



Letter to the Editor

Septic arthritis due to *Escherichia coli* in a patient with multiple myeloma and left knee pain

Artritis séptica por *Escherichia coli* en paciente con mieloma múltiple y dolor en la rodilla izquierda

To the Editor:

Multiple myeloma (MM) is a malignant monoclonal gammopathy that leads to an altered immune state, which together with other basic treatments needed to control it, causes greater susceptibility to infections. Gram positive infections are more frequent in these patients, but we know that the most common are those produced by Gram negative bacteria affecting the urinary tract once MM background treatment has begun. Arthritis due to *Escherichia coli* (*E. coli*) and other enterobacteriaceae generally represent less than 10% of adult bacterial arthritis and are more frequent in the elderly and immunosuppressed.^{1,2}

The reason for writing this letter is to describe the case of an MM patient who developed septic arthritis in her knee together with lumbar discitis after a urinary tract infection. It concerns an 81-year-old woman with a history of obstructive uropathy due to nephrolithiasis who had to be admitted to a Spanish hospital in August 2008 and had a pig-tail catheter fitted. At the time of her admission, she presented an asymptomatic bacteriuria that was treated with oral cefixime. A month later she was diagnosed with lambda light chain MM at a Durie-Salmon stage IIIB, International Score System 3, and treatment with monthly dexamethasone pulses and weekly erythropoietin support was started. In January 2009 she came to our centre's Accident and Emergency department due to a pain in her left knee that had evolved over the previous 72 hours without any previous trauma, high temperature or any other symptoms. The examination on the left knee (Figure 1) showed signs of chronic venous failure, a great increase in volume, patellar shock, joint limitation and pain on moving, with pulses present. In the analyses: leukocytes $17.40 \times 10^9/L$ (N 16.300; L 420); CRP 44.79 mg/L; D3-dimer: 179; IgG 593.00; IgA 45; IgM 20 mg/dL; with ANA, ANCA and FR normal. HBV, HCV and HIV were negative. Blood cultures were taken. The knee x-ray (Figure 2) ruled out any fractures and identified an unspecific increase in periosteal enhancement in the left medial tibial plateau, no evidence of sclerosis, and an increase in periarticular soft tissue density. The Doppler ultrasound ruled out deep vein thrombosis. We carried out a diagnostic and therapeutic arthrocentesis and fitted a drainage tube, from which we extracted cloudy viscous joint fluid with increased leukocytes (95% polymorphonuclear), high protein and low glucose. The Gram showed gram-negative bacilli, which is why empiric treatment ceftriaxone³ was started and dexamethasone pulses were discontinued. The symptoms were dealt with; there was a reduction in the increase of radiological periarticular density and CRP went back to normal after four weeks of treatment. Final characterisation

revealed that the microorganism was *E. coli*. A week later the pain returned, together with an increase in volume on the same knee with a rise in the level of acute phase reactants; *E. coli* was once again isolated in the joint fluid. She also presented a fever, bilateral lumbar pain and positive spinal percussion (L2-L3).⁴ The lumbar MRI showed L3 discitis. Due to the worsening clinical condition, a knee arthrotoomy was carried out, with regular tolerance, although it later had good response. After assessing the risk/benefit of the new invasive procedure and the excellent clinical and radiological response experienced after reintroducing ceftriaxone^{5,6}—a later antibiogram confirmed sensitivity to 3rd generation cephalosporins—the disc lesion was not biopsied despite being aware that it would have been better for a proper aetiopathological characterisation. However, due to the time sequence, we presumed that the focal point of the infection was urinary and a later asymptomatic bacteraemia aided the haematogenous seeding in the knee and probably in the intervertebral disc. That is the reason why we assumed the aetiology was the same as the one that caused the monoarthritis.⁷

We found out later from hospital records that she had been admitted five months earlier and that the germ that caused the bacteriuria at that time was also *E. coli*.

In conclusion, we have tried to reflect the complexity of infection in immunosuppressed patients and highlight the exceptional coexistence in a patient with MM, having urinary, haematic, joint and probably discal affection due to the same microorganism: *E. coli*. In these types of patients, the most common forms of arthritis are those produced by *Streptococcus pneumoniae*, and the symptoms do not normally spread to multiple sites during their evolution.



Figure 1. Photograph of the left knee, where a large increase in volume can be seen.



Figure 2. X-ray of the anteroposterior view of the left knee, where the increase in soft tissue volume can be seen.

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Acknowledgements

To the *Unidad de Enfermedades Infecciosas y Microbiología* at the *Hospital Universitario La Paz* (Madrid).