

Optic perineuritis simultaneously associated with active pulmonary tuberculosis without intraocular tuberculosis

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Dear Editor,

Optic perineuritis is a rare inflammatory disorder involving the optic nerve sheath and its adjacent tissues. This condition shows dramatic responses to steroid treatment, although relapse is common. Additional characteristics of optic perineuritis include the sparing of central vision on a visual field test, relatively good initial visual acuity, and optic disc edema^[1]. Most cases are idiopathic, although some rare cases have been associated with specific infections, such as syphilis and viral infections, and autoimmune disorders including Wegener's granulomatosis, sarcoidosis, and giant cell arteritis^[1-2]. The presentation of optic perineuritis has also been described during tuberculosis treatment^[3]. However, optic perineuritis simultaneously associated with active pulmonary tuberculosis has never been published. Here, we report the case of an otherwise healthy woman who presented with optic perineuritis associated with active pulmonary tuberculosis without intraocular tuberculosis.

A 39-year-old woman presented with ocular pain and visual loss in her right eye lasting for one week. Her medical history was otherwise unremarkable, with no immunological problems. Her corrected visual acuity was 20/20 in both eyes, with a right afferent pupillary defect. The anterior segment and vitreous were normal with no cell infiltration. Color vision was intact in each eye. Ocular motility was within normal limits, but severe pain was reported during gazing. Fundusoscopic examination demonstrated optic disc edema in the right eye (Figure 1). Automated visual field testing showed a superotemporal field defect and reserved central field in the right eye (Figure 2). Magnetic resonance imaging (MRI) demonstrated an enhanced

posterior sclera and weakly enhanced optic nerve sheath in the right eye (Figure 3).

Cerebrospinal fluid analysis did not show evidence of infection or malignancy. Laboratory tests, including those to measure erythrocyte sedimentation rate, C-reactive protein, anti-nuclear antibody, angiotensin-converting enzyme, anti-neutrophil cytoplasmic antibody, complement, syphilis and viral serology, renal and liver function, and other blood parameters, showed normal results. Chest radiology revealed an ill-defined focal patchy consolidation in the right apex (Figure 4A). High-resolution computed tomography of the chest showed multiple centrilobular nodules in the right apex (Figure 4B). These findings were suggestive of active pulmonary tuberculosis. Repeated acid-fast bacillus smear and culture were negative, although real-time polymerase chain reaction of bronchial wash material showed a positive diagnostic result for *Mycobacterium tuberculosis*.

A diagnosis of optic perineuritis associated with active pulmonary tuberculosis was made. The patient was treated with 1.0 g of intravenous methylprednisolone every day for 3d, followed by oral prednisolone 1 mg/kg·d for 11d with subsequent tapering for 6wk. In addition, treatment for pulmonary tuberculosis was initiated with the combination of isoniazid, rifampin, ethambutol, and pyrazinamide. Ten days after undergoing treatment for pulmonary tuberculosis, drug-induced hypersensitivity to isoniazid and rifampin developed. Eventually, the medications for pulmonary tuberculosis were replaced by pyrazinamide, kanamycin, and levofloxacin.

The patient's symptoms of ocular pain and visual loss started to dramatically improve after she received intravenous methylprednisolone for 3d. Two months after her initial presentation, the optic disc edema had resolved and her visual field was recovered. The patient has been observed for 12mo without ocular complications and visual or tuberculosis relapse.

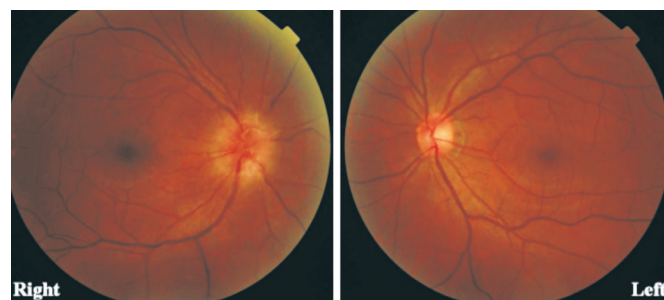


Figure 1 Right disc edema with a normal left optic disc.

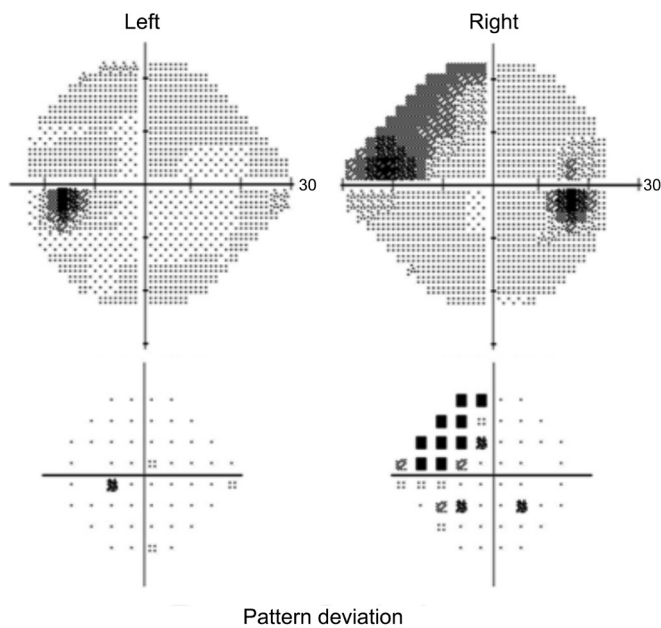


Figure 2 The automated visual field shows a superotemporal defect in the right eye and a normal visual field in the left eye.

