Case Report

Multisystem organ failure secondary to Haemophilus parainfluenzae infective endocarditis on an ICD lead: A case report

Rachel E. Bridwell, MD⁎, Amber Cibrario, DO, Brit Long, MD, Anthony M. Cho, MD

⁎ Department of Emergency Medicine, UT Health San Antonio, 7703 Floyd Curl Drive, San Antonio, TX 78229, United States of America
Department of Emergency Medicine, San Antonio Uniformed Services Health Education Consortium, 3551 Roger Brooke Drive, Fort Sam Houston, TX 78234, United States of America

1. Introduction

Implanted cardiac device (ICD) and pacemaker infection is an increasing problem, carrying an infection and mortality rate of 1–7% and 30–35%, respectively [1,2]. While causative bacteria in infective endocarditis (IE) are more commonly Staphylococci, Haemophilus parainfluenzae, a member of the HACEK family, accounts for 1–3% of endocarditis cases [3,4]. With rates of cardiac device implantation increasing, consideration of device infection in a septic patient with an ICD is needed [5]. We discuss a case of multiorgan system failure as a result of Haemophilus parainfluenzae endocarditis on right ventricular ICD leads.

2. Case presentation

A 33-year-old female with a history of polysubstance abuse and atrioventricular pacemaker was brought from jail after being detained for heroin use and possession with decreased responsiveness, defecation, and vomiting for 2 days with concern for heroin withdrawal. She was evaluated 2 days earlier and given dicyclomine. Initial vital signs were a heart rate of 89 beats per minute, blood pressure of 115/85 mmHg, respiratory rate of 20 breaths per minute (bpm), 100% oxygen saturation, physical evidence of pacemaker placement, and a core temperature of 95 degrees Fahrenheit. On exam, she was alert and oriented to name only, she had obvious track marks in antecubital fossae consistent with intravenous (IV) drug abuse, diffusely tender abdomen, and jaundiced skin with poor turgor. Resuscitation included two liters of IV fluid, 2 g IV cefepime, and 1 g IV vancomycin. Mean arterial pressure (MAP) did not improve with resuscitation, with increasing tachypnea to 30 bpm. Central venous access was obtained via the right internal jugular vein and norepinephrine started. Foley-catheter demonstrated tea-colored malodorous urine. White blood cell count (WBC) 41,700/μL, hemoglobin (Hgb) 8.4 g/dL, and platelet count 9000/mm³ were present on laboratory evaluation. Basic metabolic panel was remarkable for sodium 118 mEq/L, BUN 182 mg/dL, creatinine 4.05 mg/dL, bicarbonate 8 mg/dL, and calcium 7.3 mg/dL. Her initial lactate was 9.4. Urinalysis showed >186 WBCs and bacteria. Urine drug screen was positive for amphetamines and opiates. The patient was admitted to the medical intensive care unit for septic shock.

Due to respiratory failure and increasing lactic acidosis, the patient was intubated and started on vasopressin for additional blood pressure support. Despite continued resuscitation, arterial pH and bicarbonate declined to 7.21 and 8, respectively, despite bicarbonate infusion. Continuous renal replacement therapy was initiated due to decreased urine output. Leukocytosis peaked on hospital day one at 82,700/mm³, and hemoglobin and platelets declined below 6.1 g/dL and 9000/mm³, respectively. She received 4 units of packed red blood cells and 3 units of platelets. Hematology-Oncology determined that this profound leukocytosis, anemia, and thrombocytopenia was a leukemoid reaction. On contacting the pacemaker manufacturer, the device was placed 3 years prior for polymorphic ventricular tachycardia. Blood cultures revealed Haemophilus parainfluenzae. Transthoracic echocardiogram showed ejection fraction of 55–65% and a 1.5 × 1.3 cm mass. Transesophageal echocardiogram demonstrated 2 mobile masses, one measuring 1 × 1 cm and one 1 × 1.6 cm encased on the right ventricular lead; the ICD was subsequently removed (Fig. 1). Her hospital course was complicated by hospital acquired pneumonia with pleural effusions, requiring bilateral tube thoracostomies. The patient was discharged on hospital day 41.

⁎ Corresponding author.
E-mail address: rachelle.bridwell.mil@mail.mil (R.E. Bridwell).
3. Discussion

IE secondary to *H. parainfluenzae*, typically an oropharyngeal bacterium, represents an uncommon pathogen seen in 1–3% of cases [3]. In cases of right heart IE in intravenous drug users, *H. parainfluenzae* was isolated in combination with *S. aureus* or viridans Streptococci [6]. It is postulated that these 2 g positive bacteria facilitate the growth of *H. parainfluenzae* [7]. The presentation of IE on ICDs or pacemakers can vary widely. In a case series of 33 infected pacemakers, fever was the presenting symptom in 36% of cases, with septic shock occurring in 9% [2]. Of these 33 cases, 30 were due to *Staphylococcus*, highlighting the unusual nature of the above case presentation [2].

Clinical suspicion and positive blood cultures are crucial in the diagnosis; however, ultrasound is the best diagnostic tool in identifying and confirming IE. Transthoracic echocardiography provides a non-invasive view, while transesophageal echocardiography best identifies and measures vegetations in 96% of cases as compared to only 22% of transthoracic echocardiograms [2,8]. Early surgical management has demonstrated significant reduction in mortality in native valves, although prosthetic valves and ICD leads have not shown to have the same benefit from surgical management [9]. Current AHA guidelines recommend ceftriaxone infusion over previously chosen ampicillin as the antibiotic of choice for *H. parainfluenzae* IE [10].

4. Conclusion

The recent increase in intravenous opioid may lead to increasing numbers of patients with complications such as endocarditis. The presence of indwelling lines or other devices warrants consideration of infections involving these devices.

Funding/COI

None.

Disclaimer

The view(s) expressed herein are those of the author(s) and do not reflect the official policy or position of Brooke Army Medical Center, the U.S. Army Medical Department, the U.S. Army Office of the Surgeon General, the Department of the Army, the Department of the Air Force and Department of Defense or the U.S. Government.

Acknowledgements

The view(s) expressed herein are those of the author(s) and do not reflect the official policy or position of Brooke Army Medical Center, the U.S. Army Medical Department, the U.S. Army Office of the Surgeon General, the Department of the Army, the Department of the Air Force and Department of Defense or the U.S. Government. No funding was received for this research.

References


