CASE REPORT

Automated implantable cardioverter defibrillator lead infection in a patient with previous superior vena cava thrombosis

Tara Connelly,¹ Sadiq Siddiqui,² Walenty Kolcow,² Dave Veerasingam²

¹Department of Surgery, Galway University Hospital, Galway, Ireland

²Department of Cardiothoracic Surgery, Galway University Hospital, Galway, Ireland

Correspondence to
Dr Tara Connelly,
tara_conn@hotmail.com

Accepted 15 October 2015

SUMMARY

We present a case of a 44-year-old woman who presented with cough, pleuritic chest pain and fever leading to a diagnosis of pneumonia±pulmonary embolism. She had a history of familial hypertrophic obstructive cardiomyopathy (HOCM), for which an automated implantable cardioverter defibrillator (AICD) had been implanted, and a subsequent superior vena cava (SVC) thrombus, for which she was anticoagulated with warfarin. On admission, blood cultures grew a coagulase-negative *Staphylococcus*. CT pulmonary angiogram and transoesophageal echocardiography (TOE) were performed and revealed large vegetations adherent to the AICD leads with complete occlusion of the SVC. The infected leads were the source of sepsis. Open surgery was planned. For cardiopulmonary bypass, the venous cannula was inserted in the inferior vena cava (IVC) and a completely bloodless field was obtained in the right atrium allowing for the extraction of the AICD leads completely, along with the adherent vegetations from within.

BACKGROUND

Implanted cardiac devices such as pacemakers and automated implantable cardioverter defibrillators (AICDs) can become infected, similarly to any other foreign body. The presentation, consequences and treatment of device infection vary according to the location and extent of the infection and the clinical characteristics of the patient.¹ Despite the use of antibiotic prophylaxis at the time of device placement and revision, device-related infection is not a rare occurrence.² The majority of such infections are caused by *Staphylococcus aureus* or coagulase-negative staphylococci infections. Coagulase-negative staphylococci typically have an indolent presentation. *S. aureus* infections, particularly those complicated by bloodstream infection and AICD-related infective endocarditis, develop more rapidly and are associated with more severe systemic manifestations.^{3–5} Prompt device removal and prolonged parenteral antibiotic administration decrease mortality among patients with such infections.⁶ Lead extraction in patients having infected leads with small adherent vegetations can be performed employing percutaneous techniques, radio frequency ablation and laser. In cases with large vegetations, open-chest lead extraction may be required. This patient cohort typically manifests more severe systemic symptoms of their device

infection and have higher mortality rates compared to those undergoing minimally invasive extraction.⁷

CASE PRESENTATION

A 44-year-old non-smoking woman presented to the emergency department (ED) with a cough, right-sided pleuritic chest pain and night sweats for 4 days. She had been admitted to an outside hospital for the treatment of a lower respiratory tract infection (LRTI) twice in the previous 6 weeks. Her medical history was significant for familial hypertrophic obstructive cardiomyopathy (HOCM). This was determined during the screening of all family members after the sudden cardiac deaths of her father and a brother who died in his teens. The patient was diagnosed with HOCM and a defibrillator was placed 19 years prior to this presentation. A replacement AICD was inserted in 2006. A device failure occurred in 2008 and the box was changed. At that time, the innominate vein was thrombosed from her previous lead hence the device was implanted on the right. One appropriate shock for ventricular fibrillation was noted during the same year. In 2010, the patient was diagnosed with thrombosis at the junction of left internal jugular vein and superior vena cava (SVC), and started on warfarin, which she continued to take.

On examination in the ED, she was vitally stable and afebrile. Obvious venous distension was noted on the anterior chest wall. Her AICD scar appeared healthy with no evidence of skin erythema. Tenderness on the right anterior chest wall and under the right breast was elucidated on palpation.

Figure 1 Chest X-ray. A right hilar mass with effusion and atelectasis were demonstrated. The automated implantable cardioverter defibrillator is visible on the right side of the chest.

To cite: Connelly T, Siddiqui S, Kolcow W, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-211772

Examination of the cardiovascular system was unremarkable with no murmurs noted.

