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Case Report: Bartonella quintana-associated Neuroretinitis

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Case Report: Bartonella quintana-associated Neuroretinitis

Abstract

Background: Neuroretinitis is a self-limiting condition which typically causes monocular vision loss with good potential for visual recovery. It may be idiopathic or associated with infectious or inflammatory conditions which can carry systemic implications. Neuroretinitis classically presents with disc edema followed by development of a macular star pattern of exudates. It is most commonly attributed to Cat Scratch Disease, or *Bartonella henselae* infection. However, there have been few published reports of *Bartonella quintana* associated neuroretinitis.

Case Report: A 60-year-old patient presented with unilateral vision loss preceded by flu-like illness. The patient had exposure to a recently adopted cat. Fundus examination revealed a stellate pattern of exudates in the macula of the affected eye with questionable sectoral optic disc edema. Serologic testing revealed a positive titer for *Bartonella quintana* IgG antibody, but negative *Bartonella henselae* testing. This report highlights a rare case of neuroretinitis related to *B. quintana* infection.

Conclusion: While neuroretinitis is generally considered to be self-limiting, it may be related to a systemic infection, such as *B. henselae* and *B. quintana*. Both *B. henselae* and *B. quintana* may present with ocular findings in the setting of nonspecific systemic symptoms. A thorough ophthalmic examination and history in conjunction with serologic testing can help to establish a diagnosis and prompt consideration of further testing or treatment for concurrent systemic disease.

Keywords

Bartonella quintana, cat scratch, macular star, neuroretinitis

Introduction

A 60-year-old African American woman presented with a complaint of a "big patch" over her left eye. She described a constant, stationary dark spot in the center of her vision that had been present for the past 6 weeks. She denied ocular pain, flashes, floaters, or transient vision loss. She reported noticing mild, gradual improvement in her vision over the past few weeks. Ocular history was unremarkable. Systemic history was remarkable only for hypertension and hypothyroidism. Systemic medications included amlodipine and levothyroxine. She had no known drug allergies. She reported excellent compliance with systemic medications and blood pressure was measured as 140/90mm Hg right arm sitting.

Upon further questioning, she reported suffering from a flu-like illness a few weeks prior, with frequent headaches, chills, and fatigue. She adopted a cat approximately one month prior to the onset of her systemic illness. She could not recall any specific licks, bites, or scratches. She did not seek care for her flu-like illness or vision earlier because she did not feel well enough to leave her home and assumed the illness would run its course. At the time of her eye examination, she reported less frequent headaches and improvement in her fatigue. She was no longer experiencing any chills or fevers.

Best corrected visual acuities were 20/20 in the right eye and 20/40 in the left eye with pinhole. She exhibited normal color vision OD and OS by Ishihara and full visual fields by confrontation in both eyes. There was blur, greater temporally, on facial Amsler with the left eye. Pupils were isochoric in bright and dim illuminations. A 0.3 log unit relative afferent pupillary defect (APD) was noted in the left eye with corresponding 40% subjective red desaturation. Ocular motilities were full bilaterally. Anterior segment health was remarkable only for trace bilateral nuclear sclerosis. Her anterior chambers were deep and quiet. Intraocular pressures were 11mm Hg OU as measured with Goldmann Applanation Tonometry.

Dilated fundus examination revealed large optic discs with .8/.8 C/D OD and .75/.75 C/D OS. There was apparent sectoral edema of the inferior temporal aspect of the left optic disc. The left eye exhibited a partial stellate pattern of exudates in the macular region (Figure 1).

