Infective endocarditis associated with recurrent cardiac tamponade after cardiac resynchronization therapy

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Introduction

Infective endocarditis is among the most serious complications following the implantation of a cardiac implantable electrophysiological device (CIED) implantation. When they develop, CIED-related infections are mainly associated with right-heart infective endocarditis, which may cause tricuspid valve aneurysms or lung embolisms when vegetations detach. By contrast, cardiac tamponade rarely occurs in the context of CIED-related infection. We present the case of a patient with infective endocarditis that developed shortly after CIED implantation and led to severe cardiogenic shock and death.

Case report

A 71-year-old man was diagnosed with symptomatic complete atrioventricular block and was admitted for CIED implantation following recurrent episodes of syncope.

Two years prior to admission for CIED implantation, the patient had been hospitalized with an ST-elevation myocardial infarction and cardiogenic shock that had required intensive care admission with intra-aortic balloon pump support. During treatment, and after recovering from the cardiogenic shock, he had undergone 3 percutaneous coronary interventions for 6 severe narrowing lesions of the coronary arteries. At this time, drug-eluting stents were placed in the left anterior descending coronary artery (2 stents), left circumflex artery (1 stent), and right coronary artery (3 stents). He was discharged with a beta-blocker, angiotensin-converting enzyme inhibitor statin, diuretic, and proton pump inhibitor, and he also received 2 anticoagulant drugs. He had also received mitiglinide/voglibose tablets and insulin glargine for type 2 diabetes mellitus for 5 years.

One year after this first hospitalization, he had an episode of syncope and fell, hitting his head and developing a subarachnoid hemorrhage and subdural hematoma that required hospitalization for 3 weeks. He recovered with no adverse sequelae (ie, no limb paralysis or dysarthria), but the syncope recurred repeatedly after discharge. A 24-hour Holter electrocardiogram revealed frequent episodes of severe bradycardia and intermittent complete atrioventricular block. Moreover, transthoracic echocardiography revealed anterior, septal, inferior, and apical wall motion abnormalities in the left ventricle, with a left ventricular ejection fraction of 41%. Therefore, we determined that cardiac resynchronization therapy (CRT) was necessary to protect him against further syncope and worsening heart failure.

The patient underwent CRT-defibrillator (CRT-D) implantation. However, we had difficulty in inserting a lead of the CRT-D into the lateral branch of the great cardiac vein in the left ventricle because it was narrowed and had a crooked bifurcation. The insertion remained impossible even using double guiding catheters, and the vein eventually dissected. Thus, we attempted to use the mid-cardiac vein, but this produced twitching of the diaphragm under 2 V of pacing and was not feasible. The first session therefore ended incompletely. He subsequently received cefmetazole injections (2 g/day) for 4 days. An echocardiogram obtained after the procedure did not show a pericardial effusion.

Ten days after the first attempt, we had a second attempt at CRT-D implantation. A contrast agent infused into the great cardiac vein showed complete recovery from the dissection; therefore, we were able to place the left ventricular lead in the anterior interventricular vein to complete the CRT-D implantation. After implantation, he received cefmetazole injections (2 g/day) for 4 days and oral cefdinir (300 mg/day) for 5 days

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KEY TEACHING POINTS

- Cardiac implantable electrophysiological device–related infection primarily causes right-heart infective endocarditis (IE), which leads to either tricuspid valve aneurysms or lung embolisms. Cardiac tamponade is a rare complication of right-heart IE.
- Right-heart IE forms multiple abscesses on the epicardium of the right ventricle, and their rupture repeatedly causes cardiac tamponade.
- Fibrotic loose connection between the pericardium and ventricular muscle prevented excessive pooling around the ventricle, and it could be masked with the existence of recurrent cardiac tamponade.

after the second session. One week after the second attempt, the patient was discharged from the hospital.

Seven days after discharge, the patient developed a fever and a marked inflammatory reaction (C-reactive protein, 31.6 mg/dL) requiring admission to our hospital. Transesophageal echocardiography revealed vegetations on the tricuspid valve and atrial lead. Therefore, the CRT-D device and all leads were urgently removed and a temporary pacemaker lead was placed in the right ventricle. He was also started immediately on intravenous ceftriaxone (2 g/day) and gentamicin (120 mg/day). Peripheral blood cultures revealed methicillin-susceptible *Staphylococcus aureus*, which was confirmed on the cultures from all the leads. Therefore, the antibiotic regimen was adjusted to cefmetazole (6 g/day), and gentamicin (120 mg/day) was continued.

