Tubercular retinal vasculitis A case report

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ABSTRACT
Intraocular tuberculosis (TB) infection can have different clinical manifestations including retinal vasculitis. It more frequently involves the veins and is associated with neovascularisation. The diagnosis may be difficult and presumptive being based on clinical findings and evidence of systemic TB infection. We present a case of a 32-year-old man with a 2 months history of progressive decreased visual acuity and blurred vision in the left eye. His best-corrected visual acuity (BCVA) was 2/10. Fundoscopic examination showed vascular sheathing, neovascularisation and capillary non-perfusion on the left eye, suggestive of retinal vasculitis. Fluorescein angiography revealed focal diffuse staining and late leakage from the retinal vasculature, mainly from the veins corresponding to neovascularisation. Tuberculin skin test and Interferon gamma release assay (IGRA-QuantiFERON-TB Gold) were positive and antituberculosis treatment (ATT) was started. His left eye final BCVA was 9/10, without recurrences over a follow-up of 12 months.

Keywords: Tuberculosis, Vasculitis, Inflammation

INTRODUCTION
Tuberculosis can affect any organ of the body. The initial infection usually occurs in the lung or alimentary tract and is characterised by a primary complex, which is the combination of a focal lesion with a caseating granuloma, and regional lymphadenopathy. Ocular tuberculosis has a very important clinical polymorphism, however some forms are very suggestive of the disease, including retinal vasculitis.

Retinal vasculitis is a form of tuberculosis presentation involving primarily retinal veins in an occlusive pattern[1,2]. The exact prevalence of Intraocular tuberculosis is unknown, but it is reported to be 0.2% to 10.5% among all uveitis patients at tertiary referral eye care centers around the world [3].
A 32 years old Male patient with BCG vaccination and no history of previous systemic illness, presented with a 2 months history of progressive decreased visual acuity and blurred vision in the left eye.

On the ophthalmic examination: BCVA in the left eye was 2/10 and 10/10 in the right one. Slit-lamp biomicroscopy of both eyes revealed no anterior segment inflammation. Fundoscopic examination showed vascular sheathing, neovascularisation and capillary non-perfusion on the left eye, suggestive of retinal vasculitis (Figure 1).

Fluorescein angiography revealed focal diffuse staining and late leakage from the retinal vasculature, mainly from the veins corresponding to neovascularisation (Figure 2).

Optical Coherence Tomography showed a Cystoid macular edema (Figure 3). The chest X-ray was normal.

Both tuberculin skin test and QuantiFERON-TB Gold was positive and the patient had started an antituberculosis treatment with ERIP K4* (Rifampicine, Isoniazid, Pyrazinamide and Ethambutol) and pyridoxine supplementation for 2 months, maintained with isoniazid and rifampin for 4 months. Sectorial laser treatment was performed on retinal ischemic areas. His final visual acuity was 9/10 in both eyes with no recurrences over a follow-up of 12 months

**DISCUSSION**

The World Health Organization estimated that one-third of the world population is infected with Mycobacterium tuberculosis and 10% of infected people have a lifetime risk of manifesting tuberculous disease. TB mostly affects young adults especially immunocompromised individuals due to diabetes mellitus, malnutrition, HIV infection and tobacco smokers [4].

Ocular TB is most likely to occur as part of a post primary infection due to direct haematogenous spread or by hypersensitivity responses. According to this hypothesis, Mycobacterium tuberculosis may persist in the ocular tissues and initiate an immune mediated response that manifests clinically as vasculitis, the latter being usually treated with systemic corticosteroid therapy.

Retinal vasculitis is the commonest manifestation of intraocular TB. It more frequently involves the veins and is characterised by vitreous infiltrates (vitritis), retinal haemorrhages and neovascularisation [5].

Although ocular tuberculosis does not present pathognomonic lesions, some signs during ophthalmic examination may be considered indicators of this etiology. In the presence of anterior granulomatous uveitis, multifocal /serpiginous choroiditis, choroidal nodules or retinal vasculitis, TB should be regarded as a probable cause.

In a retrospective study, Gupta and al analyzed the ophthalmological findings of 386 patients diagnosed with uveitis and concluded that the presence of retinal vasculitis, as well as broad-based posterior synechiae and serpiginous choroiditis, was significantly more frequent in patients with TB uveitis [6].

In fact, peripheral occlusive retinal vasculitis has been associated with TB. Agrawal [7] reported the clinical features and outcomes of 110 patients with peripheral retinal vasculitis and 69 (62.72%) of them had presumed tubercular retinal vasculitis.
It is important to highlight that retinal vascular involvement, especially if occlusive and associated with inflammation, is quite suggestive of ocular TB. It represents a challenging diagnosis since 60% of patients with extrapulmonary TB have no evidence of pulmonary infection.

The gold standard for establishing the diagnosis of TBU is the detection of Mycobacterium TB in ocular tissues or fluids. However, demonstration of the bacillus by smear or culture from ocular samples is seldom achieved because of the low tissue load of M. tuberculosis and small size of ocular tissue biopsy samples [3].

The diagnosis remains presumptive, based on local epidemiologic factors, ocular phenotype, and corroborating immunologic tests (tuberculin skin test), interferon-g release assay (IGRA) (QuantiFERON-TB Gold), or both) especially in selected patients with clinical findings characteristic of intraocular TB and no symptoms or signs suggestive of other origin. Radiologic tests were defined as chest radiography or computed tomography, suggestive of healed or active pulmonary tuberculosis.

TB retinal vasculitis shares with Eales disease extensive peripheral capillary non-perfusion and periphlebitis, although by definition Eales disease is characterised by recurrent vitreous haemorrhages in young adults [6]

The decision to initiate anti-tubercular therapy (ATT) usually is made by the ophthalmologist, in collaboration with pulmonologists and infectious disease physicians, based on local management protocols. [8]

The role of ATT in reducing the rate of recurrences in patients diagnosed with Tubercular uveitis has been well established. However, the lack of international agreement on the minimum clinical dataset required to start ATT in a disease characterized by a wide spectrum of phenotypes indicates an unmet medical need in the management of this disease [9].

The treatment also includes oral corticosteroid therapy.

Neovascularization may complicate the large area of capillary fall-out leading to vitreous haemorrhage and panretinal photocoagulation has been shown to be successful in treating and preventing this complication [10].

**CONCLUSION**

Tuberculosis is a curable disease, it incidence is declining but easily missed in its extra-pulmonary form. The consequences of delay in either ocular or systemic diagnosis can be very serious for the patient.
REFERENCES


ICONOGRAPHY:

Figure 1 Ocular fundus of the left eye with showing neovascularisation, and Perivascular fluffy sheathing
Figure 2: Fluorescein angiography showing capillary non-perfusion, vascular sheathing and focal diffuse staining and leakage from the retinal vasculature.

Figure 3: Optical Coherence Tomography showing cystoid macular edema.