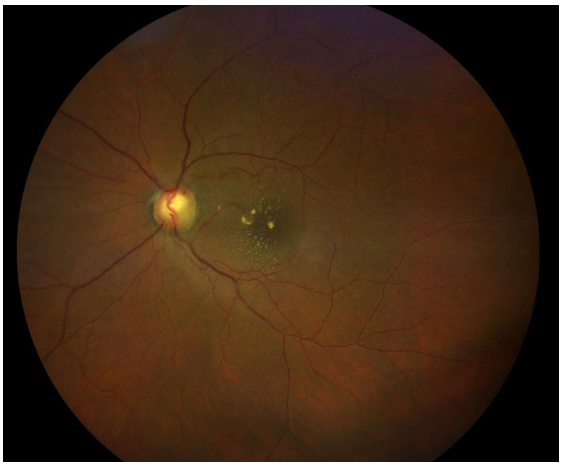

Figure 1: Color fundus photograph of the left eye with partial stellate pattern of exudates within the macular region. Note appearance of inferior temporal optic disc, suspected to be consistent with likely resolving optic disc edema.

The right eye macula was unremarkable with no exudates (Figure 2).

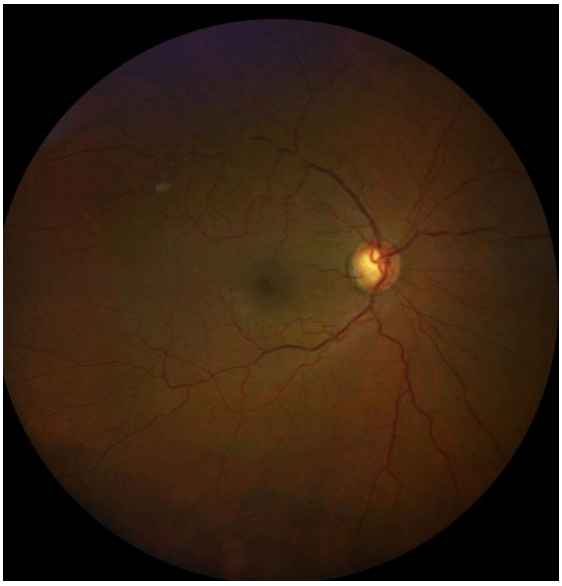

Figure 2: Color fundus photo of the right eye demonstrating a large optic disc with large cupping without retinopathy.

Optical coherence tomography (OCT) of the left macula (512x128) revealed subretinal fluid beneath the fovea, a few subretinal hyperreflective areas, as well as perifoveal intraretinal exudates nasal to the fovea (Figure 3).

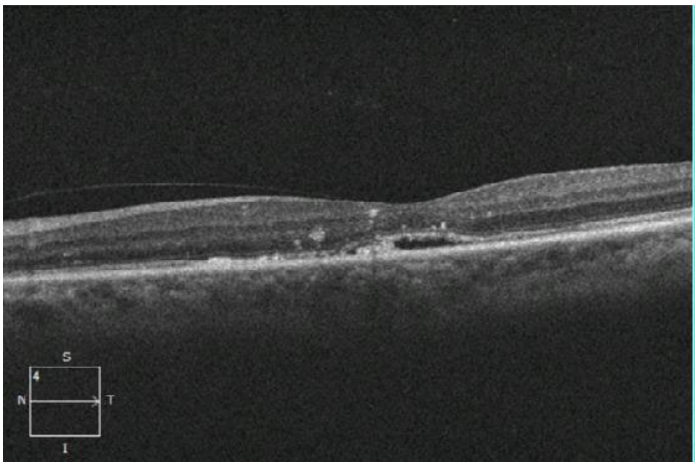

Figure 3: Cross-section of Macular OCT (512x128) of the left eye demonstrating subretinal fluid beneath the fovea, hyperreflective areas consistent with exudates primarily in the outer plexiform layer, and subretinal hyperreflective areas consistent with vitelliform deposits.

The vitreous was clear with no cells in either eye. The retinal arterioles were mildly attenuated in both eyes. The peripheral retinal examination was unremarkable in both eyes.

Tentative diagnosis at this time was neuroretinitis based on the presence of an APD, a macular star, and questionable inferior temporal sectoral disc edema. We did not have previous records; therefore, we were unable to assess for interval change in the disc appearance to confirm the presence or absence of optic disc edema. Regardless, optic nerve involvement was suggested by the presence of the APD. Differential diagnoses also included branch retinal vein occlusion (BRVO) and hypertensive retinopathy. The preceding systemic symptoms along with the lack of retinal hemorrhages or cotton wool spots in either eye made these differentials less likely.

We recommended prompt lab testing to assess for infectious or inflammatory causes of neuroretinitis given her preceding flu-like illness. We specifically ordered *Bartonella henselae* antibodies with reflex titers, *Bartonella quintana* antibodies with reflex titers, Lyme titer, ACE, RPR, FTA-ABS and ANA (see Table 1). In this case, we chose a targeted work-up and did not test for all potential causes of neuroretinitis. We specifically left out toxoplasmosis due to the absence of typical clinical features such as vitritis. We were not highly suspicious of Rocky Mountain Spotted Fever due to geographic area and presentation during winter months. We asked the patient to return for follow-up and monitoring in 1 month, or sooner if she noted any worsening of her vision.

Etiology	**Testing***
Cat-Scratch Disease/ Bartonellosis	*Bartonella henselae* IgG, IgM *Bartonella quintana* IgG, IgM
Lyme Disease	Lyme Titer
Syphilis	FTA-ABS, RPR
Tuberculosis	PPD, Chest X-ray, Quantiferon Gold
Sarcoidosis	ACE, Chest X-ray
Toxoplasmosis	*Toxoplasma gondii* antibodies
Rocky Mountain Spotted Fever	*Rickettsia rickettsii* (RMSF IgG)
Leptospirosis	Leptospira IgM

Table 1: Infectious Causes of Neuroretinitis with associated lab testing *Tests may vary by lab

Serological Results

Lab testing results indicated that the *B, quintana* IgG antibody screen was positive with a >=1:1024 titer (reference <1:64). *B. quintana* IgM antibody screen was negative. Her antibody results were consistent with the reported 6-week duration of her symptoms. All other lab tests were normal. Her primary care provider was notified of her ophthalmic presentation and tentative diagnosis. She was also referred to an infectious disease specialist for consideration of further testing and treatment.

1 month follow up

The patient returned for a one-month re-evaluation with continued subjective gradual improvement in her vision. She denied any change to her medical history. She was not on any antibiotic therapy. She had been evaluated by her primary care provider who discussed her lab results; however, she did not initiate any treatment. She had not seen an infectious disease specialist as requested. The patient rescheduled her appointment with the infectious disease specialist to a later date

and was again educated on the importance of maintaining her appointment.

Blood pressure upon exam measured 127/93mm Hg right arm sitting. Best corrected visual acuities were 20/20 in both eyes. She exhibited full confrontation visual fields with normal facial Amsler bilaterally. She no longer reported blur on facial Amsler in the left eye. Extraocular motility was full in both eyes. She continued to demonstrate isochoric pupils in bright and dim illuminations. She no longer exhibited an afferent pupillary defect.

Anterior segment examination was stable and unremarkable. Intraocular pressures measured with Goldmann Applanation Tonometry were 11mm Hg OD and 12mm Hg OS.

Dilated fundus examination revealed mild improvement in the density of the exudates (Figure 4).

