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CASE REPORT

Cat Scratch Neuroretinitis

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Abstract

Cat scratch disease (CSD) is caused by a gram-negative bacterium, *Bartonella henselae*. This uncommon disease is believed to be transmitted by a cat scratch or bite, when the bacterium is present on the cat's claw or resides in the oral cavity. There are approximately 22,000 cases of CSD diagnosed in the United States annually. Neuroretinitis (NR), which occurs in 1%-2% of CSD cases, is characterized by acute vision loss, optic disc edema, and a macular star. Diagnosis can be aided by fundus examination, optical coherence tomography (OCT), fluorescein angiography (IVFA), and serological testing for *B. henselae* infection. Cat scratch disease is usually self-limiting; however, oral antibiotics may shorten the duration of the disease.

The following case describes a dramatic presentation of a 13-year-old Hispanic female diagnosed with CSNR. The patient reported an earlier skin rash and lymphadenopathy and presented with monocular vision loss. Examination revealed optic disc edema and a macular star in the left eye. The diagnosis was confirmed by positive serology for *B. henselae*. She was treated with trimethoprim-sulfamethoxazole (Bactrim 400 mg/80 mg tablets) for three weeks. After six weeks, her systemic signs and symptoms resolved. Her visual acuity recovered but visual distortion remained.

Keywords

Bartonella henselae, Cat scratch disease, Macular star, Neuroretinitis, Optic disc edem

Introduction

Neuroretinitis (NR), secondary to cat scratch disease (CSD), is typically a self-limiting condition caused by an infectious and inflammatory reaction of the optic nerve, followed by the formation of a macular star [1,2]. The gram-negative bacillus, *Bartonella henselae*, the primary etiological cause of CSD, is transmitted to humans through scratches, bites, or saliva from an infected cat. Direct exposure to *Bartonella henselae* can cause optic disc inflammation, [3,4] with infiltration of lipid-rich fluid though the prelaminar optic disc vasculature. Once this fluid migrates into the outer plexiform layer around the macula, the exudates precipitate and form a partial or complete stellate pattern [5,6].

Systemic signs and symptoms usually precede the ocular manifestations and may include rashes, regional lymphadenopathy, fever, headache, nausea, anorexia, vomiting and sore throat. Other ophthalmic signs include reduced visual acuity, mild color defects, and a mild to moderate relative afferent pupillary defect (RAPD). While there is no race predilection for CSD, males are slightly more affected than females (60% vs. 40%), and children and young adults are at an increased risk of infection. A history of exposure to cats, especially kittens, was reported in over 90% of cases.

Ancillary testing for CSD includes serology for *B. henselae*, optical coherence tomography (OCT), visual fields, and fluorescein angiography [1]. Treatment for CSD is controversial due to its self-limiting nature [1,7,8]. However, studies have shown that oral antibiotics may shorten the recovery period, especially in moderate to severe cases [9].

Case Report

A 13-year-old Hispanic female was in her usual state of good health, until she presented with a chief complaint of gradual, but extremely blurry vision in the left eye. The symptoms began one week earlier and the vision had progressively worsened since onset. Five days prior, the patient went to an emergency department (ED), where op-

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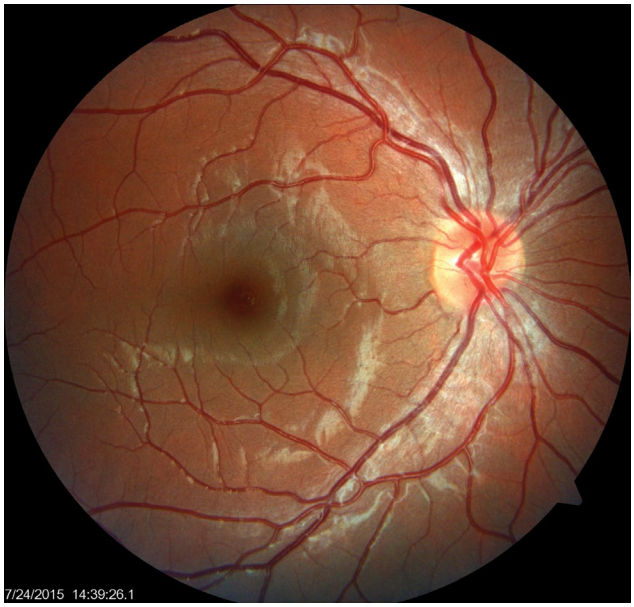

Figure 1a: Fundus photograph OD showing no abnormalities.

