CASE REPORT

Expressive Aphasia as a Presentation of Encephalitis with *Bartonella henselae* Infection in an Immunocompetent Adult

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**Objective:** To show the first clinically reported case of Cat Scratch Disease (CSD†) presenting as a focal neurologic deficit in an immunocompetent adult.

**Patient:** 59-year-old male with a history of a previous stroke.

**Results:** Examination showed an expressive aphasia, word substitution errors, and impaired repetition. A head CT and MRI showed no acute changes. The EEG findings were non-focal and did not show any epileptiform activity. The patient had a history of contact with stray kittens and previous axillary lymphadenopathy. *Bartonella henselae* serology titers were IgG positive 1:1024 (< 64) and IgM positive 1:20 (< 16). After antibiotic administration, the patient’s symptoms and aphasia resolved.

**Conclusions:** Focal presentations concerning for stroke or partial seizure activity may have underlying infectious etiology. We recommend consideration of CSD in the differential diagnosis of any adult with a history of lymphadenopathy, fever, and recent contact with a cat who presents with neurologic complications.

**INTRODUCTION**

*Bartonella henselae* is the primary cause of Cat Scratch Disease (CSD) [1]. CSD occurs predominantly in the fall and winter months. Regional lymphadenopathy is noted in 85 percent of patients, most commonly in the neck, axilla, and inguinal region [2]. Typically, the disease is benign and self-limited with an associated fever usually occurring one to three weeks after a scratch, bite, or lick from a kitten [3,4]. In immunocompetent patients, the disease is usually self-limited with rare serious com-

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†Abbreviations: CSD, cat scratch disease.

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Applications and often no need for antibiotics [1,5]. Diagnosis is usually based on serologic tests used in the appropriate clinical setting, though PCR of tissue specimens is sometimes helpful [6,7].

CASE

A 59-year-old man presented with the sudden onset of speech difficulties. He reported no other associated symptoms such as headache, dysarthria, dysphagia, weakness, paresthesias, or difficulty understanding speech. Additional history was remarkable for a left frontal stroke in March 2007 that presented with mutism and no other deficits. He had recently adopted several stray kittens. Ten days prior to presentation, he had a skin infection that progressed to right axillary lymphadenopathy, for which he received a five-day course of doxycycline.

The initial examination, two hours after the onset of speech difficulties, revealed an afebrile man with a painless 4mm erythematous papule with a central scab on his right forearm. His neurologic exam was remarkable for an expressive aphasia, word substitution errors, and impaired repetition. A head CT in the emergency department showed a low-density lesion in the region of the left insula, with well-defined borders and no mass effect (Figure 1). The following morning, the patient had an MRI of the brain that showed his prior stroke but no areas of acute ischemia (Figures 2a-e). An EEG showed moderate generalized slowing with no epileptiform activity.

The aphasia fluctuated between making paraphasic errors and a frank expressive aphasia, and he was becoming more confused. Shortly after his EEG and approximately 18 hours from the onset of symptoms,
The patient had a witnessed generalized tonic-clonic seizure. A lumbar puncture showed 133 red blood cells (RBC)/microliter (0 cells/microliter) in tube 1 with no white blood cells (WBC). There were no RBCs in tube 4 with one WBC/microliter (normal 0-5 cells/microliter). The CSF glucose was 60mg/dl (40-70mg/dl), and the protein was 64mg/dl (15-45mg/dl). HSV, VZV, Enterovirus PCR, West Nile and Lyme antibodies, as well as VDRL and CSF culture, were all negative or absent.

