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Letters to the Editor

Septic Arthritis Caused by *Sphingomonas paucimobilis* in an Immunocompetent Patient☆

Artritis séptica por Sphingomonas paucimobilis *en un paciente inmunocompetente*

To the Editor,

Septic arthritis is an inflammatory joint disease produced by invasion and multiplication of pathogenic microorganisms. Most cases of septic arthritis are caused by microorganisms of the genus Staphylococcus and Streptococcus. Non-fermenting Gram-negative bacilli (Pseudomonas, Stenotrophomonas, Acinetobacter, and Burkholderia) are a frequent causes of nosocomial infection associated with immunosuppression situations, and *Sphingomonas paucimobilis* (*S. paucimobilis*) (formerly *Pseudomonas paucimobilis*) is also a Gram-negative aerobic fermentor that now emerges as an opportunistic pathogen.

We present a case of septic arthritis by *S. paucimobilis* in a 41-year-old man with a history of frequent episodes of hyperuricemia and gout in his left knee. In the past year, he presented several episodes of acute monoarthritis treated with nonsteroidal anti-inflammatory drugs, colchicine, and intraarticular infiltration of triamcinolone. The patient presented with symptoms of pain and swelling of left knee which had lasted for 3 months without fever. Physical examination showed: temperature 36.5 °C and left knee arthritis with preserved but painful active and passive mobility. Arthrocentesis was performed, resulting in inflammatory synovial fluid, without evidence microcrystals under the polarized light microscope. The fluid was sent to the microbiology department in a sterile syringe and blood culture bottles for aerobic and anaerobic culture.

Gram stain showed abundant polymorphonuclear leukocytes and intracellular Gram-negative bacilli, and the culture-negative bacilli isolated were identified as *S. paucimobilis* by ID32GN Api (bioMerieux, Marcy L'etoile 3. France), sensitive to beta-lactams, aminoglycosides, quinolones, and cotrimoxazole. Laboratory analysis upon the patient's admission showed 8070 leukocytes/L (70% neutrophils and 30% lymphocytes), erythrocyte sedimentation rate of 42 mm the first hour and CRP 8.04 mg/dl. CBC, coagulation, and biochemistry were normal. Echocardiogram was normal and X-rays showed a slight increase in soft tissue suprarrotulian density, indicative of effusion; the CT with intravenous contrast observed loosening of articular recesses, with slight enhancement of the synovium.

Daily articular drainage was performed and the patient was treated with ceftazidime (6 g/24 h) plus gentamicin (240 mg/24 h), with clinical improvement, so gentamicin was discontinued after 8 days of treatment, continuing only with ceftazidime. After 15 days and with several negative cultures we reisolated *S. paucimobilis*, so treatment was changed to meropenem and ceftazidime (4 g/24 h/7 days) to complete 21 days of intravenous antibiotic therapy in total. The patient underwent surgical treatment consisting of joint lavage and synovectomy. After 22 days of treatment, it was substituted to oral therapy with ciprofloxacin (1.5 g/24 h) plus trimethoprim-sulfamethoxazole (800/160 mg/12 h) for 3 months.

S. paucimobilis is isolated in nature from soil and water,[1] and in hospital settings from distilled water equipment, dialysis fluids, nebulizers, and other instruments used for respiratory therapy.[2–4] It can cause a variety of nosocomial infections such as community acquired pneumonia, bacteremia, catheter-related infection, osteomyelitis, septic arthritis, meningitis, peritonitis, postoperative endophthalmitis, pleural empyema, and infections of the urinary tract, and bile ducts. These infections are manifested mainly in patients with some form of immunosuppression, patients undergoing an invasive procedure or patients with peritoneal[5–7] dialysis catheters. The microorganism has not demonstrated a high degree of virulence and no cases of death from infection due to *S. paucimobilis* are reported. Its low pathogenicity is due to the lack of lipopolysaccharide in the outer membrane of the Gram-negative cell wall, which is associated with endotoxic activity, and this would explain the good condition of the patient despite successive positive cultures and the prognosis of most cases of infection described by *S. paucimobilis*.[8] There was no knowing what was the origin of the infection by *S. paucimobilis* was. It is possible that the patient was colonized and transient bacteremia allowed the organism to reach the joint, or that the patient became infected due to an improper procedure (direct inoculation) in one of the infiltrations to which he was subjected.

Particularly striking is the extreme difficulty in eradicating *S. paucimobilis*, despite antibiotic therapy and adequate daily articular drainage. Although there are only two cases of arthritis described by *S. paucimobilis*, both in immunocompromized patients,[9,10] it seems necessary to monitor infections caused by opportunistic pathogens in immunocompetent patients, if only because of the high hospital cost involved.

References

1. Smalley DL, Hansen VR, Baselski VS. Susceptibility of *Pseudomonas paucimobilis* to 24 antimicrobial agents. Antimicrob Agents Chemother. 1983;23:161–2.
2. Ryan MP, Adley CC. *Sphingomonas paucimobilis:* a persistent Gram-negative nosocomial infectious organism. J Hosp Infect. 2010;75:153–7.
3. Lin JN, Lai CH, Chen YH, Lin HL, Huang CK, Chen WF, et al. Sphingomonas paucimobilis bacteremia in humans: 16 case reports and aliterature review. J Microbiol Immunol Infect. 2010;43:35–42.
4. Toh HS, Tay HT, Kuar WK, Weng TC, Tang HJ, Tan CK. Risk factors associated with Sphingomonas paucimobilis infection. J Microbiol Immunol Infect. 2011;44:289–95.
5. Calubiran OV, Schoch PE, Cunha BA. *Pseudomonas paucimobilis* bacteraemia associated with haemodialysis. J Hosp Infect. 1990;15:383–8.

