Ventricular capture failure in a pacemaker-dependent patient due to *Cardiobacterium valvarum* lead-related endocarditis

Valeria Dall'Ospedale, MD,* Davide Donelli, MD,* Marta Zatti, MD,†
Gianluca Gonzi, MD,* Antonio Crocamo, MD,* Daniele Sartorio, MD, PhD,*
Giampaolo Niccoli, MD, PhD,* Maria Francesca Notarangelo, MD, PhD*

*From the *Cardiology Division, Azienda Ospedaliero-Universitaria di Parma, Parma, Italy, and †Infectious Diseases Division, Azienda Ospedaliero-Universitaria di Parma.*

Introduction

We report a case of a pacemaker-dependent patient suffering from lead-related infective endocarditis, triggered by *Cardiobacterium valvarum*—a rare HACEK group bacterium. This condition precipitated increasing pacing thresholds, leading to a loss of right ventricular capture. It necessitated a protracted antibiotic regimen, lead extraction, and a bridging solution to completely eliminate the infection prior to the implantation of a new endocavitary pacemaker. This complex case required a multidisciplinary approach.

Case report

History of presentation

A 32-year-old patient arrived at the emergency department with a 2-day history of intermittent chest discomfort, palpitations, and epigastric pain. The physical examination did not reveal any abnormalities. The patient's vital signs were stable and he was afebrile. His chest auscultation was clear and he showed no localized signs of infection or dependent edema.

Past medical history

The patient had been diagnosed at a young age with a bicuspid aortic valve, ascending aortic dilatation, and descending aortic coarctation. At 23 years old, he underwent Bentall's aortic valve and ascending aorta replacement, concurrently with descending aortoplasty. Shortly after operation, the patient developed a complete atrioventricular block, necessitating a permanent dual-chamber pacemaker configured to DDD mode.

KEYWORDS HACEK bacteria; Pacemaker endocarditis; *Cardiobacterium valvarum*; Capture failure; Lead extraction
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Address reprint requests and correspondence: Dr Davide Donelli, Cardiology Division, Azienda Ospedaliero-Universitaria di Parma, Viale Antonio Gramsci, 14, 43126 Parma (PR), Italy. E-mail address: donelli.davide@gmail.com.

KEY TEACHING POINTS

- Infective endocarditis associated with pacemaker leads presents challenges in pacemaker-dependent patients, requiring careful management and infection eradication.
- Effective management of pacemaker-related infections in pacemaker-dependent patients involves a multidisciplinary approach to guide the use of bridging solutions.
- *Cardiobacterium valvarum*, a rare HACEK bacterium, can cause lead-related infective endocarditis in pacemaker-dependent patients.

Investigations and differential diagnosis

The patient's electrocardiogram revealed an atrium-driven ventricular-paced rhythm at 78 beats per minute (bpm). Electrocardiographic monitoring recorded a loss of ventricular capture leading to a total atrioventricular block for 6 seconds (Figure 1). Pacemaker interrogation indicated a notable increase in lead impedance and ventricular threshold, from 501 ohms and 1 mV @ 0.4 ms in the week prior to hospital admission to 1953 ohms and 3.5 mV @ 0.4 ms. The thresholds obtained through the auto-tests were within the limits in the previous 10 days.

The possibility of pacemaker lead fracture or lead dysfunction owing to cardiac perforation was considered. A chest radiograph revealed no macroscopic breaks in the lead. A chest computed tomography (CT) scan was performed, revealing no abnormalities in the pacemaker lead or signs of pericardial effusion. However, a nonhomogeneous, hyperdense right paratracheal mediastinal mass with a maximum diameter of 35 mm was identified, with no aortic periprosthetic leaks found (Supplemental Video 1).

