Case Report

The Cat's Meow: A Triad of Optic Neuritis, Iritis, and a Parotid Mass

Lauren Baker, BS[1], Zane Boerner, BS[1], Michael Strong, MD[2], Bethany Nunez, MD[2]

Abstract

Cat Scratch Disease is an infectious process caused by the bacteria *Bartonella henselae*. It classically presents as a self-limited lymphadenopathy after inoculation through a cat scratch. This case details an atypical presentation of optic neuritis, iritis, and a parotid mass. The purpose of this discussion is to promote the inclusion of Cat Scratch Disease in the diagnostic workup of a pediatric patient presenting with these symptoms.

Keywords: Bartonella henselae, Cat Scratch Disease, Parotid mass, Optic neuritis, Iritis

The Condition

In most pediatric patients, cat scratch disease (CSD) presents as a localized cutaneous and lymph node disorder around where the causative organism, *Bartonella henselae,* was introduced. CSD begins with a primary lesion at the site of inoculation 3 to 10 days after the organism enters the skin and then goes through vesicular, erythematous, and papular phases.[1] The hallmark of the disease is lymphadenopathy near the location where the organism entered the skin around 1-2 weeks later. Usually, CSD is self-limited, but in some patients, it can spread to infect the liver, spleen, eye, or central nervous system. The direct mechanism of neurological involvement is unknown. It is thought that *Bartonella henselae* produces a neurotoxin that causes direct damage to cells in the nervous system, or it may induce an autoimmune response.

The different neurological manifestations are neuroretinitis, encephalopathy, and seizures. The less common neurological symptoms include paresthesias, meningomyeloradiculopathy, sphincter dysfunction, facial nerve palsy, cerebral arteritis, transverse myelitis, acute hemiplegia, and Guillain-Barre syndrome.[2] Bartonella, when associated with neuroretinitis, presents with optic disk edema, retinal detachment, and exudates that arrange in the pattern of a macular star. The absence of a macular star suggests a less common ocular neuropathy such as optic neuritis instead of neuroretinitis.[3] Cat-scratch disease rarely manifests with ocular or parotid involvement. Only 1-2% of patients with this disease have ocular involvement.[4] Ocular involvement can result in acute left eye vision loss due to papillitis/optic neuritis and iritis.

Corresponding Author:
Lauren Baker, BS
Texas Tech University Health Sciences Center School of Medicine, Amarillo, Texas
Email: lauren.baker@ttuhsc.edu

Lymphadenitis can result in swelling of the parotid tissue producing a tumor-like mass.[5] This can precipitate a painful mobile swelling over the left parotid area with no other lymphadenopathy.

Case Presentation

A 14-year-old girl presents to the emergency department with a tender nodule over her left parotid gland. The onset of the nodule was six days prior to admission. Two days after the onset of the nodule her vision became blurry, she developed blind spots in her visual fields, and had eye pain with ocular motion. A maxillofacial CT of the nodule in the emergency department was concerning for a parotid adenoma. She was referred to outpatient ophthalmology for evaluation of her visual changes. At the ophthalmology appointment, she was noted to have acute left optic neuritis and bilateral iritis and was referred for admission to the hospital.

Further history on admission reveals that she is sexually active, has no recent travel history, and has regular contact with cats. On admission, her visual acuity is 20/20 in the right eye and 20/50 in the left. An afferent pupillary defect is noted in the left eye with a round pupil of 4.0 mm and 2+ reactive. Her visual fields are full. The nerve margins of the left optic nerve are blurred and have two small hemorrhages. A 2 cm nodule is noted over the left parotid gland and is tender to palpation, mobile, and firm.

Discussion

Differential Diagnosis

The differential diagnosis for the patient's optic neuritis, iritis, and parotitis included malignant, infectious, and autoimmune processes. A mass in the parotid gland in combination with abnormal neurologic symptoms raised suspicion for a malignant process. Many infectious agents, such as Cytomegalovirus (CMV), Epstein-Barr Virus (EBV), Human Immunodeficiency Virus (HIV), Bartonella, and Toxoplasma may present with reactive lymphadenopathy which can lead to an enlarged parotid gland depending on the location of the inflamed node. Although the patient did not have classic signs of bacterial meningitis such as headache or meningismus, her abnormal neurologic findings should prompt the consideration of bacterial meningitis or aseptic meningitis as the underlying etiology.

