IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Presumed Ocular Tuberculosis About One Case

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Abstract

Ocular manifestations of tuberculosis are rare and diverse, the most common manifestation of ocular tuberculosis is choroiditis.

We describe a patient who presented a unilateral active multifocal choroiditis with panuveitis, and was diagnosed with presumed ocular tuberculosis.

This case shows that the diagnosis of ocular tuberculosis can be made in the presence of a number of clinical and biological epidemiological arguments, and treatment must be started as soon as possible.

Keywords: presumed ocular tuberculosis, multifocal tuberculosis, uveitis.

I. INTRODUCTION

Ocular tuberculosis (TB) is a challenging clinical entity, presenting hurdles in diagnosis and management for both ophthalmologists and infectious disease specialists. Early diagnosis and prompt treatment may be sight-saving in patients with ocular TB.

II. Case report

A 34-year-old patient with notion of tuberculosis contact, presented with a progressive decrease in vision, a painful red eye in the right eye, with a fever and night sweats for 1 month. The ophthalmological examination showed a visual acuity of 1/10 in the right eye and 10/10 in the left eye. The right eye had conjunctival hyperemia with some keratic precipitates, a significant inflammatory response in the anterior chamber, posterior synechiae at 5 hours, and pigment deposition on the lens. The ophthalmological examination in the left eye was normal. Intraocular pressure measured 16 mmHg OU.

Indirect fundoscopic examination revealed vitreitis (1+), papilloedema with a macular edema and occlusive periphlebitis with tortuous vessels (fig 1 B).

Pupillary reactions, extraocular motility and color vision were normal.

A complete blood count, serum calcium, serum lysozyme, serum angiotensin-converting enzyme, aminotransferases, a PPD skin test, serologies: syphilis, cytomegalovirus, toxoplasmosis, HIV, herpes virus, HLA-B27, antiSS-A/Ro and antiSS-B/La antibodies, anti-neutrophil cytoplasmic antibody (ANCA), complement factors levels, rheumatoid factor, antinuclear factor and C-reactive protein were ordered. Cerebrospinal fluid analysis, chest X-ray and chest tomography were also requested. The results of the above blood tests, including serologies, the chest X-ray and chest tomography were all normal. The PPD skin test had an induration size of 30 mm (strong reactor).

The retinal fluorescein angiography shows multiple deep lesions in the right eye, becoming hyper fluorescent. The left eye shows a few scattered hyperfluorescent lesions with a vasculitis and papillitis (Fig 2A and B). Optical coherence tomography shows a macular oedema (fig 3).

A presumptive diagnosis of ocular TB with posterior uveitis and choroidal tubercles was made.

Thereafter, specific treatment for ocular TB with a triple scheme (Isoniazid, Rifampicin and Ethambutol) was initiated. After one week of treatment, a systemic corticosteroid therapy based on prednisone at a dose of 1mg/kg/d was prescribed.

There was significant clinical improvement after 15 days of therapy with the triple strategy. The patient's visual acuity increased and the signs of inflammation, such as vitreitis, were less severe, the vitreous opacification and choroidal tubercles gradually resolved.

The patient was monitored jointly by the Infectology and Ophthalmology

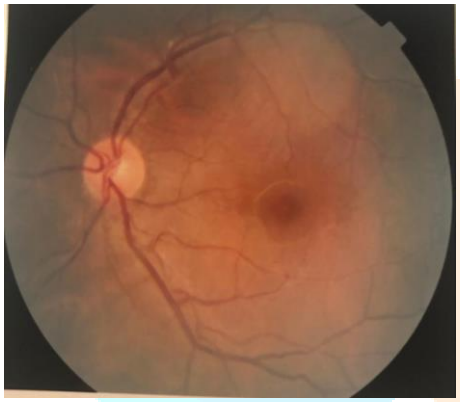

Fig 1 A: Color fundus photograph of the left eye without anomalies.