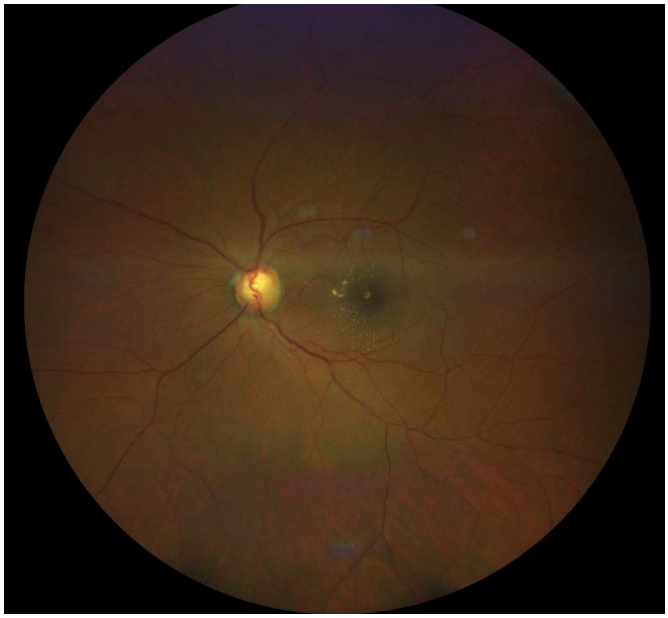

Figure 4: Color fundus photograph of the left eye at 1 month follow up showing improvement in the density of the parafoveal and subfoveal exudates. Optic disc appearance is stable with her previous visit.

Macular OCT (512x128) also demonstrated resolution of the subretinal fluid (Figure 5). Given the improvement in her presentation, we again recommended she return for ocular re-evaluation in 1 month.

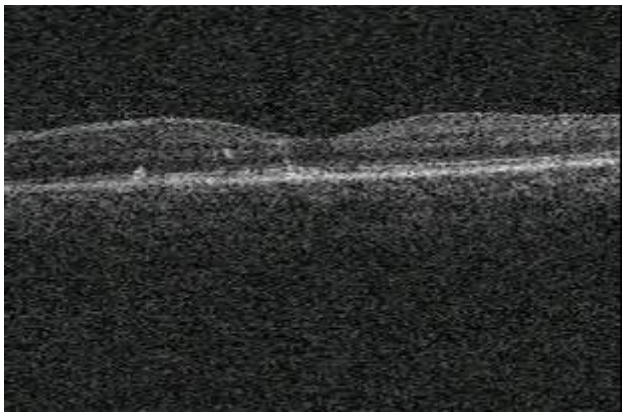

Figure 5: Macular OCT (512x128) showing resolution of subretinal fluid with few residual intraretinal and subretinal hyperreflective areas.

Unfortunately, despite patient education, she repeatedly rescheduled or no-showed appointments with the infectious disease specialist. She was, however, compliant with her ocular re-evaluations and demonstrated clinical resolution after 4 months, or approximately five and a half months from onset of visual symptoms.

DISCUSSION

Neuroretinitis was initially thought to be a disease of the macula. "Stellate maculopathy" was first described by Theodor Leber in 1916.[1] In 1977, Gass suggested that the condition was more closely related to a disorder of the optic disc based on the fact that optic disc edema precedes maculopathy.[1] With fluorescein angiography, he demonstrated that leakage was in fact from the optic disc, further endorsing his hypothesis. Inflammation leads to leakage of fluid from the optic disc vasculature into the peripapillary retina. Serial monitoring with OCT has demonstrated that fluid accumulates within and below the retina as a peripapillary serous detachment.[2] OCT may also show peripapillary or macular retinal thickening. Over time, as the fluid resorbs, deposits composed of lipid and protein collect in the outer plexiform layer to produce the characteristic macular star appearance. These exudates appear as intraretinal hyperreflective foci on OCT.[3] Once the exudates have resolved, persistent outer retinal layer disruption (of the external limiting membrane, ellipsoid zone and the foveal interdigitation zone) may persist.[3]

Patients with neuroretinitis typically report painless unilateral loss of vision. Clinically, patients often demonstrate reduced visual acuity, a small magnitude relative afferent pupillary defect, and a visual field defect. The pattern of visual field loss is most commonly a central or cecocentral scotoma. Patients may also demonstrate blind spot enlargement. The level of vision may be 20/20 to finger

counting at initial presentation. In some cases, neuroretinitis may be accompanied by anterior, intermediate, or posterior uveitis.