INVESTIGATIONS

Full blood count revealed an elevated white cell count of $15.3 \times 10^9/l$. C reactive protein was 133 mg/dl. International normalised ratio (INR) was 5.5. mg/dl Warfarin was stopped due to the high INR.

A right hilar mass with effusion and atelectasis were demonstrated on chest X-ray (figure 1). Broad spectrum antibiotic treatment for a LRTI was started.

The evening following admission, the patient became febrile. Transoesophageal echocardiography (TOE) and CT pulmonary angiogram (CTPA) were ordered. TOE showed calcified and mobile leads in the right atrium and ventricle and large, mobile vegetations adherent to the AICD lead in the right ventricle (figure 2). CTPA showed a 2.8×3 cm expansile embolus in the right main pulmonary artery suggestive of an infective or tumour embolus. The AICD leads in the SVC were also visualised. As the SVC was thrombosed, no blood flow was appreciable (figure 3).

Blood cultures grew *Staphylococcus hominis* and microbiology advised the addition of vancomycin.

DIFFERENTIAL DIAGNOSIS

When a thorough head to toe physical examination revealed no obvious source of infection it became apparent that the AICD was the source of sepsis. Differential diagnoses would include an incidental finding of thrombus or cardiac tumour, for example, a myxoma. However, cardiac tumours are rarer in the right atrium and the mass was adherent to the lead, making both differentials unlikely.

TREATMENT

Owing to the complexity of the case, after joint discussion between cardiology, cardiothoracic surgery and infectious diseases, the decision was made to remove the AICD along with its leads. Owing to the size of the vegetations and the SVC thrombosis, open surgery was performed.

Under general anaesthesia, a median sternotomy and a separate, right upper chest transverse incision were performed for cardiac access and device removal, respectively. On full cardiopulmonary bypass, the right atrium was opened in a bloodless field. Venous drainage was through the IVC, as the SVC was completely thrombosed, and cardiopulmonary bypass was

achieved. Despite the SVC thrombosis, mobilisation of the leads from both ends was achieved, and the infected leads and vegetations were removed from within the heart (figure 4). On examination, the tricuspid valve was intact with no evidence of vegetations. The AICD box was then removed from the chest wall. Thus the foreign material was removed in its entirety. The vegetations and leads were sent for histopathology, and culture and sensitivity.

OUTCOME AND FOLLOW-UP

Microbiology results showed that the lead tips grew coagulase-negative *Staphylococcus* and the vegetations grew *Staphylococcus epidermidis* sensitive to gentamycin and erythromycin. Histology of the vegetations showed fibrinopurulent exudate.

Postoperatively, once the wounds were healing well and the drains were removed, the patient was transferred to the care of the infectious disease department. Her postoperative course was uneventful and she was continued on intravenous antibiotics for 6 weeks. Another AICD was placed again, subcutaneously, 6 weeks later. During this period, the patient was treated with amiodarone to prevent arrhythmias and her warfarin was restarted.

DISCUSSION

Although AICD implantation is potentially life-saving, infection is an uncommon but devastating complication with a mortality rate of up to 33%.² Device infections can be classified by location and/or source. Pocket infections involve the subcutaneous pocket containing the device and the subcutaneous segment of the leads but not the transvenous segment. Deeper infections involve the transvenous portion of the lead, typically with associated bacteraemia and/or endovascular infection. Deep infection can occur with or without involvement of the generator pocket and can include device-related endocarditis in which vegetations may be found on the intracardiac portion of the lead. The above described case is that of a deeper type of AICD infection. In primary infections, the device and/or pocket itself is the source of infection. This type of infection is usually due to contamination at the time of implantation. Secondary infection is caused by seeded bacteraemia from a different, non-AICD. As in our case, coagulase-negative *Staphylococcus* is the most common cause of implantable cardiac device-associated infection. *S. hominis* was cultured in our patient's blood making the foreign body, the AICD, the most likely source, as implantable cardiac device-associated infection rates rise with the duration that the device has been implanted.³

Risk factors associated with AICD infections include renal failure, complications such as haematoma formation at the generator incision site and the implantation of devices with multiple leads.⁴ Blood cultures are recommended for all patients and TOE is recommended for patients with signs of sepsis and systemic illness. Complete removal of the device including its leads is required for cure. Patients should be cared for in centres with multidisciplinary expertise in the management of such infections.^{5 6} In 2010, the American Heart Association (AHA) published guidelines that addressed the diagnosis, treatment and prevention of AICD infections.⁸ These guidelines consider complete device and lead removal to be mandatory in all cases of AICD infection.