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6. Morrison Jr AJ, Shulman JA. Community-acquired bloodstream infection caused by *Pseudomonas paucimobilis:* case report and review of the literature. J Clin Microbiol. 1986;24:853–5.
7. Reina J, Bassa A, Llompart I, Portela D, Borrell N. Infections with *Pseudomonas paucimobilis:* report of four cases and review. Rev Infect Dis. 1991;13:1072–6.
8. Kawasaki S, Moriguchi R, Sekiya K, Nakai T, Ono E, Kume K, et al. The cell envelope structure of the lipopolysaccharide-lacking Gram-negative bacterium *Sphingomonas paucimobilis*. J Bacteriol. 1994;176:284–90.
9. Kuo IC, Lu PL, Lin WR, Lin CY, Chang YW, Chen TC, et al. *Sphingomonas paucimobilis* bacteraemia and septic arthritis in a diabetic patient presenting with septic pulmonary emboli. J Med Microbiol. 2009;58:1259–63.
10. Charity RM, Foukas AF. Osteomyelitis and secondary septic arthritis caused by *Sphingomonas paucimobilis*. Infection. 2005;33:93–5.

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Resolution of Refractory Uveitis, Switching Anti-TNF Treatment☆

Resolución de uveítis refractaria, cambiando el tratamiento anti-TNF

Mr. Editor,

TNF-α plays a role in the induction and maintenance of inflammation in autoimmune disease, hence the TNF-α inhibitors are used successfully in the control of certain systemic diseases or autoinmunitarias.[1]

Uveitis is an intraocular inflammation-associated to autoimmune systemic diseases, in which effectively blocking TNF-α[2] constitutes one of the most important advances in recent years in the treatment of non-infectious uveitis.

The different anti-TNF-α agents do not have the same efficacy on ocular[2] inflammation and 3 major questions remain to be resolved

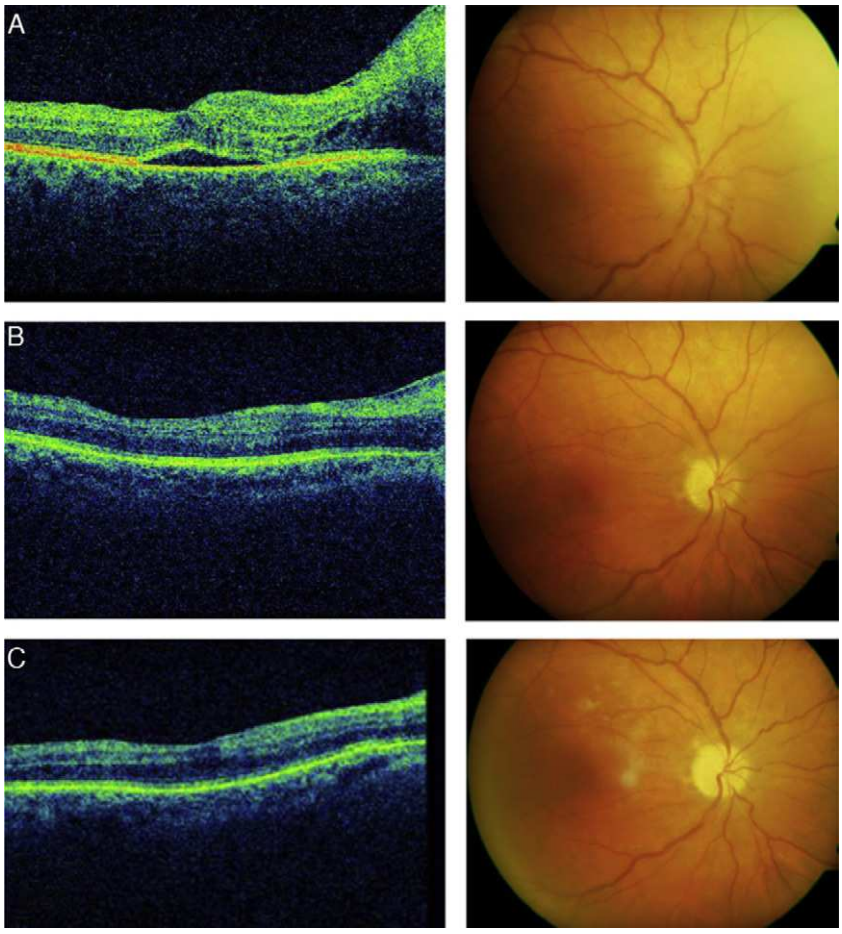

Fig. 1. Optical coherence (left) and retinal tomography (right) of the patient's right eye at 7 months after treatment with infliximab (A) and 2 (B) and 4 (C) months after switching anti-TNF -α treatment to adalimumab.

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