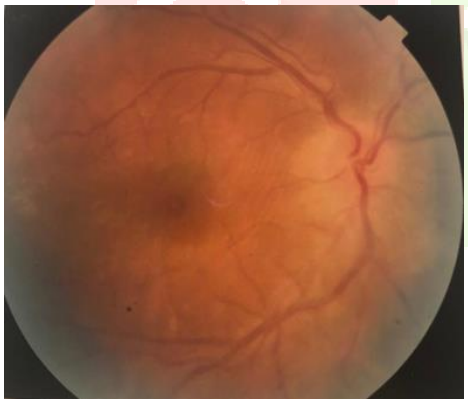

Fig 1B: Color fundus photograph the right eye demonstrating a papillitis

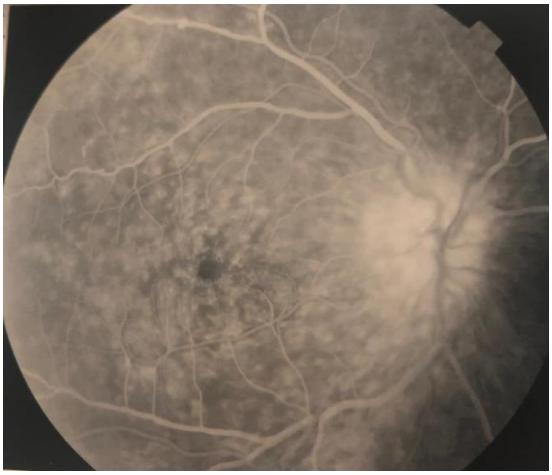

fig 2 A: fluorescein angiography of the right eye shows a macular and papillary oedema and choroidal hyperfluorescent lesions at a late stage.

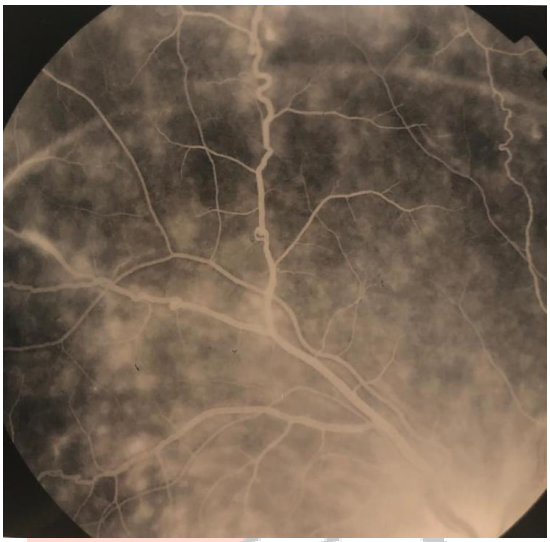

FIG 2 B: fluorescein angiography of the right eye shows vascular diffusion of fluorescein and tortuous vessels.

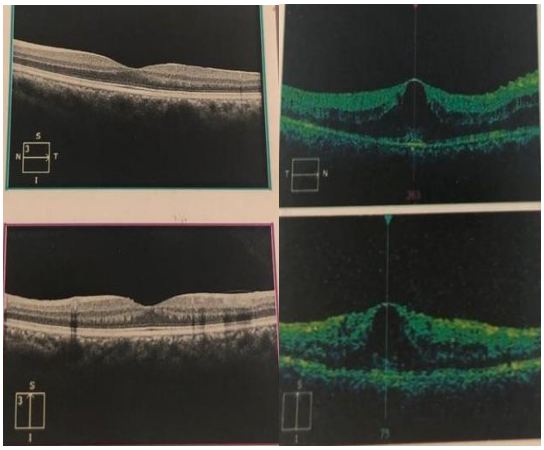

Fig 3: Optical coherence tomography shows a macular oedema in the right eye.

III. Discussion:

Tuberculosis is a chronic infection caused by

Mycobactrium tuberculosis (MT), that is still endemic in Morocco. Eye involvement, however, is quite rare with very variable clinical pictures [1].

Choroidal tubercles were first anatomically described in 1855 and identified with an ophthalmoscope in 1867. One year after the discovery of the organism, TB was identified in the eye in 1883.[2] An autopsy study of military TB in 1950 even reported that eye examination exceeded chest radiography in diagnostic sensitivity.[3]

Tuberculosis is a major cause of morbidity and mortality, with an estimated prevalence of around 25,000 new cases / year [4]. The extra-pulmonary locations of tuberculosis represent 35% of all cases, including 1 to 2% are ocular damage [5].

Approximately one-third of the global population is impacted by tuberculosis. Patients with this condition may experience scleritis, which can manifest as focal, nodular, or diffuse inflammation, with or without keratitis, anterior granulomatous uveitis may occur, in the posterior segment; vitritis, choroiditis, papilledema, macular oedema, retinitis, pseudo-serpiginous choroiditis and other entities can be observed [6]. The diagnosis of ocular tuberculosis is made by isolation of Mycobacterium tuberculosis on Löwestein–Jensen medium or by PCR. The diagnosis is supported by the clinical findings, imaging techniques including optical coherence tomography, fluorescein angiography, indocyanine green and ultrasonography. Tuberculin skin test helps to confirm the diagnosis [7].

Tuberculosis has a proclivity for causing caseating granulomatous inflammation primarily in the lungs, but it can also affect the gamut of organs in the human body.

Additionally, some forms of ocular tuberculosis, such Eales' disease, are thought to be the result of a hypersensitivity reaction. [8.9].

According to Rich's law, the size of a tuberculous lesion is directly linked to the number and potency of the bacteria, as well as the level of sensitivity of the affected tissue. It is also inversely associated with the host's innate and acquired resistance to the pathogen. [10]

The process of diagnosing ocular TB is challenging due to the complications involved in obtaining an ocular sample for microbiologic analysis. Typically, a tentative diagnosis is made based on the identification of acid-fast bacilli under a microscope (known as acid-fast bacilli microscopy) in a diagnostic sample, such as a smear of vitreous aspiration or aqueous. However, a conclusive diagnosis depends on isolating the organism from a diagnostic specimen through a positive culture, which is a laborious and time-consuming procedure [5.11].

The tuberculin skin test is limited due to its low sensitivity and specificity, particularly in nations where the bacille Calmette-Gue´rin vaccine is extensively employed. As a result, one of the most encouraging diagnostic approaches involves amplifying and detecting specific DNA segments using the polymerase chain reaction. This technique was employed to diagnose an M. tuberculosis infection [12]. Polymerase chain reaction is a rapid diagnostic test with high sensitivity and specificity. Moreover, it is especially useful for diagnosing primary ocular TB.

The tuberculin skin test and the Quantiferon-TB Gold are useful screening tests for latent TB especially in selected patients with clinical findings characteristic of intraocular TB and no symptoms or signs suggestive of other origin [13].

Ocular tuberculosis can arise through the hematogenous spread of the pathogen from the primary complex or reactivated post-primary lung lesions, or it may manifest as immune-mediated inflammation. [14]. The exact association of uveitis with latent tuberculosis (TB) is not known. However, once the clinical features and corresponding tests suggest a diagnosis of presumptive ocular TB, a typical treatment regimen is initiated with a combination of both systemic corticosteroids

Addition of anti-tubercular therapy (ATT) to corticosteroids in uveitis patients with latent/manifest TB is known to result in significant reduction in recurrences of uveitis, but there is a lack of clarity regarding the impact and duration of ATT in these cases [15].

As with pulmonary TB, ocular TB often requires multi-drug TB therapy (rifampicin, isoniazid, pyrazinamide, and ethambutol).[16] Treatment for ocular TB is similar to pulmonary TB and may require four-drug therapy for up to two months followed by two-drug therapy (rifampin and isoniazid) for up to four months.[17] The role of steroids for ocular TB (systemic or topical) remains controversial.[17]

A meta-analysis showed that multi-drug anti-TB therapy resulted in 92% improvement in ocular inflammation with 69% improved visual acuity, and 84% without recurrence of their TB after treatment. [17]

Corticosteroids may be required when the inflammation is sight-threatening: posterior, severe, hemorrhagic vasculitis with vitritis and macular edema, which probably indicates an added immune response to bacterial infection. [5.18].

i. CONCLUSION :

Ocular tuberculosis is often a challenging diagnosis. Morocco remains an endemic country for tuberculosis, with various presentations such as ocular of an ophthalmological examination in case of suspicion of tuberculosis and not to dismiss the diagnosis even in the absence of a clinical picture evocative.

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