

## Ocular tuberculosis presenting as a central retinal vein occlusion

Raid Soumaya<sup>1</sup>, El Moataz billah Salma<sup>2</sup>, Mchachi Adil<sup>3</sup>, Benhmidoune Leila<sup>4</sup>,  
Chakib Abderrahim<sup>5</sup>, Rachid Rayad<sup>6</sup>, El Belhadji Mohamed<sup>7</sup>

<sup>1, 2</sup> Resident doctor, <sup>4, 5, 6, 7</sup> Associate professor

Department of ophthalmology, 20 Aout 1953 teaching hospital, University hospital center Ibn Rochd,  
Casablanca, Morocco

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### Summary:

Ocular tuberculosis still represents a major diagnostic and therapeutic challenge, due to its heterogeneous clinical manifestations, mixed ocular tissue involvement, lack of diagnostic criteria and gold standard tests, and lack of international agreement on the therapeutic approach. We report the case of ocular tuberculosis presenting initially as central retinal vein occlusion. A 27 years old female patient presented to the ophthalmology emergency department for an acute painless unilateral vision loss. The examination findings were compatible with a central retinal vein occlusion. The suggestive chest Xray findings, associated with the positive tuberculin skin test and the good response to the anti-tuberculosis treatment were in favor of the diagnosis of tuberculosis. Central retinal vein occlusion in a young patient warrants extensive investigation to look for an underlying cause.

**Key words:** Tuberculosis, central retinal vein, occlusion, macular edema, anti-Vegf

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Date of Submission: 02-07-2021

Date of Acceptance: 16-07-2021

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### I. Introduction:

In Morocco, tuberculosis remains a major public health problem in spite of the efforts of the Ministry of health to alleviate it. Ocular TB is a rare extra pulmonary form of the disease which can have a considerable potential impact on visual loss. Ocular tuberculosis still represents a major diagnostic and therapeutic challenge, due to its heterogeneous clinical manifestations, mixed ocular tissue involvement, lack of diagnostic criteria and gold standard tests, and lack of international agreement on the therapeutic approach. Much like the ability of syphilis to mimic various skin conditions, tuberculosis should be thought of as “the great imitator” of ocular pathologies. Patients with ocular tuberculosis present with a wide spectrum of clinical signs; however, central retinal vein occlusion associated with ocular tuberculosis is rare and only a few cases have been reported. We report the case of ocular tuberculosis presenting initially as central retinal vein occlusion.

### II. Case Report:

A 27 years old female patient presented to the ophthalmology emergency department for an acute painless unilateral vision loss.