The case described above is interesting for several reasons. An overlap in symptoms from the infected device and an LRTI, brought about the treatment of the LRTI and a missed diagnosis of an AICD lead infection. Additionally, the previous total occlusion of the SVC, necessitating venous drainage through the

Figure 2 Transoesophageal echocardiography showing calcified and mobile leads in the right atrium and ventricle (small arrows), and large, mobile vegetations adherent to the automated implantable cardioverter defibrillator lead in the right ventricle (large arrows).

Figure 3 CT pulmonary angiogram showing a 2.8×3 cm expansile embolus in the right main pulmonary artery suggestive of an infective or tumour embolus (large arrow). The automated implantable cardioverter defibrillator leads in the superior vena cava (SVC) are also visualised. As the SVC is thrombosed, no blood flow is appreciable (small arrow).

IVC during extraction of the infected device, and the concomitant presence of a mycotic embolus in the right pulmonary artery, were unusual and challenging aspects of this case.

Treatment of the embolus in the right main pulmonary artery was considered at the time of surgery. However, to open the

pulmonary artery would require the institution of deep hypothermic circulatory arrest, which would complicate the surgery further and add to operative risk. Echocardiogram and ECG findings did not demonstrate right ventricular strain; this showed that the embolus was not haemodynamically significant. At the time, the operating surgeon felt that, as the patient would have long-term antibiotic treatment and would be on anticoagulation for treatment of previous venous thromboembolism, the risks of removing this embolus would outweigh the benefits. The vegetation in the right pulmonary artery could potentially be a source of a repeated AICD endocarditis. Thus, the patient will require careful lifelong monitoring.

Learning points

- ▶ Diagnosis of automated implantable cardioverter defibrillator (AICD) lead infection can be delayed due to misdiagnosis secondary to overlapping symptoms from other diseases such as pneumonia.
- ▶ AICDs with recurrent infections may occlude or cause thrombosis occluding large vessels including the superior vena cava.
- ▶ AICD infection requires removal of the infected device.
- ▶ Large vegetations adherent to AICD leads are not amenable to percutaneous extraction and require an open surgical procedure.

Figure 4 Pathology specimen of explanted automated implantable cardioverter defibrillator with two large vegetations adherent to the leads.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Baddour L, Cha Y, Wilson W. Clinical practice. Infections of cardiovascular implantable electronic devices. *N Engl J Med* 2012;367:842–9.
- 2 Mond H, Proclemer A. The 11th world survey of cardiac pacing and implantable cardioverter-defibrillators: calendar year 2009—a World Society of Arrhythmia's project. *Pacing Clin Electrophysiol* 2011;34:1013–27.
- 3 Anselmino M, Vinci M, Comoglio C, et al. Bacteriology of infected extracted pacemaker and ICD leads. *J Cardiovasc Med (Hagerstown)* 2009;10:693–8.
- 4 Le K, Sohail M, Friedman P, et al. Clinical features and outcomes of cardiovascular implantable electronic device infections due to staphylococcal species. *Am J Cardiol* 2012;110:1143–9.
- 5 Voigt A, Shalaby A, Saba S. Continued rise in rates of cardiovascular implantable electronic device infections in the United States: temporal trends and causative insights. *Pacing Clin Electrophysiol* 2010;33:414–19.
- 6 Uslan DZ, Sohail MR, St Sauver JL, et al. Permanent pacemaker and implantable cardioverter defibrillator infection: a population-based study. *Arch Intern Med* 2007;167:669–75.
- 7 Guimaraes M, Schonholz C, Hannegan C, et al. Radiofrequency wire for the recanalization of central vein occlusions that have failed conventional endovascular techniques. *J Vasc Interv Radiol* 2012;23:1016–21.
- 8 Baddour L, Epstein A, Erickson C, et al. Update on cardiovascular implantable electronic device infections and their management: a scientific statement from the American Heart Association. *Circulation* 2010;121:458–77.

Copyright 2015 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow