the low cellularity of the SF, induced us to continue to suspect septic arthritis.

References


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Serratia marcescens septic sternoclavicular joint arthritis: A case report

Artritis séptica esternoclavicular por Serratia marcescens: a propósito de un caso

To the Editor,

Septic arthritis of the sternoclavicular joint accounts for less than 1% of all the cases of septic arthritis. It is frequently associated with predisposing conditions, such as intravenous drug abuse or diabetes. Given the infrequency of the disease, the diagnosis is often delayed.1 Serratia marcescens is a Gram-negative enterobacterium associated with a wide range of nosocomial infections.2

We report a case of sternoclavicular joint septic arthritis caused by this enterobacterium. The patient was a 70-year-old man diagnosed with hypertension, type 2 diabetes, dyslipidemia and chronic ischemic heart disease in the form of unstable angina, with percutaneous revascularization of anterior descending and circumflex arteries. On admission to the hospital for unstable angina, cathe
terization revealed no evidence of new coronary lesions. On the left shoulder and dysphagia. On clinical examination, the only notable findings were an arterial blood pressure of 150/76 mmHg, body temperature of 38 °C, edema and erythema in left sternoclavicular joint, and pain on moving his left arm. The analytical findings included a hemoglobin level of 11.2 g/dL and leukocyte count at 3600/mm3, with 7.5% lymphocytes and 85.8% neutrophils, platelet

count of 84,000/mm3 and C-reactive protein at 325 mg/L. Blood cultures revealed the presence of Serratia marcescens sensitive to quinolones, carbapenems, aminoglycosides and third-generation cephalosporins. Computed tomography of his neck and chest (Fig. 1) confirmed the presence of an infectious process in the sternoclavicular joint, with no signs of local complications. The results of an otorhinolaryngological examination were normal. It was not possible to obtain a sample of joint fluid. There was no evidence of endocarditis on transthoracic echocardiography. He was treated with 1 g/day of intravenous ertapenem for 4 weeks, followed by a

Fig. 1. Augmentation of soft tissue and obliteration of fat planes associated with the inflammatory-infectious process in left sternoclavicular joint (arrows).

Rheumatoid arthritis and T cell large granular lymphocyte leukaemia: A case report

Arthritis reumatoide y leucemia de linfocitos grandes granulares T. A propósito de un caso

To the Editor,

Large granular lymphocytic (LGL) leukaemia was described by Loughran et al. in 1985. It is characterized as an unusual heterogeneous disorder with clonal expansion of mature T lymphocytes. Although the cause is unknown, antigenic stimuli responsible for inducing the activation of large granular CD8+ effector lymphocytes via different signaling pathways have been implicated. It has been associated with a wide spectrum of signs and symptoms that can be the first or only manifestation of the disease, including asymptomatic periods, splenomegaly, cytopenias, recurrent bacterial infections, B symptoms, hepatomegaly, lymph node involvement, neuropathy and pulmonary hypertension. In addition, an association has been established between LGL leukaemia and autoimmune diseases, forming part of an entity known as pseudo-Felt’s syndrome. We report the case of a 62-year-old woman who developed LGL leukaemia 30 years after being diagnosed with seronegative rheumatoid arthritis (RA).

When she presented to our hospital, the patient was being treated with 5 mg prednisone and 150 mg ranitidine. On physical examination, she had pale skin and mucous membranes, deformed metacarpophalangeal and interphalangeal joints, and splenomegaly. Her laboratory tests were normal, with the exception of a leucocyte count of 1.82 × 10^9/L; neutrophils, 0.877 × 10^9/L; iron deficiency anaemia; platelets, 130 × 10^9/L; complement C3, 70.9 mg/dL; complement C4, 5.1 mg/dL; and positive antinuclear antibodies with a homogeneous pattern. Oral iron therapy and weekly methotrexate were started and her prednisone dose was raised. In view of the clinical course (especially RA and neutropenia), as well as the presence of splenomegaly, we considered a diagnosis of Felty’s syndrome (FS). Computed tomography confirmed the splenomegaly and a bone marrow study revealed the presence of an interstitial and nodular infiltrate of T lymphocytes expressing CD3, CD8, T-cell receptor (TCR) βF1, and CD57, suggestive of infiltration by LGL leukaemia. Four months after the initiation of treatment with methotrexate, the patient developed an abdominal wall abscess requiring antibiotic therapy and surgical drainage.

Large granular lymphocytic leukaemia is an uncommon clinical condition, characterized by an indolent, nonprogressive clinical course. The symptoms present during the sixth decade of life, and it affects both sexes equally. It constitutes 2–5% of all T/natural killer (NK) cell neoplasms. To date, 400 cases have been reported in the literature. Given the criteria established for LGL leukaemia, which require the presence of clonal expansion of LGL in peripheral blood >0.5 × 10^9/L and or bone marrow and a study showing

References


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