Candida albicans endocarditis in a patient with an implantable cardioverter defibrillator (ICD)

Endocarditis por candida albicans en paciente portadora de daf

The incidence of infective endocarditis of implantable cardioverter defibrillators has increased in recent years, which is similar to the increase in the occurrence of fungal endocarditis. We here report the case of an ICD carrier female patient who presented an infection of the lead by Candida albicans.

A 65-year-old woman with history of hypertension, hypercholesterolemia, chronic renal failure under hemodialysis treatment and dilated myocardopathy of ischemic origin had an ICD implanted after an episode of cardiac arrest due to ventricular fibrillation in April 2012. In May 2013, she was admitted to the Service of Intensive Care Medicine because of septic shock in relation to a pelvic abscess secondary to perforated diverticulosis disease. Sigmoidectomy and terminal colostomy was performed, and the patient was discharged from the hospital 6 weeks after surgery. A month later, she was readmitted to the intensive care unit with clinical manifestations of septic shock of unknown origin. Three blood cultures were obtained and in all of which C. albicans was isolated. The patient was treated with liposomal amphotericin B (5 mg/kg/day), supportive vasoactive drugs and continuous veno-venous hemofiltration. In transesophageal echocardiography a 23 mm size mass was observed attached to ICD electrode (Figs. 1 and 2). Despite the size of the vegetation and given the high surgical risk and multiorgan involvement of the septic process, early removal of the device was considered adequate and assuming the risk of pulmonary embolism. The patient presented various bacteremic febrile episodes over the course of the following weeks but all blood cultures after removal of the ICD were negative. Given the persistence of fever and elevated sepsis-related laboratory parameters, caspofungin was added to treatment with liposomal amphotericin B (loading dose of 70 mg i.v.; maintenance 50 mg/daily). Repeated echocardiograph studies excluded the presence of endocarditis and/or heart valvular involvement. Bilateral pulmonary septic emboli, which may justify persistent fever, were observed on the thoracic computed tomography (CT) scan. The clinical condition of the patient improved, with hemodynamic stability and decrease of sepsis-related analytical results. Treatment with vasoactive drugs and continuous veno-venous hemofiltration was discontinued. Fours weeks after ICD explantation and after more than 72 h with normal temperature, a new device was reimplanted. The patient received the double antifungal treatment for 6 weeks and was discharged home 10 weeks after the admission.

There are a few studies of fungal endocarditis, particularly in the era of the new antifungal agents, such as the latest generation of azoles or echinocandins. Although staphylococcus are the pathogens most frequently isolated in ICD-related endocarditis (60–80%), fungal endocarditis accounts for 1–6% reaching up to 10% in some clinical series. In carriers of ventricular-assisted devices, Aslam et al. reported that fungi were responsible for up to 21% of infections. Fungal endocarditis is predominantly caused by Candida, especially C. albicans followed by Candida glabrata, Candida parapsilosis and Candida tropicalis.

Risk factors for fungal endocarditis include debilitating diseases, previous use of broad-spectrum antimicrobials, parenteral nutrition and use of intravascular devices, such as catheters, prosthetic heart valves or ICD. Fungal endocarditis is frequently associated with major complications, such as embolism, in relation to the ability to produce large vegetations, and has a high mortality rate (up to 91% in some series).

Some aspects of the management of ICD-associated fungal endocarditis are still controversial, and given the paucity of randomized clinical studies, therapeutic decisions in this scenario are challenging for the clinician.

The antifungal regimen recommended by most of the current clinical guidelines includes the use of liposomal amphotericin B

Fig. 1. Transesophageal echocardiography (TEE) where a mass sized 18 mm is observed in the right atrium (RA).

Fig. 2. Image of ETE with a 17 mm x 23 mm mass attached to de ICD electrode in RA.
Meliodosis importada desde Colombia a España

Meliodosis imported from Colombia to Spain

Sr. Editor:

La meliodosis es una enfermedad tropical causada por Burkholderia pseudomallei. Es endémica, con gran importancia para la salud pública, en el sureste de Asia y el norte de Australia\(^1\). En las últimas décadas la epidemiología ha ido cambiando, con crecientes informes de casos confirmados autóctonos adquiridos en dichas zonas y pequeños brotes y casos esporádicos en áreas de América del Sur, algunas islas del Caribe y algunos países africanos\(^2\). Las manifestaciones clínicas incluyen: la forma bacterémica, con o sin shock séptico, y la forma no bacterémica, incluyendo la neumonía, la infección del tracto genitourinario y las infecciones de piel y tejidos blandos\(^3\). Puede presentarse con afectación del músculo esquelético en el 2-10% de los pacientes. La artritis séptica causada por B. pseudomallei es más común entre los pacientes con enfermedades subyacentes, como diabetes, insuficiencia renal y cirrosis\(^4\).

Presentamos el caso de un varón de 28 años de edad, nacido en España, sin antecedentes patológicos ni factores de riesgo de interés, que fue atendido en nuestro hospital en agosto del 2005, con historia de 7 días de fiebre de hasta 40 °C, malestar general, dolor de garganta y linfadenopatía submandibular dolorosa. Los síntomas se iniciaron 2 semanas después de regresar de un viaje turístico de 4 semanas a través de la selva en Colombia. El paciente fue tratado inicialmente con azitromicina, 500 mg/24 h vía oral, durante 3 días. Al no mostrar mejoría, regresa al hospital, requiriendo ingreso. Durante su estancia en el hospital desarrolló un intenso dolor e hinchazón del tobillo derecho. Las pruebas de laboratorio revelaron un recuento de leucocitos de 11.300/mm\(^3\), una proteína C reactiva de 14.50 mg/dl, una VSG de 71 mm/h, y serología de VIH, VHB y VHC negativas. Las pruebas de función hepática resultaron alteradas (AST,

---


\(^{7}\) Itziar Eguibar-Villimar\(^{\text{a}}\), Jose Manuel Porres-Aracama\(^{\text{b}}\), Begoña Azcarate-Ayerd\(^{\text{c}}\), Ruth Salaberri Udabe\(^{\text{a}}\)

\(^{\text{a}}\) Department of Medical Intensive Care, Hu Donostia, San Sebastian 20080, Spain

\(^{\text{b}}\) Department of Medical Intensive Care and Arritmia Unit, Hu Donostia, San Sebastian 20080, Spain

\(^{\text{c}}\) Department of Medical Intensive Care, Hu Donostia, San Sebastian 20080, Spain

---

E-mail address: xixi-@hotmail.com (I. Eguibar-Villimar).

http://dx.doi.org/10.1016/j.aimc.2014.06.004