POSTERIOR UVEITIS OR WET AGE-RELATED MACULAR DEGENERATION? CASE REPORT

Filip Mircea, Moisescu Raluca
*Ama Optimex Eye Clinic, Bucharest, Romania

Correspondence to: Mircea Filip, MD, PhD, Associate Professor, FEBO
Ama Optimex Eye Clinic, Bucharest, Romania
54 Toamnel Street, District 2, Bucharest, Romania
Phone: +4021 211 16 22, E-mail: mirceafilip@amaoptimex.ro

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Abstract
We present the case of a 61-year-old patient without previous ophthalmic or general history, who developed unilateral posterior pole granuloma and was diagnosed with posterior uveitis most likely due to a systemic Toxocara canis infection. Clinical examination and ancillary investigations showed elements that were also consistent with wet ARMD, but laboratory tests and successful use of oral anti-helminthic and corticosteroid therapy in decreasing the macular lesion and improving visual acuity, confirmed the diagnosis of posterior uveitis.

Keywords: Posterior uveitis, wet age-related macular degeneration, choroidal neovascularization

Case report
A 61-year-old woman presented to our practice complaining of about a 15 day decreased vision in the right eye (OD). There was no previous ophthalmic history and her general health was good. The patient denied having eye pain, redness, photophobia or irritation. She was not taking any medication and denied any medications allergies. On presentation, the best corrected visual acuity (BCVA) in the affected eye was counting fingers (CFs) and 20/20 in the left eye (OS). Results of slit-lamp examination were normal. Pupils were normal in size and shape and reactive to light, with discrete lens opacification present in both eyes. Intraocular pressure (IOP) values measured with the Goldmann tonometer were 12 and 15 mmHg respectively. The Amsler grid test was positive OD.
Fundus examination revealed a 2 DD round, raised, yellowish-white sub-retinal lesion adjacent to the fovea, surrounded by retinal hemorrhage OD and several white macular lesions and pigment mottling OS (Fig. 1). The optic disc, retinal vessels and vitreous were normal in both eyes. The patient had an optical coherence tomography (OCT) exam performed 3 days before, which showed a highly reflective mass located above the retinal pigment epithelium (RPE), intraretinal fluid with central retinal thickness (CRT) of 425 µm OD and small highly reflective lesions located at the level of Bruch membrane, consistent with drusen OS.

Fundus fluorescein angiography (FFA) revealed late hyperfluorescence consistent with CNV OD (Fig. 2) and late staining of multiple small lesions and underlying choroidal vasculature consistent with drusen and RPE atrophy OS (Fig. 3).
B-mode ultrasonography (USG) showed a discrete elevation of the posterior pole without fluid accumulation or edema in the episcleral space or around the optic nerve (Fig. 4).

We decided to immediately start therapy with topical NSAID drops 3 times per day OD and oral ceftriaxone 500mg twice per day and a 3-day course of oral methylprednisolone 32 mg per day, starting 48 after antibiotics. The patient was advised to undergo a series of laboratory tests, which included blood count, erythrocyte sedimentation rate, leukocytes, antinuclear antibodies, rheumatoid factor, purified protein derivative of tuberculin, Toxoplasma IgG and IgM and Toxocara enzyme-linked immunosorbent assay (ELISA), and to return for follow-up in 2 weeks.

The patient did not report as scheduled, but after 1 month from the initial visit, she returned with the blood work. It revealed mild leukocytosis with eosinophilia, increased fibrinogen levels and a positive ELISA test for Toxocara IgG. The Borrelia IgM was negative, there was no evidence of an active infection with cytomegalovirus (CMV), HSV, VZV and Rubella and the rheumatology tests were normal. The patient stated that she had undergone a 10-day treatment course with oral albendazole 400mg per day (10mg/ kg of body weight in 2 divided doses) and oral clarithromycin 500 mg two times per day, as recommended by the parasitology specialist.

BCVA was 1/20 OD and 20/20 OS, results of slit-lamp examination were again normal, as was IOP in both eyes. Fundus examination revealed decreased size of macular lesion, with persistent surrounding hemorrhage OD. OCT performed in our clinic showed a decrease in CRT with persistent highly reflective mass above the RPE OD (Fig. 5).

The patient was advised to start a 14-day oral steroid treatment course by using progressively decreasing doses of methylprednisolone, in association with topical NSAID drops 3 times per day OD. The next visit was scheduled in 2 weeks.
Discussion

The above-presented case raised the issue of differential diagnosis between posterior uveitis and wet ARMD in a patient with macular edema and CNV.