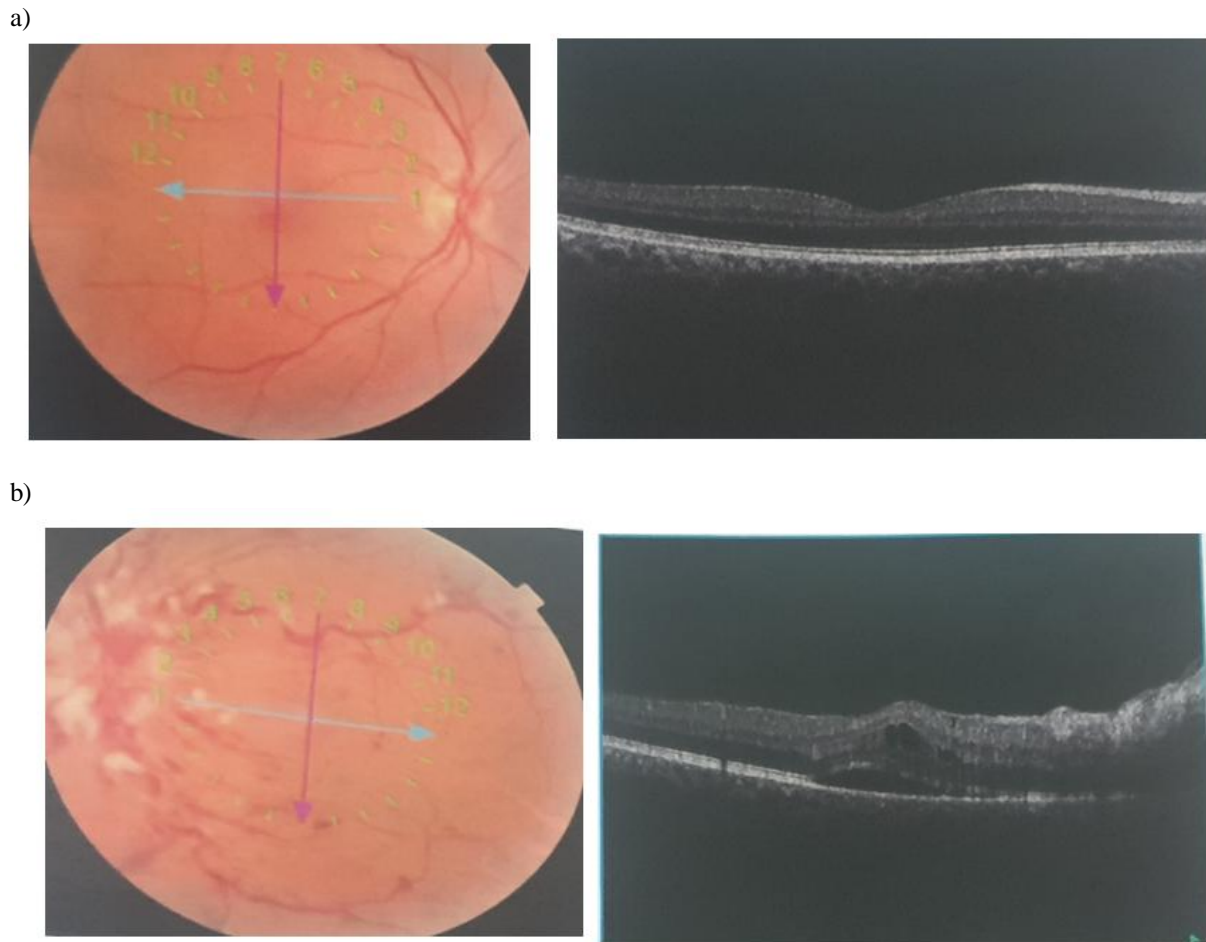
The patient reported a history of fever and night sweat evolving during three weeks with no respiratory or systemic symptoms. There was no premorbidities and no significant family history suggestive of tuberculosis or other illness.

Best-corrected visual acuity in the left eye was counting fingers close to face. A relative afferent pupillary defect was observed. Ocular pressure was within normal range

Anterior segment examination of the left eye revealed fine keratoprecipitates, a mild cell infiltration into the anterior chamber and a clear lens. Dilated ocular fundus examination found no vitreous inflammation, dotted and flame-shaped intraretinal hemorrhages throughout the fundus in all 4 quadrants, engorgement and tortuosity of the major retinal veins, papillary edema, telangiectatic capillary bed, and small cotton wool spots located in the area of the optic nerve head and alongside the vessels. These findings were compatible with a central retinal vein occlusion. The examination of the right eye was normal.

Fundus fluorescein angiography showed hyperfluorescence in the optic disc and a petaloid pattern of fluorescence pooling in the macula indicating cystoid macular edema.

The macular optical coherence tomography revealed cystoid macular edema, sub retinal fluid with serous detachment of the macula and thickening of retina up to 440 microns.



**Figure 1** Radiophotography and macular OCT: a) right eye b) left eye

The patient reported no recent weight loss. On examination, small painless lymphnodes were found in the left cervical chain

Initial investigations showed an inflammatory syndrome with C-reactive protein at 60 mg/l, erythrocyte sedimentation rate at 120 mm/hour and fibrinogen at 6g/ml The blood count cell revealed a hyperleukocytosis at 16000 cell /mm<sup>3</sup>. The results of clotting test were normal.

She had negative results for the following: rheumatoid factor, anti-nuclear antibody, lupus anti-coagulant, IgG and IgM phospholipid antibody, neutrophil cytoplasmic antibody and cry globulins. A virology test for both *Herpes* and *Varicella* IgG and IgM were negative. Cytomegalovirus, toxoplasmosis, VDRL/TPHA and HIV tests were also negative.

