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Cat Scratch Disease Due to *Bartonella henselae* Serotype Marseille (Swiss Cat) in a Seronegative Patient

Bartonella henselae has been reported to cause various clinical syndromes, including meningoencephalitis (9), endocarditis (6), bacillary angiomatosis (4, 5, 8), visceral peliosis (5), and cat scratch disease (CSD) (7). However, because of the fastidious nature of the bacterium, few clinical strains have being isolated, especially from patients with CSD. So, the diagnosis of *B. henselae* infection is often achieved by PCR-based methods or, more often, serology.

We report the isolation and characterization of *B. henselae* serotype Marseille (Swiss cat) from a patient seronegative for CSD. A 23-year-old man, with Wilson's disease treated with D-penicillamine and pyridoxine, was admitted to Fondation Hôpital Saint-Joseph, Paris, France, after a 2-week history of left axillar adenomegaly. He had been bitten on the left hand by a rat 1 year ago and on the left elbow by an insect 1 month ago; this latter bite was followed by an inflammatory local reaction. He was also regularly scratched on the forearms by his cat, on which many fleas have been detected. Physical examination showed a voluminous inflammatory adenomegaly. The patient had a temperature of 38°C. Puncture revealed the presence of pus with numerous leukocytes but without visible organisms on Gram, Gimenez, and Ziehl stainings. Treatment with ciprofloxacin (400 mg/day, intravenously [i.v.]) plus oxacillin (3 g/day, i.v.) was begun. All bacterial, including mycobacterial, parasitic, and fungal cultures were negative. Serology for tularemia gave negative results. Despite antibiotic treatment, the fever and the purulent adenomegaly persisted. Surgical excision of the adenopathy was performed. Histological examination showed microabscesses without caseation necrosis, giant cells, or tumor cells. After 3 days, the treatment was changed to amoxicillin (3 g/day, i.v.) plus ciprofloxacin. Cat scratch disease was suspected, but two serological tests against *Bartonella quintana* and *B. henselae* gave negative results. However, when the same two serum specimens were tested against *B. henselae* serotype Marseille, an immunoglobulin G titer of 1:100 was determined. Moreover, the presence of *B. henselae* in the adenomegaly was shown by base sequencing of PCR-amplified *gltA* gene fragments (3). Biopsy material yielded *Bartonella*-like organisms on human endothelial cell line ECV 304 (5), and these organisms were identified as *B. henselae* serotype Marseille by using rabbit antisera raised specifically against this serotype. The strain did not react with antisera raised against serotypes Houston and London.

After surgery, a significant clinical improvement was noted and the patient was discharged on treatment with azithromycin (500 mg/day) for 10 days. Three months later, the patient was considered cured. Only two previous studies have reported the responsibility of this *B. henselae* variant for CSD (1, 2). As with the case reported by Drancourt et al. (2), the patient had a negative serology result when Houston-1 antigen was used. The study of Bergmans et al. (1), using PCRs which amplified part of the 16S rRNA gene, showed that Dutch patients with CSD carry a limited number of *B. henselae* variants. The demonstration of antigenetic variability within the species provides one possible reason for inconsistent results in the serological diagnosis of CSD.

This case underlines the necessity to incorporate *B. henselae* serotype Marseille into immunofluorescence assays to improve the indirect diagnosis of CSD and to improve knowledge of the prevalence of different *B. henselae* variants in patients with CSD.

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