Unilateral visual loss secondary to cat scratch disease in a healthy young man

Norfarizal Ashikin Abdullah, Umi Kalthum Md Noh*, Rona Asnida Nasaruddin, Ayesha Md Zain, Hanizasuriana Hashim, Jemaima Che Hamzah

Department of Ophthalmology, Universiti Kebangsaan Malaysia Medical Centre, Jalan Yaacob Latiff, 56000 Cheras, Kuala Lumpur, Malaysia

1. Introduction

Cat scratch disease (CSD) caused by Bartonella henselae (B. henselae) is one of the etiology for neuroretinitis[1]. Almost 33% of patients who had exposure to cats will have serologic evidence of previous B. henselae infection. During the initial stages, patients may present with lesion at the inoculation site, followed by painful lymphadenopathy of the draining lymph node. These presentations are usually accompanied by constitutional symptoms such as malaise, fever and myalgia which were common in CSD but uncommon in recurrence cases[2]. However, the diagnosis of CSD is not precluded by the absence of history of cat bite[3]. Although there are many species of Bartonella, B. henselae is a Gram negative bacillus known to be the major causative agent of CSD. This particular strain induce retinal infection with classical presentations of necrotizing granulomatous lesions seen typically in immunocompetent patients[4–6]. This case report illustrates a serologically proven case of CSD in an immunocompetent male which responded well to treatment.

2. Case report

A 36-year-old healthy young man had presented with gradual worsening of central vision in the left eye for a month. He had history of recurrent low grade fever that was associated with myalgia. He has three healthy and well kept cats at home. He sustained minor abrasion while handling
them. On examination, his visual acuity was 6/24 improved to 6/9 with pinhole in the right eye and hand movement in the left. Visual field test revealed left eye central scotoma. The left relative afferent pupillary defect was present. Result of anterior segment examination was quiet. The left eye fundus examination revealed presence of generalised hyperaemia and swelling of the optic disc with area of pre-retinal hemorrhage superior to the optic disc (Figure 1). There was presence of macula star with surrounding edematous area. Peripheral retina showed multiple choroiditis with subhyaloid hemorrhage at the inferotemporal arcade. There was no vasculitis or retinitis. The right anterior segment and fundus were normal. Result of systemic examination was unremarkable.

Indocyanine green revealed hyperfluorescence areas during choroidal filling phase followed by hypofluorescence during later stage, with evidence of generalised choroiditis (Figure 2). There was no retinal vasculitis. Optical coherent tomography showed significant macula edema with subretinal fluid accumulation (Figure 3). Full blood count was normal. Erythrocyte sedimentation rate was raised to 93 mm/h. Screening for tuberculosis was negative. Both immunoglobulin G (IgG) and immunoglobulin M (IgM) serology for B. henselae were raised (IgG 1:512 and IgM 1:96). The diagnosis of left neuroretinitis secondary to B. henselae was made.

Patient was treated with intravenous cefazidime 1 g three times daily for 1 week followed by oral rifampicin 300 mg once daily and oral doxycycline 100 mg twice daily for 6 weeks duration.

After 1 month of treatment, the visual acuity improved to 6/9 with resolving macula star and optic disc edema. Three months later, his vision returned back to baseline of 6/6 and N6, with complete resolution of macular edema and choroiditis.

3. Discussion

Neuroretinitis is an uncommon inflammatory disorder of the optic nerve head and retina with formation of macula star following resolution of the peri-papillary exudates[1]. Neuroretinitis can be of infective causes such as CSD, Lyme disease, tuberculosis, mumps, syphilis, toxoplasmosis and toxocariasis, inflammation such as sarcoidosis and periarteritis nodosa as well as retinal related pathology such as idiopathic retinal vasculitis, aneurysms, and neuroretinitis syndrome and Parry–Romberg syndrome.

Neuroretinitis is associated with inflammation of the vascular walls at optic discs, resulting in fluid exudation into the surrounding retina. Eventually, lipid–rich exudation on the form of macular star develops following the configuration of the outer plexiform layer at the posterior pole[2]. This becomes apparent at approximately 2 weeks after the development of optic disc swelling and resolution of serous fluid[3]. The dissapearance of the macula exudates takes about few months which resulted in retinal pigment epithelium defect[3].

Unilateral conjunctivitis associated with lymphadenopathy of preauricular nodes, termed Parinaud oculoglandular syndrome is the commonest ocular manifestation of CSD. Despite being atypical to systemic manifestations of the CSD, infections of the retinal and choroidal layer of the eye may lead to devastating consequences due to significant visual impairment[4,5]. Neuroretinitis secondary to CSD is usually a clinical diagnosis especially in patient with history of contact with cats. However, positive serology evidence of B. henselae helps in supporting the diagnosis and eventually directing
the correct treatment. Laboratory methods for detection of B. henselae include ELISA, PCR and immunofluorescence assay. IgM level of B. henselae using immunofluorescence assay and ELISA was highly specific compared to IgG level in order to make diagnosis of CSD. In immunocompetent patients, the serologic test yield 90% specificity and sensitivity while in immunocompetent patients it is only 70%[6]. Observation in one study found that patients with optic disc oedema associated with peripapillary serous retinal detachment and macula star showed high antibody titre for B. henselae[7].

CSD usually follows a benign and self-limiting course in the immunocompetent patients, despite no treatment with antibiotic[8]. The course of disease may take two to six months to resolve on its own. Those who developed neuroretinitis have been observed to gain back their baseline visual acuity and resolution of macular oedema even without treatment; although the clinical improvements may take a longer duration[9]. Hence, the institution of antibiotic is mainly to speed up the resolutions of disease, and does not alter the final outcome in healthy individuals. However, it has been shown to induce dramatic response in the immunocompromised patients. Doxycycline is the antibiotic of choice because of its ability to penetrate the blood-ocular and blood–brain barrier, with resulting good concentration in the eye, and is usually constituted up to 4 weeks in affected healthy patients. In the immunocompromised, the course of treatment may necessarily take up to 4 months to ensure complete resolution of disease. Other biological agents that have been shown to be active against B. henselae includes other aminoglycoside group such as gentamycin and erythromycin, and also of sulphur drug such as the combination of trimethoprim and sulphamethoxazole. These alternatives provide a good substitute for patients with allergies or contraindications to certain group of medications.

The final visual prognosis of CSD affecting the eye is excellent. Most patients regain useful vision, however, some may still retain residual and mild blurring of vision, optic disc pallor or developed consequent retinal pigmentary changes.

As a conclusion, even though neuroretinitis is an atypical presentation of CSD, its presence may cause marked visual deterioration. Ophthalmologist should be aware of neuroretinitis in a patient presented with reduced vision with optic disc edema and macula star especially after history of cat’s bite or scratch. Although it is self limiting in immunocompetent patient, with antibiotic treatment, it may hasten the visual recovery and speed up the resolution of the disease.

Conflict of interest statement

We declare that we have no conflict of interest.

Related reports

There are not many cases of neuroretinitis secondary to cat scratch published. This is one of the good presentations with properly investigation. This case was treated accordingly and having good outcome.

Applications

It is important to share the clinical presentation and the management of this case. All patient presented with neuroretinitis should rule out B. henselae. Proper treatment will hasten the recovery and prevent further complication.

Peer review

Overall, this is a good case report. The topic is interesting and well written. It is important to share the clinical presentation and the management of this case. Proper treatment will hasten the recovery and prevent further complication.

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