Seronegative cat scratch disease in a patient with systemic lupus erythematosus

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To the editor,

Cat scratch disease (CSD) is an infectious disease caused by *Bartonella henselae*, which is transmitted through cat bites or scratches. The most typical presentation is regional subacute lymphadenitis, that usually affects children and has a benign and self-limited clinical course[1]. However, immunosuppression is a known risk factor for the development of serious and atypical forms of disease[1].

We present the case of a 21-year-old woman with systemic lupus erythematosus (SLE) with cutaneous, articular and hematological manifestations and class IV lupus nephritis diagnosed at the age of 16 years. She was treated with methylprednisolone pulses (MP) and mycophenolate mofetil (MMF), as induction and maintenance therapy, along with hydroxychloroquine. The patient had a favorable clinical course with complete nephritis remission for 2 years, when she presented with a lupus nephritis flare due to nonadherence to treatment, successfully managed with MP and MMF dose adjustment.

Six months later, under MMF 3g/day and prednisolone 15 mg/day, she presented in the Rheumatology Clinic with a one-week history of daily high fevers (up to 39.5ºC), myalgia, nausea and mild colicky abdominal pain. Physical examination was unremarkable. Laboratorial workup showed white cell count of 11.470×10^9/l, C-reactive protein (CRP) 41.9 mg/L, normal blood urea nitrogen and creatinine, unremarkable urinalysis, negative cultures of urine and blood, normal complement levels, and negative anti-double-stranded-DNA antibodies. Chest radiography and abdominopelvic ultrasound showed no particular findings. One week later the patient was admitted for persistent symptoms. On the fifth day of hospitalization the patient complained of bilateral inguinal pain and multiple tender enlarged lymph nodes were palpable in the groin area. Upon further questioning she admitted exposure to a pet cat and many stray cats. Laboratory tests showed leukocytosis with neutrophilia and rising CRP (168.0 mg/L). Inguinal ultrasound confirmed the presence of superficial inguinal lymphadenopathy with 40 mm of maximum diameter, and abdominopelvic computed tomography confirmed enlarged lymph nodes in inguinal and iliac chains without other significant findings (Figure 1). A fine-needle aspiration biopsy of an inguinal lymph node was performed with a positive result of polymerase chain reaction (PCR) assay for *Bartonella henselae*. All other microbiological tests were negative, including serology for Bartonella. Echocardiogram and ophthalmological evaluation were unremarkable. She was treated with azithromycin 500 mg/day for 5 days with marked improvement.

There are very few reports of CSD in SLE patients[2,3]. The majority of cases affecting immunocompetent patients are self-limited and antibiotics are generally not required. However, in immunocompromised patients there can be local or systemic complications including suppurative lymphadenitis, retinitis, endocarditis and hepatosplenic disease[1]. In this case, considering the immunosuppressed state, duration of the disease and a

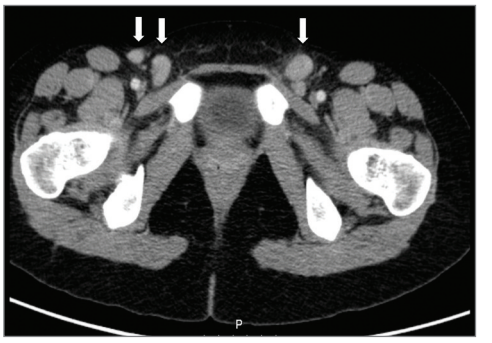

FIGURE 1. Abdominopelvic computed tomography showing bilateral inguinal lymphadenopathy (white arrows)

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well-appearing patient, we decided to treat with a short course of antibiotherapy, with excellent response. This fact is noteworthy as many of these cases require prolonged treatment[2,3].

Serologic testing is the most extensively used technique for the diagnosis of CSD, but its sensitivity is variable, ranging from <30% to 100%[4,5]. PCR sensitivity with samples of lymph node tissue or aspirates is 30-60%, but it has high specificity and allows rapid identification[4]. Other cases of seronegative disease diagnosed by PCR detection of *Bartonella hensellae* DNA have been described[6,7,8].

In conclusion, CSD should be considered in SLE patients presenting with fever and lymphadenopathy and in highly suspicious seronegative cases PCR might be helpful.

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