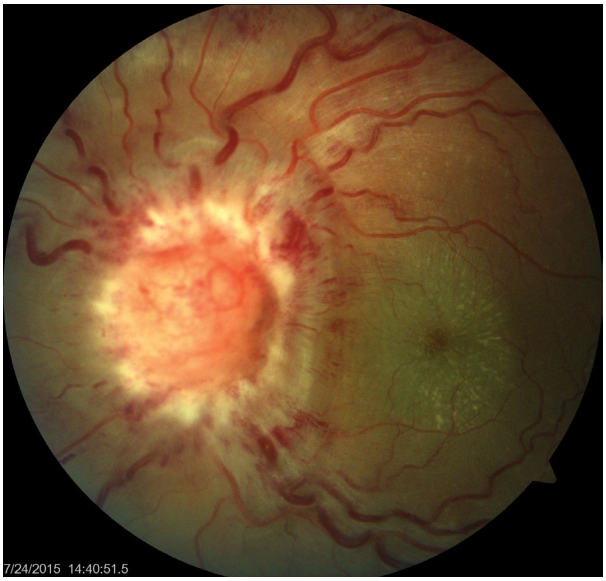

Figure 1b: Fundus photograph OS documents severe swelling of the optic nerve with macular exudates.

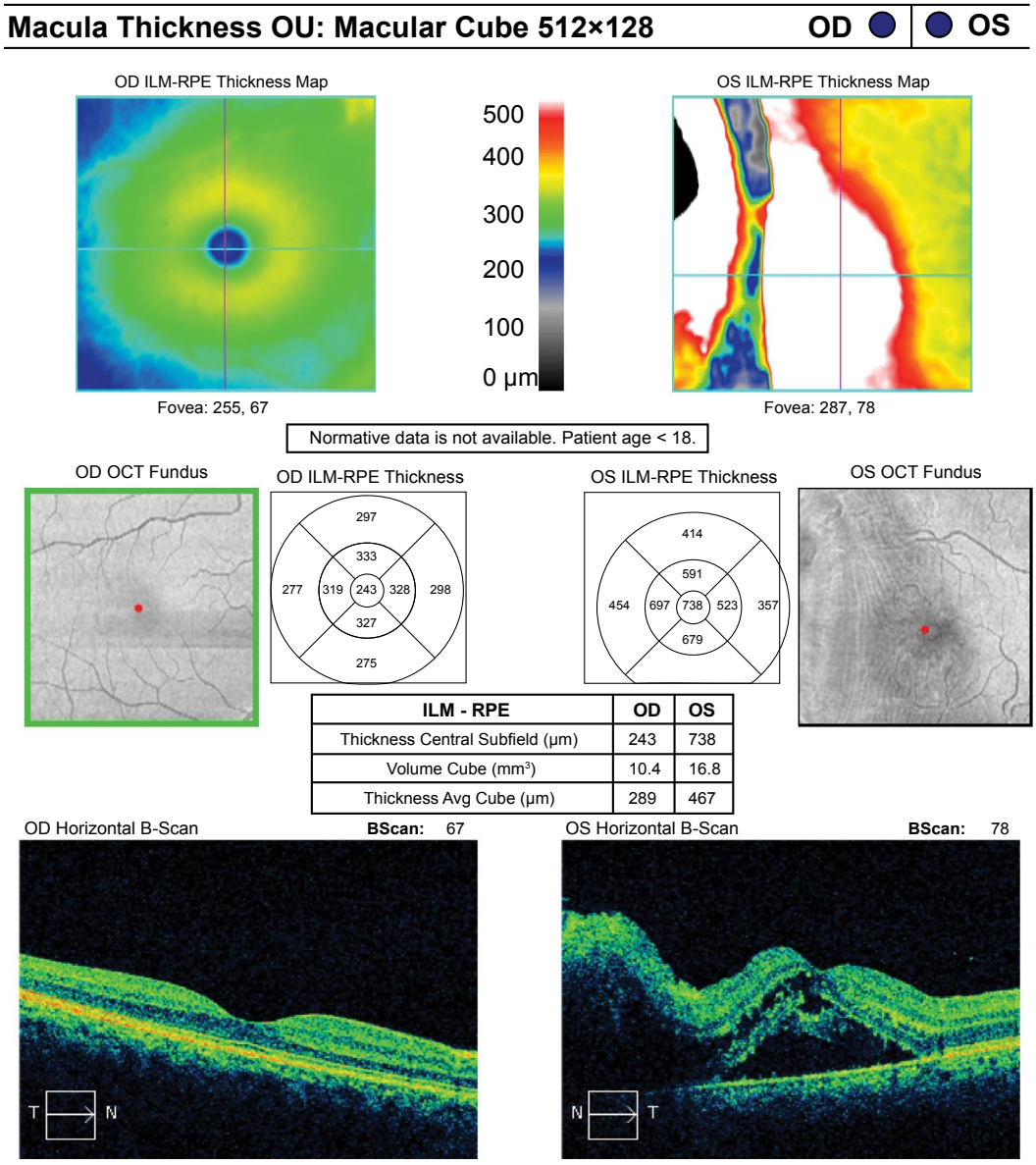

Figure 2: SD-OCT reveals no abnormalities of the right eye but demonstrates extensive macular thickness, due to intraretinal edema and a large serous detachment in the left eye.

tic nerve swelling was detected in the left eye. Magnetic resonance imaging (MRI) of the brain, performed at the emergency visit, was within normal limits. The patient was then referred to a neuro-ophthalmologist for further consultation.

At the consult visit, the patient denied any associated symptoms or recent travels outside her hometown. Her father reported no relevant medical or ocular history and her family history was non-contributory. She took no medications and had no known drug allergies, including sulfa derivatives. She was a seventh grader doing well in school and had three dogs and one cat in the home.

The patient was alert, oriented and in no apparent distress; her vital signs were normal. Her best corrected visual acuity was OD: 20/20-2 and OS: 20/200+1. Extra ocular motility and cover testing were within normal limits, with no pain on eye movement. Confrontation visual fields demonstrated superior-temporal and inferior defects in the left eye, which were supported by the findings on Amsler grid testing. A 1.2 log unit relative afferent pupillary defect (RAPD) was found in the left eye. While the color vision in the right eye was normal (14/14 plates), a mild color vision defect (12/14 plates) was noted in the left eye, using the Hardy-Rand-Rittler (HRR) pseudo-isochromatic test.