Despite high-dose diuretic therapy, the patient's heart failure had progressed, and by the sixth day of admission he required intensive care with mechanical ventilation and continuous hemodiafiltration to maintain his blood oxygen concentration and to improve acidosis. On the seventh day, his blood pressure decreased rapidly and he progressed to cardiac arrest, necessitating immediate percutaneous cardiopulmonary support with an intra-aortic balloon pump. Transthoracic echocardiography revealed a large pericardial effusion that was causing a cardiac tamponade, and after pericardiocentesis, his blood pressure rose immediately. Angiography did not reveal any evident perforations of the coronary arteries, great cardiac vein, or mid-cardiac vein. A computed tomography scan also showed that there was no pacing lead perforation. In total, 1700 mL of the bloody pericardial effusion was collected with a hemoglobin concentration of 2 g/dL. A drainage catheter was placed into the pericardial cavity for 3 days. However, the cardiogenic shock recurred 2 days after removing the drainage catheter. Transthoracic echocardiography failed to reveal a pericardial effusion immediately (Figure 1A and B), and it was only on the following day that a contrast-enhanced computed tomography scan showed pooling of a pericardial effusion in front of the right atrium (Figure 1C). After treatment by pericardiocentesis, blood

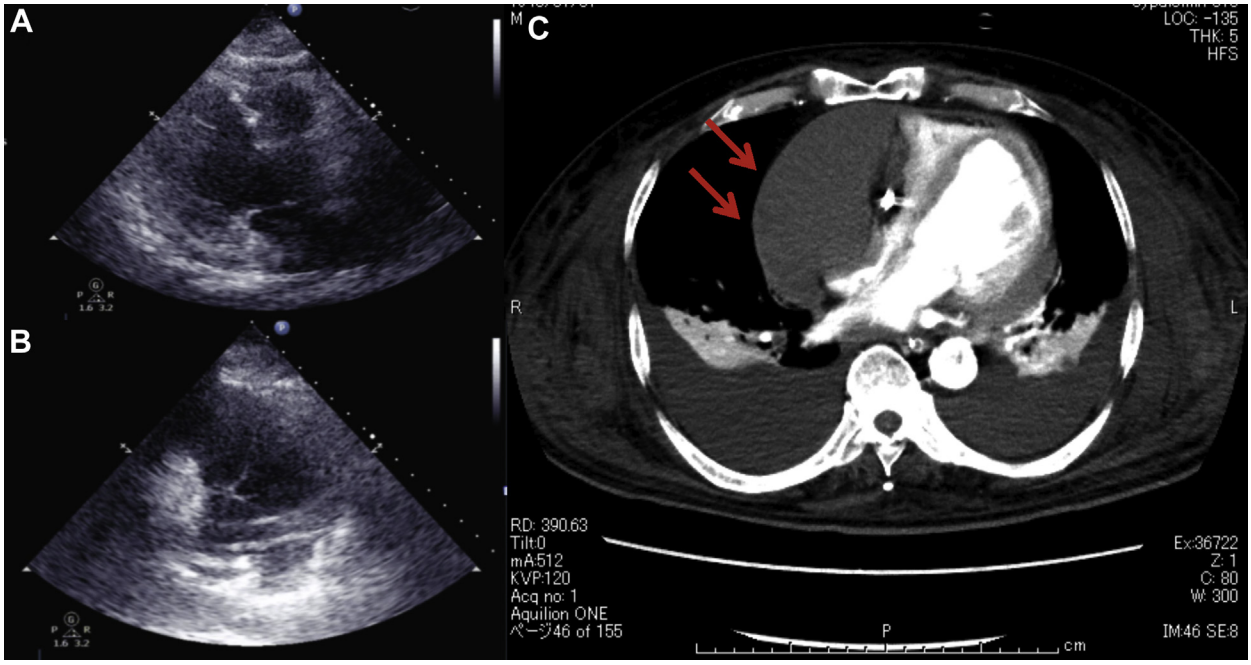

Figure 1 **A:** The normal parasternal long-axis echocardiogram did not show the pericardial effusion. **B:** A long-axis view at the level of the second intercostal space revealed pericardial effusion that had clearly pooled in front of the right atrium. **C:** Axial contrast-enhanced computed tomography showing pericardial effusion, consistent with the echocardiogram. The pericardial effusion (*red arrows*) compressed the right atrium and caused hemodynamic collapse. Effusion and atelectasis are evident in both thoracic cavities.

