Bartonella henselae in a Patient with Cat Scratch Disease

Clinical presentation of infection due to Bartonella henselae ranges from a relatively mild lymphadenopathy with few additional symptoms, seen in cat scratch disease (CSD), to life-threatening systemic disease in immunocompromised individuals [1]. The more severe clinical manifestation in immunocompromised hosts points to a role of T cells in the pathogenesis of these infections. We describe a patient with CSD and Reiter’s syndrome, whose cellular immune response to B. henselae was investigated by using a lymphoproliferation assay.

A 27-year-old woman presented with a 4-week history of inguinal lymphadenopathy, and a 1-week history of fever. Despite therapy with trimethoprim-sulfamethoxazole for 10 days, the lymph node had continued to enlarge. Therefore, she was admitted to the hospital. Physical examination was unremarkable except for a tender, dolent, 8-cm × 4-cm right inguinal lymph node and cat scratch lesions on the right leg. She had acquired a young stray cat during her holiday in Sicily 3 months earlier. Serologies for common agents of lymphadenopathy were negative. Fine needle aspiration of the lymph node yielded sterile pus. During the follow-up, erythema nodosum of the left leg and arm, arthritis of the left ankle joint, and conjunctivitis of the right eye developed. Reiter’s syndrome was suspected and testing for HLA-B27 antigen was positive. Cultures of stool, urine, and cervical smear specimens did not reveal any pathogens. The patient was treated with oral antibiotics.

**Detection of Specific Cellular Immune Response to Bartonella henselae in a Patient with Cat Scratch Disease**

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**Figure 1.** Stimulation of peripheral blood lymphocytes of the patient (black bars) and of two representative healthy donors (white and hatched bars) with heat-killed *Bartonella henselae* in the indicated concentrations. Proliferation was determined after incubation for 5 days. Results are presented as stimulation index (thymidine uptake after antigen stimulation divided by the spontaneous uptake).
B. henselae DNA was isolated from the lymph node aspirate by using PCR amplification of citrate synthase gene and restriction fragment length polymorphism (RFLP) analysis [2]. Culture for Bartonella species performed on solid agar media and incubated for up to 6 weeks did not yield B. henselae. Serology was performed by using an indirect fluorescence assay (MRL, Cypress, CA). Diagnostics serum samples collected at 2 and 6 weeks and 8 months after admission yielded titers of 256, 128, and 32, respectively, for IgG antibodies to B. henselae.

Peripheral blood lymphocytes were evaluated for B. henselae—specific stimulation using a whole-blood lymphoproliferation assay [3]. Heparinized blood was collected from the patient 2 weeks after admission, and blood samples from five healthy donors were analyzed in parallel. Heat-killed B. henselae (ATCC [American Type Culture Collection] 49793) was used as a specific stimulus, whereas lectin mitogens served as positive controls. We found a specific proliferation of the patient’s lymphocytes upon stimulation with B. henselae, whereas lymphocytes from the five healthy donors did not respond to equivalent stimulation (figure 1).

To our knowledge, there are no data available concerning B. henselae—specific cellular immune responses in vitro in patients with CSD. The CSD skin test used previously as one of four criteria for clinical diagnosis of CSD [4] determined a delayed-type hypersensitivity to tissue homogenates containing the (at that time) unknown agent of CSD. The skin test antigen is, however, not commercially available and tissue homogenates bear the risk of transmitting pathogens. Thus, an in vitro lymphoproliferation assay might be a useful diagnostic adjunct, especially in cases for which serologic results are equivocal. (After preparing this article, we studied the peripheral blood lymphocytes of another CSD patient, and found, as described in this report for the first time, a Bartonella-specific proliferation of her cells.)

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References

Mycoplasma pneumoniae and Second-Degree Heart Block

Cardiac manifestations of infection due to Mycoplasma pneumoniae have been described previously in association with myocarditis and pericarditis [1–3]. M. pneumoniae infections have been associated with nonspecific electrocardiographic modifications and are not believed to be responsible for potentially serious high-level heart blocks. Herein we describe a patient with second-degree heart block associated with M. pneumoniae.

A 45-year-old man was admitted to our intensive care unit because of mild respiratory distress, fever (temperature, 39.8°C), myalgia, cough, and diarrhea. At the time of admission, his blood pressure was 120/85 mm Hg, his pulse was 85/minute, and his respiratory rate was 27/minute. One week before admission, he had a mild illness with cough and fever. He had no history of cardiovascular disease, and had not been taking any medications. A chest radiograph demonstrated bilateral interstitial infiltrates. Arterial blood gas determinations while the patient was breathing room air included the following values: pH, 7.43; PaO₂, 8.32 kPa; PaCO₂, 4.29 kPa; bicarbonate, 19.5 mmol/L; base excess, 2.5; and Sao₂ 88%. Laboratory evaluation revealed the following values: WBCs, 15,600/mm³ (80% neutrophils); hemoglobin, 10 g/dL; and C-reactive protein, 226 mg/L. Blood electrolyte (potassium, sodium, and calcium), blood urea and nitrogen, and creatinine levels and platelet counts were normal. Cultures of blood were sterile, and serological investigations were negative for antibodies to Chlamydia and Legionella species and HIV. Conversely, a serology for Mycoplasma pneumoniae with use of ELISA was positive according to the following results: on the first serum sample (hospital day 2), positive for IgM antibodies with IgG antibodies; and on the second sample (20 days later), a significant increase in IgG antibodies coinciding with the strong decrease in IgM. An electrocardiogram showed no abnormality on the first hospital day. According to the clinical, biological, and radiographic findings, atypical pneumonia was diagnosed, and the initial treatment consisted of iv erythromycin and iv cefotaxime. Oxygen was delivered at a rate of 8 L per minute.

On the second hospital day, the patient experienced dizziness and was bradycardic. An electrocardiogram showed a slow ventricular rate (45/minute) and a constant long PR space (36 milliseconds), with one narrow QRS complex observed every two P nodes. In addition, we noted the absence of a bundle branch block, which was compatible with a second-degree heart block. A 24-hour Holter electrocardiogram showed episodical Luciani-Wenckebach period (figure 1). Transthoracic echocardiography was normal. On the basis of the positive initial serological findings (IgM), the heart block was determined to be of infectious origin; cefotaxime therapy was discontinued. Because of the patient’s excellent tolerance of the bradycardia, the probable nodal level of the heart block (narrow