Both anterior and posterior segments were normal in the right eye (Figure 1a); however, biomicroscopy revealed 1+ flare in the anterior chamber and 1+ cell in the anterior vitreous of the left eye. Fundus exam of the left eye showed macular edema, a stellate exudative maculopathy, venous tortuosity, and grade IV optic disc edema (ODE), equivalent to a Modified Frisén Scale of Papilledema (Figure 1b). Cirrus optical coherence tomography (Carl Zeiss Meditec, Dublin Calif) of the left eye documented cystoid macular edema (CME) and a serous macular detachment. The central macular region in the left eye (738 um) was three times that of the right eye (243 um) (Figure 2). Humphrey 30-2 SITA-Fast visual field (VF), with a size III stimulus, showed a nasal hemisphere depression in the right eye, most likely due to low test reliability. Humphrey 30-2 FASTPAC VF, of the left eye, using a size V target, revealed a significantly enlarged blind spot, a superior- temporal quadrantanopia, and a mild to moderate inferior nasal step (Figure 3).

Based on clinical findings, neuroretinitis (NR) was the tentative diagnosis, with optic neuritis and ischemic optic neuropathy as differentials. Since the patient reported having multiple dogs and a cat at home, additional history was obtained. The patient stated that the kitten was a new stray and was rescued from the woods one month prior. About a week later, the cat scratched her arms and she subsequently developed a skin rash and a tender anterior cervical lymphadenopathy. Three weeks later, she noted blurry vision in her left eye, which prompted the emergency room visit.

Due to the new information, the patient was tested for *Bartonella* antibodies. The immunoglobulin G (IgG) titer for *B. henselae* was positive, with a ratio of 1:1280. The serologic tests confirmed the diagnosis of NR secondary to cat scratch disease (CSD), and sulfamethoxazole/trimethoprim (Bactrim 400 mg/80 mg tablets) was prescribed, one tablet every 12 hours for three weeks. She was asked to return in 4-6 weeks.