The following morning the patient was aphasic, febrile, had an elevated WBC count to 12,000/microliter, and his level of alertness worsened. A repeat EEG showed non-focal findings, including a moderate generalized slowing and frontal intermittent polymorphic delta slowing with no epileptiform activity. Considering the history of stray kittens in his home, a suspicious skin lesion, and previous axillary lymphadenopathy, the possibility of *Bartonella henselae* infection was raised. At approximately 48 hours from symptom onset, *B. henselae* serology studies were sent, and the patient was given one dose of Azithromycin 500mg IV. The next morning, the patient’s symptoms and aphasia had resolved. He was switched to doxycycline and rifampin, based on CSF penetration profiles, and discharged home with a 14-day course of the above antibiotics. *B. henselae* titers were reported as IgG positive 1:1024 (nl < 64) and IgM positive 1:20 (nl < 16). Follow-up imaging several weeks later showed no interval change (Figures 3 a-e).

Today, the patient has resumed his occupation as a musician, singer, and songwriter. He is teaching classes and currently in the recording studio working on an album.

**DISCUSSION**

Our patient is the first adult case of serologically confirmed *B. henselae* infection presenting with a recurring expressive aphasia that we have encountered. The patient had a prior stroke in a region associated with speech apraxias and expressive aphasias, and he presented with mutism at that time. Several studies suggest that the blood brain barrier is dysfunctional in regions of prior infarct, which provides a potential portal of entry to the central nervous system [8,9]. Entry of *B. henselae* and inflammatory mediators at this site could explain initial symptoms of recrudescence of his previous focal deficit. However, while initially presenting with an expressive aphasia, he went on to develop several symptoms inconsistent with a purely ischemic cause or solely recrudescence of a prior stroke. While epileptiform phenomenon can develop in the setting of old cortical ischemic lesions, this is less likely given the non-focal and non-epileptiform EEG findings, the waxing and waning consciousness in the setting of a
febrile patient with an elevated WBC count, and the recent exposure history. CSD encephalopathy was considered more likely. Focal neurologic presentations in a population this age often prompt concern for stroke. When this has been thoroughly evaluated, it is important to consider that systemic infections such as CSD in the proper clinical setting also can present with focal neurologic symptoms.

Most commonly, CSD is diagnosed in patients younger than 21 [10]; in one study, 79.2 percent were younger than 18 [2]. The majority of reports involving neurologic presentations of CSD are from the pediatric literature. In one report, roughly 12 percent had neurologic complications [4], and other reports include severe encephalopathy [11], status epilepticus [12], anti-epileptic drug resistant seizures [11,13], recurrent status epilepticus and prolonged encephalopathy with hemiparesis and chorea [13], pleural effusions and encephalopathy [14], fatal meningitis and encephalitis [15], fatal disseminated systemic disease and encephalitis [16], and acute hemiplegia [17]. There has been one report of an adolescent boy whose CSD encephalopathy presented with acute-onset, self-resolving, recurrent expressive aphasia [18].

A comprehensive look at the neurologic complications of *B. henselae* since 1995 reported that encephalopathy, in particular, was noted, though there have been associations with aseptic meningitis and dementia in patients co-infected with human immunodeficiency virus [19]. More recent reports describe MRI changes associated with CSD neurologic manifestations [17,20,21,22]. Larger studies estimate between 0.17 percent to 2 percent of patients develop encephalopathy [3,23]. Over the past two decades, there have been several case reports of CSD with encephalopathy or meningoencephalitis and status epilepticus [13,24-29]. Such reports have prompted recommendations for the consideration of CSD in the differential diagnosis of any previously healthy adult presenting with neurologic complications who has a history of lymphadenopathy, especially if he owns a cat [1]. One study showed encephalopathy in 61 of 76 patients with neurologic complications of CSD, with 15 showing cranial or peripheral nerve involvement. Of these patients, 78 percent recovered within one to 12 weeks, and 100 percent recovered within one year, with roughly 50 percent receiving antibiotics. None had any neurologic sequelae [30].

Our patient responded to antibiotics rapidly. The self-limited form of CSD is often unaffected by antibiotic treatment [3]. With severe or persistent CSD infection, several antibiotics are effective, including erythromycin and doxycycline [1]. While there is little evidence based treatment for neurologic disease of CSD, expert opinion recommends a combination of doxycycline plus rifampin for 10 to 14 days. Most serious and potentially life-threatening infections respond well to antibiotic therapy [1].

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