Letter to the Editor

Olecranon bursitis caused by group C streptococcus in a patient with tophaceous gout

Dear Editor,

Non-A or B beta-hemolytic streptococci include mainly the C and G groups. Even though rarely classified to the species level by the laboratories, such groups are currently named Streptococcus dysgalactiae subspecies equisimilis (SDSE). In clinical terms, community-acquired pharyngitis and skin lesions largely predominate, and these infections are particularly diagnosed in individuals with comorbidities.1 We herein describe an unusual case of a patient with tophaceous gout who developed an olecranon bursitis due to a group C streptococcus.

The patient, a 72-year-old white male, has presented tophaceous gout for the last three decades, with involvement of feet, elbows, and hands. In the last years, the patient evolved to chronic renal disease due to urate interstitial disease and anti-inflammatory overuse. He was recently admitted to the hospital because of fever and a sudden enlargement of the right olecranon bursa. Physical examination revealed diffuse tophaceous deposits in feet, hands, and left elbow, and an intense redness and swelling of the right olecranon bursa. Hemoglobin level was 9.3 g/L, and the leukocyte count was 9490 cells/mm³. The uric acid level was slightly elevated (7.6 mg/dL). Creatinine level was 1.9 mg/dL. Echography of the right elbow demonstrated an inflamed olecranon bursa. The bursa aspiration yielded a milky synovial fluid (SF); cell count revealed 19,200 leukocytes/mm³, 70% being neutrophils. The SF Gram staining showed Gram-positive cocci, and a search for crystals was positive for monosodium urate. Oxacillin (2 g 6 times a day) was promptly started. Two days afterwards, the SF grew a beta-hemolytic streptococcus of group C. Oxacillin was replaced by G penicillin (2,000,000 IU every 4 h). In parallel, bursal fluid drainage was carried out. After three weeks, the outcome was favorable.

Staphylococcus aureus is identified in the large majority of cases of septic olecranon bursitis.2 The current case is probably the first describing an olecranon bursitis caused by a group C streptococcus in a patient with tophaceous gout.

Septic arthritis due to group C streptococcus is, perse, far uncommon. Ten cases of septic arthritis due to group C streptococcus were reviewed in 1990: polyarticular presentation (5/10), organic dysfunction (4/10), and fatal outcome (3/10 patients) distinguished this infection from the usual streptococcal arthritis. No patient had concomitant bursitis.3 Although our patient presented morbidities as chronic tophaceous gout and renal disease, his infection involved only one site, did not cause further organic damage and was rapidly responsive to G penicillin.

In 1991, a novel case of septis arthritis due to a SDSE was described.4 The last description of a case of musculoskeletal infection (myositis plus septic arthritis) due to a group C streptococcus dates from 1998.5

In summary, probably not more than 15 cases of arthritis caused by group C streptococci have been described to date,3-5 and septic bursitis is so far unreported. Although rare, infection by this streptococcus strain should be part of the differential diagnosis of septic arthritis or bursitis. Given the potential severity of the clinical picture, early use of G penicillin and drainage of the involved structure are highly recommendable.

Conflict of interest

The authors have no conflict of interest to declare.

REFERENCES


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