

CASE REPORT

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Intravitreal Sustained-release Dexamethasone Implant in Treatment for Chronic Macular Edema in Syphilis Infection: a Case Report

Azra Mehmedovic¹, Vernesa Sofic-Drino¹, Alma Biscevic^{1,2}, Melisa Ahmedbegovic-Pjano¹**ABSTRACT**

Background: Macular edema results from many conditions, such as diabetic retinopathy, macular degeneration, inflammatory diseases, cataract operation, trauma, and tumors. Specifically, the capillary filtration rate should equal the speed of fluid removal from extracellular retinal tissue, such as the glial and retinal pigment epithelium cells layer (RPE). Once these forces are imbalanced, fluid accumulates in cystoid spaces within the inner layers of the retina. **Objective:** The main purpose of this case report is to show that macular edema caused by any inflammation, either bacteria, virus, or autoimmune origin, can be treated successfully, even if it is chronic. **Case report:** A 31-year-old man has been reported to our clinic with symptoms of blurry vision in the left eye, which occurred during the last year. Essential examinations included CDVA, IOP measurement, slit-lamp examination, indirect ophthalmoscopy, and OCT scan that showed significant macular edema (central foveal thickness of 353 microns). We initiated laboratory searches, such as blood, serology, and immunology testing for the next three months after his first visit, together with prescribed topical and periocular corticosteroid therapy. The test to VDRL (venereal disease research laboratory) for Syphilis and Toxocariasis came positive. We took the best decision and recommended further treatment with the intravitreal application of Dexamethasone Implant 0.7mg. One week after the intravitreal application of corticosteroids on the control exam, there were normal findings on the posterior segment with no macular edema (central foveal thickness of 269 microns). **Conclusion:** It is unexclusive that infection by *Treponema pallidum* (TP) causes isolated macular edema without any other symptoms on the anterior segment of the eye. It has indirect action on the macula, not just causing papilledema, retinal vasculitis, retinochoroiditis, and inflammatory disc edema, as expected. TP or the bacteria transmembrane protein (treponemal ligands) directly acting on vascular endothelial cells of the RPE cells, will be the key to the most certain mechanism of this condition. It is related to the possibility of the secretion of cytokines and the interactions between immune cells indirectly.

Keywords: *Treponema pallidum*, macular edema, retina, Dexamethasone, inflammation

1. BACKGROUND

Cystoid Macular Edema (CME) is a retinal thickening of the macula that disrupts the normal blood-retinal barrier. This causes leakage from the perifoveal retinal capillaries and accumulation of fluid within the intracellular spaces of the retina, primarily in the outer plexiform layer (1). The ocular presentation of the Syphilis infection can be various. As known, it can infect the anterior and posterior segment of the eye, but the most common is posterior uveitis and panuveitis (2). Eye infection can occur in any stage of the primary systemic disease. It can also be delayed over the years until the first signs of the illness appear. However, the leading role is the immunodeficiency of the host. Some cases of Syphilis infection can lead to unilateral sight loss, but others can also be treated well without complications. Most complications are usually connected with HIV infection. Autoimmune diseases frequently cause macular edema along with leading factors, such as diabetic retinopathy, macular degeneration, vein occlusion, macular traction, trauma, and tumors.

