

Optochiasmatic Tuberculomas: A Vision-Threatening Paradoxical Response in Tuberculous Meningitis

Optokiazmatik Tüberküloma: Tüberküloz Menenjitte Karşı Oluşan Beklenmedik Yanıta Bağlı Olarak Görmenin Tehlikeye Girmesi

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ABSTRACT

The single main cause of death from infectious diseases worldwide is tuberculosis, which is more prevalent in developing countries. Tuberculous meningitis (TBM) is a severe form of TB in which morbidity and mortality depend upon the stage of presentation. Intracranial brain parenchymal tuberculomas may form paradoxically, days to months after starting antituberculous drug therapy. Tuberculomas may develop in and around the optic chiasm and optic nerves after institution of antituberculous treatment as a quite rare occurrence in TBM; this may lead to severe visual loss if not treated properly. We describe a 56-year-old man with documented TBM being treated with first line antituberculous drugs who developed visual impairment 2 months after starting the treatment. MRI after gadolinium administration revealed multiple perichiasmatic and a few parenchymal ring enhancing lesions due to tuberculomas. The patient was treated with corticosteroids and antituberculous therapy was continued after which he recovered his vision gradually. Visual impairment developing in a patient on treatment with antituberculous drugs should give rise to a suspicion of rare optochiasmatic tuberculomas; this necessitates urgent contrast-enhanced MRI of the brain and prompt treatment with steroids.

KEYWORDS: Optochiasmatic tuberculoma, Paradoxical response, Tuberculous meningitis, Tuberculoma, MRI

ÖZ

İnfeksiyöz hastalıklardan ölümlerin en sık görülen nedenlerinden birisi olan tüberküloz hastalığı gelişmekte olan ülkelerde daha sık görülmektedir. Tüberküloza bağlı gelişen menenjit, tüberküloz hastalığının ciddi bir şeklidir. Menenjit gelişen tüberküloz hastalığında morbidite ve mortalite hastanın geliş bulgu ve semptomlarına bağlıdır. Beyin parankimi tutulumu gösteren tüberküloz hastalığında beklenmedik bir şekilde beyinde tüberküloz oluşumu antitüberküloz tedaviden günler veya aylar sonra ortaya çıkabilir. Tüberküloz menenjitinde oldukça nadir olmakla beraber optik kiazma ve optik sinirler çevresinde nadiren tüberkülomlar gelişebilir ve bu tutulum sonucunda uygun tedavi yapılmaz ise görme yeteneği kaybolur. Birinci basamak antitüberküloz tedavisinden 2 ay sonra görme bozukluğu gelişen 56 yaşındaki erkek hasta olgu olarak sunulmaktadır. Gadolinyumlu manyetik rezonans görüntülemesi yapılan olguda perikiazmatik bölgede çok sayıda, beyin parankiminde ise daha az sayıda olmak üzere halka şeklinde kontrastlanma gösteren tüberkülomla uyumlu lezyonlar saptanmıştır. Hastaya kortikosteroid tedavisi başlanmış, antitüberküloz tedavisi sürdürülmüştür, hastanın görme kaybı tedavi ile tedrici olarak düzelmiştir. Antitüberküloz tedavi alan hastalarda görme kaybı gelişmesi durumunda optokiazmatik tüberküloma gelişimi düşünülmeli ve acil olarak beyinden kontrastlı manyetik rezonans görüntüleme yapılmalı ve en hızlı şekilde steroid tedavisine başlanmalıdır.

ANAHTAR SÖZCÜKLER: Optokiazmatik tüberküloma, Beklenmedik yanıt, Tüberküloz menenjit, Tüberkülom, MRG

INTRODUCTION

Central nervous system (CNS) involvement is seen in about 10% of patients with tuberculosis, and this is more common in developing countries (3, 10). CNS tuberculosis may manifest in diffuse form with meningitis or in focal form as intracranial tuberculomas (3). Neuro-ophthalmologic complications of tuberculous meningitis include oculomotor nerve palsy, pupillary abnormalities and optic disc changes of papillitis, papilledema, or optic atrophy (1). These complications are seen in the late stage of tuberculous meningitis (TBM), usually manifesting in the elderly (5). Intracranial tuberculomas may enlarge or develop afresh as a paradoxical response

to ongoing antituberculous chemotherapy for tuberculous meningitis (TBM). Tuberculomas may rarely develop during treatment of TBM around the anterior optic pathway including the optic nerve and optic chiasm, threatening vision (8). We describe paradoxical development of tuberculomas in the anterior optic pathway demonstrated by MRI in a case of TBM who complained of progressive loss of vision during the 2nd month of antituberculous therapy (ATT).