The patient returned six weeks later and stated that the vision in the left eye was much better, but that mild visual distortion remained. On testing, the VA in the left eye had improved from 20/200+1 to 20/20-2. The macular edema had decreased three fold in comparison to the initial OCT scan-central macular thickness was reduced from 738 um to 202 um (Figure 4). Although the sub-reti-

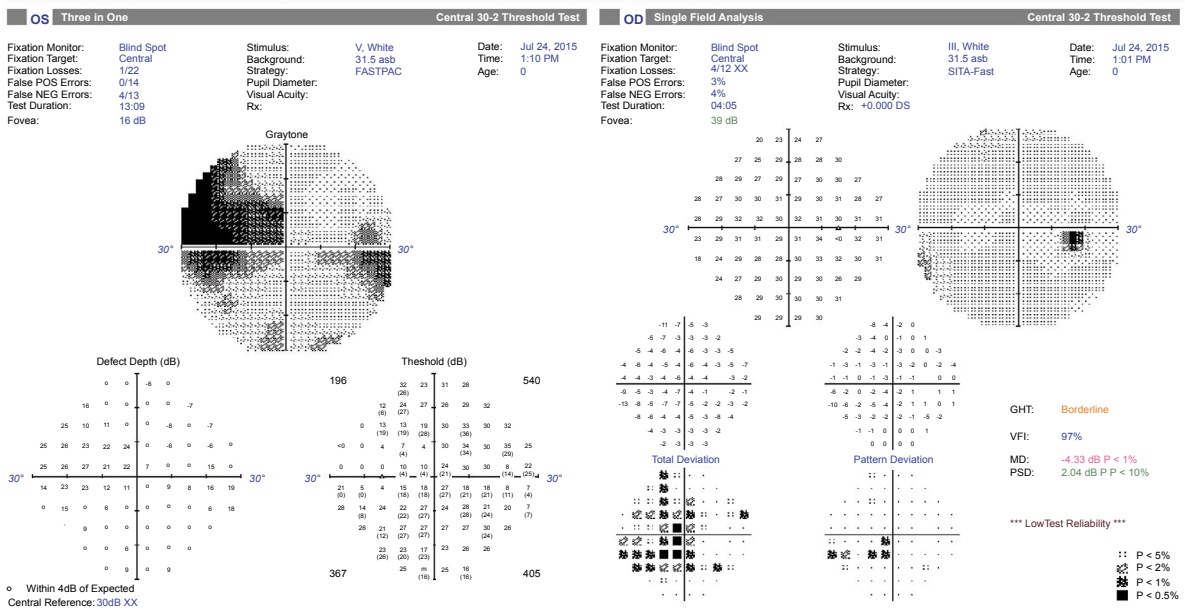

Figure 3: HVF of the right eye, while demonstrating a mild nasal depression, was essentially normal, with a VFI of 97%. The left eye, even with a size V stimulates, had a dense temporal field loss, with an inferior nasal step.

