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Case Report

Intraocular Jarisch-Hexreihmer Reaction After Initiation of Antibacillary Treatment in Immunocompetent Patients: About Two Cases

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Abstract

Ocular tuberculosis is a significant infection caused by Mycobacterium Tuberculosis (MTB) that impacts the eye, its surrounding areas, or its surface. We present two definitive cases of ocular tuberculosis where the ocular findings notably worsened following the initiation of anti-tuberculosis treatment. This deterioration is clearly linked to the Jarisch-Herxheimer reaction, which we successfully managed with corticosteroids.

The Jarisch-Herxheimer reaction is a well-documented complication that can occur after the treatment of various infectious diseases. This reaction arises when bacteria are destroyed, releasing toxins that provoke a substantial inflammatory response. It is essential for clinicians to be highly aware of this condition, as prompt recognition and immediate corticosteroid intervention can effectively restore vision. In these cases, there is absolutely no need to discontinue anti-tubercular therapy.

Keywords: Uveitis, Ocular tuberculosis, Jarisch-Herxheimer reaction, Corticosteroids

Introduction

Ocular tuberculosis is defined as an infection caused by Mycobacterium Tuberculosis (MTB) that affects the eye, its surroundings, or its surface [1]. The Jarisch-Herxheimer (JHR) reaction was initially described in patients with syphilis who experienced exacerbations of their skin lesions following treatment with penicillin. This is a temporary clinical phenomenon characterized by systemic manifestations but is rarely observed as an ocular complication [2].

We present two cases of ocular tuberculosis in which the ocular findings worsened after the initiation of anti-tuberculosis treatment. We believe this worsening is attributed to the Jarisch-Herxheimer reaction.

Case Presentation

Case 1: A 28-year-old patient was referred for a progressive painless visual deterioration that had been developing for 06 months, more accentuated in the right eye. Clinical examination in the admission revealed visual acuity of 1/10 (Snellen chart) in the right eye and 7/10 (Snellen chart) in the left eye. Examination of the anterior segment found a clear cornea in both eyes and no sign of anterior uveitis.

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Fundus examination revealed multifocal choroiditis with extensive scar lesions at the posterior pole of the right eye, and serpiginous-like choroiditis in the left eye (Figures 1 and 2). The vitreous examination was unremarkable.

Fluorescein angiography showed inactive scar and atrophic lesions in the right eye and signs of active lesions in the left eye with hypo fluorescence in the center and hyper fluorescence at the edge with peri-papillary location and centrifugally evolution sparing the center of the macula (Figures 3 and 4).

The macular OCT scan revealed the presence of macular atrophy in the right eye and central macular edema with hyper reflectivity of the choriocapillaris related to the foci of choroiditis on the left. The blood count, the inflammatory assessment, and the chest x-ray were normal. The intradermal reaction to tuberculin was phlyctenular at 15 mm, the QuantiFERON –TB GOLD was positive and the rest of the etiological assessment was normal. The diagnosis of intraocular tuberculosis was made and antibacillar treatment was recommended combining Rifampicin (R) 10 mg/kg/day, isoniazid (H) 5 mg/kg/day, ethambutol (E) 15 mg/kg/day and pyrazinamide (Z) 20 mg/kg/day. The evolution was marked by a worsening of the symptoms in the left eye, 48 hours after the initiation of treatment, with acute visual loss: 2/10 (Snellen chart), the presence of a anterior chamber cells grade 3+ of the SUN classification and a severe vitreous haze. Fundus details were not visible. Ultrasound B-scan showed inflammatory foaters in the left eye, with retinal choroid scleral complex thickening.

Bolus of corticosteroid therap: 1g/day for 03 days was initiated, followed by a relay of oral prednisone at a rate of 1mg/kg/day for 01 month then progressive reduction.

The evolution was marked by an improvement of the visual acuity in the left eye measured at 10/10 (Snellen Chart), and the disappearance of signs of uveitis.