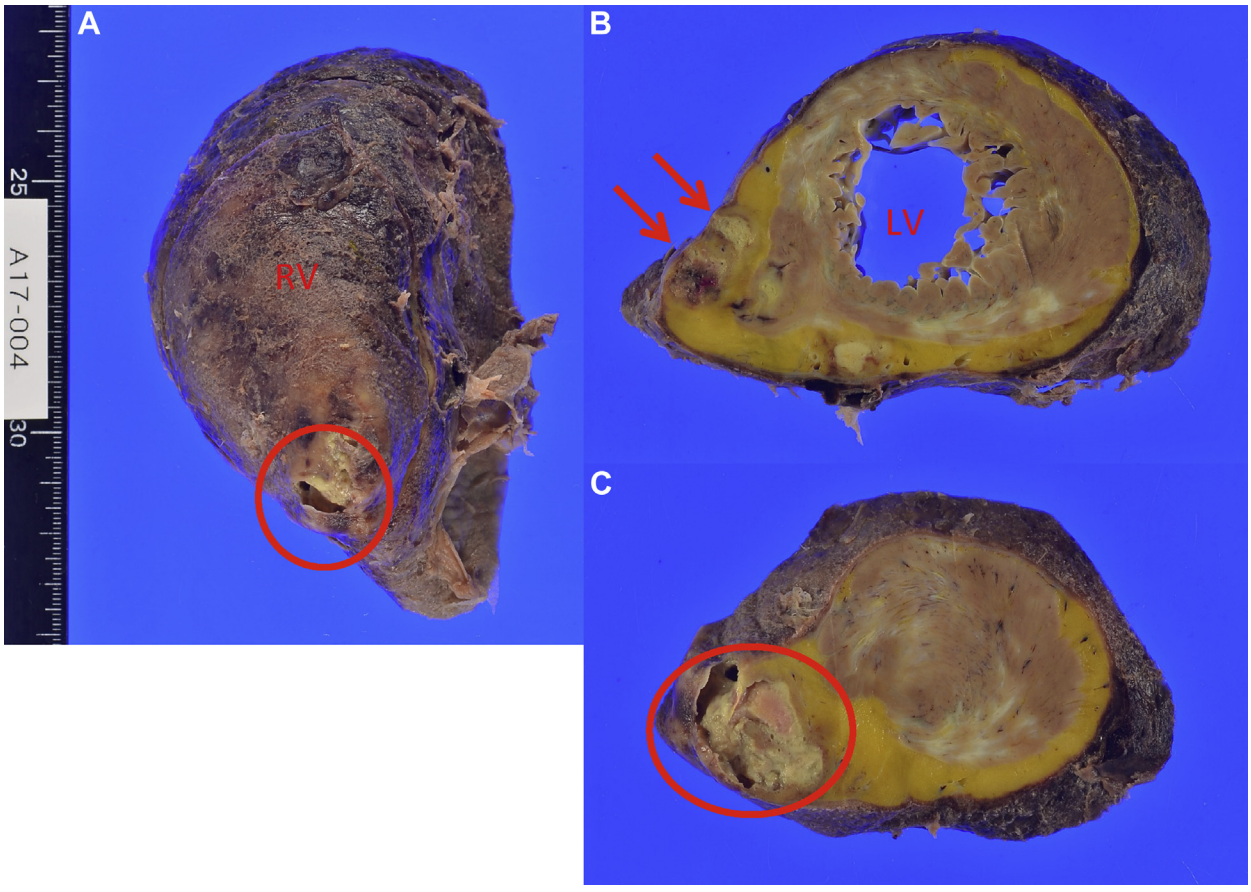

Figure 2 **A:** Image shows an epicardial tear in the apical area (*red circle*) of the right ventricle (RV), which was the remnant of an abscess rupture, with fibrotic tissue adherent to the right and left ventricles. **B:** This image shows a slice of the ventricle at the level of the papillary muscles. The *red arrows* point to the multiple abscesses present in the epicardium. **C:** The debris of abscess rupture (*red circle*) did not connect to the right ventricular chamber, indicating that the cardiac tamponade had been caused by abscesses rupture. LV = left ventricle.

pressure rose again, but multiple organ failure and disseminated intravascular coagulation developed. The patient died on day 15 of admission.