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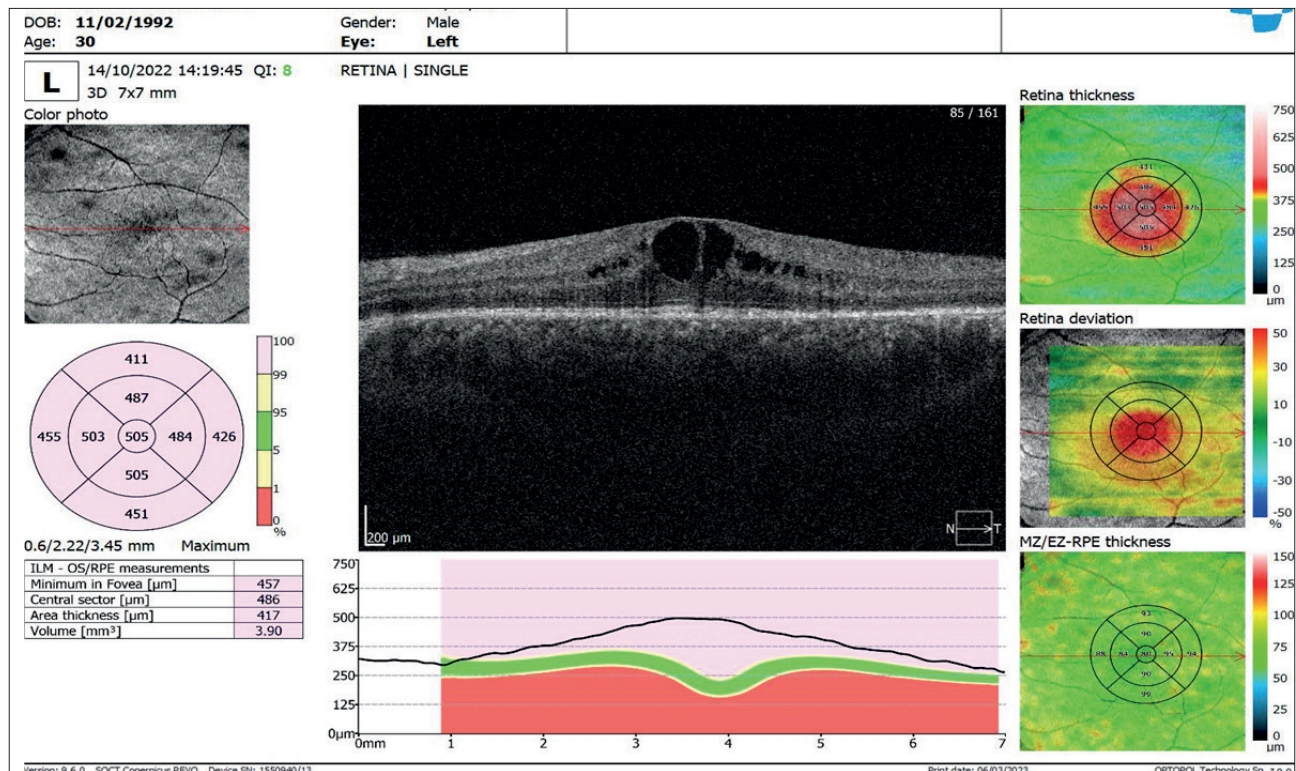


Figure 1. The OCT image shows the macular edema in total, its central foveal thickness measuring 397 microns, the retinal pigment epithelium layer (RPE) layer's disorder bit on the foveola's temporal side.

This is useful for understanding our decision in what way to treat this inflammatory-induced cystoid macular edema (CME). The initial treatment for macular edema caused by inflammation is topical and periocular corticosteroids. CME, which did not respond to periocular corticosteroid, can be resolved with an intravitreal sustained-release dexamethasone implant (3). It can also be a big challenge because of the interaction of the therapy with the systematic treatment that requires immunosuppression. Elevated levels of proinflammatory cytokines and vascular endothelial growth factors were found in all types of cystoid macular edema (4). Ocular involvement is a rare complication of syphilis, occurring in approximately 1% of cases (5).

2. OBJECTIVE

The main purpose of this case report is to show that macular edema caused by any inflammation, either bacteria, virus, or autoimmune origin, can be treated successfully, even if it is chronic.

3. CASE REPORT

A 31-year-old man has been reported to our clinic with symptoms of blurry vision in the left eye, which occurred during the past year. We started with essential examinations such as visual acuity testing, intraocular pressure measurements (IOP), slit-lamp examination, and indirect ophthalmoscopy (fundus examination). We found normal findings of the anterior segment: clear cornea, deep and clear anterior chamber, normal iris and pupil reaction, and clear lens. Distance visual acuity was measured using a standard Snellen acuity chart at 6m and presented in decimals. Corrected distance visu-

al acuity (CDVA) in the left eye was 0.55 Snellen lines, and IOP was 15 mmHg. In the posterior segment, there were no signs of vitritis and retinitis. The optic disc had regular sharp-shaped borders with no edema; the macula showed no clinical edema, blood, or exudation. The OCT (SOCT Copernicus REVO, Optopol, Zawiercie, Poland) scan showed significant edema that measured 397 microns in central foveal thickness, with a slight disorder of the retinal pigment cell layer (Figure 1). Macular edema persisted during all control exams, with slight vision deterioration with a CDVA of 0.35.