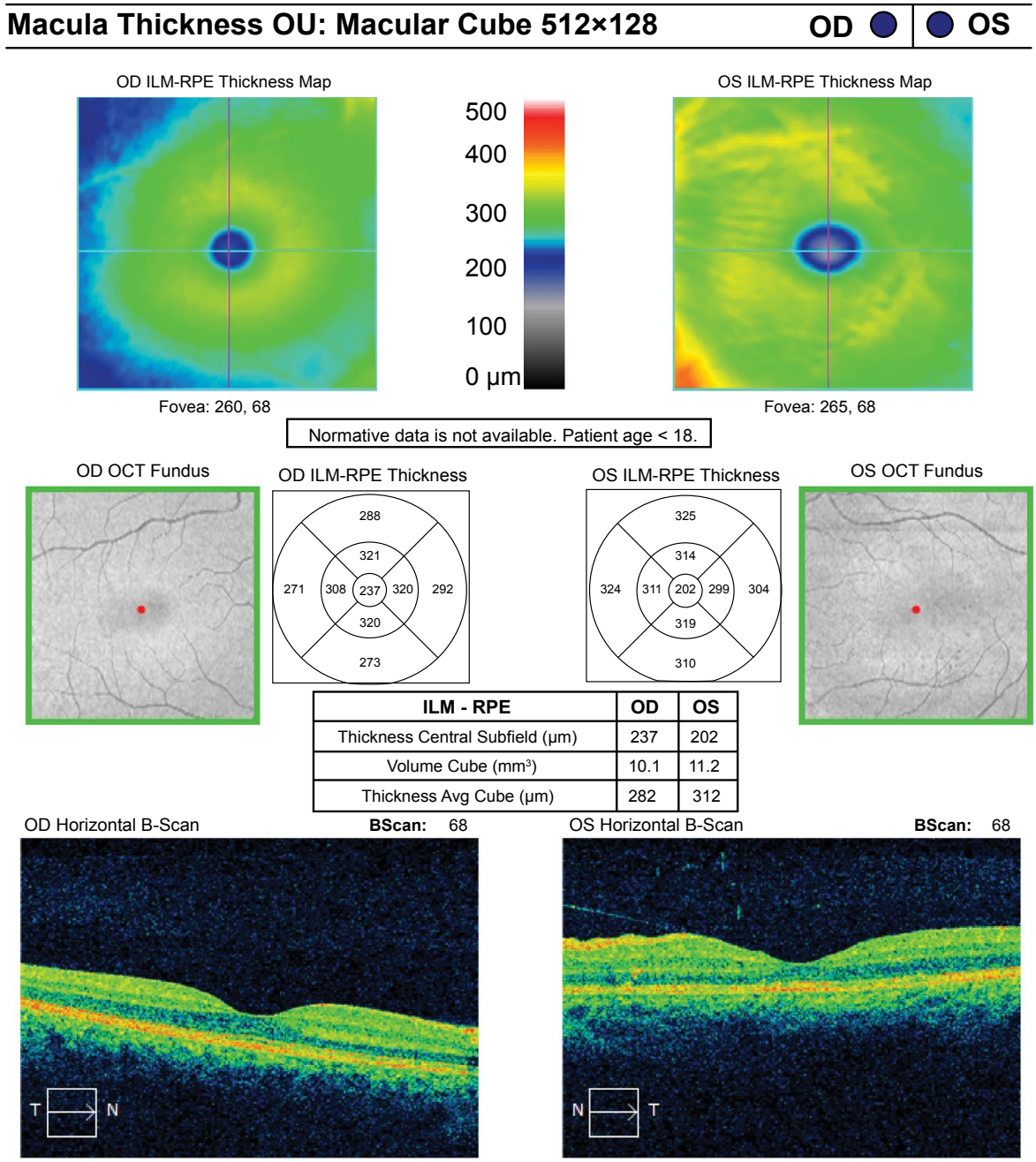

Figure 4: SD-OCT of the right eye remained stable; the macular contour and thickness in the left eye was much improved.

nal fluid had resolved and the macula appeared intact, residual stellate exudates were still present on ophthalmoscopy (Figure 5a), while the right eye remained unchanged (Figure 5b). The patient showed improvement on visual field testing, using a size III target in the left eye. However, an inferior altitudinal defect, with a scattered superior depression was found and a slightly enlarged blind spot remained (Figure 6). A 0.9 log unit RAPD was measured and color vision testing showed a one plate improvement, compared to the consult exam. Improved, but residual metamorphopsia was confirmed by Amsler grid. Repeat serology continued to show an elevated *B. henselae* IgG titer (1:2560). Overall, the patient demonstrated considerable improvement; consequently, she was advised to return in two months for a scheduled follow-up, or as needed if any new concerns should manifest.

Discussion

Neuroretinitis (NR) secondary to cat scratch disease (CSD), now referred to as cat scratch neuroretinitis (CSNR), is one of the three most common forms of NR (the other being idiopathic NR and recurrent NR) [1]. There are an estimated 22,000 new cases of CSD reported yearly in the U.S. (6.6 cases per 100,000) and CSNR is the most common form of NR associated with an infectious agent [2,10-12]. The primary etiological organism in CSD, *B. henselae,* can be associated with ocular complications such as NR, Parinaud oculoglandular syndrome (POGS), and focal retinochoroiditis [3].

