CASE REPORT

A challenging differential diagnosis of optic neuropathy in ED: CSD

Rohat Ak, Fatih Doganay, Ebru Unal Akoglu, Tuba Cimilli Ozturk

Department of Emergency Medicine, Fatih Sultan Mehmet Education and Research Hospital, Istanbul, Turkey

Correspondence to
Dr Ebru Unal Akoglu,
ebryunal@gmail.com

Accepted 27 May 2015

SUMMARY

Optic neuropathy due to cat scratch disease (CSD) is a rare occurrence associated with macular star formation and is characterised by sudden mostly unilateral painless loss of vision. The aetiological agent in CSD is *Bartonella henselae*. Ocular complications present in up to 10% of patients and include neuroretinitis, conjunctivitis or uveitis. Ocular bartonellosis is usually self-limited. A case of a man patient with neuroretinitis caused by *B. henselae* is reported.

BACKGROUND

Optic neuropathy refers to a clinical disorder characterised by sudden-to-chronic loss of vision in one or both eyes due to optic nerve dysfunction that might be idiopathic, ischaemic, primary demyelinating, infectious or inflammatory in aetiology.¹ Optic neuropathy due to cat scratch disease (CSD) is a relatively infrequent occurrence caused by *Bartonella henselae*. Other infectious causes include syphilis, Lyme disease, viruses and diffuse unilateral subacute neuroretinitis. CSD has been presented as a syndrome of regional lymphadenopathy and fever with some patients having a skin lesion at the site of inoculation.^{2–3} *B. henselae*, which is intracellular, aerobic, oxidase-negative and Gram-negative bacilli, was identified as the aetiological agent.² Cats are the major reservoir for *B. henselae*. The cat flea, saliva, scratch or bite are the major routes of transmission. Once transmitted to humans, following the scratch or bite of a kitten or adult cat, *B. henselae* invades haematopoietic progenitor cells. The response-to-infection depends on the immune status of the host. The typical CSD is characterised by lymphadenopathy with fever, benign and self-limited, with no other signs or symptoms.⁴ Other manifestations may present in a wide spectrum, but fortunately are seen less commonly (hepatosplenomegaly, granulomatous hepatitis, osteomyelitis, encephalitis, glomerulonephritis, endocarditis and pneumonia). After the lymphatic system, the eye is the most commonly affected organ.³ Ocular involvement occurs in 5–10% of cases and can be subclinical, and visual symptoms may not be a common presenting symptom.⁵ The most common ocular manifestations are Parinaud syndrome, which consists of fever, regional lymphadenopathy and follicular conjunctivitis, followed by neuroretinitis.² Neuroretinitis typically presents with sudden loss of vision after a febrile prodromal illness. Optic disc swelling and macular star formation can be diagnosed by funduscopic examination up to 2–4 weeks period after the infection.⁶ We describe a case of ocular bartonellosis in an adolescent man

patient with unilateral sudden painless vision loss and optic neuropathy.

CASE PRESENTATION

We report a case of a 25-year-old man who presented to the emergency department with acute loss of vision in his right eye. Vital signs were as follows: fever: 36.5°C, SpO₂: 99%, pulse: 107 bpm and blood pressure: 135/61 mm Hg. He had neither a trauma nor an existing disease history. He had a history of fever 1 week prior to the eye symptoms. On examination, his pupils were equal and reactive to light; extraocular muscle movements were normal. Measured visual acuity was 1/20 in the right eye with a relative afferent pupillary defect and 20/20 in the left eye. Other systemic and neurological examination was normal. Chest X-ray, complete blood count, C reactive protein and erythrocyte sedimentation rate were normal. Cranial CT and MRI were unremarkable. The patient was referred to ophthalmology for consultation. Ocular examination showed papillary oedema. Intraocular pressures were normal. On funduscopic examination, optic disk swelling associated with macular oedema and splinter haemorrhages were revealed; peripheral retinal examination was normal. There was an initial suspicion for optic neuropathy and intravenous methylprednisolone therapy was started. Serological studies for cytomegalovirus, HIV and antinuclear antibodies were negative. The serological titres for anti-*B. henselae* were found to be 1:100. The patient was started on oral tetracycline and rifampicin for 4 weeks. At follow-up at 1 month, there was improvement of visual acuity.