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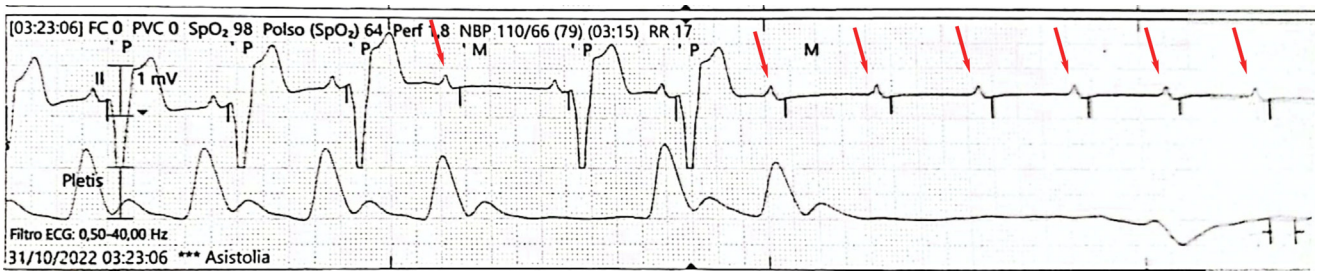

Figure 1 Emergency Department monitor electrocardiogram tracing. The arrows show the loss of capture from the ventricular lead.

Lab results indicated a white blood cell count of 10.6 $\times$ 10^3/mL, hemoglobin of 12.5 g/dL, a platelet count of 236 $\times$ 10^3/mL, normal high-sensitivity troponin levels, creatine phosphokinase and transaminases (TnI hs 6.9 ng/L, CPK 159 U/L, GOT 28 U/L, and GPT 29 U/L), lactate dehydrogenase and C-reactive protein levels slightly above the upper limit (respectively, 300 U/L and 16.1 mg/L), and procalcitonin within range (0.34 ng/mL). Toxicological exams were negative. A transthoracic echocardiography showed a normal prosthetic aortic valve without paravalvular regurgitation and the absence of pericardial effusion. Thus, while lead displacement and major aortic prosthesis malfunctions were ruled out, the possibility of mediastinitis of unknown origin was considered.

Given the suspicion of descending mediastinitis originating from an esophageal fistula, perforation, or pharyngeal phlegmon, oropharyngoscopy, fiberoptic laryngoscopy, upper gastrointestinal tract radiography, and neck and head CT were conducted, but no abnormalities were found. Blood cultures were collected despite the absence of fever owing to the suspicion of an infectious cause for the mediastinal collection.

A traumatic etiology, potentially causing both a lead fracture and a spontaneous mediastinal hematoma, facilitated by warfarin anticoagulant therapy, was considered.[1] To check for increases in the volume of the mediastinal mass suspecting a hematoma, a repeat chest CT was performed, showing no significant differences.

Subsequently, previously collected blood cultures tested positive for *Cardiobacterium valvarum*, a rare HACEK bacterium, from 2 samples. This finding shifted the differential diagnosis, strongly suggesting bacterial infective endocarditis.

A transesophageal echocardiography displayed a mobile formation (1.3 $\times$ 0.5 cm, Figure 2) attached to the ventricular pacing lead near the superior vena cava outlet. There was no evidence of vegetation in the aortic valvular apparatus. (18)F-FDG positron emission tomography was also conducted, showing no areas of elevated metabolic activity in the mediastinum and in the right ventricle (Supplemental Video 2). Consequently, the Duke Criteria for infective endocarditis were met (2 major criteria), leading to a diagnosis of pacemaker lead–related *Cardiobacterium valvarum* endocarditis.

Management

Initially, to prevent suboptimal ventricular capture in a pacemaker-dependent patient, the device was reprogrammed by increasing the output to 7.5 V @ 1 ms. However, cardiac monitoring continued to record sporadic episodes of ventricular capture deficit. Consequently, an additional permanent active fixation lead was implanted via the right subclavian vein, connected to an external permanent pacemaker, and set in VVI backup mode at 40 bpm. Figure 3 displays the chest radiograph after implantation. This technique was chosen to preclude lead displacement.