Disc edema is usually present upon initial examination; however, the classic macular star appearance develops over a few-week period. The disc edema typically resolves within three months; however, the macular star persists for a longer period of time and usually results in slower recovery of visual acuity.[4] Depending on the time of presentation, patients may exhibit disc edema and a macular star or either in isolation. Disc edema in the absence of a macular star should not exclude neuroretinitis from the list of differential diagnoses. Neuroretinitis often presents as vision loss following nonspecific systemic symptoms such as headaches, lymphadenopathy, arthralgia, and rashes particularly in infectious etiologies. Differential diagnoses of neuroretinitis may include hypertensive retinopathy, diabetic retinopathy, anterior ischemic optic neuropathy, retinal vein occlusion or papilledema.

Neuroretinitis may be idiopathic or secondary to infectious or inflammatory conditions. Infectious etiologies may be due to bacterial, viral, or fungal etiologies including, but not limited to, *Bartonella* species, syphilis, Lyme disease, toxoplasmosis, tuberculosis, West Nile, Zika, histoplasmosis, herpes simplex, and herpes zoster. Infectious neuroretinitis is more commonly unilateral but may rarely be bilateral. The presence of bilateral macular stars should raise suspicion for an alternative etiology such as malignant hypertension or papilledema due to increased intracranial pressure. Inflammatory conditions, including sarcoidosis, polyarteritis nodosa and systemic lupus erythematosus may also produce neuroretinitis.[5] Idiopathic and recurrent cases of neuroretinitis have also been described. Recurrent forms of the disease tend to be idiopathic with a poorer visual prognosis. Lack of preceding systemic symptoms and severe visual field loss tend to be associated with recurrent disease and less favorable visual prognosis.[6] A thorough history inquiring about concurrent or preceding symptoms such as rashes, animal exposure, fevers, malaise, and headaches may be helpful in establishing a list of differential diagnoses and when considering the need for additional serologic testing. Consideration of geographic location as well as recent travel can be helpful in developing a thorough, yet targeted, work-up.

Although ocular complications of *Bartonella* infection are rare, Cat Scratch Disease, or *Bartonella henselae* infection, is the most commonly diagnosed cause of neuroretinitis. Patients exposed to *B. henselae* may develop a hyperemic papule at the site of inoculation with development of regional lymphadenopathy over a period of several weeks. They may later develop a prodromal flu-like illness prior to onset of visual changes. Five percent of symptomatic patients with *B. henselae*

develop Parinaud's oculoglandular syndrome characterized by unilateral follicular conjunctivitis with regional lymphadenopathy and possible conjunctival granulomata.[7] This condition is most commonly related to Cat Scratch Disease, but it can arise secondary to other infectious etiologies and is therefore not pathognomonic. Only 1-2% of patients with symptomatic *B. henselae* infection develop neuroretinitis.[7] It is uncommon for patients to present with both conjunctivitis and neuroretinitis. Pathogenesis of neuroretinitis is thought to be related to an immune response, intraocular infection, or combination of both.

While *B. henselae* has a well-documented association with neuroretinitis, *B. quintana* is less commonly identified as an etiology with few published case reports.[3] In this case, our patient reported contact with a cat, which initially raised suspicion for *B. henselae* and cat-scratch disease. However, it is important to note that our patient tested positive for *B. quintana*, not *B. henselae*. In addition to neuroretinitis, *B. quintana* may manifest with other anterior and posterior segment findings as presented in Table 2.[8,9] *B. quintana* can present similarly to *B. henselae*, although *B. quintana* does not typically produce retinal vascular occlusions, whereas *B. henselae* may. Therefore, it is important to consider *B. quintana* as a potential etiology in many cases where *B. henselae* is considered.