We treated the patient with topical corticosteroids, Dorzolamide, Bromfenac sodium-sesquihydrate, and periocular corticosteroids (Triamcinolone acetonide). Therapy was repeated for nine weeks, and there was no improvement. We initiated laboratory searches, such as blood, serology, and immunology testing. Testing was done for rheumatoid arthritis, paranasal sinusitis, HLA b27 typing for ankylosing spondylitis, testing for venereal diseases (VDRL), CMV, Toxocariasis, HIV, and EBV. We got the results Reactive 20.96 S/CO for Syphilis, Toxocara canis IgG:11.5 Positive, EBV:). Guided by the findings, we took the best decision and recommended further treatment with the intravitreal application of a sustained-release Dexamethasone implant in the left eye. Seven days after treatment, we had satisfactory results: macular edema (central foveal thickness of 288 microns), slight retinal pigment cell layer disorder, and improved CDVA of 0.75 Snellen lines (Figure 2).

OCT imaging was an essential and central significant diagnostic procedure. In figures one and two, we can establish and monitor the stages of macular edema for an extended period and evaluate its treatment by showing

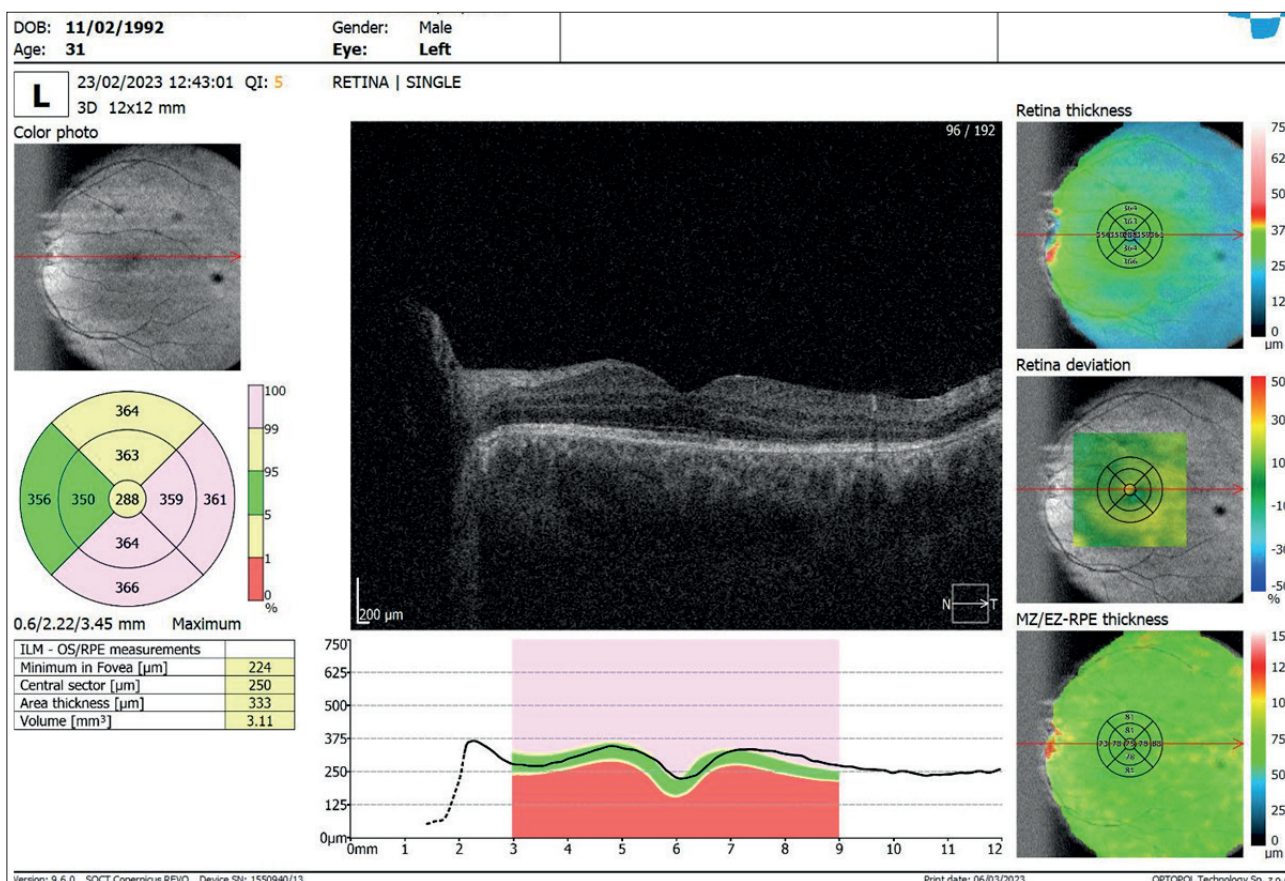


Figure 2. OCT image shows the same eye after the treatment in full; its central foveal thickness measuring 288 microns, and slight disorder of the RPE layer bit on the temporal side of the foveola.

how it affects improvement. However, this doesn't show any other significant diagnostic feature that can be very significant to assessing other treatment improvement signs.

4. DISCUSSION

Cystoid macular edema is the leading cause of vision loss in patients with inflammatory diseases such as syphilis. It's challenging to treat because it's not clear the mechanism by which it forms. Still, we can presume it is the inflammatory response for proinflammatory cytokines, and vascular endothelial growth factor (VEGF) can be very stubborn, lasts for various periods, and is recurrent in most cases with very small or no response for therapy. It is probably mainly because of the immune response of the combined inflammatory processes on usually not just one infection cause. Interleukin-6 [IL-6] is produced by multiple cells of the innate immune system and leads to a higher likelihood of developing autoimmune inflammatory diseases, such as non-infectious uveitis, through various mechanisms. These include increasing the helper T-cell population over the regulatory T-cell population, leading to the increased expression of inflammatory cytokines, such as tumor necrosis factor-alpha, and being vital in generating uveitis and subsequent macular edema through these inflammatory pathways (6). Treatment with anti-inflammatory and anti-vascular endothelial growth factor (anti-VEGF) drugs is widely applied for all forms of cystoid macular edema. Usually, it has a beneficial but temporary effect. The ef-

