

Unilateral ocular manifestation of cat-scratch disease in a 13-year-old child: Case report

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ABSTRACT

Cat-Scratch Disease (CSD) is a zoonotic infection caused by gram-negative facultative intracellular bacillus *bartonella henselae*. Ocular manifestations of Cat-scratch disease include Parinauds oculoglandular syndrome, uveitis, vitritis and neuroretinitis. Although there is wide range of ocular manifestations of Cat-scratch disease, the concurrent presentation of Parinaud's oculoglandular syndrome and neuroretinitis is rare. We present a case of a 13-year-old girl with cat-scratch disease who developed both conditions in the left eye following multiple cat scratches. The patient was treated with intravenous gentamycin, oral doxycycline and tapering oral prednisolone. Her visual acuity improved from hand movement to 6/9 in the left eye and ocular inflammation resolved by discharge.

Key words: Cat-scratch disease, Neuroretinitis, Parinaud's oculoglandular syndrome

INTRODUCTION

Cat-Scratch Disease (CSD) is one of the zoonotic infections caused by a gram-negative facultative intracellular bacillus *bartonella henselae*¹⁻³. Other less common possible causative agents include *afipia felis* and *bartonella clarridgeiae*. Flea faeces are the major sources of these bacteria, through scratch, bite, or lick from infected cats, specifically kittens, humans are usually infected, or less commonly through ticks and other animal scratches and bites^{3,4}. Overall, the disease manifests itself as a self-limiting illness marked by regional lymphadenopathy, pustular skin lesions, mild fever, and generalised malaise. Most cases recover without complications, though some atypical presentations can occur especially in immunocompromised persons. In such cases, there is the risk of systemic spread, with the liver, spleen and bones being affected or developing neurological and ocular complications⁵. Due to lack of randomized clinical trials the treatment of CSD is not standardized and it varies among clinicians. However observational data describe potential clinical response with trimethoprim-sulfamethoxazole, rifampin, azithromycin, gentamicin and ciprofloxacin⁶. Furthermore, various combinations of doxycycline, rifampin and gentamicin have been used for encephalopathy, neuroretinitis and hepatosplenic disease; in some instances of the latter conditions, resolution has been achieved in combination with steroid use⁷. Despite generally good outcomes, potential long-term sequelae include disc pallor, afferent pupillary defects, retinal pigmentary changes, and mildly decreased visual acuity and mild postinfectious optic neuropathy⁸.

The ocular manifestations of CSD can be divided into two main groups. The first, Parinaud's Oculoglandular Syndrome (POGS), will have conjunctivitis and will often have preauricular lymphadenopathy⁹. The second is neuroretinitis characterized by optic nerve swelling, granulomatous inflammation of the retina and optic nerve, the presence of macular stars, and subsequent possible vasculitis¹⁰. Overall, ocular involvement will be unilateral, although there are rare reports of bilateral disease. We present a case of a female child with CSD who developed unilateral POGS and neuroretinitis.

CASE REPORT

A 13-year-old girl presented to Lions Sight First Eye Hospital in Mzuzu, Malawi on February 12th 2025. She complained of redness in both eyes for the past month, followed by swelling of the left eyelids, which began one week prior. Her symptoms were associated with tearing and occasional left eye pain. She also reported reduced vision in the left eye, which was initially normal when the redness started but began to decline over the past week. Additionally, she experienced occasional headaches. Upon further questioning, the patient revealed a history of multiple exposures to cat scratches. Most recently, she was scratched by a juvenile cat six days prior, sustaining injuries on her left arm and right (Figure 1). The patient reported no history of systemic diseases. She had a history of eye conditions including bacterial conjunctivitis and allergic conjunctivitis.

On examination, the patient's visual acuity was 6/9 in the right eye and hand movement in the left eye. The

pupils were reacting to light and ocular motilities were full in both eyes. Furthermore, intraocular pressure was 9mmHg in the right eye and 10mmHg in the left eye.

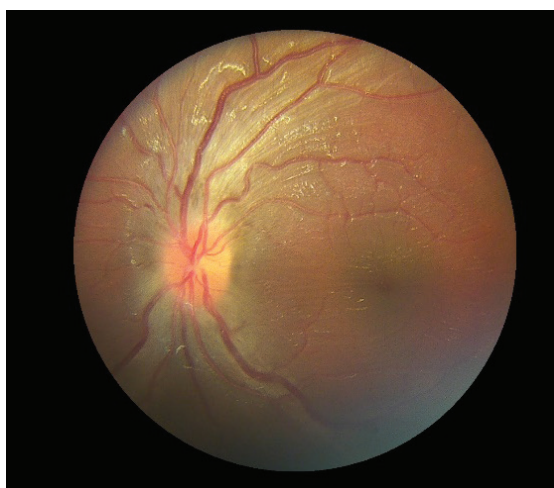
Right eye: The right eye showed mild conjunctival hyperaemia, but the other anterior segment structures were healthy. Fundus examination of the right eye the cup to disc ratio was 0.3 with a healthy retina.