Anterior Segment	**Posterior Segment**	**Systemic**
Parinaud's Oculoglandular syndrome	Neuroretinitis	Endocarditis
Anterior Uveitis	Retinitis	Trench fever
	Vasculitis	Bacillary angiomatosis
	Intermediate uveitis	Chronic Bacteremia
	Posterior uveitis	

Table 2: Manifestations of *Bartonella quintana* Infection

There are no well-established guidelines for treatment of *Bartonella*-associated neuroretinitis. Treatment considerations are often based on case series and retrospective reviews due to lack of randomized clinical trials. Immunocompetent patients tend to have favorable visual outcomes given the self-limiting disease

course, regardless of antibiotic treatment, in both *B. henselae* and *B. quintana* neuroretinitis. Immunocompromised patients or patients with severe systemic infections may benefit from antibiotic treatment. Treatment with a broad-spectrum antibiotic may be considered while lab results are pending. Antibiotics such as doxycycline, rifampin and azithromycin have been reported to reduce the severity and duration of visual manifestations in neuroretinitis associated with Bartonellosis (Table 3).[9] However, some clinicians recommend treatment only for severe visual loss or immunocompromised individuals.[10] If treatment is to be employed in pediatric patients, typically azithromycin and/or rifampin are preferred over doxycycline due to risk of tooth discoloration. Corticosteroids, with or without antibiotics, may also be considered in the treatment of neuroretinitis, particularly in cases of severely decreased vision or significant intraocular inflammation. One study reported better visual outcome with antibiotic plus steroid therapy compared with antibiotics alone in *B. henselae* related ocular conditions (including neuroretinitis as well as uveitis and retinal vascular occlusions).[11] Corticosteroids alone may be considered in cases of recurrent or idiopathic neuroretinitis. Alternatively, immunosuppressive treatment may be prescribed for patients with recurrent disease. Ultimately, risk versus benefit of therapy must be weighed due to possibility of side effects and adverse reactions with pharmacologic therapy. We elected not to initiate treatment in our patient because she presented non-acutely with improving visual symptoms.

Antibiotic	**Dose (Adult and Children >45kg)**	**Dose (Children <45kg)**
Azithromycin	500mg PO for 1 day, then 250mg PO for 4-6 weeks	10mg/kg PO for 1 day, then 5mg/kg PO for 4-6 weeks
Doxycycline[22]	100mg PO BID for 4-6 weeks	2.2mg/kg BID for 4-6 weeks
Rifampin	300mg PO BID for 4-6 weeks	10mg/kg BID for 4-6 weeks

Table 3: Treatment Options for *B. henselae* or *B.quintana*-associated neuroretinitis*
*Duration of treatment may be prolonged in immunocompromised patients

Long-term visual prognosis of *Bartonella*-associated neuroretinitis is thought to be quite good. However, there have been reports of poor visual outcomes. Development of full-thickness macular holes following *Bartonella*-associated neuroretinitis have been reported in few case reports.[12] A small case series

published in 1998 found that the majority of patients experienced recovery of visual acuity and visual fields, although some patients demonstrated persistent fundus changes such as macular pigmentary abnormalities and disc pallor.[13] In the case of our patient, visual acuity returned to 20/20 and she no longer exhibited an APD at her one-month follow-up visit without treatment. Four months after her initial presentation, the retinal exudates had resolved completely. As of her four-month follow-up, the area concerning for sectoral disc edema in the affected eye persisted.

Bartonella henselae and *Bartonella quintana* may be diagnosed with serologic testing; however, there is a potential for false negatives. Acute infections may show normal titers; if suspicion is high despite normal titers, lab testing may be repeated after several weeks. There is also cross reactivity between the two organisms, especially in immunoglobulin G (IgG) titers.[14] In most cases, the infecting organism will have the higher titer. Diagnosis may also be aided by culture, immunohistochemistry, or polymerase chain reaction (PCR) based testing. High IgG titers usually indicate active or recent infection while lower titers may indicate chronic infections.[14] Immunoglobulin M (IgM) titers are positive in cases of recent infection, whereas IgG titers are positive in current or past infection. Ultimately, the role of the optometrist includes referring the patient to the proper provider(s) to receive care of non-ocular manifestations. In our case, *B. quintana* IgG titers were positive in the setting of normal IgM titers to suggest a more chronic infection. Our patient had a nonreactive *B. henselae* titer; therefore, we did not suspect that the *B. quintana* titer was a result of cross-reactivity. The patient did not present during the acute phase of her symptoms. However, since the IgG titer was quite elevated (>=1:1024), she was promptly referred to an infectious disease specialist for consideration of further testing for systemic complications such as endocarditis or chronic bacteremia which would require treatment.

