

Rupture of a pacemaker lead during the course of infective endocarditis

Enfektif endokardit seyrinde kalp pili elektrodunun kopması

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Summary—A 23-year-old male who had a VDDR pacemaker implanted seven years ago due to sick sinus syndrome and recurrent syncope episodes was admitted with symptoms of dyspnea, fever, and tachycardia, which were present for a few days. He was suspected to be suffering from pneumonia and underwent computed tomography scanning of the thorax, which revealed widespread infiltration in the lung parenchyma and pulmonary emboli. Transthoracic echocardiography revealed an extremely mobile echogenic structure in the right atrium, which was determined to be the free portion of a ruptured pacemaker lead. There was an overlying thrombus and/or vegetation-like organized soft tissue within the right ventricle around the lead component. In this article, the rupture of a permanent pacemaker lead, which complicated the course of infective endocarditis associated with pulmonary embolism and pneumonia is reported. We hypothesize that the underlying mechanism for the rupture is soft tissue entrapment within the right ventricle. Unfortunately, this rare and life-threatening situation led to the death of our patient after the surgical removal of the device and its components.

Özet—Yedi yıl önce sinüs sendromu ve tekrarlayan senkop atakları nedeni ile VDDR kalp pili yerleştirilmiş olan bir hasta kliniğimize son birkaç gündür devam eden ateş yüksekliği, nefes darlığı ve çarpıntı yakınmalarıyla başvurdu. Pnömoniden şüphelenilerek toraksın bilgisayarlı tomografisi yapılan hastada akciğer parankiminde yaygın infiltrasyon ve pulmoner emboli ile uyumlu bulgular saptandı. Transtoraksik ekokardiyografide sağ atriyum içerisinde, kopmuş kalp pili elektroduna ait olduğu anlaşılan, ileri derecede hareketli yapı saptandı. Sağ ventrikül içerisindeki diğer elektrot parçasının etrafında pıhtı ve/veya vejetasyon ile uyumlu görünüm saptandı. Bu yazıda, pulmoner emboli ve pnömoninin eşlik ettiği, enfektif endokardit komplikasyonu olarak gelişen kalıcı kalp pili elektrodunun tamamıyla kırıldığı bir hasta sunuldu. Kırılmanın altında yatan mekanizma olarak sağ ventrikül içerisindeki yumuşak dokuda sıkışma (tuzaklanma) öne sürülmektedir. Ne yazık ki bu nadir ve hayatı tehdit eden tablo, cihazın ve parçalarının cerrahi olarak çıkarılmasını takiben hastanın ölümüne yol açmıştır.

Lead fracture is a life threatening complication of permanent pacemaker implantation and has an incidence of 4%.^[1] An untethered lead can cause perforation of major vasculature or cardiac chambers, can cause penetration or fistulization through the adjacent tissues, including the bronchi, and can cause thromboembolic events as a result of fragmentation or thrombus formation. In this report, we describe a case of pacemaker lead rupture

accompanied by infective endocarditis (IE) and pulmonary embolism.

CASE REPORT

A 23-year-old male who had a VDDR pacemaker implanted seven years ago due to sick sinus syndrome and recurrent syncope episodes was admitted with symptoms of dyspnea, fever, and tachycardia, which were present for a few days. Electrocardiogram revealed an atrial flutter with a rapid ventricular rate, and his body temperature was 38.5 °C. Chest radiography demonstrated multiple cavitory lesions in the

Abbreviations:

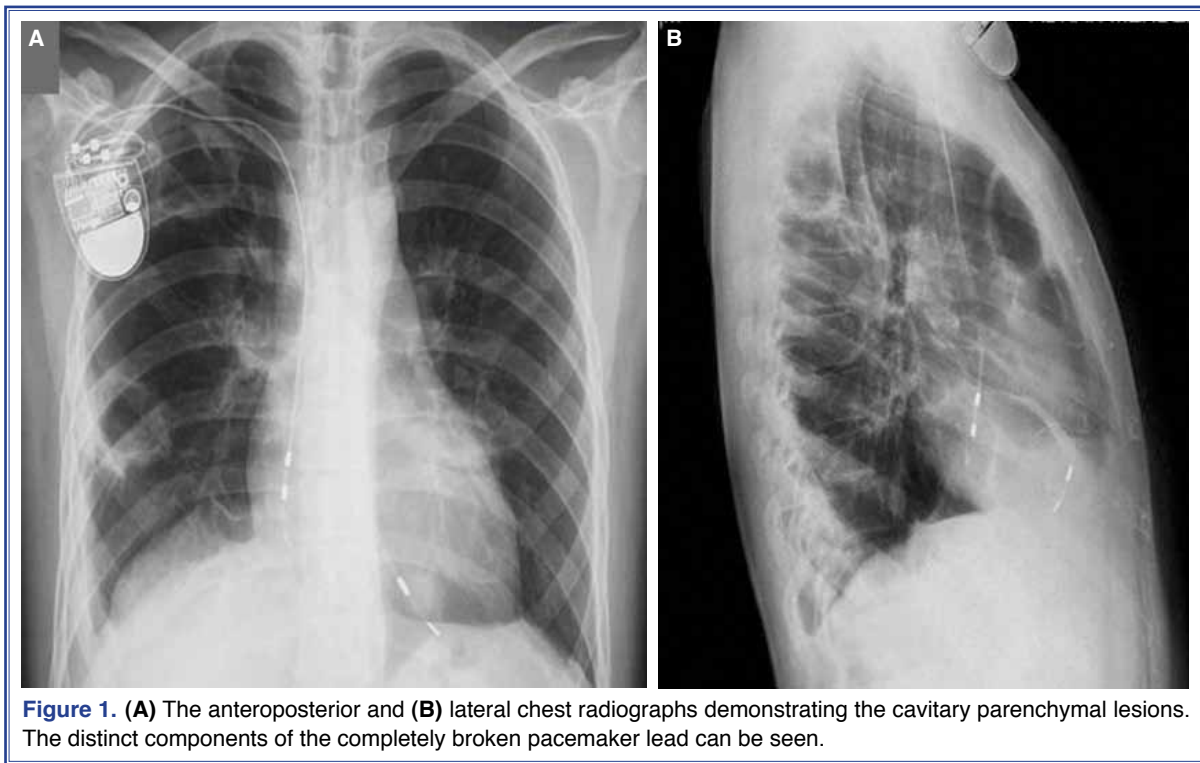
CDRIE	Cardiac device-related infective endocarditis
CT	Computed tomography
IE	Infective endocarditis
MRSA	Methicillin-resistant Staphylococcus aureus
TTE	Transthoracic echocardiography

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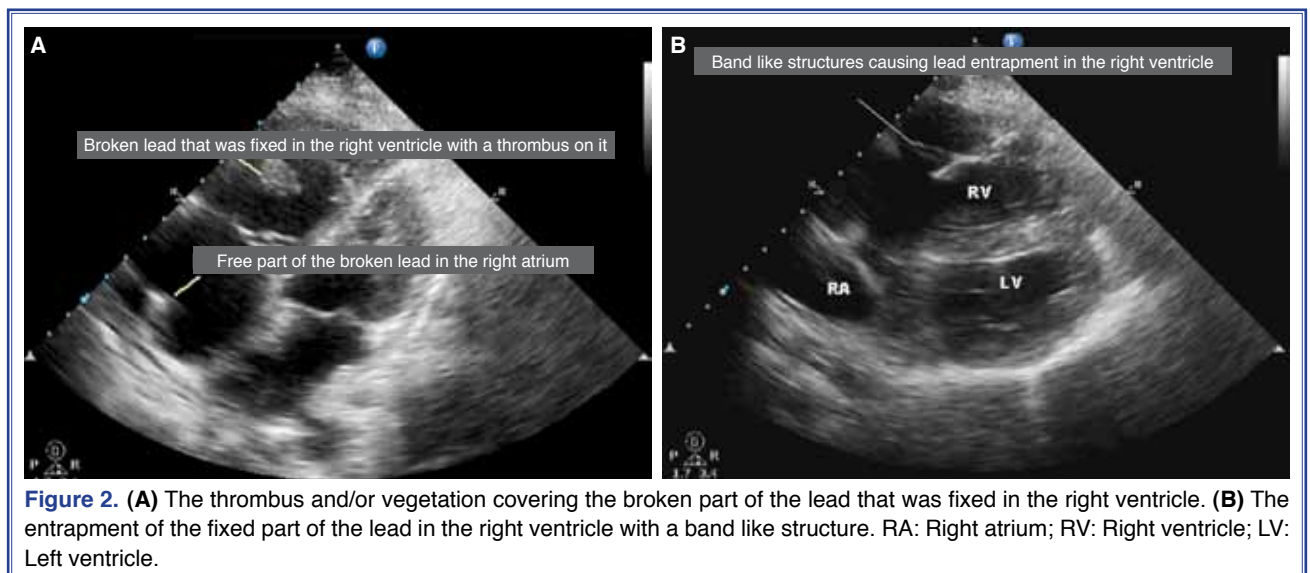
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lung parenchyma (Fig. 1a, b). He was suspected to be suffering from pneumonia and underwent computed tomography (CT) scanning of the thorax, which revealed widespread infiltration in the lung parenchyma and pulmonary emboli. Transthoracic echocardiography (TTE) revealed an extremely mobile echogenic structure in the right atrium, which was determined to be the free portion of a ruptured pacemaker lead. The other part of this lead was connected to the battery.