In the following days, the dual-chamber pacemaker continued to fail in ventricular capture despite a high-output setting. Thus, the backup pacemaker was set in VVI mode at 60 bpm as the primary pacing device, and the dual-chamber pacemaker was deactivated.

Upon diagnosis of lead-related *Cardiobacterium valvarum* endocarditis, the extraction of malfunctioning leads and the subsequent implantation of a new device after infection eradication were planned by a multidisciplinary team of electrophysiology and infectious diseases consultants.

Empirical antibiotic therapy with ciprofloxacin and ceftriaxone was promptly initiated following the blood culture results. Subsequent antibiograms confirmed the bacterium's susceptibility to these antibiotics. Per infectious disease consultants' recommendation, the patient was monitored with sequential blood cultures, and the implantation of a definitive endocavitary pacemaker was scheduled after 6 weeks of antibiotic therapy.

The treatment strategy necessitated a bridging solution to pace the patient while the patient was undergoing the lengthy antibiotic course needed to eliminate the infection, prior to implantation of a new permanent endocavitary pacemaker.

The extraction procedure was performed using an excimer laser technique, and an epicardial lead with an abdominal generator was implanted as a pacing bridge-to-permanent solution. Following implantation, the lead exhibited a threshold of 3.5 V @ 1.5 ms, sensitivity of 2.8 mV, and impedance of 880 ohms. Thresholds were tested every other day. On the first day after implantation, the epicardial lead experienced an increase in capture threshold, likely owing to postsurgical tissue edema, leading to an output increase to 5.5 V @ 1 ms. After 3 days from the implantation, a further increase in the

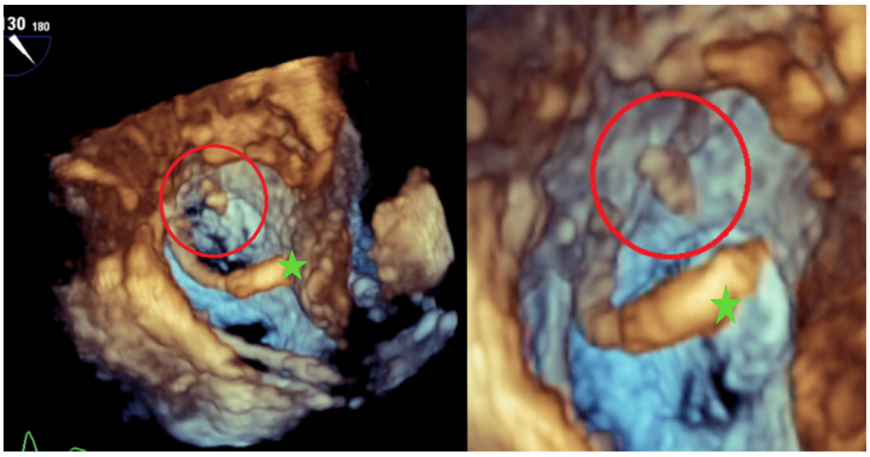

Figure 2 Three-dimensional transesophageal echocardiography reconstruction of the mobile endocarditis vegetation adhering to the pacemaker lead, captured via RATLe-90 (Rotate-Anticlockwise-Tilt-Left-90) maneuver for assessing leads in the right atrium. The red circle shows the vegetation adhering to the pacemaker lead (*green star*).

capture threshold was observed, and the output was set at 7.5 V @ 1.5 ms, resulting in stable ventricular capture (in the following weeks the threshold decreased to 3 V @ 1.5 ms).

Once ventricular capture was stabilized, the backup pacemaker was removed, and the patient was transferred to a long-term care facility to complete his antibiotic course. Serial blood cultures remained negative.

Lastly, regarding the mediastinal collection, which was likely to be considered as a self-limiting spontaneous hematoma, given that the patient was on anticoagulant therapy and since mediastinitis was ruled out because the positron emission tomography scan was negative, a watchful waiting strategy was decided upon, as it did not increase in size.

