CASE REPORT

Pulmonary Tuberculosis with Neuromyelitis Optica: An Uncommon Association of a Common Disease

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ABSTRACT

Systemic tuberculosis has been reported with varying neurological manifestations like meningitis, tuberculomas, myositis and neuropathy. Neuromyelitis optica (NMO) is a well known neurological entity which has been described in association with several systemic disorders like systemic lupus erythematosis, diabetes mellitus, hypothyroidism, exposure to insecticides etc. However, only a few cases of NMO have been reported in association with *Mycobacterium tuberculosis*. Here, we report a case of pulmonary tuberculosis in association with NMO to highlight the under-reported association of NMO with pulmonary tuberculosis presenting in a peculiar anatomical fashion i.e. longitudinal myelitis with predominant posterior column involvement.

Key words: *Pulmonary tuberculosis. Neuromyelitis optica. Optic neuritis. Transverse myelitis.*

INTRODUCTION

Neuromyelitis optica or Devic's disease is a rare disease which affects the optic tract and the spinal cord. It is characterized by transverse myelitis with successive or simultaneous optic neuritis which can either be monophasic or multiphasic. It has been described in association with many systemic disorders including pulmonary tuberculosis. Its association with evolving pulmonary tuberculosis, however, has been noted in a handful of case reports only.[1-5]

We report this particular case, to highlight an uncommon association of a common disease presenting in an extremely unusual fashion i.e., longitudinal myelitis with posterior column involvement.

CASE REPORT

A 46 years old, hypertensive and diabetic male, presented with a history of painless visual loss in right eye 2 months ago. Fifteen days after the onset of initial symptom, he had developed visual loss on the left side as well accompanied by right sided weakness without facial involvement or sphincter or any sensory impairment. Systemic review was significant for dry cough and weight loss. He was taking anti-tuberculous therapy (ATT) for a week and had a positive sputum smear for *Mycobacterium tuberculosis* (MTB). He had been treated successfully for pulmonary tuberculosis 2 years ago.

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His neurological examination revealed optic atrophy and complete visual loss in the right eye and relative afferent papillary defect, papilloedema with preservation of light perception on the left side. Examination of extremities showed right hemiparesis and severe proprioceptive loss with contralateral loss of pin prick sensation, consistent with Brown Sequard syndrome. Investigations revealed a high ESR (67 mm after first hour) with mild normochromic normocytic anaemia (11 gm/dl). Serum levels of calcium, albumin, LDH and ANA were within normal ranges. Sputum smear for AFB was positive.

MRI cervical spine showed high signal intensity areas on T2WI mainly involving posterior column extending from 2nd to 4th cervical spine level (Figure 1) with marginal enhancement on post-gadolinium images. MRI brain with gadolinium showed enhancement of optic chiasma (Figure 2) with normal brain parenchyma. Analysis of cerebrospinal fluid (CSF) was only remarkable for raised

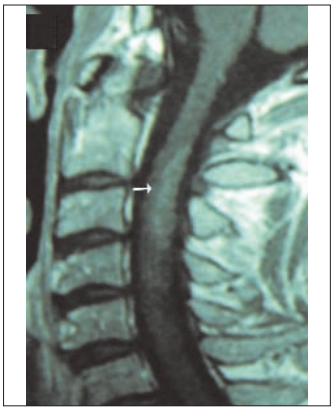

Figure 1: T-1 weighted post-gadolinium sagittal image showing patchy enhancement in the cervical cord extending from C2 to C4 more marked posteriorly with sparing of anterior column.

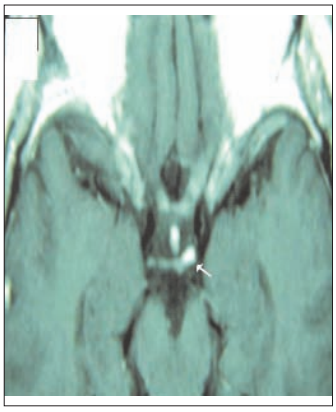

Figure 2: T-1 weighted post-gadolinium axial image showing enhancement of extra canalicular part of optic nerve and optic chiasma.

protein (74 mg/dl). Oligoclonal bands, AFB smear and MTB PCR were not detected in the CSF. Serum antinuclear antibody (ANA) was negative.

A diagnosis of neuromyelitis optica was made based on Wingerchurk's criteria.[7] He was started on high dose methylprednisolone, while ATT was continued. On follow-up visit at 2 weeks after discharge, he had some improvement in the motor strength and proprioception, though there was no improvement in the vision.

DISCUSSION

NMO is a severe demyelinating syndrome characterized by acute optic neuritis and transverse myelitis, either successive or simultaneous, sparing the cerebral white matter. The illness can be monophasic, multiphasic or relapsing in nature. Since, its first description in 1894 by Devic, NMO remained controversial as a separate entity or as a variant of multiple sclerosis.

Collagen vascular disorders like systemic lupus erythematosus; endocrinopathies like diabetes; and exposure to insecticides and systemic infections like pulmonary tuberculosis have been implicated as the possible associations of NMO.[6] Wingerchurk's criterion,[7] has been traditionally used for the diagnosis of NMO even in the presence of tuberculosis as well.[8] The criterion includes clinical evidence of events involving optic nerve(s) and spinal cord along with 2 out of the 3 of the following parameters: long extensive spinal cord lesions (> 3 spinal segments), brain MRI normal or not meeting criteria for multiple sclerosis or NMO IgG seropositive status.

Sporadic reports of NMO in association with pulmonary tuberculosis have been published. The CSF is often inflammatory with mild to moderate polymorphonuclear pleocytosis, though; *Mycobacterium tuberculosis* had not been isolated.[6] MRI of spinal cord is typically abnormal with high signal intensity areas on T2WI and contrast-enhancement with or without oedema/ cavitation in the affected areas. However, predominant involvement of posterior column, as seen in our patient has not been described before. Optic nerves can also show contrast-enhancement on T1WI. MRI brain is essentially normal or shows non-specific changes.[6] This patient had both of these reported radiological findings i.e. an enhanced-optic nerve as well as a normal MRI brain.

NMO carries a poorer prognosis;[3] especially when seen concomitantly with a systemic illness, in this case, recurrence of pulmonary tuberculosis. Though, the functional motor status has been shown to improve, the visual loss remained unchanged in most of the reported cases, as observed in our patient.

The possible mechanism of this association may be a direct tuberculous involvement of the nervous system or a reaction to antituberculous therapy. However, lack of evidence for direct invasion of CSF or brain by the *Mycobacterium tuberculosis* in our case suggests an immune-based reaction triggered by either ATT or the *Mycobacterium* itself. A small cohort of Chinese patients showed improvement after receiving ATT in steroid-resistant NMO.[9] This may, therefore, suggest a role of the *Mycobacterium* itself in triggering the inflammatory cascade leading to NMO. This nevertheless remains a speculative plausibility and further studies are needed to confirm the pathophysiology behind this association in such patients.

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