The untethered segment of the lead was observed to be streaming between the right atrium, the vena cava inferior, and the hepatic veins in a whipping fashion. The other part of the lead was stabilized with an overlying thrombus and/or vegetation-like organized soft tissue within the right ventricle (Fig. 2a, *see supplementary video file 1), which appeared to encircle and wrap the lead (Fig. 2b, *see supplementary video file 2). These findings were confirmed upon



fluoroscopic examination (*see supplementary video file 3). Blood cultures were obtained and empirical antibiotherapy of intravenous ampicillin-sulbactam (12 gr/day delivered in four equal doses) was initiated to suppress the systemic infection and to allow us to remove the pacemaker system as soon as possible. Methicillin-resistant *Staphylococcus aureus* (MRSA) was detected in two distinct blood cultures, and therefore during the third day of hospitalization his therapy was switched to a combination of vancomycin (30 mg/kg/day), rifampicin (1200 mg/day), and gentamycin (3 mg/kg/day). Signs of infection progressively diminished, and during the sixth day of the therapy, the infection was under control, although his pulmonary artery pressure was still high (70 mmHg). We planned to remove the entire pacemaker system, and we discussed the extraction method with the surgeons. Although the surgical approach has the advantage of avoiding inadvertent injury to the right ventricle and tricuspid valve, which may occur during percutaneous extraction, it does pose a high risk of morbidity and mortality due to the patient's pulmonary hypertension (70 mm/Hg) and poor respiratory condition as a consequence of pulmonary embolism. Thus, we decided to snare and extract the broken lead in the right ventricle, and then remove the battery and the anchored lead by a percutaneous method with a standby surgical backup. However, the patient was transferred to another center upon his will. The pacemaker system was entirely removed by a surgical method at this center, which was accompanied by debridement. Surgical observation confirmed our TTE findings that the fixed part of the lead in the right ventricle was entrapped by an excessive soft tissue. The lead was at the right ventricular apex, a little horizontal to the interventricular septum, and the thrombus and/or vegetation was covering 2/3 of the distal part, with a little spreading to the mid cavity. Soft tissue around 1/3 of the apical part and the adherent bands between the lead and the interventricular septum were found to be more dense and fibrous. During the operation, the extraction of the lead was not easy, and the traction was troublesome. The tricuspid valve was intact (unfortunately images from the operation were not taken at this center). The pathological examination of the surgical specimen was consistent with the histological features of IE, and a microbiological examination revealed the colonization of MRSA. The patient survived the operation, but died on the fifth postoperative

day due to ongoing septicemia and respiratory failure.

DISCUSSION

Pacemaker lead fracture is a serious complication that typically occurs at the costoclavicular region due to soft tissue entrapment.^[2-4] Thoracic outlet syndrome, repetitive and frequent arm movements, iatrogenic lead torsion, and fibromuscular bands between the clavicle and the first rib are risk factors for fracture. Soft tissue entrapment exerts static load upon the leads, and repeated bending at the point of entrapment is suggested to be responsible for the damage,^[4,5] which often leads to fracture of the lead, while rupture is extremely rare. The possible mechanism of the unusual lead rupture in the right ventricle in our case may be the soft tissue entrapment of the lead within the organized thrombus and/or vegetation.

Infection of cardiac devices, including permanent pacemakers, is associated with high mortality.^[6] Cardiac device-related infective endocarditis (CDRIE) is an infection involving the leads, cardiac valve leaflets, and endocardial surface. Blood cultures are positive in 77% of cases with CDRIE^[7] and *Staphylococci* are the most common causative pathogens.^[8] In the present case, a positive blood culture, pulmonary embolism, pneumonia, and TTE results are suggestive of CDRIE.

CDRIE necessitates the removal of the device since medical therapy alone is associated with high mortality and risk of recurrence.^[7] Device removal can be achieved with either percutaneous or surgical methods. Pulmonary embolism is a frequent complication of the percutaneous approach, but it continues to be the preferred method, even in the presence of large vegetations, since the overall risks are higher for surgical extractions, especially in cases with associated co-morbidities.^[7-9] Surgery is usually recommended when percutaneous extraction is technically impossible or when severe tricuspid valve IE is present.^[7] The recommendations concerning the timing of the surgery are indefinite in such a situation, but if the infection is suppressed, it may be safer to plan the intervention after the reduction of pulmonary artery pressure. In conclusion, the strategy of pacemaker removal in cases of CDRIE should be decided after reviewing the individual characteristics of each patient.

Conflict-of-interest issues regarding the authorship or article: None declared

***Supplementary video files associated with this article can be found in the online version of the journal.**

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Key words: Endocarditis, bacterial; equipment failure; heart valve diseases; pacemaker, artificial /mortality; prosthesis-related infections; pneumonia; pulmonary embolism; Staphylococcus.

Anahtar sözcükler: Endokardit, bakteriyel; ekipman bozulması; kalp kapak hastalıkları; kalp pili/mortalite; protez ile ilişkili enfeksiyon; pnömoni; pulmoner emboli; stafilokok.