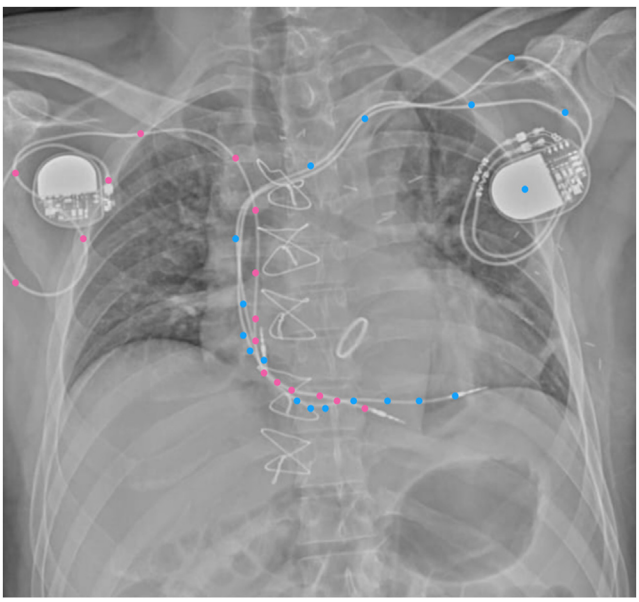

Figure 3 Chest radiograph after active fixation lead insertion by right subclavian vein connected to an external permanent pacemaker (*pink dots*) and the dual-chamber pacemaker (*blue dots*).

Follow-up

Upon completing the antibiotic therapy, the patient was discharged. During follow-up interrogations of the epicardial pacemaker (which was programmed in VVIR 60 bpm), acceptable ventricular thresholds were recorded, enabling a decrease in output to 1 V @ 1 ms. A CT scan performed 1 month later confirmed the complete resolution of the mediastinal collection.

After completing specific antibiotic therapy, achieving normalization of inflammatory blood markers, and consistently obtaining negative blood cultures, the patient was discharged. A new pacemaker was implanted, but the procedure was delayed by 6 months owing to the patient's personal reasons. This pacemaker included a lead in the coronary sinus and an atrial lead, with the generator positioned at the right subclavian level. During the same hospital stay, the abdominal generator was removed and the epicardial lead, which was placed on the right ventricle, was connected to the new subclavian generator.

Discussion

HACEK infections occur when bacteria from the HACEK group (*Haemophilus* species excluding *H. influenzae*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella*, and *Kingella*), which typically reside in the normal oral cavity and respiratory tract flora, enter the bloodstream through various mechanisms. These can include dental procedures resulting in bacteremia, inhalation of respiratory secretions, or trauma. While HACEK bacteria are generally associated with endocarditis, accounting for approximately 1%–5% of all bacterial endocarditis cases, they can also cause infections in other body parts. Few instances of mediastinitis have been described as well.

Individuals with underlying heart conditions, such as heart valve abnormalities, the presence of a cardiac implantable electronic device (CIED), artificial heart valves, or a prior

history of endocarditis, are at a higher risk for HACEK infections.

Cardiobacterium valvarum is a rare species within the *Cardiobacterium* genus, with *Cardiobacterium hominis* being more common.[2]

According to the 2019 International Criteria for CIED Infections, a definite diagnosis was established.[3] These guidelines recommend the removal of the generator and pacing leads, coupled with a 4-week course of antibiotics for native valve HACEK infective endocarditis. For prosthetic valve HACEK infective endocarditis, a 6-week course of antibiotics is advised, with cephalosporins as the primary treatment option.

Conclusion

To the authors' knowledge, this is the first reported case of infective endocarditis caused by *Cardiobacterium valvarum*, leading to increased pacing thresholds and capture failure, likely owing to an exit block from a local inflammatory response and subsequent fibrosis. This case emphasizes the complex management of CIED infection in pacemaker-dependent patients with subtle infective clinical manifestations and underscores the necessity of a multidisciplinary team treatment strategy.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.03.008.

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