Case 2: We report the case of a 25-year-old patient, referred to the ophthalmology department for an acute visual loss in both eyes

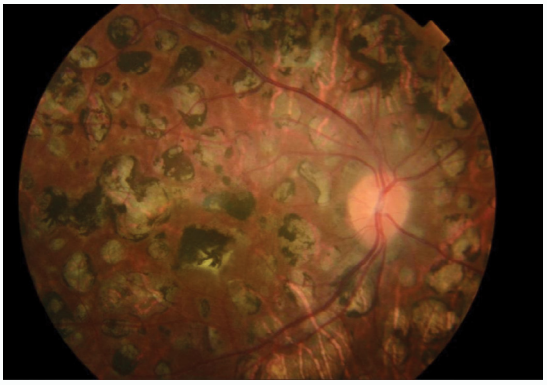

Figure 1: Retinal photography of the right eye showing multifocal choroiditis.

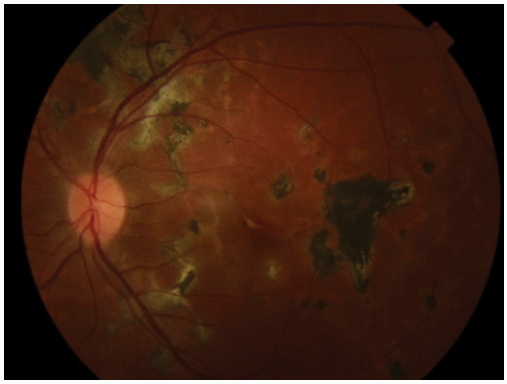

Figure 2: Retinal photography of the left eye showing serpiginous-like choroiditis.

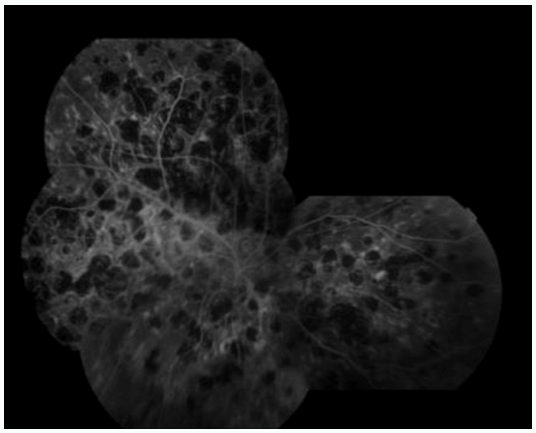

Figure 3: Retinal Retinal angiography of the right eye.

without associated signs. On examination, visual acuity was: 6/10 (Snellen Chart) in the right eye and 10/10 (Snellen Chart) in the left eye. Examination of the left eye was characterized by the presence of signs of non-granulomatous anterior uveitis.

Fundus examination of both eyes revealed the presence of moderate vitreous haze, an exudative, hemorrhagic posterior retinal vasculitis associated with peri phlebitis. There were also retinal hemorrhages in the lower and nasal quadrants of the right eye. There was no papilledema or choroidal lesions. The macula was normal in both eyes. Fluorescein angiography revealed active vasculitis and peri

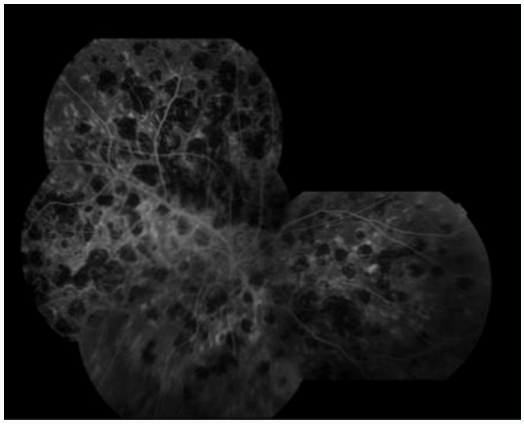

Figure 4: Retinal angiography of the left eye showing active lesions.

phlebitis associated with peripheral sea fan neovascularization in both eyes. Macular OCT scan showed normal macular thickness in both eyes.

Blood tests were positive for QuantiFERON-TB Gold. The Mantoux test was performed and was positive. All other serologies were normal. Chest x-ray was also normal.

