

### Septic Arthritis Due to *Enterococcus faecalis* in a Patient With a Tunnelized Hemodialysis Catheter

**To the Editor:** *Enterococcus faecalis* spp is one of the main causes of bacteremia, endocarditis, and nosocomial infection; nevertheless, very few cases of infectious arthritis by *Enterococcus* spp have been described. It can be due to its low affinity for osteoarticular tissue or to the fact that cases described before 1984 were classified like septic arthritis due to group D *Streptococcus*. We present the case of a 78 year-old man with chronic renal insufficiency in hemodialysis with a tunnelized (TC) ShonCath™ type (AngioDynamics®) catheter located in the jugular right, a definitive bicamera pacemaker for a complete atrioventricular block, and a aorto-aortic bypass for an abdominal aorta aneurism. Three months before his hospitalization he underwent a colonoscopy in which a polyp was extirpated and later anatomopathologically classified as an infiltrating adenocarcinoma. Nine days later he presented fever. In the peripheral blood cultures and the cultures of the TC that were obtained we isolated *Enterococcus faecalis* sensible to ampicilline and vancomicine, with sinergy for aminoglicosides (time growth differential, 200 min). He was diagnosed with bacteremia related to TC (BRCHD) and received intravenous treatment with vancomicine, 1.5 g in a single dose, amoxicilline 1 g/12 h for 21 days, and gentamicine 80 mg/24 h for 11 days, and the CT was “sealed” between dialysis with 2 mg of vancomicine. He was feverless after 48 hours and the posttherapeutic blood culture was negative. Three months later he was hospitalized again for an inflammation of the right knee, and the examination found pain, swelling, an increase in temperature, functional incapacity and limitation upon mobilization in all planes. In the blood chemistry we found: urea, 135 mg/dL; creatinine, 6.7 mg/dL, and CRP, 139 mg/L; in hemogram, 8670 leucocytes/ $\mu$ L (71% polymorphonuclear); hemoglobin, 10.9 g/dL, and ESR, 56 mm/h. The synovial fluid showed 52 356 cells/ $\mu$ L (95% polymorphonuclear); proteins, 96 mg/dL, and glucose, 10 mg/dL; Gram’s stain was negative and in the culture *E. faecalis*, with an identical antibiotype to the one identified in the BRCHD, was found. The peripheral blood and CT cultures obtained as well as the urine culture were negative.

A heart ultrasound was done, which discarded endocarditis and infection of the pacemaker, and an abdominal echography where intraabdominal septic centers were not visualized. We decided to conserve the CT and to undertake intravenous treatment with ampicilline 6 g/day and ceftriaxone 2 g/day for 4 weeks, with which the cultures

of synovial fluid were negative after 48 h. At discharge he continued feverless and without joint manifestations. *E. faecalis* causes a 2.4%–8% of BRCHD. Philipneri et al<sup>1</sup> emphasizes the importance of retrieving the central catheter before an episode of bacteriemia. They observed a clear relation between maintaining the infected catheter and the appearance of osteoarticular complications in spite of the correct antibiotic treatment, which can appear up to 5 weeks after initiating the treatment. Nevertheless, the lack of vascular access, as happened in our patient, forced us to try and “save” the TC by means of systemic and “sealed” treatment of the TC with a high antibiotic concentration in the period between dialysis.<sup>2</sup> In our case, the isolation of *E. faecalis* with the same antibiotype in blood and synovial fluid indicate that the origin of arthritis was the BRCHD, although the blood cultures after he treatment confirmed their eradication. The microorganism can nest in the knee during the acute phase and “increase” after suspending antibiotic treatment. *Enterococcus* spp offers intrinsic resistance to cephalosporines and is moderately sensible to penicillins, carboxipenicillins, ureidopenicillins, and carbapenems. For that reason, we used a synergic combination of penicillin and aminoglicoside<sup>3,4</sup> for treatment. Gavalda et al<sup>5,6</sup> has demonstrated, in vitro and in vivo, that the combination of ampicillin and ceftriaxone is synergic and of equal effectiveness than ampicillin and gentamicine in a humanized experimental model of endocarditis. Our case demonstrates that this guideline can constitute an alternative to the classic guideline in patients with renal insufficiency or intolerance to aminoglicosides.

Julio Ramírez García,<sup>a</sup> Mercedes Toro Ramos,<sup>b</sup>  
Rafael Luque Márquez,<sup>c</sup> and Aristides de Alarcón González<sup>c</sup>

<sup>a</sup>Servicio de Reumatología, Hospital Universitario Virgen del Rocío, Sevilla, Spain

<sup>b</sup>Servicio de Nefrología, Hospital Universitario Virgen del Rocío, Sevilla, Spain

<sup>c</sup>Servicio de Enfermedades Infecciosas, Hospital Universitario Virgen del Rocío, Sevilla, Spain

### References

1. Philipneri M, Al-Aly Z, Amin K, Gellens ME, Bastani B. Routine replacement of tunneled, cuffed, hemodialysis catheters eliminates paraspinal/vertebral infections in patients with catheter-associated bacteremia. *Am J Nephrol*. 2003;23:202-7.
2. Saxena AK, Panhotra BR. Haemodialysis catheter-related bloodstream infections: current treatment options and strategies for prevention. *Swiss Med Wkly*. 2005;135:127-38.
3. Zwillich SH, Hamory BH, Walker SE. Enterococcus: an unusual cause of septic arthritis. *Arthritis Rheum*. 1984;27:591-5.
4. Raymond NJ, Henry J, Workowski KA. Enterococcal arthritis case report and review. *Clin Infect Dis*. 1995;21:516-22.
5. Gavalda J, Torres C, Tenorio C, López P, Zaragoza M, Capdevila JA, et al. Efficacy of ampicillin plus ceftriaxone in treatment of experimental endocarditis due to *Enterococcus faecalis* strains highly resistant to aminoglicosides. *Antimicrob Agents Chemother*. 1999;43:639-46.
6. Gavalda J, Onrubia PL, Gómez MT, Gomis X, Ramírez JL, Len O, et al. Efficacy of ampicillin combined with ceftriaxone and gentamicin in the treatment of experimental endocarditis due to *Enterococcus faecalis* with no high-level resistance to aminoglicosides. *J Antimicrob Chemother*. 2003;52:514-7.