2-week regimen of oral ciprofloxacin (500 mg/12 h). In view of the favorable clinical response and analytical findings, surgical treatment was ruled out.

Septic arthritis of the sternoclavicular joint is an uncommon disease[3] in both immunocompetent and immunocompromised individuals.[4] The risk factors are diabetes mellitus, rheumatoid arthritis, intravenous drug abuse, neoplastic diseases, chronic kidney disease, human immunodeficiency virus infection, cirrhosis, local trauma and central line infections.[4] The fact that our patient was a diabetic and, moreover, had undergone cardiac catheterization is important. *Staphylococcus aureus* is the most common causative agent.[5] Until now, there has been only one case attributed to infection by *Serratia marcescens* in the medical literature.[6] The most common mechanism of infection is bacteremia.[7] Patients may complain for days or even months of pain in shoulders, neck or chest, with limited arm mobility, associated with fever. The clinical picture in our patient was similar to those reported by other authors. However, we consider that the dysphagia was related to extrinsic compression of the esophagus.[1] Joint inflammation and erythema can also be present. Sternoclavicular arthritis is generally unilateral, and is right-sided in 60% of the cases. Bacteremia is found in 62% of the patients. Computed tomography is the initial imaging technique that can identify bone involvement and detect retrosternal dissemination. The most serious complication is mediastinitis, which occurs in 15% of the cases.[7] The initial therapeutic approach includes prolonged antibiotic therapy when there are no complications. However, in the presence of extensive osteomyelitis, abscesses or mediastinitis, surgical treatment is recommended.[8] Debridement is the surgical technique associated with the lowest incidence of complications.[9] In conclusion, septic arthritis of the sternoclavicular joint is uncommon, especially that caused by enterobacteria. However, it is potentially disabling and fatal, and should be suspected in any condition that affects the sternoclavicular region.

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

Artritis 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.[1] In addition, an association has been established between LGL leukaemia and autoimmune diseases, forming part of an entity known as pseudo-Felty's syndrome.[2] 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 leukocyte count of 1.82×10^9/L; neutrophils, 0.877×10^9/L; iron deficiency anemia; platelets, 139×10^9/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.[1] 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

☆ Please cite this article as: Herráez-Albendea MM, Jarilla-Fernández MC, Jiménez-Burgos F, Sánchez-Rodriguez E. Artritis reumatoide y leucemia de linfocitos grandes granulares T. A propósito de un caso. Reumatol Clin. 2016;12:239–240.

the characteristic immunophenotype (CD3+/CD8+/CD57+ and/or CD16+), as well as clonal TCR gene rearrangement,[3] we considered a possible diagnosis of this disease, as our findings were consistent with the reported features. In view of the patient's medical history, we ruled out other conditions like FS, a systemic complication of RA characterized by the triad of RA, persistent neutropenia and splenomegaly, which is closely associated with the HLA-DR4 haplotype (nearly 95% of the patients), although some authors have suggested that FS and LGL leukaemia associated with RA are expressions of a single entity characterized by LGL proliferation.[2,4] It must also be distinguished from infections, hematologic neoplasms, solid tumors and autoimmune diseases. We consider it of interest to highlight the association between LGL leukaemia and certain autoimmune diseases, including RA, Sjögren's syndrome, polymyositis, rheumatic polymyalgia, vasculitis,[5] endocrine disorders, celiac disease and autoimmune polyendocrinopathy-mucocutaneous candidiasis-ectodermal dystrophy syndrome, among others, resulting in a condition known as pseudo-Felty's syndrome.[2]

In conclusion, the diagnosis of LGL leukaemia associated with RA requires a high index of suspicion, based on the connections between patient history and analytical and radiological criteria, as well as the confirmation of a characteristic immunophenotype in peripheral blood and/or bone marrow.[6] Detailed descriptions of new cases could contribute to the achievement of a better understanding of this condition and to the awareness of the importance of early diagnosis and therapeutic intervention.

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