The patient was treated with systemic anti-tuberculosis RHZE for two months, then RH and argon laser photocoagulation in the ischemic areas. The evolution was marked by a worsening of clinical symptoms three days after the initiation of treatment, with a drop in visual acuity measured at 5/10th in both eyes, an aqueous flare grade 3+ of the SUN classification. On fundus examination, we noted worsening vitreous haze associated with papilledema in the right eye.

Fluorescein angiography revealed the presence of papillitis in the right eye and the table appearance of the initial lesions in both eyes (Figures 5 and 6). The macular OCT scan was normal in both eyes.

The evolution after the initiation of corticosteroid therapy was marked by good clinical improvement: visual acuity at 10/ 10th in both eyes, disappearance of signs of retinal vasculitis and pre-retinal neovascularization.

Discussion

Ocular tuberculosis can occur through two main mechanisms: hematological, which induces a direct mycobacterial infection, and immunological, which results from a hypersensitivity response to mycobacteria [3].

The Jarisch-Herxheimer reaction is a complication that can arise after the treatment of various infectious diseases. This reaction occurs when the bacilli are destroyed, releasing toxins that trigger an inflammatory response. This reaction can often be managed with corticosteroid therapy [4]. Recent studies suggest that a rapid and substantial stimulation of the patient's immune system could lead to this reaction.

This is distinct from a paradoxical reaction, which is characterized by the clinical or radiological worsening of pre-existing tuberculosis. In this case, new lesions develop that are not due to the normal progression of the disease in a patient who initially shows improvement after anti-tuberculosis treatment [4].

The management of the Jarisch-Herxheimer reaction generally involves administering analgesics and anti-inflammatory medications.

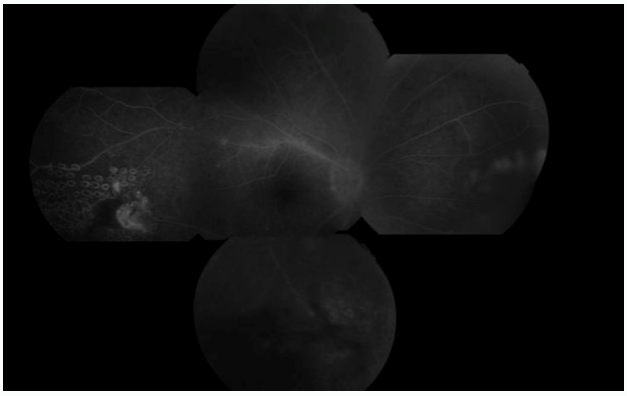

Figure 5: Retinal angiography of the right eye showing active papillitis and vasculitis.

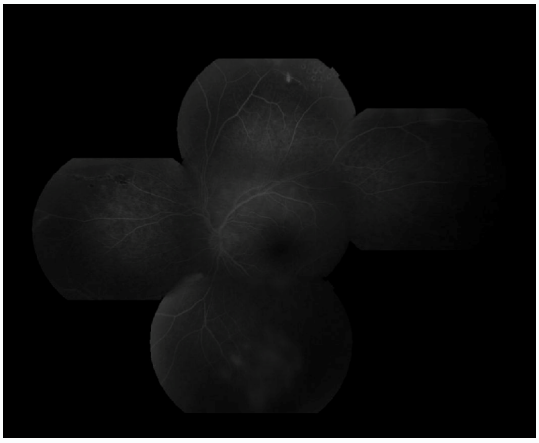

Figure 6: Retinal angiography of the left eye.

However, there are no standardized guidelines regarding the dose and duration of corticosteroid therapy. In situations where systemic steroid therapy is not advisable-such as in patients with uncontrolled diabetes, active or latent infections, severe hypertension, or unstable mental health-intravitreal injection of ranibizumab may be a viable alternative [5].

Conclusion

The ocular JHR is a rare condition that can occur in patients who have been treated for ocular tuberculosis, potentially resulting in sudden vision loss. Clinicians need to be aware of this condition because prompt recognition and timely initiation of corticosteroid treatment can help restore vision. In these cases, discontinuing anti-tubercular therapy may not be necessary.

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