Neuroretinitis as the only manifestation of Lyme disease: A case report

Luis Felipe Canova Ogliari[1], Mateus Pimenta Arruda[1], Marcio A. Nogueira Costa[1], Heitor Santos Nogueira[1], Jacqueline Coblentz[2], Miguel N. Burnier Jr[2,3]

[1] Department of Ophthalmology, Penido Burnier Institute, Campinas-SP, Brazil
[2] MUHC-McGill University Ocular Pathology and Translational Research Laboratory
[3] Department of Ophthalmology and Pathology, McGill University, Montreal, QC, Canadá

PURPOSE:

We herein present a patient who had neuroretinitis related to a previous diagnosis of Lyme disease. The presence of neuroretinitis in a setting of Lyme disease represents a very uncommon manifestation of this particular infection.

METHODS:

Case Report.

RESULTS:

A 23-year-old woman presented on May 13, 2020 complaining of low visual acuity in the left eye (LE) for 30 days. She had no history of trauma or medication use.

VISUAL ACUITY:

OD: 20/20
OS: 20/400

BIOMICROSCOPY:

OU: clear conjunctiva, transparent cornea, phakic, and the absence of anterior chamber reaction in both eyes

FUNDOSCOPY:

OD: physiologic cupping, and preserved macular brightness

OS: 4+/4+ papilledema, increased vascular tortuosity, presence of exudates and edema in the macular region, applied retina (Figure 1).

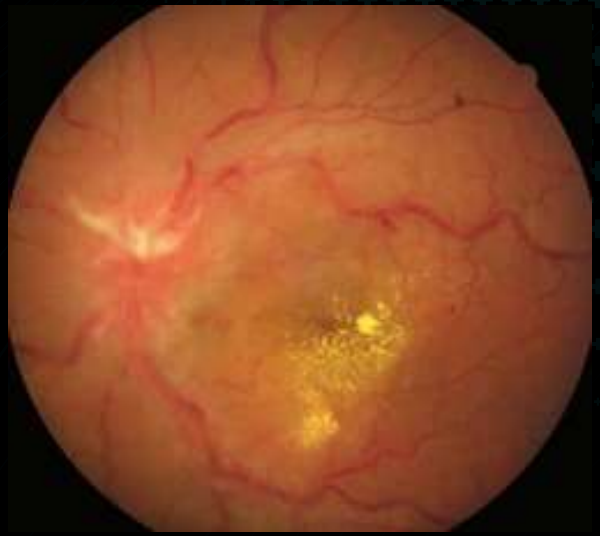

Figure 1: Fundus picture of OS showing significant pailledema, increased vascular tortuosity, exudates and macular edema

Optical coherence tomography (OCT) (Figure 2), angiography (Figure 3), and retinography were performed. Requested erythrocyte sedimentation rate, complete blood count, serology for toxoplasmosis(immunoglobulin G [IgG], immunoglobulin M [IgM]), tuberculin skin test, venereal disease research laboratory, fluorescent treponemal antibody absorption, herpes simplex virus test (IgG and IgM), rheumatoid factor, antinuclear antibody, serology for Bartonella, chest radiography and angiotensin-converting enzyme. Since serologies results were pending, empirical treatment with doxycycline 100 mg 12/12 h was started, considering a probable neuroretinitis due to cat scratch disease. The serology for Bartonella was negative; however, the IgM serology was 1:116 positive for Borrelia and was confirmed by the Western blot test. The diagnostic hypothesis was neuroretinitis caused by Lyme disease. It was decided to supplement with prednisolone 40 mg/day, as an anti-inflammatory, because of the disc edema, and the patient was followed up being observed clinically and on imaging tests. OCT and retinography were requested as follow-up.

