debridement and, frequently, the prosthetic exchange in 1 or 2 sessions; this last one with better results.[19,21] In patients with an unfavorable progression and without the possibility of a prosthesis exchange, arthrodesis is an option, which, in spite of its bad functional results, is followed by a high rate of bacteriological resolution. In relapsing cases, which threaten the life of a patient, dearticulation or amputation are the only alternative.[1,12,19]

We have read about only one other patient with microcrystalline and infectious arthritis on a patient with a joint prosthesis.[11] It presented in a 46-year-old male with a knee prosthesis infected by *S aureus* which was resistant to oxacyllin and who required an arthrodesis. We consider that, because prosthesis are more susceptible to infectious complications than native joints,[5,12] this possibility must be the first one to be investigated when faced with pain and inflammatory signs, even in patients with a history of microcrystalline arthritis.

Norberto Gómez Rodríguez, Jesús Ibáñez Ruán,
and Marisol González Pérez

Servicio de Reumatología, Hospital Povisa, Vigo, Pontevedra, Spain

References

1. Infection in total joint replacement. En: Norden C, Guillespie W J, Nade S, editores. Infections in bones and joints. Boston: Blackwell Scientific Publications; 1994. p. 291-319.
2. Steckelberg JM, Osmon DR. Prosthetic joint infections. En: Bisno A, Waldvogel FA, editores. Infections associated wtih indwelling medical devices. 2.a ed. Washington: American Society for Microbiology; 1994. p. 259-90.
3. Bengtson S, Knutson K. The infected knee arthroplasty. Acta Orthop Scand. 1991;62:301-11.
4. Poss R, Thornhill TS, Ewald FC, Thomas WH, Batte NJ, Sledge CB. Factors influencing the incidence and outcome of infection following total joint arthroplasty. Clin Orthop. 1984;182:117-26.
5. Kaandorp CJE, Dinant HJ, van der Laar MAFJ, Moens HJB, Prins APA, Dijkmans BAC. Incidence and sources of native and prosthetic joint infection: a community based prospective survey. Ann Rheum Dis. 1997; 56:470-5.
6. Batlle E, Tovar JV, Pascual E, Salas E. Artritis séptica con microcristales: una causa de error diagnóstico. Med Clin (Barc). 1987;89:281-4.
7. Baer PA, Tenenbaum J, Fam AG, Little H. Coexistent septic arthritis and crystal arthritis. Report of four cases and literature review. J Rheumatol. 1986;13:604-7.
8. Pérez D, Olivé A, Tena X. Coexistencia de artritis infecciosa y artritis microcristalina. An Med Intern (Madrid). 2000;17:329-30.
9. Gómez N, Ibáñez J, González M. Artritis séptica por *Escherichia coli* en una mujer con artropatía gotosa. An Med Intern (Madrid). 2005;22:349-50.
10. Gómez N, Rodríguez E, Ferreiro JL, Vilar M. Artritis séptica por *Staphylococcus epidermidis* en paciente con insuficiencia cardíaca y gota. Med Clin (Barc). 1994;102:317-8.
11. Yu KH, Luo SF, Liou LB, Wu YJJ, Tsai WP, Chen JY, Ho HH. Concomitant septic and gouty arthritis – an analysis of 30 cases. Rheumatology. 2003;42:1062-6.
12. Muñoz P, Bouza E. Infecciones causadas por estafilococos resistentes a β-lactámicos sobre prótesis articulares: diagnóstico y tratamiento. Rev Clin Esp. 1997;197:38-47.
13. Dubost JJ, Soubrier M, De Champs C, Ristori JM, Bussière JL, Sauvezie B. No changes in the distribution of organisms responsible for septic arth-ritis over a 20 year period. Ann Rheum Dis. 2002;61:267-9.
14. Bouza E, Muñoz P. Micro-organisms responsible for osteo-articular infections. Baillieres Best Pract Res Clin Rheumatol. 1999;13:21-5.
15. Meghji S, Crean SJ, Nair S, Wilson M, Poole S, Harris M, et al. *Staphylococcus epidermidis* produces a cell-associated proteinaceous fraction which causes bone resorption by a prostanoid-independient mechanism: relevance to the treatment of infected orthopaedic implants. Br J Rheumatol. 1997;36:957-63.
16. von Eiff C, Peters G, Heilmann C. Pathogenesis of infections due to coagulase-negative staphylococci. Lancet Infect Dis. 2002;2:677-85.
17. Berbari EF, Hanssen AD, Duffy MC, Steckelberg JM, Ilstrup DM, Harmsen WS, et al. Risk factors for prosthetic joint infection: case-control study. Clin Infect Dis. 1998;27:1247-54.
18. Shirtliff ME, Mader JT. Acute septic arthritis. Clin Microbiol Rev. 2002;15:527-44.
19. Rodríguez-Merchan EC. Artrosis de rodilla: prótesis totales. In: Rodríguez-Merchan EC, editor. Artrosis. Madrid: Medical & Marketing Communications; 2002. p. 137-64.
20. Rosseau I, Cardinal EE, Raymond-Tremblay D, Beauregard CG, Braunstein EM, Saint-Pierre A. Gout: radiographic findings mimicking infection. Skeletal Radiol. 2001;30:565-9.
21. Boxen I. Inability of bone and gallium imaging to differentiate acute gout arthritis from septic arthritis. Clin Nucl Med. 1986;11:443-4.
22. Blackburn WD, Alarcon GS. Prosthetic joints: a role for prophylaxis. A review. Arthritis Rheum. 1991;24:110-7.
23. Flores X. Cirugía de rescate en implantes articulares. In: Herrera A, Ferrández L, Herrero-Beaumont G, Rodríguez de la Serna A, editors. La rodilla. Tomo II. Monografías médico-quirúrgicas del aparato locomotor. Barcelona: Masson; 2001. p. 75-104.