The sexual history of the patient also indicated the possibility of sexually transmitted infections (STIs) such as chlamydia, gonorrhea, and syphilis. The patient's presentation with optic neuritis and iritis prompted the consideration of a variety of autoimmune diseases such as juvenile idiopathic arthritis (JIA), multiple sclerosis, systemic lupus erythematosus (SLE), and sarcoidosis. Optic neuritis is typically secondary to demyelination, such as is seen in multiple sclerosis.[6] Ocular findings alone would not be sufficient for the diagnosis of SLE but should prompt a thorough history and physical exam for the evaluation of other underlying symptoms. Lymph node involvement with the presence of ocular symptoms may lead to a concern of sarcoidosis. Autoimmune diseases such as rheumatoid arthritis and systemic lupus erythematosus can be a cause of aseptic meningitis.[7,8]

Actual Diagnosis

To evaluate the diagnosis, imaging of the head and neck were obtained, sexually transmitted infection (STI) tests were done, and a lumbar puncture was performed. CSF cell count revealed pleocytosis consistent with aseptic meningitis.

The MRI showed that the nodule over the parotid gland was a reactive lymph node. This along with the visual changes due to optic neuritis and iritis indicated that the patient had an infection rather than a parotid adenoma. With STIs such as chlamydia, gonorrhea, and syphilis, the patient would be expected to present with genitourinary symptoms. The negative tests with the absence of normal STI presentation lowered the suspicion of these infectious agents. Laboratory tests for toxoplasma, CMV, EBV, and HIV were all negative. CSF culture x 24 hours was negative. These results decreased the likelihood of viral or bacterial meningitis. Negative anti-neutrophil antibody (ANA), rheumatoid factor (RF), and erythrocyte sedimentation rate (ESR) tests and no evidence of demyelination on imaging lowered the possibility of optic neuritis due to an autoimmune disease.

The MRI indicative of an infectious etiology, history of cat scratches, lymphadenopathy, and ocular involvement suggested the possibility of infection with *Bartonella henselae*. Cat scratch disease is characteristically diagnosed by meeting 3 out of 4 criteria including recent cat exposure, a positive test for *Bartonella henselae* antibodies, lymph node lesions, and lymphadenopathy with negative lab results for other causes [4]. The diagnosis was confirmed as cat-scratch disease post-discharge by elevated *Bartonella henselae* IgG titers at 1:128 (Reference Range: < 1:64) on 10/15/2018.

In most patients, Bartonella infection is self-limited, and treatments are supportive. In vitro testing has shown that various antibiotics are effective for the treatment of Bartonella. The indication for treatment with antibiotics is lymphadenitis to prevent the dissemination of infection. In CSD with lymphadenitis, azithromycin is the preferred treatment with clarithromycin, rifampin, and trimethoprim-sulfamethoxazole as alternative options. There is limited evidence for the specific treatment of cat scratch disease with neurological involvement.[9] The combination of doxycycline and rifampin is recommended for children over 8 years of age and has shown to promote the resolution of neurological presentations. The addition of systemic corticosteroids can also aid the resolution of the symptoms especially in patients with severe or persistent disease.[10]

The suspicion of cat-scratch disease in this patient led to empiric treatment with azithromycin, but the patient was transitioned to a regimen of doxycycline, rifampin, and prednisone with recognition of neurologic extension. Following medical treatment, the patient's bilateral iritis resolved, and optic neuritis improved. The lymph node enlargement gradually subsided. The patient's symptoms continued to improve on empiric therapy for Bartonella infection and she was discharged. The patient made full recovery with completion of antibiotic regimen.

Conclusion

Consider *Bartonella henselae* infection with the presentation of optic neuritis, iritis, and a parotid mass. In addition, Bartonella antibody testing is recommended to avoid unnecessary biopsy of a parotid mass or prior to beginning therapy for optic neuritis or neuroretinitis.

Optic neuritis secondary to infection can be misdiagnosed as demyelinating optic neuritis in young patients. To help avoid a misdiagnosis, providers should obtain a full and complete history including contact with pets or other animals because it is important to rule out bacterial infection prior to a diagnosis and treatment.

Affiliations:
[1]Texas Tech University Health Sciences Center School of Medicine, Lubbock, Texas
[2]Department of Pediatrics, Texas Tech University Health Sciences Center School of Medicine, Lubbock, Texas

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