Left eye: Anterior segment examination revealed swollen eyelids and a hyperaemic conjunctiva with follicles. Fundus examination of the left eye showed a swollen optic nerve head with blurred disk margin, a hyperaemic optic disc and exudates around the macular (Figure 2). A full blood count was ordered which revealed an elevated white blood cell count of 12,400 and neutrophils levels were markedly elevated at 71.6%, consistent with a bacterial infection.

Figure 1: Images showing cat scratches (Black arrow) and left eye inflammation. Image showing a cat scratch on the leg (A). Image showing a cat scratch on the arm (B). Image showing left eye swollen eyelids (C)



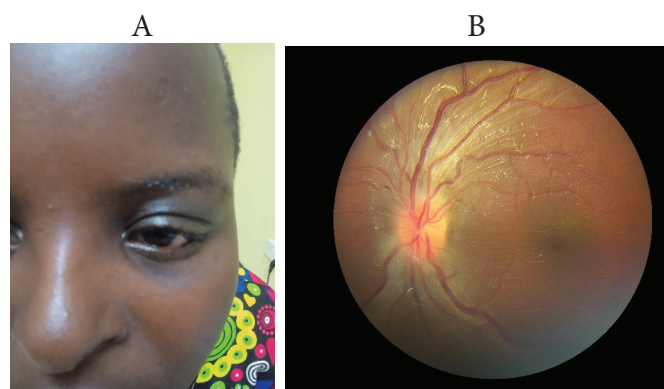
Figure 2: Retinal image of the left eye on day of presentation



The diagnosis was Parinaud's oculogranular syndrome and neuroretinitis in a female child with CSD. The patient was admitted in the paediatric ward at Lions Sight First Eye Hospital in Mzuzu, Malawi. The patient was prescribed gentamicin IV 80mg once daily for 14

days, doxycycline PO 100mg twice daily for 6 weeks and tapering prednisolone was also prescribed (40mg-5mg) for 7 days. On the day of discharge, the visual acuity was 6/9 in the left eye and the eye inflammation was resolving (Figure 3).

Figure 3: Left eye images on the day of discharge. Image depicting the resolved eye inflammation (A). Image illustrating the resolving neuroretinitis (B)



DISCUSSION

Our patient presented with Parinaud's oculoglandular syndrome and neuroretinitis which led to the clinical diagnosis of Cats Scratch Disease (CSD). She had remarkably reduced vision, with a visual acuity of Hand Movement (HM) in the left eye, follicular conjunctivitis and neuroretinitis. The reduced vision could be attributed to macular exudates and optic neuritis. Other ocular presentations of CSD reported in literature include multi focal retinitis, uveitis, retinal vessel occlusion and retinal detachment¹¹.

Ocular manifestations of CSD are divided into two groups namely Parinaud's oculoglandular syndrome and neuroretinitis¹². Features of Parinaud's oculoglandular syndrome include conjunctivitis and preauricular lymphadenopathy while features of neuroretinitis are optic nerve swelling, granulomatous inflammation of the retina and optic nerve, the presence of macular stars, and subsequent possible vasculitis¹³. Overall, ocular involvement is unilateral, although there are rare reports of bilateral disease⁸. Most case reports describe neuroretinitis as the ocular manifestation of CSD while POGS is uncommon, occurring in only about 6% of patients¹⁴. Our patient uniquely showed concurrent Parinaud's oculoglandular syndrome and neuroretinitis as evidenced by follicular conjunctivitis, optic nerve head swelling, hyperaemic optic disk, blurred optic margins and macular exudates.

Exposure to young cats or kittens is a significant risk factor for CSD¹⁵. One week before presentation the patient sustained cat scratches and lived with cats in her home. No *Bartonella* specific laboratory tests were performed which is a limitation of the case report although clinical findings strongly supported CSD.