Pathologic assessment postmortem revealed the presence of multiple abscesses on the epicardium of the right ventricle, with some having ruptured (Figure 2). There was no macroscopic perforation of the great cardiac vein (Figure 3A) and hematoxylin-eosin staining showed that its inner membrane was thickened, consistent with natural repair (Figure 3B). Also, there was no perforation of the right ventricle either macroscopically or microscopically. Gram staining of the epicardial and lung abscesses revealed the presence of methicillin-susceptible *S. aureus*, despite blood cultures taken just before his death showing the presence of extended-spectrum beta-lactamase-producing *Escherichia coli*.

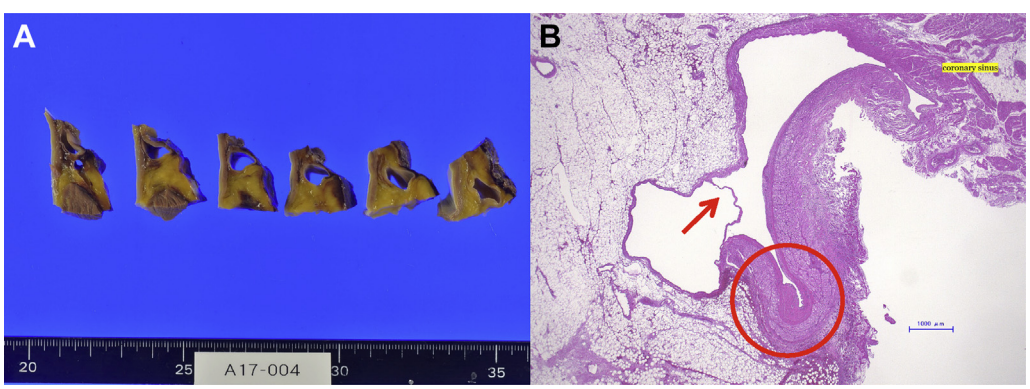

Figure 3 **A:** There was no macroscopic rupture site in the great cardiac vein. **B:** A hematoxylin-eosin stain of the great cardiac vein shows that the inner membrane (*red circle*) had repaired naturally and had become thicker than the other site. Thus, rupture of the great cardiac vein could not have caused the cardiac tamponade. The *red arrow* indicates the valve in the coronary sinus.

Discussion

Recent data indicate that the incidence of CIED-related infection has increased as the use of implantable cardioverter-defibrillators has increased.[1] In a prospective multicenter registry, the risk factors of CIED-related infection were reported as fever 24 hours before implantation, preceding temporary pacing wire implantation, and early reintervention.[2] In this case, the possible relationship between CIED-related infection and both the prolonged initial procedure time and the repeat attempts in a short period cannot be denied. We are unaware of any previous reports of repeated cardiac tamponade resulting from CIED-related infection. In this case, the recurrent cardiac tamponades that triggered the clinical emergencies and cardiogenic shock had a major impact on the patient's condition and complicated the intensive care.

Pathologically, multiple abscesses were noted to have formed on the epicardium of the patient's right ventricle apex (Figure 2A), but none of these connected to the right ventricular chamber directly (Figure 2B and C). This indicates that perforation by temporary pacemaker placement was not the cause of the cardiac tamponade. Instead, we consider the etiology of the cardiac tamponade to be as follows: (1) vegetation formed on the tricuspid valve and the atrial lead; (2) the infection moved to the apex of the right ventricle when the temporary pacing lead was inserted; (3) multiple abscesses formed on the epicardium; and (4) the pericardial effusion occurred when the abscess ruptured, leading to cardiac tamponade. The low hemoglobin concentration of the pericardial effusion suggested that the first tamponade occurred slowly and was caused by rupture of the abscess rather than the cardiac muscle. In the second tamponade, the effusion pooled in front of the right atrium (Figure 1C), and pathology showed that fibrotic tissue had adhered around the ventricle (Figure 2A). These findings suggested the possibility of a loose connection between the pericardium and the ventricular muscle that prevented excessive pooling around the ventricle, making the diagnosis of effusion more difficult.

Conclusion

We conclude that infection after CIED implantation or replacement can cause multiple epicardial abscesses and fibrosis. When these abscesses rupture, they can result in cardiac tamponade that may initially be masked and recur despite appropriate therapy.

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