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Septic Shock due to Implantable Cardiac Defibrillator Related Infection

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Abstract

Infection is an important complication of cardiac device implantation. We report the case of a 61 year old patient presenting with septic shock caused by cardiac device infection (CDI) three-weeks after device implantation. At initial presentation, there was an absence of both localising signs and echocardiographic evidence of CDI. Later, Staphylococcus aureus was cultured from blood and the pre-pectoral pocket. 48 hours after admission the device and leads were explanted in theatre by simple traction. Despite appropriate antibiotics and full supportive care (including haemofiltration, ventilation and inotropic support), the patient died on day six. Cardiac device infection may present with septic shock in the absence of localising features. A high index of suspicion is required, particularly for early CDI.

Introduction

AMortality and morbidity associated with cardiac device infection (CDI) is likely to increase as the indications for device therapy in cardiac disease broaden [1]. Septic shock is a rare presentation of CDI. Early explantation and antimicrobial therapy can avert an adverse outcome but clinical suspicion must be high particularly if, as in the illustrated case (below), there is an absence of both localising signs and echocardiographic evidence of infection (at presentation) .

EA 61-year-old male presented with a 10 day history of general malaise with fever, flu-like symptoms, mild abdominal pain and watery diarrhoea. He had received a course of antibiotics 12 days previously for sinusitis. His past medical history included type II diabetes mellitus treated with gliclazide.

Three-weeks previously he underwent dual chamber implantable cardioverter defibrillator (ICD) implantation (Boston Scientific Teligen™ 100 dual chamber ICD). He was presenting with palpitations and, ventricular tachycardia (VT) was documented. Further investigation revealed moderate to severe left ventricular systolic dysfunction (estimated ejection fraction by echocardiography 30%) and extensive coronary artery disease. The coronary artery disease was not amenable for revascularisation and managed medically. A VT stimulation study was positive. The ICD was implanted in the left pre-pectoral pocket with single-dose cefuroxime 1.5g intravenous antibiotic prophylaxis.

On his latest presentation his blood pressure was 85/40mmHg and pulse 115bpm. He was afebrile.

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There were neither cardiac murmurs nor clinical stigmata of infective endocarditis. His generator site at presentation was not swollen and there were no signs of induration or inflammation.

He had a thrombocytopenia and anaemia (platelets 67, Hb 11.0g/dl) but his white cell count was normal. There was renal impairment with urea and creatinine of 18.4 and 376 mmol/l respectively. His cardiac rhythm was atrial fibrillation. There was no consolidation of lung fields on chest x-ray and both leads of the ICD were in satisfactory positions.

The initial working diagnosis was gastroenteritis with dehydration, precipitating acute kidney injury and atrial fibrillation. Clostridium difficile infection was considered because of the recent antibiotic exposure.

In the initial hours following presentation he received fluid resuscitation in the accident and emergency department, but remained hypotensive and anuric. He was transferred to the critical care unit where infusions of noradrenaline, enoximone and dopexamine, and con tinuous veno-venous haemofiltration were commenced. His respiratory function deteriorated and he required intubation. Empirical antibiotics (cefuroxime and metronidazole) were given.

The differential diagnosis was now considered to be CDI, or an intra-abdominal focus of sepsis.

A CT scan of his abdomen was performed, which demonstrated significant free fluid, particularly around the pancreatic head. A subsequent exploratory laparotomy confirmed free fluid, but was otherwise unremarkable.

Blood cultures grew a gram positive cocci (day two). An ultrasound scan revealed a small fluid collection around the ICD, which was aspirated and this also grew gram positive cocci on culture. The antibiotic regime was adjusted to vancomycin, clindamycin and linezolid, pending sensitivities of the organism. Transthoracic echocardiogram did not identify any vegetations on the valves nor on the ventricular leads although shielding by the distal coil of the ICD ventricular lead made interpretation difficult. 48 hours after admission the ICD system was explanted in theatre in its entirety. There was purulent fluid released from the generator site. The organism in the blood culture and collection around the ICD was identified as Staphylococcus aureus. The antibiotic regime was changed to flucoxicillin monotherapy administered intravenously

By day six the patient had remained noradrenaline dependent, had shown no recovery in renal function and had developed an ischaemic liver injury. His temperature was persistently > 39°C despite appropriate antibiotic treatment. Finally, through sepsis and high-dose noradrenaline, his fingers and feet had become critically ischaemic and no longer viable. After discussion with the patient's family, care was withdrawn.

Discussion

TThere are relatively few reports in the literature describing septic shock as the mode of presentation for CDI and no previous detailed report, as far as we are aware, of Staphylococcal aureus as the causative organism.

The main learning points from this case are that (i) vigilance and high suspicion of index is required for CDI, (ii) clinical localising signs and echocardiographic evidence of infection may be absent at presentation, and (iii) prompt explantation of the entire device (generator and leads) is important. In the case described, CDI was not considered at first presentation because of the absence of clinical localising signs. Explantation of the device was carried out, but only after the patient had developed established multi-organ failure.

Criteria of a CDI, as opposed to secondary infection of the device, as indicated by Chamis et al. [2] are fulfilled in this case. There was no other source of bacteraemia identified, there was positive culture from the generator site and the implantation was less than one year ago. However, there were no strong pointers to CDI at presentation and the delay in diagnosis is likely to have contributed to the adverse outcome.

The presentations of CDI are diverse. Pocket infection may present with pain, swelling and discharge, while pacemaker lead endocarditis may present with systemic features (e.g. fever)

and pulmonary features (e.g. cough, shortness of breath). The absence of localising features at presentation in this case remains unexplained.

The rate of CDI rates varies between 1.6% to 6.7% [1;3;4]. In a large retrospective series of CDI the overall infection rate was 1.6% and the rate of confirmed pacemaker endocarditis was 0.3% [1]. The most commonly identified pathogens are those of the Staphylococci family - usually S. aureus or S. epidermidis. Other organisms - including fungi [5] - are occasionally implicated.

In a comprehensive series of 33 patients with 'definite' pacemaker endocarditis (based on surgery or autopsy histologic findings or bacteriologic findings or electrode-tip wire vegetation), three patients presented with septic shock [6]. All three patients had lead vegetations, with two out of the three cases surviving. Time to explantation and its possible influence on outcome was not detailed.

Explantation of device has been shown to be important [2] for outcome and it is fair to say that this is time dependent. Our case highlights the possibility of fulminant sepsis in CDI with Staphylo coccus aureus bacteraemia in the absence of localising signs or symptoms at presentation and in the absence of (transthoracic) echocardiographic evidence of vegetations. A high index of suspicion is important, particularly for early CDI (<1 year) [2].

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