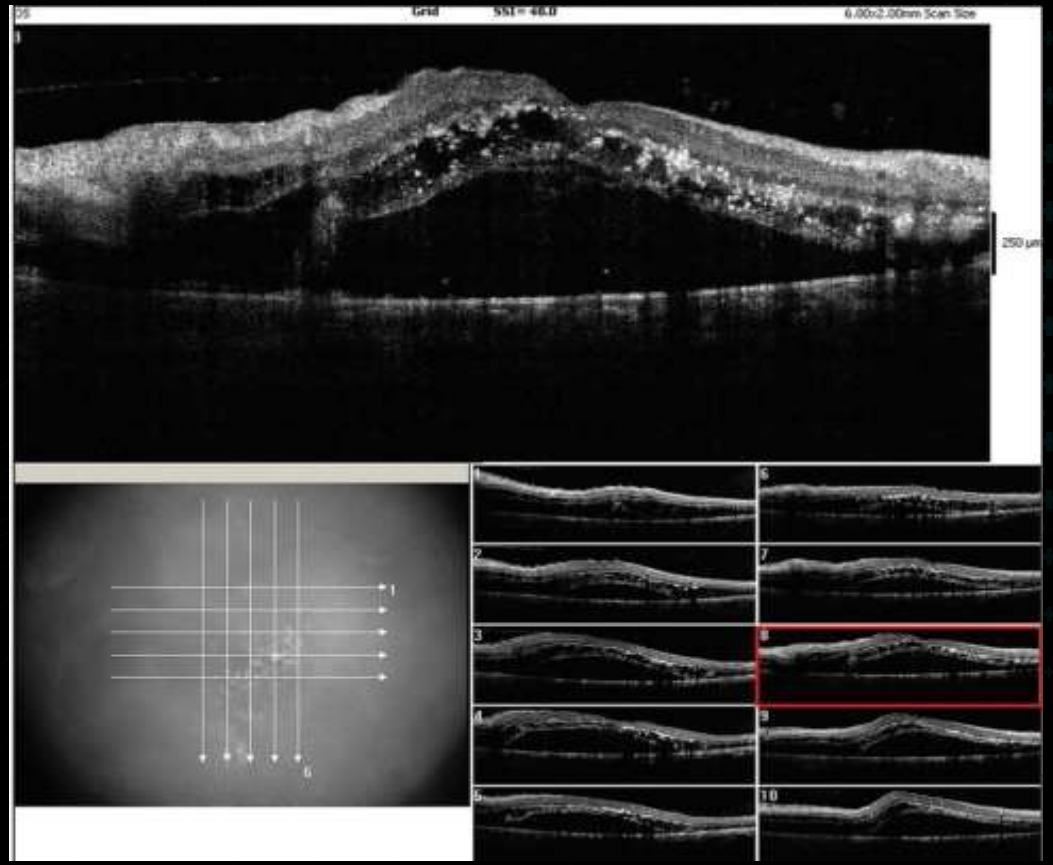

Figure 2: Optical coherence tomography of left eye (before treatment) showing loss of foveal depression, “wrinkling” of the inner limiting membrane, with an increase in the diffuse retinal thickness. There is the presence of intraretinal and subretinal fluid, leading to sensory retinal detachment

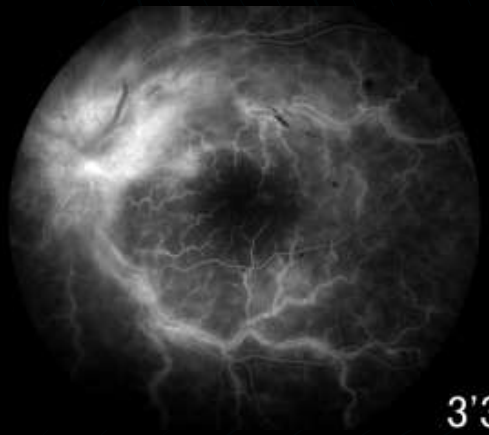

Figure 3: Angiography (late venous phase) of the left eye showing hyperfluorescence with blurring of the optic disc. Progressive hyperfluorescence is also evidenced by staining in the arcades. The macular region, as well the temporal periphery are hypofluorescent

After 60 days, there was an improvement in the disc edema, exudation, and macular edema, with an important improvement in visual acuity [Figure 4].

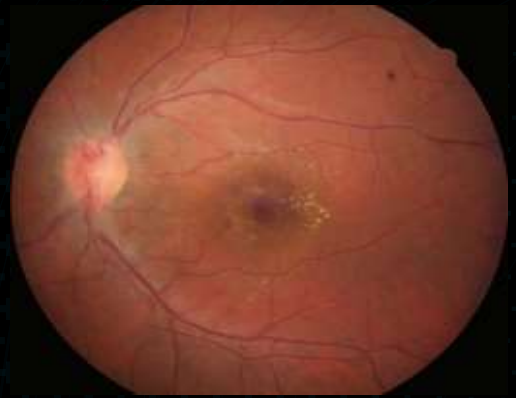

Figure 4: Fundus picture of left eye after treatment showing improvement in the papilledema, exudation, and macular edema

LE OCT (after treatment): vitreoretinal interface without changes, with vitreous adhesion in the macular region. Thinning of the inner layers of the retina. The absence of subretinal fluid. Hyperreflective spots between inner and outer plexiform nuclear layers, temporally to the macula, and choroidal thickness preserved [Figure 5].

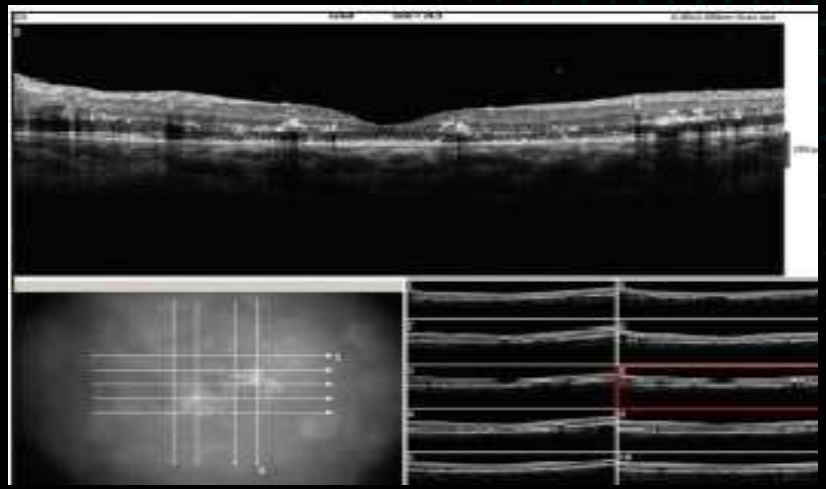

Figure 5: Optical coherence tomography of left eye after treatment showing thinning of the inner layers of the retina, absence of subretinal fluid, and hyperreflective spots between inner and outer plexiform nuclear layers, temporally to the macula

DISCUSSION AND CONCLUSION:

The infectious agent of Lyme disease is B. burgdorferi, a spirochete bacterium detected in blood, synovial fluid, cerebrospinal fluid, retina, and vitreous humor.[1,2]
The most common and initial manifestation is the skin lesion called erythema migrans, which is present in 70%–80% of patients and may be related to constitutional symptoms such as fever, myalgia, and headache.[1,2]
The absence of skin lesions, and also neuroretinitis as the only presentation of this infection, is indeed very uncommon; although, it has been reported in some cases.[1,3] The similarity of these cases to our, was that the patient's outcome was sudden and painless blurring vision, without cutaneous manifestation and three of them had a serology confirmation by the enzyme-linked immunosorbent assay (ELISA) and Western blot.

Neuroretinitis is an inflammatory disorder characterized by optic disc edema and subsequent macular star imaging.[1,2] It is one of its possible manifestations, commonly in the secondary stage,[1,4] but neuroretinitis can also occur in cat-scratch disease, syphilis, mumps, toxoplasmosis, salmonellosis, tuberculosis, and histoplasmosis.

It is recommended that the diagnosis be performed by serology using an immunoenzymatic test, and confirmed by the Western Blot test.[2]

Once the diagnosis is confirmed, the indicated therapy is doxycycline 100 mg 12/12 h or 200 mg once a day for 14–21 days, or amoxicillin 500 mg three times a day for 14–21 days.[2] Allergic patients may use erythromycin or tetracycline.

In our patient, oral doxycycline and corticosteroids were prescribed, with significant improvement in vision after 60 days (BCVA: 20/30) and also in OCT (which is possible to monitor the evolution of macular edema and optic nerve).

References:

1: Guliani BP, Kumar S, Chawla N, Mehta A. Neuroretinitis as presenting and the only presentation of Lyme disease: Diagnosis and management. Indian J Ophthalmol 2017;65:250-2.

2: Sanchez E, Vannier E, Wormser GP, Hu LT. Diagnosis, treatment, and prevention of Lyme disease, human granulocytic anaplasmosis, and babesiosis: A review. JAMA 2016;315:1767-77.

3: Vanya M, Fejes I, Jako M, Tula A, Terhes G, Janaky M, et al. Lyme disease associated neuroretinitis – Case report. Acta Microbiol Immunol Hung 2015;62:403-8.

4: Purvin V, Sundaram S, Kawasaki A. Neuroretinitis: Review of the literature and new observations. J Neuroophthalmol 2011;31:58-68.