DISCUSSION

The classic presentation of CSD is as a febrile illness, in conjunction with a history of feline exposure, lymphadenopathy and cutaneous lesions. Fever and lymphadenopathy have been considered diagnostic criteria of CSD.² Cases of CSD neuroretinitis lacking lymphadenopathy have been reported.⁷ Tsuneoka and Tsukahara found that up to 16% of patients serologically diagnosed as having CSD presented with no lymphadenopathy. In total, 3 of 30 such individuals were also afebrile. However, unlike the cases reported here, two of these patients had symptoms suggestive of systemic illness.⁸ A large retrospective study by Carithers³ demonstrated that as many as 41% of patients with CSD may have no documented fever. Leber stellate neuroretinitis is a kind of CSD that is characterised by unilateral painless visual loss associated with optic disc swelling and macula star exudates.

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To cite: Ak R, Doganay F, Akoglu EU, et al. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2015-210252

Munteanu and Berghian reported two cases of Leber stellate neuroretinitis, represented with decreased visual acuity, 2–3 weeks after a flu-like syndrome. Typical optic disc swelling and infectious process before visual loss were described in both cases, as in our case.⁹ Serological tests are the cornerstone of the differential diagnosis.

In the emergency department, optic neuropathy can be due to various causes that show significant overlap in clinical presentation and clinical examination.¹ The diagnosis of the underlying cause of optic neuropathy should be established as soon as possible. The optic neuritis treatment trial has shown that intravenous administration of high-dose steroids decreases the risk of progression of optic neuropathy to multiple sclerosis within 2 years of follow-up. Intravenous methylprednisolone does not change the long-term prognosis of optic neuritis, but it does hasten short-term visual recovery. Corticosteroid use in CSD with eye involvement has had mixed results. There was good response in multiple studies¹⁰ but, conversely, recent case reports have found no improvement in symptoms with corticosteroids.² The diagnosis of infection is clinical. Laboratory evaluation is usually applied to confirm the initial suspicion.² On MRI, the characteristic optic-globe nerve junction is specific for *B. henselae* as cause of optic neuropathy. After the resolution period of the macular exudates, which usually takes months,

patients may experience abnormal colour vision, subnormal contrast sensitivity and decreased visual acuity.²

In healthy individuals with ocular CSD, the treatment is still controversial since the progression of full recovery occurs in a few months. Systemic antibiotics seem to shorten the disease duration and also hasten visual recovery.¹¹ Alternatives to azithromycin include rifampicin, ciprofloxacin, gentamicin and trimethoprim-sulfamethoxazole, in order of increased effectiveness.² In most cases, the prognosis is good, but there may be residual loss of visual acuity and retinal pigmentary changes.

Contributors RA and FD attended in the progression of diagnosis, had the idea for the manuscript, collected the data and patient information, and followed up the conclusion of the case. TCO and EUA supervised the case and reviewed the article before submission. All authors were involved in the writing process and approved the final version of the article before submission.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- In the emergency department, sudden painless unilateral loss of vision in young adults has a wide spectrum of differential diagnoses. Optic neuritis, as a presenting sign of multiple sclerosis or cerebrovascular events, is usually considered as the initial diagnosis especially in non-traumatic young adults. Cat scratch disease is usually diagnosed based on history and accompanying symptoms. The diagnosis should be confirmed by serological tests. Cat scratch disease neuroretinitis is one of the differential diagnoses of unilateral central scotoma and should be considered even though there are no prodromal signs.

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