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CASE REPORT

Cat scratch disease: an unusual presentation

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Abstract:

Cat-scratch disease (CSD) is a zoonotic infection caused by Bartonella henselae occurring in about 24000 patients annually in the United States. It typically presents with fever and regional lymphadenopathy and is considered as one of the most common causes of chronic lymphadenitis in pediatric population. It generally has a benign and self-limited course, though this is not always the case. Rare forms of extra-nodal involvement, and fatalities in immunocompetent patients have been reported. We report a case of cat scratch disease presented with encephalitis without the usual prodromal symptoms. Cat-scratch encephalopathy should always be considered in patients with an altered mental status of unknown etiology.

Keywords: Cat scratch, Bartonella henselae, lymphadenitis, encephalopathy

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CASE REPORT

A 12 year old Caucasian male with history of attention deficit hyperactivity disorders on amphetamine-dextroamphetamine combination presented with an altered mental status. Patient has no preceding febrile illness. No history of trauma or known intentional ingestions. The family lives in a rural area and they have cat, dog, cockatiel and guinea pig. Parents were not aware of history of scratches or bites by an animal. On presentation the patient was febrile, delirious with writhing movements and posturing. Patient was intubated for airway protection. A complete blood count, electrolytes, liver and kidney functions were unrevealing. Cerebrovascular insult was excluded by computed tomography. Urine drug screen was positive for amphetamines which was expected as he was on amphetamine-dextroamphetamine combination. Antibiotics and antiviral were

initiated to cover a possible meningitis and/or encephalitis. Initial cerebrospinal fluid (CSF) analysis was normal. On the second of hospitalization, the patient had a partial seizure which was controlled with Lorazepam and maintained on levetiracetam. A brain magnetic resonance imaging was normal. Electroencephalography showed non-specific wave changes. By day 3 of hospitalization, sedation was weaned off and the patient was extubated. Post extubation course was complicated by aphasia, ataxia and visual hallucinations. CSF cultures came back negative. Cerebrospinal fluid was also sent for polymerase chain reaction analysis for herpes simplex virus, enterovirus, arbovirus, mycoplasma and West Nile virus, in addition to NMDA receptor antibody, all these studies were negative. A serologic panel for St Louis, eastern and western encephalitis viruses were also negative. Serology for B henselae showed high titer with IgG 1:512

and IgM 1:64. Given that the patient had some new neurological manifestations after extubation, he was started on rifampicin for a total of 10 days. Patient improved gradually and his mentation returned to normal. He continued to do well in post discharge follow up and was weaned off levatiracetam.

Discussion:

Cat-Scratch Disease (CSD) is a self-limiting disease caused by *Bartonella henselae* [1]. It was first reported by Debre' et al in 1950 in a 10 year old boy with lymphadenitis [2]. *Bartonella henselae* is a gram-negative, fastidious, intracellular bacteria tends to cause granulomatous infection of the skin and the regional lymph nodes. There are two identified genotypes of *B. henselae*; Houston (type 1) and Marseille (type 2). Type 2 is more prevalent in North America [3]. CSD tend to occur most often in pediatric age group [4]. *B. henselae* has an incubation period of 1-3 weeks, and generally starts as a papulo-pustular lesion which lasts for 1-3 weeks, followed typically by fever and regional lymphadenopathy in approximately 90% of patients [5]. A 0.17%–2% of cases present with central nervous system involvement usually manifests as an encephalopathy with sudden-onset seizures or altered consciousness as in our reported case [6]. Rarer neurological manifestations include facial palsy, myelitis, peripheral neuritis, paresis, sphincter dysfunction, chorea and cerebellar ataxia [7-10]. Other reported organs involvement include endocarditis, hemolytic anemia, and thrombocytopenia [11]. Gastroenterological manifestations include granulomatous hepatitis and hepatosplenomegaly [12]. Also, one tenth of patients with CSD have musculoskeletal manifestations such as myalgia, arthritis, tendinitis, and osteomyelitis [13]. The pathogenesis of CSD encephalopathy remains to be elucidated. Theories put forth to explain the mechanism include direct invasion of the CNS by the organism. This theory is supported by the abrupt onset and prompt resolution of symptoms, in addition to the positive PCR in CSF and isolation of the organism from, the brain and

other organ tissues [14-16]. A toxic theory was suggested. However, up to our knowledge, no toxins were identified [8]. Immunologic theory was postulated based on the time-lag between the initial presentation of the disease and the development of symptoms [7]. This theory was also supported by identification of *B. henselae* in patient with immune-mediated cerebral vasculitis and another patient with Guillain-Barré syndrome [17-18]. Despite the aforementioned theories, the rarity of the condition as well as lack of tissue diagnoses make a unifying explanation difficult.

CSF analysis in patients with CSD usually yields no abnormalities. Neuroimaging and EEG in patients with encephalopathy are usually normal. Non-specific changes reported in severe cases still ambiguous and gives no clue to the diagnosis [8, 19]. Treatment of cat scratch disease is still controversial. Antibiotics should always be considered in immunocompromised patients. For most immunocompetent patients with mild or moderate disease, only symptomatic treatment is recommended. A five days of Azithromycin was found to be effective in reducing the size of lymph nodes and may be considered in severe and painful lymphadenopathy [20]. Margileth *et al.* [21]. studied 18 different antibiotics on patient with confirmed CSD in both adult and pediatric patients. Rifampin and ciprofloxacin were the most effective, 87% and 84% respectively [21]. The role of steroids is still unclear. High dose steroids were successfully used in severe cases with neurological involvement and in immunocompromised patients [22-23].

Children presenting with acute mental status changes are more likely to be diagnosed with bacterial or viral meningitis, head trauma, ingestion, herpes encephalitis, or tick borne illness. It is important to consider less common diagnoses as well such as bartonella encephalitis even if patients do not display the typical prodromal symptoms.

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