The Tuberculin skin test was highly positive with an induration over 15 mm .Chest X-ray showed diffuse small nodular shadows with pleural effusion and hilar lymphadenopathies consistent for tuberculosis. A computed tomography scan of the thorax showed significant lymphadenopathy in supraclavicular region as well as the hilum, and paratracheal regions. The paratracheal lymph nodes were heavily calcified.

Anti-tuberculosis therapy was started with a regimen as following: rifampin, isoniazid, pyrazinamide, and ethambutol for 2 months and continuing rifampicin and isoniazid for four additional months.

Monthly intravitreal bevacizumab injections were instated. After 3 intravitreal injections, fundus findings associated with CRVO improved markedly, and macular edema was resolved

### III. Discussion:

Ocular manifestations of tuberculosis are nonspecific . They are usually associated with systemic disease and are therefore considered to represent a secondary infection. The predominant route by which tubercle bacilli reach the eye is through the bloodstream, after infecting the lung(1).

Intraocular tuberculosis may present as granulomatous anterior uveitis, intermediate mild-to-moderate vitritis with snowball, snow banking, peripheral vascular sheathing, and/or peripheral retinochoroidal granulomas. Posterior uveitis remains the most common presentation including tubercles or multifocal

choroiditis progressing to a diffuse, contiguous pattern called a serpiginous-like lesion. Occlusive retinal vasculitis may also occur and induce neovascularization. Optic neuropathy develops either from direct infection induced by tuberculosis or a hypersensitivity reaction to the infectious agent. It may present as an optic nerve tubercle, papillitis, or papilledema (2).

A first presentation of CRVO attributed to tuberculosis is rare. Few cases associated with underlying pulmonary tuberculosis were reported. Fullerton et al reported the case of a 28-year-old Asian woman with CRVO as the only manifestation of ocular tuberculosis(3). Fountain et al reported as well a case of CRVO associated with pulmonary tuberculosis(4).

Muiz et al described the case of a CRVO with no systemic infection. The diagnosis was established after a polymerase chain reaction (PCR) analysis of vitreous fluid was positive for *Mycobacterium tuberculosis*(5).

Most of these cases of ocular tuberculosis are presumed, as unequivocal evidence of the infection is often not available(6). Ang et al. proposed an approach depending on the clinical situation/suspicion which also impacts in the decision for invasive diagnostic procedures(7). In our case, the suggestive chest Xray findings, associated with the positive tuberculin skin test and the good response to the anti-tuberculosis treatment were in favor of the diagnosis.

In general, the treatment of ocular tuberculosis is the same as for pulmonary tuberculosis. Treatment consists of a four-drug regimen, administered in two phases: rifampicin, isoniazid, pyrazinamide, and ethambutol daily for two months, followed by rifampicin and isoniazid for four months. Corticosteroids are often used to treat intraocular tuberculosis. Oral corticosteroids are often adopted for patients with posterior segment inflammation, whereas topical steroids are used for those with anterior segment inflammation. However, a meta-analysis revealed that the concurrent use of corticosteroids had no significant beneficial effect on treatment outcome(8)

There are few cases reporting the use of intravitreal injection of anti-VEGF as an adjunct to anti-tuberculosis therapy for the management of CRVO in tuberculosis. Intravitreal injection of anti-VEGF improves vascular permeability and reduces progression of retinal non perfusion in CRVO. Yuksel et al reported a case where the anti VEGF therapy was ineffective with little change three months after the anti-VEGF injection and there were vitreous hemorrhages after five months(9). In another case the treatment was not effective for CRVO in tuberculosis-associated uveitis(10). In our case, the treatment was effective inducing the macular edema resorption.

#### **IV. Conclusion:**

Central retinal vein occlusion in a young patient warrants extensive investigation to look for an underlying cause. This case illustrates the possible need for young patients with central retinal vein occlusion to undergo an invasive procedure to reach a definitive diagnosis, especially in areas in which tuberculosis cases show resurgence.

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Raid Soumaya, et. al. “Ocular tuberculosis presenting as a central retinal vein occlusion.” *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, 20(07), 2021, pp. 14-16.