Direct bacterial invasion, or autoimmune response against the optic nerve, may cause optic nerve vascular inflammation, with a secondary inflammatory reaction in the nerve

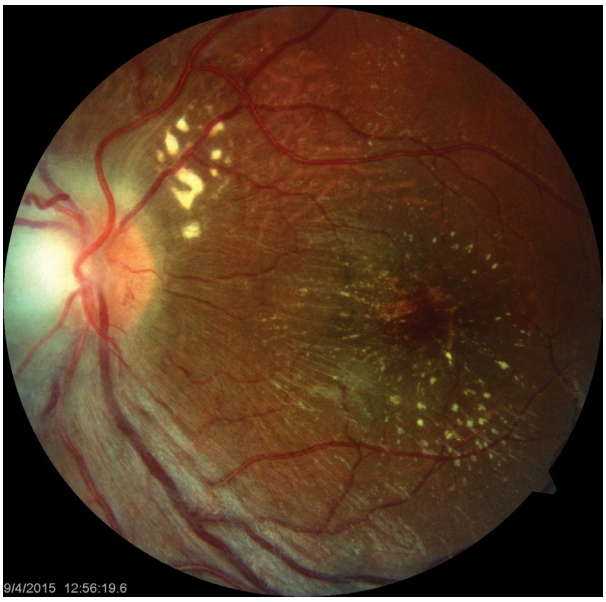

Figure 5a: Fundus photograph OS showing reduced swelling of the optic nerve with residual macular exudates.

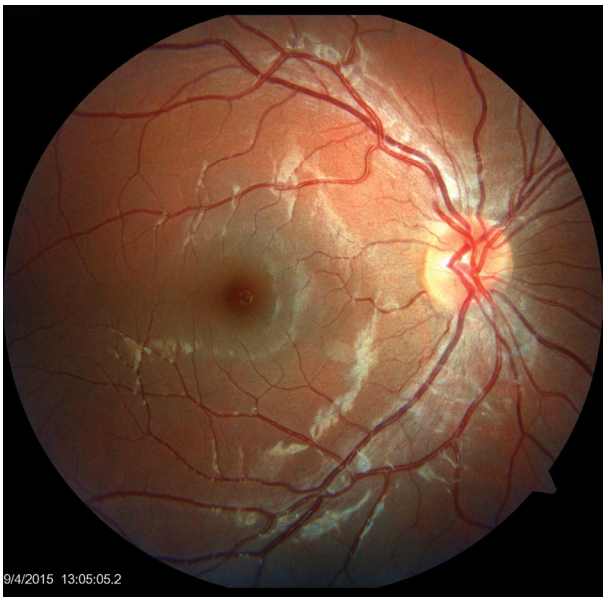

Figure 5b: Fundus photograph OD demonstrating normal findings.