Bartonella henselae is a gram-negative intracellular bacillus that cause CSD and spread via scratches, bites, or flea faeces. The bacteria induce regional lymphadenopathy and occasionally systemic lymphadenopathy after entering the lymphatic system¹⁶. When the *Bartonella* spreads haematogenously to the optic nerve and retina, it causes a granulomatous inflammatory reaction that involves the eyes. Conjunctivitis and preauricular lymphadenopathy are the hallmarks of POGS, whereas optic disc oedema, macular star formation and vasculitis are the hallmarks of neuroretinitis¹¹.

CSD related neuroretinitis must be differentially diagnosed for the inflicting conditions of infectious, inflammatory and neoplastic processes. The most common immune mechanisms involving neuroretinitis include syphilitic perineuritis, toxoplasmosis, tuberculosis, and leptospirosis. Autoimmune and inflammatory opticulate conditions e.g. optic neuritis, sarcoid papillitis, or pseudotumor cerebri cause swelling of the optic nerve

and need to be ruled out carefully. Neoplastic causes like lymphoma ought to be suspected when associated with systemic symptoms¹⁷.

In order to confirm the diagnosis, laboratory tests are essential. The Enzyme Linked Immunosorbent Assay (ELISA) and Immunofluorescence Assay (IFA) are the two serological assays for *Bartonella henselae* that aid in the detection of IgM or IgG antibodies where high titers indicate a recent infection¹⁸. *Bartonella* DNA can be found in blood or ocular fluids with great specificity using Polymerase Chain Reaction (PCR) testing, however blood cultures are less accurate because of the bacteria picky character¹⁹.

Other diagnostic measures offer considerable support for the diagnosis of CSD induced neuroretinitis. Some preferred indicators include retinal vasculitis, macular exudate, and disc oedema, which can be visualized through fundoscopy. Optical coherence tomography reveals any retinal thickness and macular oedema, while fluorescein angiography detects leakage from the optic disc. MRI contrast is used to exclude optic neuritis or lesions threatening the brain in case of neurological involvement²⁰.

Management of CSD mainly comprises of antibiotic therapy²¹. Our patient was managed with antibiotics and glucocorticosteroids. While many studies report the use of antibiotics as the primary management for CSD few studies had included a steroid in their treatment regimen²⁰. Our patient's vision improved to 6/9 in the left eye within 10 days with resolution of conjunctivitis, neuroretinitis and macula exudates. Follow up in CSD is necessary to note disease progression and complications²². Our patient was followed for a period of two weeks during admission, and a follow up appointment was scheduled for two weeks and then six months after discharge, however the patient missed the first outpatient visit.

Overall, CSD resolves on its own, however if there is ocular phenotype involved, it has to be treated to prevent vision loss. Oral doxycycline, historically prescribed as the most efficacious treatment at 200mg once a day for 14-28 days. In addition, some other antibiotics have also been used in the therapy of CSD and may include ciprofloxacin, rifampin, trimethoprim and gentamicin²³. Alternative treatments include erythromycin for children under 12 years to prevent tooth discoloration and azithromycin for rapid lymphadenopathy. In severe cases, intravitreal anti-VEGF therapy may be considered for neovascularization or macular oedema²⁴. Depending on the severity of neuroretinitis, the prognosis of vision varies. According to some studies, 80% of the affected eye achieve better than 20/40 vision at diagnosis, 14% have moderate visual impairment (20/40-20/200), and 6% have severe visual impairment (<20/200)²⁰. Recovery is poor when the optic nerve is heavily involved.

CONCLUSION

Parinaud's oculoglandular syndrome and neuroretinitis are rare findings in patients with CSD. This case report emphasizes on how important it is to identify POGS and neuroretinitis as abnormal ocular signs of CSD and therefore suggests that CSD should be included in the differential diagnosis of patients presenting with POGS and neuroretinitis. In order to rule out other infectious and inflammatory diseases, laboratory and imaging tests are required. Early antibiotic and glucocorticosteroid therapy are important for better visual outcome. Public awareness campaigns should be conducted to educate communities about the transmission of CSD through cat scratches, bites, and flea faeces. Comprehensive flea treatment for cats can help lower the risk of infection in people because CSD is a zoonotic infection that is sustained and spread among cats by fleas. Handwashing after interacting with cats can also lower the danger because flea faeces can burrow into injured skin.

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Authors' contributions

Dr Patty Mopamboli Mboli conceptualized the case report and was the primary author responsible for drafting the manuscript. Benjamin Tenson Msopole was responsible for the taking all patient photographs and their subsequent incorporation into the manuscript as figures. Phillip Chisomo Nyambalo conducted the proofreading of the manuscript, ensuring accuracy and clarity and was responsible for the formatting and referencing.

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