal any perfusion defects.

Because of the thrombus size and mobility surgical removal of thrombus was recommended to our patient, but was refused. Thrombolytic therapy was not administered because of the recent stroke, and the patient was treated with low-molecular weight heparin followed by *per os* anticoagulant therapy with target INR within the range of 2.5 to 3.5. Echocardiographic follow-up confirmed gradual dissolution of the thrombus after 6 weeks. After complete resolution of thrombus chronic anticoagulant therapy was continued according to European Society of Cardiology (ESC) guidelines for permanent atrial fibrillation with recommended INR between 2.0 and 3.0.

Discussion

Serious thrombotic and embolic complications are reported to occur in 0.6–3.5 % of patients with permanent transvenous pacing leads[1]. Right atrial pacemaker lead thrombosis has been less frequently described, presenting either as an incidental echocardiographic finding[3] or by symptoms of right-sided heart failure[4], obstruction or embolization of pulmonary artery[5].

Several mechanisms have been postulated in the pathogenesis of pacemaker lead induced thrombosis[6]. The long-term residence of permanent pacemaker leads may act as a continous nidus for the formation of thrombus. In cases of dual-chamber pacing multiple pacemaker wires increase the total surface area of foreign material at risk for thrombus formation. Pacemaker leads may produce a foreign-body-type reaction with subsequent inflammation and fibrosis along the course of the wire, and this may lead to thrombus propagation along the pacing lead. Furthermore, congestive heart failure, hypercoagulable states such as antithrombin III, protein C and S deficiencies, thrombocytosis, malignancies, and possibly pacemaker lead material itself, may predispose to thrombosis[7–9]. It has been shown that pacemaker leads with different types of insulation have various thrombogenicity. Namely, thrombogenicity of polyurethane leads may be lower than that of silicone leads[9].

Intracardiac, generally right atrial lead thrombosis is more dangerous and difficult to manage. The hemodynamic significance of the intracardiac clot depends on its size, ranging from one to several centimeters in diameter, and location[10]. Atrial arrhythmias can occur if the sinoatrial area is involved. Similar to atrial myxomas, intracardiac thrombus may cause functional tricuspid stenosis or insufficiency, and should be considered when refractory right ventricular failure is present[4].

Systemic embolization can be related with permanent pacing in several ways. Paradoxical embolus can result in the presence of an established or transient intracardiac right-to-left shunt (i.e. latent *foramen ovale*)[11]. Transvenous pacemaker leads may be placed unintentionally into the left ventricle via a patent foramen ovale or through atrial septal defect, resulting in thromboembolic complications, primarily embolic stroke[12,13]. Finally, patients in a VVI pacing mode have a higher incidence of paroxysmal and permanent atrial fibrillation, which independently predisposes to embolic events. Patients with VVI pacing mode have a significantly higher incidence of reduced left atrial appendage function than patients with atrioventricular sequential (VDD, DDD) pacing[14–16].

Despite accepted clinical guidelines for the stroke prevention in patients with atrial fibrillation, our patient was treated only with aspirin 100 mg daily. Carlsson et al.[17] have reported that only 28% of the patients with permanent pacemaker and atrial fibrillation were anticoagulated; only 10.8% of those who were not anticoagulated had contraindications to the therapy. They concluded that the majority of pacemaker patients with atrial fibrillation, for whom anticoagulation is indicated, failed to receive it. Those caring for these patients are urged to ensure its much widely use.

In spite of the considerable size of the thrombus our patient was asymptomatic, and thrombosis of the pacemaker lead was detected incidentally. The position of the pacemaker lead in the right ventricle was confirmed by TEE, and the existence of a right-to-left shunt was excluded by contrast echocardiography. Therefore, the pacemaker lead thrombus could not be related with the previous ischemic strokes in our patient. Probable sources of recurrent stroke were thromboemboli due to permanent atrial fibrillation, as suggested by the reduced emptying flow velocity of the left atrial appendage (0.22 m/s), the spontaneous echocontrast in left atrium and left atrial appendage defined as a 3+ = moderate (dense swirling pattern during the entire cardiac cycle), according the criteria of Fatkin et al.[18]. Our patient was treated with a low-molecular weight heparin and gradual dissolution of the thrombotic formation was documented by echocardiography. Chronic anticoagulant therapy with warfarin was administered because of the increased risk of a repeated ischemic stroke.

We recommend that all patients with permanent pacemakers who have an increased risk for lead thrombosis, including those with congestive heart failure, hypercoagulable states, and immobilization, should be considered for systemic anticoagulation. However, because a case of the pacemaker lead thrombosis with massive pulmonary embolism within 12 hours after a permanent transvenous pacemaker implantation has been reported[19], anticoagulant therapy should be started early.

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TROMB NA VODIČU ELEKTROSTIMULATORA SRCA U DESNOM ATRIJU – SLUČAJAN NALAZ TRANSEZOFAGUSNOM EHOKARDIOGRAFIJOM

SAŽETAK

U 62-godišnjeg muškarca s permanentnom fibrilacijom atrija i ponavljajućim moždanim infarktom, transezofagusnom ehokardiografijom je u desnom atriju na elektrodi trajnog elektrostimulatora srca prikazan veći ugrušak. Zbog veličine ugruška preporučen je operativni zahvat kojeg je bolesnik odbio, pa je ugrušak otopljen primjenom antikoagulansa. U radu se razmatra patogeneza ugruška na elektrodi elektrostimulatora, kliničke posljedice, dijagnostičke i terapijske mogućnosti.