Implantable Cardioverter Defibrillator Lead Thrombosis in a Patient With Recurrent Pocket Infection: A Case Report

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Abstract

Introduction: Pacemaker (PM)-related thrombosis is an infrequent complication of cardiac pacing.
Case Presentation: Here is presented the case of a 21-year-old female with long QT syndrome (LQTS) and history of aborted sudden cardiac death who inserted implantable cardioverter defibrillator (ICD) several years ago, but with recurrent pocket infection. The patient was admitted to the hospital due to pusy drainage from ICD pocket and fever two weeks prior to admission. Echocardiography revealed moderate tricuspid regurgitation and also mild right ventricle (RV) enlargement, besides a large echogenic hypermobile mass in right atrial (RA) cavity with intermittent protrusion to RV chamber via tricuspid valve. Transesophageal echocardiography demonstrated a large, mobile thrombus in the right atrium attached to the ICD lead. The patient underwent open surgery with successful thrombectomy. Then pocket of generator was revised and extensive debridement was performed.
Conclusions: RA thrombus associated with PM leads is an unusual complication of PM insertion. Although there are several management strategies for RA thrombus, including thrombolytic therapy, surgical thrombectomy and anticoagulation, the most appropriate therapeutic option remains controversial. The type of treatment should be established according to the dimension, mobility, and location of the RA thrombus. In conclusion, coagulopathy, heart failure and atrial fibrillation could be the nidus for PM (or ICD) lead thrombosis and treatment should be individualized.

Keywords: Echocardiography, Pacemaker, Implantable Cardioverter Defibrillator, Lead Thrombosis, Pocket Infection

1. Introduction

Pacemaker (PM) leads are known to be predisposing factors for thrombosis (1, 2). PM lead thrombosis is a rare but potentially fatal event. It is usually diagnosed as a coincidental echocardiographic finding and may be associated with serious complications such as pulmonary artery embolism and stroke (3, 4).

The current report is on a rare case of a 21-year-old female who presented recurrent episode of generator pocket infection and implantable cardioverter defibrillator (ICD) lead thrombosis coincidentally.

2. Case Presentation

The patient was a 21-year-old female who inserted ICD at the age of fourteen as she was diagnosed with long QT syndrome (LQTS) in the setting of an aborted sudden cardiac death. It was complicated by two further re-explorations, the first one for primary pocket erosion which required pocket revision, and the second for ICD generator re-implantation. At that time, she was afebrile, with negative serial blood cultures, normal level of C-reactive protein (CRP) and no evidence of vegetation on transesophageal echocardiography (TEE). It was followed by infection of the ICD pocket caused by Staphylococcus epidermidis requiring generator removal two years later. A fresh dual chamber ICD was implanted in the right infraclavicular pocket, followed by an elective pulse generator change.

The patient referred to the hospital in 2014 with fever for two weeks. Heart auscultation revealed a systolic murmur in the tricuspid focus, while the rest of the physical examination was unremarkable. Electrocardiogram revealed sinus rhythm with very long QT interval (QTc = 620 ms). Thoracic observation showed multiple scar of healing wound on both subclavian regions with yellowish brown drainage from left sided subclavian area. Leukocytosis was detected in the lab tests with neutrophil preference, the erythrocyte sedimentation rate (ESR) was 60 mm/h and all of the three blood cultures were negative. Chest X-ray con-
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firmed three leads in superior vena cava (SVC), with two leads in RA and one lead in RV, besides a pulse generator in the right clavicular fossa only (Figure 1).

Bacterial culture of wound discharge confirmed *Staphylococcus epidermidis* infection; therefore, intravenous vancomycin was prescribed.

Transthoracic echocardiography (TTE) showed dual coil leads in RA and RV position resulting in moderate tricuspid regurgitation besides mild RV enlargement. There was also a large echogenic hyper mobile mass, measured 1.6 × 2 cm in RA cavity with intermittent protrusion to RV chamber via tricuspid valve, but its attachment was not obvious on TTE; hence, TEE was performed. On TEE, a single mass with the same morphologic features was described; it was attached to one of the coiled leads in RA via its very narrow stalk (Figure 2).