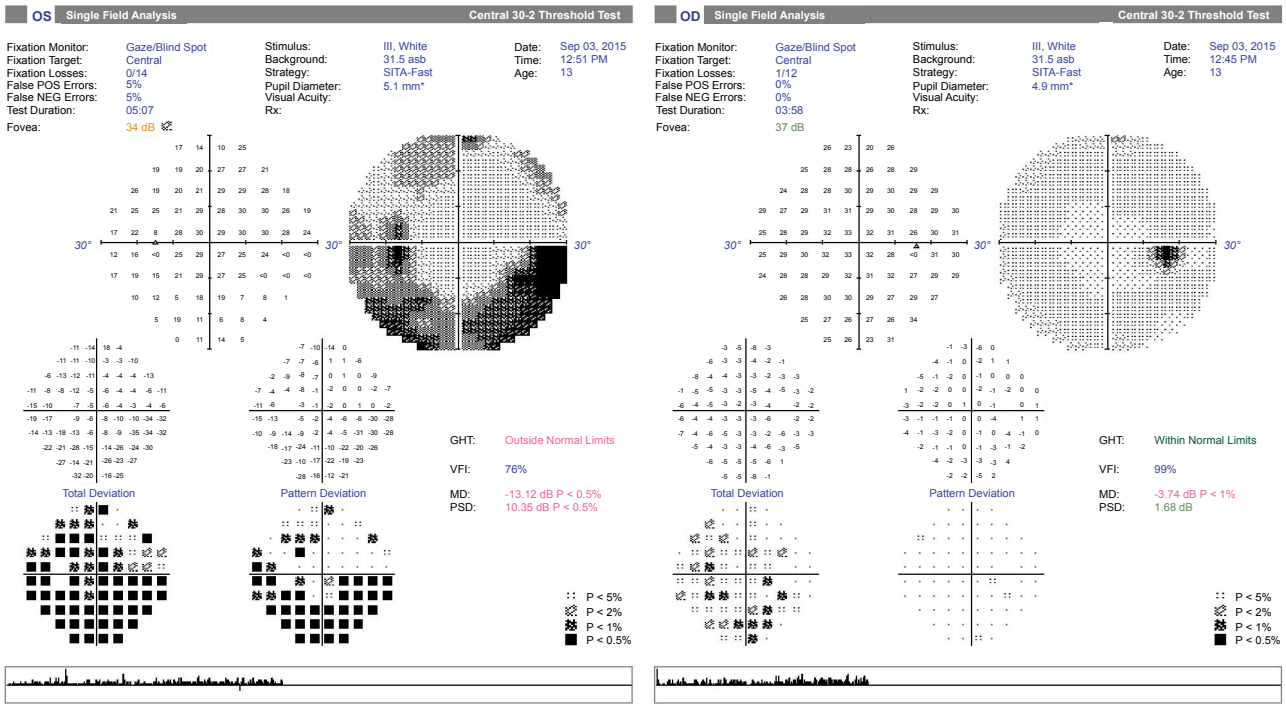

Figure 6: Reliable HVF, 6 weeks after presentation, disclosed a persistent inferior altitudinal defect in the left eye. The right eye remained within normal limits.

fiber layer of the retina [1]. The first case of NR was reported in 1916 by Theodor Leber, a German ophthalmologist, who described a condition with vision loss, optic disc edema (ODE) and stellate maculopathy [13]. However, the term NR was not employed until 1977, when Gass proved the temporal sequence of the leakage sites by using fluorescence angiography (FA) [14]. The fact that optic nerve edema precedes the macular star (MS), has been confirmed in many case studies by combining FA and OCT imaging [15,16]. As OCT is non-invasive, it has become the standard means to provide reliable evaluation and management of CSNR [1,17-22].

The diagnosis of CSD or CSNR relies on known cat or flea exposure, lymphadenopathy, and a positive *B. henselae* titer [3,10]. In the patient reported, a skin rash and lymphadenopathy occurred prior to the ocular symptoms and consequently, were not documented by a medical professional. Another common finding in CSD, fever, was not manifest in this case [19]. Patients with CSNR can sometimes report ocular discomfort and it is important that other neurological conditions, where pain is more common, such as neuritis and neuromyelitis optica (NMO) be excluded [23,24]. Unlike optic neuritis and NMO, pain occurs in only in 25% of NR cases and is usually mild in nature [1].

In our patient, no pain was reported or associated with eye movement. In addition, the presence of a macular star was critical in the differential diagnosis, as this finding is atypical in demyelinating conditions.

Catch scratch disease is self-limiting, [1,2] thus treatment has been controversial [7,8]. Oral antibiotics are more likely to speed recovery, [9] if specific antigens are identified. It is also recommended to treat complicated cases of CSD, when other organ systems or atypical presentations are involved [7]. Although doxycycline and ciprofloxacin have shown efficacy in treating CSNR, [1,25,26] they were not prescribed due to the potential adverse effects in a young patient. According to Purvin, et al. [1] sulfamethoxazole/trimethoprim, or azithromycin are suggested to treat young children or adolescents with CSNR; therefore, Bactrim was prescribed in this case.

Studies have shown that CSNR has an excellent prognosis. Final visual acuity improved in almost all reported cases, with 93% of patients recovering to 20/40 or better, with an average of 7.7 lines gained [1]. Our patient showed a significant improvement, from 20/200+1 to 20/20-2 in six weeks. By contrast, the visual fields showed a slower recovery, with a dense inferior altitudinal scotoma seen at the follow up visit. This defect was probably due to the severe swelling of the optic disc, which will be monitored in follow up visits.

Conclusion

With a presentation of optic disc edema and a macular star (ODEMS), combined with a history of a cat scratch or contact with cats, CSNR should be considered in the differential diagnoses. Ancillary tests, such as serology, OCT, and VF's help confirm the etiology, stage the severity of the disease, and assist in following patients with CSNR to resolution. As in the case presented, it is often beneficial for patients with severe symptoms and significant clinical signs to initiate oral antibiotics and therefore, shorten the duration and speed the resolution of the disease.

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