tive. The MGIT culture was positive 12 days after incubation, confirming the presence of acid-alcohol fast bacilli by Zhiel-Nielssen staining. The LJ medium culture could not be interpreted because it was contaminated. Identification by MALDI-TOF MS (Bruker Daltonics Inc., Germany) with a score of 1909 was *M. heckeshornense*. The strain was sent to the National Micobacteria Centre (Instituto Carlos III [Carlos III Institute], Majadahonda, Madrid) to confirm its identification by molecular methods and to carry out antibiotic susceptibility studies using the proportion method.

Identification was carried out through PRA of the *hsp*65 gene and digestion with BstEII and HaeIII enzymes. The pattern that was obtained was 3 bands 235/120/100 after digestion with the first enzyme and 3 other bands after digestion with HaeIII 160/105/60. This presumptive identification was confirmed by 16S rRNA gene sequencing. The strain with which 100% homology was presented in the GenBank was with the *M. heckeshornense* strain S369 (NR_028759), comparing 1360 pb. The antibiotic susceptibility test was carried out using MIC in solid medium; the strain was sensitive to cycloserine, ethionamide, rifampin, capreomycin, streptomycin and kanamycin, and resistant to ethambutol, isoniazid, PAS, pyrazinamide, TCH, and thiosemicarbazone.

Isolation of non-tuberculous mycobacteria in synovial fluid is unusual, preferentially occurring in immunosuppressed patients. In our case, as in the work of Yokohama et al.,[5] identification by MALDI-TOF was conducted, which is why it can be considered a quick, cost-effective and accurate tool for the identification of *M. heckeshornense*. We presented a fifth case of osteoarticular involvement in addition to a case of tenosynovitis[2] and 3 of spondylodiscitis.[2,3] Males (4 cases) and the existence of any risk factor (2 HIV infections and 2 immunosuppressive treatment) predominated, and all achieved a cure. The association of biological drugs used in rheumatoid arthritis and the increased risk of serious infections provide controversial evidence. According to a meta-analysis published in *The Lancet*,[6] biological medicines showed a significant increase in the risk of serious infections at usual doses compared to disease-modifying antirheumatic drugs (DMAD), with the combination of biological drugs presenting the highest risk.

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Parvimonas micra infective endocarditis

Endocarditis infecciosa por Parvimonas micra

Parvimonas micra, formerly *Peptostreptococcus micros* and *Micromonas micros,* are anaerobic, gram-positive cocci that belong to the abdominal, oropharyngeal and genitourinary flora, commonly related with periodontal diseases.[1] Its pathogenicity has been also documented in disseminated diseases such as septic arthritis, spondylodiscitis or abscesses in different organs.[2–4] In 2015 the first case of endocarditis was described, in a 71-years-old male patient with valve abscess.[1] A literature review showed two cases of infectious endocarditis due to *P. micra*[1,5] and three cases related to their ancestor *P. micros*.[6–8]

We present a case of infectious endocarditis in a woman with a pacemaker and prosthetic mitral valve.

A 63-year-old female presented at the emergency department with a history of one month of persistent fever at dawn and noon, with chills, diaphoresis, asthenia and weight loss (5 kg). She denied dental procedures, recent interventions or invasive diagnostic techniques. Oral evaluation was made with no signs of periodontal disease other than edentulism. There were no respiratory, gastrointestinal, musculoskeletal or urinary symptoms and no cutaneous manifestation were found.

She had frequent follow-ups with neurosurgery and neurology for a right cerebral subependymoma and a mid-cerebral artery cardioembolic ictus performed eleven months prior to this episode. She had had a mechanical prosthetic mitral valve replacement in 1990. She had frequent checkups with urology for kidney angiomyolipomas.

On examination, temperature was normal, blood pressure was 130/62 mmHg, pulse of 70 beats per minute, respiratory rate of 18 breaths per minute, and oxygen saturation 100%. Auscultation was clear with no remarkable finding except for a click in the mitral valve. No lymphadenopathies were noted, as well as no edemas, with low muscle mass. The remainder examination was normal.

The patient was hospitalized to study a fever of unknown origin, and an echocardiography and positron emission tomography/computed tomography (PET/CT) were done. The PET/CT showed metabolic signs of infection in mitral prosthetic and

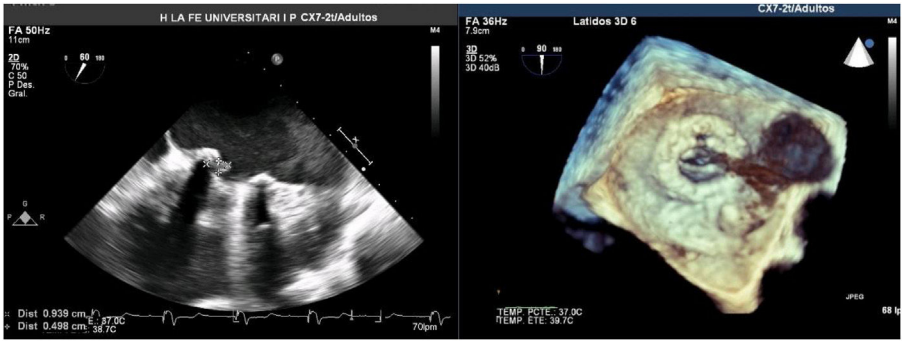

Fig. 1. Transesophageal echocardiography, shows vegetation on the medial side of the prosthetic ring of about 8 mm.

periprosthetic tissue with moderate increased tracer uptake at the right carotid artery, aortic arch, thoracic aorta and pulmonary arteries compatible with active vasculitis on the large vessels. No pacemaker or extracardiac deposites were found.

A Transesophageal echocardiography (TEE) showed a vegetation on the medial side of the prosthetic ring of about 8 mm (Fig. 1). Cardiac and aortic CT discarded the possibility of valvular abscess or aortitis.

During the first 5 days of hospitalization 3 blood cultures were taken and incubated in BACT/ALERT® VIRTUO® (bioMérieux). After 43.94 h of average incubation (44.88 h – 42.82 h – 44.13 h) in the anaerobic media growth of gram-positive cocci identified by MALDI-TOF® as *P. micra*. The antibiotic susceptibility was done using Epsilon test in Columbia blood agar supplemented with 5% lamb defibrinated blood, in anaerobic atmosphere, obtaining the following MIC (mg/L) profile: penicillin 0.016, ampicillin 0.032, amoxicillin-clavulanic 0.047, ceftriaxone 0.125, imipenem 0.012, clindamycin 0.094, vancomycin 0.38, levofloxacin 0.19, rifampicin 0.002.

Empiric treatment was started with ceftriaxone and gentamycin and later modified to penicillin and clindamycin after culture isolation. The first negative blood culture was 4 days after initial treatment. TEE showed vegetation stability without complications, so it was decided to manage the case conservatively, with outpatient orally treatment with ceftriaxone and rifampicin for 6 more weeks. The evolution was favorable with negative control of blood cultures up to one year after the episode. After a year follow up by the Rheumatology Department the conclusion was that, findings on the PET/CT scan were due to the inflammatory response to the infection, which diminished on subsequent scans.

Endocarditis is mainly caused by species of the genus *Streptococcus* and *Staphylococcus*, comprising around two-thirds of the total of infectious endocarditis, while anaerobic organisms are only isolated in 2–16% of the cases.[9] Predominant anaerobic species are *Cutibacterium acnes*, *Bacteroides fragilis* and *Clostridium* spp.[10] Endocarditis due to *Peptostreptococcus* spp. are infrequent[10] with *P. magnus* being the most common species.[9] If we focus on *P. micra*, until now five cases have been described in the literature,[1,5–8] only two of them encompassed within this terminology.[1,5]

Conflict of interest

None.

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