Septic Bursitis Due to *Eggerthella lenta*

To the Editor: Septic bursitis fundamentally affects superficial bursae (olecranon and prepatellar). Usually it is due to microorganisms that penetrate through trauma because both localizations are exposed to it, be it by accidental puncture or, less frequently, due to intrabursal infiltration with steroids. More than 80% of cases of septic bursitis are produced by *Staphylococcus aureus* and the rest by *Streptococcus* spp, gramnegative bacteria, micobacteria and fungi, and anaerobes are infrequently isolated. On the contrary, cases of arthritis due to anaerobes have been described.[1-4]

We present the case of olecranon bursitis due to *Eggerthella lenta* in a 70-year-old male with a history of stroke, hypertension, hyperuricemia, mild chronic renal failure due to nephroangiosclerosis, ischemic heart disease, bilateral carotid stenosis, treatment resistant rheumatoid arthritis, and receiving steroids and etanercept and chronic olecranon bursitis of a mechanical origin. The patient presented rheumatoid nodules on both olecranon bursae for which he had been operated years before. He came to the clinic due to right olecranon swelling, without any fever or chills. A bursocentesis was performed and an inflammatory synovial fluid was obtained, without any evidence of crystals under polarized light microscopy. The samples were sent for culture in blood culture media (Bact-Alert, Organon-Technika), resulting negative after 5 days

Figure 1. *Anteroposterior x-ray of the elbow.*

Figure 2. *Lateral x-ray of the elbow*

of incubation. Four days later the patient he returned to the clinic due to worsening and, upon examination, he was found to have an increase in local temperature, erythema and pain when palpated. His blood pressure was 124/84; temperature, 36.4°C. His blood analysis showed 9960 leukocytes/μL, 76.4% neutrophils, 15% lymphocytes; erythrocyte sedimentation rate, 19 mm in the first hour; C-reactive protein, 0.53 mg/dL. The right elbow x-ray there was an important increase of the soft-tissue of the posteromedial zone (Figures 1 and 2), with radiolucid images in its interior. The bursocentesis was repeated showing a macroscopic purulent aspect and was sent to the laboratory of microbiology for culture in a sterile syringe and with blood culture media specific for both aerobes and anaerobes. Gram staining showed abundant polymorphonuclear leukocytes and intracellular grampositive bacteria. Treatment was started with amoxacyllin-clavulanate 1 g/8 h iv. After 48 hours the anaerobe culture was positive and subcultures in blood agar and chocolate agar (5% CO_2), were done as well as in Sabouraud agar and Schaedler agar (anaerobic media); in this last plate there was a growth of grampositive bacilli, identified through a Api 20 A gallery (BioMérieux) as *Eubacterium lentum* (currently, *E lenta*).[5] Sensitivity to antibiotics were carried out using E-test strips (AB-Biodisk, Solna, Sweden) in *Brucella* agar, and turned out sensitive to penicillin G, amoxicillyn-clavulanate, cephoxitine, imipenem, clindamycin, and metronidazole. Daily extraction of synovial fluid was carried out which were positive for the first three days of hospitalization and then were negative.