Due to high risk of embolization, surgical removal of the mass and retraction of its corresponding lead was done. Tricuspid valve was repaired at the same time. Grossly, it was a pinkish round homogenous mass with attachment to one lead left abandoned in heart. Subsequently, pocket revision and extensive debridement was done, generator was removed and residual leads were extracted. Pocket was closed after irrigation.

No residual mass was detected by both intra operative TEE and three days later TTE.

Pathological report described the mass as a large organized thrombosis with no evidence of vegetation. The microbiological culture of mass was negative.

Intravenous (IV) antibiotic and oral anticoagulation were started. After two weeks of antibiotic therapy, a single chamber ICD was implanted at the other side. Antibiotic therapy was continued for one month.

3. Discussion

Right atrial (RA) thrombus associated with PM leads is an unusual complication of PM insertion. Previous studies reported that significant thrombotic and embolic complications occur in 0.6%-3.5% of patients with permanent transvenous pacing leads (1).

Pacemaker lead-associated thrombus formation is multifactorial and several possible mechanisms may contribute to endothelial injury, inflammation, hypercoagulability and foreign body-type reaction (5). Heart failure, atrial fibrillation and hypercoagulability may also cause thrombus formation. Implantation procedure per se probably causes a varying degree of venous endothelial injury, which can be further exacerbated by inflammation and irritation induced by friction of the transvenous lead over time.

In the current case, this may be attributed to the presence of multiple leads, and the use of dual coil leads. The presence of inflammatory state due to pocket infection may be a predisposing factor to develop thrombosis.

Inherited thrombophilic disorders increase the risk of venous thrombosis. Sabbagh et al. (6), reported that the prevalence of the methylenetetrahydrofolate reductase (MTHFR) heterozygous mutation is very high (34.6%) in the general population. Patients with the MTHFR gene mutation may have elevated homocysteine levels, often related to an increased risk of venous thrombosis (7). However, homocysteine levels may also be normal in patients with MTHFR gene mutation, as was in the current case (8).

Other coagulopathies such as protein C and S deficiency, factor V leiden and protein 20210A could be responsible for PM (or ICD) lead thrombosis. Consequently, any thrombus
might serve as a nidus for further thrombosis. However in the current case all lab tests for coagulopathy were negative.

Although there are several strategies to manage RA thrombus, including thrombolytic therapy, surgical thrombectomy, and anticoagulation, the most appropriate therapeutic option remains controversial (9, 10). Rose et al. (11), indicated that thrombolytic therapy was associated with an improved survival rate when compared with anticoagulation therapy or surgery in this clinical setting. However, bleeding and massive pulmonary embolism are some of the most feared complications of thrombolytic treatment. Surgical thrombectomy is a therapeutic option for patients with massive pulmonary embolism and open foramen ovale, while anticoagulant therapy is accepted as a potent therapeutic option to treat immobile thrombus and the patients with hemodynamic instability. Several reports showed that using anticoagulants to treat RA thrombus yields favorable results (12).

However, the type of treatment should be established according to the dimension, mobility, and location of the RA thrombus. In the current case, open heart surgery approach allowed both direct visualization and complete removal of all device components (including leads) while avoiding dissemination of large thrombus as well as repair of tricuspid valve.

Thrombolytic therapy remains the first line of treatment for the majority of patients. Streptokinase and recombinant tissue plasminogen activator (r-tPA) are effective to treat RA thrombus associated with PM leads (13, 14). However, there is no standard protocol on how to apply thrombolytic agents to treat intracardiac thrombus, since they are used in various doses and with different protocols in the literature.

In conclusion, coagulopathy, heart failure and atrial fibrillation could be the nidus for PM (or ICD) lead thrombosis and treatment should be individualized.

References