fect of intravitreal injection on the long-term prognosis of CME is challenging to evaluate; in some patients, a single injection will be effective for an extended period, while others will require multiple injections for relapsing CME (7).

5. CONCLUSION

This case report demonstrates that initial therapy of intravitreal sustained-release Dexamethasone implant in treatment for cystoid macular edema caused by Syphilis infection has a good improvement for decreasing macular edema in full of its thickness and improving the visual accuracy. However, corticosteroids have maximum efficacy between 3 and 6 months, can cause high eye pressure, and can lead to cataract formation in the long term. Upcoming data on aqueous constituents in cystoid macular edema and imaging with the new generation of optical coherence tomography offer the hope that a better treatment strategy will soon be established. Otherwise, performing and following the treatment in any other way will take much work. It is well known that corticosteroids reduce inflammation by suppressing immune system activity. The effect of vitrectomy on inflammatory cystoid macular edema is unclear and might become more critical in the future (4). OCT imaging is the primary diagnostic procedure in these cases; it is straightforward and noninvasive and can be performed in every stage of the disease. However, it doesn't show us any other parameter of the benefit of the treatment, which can be very useful for evaluating progression. As

known, visual accuracy, intraocular pressure, and blood testing are also significant. Corticosteroid treatment can cause eye pressure elevation in the long term, so it needs to be followed and controlled often. Following the case, we perform to include eye drops that reduce eye pressure and anti-inflammatory eye drops (such as topical corticosteroids) in therapy. Cystoid macular edema can be recurrent. Predominantly, it is self-limiting and spontaneously resolves within 3-4 months. If the edema is chronic (more than 6-9 months), permanent damage to the photoreceptors with retinal thinning and fibrosis can occur (1). Knowing all of this, it is crucial to make all diagnostic procedures, establish the correct diagnostic problem, and start the treatment on time to improve visual acuity.

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