Letters to the Editor

Septic Arthritis Caused by Staphylococcus lugdunensis

Artritis séptica por Staphylococcus lugdunensis

To the Editor:

We present the case of infectious arthritis caused by Staphylococcus lugdunensis, after arthrocentesis, in a patient with psoriatic arthritis undergoing treatment with anti-TNF. The interest of this case lies in how infrequent this germ is as a cause of septic arthritis; to our knowledge, this is the first case described of joint infection by S lugdunensis after performing the above mentioned procedure.

The patient is a 41-year-old male with a personal history of psoriatic arthritis undergoing treatment with etanercept 25 mg twice a week and deflazacort 6 mg/24 h. He came to the hospital due to fever which had presented 48 hours prior, as well as pain, swelling, and functional limitation of the right knee. The week before hospitalization, arthrocentesis and intraarticular infiltration with 1 mL of triamcinolone acetonide had been performed.

Upon hospitalization the patient had a 38°C fever with no systemic consequences. Physical examination revealed descamative erythematous lesions on the skin of the trunk and lower extremities and signs of arthritis of the left knee, with no other data of interest. Afterward, an arthrocentesis was performed, obtaining 30 mL of purulent joint fluid. Samples were sent to the laboratory for biochemistry, microbiology, and microcrystal analysis.

Complementary testing, such as hemogram, basic biochemistry, coagulation studies and chest x-ray were all normal. An erythroesedimentation rate of 40 mm/h and a C-reactive protein of 52.9 mg/L were noticed. Simple x-rays of the joint showed an asymmetrical reduction of the joint line and an increase in the density of soft tissue.

Faced with the possibility of septic arthritis secondary to joint infiltration, treatment with etanercept was suspended and empirical antibiotics (IV cloxacillin 2 g/6 h and IV ceftriaxone 2 g/24 h) were started.

The biochemical analysis of the synovial fluid presented more than 50 000 cells, 98% polymorphonuclears, and low glucose. No microcrystals were observed upon examination with polarized light microscopy. The microbiological analysis revealed coagulase negative (CN), S lugdunensis resistant to beta-lactamatics and sensitive to macrolides and quinolones.

After identifying the germ and following the antibiogram, treatment with IV ciprofloxacin 400 mg/12 h and oral rifampicin 600 mg/24 h for 4 weeks was started, with daily arthrocentesis and joint lavages. Echocardiographic imaging and serial blood cultures were negative in their findings.

In spite of the patients' clinical improvement and the negative cultures after the start of specific antibiotic therapy, the progression of the arthritis was not satisfactory, with noticeable compartmentalization of the effusion, requiring surgical drainage and open-sky total synovectomy. Upon discharge the patient was asymptomatic and later underwent a total right knee arthroplasty. S lugdunensis was described in 1988 by Freney et al1; It is a CN germ similar to S aureus. Its incidence has increased during the past few years due to its correct microbiologic identification. It is part of the bacterial flora of the skin and is frequently isolated in the perineal region.2 It leads to skin and soft tissue infections, although some infections on native heart valves have been reported, as well as breast abscesses, osteomyelitis, etc.1,3 It is related to recurring infections of joint prosthesis and after procedures such as arthroscopy.1,4

S lugdunensis fundamentally leads to infections in immunocompromised patients.1,4 Among its virulence factors, the capability of binding to the extracellular protein matrix, fibronectin, and fibrinogen stand out. As with S aureus, it produces a secretion that facilitates periprosthetic infections and impedes penetration by antibiotics, explaining the tendency of these infections to reappear.5

In order to identify it, certain biochemical tests allow its differentiation from the rest of the CN germs, such as ornitine decarboxylase, the pyrroldilarylamidase test, and the mannose acidification test.

Sensitivity of CN germs to antibiotics depends on the species and the nosocomial or extrahospital orig of the strain. S lugdunensis shows sensitivity to all groups of antibiotics used for treatment of staphilococcal infections, including penicillins.6 In our setting, S lugdunensis presents good sensitivity to beta-lactamatics; betalactamaste producing strains are found in 4% compared with 29% of strains producing betalactamase in american cases.7 The existence of macrolide and fluoroquinolone resistant isolates has been communicated.

Face with the isolation of a CN germ and S lugdunensis in particular, we should be alert to its action as a aggressive pathogenic germ in the immunocompromised patient. The interest of this case lies in how infrequent this germ appears to be, the pattern of resistance to beta-lactamic drugs and its progression to joint deterioration, finally meriting total knee arthroplasty.