INTRODUCTION

Neuromyelitis Optica (NMO) is diagnosed on the basis of Wingerchurk's criteria, including clinical evidence of events involving optic nerve(s) and spinal cord along with two out of the three of the following parameters: long extensive spinal cord lesions (> 3 spinal segments), brain MRI normal or not meeting criteria for multiple sclerosis, NMO IgG seropositive status. Systemic diseases like systemic lupus erythematosus, Diabetes, insecticide exposure and infections like pulmonary tuberculosis have been implicated as the possible associations of NMO.

Here we report a case of young girl who during the course of NMO treatment, revealed to have active abdominal tuberculosis.

CASE REPORT

A 19 years female with past history of pulmonary TB, 8 years back, for which she had completed full course of anti-tuberculosis medication, admitted in the hospital with complaints of sudden onset of weakness of lower limbs for 4 days along with urinary retention and constipation for 3 days. These complaints were not associated with any backache or paraesthesias. Her systemic examination revealed power of 0/5 in both lower limbs, increased tone, absent reflexes with upgoing plantars with no definite sensory level and no gibbus or spinal deformity noted and normal eye examination. Her MRI spine was carried out; lower cervical and the entire cord till conus demonstrated thickened cord and T2W hyperintensity without evidence of cord compression or enhancement and on post-contrast features were suggestive of vertical myelitis (Figure 1). MRI brain with gadolinium enhancement was unremarkable for any demyelinating lesion. Her CSF studies showed normal glucose (68 mg/dl), protein (32 mg/dl), and cell count (30/cumm with 90% lymphocytes). CSF was negative for AFB smear, MTB PCR, oligoclonal bands and AFB. Her Visual Evoked Potential (VEP) showed delayed latencies bilaterally, indicating visual pathway demyelination. On these clinical and laboratory background she was diagnosed as a case of Neuromyelitis Optica (NMO). She was started on pulses of intravenous methylprednisolone followed by oral steroids but without any significant improvement.

A week later, she suddenly developed high grade fever with severe abdominal pain along with abdominal distention. On examination, abdomen was tense and tender with shifting dullness. Routine investigations and ANA, Anti ds DNA antibodies were normal. Her U/S abdomen showed mild ascites with internal echoes. Her CT abdomen was carried out that showed dilated ileal loops with thickened edematous folds, fluid in cul-de-sac, which could be due to abdominal tuberculosis. Frank pus was aspirated under ultrasound guidance. Diagnosis of acute abdomen was made; exploratory laparotomy was done and 500 ml Frank pus was drained. Mesenteric lymph nodes were taken for histopathology as they were found to be enlarged while there was no perforation of any viscus'. Pus for AFB smear and ordinary C/S were negative and histopathology of lymph nodes showed reactive hyperplasia (Figure 2).

Steroid was stopped when she developed acute abdomen and on empirical grounds she was started on anti-tuberculosis therapy. She showed dramatic response after 4 weeks of therapy and could walk without support. After 6 weeks her AFB culture report was received which showed growth of Mycobacterium tuberculosis.
DISCUSSION

NMO or Devic’s disease is a rare acute inflammatory disease characterized by demyelination affecting the spinal cord and optic nerves. Many case reports and series are available suggesting the association of NMO and active pulmonary tuberculosis. In our patient, there was past history of pulmonary tuberculosis. This time she presented with NMO and during course of steroid developed abdominal symptom which proved to be abdominal tuberculosis. Her abdominal and neurological symptoms markedly improved with a course of steroid which was used only for one week and antituberculous therapy.

A retrospective case-control study was conducted in Cape Town, South Africa to investigate the possible association between NMO and TB and the results showed an association between NMO and pulmonary tuberculosis in the study population. Seventy nine percent patients had preceding or simultaneous diagnosis of pulmonary tuberculosis. The most likely mechanism proposed by them was immune-mediated inflammatory demyelination of the optic nerves and spinal cord triggered by pulmonary tuberculosis.\(^3\)

Rafai et al. published two case reports of NMO in the course of pulmonary tuberculosis. One had sputum smear positive pulmonary TB before developing features of Devic - like syndrome while the other patient showed sputum smear-positive pulmonary TB later in the course of his illness.\(^4\) They also emphasized on immune dysfunction triggered by Mycobacterium infection.

A case report from Pakistan also showed association of NMO with sputum smear positive pulmonary TB in a 46-years diabetic and hypertensive male. According to authors the possible mechanism for this association may be a direct tuberculous involvement of the nervous system or a reaction to anti tuberculous therapy.\(^5\) However, they had no evidence of direct invasion of CSF or brain by MTB. So they also concluded on immune based reaction triggered by either ATT or the Mycobacterium itself. Lrzezioui et al. reported a case of NMO in association with multisystemic tuberculosis in a 36 years female patient who initially responded well to anti-tuberculous therapy. The author concluded to search tuberculosis in patients with NMO, especially in countries where tuberculosis is endemic and a great problem of public health, and not to consider it only as a variant of multiple sclerosis because of different therapeutical options.\(^6\)

There are no specific treatments for patients with acute, severe neurological deficits caused by neuromyelitis optica (NMO) who fail to recover after treatment with high-dose corticosteroids. A prospective, controlled study in southern China evaluated the clinical response of anti-tuberculosis treatment (ATT) in patients suffering from steroid-refractory NMO, and investigated the correlation between NMO and tuberculous infection of the central nervous system (CNS). Twelve patients with steroid-refractory NMO were monitored during ATT and compared with a control group of 13 patients with the same type of NMO who received current standard therapies. A molecular diagnostic test was carried out and Extended Disability Status Scale (EDSS) score analysis, visual acuity, the number of relapses and MRI changes were evaluated at study entry and after 1 and 2 years of therapy. ATT also significantly reduced the rate of relapse. By comparison, in the control group, a significant clinical deterioration was observed, and patients did not show favorable EDSS scores and MRI changes. This study suggested that CNS infection with Mycobacterium tuberculosis is an important cause of NMO.\(^7\)
As tuberculosis is endemic in Pakistan, there is need to extensively search for presence of active tuberculosis in patient presenting with NMO. In addition, even if no active tuberculous infection is diagnosed, patients of NMO who are refractory to steroid and have no other underlying systemic disease could be given a trial of anti tuberculous drugs for their neurological symptoms.8

REFERENCES