Bartonellosis in humans is most commonly caused by *Bartonella henselae, B. quintana* and *B. bacillioformis*.[15] *B. henselae* is most commonly associated with ocular manifestations; however, *B. quintana* has also been implicated in cases of ocular inflammation including cases of neuroretinitis.[3,9] Bartonellosis due to *B. quintana* may result in systemic manifestations such as lymphadenopathy, endocarditis or trench fever.[14] Trench fever was named in the early 1900s due to the high number of illnesses in soldiers in World War I. It is characterized by recurrent fever, headache, weakness, and bone pain, often in the shins.[16] Immunocompromised patients typically have more severe clinical manifestations of Bartonellosis than patients with normal immune systems; some patients do not develop any symptoms and many cases are self-limiting within a few months. Alternatively, some patients develop chronic manifestations including bacteremia, which may be asymptomatic.

Both *B. henselae* and *B. quintana* may be transmitted to humans via human lice or cat scratches, licks or bites, or potentially from infected cat fleas or contact with flea feces. Cats are considered the reservoir for *B. henselae* while humans are considered the reservoir for *B. quintana*. However, both species have been isolated from cats.[17] Cats are not routinely screened for *Bartonella.* Infected cats may appear asymptomatic, or may manifest symptoms including fever, lethargy, or vomiting. *Bartonella* species are more likely to be transmitted by kittens than by adult cats. Since both *B. henselae* and *B. quintana* have been isolated from cats, it is important to test for both organisms in cases with history of contact with cats. Homelessness and living in unsanitary or crowded conditions is also associated with *B. quintana* infection which can be transmitted by the human body louse.

It is also important to recognize that treatment may be necessary for other systemic manifestations. *B. quintana* has been found to infect erythrocytes and endothelial cells and can manifest as acute, chronic, or asymptomatic disease states affecting various organ systems, including the eye.[18] There is also a lack of well-established guidelines for manifestations affecting other organ systems.

Systemic antibiotic therapy for *Bartonella* infections varies depending on the systemic manifestations and severity of clinical findings. A systematic review and meta-analysis from 2012 found no evidence to support a better cure rate or time to cure with antibiotics for Cat Scratch Disease, or *B. henselae* infection.[18] However, it is important to note that infections caused by *Bartonella* species may cause acute or chronic disease with variable severity and prognosis. More severe manifestations of infection, such as endocarditis and chronic bacteremia, warrant antibiotic treatment although there is little consensus on a specific optimal regimen. Given the wide variety of systemic manifestations and complications, it is important for the optometrist to co-manage the patient with other providers to assess for concomitant systemic disease and treat appropriately.

When neuroretinitis is suspected, consider ordering serologic testing for underlying systemic etiologies. While the ocular condition is considered self-limiting, patients may suffer from systemic consequences as mentioned above. The presence of ocular findings may be the first indication of systemic manifestations. An idiopathic recurrent form of neuroretinitis has also been described in which patients present with more severe visual field defects and less improvement in visual function. Some patients with recurrent forms of the condition may require long-term immunosuppression to reduce frequency of attacks.[19]

CONCLUSION

This case demonstrates a rare case of *B. quintana* associated neuroretinitis and stresses the importance of obtaining a detailed history and using a targeted approach to diagnosis. While *B. henselae* is the most common cause of neuroretinitis, it is important to maintain a thorough list of potential etiologies, including *B. quintana,* when considering further serologic testing to improve diagnostic yield. Clinicians should recognize the potential ocular manifestations of *B. quintana* and include it in their work-up along with *B. henselae*. This case also highlights the importance of communication and co-management with other providers including primary care physicians and infectious disease specialists, to ensure that potential systemic manifestations are addressed.

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