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Olgu Sunumu ***Case Report***

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Pacemaker İnduced Superior Vena Cava Syndrome and Subclavian Vein Stenosis: Case Report and Systematic Review

Pacemaker'a Bağlı Vena Cava Superior ve Subklaviyan Ven Darlığı: Olgu Sunumu ve Sistematik Derleme

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Özet

Vena kava superior (VKS) baş, üst ekstremiteler ve toraksın üst kesiminin venöz drenajını sağlayan ana toplardamardır. Kalp pili takılmasında neredeyse her zaman subklavian ven tercih edilmektedir. Pil implantasyonu sonrasında gelişebilen venöz darlık önemli bir problem olsa da genellikle sessiz seyreder. VKS sendromu, kalp pili takılması sonrası gelişen nadir ama önemli bir komplikasyondur. Burada, 20 yıl önce kalp pili takılan bir hastada gelişen VKS sendromu ve eşlik eden subklavian ven stenozu vakası bildireceğiz. Bir çok farklı tedavi seçeneğimiz olsa da; tedavi, vakamızda da olduğu gibi zor olacaktır.

Anahtar kelimeler: Vena kava superior sendromu, kalp pili, subklavian ven darlığı

Abstract

Superior vena cava (SVC) is the biggest venous channel carrying blood from head, upper extremities and upper thorax to the heart. Subclavian vein is almost always utilized during pacemaker implantation. Although venous stenosis after pacemaker implantation is a common complication, most of the patients remain asymptomatic. SVC syndrome is a serious but very rare complication of pacemaker implantation. Here, we report a patient with SVC syndrome and concomitant subclavian stenosis due to pacemaker implantation 20 years ago. Different treatment strategies are available but sometimes as in our case, it may be difficult to treat.

Keywords: Superior vena cava syndrome; cardiac pacemaker; subclavian vein stenosis

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Introduction

SVC is the major venous vessel that drains blood from head, neck, upper extremities, and upper thorax. SVC syndrome arises in the event of decreased or obstructed blood flow through SVC. Obstruction of SVC results in interstitial edema, and collateral blood flow due to increased venous pressure (1).

Although venous thrombosis or stenosis after pacemaker implantation is common, the patients are usually asymptomatic. The reported incidence of venous stenosis is 30-64% in literature (2), whereas the incidence of SVC syndrome is rare, ranging from 1 in every 250 to 40,000 patients (3). Several mechanical, vascular, and general risk factors for the development of SVC syndrome have been identified. Haemotological disorders such as protein C/S, antithrombin 3, factor V Leiden mutations, and thrombophilia, as well as hormone therapy, infection, and number of leads in venous vessel are important risk factors. Mechanical stress due to pacemaker lead causes vessel wall inflammation, thrombus formation and finally venous occlusion (4).

SVC syndrome is a slowly progressing process that allows formation of collateral circulation. Therefore, venous obstruction may remain asymptomatic for years. Depending on the severity of the symptoms and etiology treatment is indicated. Balloon angioplasty with or without stenting, surgery, thrombolysis, and mechanical thrombectomy are available treatment options.

Case Report

A 56 year-old male patient with the diagnosis of sick sinus syndrome who underwent dual-chamber permanent pacemaker implantation at the right infraclavicular area twenty years ago was hospitalized due to depletion of battery. The patient complained of inability to bend over without flushing and headache before the process. On physical examination, engorged neck and upper thoracic veins were seen especially in bend over position. We performed venography using right antecubital vein with the suspicion of SVC syndrome. Venography revealed obstruction of right subclavian vein and SVC with evidence of collateral flow (Figure 1). Computed tomography (CT) of the chest and neck showed significant occlusions of the proximal part of SVC and the distal part of right subclavian vein (Figures 2 and 3). Stenosis of the VCS looked like an ''aortic interruption''. Lymphadenopathy was not present on CT. Additionally, we investigated hematological parameters that may accelerate venous thrombosis. Results were as follows: homocysteine: 18,6 µmol/L (N: 0-12 µmol/L), protein C: 45 IU/dL (N: 65-140 IU/dL, protein S: 39 (N: 55-160%), anti-thrombin III: 0,31 mg/mL (N: 0,19-0,31). Factor V Leiden mutation was not present.

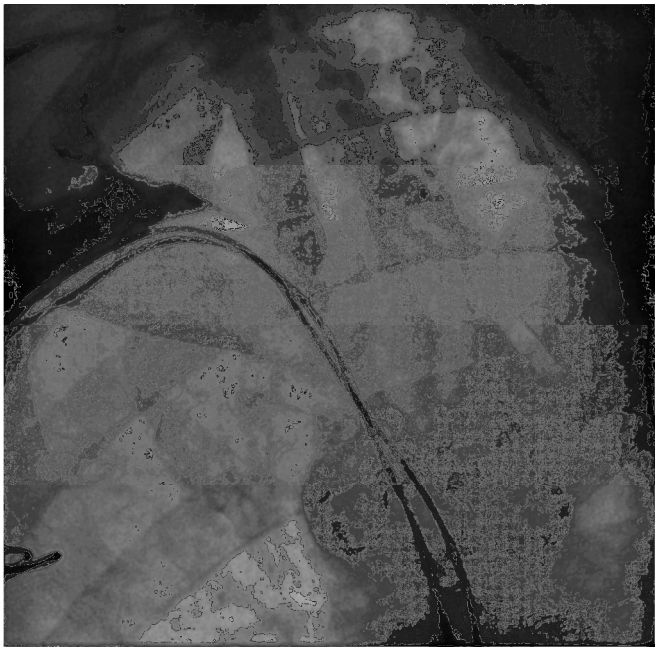

Figure 1. Severe stenosis of right subclavian vein, total occlusion of SVC, and marked increase in collateral drainage during venography.

