*Rev. Inst. Med. Trop. Sao Paulo*
*54(4):229-230, July-August, 2012*
*doi: 10.1590/S0036-46652012000400008*

## CASE REPORT

# OPTOCHIASMATIC TUBERCULOMA AS THE SOLE MANIFESTATION OF LATE RECURRENT TUBERCULOSIS

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**SUMMARY**

Brain tuberculomas account for 10-20% of space occupying brain lesions in developing countries. Most lesions are observed at time of tuberculosis diagnosis or soon after starting treatment. We herein describe a 32 year-old patient with a 14-month history of headache and progressive visual loss. Her past medical history revealed pulmonary tuberculosis treated eight years before. A brain MRI showed a T1- and T2-weighted isointense contrast-enhancing lesion in the optic chiasm. A presumptive diagnosis of optochiasmatic tuberculoma was made and isoniazid, rifampin, pyrazinamide, and ethambutol were started. Despite treatment, the patient evolved to blindness. The prompt recognition of this condition is extremely important since the presence of optochiasmal enhancement is associated with blindness in patients with tuberculosis.

**KEYWORDS:** Tuberculosis; Meningitis; Optic chiasm.

## INTRODUCTION

Brain tuberculomas are caseous foci with fibrous encapsulation resulting from hematogeneous dissemination of bacilli. They account for 0.15-4% of space occupying brain lesions worldwide[6], but can reach 10-20% in developing countries[7]. Most lesions are observed at time of tuberculosis diagnosis, either meningeal or systemic, or soon after starting treatment (paradoxical reaction). They are present in 1% of patients with active tuberculosis and up to 28% of patients with tuberculous meningitis[4]. The inflammatory reaction observed in central nervous system tuberculosis has a strong predilection for basal parts of the brain, especially in interpeduncular, suprasellar and Sylvian cisterns, which can result in optochiasmatic arachnoiditis and tuberculoma[2]. Optochiasmatic arachnoiditis and tuberculoma are devastating forms of tuberculous meningitis and are often associated with blindness or severe vision impairment. We herein describe a patient with optochiasmatic tuberculoma as the sole manifestation of tuberculosis reactivation eight years after treatment of pulmonary tuberculosis.

## CASE REPORT

A 32 year-old lady was seen at the outpatient brain tumor clinic at the National Institute of Cancer (Rio de Janeiro, Brazil) due to a possible optic chiasm glioma. She reported progressive bilateral visual loss over 14 months associated with intense headache and vomiting in the last month. She denied fever or any other systemic or neurologic complaints. The neurologic examination was unremarkable and the first ophthalmic examination revealed no perception of light in the left eye and vision 20/60 in the right eye. Her past medical history revealed pulmonary tuberculosis treated eight years before. At that time, culture from the sputum was positive for *M. Tuberculosis* in the sputum and the isolated strain was sensitive to isoniazid, rifampicin, pyrazinamide, ethambutol and etionamide. She was treated with isoniazid, rifampicin and pyrazinamide for six months with good compliance and improvement of the symptoms. HIV serology was negative at that time as well as at the onset of visual complaints.

A brain MRI showed a T1- and T2-weighted isointense contrast-enhancing lesion in the optic chiasm (Fig. 1). A lumbar puncture revealed mild pleocytosis (8 cells/mm$^3$), protein 55 mg/dL and glucose 40 mg/dL. Stains and cultures for bacteria, mycobacteria and fungi were negative. A lung CT scan did not reveal any active tuberculosis lesions. A presumptive diagnosis of optochiasmatic tuberculoma was made and a four-drug regimen (isoniazid, rifampin, pyrazinamide, and ethambutol) was started. The headache and vomiting quickly resolved but despite tuberculosis treatment and steroids she evolved to blindness. Fourteen months later a new MRI showed complete resolution of the lesion.

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Conflict of Interest: none.
Financial Support: none.
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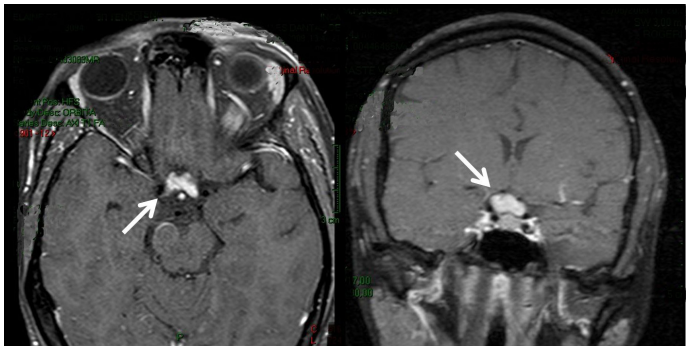

**Fig. 1 -** Axial (A) and coronal (B) contrast enhanced T1-weighted MRI sequences showing an optochiasmatic tuberculoma (white arrows).

## DISCUSSION

Vision loss occurs in about 10% and impairment in 27% of patients with central nervous system tuberculosis[1]. It is usually secondary to hydrocephalus, optochiasmatic arachnoiditis, optic nerve involvement, tuberculomas affecting the optochiasmatic region and ethambutol toxicity[8]. Optochiasmatic tuberculomas are uncommon and can be present at diagnosis of pulmonary tuberculosis or develop paradoxically during treatment[1] raising the question of treatment failure. According to GARG *et al.,* a multivariate logistic regression analysis revealed that younger age, female gender, and an elevated cerebrospinal fluid protein content are associated with development of optochiasmatic tuberculomas[2].

An important issue is that as brain tuberculomas can present without concomitant meningeal involvement, as observed in our patient, the diagnosis becomes even more difficult. They can be easily confounded with other space-occupying lesions such as brain abscess, sarcoidosis, metastasis or primary brain tumors, especially if the diagnosis of tuberculosis was not established in other sites[7]. Biopsy may be required in such cases. Stereotatic biopsy may be superior to open craniotomy since it is associated with lesser complications and its diagnostic efficacy is around 85%[3].

Steroids have been traditionally used in attempt to reduce the inflammatory reaction and secondary neurologic damage, but despite treatment patients can evolve to complete visual loss. Recently, some authors have used thalidomide with improvement of brain lesions and recovery of vision[5].

The prompt recognition of this condition is extremely important since the presence of optochiasmal enhancement is associated with blindness in patients with tuberculosis.

## RESUMO

### Tuberculoma opto-quiasmático como única manifestação de tuberculose recorrente tardia

Tuberculomas cerebrais são responsáveis por 10-20% das lesões parenquimatosas em países em desenvolvimento. A maioria destas lesões é observada ao diagnóstico de tuberculose ou logo após o início do tratamento. Descrevemos um caso de uma paciente de 32 anos com história de 14 meses de evolução de perda visual progressiva e cefaleia. A história patológica revelou tuberculose pulmonar 8 anos antes. A ressonância magnética do crânio mostrou uma lesão isointensa nas sequências T1 e T2 captantes de contraste no quiasma óptico. Fizemos o diagnóstico presuntivo de tuberculoma ótico-quiasmático e inciamos isoniazida, rifampicina, pirazinamida e etambutol. Apesar do tratamento, a paciente evoluiu para amaurose bilateral. O rápido diagnóstico desta condição é extremamente importante já que a presença de captação de contraste está associada à amaurose em pacientes com tuberculose.

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Received: 12 December 2011
Accepted: 17 April 2012