CASE REPORT

A 56-year-old man, taking ATT since 2 months for tuberculous meningitis, presented with progressive diminution of vision in both eyes and headache for the last 15 days. He had

started taking ATT as a combination of rifampicin, isoniazid, pyrazinamide and ethambutol. The patient reported full compliance with the antituberculous drug regime and his clinical condition improved gradually. After the initial improvement with ATT, he had suffered loss of vision 15 days ago. On examination at presentation with diminished vision, fundoscopy showed bilateral papilledema. Visual acuity was decreased in both eyes with best corrected visual acuity of 20/200 in right eye and 20/200 in the left. Visual field testing (perimetry) depicted visual loss in bilateral temporal fields. Bilateral oculomotor nerve palsy was present with pupils sluggishly reacting to light on both sides. His serology was negative for human immunodeficiency virus (HIV). Lumbar puncture revealed *Mycobacterium tuberculosis* within the cerebrospinal fluid (CSF) on culture to be sensitive to the first line antituberculous drugs the patient was taking. MRI brain was performed on a 1.5 tesla MR system using T1-weighted (T1W) and T2W sequences in axial, coronal and sagittal planes; this was followed by intravenous administration of gadolinium containing contrast agent and subsequent T1W imaging in all the planes. MRI showed multiple rounded lesions with hyperintense signal intensity wall on T1W sequence in the suprasellar and perimesencephalic cisterns (Figure 1). T2W images showed innumerable round lesions filling suprasellar and perimesencephalic cisterns; multiple small rounded lesions were seen in the brain parenchyma also with vasogenic edema in midbrain and in both temporal lobes (Figure 2). Post-gadolinium enhanced T1W images depicted multiple ring enhancing lesions clustered around optic chiasma in the suprasellar cistern and in the sylvian fissures and perimesencephalic cistern and brain parenchyma (Figure 3, 4, 5). Clinical features and imaging findings were compatible with a diagnosis of TBM with optochiasmatic tuberculomas resulting in neuro-ophthalmologic manifestations, possibly due to paradoxical response to treatment. Patient was continued with he ATT and intravenous dexamethasone was added that was followed by oral prednisolone. Steroids were continued at full dose for 8 weeks and then tapered gradually in 4 weeks as the patient refused surgical intervention. He was advised to continue ATT for 24 months. Visual acuity started to improve after combined antituberculous and steroid treatment; his best corrected visual acuity was 20/60 in the right and 20/70 in the left eye 5 months later. CSF was normal and no *Mycobacterium tuberculosis* could be grown at 6 months. After stopping steroids and with continued ATT, the patient had no ocular muscle weakness at 8 months.

DISCUSSION

Visual loss in TBM resulting from involvement and inflammation of the optic chiasm and optic nerves is a rare occurrence (7, 9). Visual loss developing during treatment of TBM may be related to drug toxicity and arachnoiditis besides tuberculomas involving the anterior optic pathway. The optochiasmatic region may be a rare location involved by paradoxical development of intracranial tuberculomas during ATT for CNS or pulmonary tuberculosis (6, 7, 9).

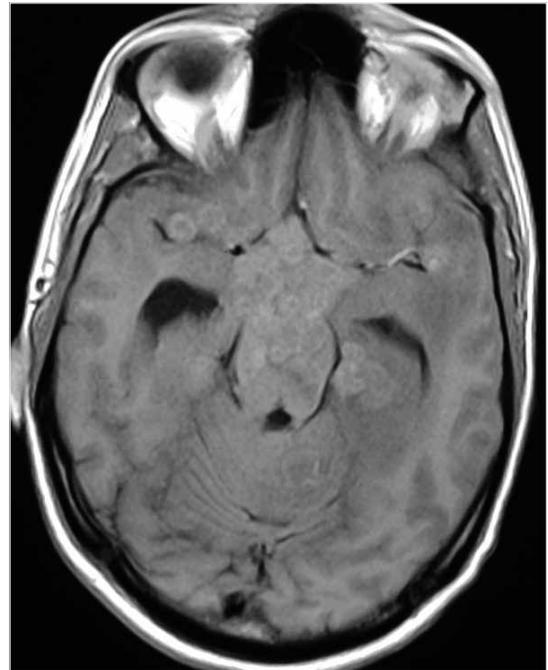


Figure 1: Axial T1-weighted MR image of brain showing multiple rounded lesions in the suprasellar cistern with hyperintense signal intensity wall and hypointense center.

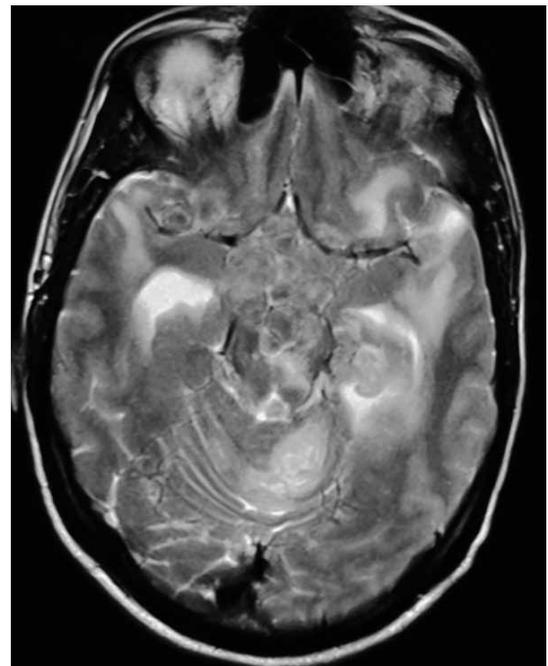


Figure 2: Axial T2-weighted MR image shows multiple rounded lesions around midbrain predominantly in anterior CSF space; vasogenic edema is seen in temporal lobes and in midbrain.