According to patient's symptoms and clinical parameters, an initial medical treatment using oral anticoagulation (OAC) was decided. Despite a 2-month OAC treatment, clinical picture was unchanged therefore, venoplasty was planned. Venoplasty was not successful due to inability to pass guidewire through total occlusion. Finally surgery was advised to the patient; however the patient did not accept surgery due to associated risks. He is currently under medical treatment with OAC.

Discussion

We present a pacemaker-induced totally occluded SVC syndrome with ineffective medical or percutaneous treatment. SVC syndrome is a rare but serious complication of pacemaker implantation. The diagnosis of the SVC syndrome is based on patient symptoms and clinical signs. Venography is necessary for correct diagnosis. Venography not only shows stenosis of the vessel but also characterizes venous anatomy, site

and extent of the venous obstruction which is required for therapeutic strategy. Despite the limitations in delineating venous anatomy, CT is an essential tool for excluding extrinsic reasons (5), since most of the SVC syndrome cases are caused by malignant lung tumors and lymphoma (6).

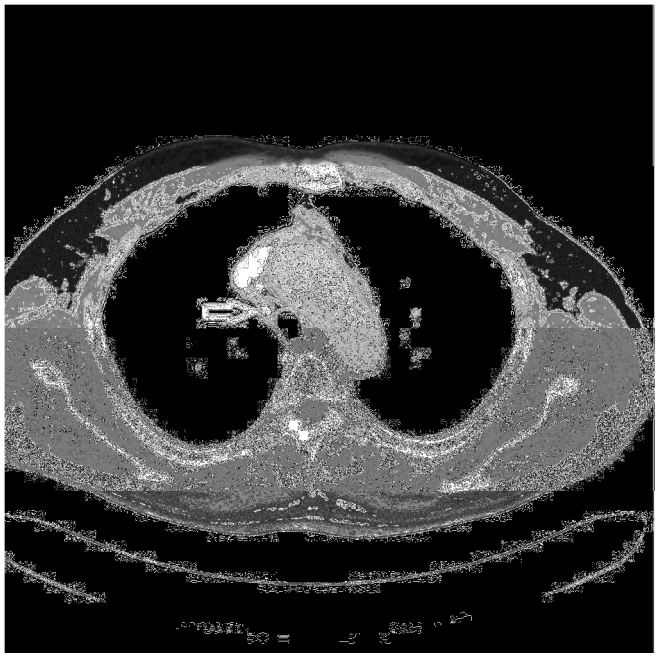

Figure 2. Occlusion and pacemaker leads (arrow) in lumen of superior vena cava.

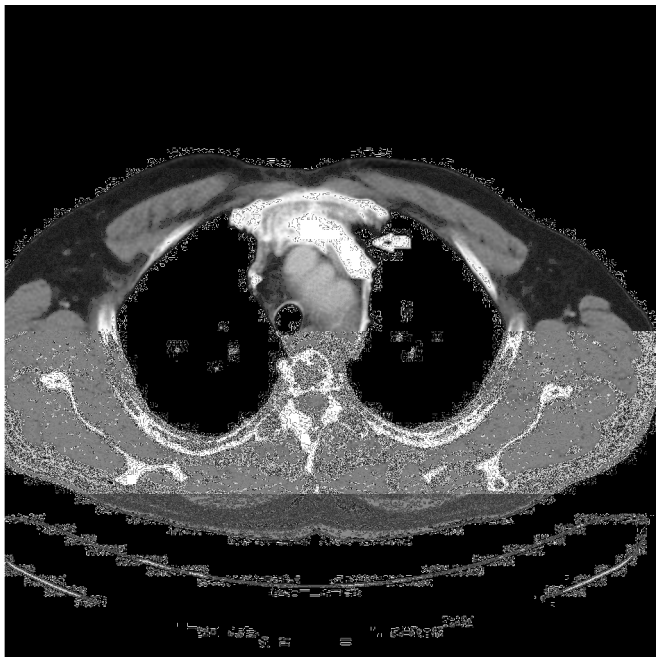

Figure 3. Enlarged brachiocephalic vein (arrow) draining into hemiazygos vein. Occluded superior vena cava and pacemaker leads in obliterated lumen are seen.

Additionally we investigated hematological parameters that accelerate venous thrombosis such as protein C/S, antithrombin 3 and factor V Leiden mutations. Homocysteine and antithrombin-3 levels were high which would facilitate propagation of venous thrombosis. Factor V Leiden mutation was not detected.

Because of the anatomical variations in occlusion site and lesion characteristics, there is no standard treatment strategy. In most patients, satisfactory results cannot be achieved by anticoagulation therapy alone, especially in totally occluded veins as in our case. Anticoagulation proves effective only in subtotal or milder occlusions. Venoplasty is the treatment of choice, and can be performed successfully in majority of patients with SCV syndrome. Many authors report successful treatment in most of the cases by venoplasty with or without stenting (7,8). However, venoplasty may also be unsuccessful, mostly due to nature of the venous occlusion, as in our patient, percutaneous intervention failed because of lesion characteristics. Surgical treatment is another option in this scenario. Surgery was recommended to the patient as the last treatment strategy. But the patient did not accept surgical treatment. Now the patient is under medical treatment which is the only way to treat. Importantly, life-long anticoagulation should be continued after successful percutaneous intervention to reduce re-occlusion.

Despite failed venoplasty and patient's reluctance to surgical treatment, during a two-year follow-up using anticoagulants our patient remained in good health with relatively minimal symptoms. Thus, our case emphasizes the difficulties in treatment of pacemaker induced SVC syndrome.

Conflict of Interest

None declared

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