Transverse Myelitis and Guillain-Barré Syndrome Associated with Cat-Scratch Disease, Texas, USA, 2011

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We describe a case of coexisting transverse myelitis and Guillain-Barré syndrome related to infection with Bartonella henselae proteobacterium and review similar serology-proven cases. B. henselae infection might be emerging as a cause of myelitis and Guillain-Barré syndrome and should be considered as an etiologic factor in patients with such clinical presentations.

A child with lower extremity weakness raises an increasingly complex diagnostic challenge; frequently no etiology is identified (1,2). We present a case of lower extremity weakness linked to cat-scratch disease (CSD, causative agent Bartonella henselae proteobacterium).

In 2011, a 10-year-old girl was transferred to our hospital (UT Health, Houston, Texas, USA) from another hospital, where she had been treated for 2 days for abdominal pain, vomiting, and urinary retention. Seven days before admission to UT Health, she had a left cervical lymphadenopathy. During hospitalization, the patient had urinary retention; lower extremity weakness; worsening headache; neck pain; lower back pain; and a bilateral burning sensation in the wrists, knees, ankles, and feet.

Before her illness, she was healthy and fully immunized; her exposure history only included a cat at home that frequently bit and scratched her. Physical examination revealed a palpable lymph node (3 x 4 cm) at the left cervical lymph node, lower extremity strength of 4 on a 5-point scale (https://www.ncbi.nlm.nih.gov/books/NBK436008), and decreased deep tendon reflexes. She reported hyperalgiesia in her legs.

Peripheral blood cell counts and chemistry test values were within reference ranges. Alanine aminotransferase and aspartate aminotransferase were both mildly elevated (48 U/L [0.8 µkat/L]). A magnetic resonance image (MRI) of the brain showed a focus of increased T2 signal, and an MRI of the spine showed a long centrally located segment of increased T2 signal (Figure). Cerebrospinal fluid (CSF) studies showed a leukocyte concentration of 58 cells/mm³ (reference range <10 cells/mm³), glucose of 46 mg/dL (nonfasting reference range 45–100 mg/dL), and protein of 55 mg/dL (reference range 15–45 mg/dL). We gave the patient a diagnosis of myelitis and treated her empirically with ceftriaxone and vancomycin, pending CSF culture results. On day 11 of illness, we started administering rifampin and doxycycline for a possible CSD diagnosis; the patient was positive for B. henselae IgG (1:152) and IgM (1:160). Increases in B. henselae IgG and decreases in B. henselae IgM were seen with subsequent serologic tests: day 27 (IgG 1:256, IgM 1:40) and day 41 (IgG 1:512, IgM 1:20). Evaluation for other etiologies included bacteria culture with urine, blood, and CSF samples; CSF latex agglutination for bacterial antigen; virus culture with nasal washes; rapid plasma reagin test; CSF venereal disease research laboratory testing; enterovirus, herpes simplex virus, and mycobacteria PCR of CSF sample; Epstein-Barr virus and

Figure. Magnetic resonance images (MRIs) on day 10 of illness in a 10-year-old girl with transverse myelitis and Guillain-Barré syndrome associated with cat-scratch disease, Houston, Texas, USA, 2011. A) Brain MRI. Arrow indicates focus of increased T2 signal in the left posterior periventricular and deep white matter. B) Sagittal spine MRI. Arrow indicates long segment of increased T2 signal centrally located within the spinal cord. C) Axial thoracic spine MRI. Arrow indicates increased central signal within the spinal cord.
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improvement, with resolution of urinary retention and

a substantial decrease in pain and weakness; 4 months later,

she had only residual sensory deficits.

B. henselae proteobacterium is transmitted to humans
typically through cat scratches or bites (3). Neurologic

complications, usually self-limited, develop in 0.2%–3.0%
of CSD patients (4). The first case of CSD with neurologic

manifestations was described in 1952. By 1971, ~40 cases
had been reported (5), 90% involving encephalitis and a

few myelopathy (6). The cases of myelopathy had slower

recovery courses than those of encephalitis, as well as more

residual deficits.

Four other serology-documented CSD-associated myelitis cases (3,4,7) and 1 other GBS-associated B. henselae
infection (in a 10-year-old girl) (8) have been described. Carman et al. reported a case similar to the one we de-
scribe: myelitis and GBS in a 12-year-old boy (9).

Studies of the efficacy of treatments for CSD-associated neurologic manifestations are lacking, and thus, the

optimal regimen and duration of therapy are unknown. However, we suggest that clinicians consider CSD early
in disease courses involving neurologic complications; the possibility of GBS, myelitis, or both in the setting of pos-
sible CSD should prompt clinicians to initiate antimicrobial
treatment early and consider steroid or intravenous immu-
noglobulin therapy to prevent progression of disease.

This patient had an unusual presentation of CSD, with
evidence of myelitis, brain lesions, and peripheral nerve

involvement. Although few cases of CSD-associated trans-
verse myelitis and GBS have been described, clinicians

should be aware of the existence of this clinical scenario

and include it as a differential diagnosis for these 2 syn-
dromes in the pediatric age group.

About the Author

Dr. Zakhour is an assistant professor at the American University of Beirut in Beirut, Lebanon. She has a special interest in
immune complications of infections.

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