Pathogenesis of this paradoxical development and/or expansion of tuberculomas has been attributed to exaggerated host reaction to tuberculous protein in a patient being successfully treated with ATT (3, 8). Role of corticosteroids in controlling tuberculomas in this situation is



Figure 3: Post gadolinium-enhanced T1-weighted MR image showing multiple ring enhancing lesions around the optic chiasma and fewer rings in the Sylvian fissures and in brain parenchyma.

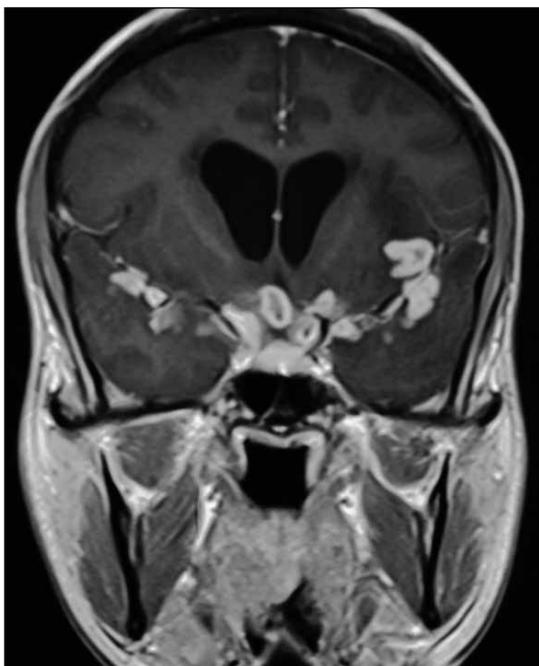


Figure 4: Contrast-enhanced T1-weighted coronal MR image showing ring enhancing lesions in suprasellar cistern around optic chiasm.

more likely due to an effect on this host response rather than reduction of associated edema. The paradoxical response resulting in optochiasmatic tuberculomas has important diagnostic and therapeutic implications (3, 7, 9).

Early recognition of this condition and continued ATT and



Figure 5: Sagittal contrast enhanced MR image showing suprasellar ring enhancing lesions in perichiasmatic location.

corticosteroid therapy or surgical decompression of optic pathway may help in preserving the visual function of the patient (3, 6, 9). The treating neurologist and ophthalmologist need to be aware of such a rare occurrence of paradoxical optochiasmatic tuberculomas as a cause of visual impairment in the spectrum of tuberculosis and its treatment. This awareness and proper clinical judgment helps in timely recognition of this phenomenon in a patient on ATT who needs to be closely followed (6). Satisfactory initial response to ATT, no associated systemic features and persistent sensitivity of isolated *Mycobacterium tuberculosis* to the instituted first line antituberculous drugs favoured a diagnosis of paradoxical response against progressive drug resistant infection in our case.

MRI helps in early detection of intracranial tuberculomas which are seen as ring enhancing lesions after contrast administration. Intracranial tuberculomas are usually parenchymal and extra-axial location in CSF spaces is exceedingly rare. Optochiasmatic tuberculomas may be intrachiasmatic or perichiasmatic in location; this differentiation is difficult even with MRI (3). Multiple ring enhancing lesions due to tuberculomas on MRI need to be differentiated from neurocysticercosis, pyogenic abscesses, metastasis and toxoplasmosis. Clinical features, serology and CSF findings are helpful besides imaging to arrive at proper diagnosis. Optochiasmatic tuberculomas also need to be considered in the differential diagnosis of optic nerve and perichiasmatic tumors in view of prevalent TB (2). Using MRI, the exact location and relationship of tuberculomas with respect to the optic nerve and chiasm can be determined. This helps in planning surgical decompression of optochiasmatic tuberculomas threatening vision.

For the treatment of paradoxical development of tuberculomas, antituberculous drugs are continued as usual and systemic corticosteroids are added (3, 8). Steroids overcome the inflammatory response of the host body to the breakdown products of killed *Mycobacterium tuberculosis* (4). ATT is continued up to 24-30 months, though a shorter course has been reported to be effective as well (3). Corticosteroid therapy is continued for 6-8 weeks and withdrawn after gradual tapering. Failure of medical therapy is an indication for urgent surgical decompression (6). Timely decompression of optochiasmatic region is important for a favorable visual recovery. Prompt surgical intervention for tuberculomas of the anterior optic pathway is facilitated by MRI by providing a roadmap.

CONCLUSION

Paradoxical development of optochiasmatic tuberculomas may present as visual impairment in a patient on ATT for TBM. MRI demonstrates ring enhancing lesions around the optic chiasm and aids in the management by helping in timely diagnosis and follow-up. Corticosteroid therapy may achieve satisfactory decompression and preserve vision.

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