Posterior uveitis describes an inflammation of the choroid, which may be referred to as choroiditis or chorioretinitis, if the retina is also involved. It is further defined as being focal, multifocal or diffuse, depending on the nature of the inflammatory lesions, usually affecting young people between 20 and 50 years [1].

Posterior uveitis causes gradual visual loss, often associated with floaters caused by the presence of cells in the vitreous. There is occasional photophobia, but little or no discomfort or redness. Inflammatory lesions may be seen on the retina or choroid, yellow when fresh, whilst older ones have more distinct edges and a whitish appearance [1,2].

A thorough diagnostic work-up directed by patient's history, symptoms and signs and clinical examination is mandatory. Ancillary investigations such as FFA, USG, macular OCT and selective laboratory investigations help in confirming the diagnosis. It is of paramount importance to identify the possible etiology, as posterior uveitis can be infective or non-infective [3,4].

Laboratory tests are more useful in infective than in non-infective conditions. It is extremely important that the patient is evaluated thoroughly by an internist, to rule out possible associated causes of his/her uveitis, because the therapy is incomplete without simultaneous treatment of the underlying systemic condition [5].

Local and systemic steroids along with immunosuppressives in selected cases are the mainstay of treatment of non-infective conditions. Infective conditions need to be treated primarily with the specific anti-infective agents along with anti-inflammatory therapy in the form of low-dose steroids. In case of infective uveitis, systemic steroids need to be initiated at least 48–72 h after the start of specific anti-infective therapy and then stopped at least 1 week prior to stoppage of specific treatment [5,6].

Elements suggesting the existence of a posterior pole granuloma in this case are:

- Relatively sudden decrease in visual acuity
- Fundus appearance showing characteristic yellow, elevated macular lesion, surrounded by hemorrhage resembling recent inflammation
- Hyperfluorescence on FFA consistent with granuloma
- Blood work revealing mild systemic inflammation and positive antibodies for Toxocara canis
- Good outcome after specific anti-helminthic and oral steroid therapy with mild increase in visual acuity

Wet ARMD is a chronic eye disease, usually occurring in patients over the age of 50, that causes central loss of vision. In this type of disease, abnormal blood vessels, known as choroidal neovascularization, grow under the retina and macula. These vessels may then bleed and leak fluid, causing macular edema and thus distorting or destroying central vision. Therefore, vision loss may be rapid and severe. Wet ARMD treatment options include intravitreal injections using Vascular Endothelial Growth Factor (VEGF) inhibitors such as
ranibizumab or potent corticosteroids such as triamcinolone acetonide [1,7,8].

Findings consistent with wet ARMD in this case are the following:
- Age over 50
- No history of ocular inflammation
- No general history consistent with infectious or inflammatory systemic conditions
- Decreased central vision with positive Amsler grid test
- Normal slit-lamp examination of anterior pole and vitreous
- Macular edema and CNV documented by OCT in the affected eye
- Drusen-type lesions consistent with dry ARMD in the other eye
- Relatively normal appearance of the choroid and sclera on B-mode USG

CNV is most commonly seen in wet ARMD, but is also a well-known complication of posterior uveitis, associated with choroidal inflammation and damage. Intermediate and posterior uveitis will also cause macular edema, therefore macular OCT became a useful tool in following the disease with greater ease and sensitivity. FFA confirms the presence of CNV in both posterior uveitis and wet ARMD. As treatment approaches for posterior uveitis and wet ARMD are significantly different, a correct diagnosis must be established immediately in order to avoid complications.

In this case, the atypical clinical features generate the need for further instrumental tests and laboratory exams. As in most situations, it was difficult to establish the diagnosis based only on clinical manifestations, because ocular symptoms may be various and inflammatory signs are not always present. In our case, there was no sign of systemic involvement, but only the presence of eosinophilia at laboratory examinations and positivity of IgG. We predict a good prognosis in this case due to immediate start of appropriate therapy and favorable results at first follow-up.

**Conclusions**

Considering all of the above, we sustain the diagnosis of posterior uveitis in this case and the need for immediate antihelminthic and oral steroid therapy in order to improve fundus appearance and visual acuity. In our opinion, the presentation occurred in the early stages of the disease with sudden decrease of vision due to posterior pole granuloma formation and CNV. It is therefore in the best interest of the patient to use every available medical resource in order to accurately diagnose and treat posterior uveitis, because the delay can cause severe complications and permanent visual loss.

**References**