*E lenta*es is a grampositive bacilli (Figure 3), a strict anaerobe, non-sporulating and catalase negative which forms a part of the normal flora of the mouth and the gastrointestinal tract in humans. It is isolated rather frequently in abscesses and wounds and is rarely isolated in blood cultures.[6] It has been considered as a pathogen in infections associated to intrauterine devices, bacteremias in patients with neoplasia, female genitourinary tract infections and abdominal infections.[7,8] Bursitis due to *E lenta* can occur as the consequence of an asymptomatic bacteremia with its origin in the intestinal tract or mouth, or as external contamination due to a procedure of bursal manipulation. The patient did not refer recent dental manipulation and no important lesions were found. Bursae manipulation was always carried out with the utmost care regarding asepsia. One month and a half after his discharge a barium enema was performed to rule out an associated colon affection and, although no signs of diverticulitis were seen in that moment, there were some diverticulae on the sigmoid and descending colon. Due tpo these findings and the nature of the causal agent, it is probable that the origin of the bursitis were a transient bacteremia of an intestinal source.

No cases of bursitis due to *E lenta* were found when we

Figure 3. *Gram stain: grampositive bacilli (×1000)*

reviewed the literature and, although anaerobes are not usually isolated in septic bursitis, it seems advisable to process synovial fluids for both aerobe and anaerobe cultures, and to carry out serial blood cultures even in the absence of fever, especially in a patient that, as this case illustrates, has some sort of immunocompromise and is the subject of frequent manipulation due to chronic procedures. In addition, in immunocompromised patients it is important to rule out infections due germs that are infrequently isolated

Fernanda Pardo Sánchez,[a] Patricia Alejandra Romero Jung,[a] Lucía Martínez Lamas,[a] and Beatriz Arca Barca[b]

[a]Servicio de Microbiología, Complejo Hospitalario Universitario Santiago de Compostela, Santiago de Compostela, A Coruña, Spain
[b]Servicio de Reumatología, Complejo Hospitalario Universitario Santiago de Compostela, Santiago de Compostela, A Coruña, Spain

References

1. Brook I. Joint and bone infections due to anaerobic bacteria in children. Pediatr Rehabil. 2002;5:11-9.
2. Sonsale PD, Philipson MR, Bowskill J. Septic arthritis of the knee due to *Fusobacterium necrophorum*. J Clin Microbiol. 2004;4:3369-70.
3. Roche M, Smyth E. A case of septic arthritis due to infection with *Gemella morbillorum*. J Infect. 2005;51:187-9.
4. Ohl CA. Artritis infecciosa de las articulaciones nativas. In: Mandell GL, Bennett JE, Dolin R, editors. Enfermedades infecciosas: principios y práctica. Madrid: Elsevier Churchill Livingstone; 2005. p. 1311-22.
5. Kageyama A, Benno Y, Nakase T. Phylogenetic evidence for the transfer of *Eubacterium lentum* to the genus *Eggerthella* as *Eggerthella lenta* gen. nov., comb. nov. International Journal of Systematic Bacteriology. 1999;49:1725-32.
6. Rodloff AC, Hillier SL, Moncla BJ. Peptostreptococcus, Propionibacterium, Lactobacillus, Actinomyces, and other non-spore-forming anaerobic Gram-positive bacteria. In: Murray PR, Baron EJ, Pfaller MA, Tenover FC, Yolken RH, editors. Manual of clinical microbiology. 7.a ed. Washington: ASM Press; 1999. p. 672-89.
7. Mascini EM, Verhoef J. Bacilos anaerobios grampositivos no esporulantes. In: Mandell GL, Bennett JE, Dolin R, editors. Enfermedades infecciosas: principios y práctica. Madrid: Elsevier Churchill Livingstone; 2005. p. 2849-52.
8. Lattuada E, Zorzi A, Lanzafame M, Antolini D, Fontana R, Vento S, et al. Cutaneous absceso due to *Eubacterium lentum* in infection drug user: a case report and review of the literature. Journal of Infection. 2005;51:e71-2.