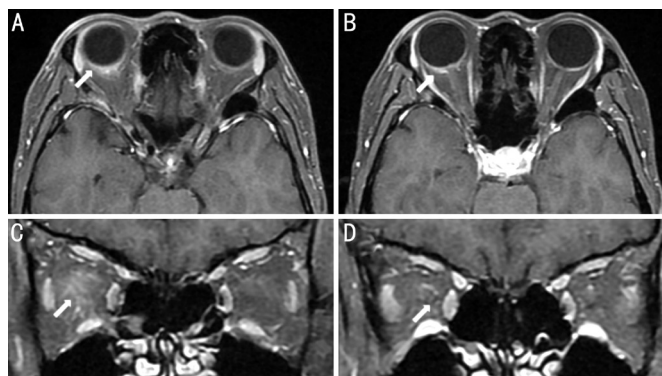


Figure 3 Magnetic resonance imaging scans of the orbit, with contrast infusion and fat suppression, showing an enhanced posterior sclera (A-C) and weakly enhanced optic nerve sheath (D).

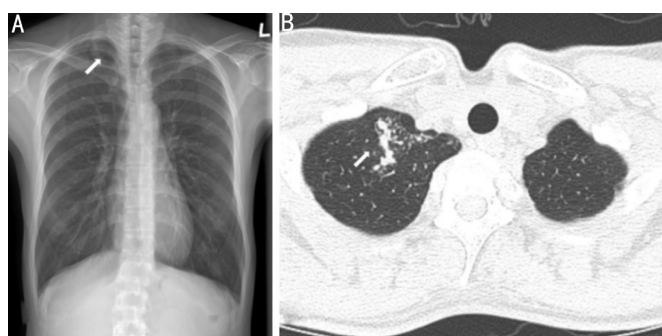


Figure 4 Chest radiograph showing ill-defined focal patchy consolidation in the right apex (A), and high-resolution computed tomography of the chest demonstrates multiple centrilobular nodules in the right apex (B).

Optic perineuritis can be difficult to clinically differentiate from optic neuritis, as both disorders may present with acute visual loss with eye pain, pain with ocular movement, and a swollen optic disc^[1]. MRI findings of enhancement around the optic nerve, termed the “tram tract” on axial cuts and a “doughnut” on coronal cuts, can aid in diagnosing optic perineuritis and

differentiate this condition from optic neuritis^[1-2]. In the patient described here, enhancement around the optic nerve was mild. However, MRI were showed enhanced posterior sclera, suggesting orbital inflammation. Ohtsuka *et al*^[4] reported that enhancement of the adjacent posterior sclera was observed in a patient with optic perineuritis. In addition, this patient clinically presented with acute visual loss, pain with ocular movement, and optic disc edema, as well as sparing of the central vision and normal color vision. These findings are typical clinical features of optic perineuritis^[1], and this patient dramatically responded to steroid treatment.

The majority of optic perineuritis cases are idiopathic, and causes related to inflammation or infection are very rare^[1]. Jacob *et al*^[3] reported a case that presented with optic perineuritis during treatment for tuberculosis, and the authors presumed that the etiology may have been caused by dysimmune neuropathy rather than infection or toxicity due to anti-tuberculosis treatment. In our case, optic perineuritis simultaneously presented with active pulmonary tuberculosis in an otherwise healthy adult; therefore, we believe that this case differs from that of Jacob *et al*^[3], with each case showing a different pathogenesis.

The mechanism for the simultaneous presentation of optic perineuritis and pulmonary tuberculosis in this case is unknown. However, posterior scleritis can occasionally be caused by systemic infection, and inflammation of the posterior sclera may extend to the optic nerve sheath. Gupta *et al*^[5] reported a patient with isolated posterior scleritis and tuberculosis in the cervical lymph node; their case also seemed to manifest as intraocular tuberculosis, although our patient did not exhibit intraocular inflammation.

This is the first reported case of optic perineuritis simultaneously associated with active pulmonary tuberculosis without intraocular tuberculosis. In particular, this condition developed in an otherwise healthy and immunocompetent adult. Thus, it is important to be aware of accompanying infective or inflammatory disorders when diagnosing optic perineuritis.

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