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A Case Report of Neuromyelitis Optica

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Abstract
Neuromyleltis Optica also called as Devic's disease is an aggressive inflammatory disorder characterized by recurrent attacks of optic neuritis and myelitis. More female predominance [9:1]. Attacks of Optic neuritis can be bilateral with severe visual loss. Myelitis can be severe and transverse and is typically longitudinally extensive involving 3 or more contiguous vertebral segments. CSF findings include pleocytosis with neutrophils and eosinophils, OCB bands are uncommon occurring in <20% of NMO. The prevalence range of NMOSD is 0.5–4/100,000 and may be up to 10/100,000 in certain racial groups. Optic neuritis or myelitis are the defining signs of NMOSD; either one may appear as the initial symptom. A highly specific and only modestly sensitive diagnostic test for NMOSD is Aquaporin-4 Immunoglobulin G (AQP4-IgG). It has been demonstrated that it can identify antibodies that are specific to the astrocyte protein AQP4.
Keywords: *Optic neuritis, Myelitis, Aquaporin – 4 Immunoglobulin G.*

Case Report

A 52 year old female presented with chief complaints of weakness of bilateral lower limbs with history of diminished vision on both eyes, decreased hot and cold sensation in lower limb and bladder incontinence for 1 month.

Patient had past history of Unilateral optic neuritis of left eye 6 years back was diagnosed as Multiple sclerosis and she was treated with on T.

Dimethyl fumarate, T. Prednisolone. No other co-morbidities. CNS Examination: Higher Mental Functions - Normal,

Cranial Nerve: Optic Nerve: Visual acuity 6/12 in both eyes, Fundus: Funduscopy examination revealed a picture of bilateral atrophic papillae.

Motor system: Tone -Spasticity in bilateral lower limb, Power: 0/5 in right lower limb and 1/5 in left lower limb. Deep tendon reflex: Knee and Ankle reflex was exaggerated.

Plantar reflex was bilaterally extensor. Sensory system: spinothalamic, posterior column, cortical sensations was decreased below the level of

umbilicus. Autonomic system: Bladder incontinence was present.

Blood Investigations was normal. Anti-Nuclear Antibody [ANA] was done which was negative.

MRI BRAIN + SPINAL CORD showed Few T2/ Flair high intensities in bilateral central Semiovale. Few of these lesions seen radiating to ventricular surface.

T2 high signal intensity at D2-D7 superior endplate in spinal cord with thinning at same level, Suggestive of longitudinally Extensive Transverse Myelitis with cord atrophy, vertebral body Hemangioma at D8, Bilateral optic nerve atrophy [Left > Right].

CSF showed lymphocytic pleocytosis. NMO WITH MOG ANTIBODY Profile done which showed NMO[AQ4] ANTIBODIES: POSITIVE, MOG ANTIBODIES: NEGATIVE, S.OCB: NEGATIVE, CSF OCB:1-3 BANDS.

Thus Diagnosis was made as Neuromyelitis Optica Spectrum Disorder. She was treated with Intravenous Corticosteroids, Immunosuppressive therapy [Rituximab] and Plasmapheresis.

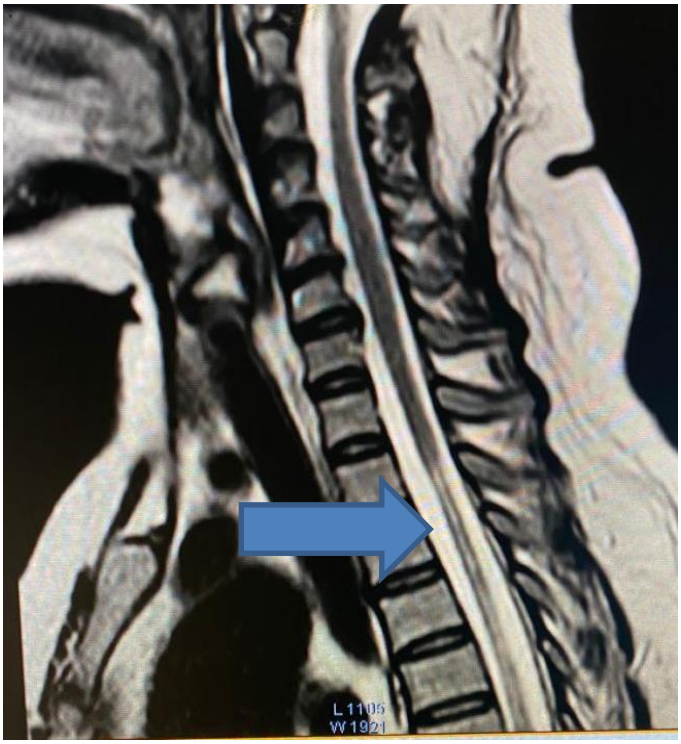

Fig 1: T2 high signal intensity at D2-D7 superior endplate in spinal cord with thinning at same level, Suggestive of longitudinally Extensive Transverse Myelitis

Discussion

AQP4-Abs cause complement- and cell-mediated astrocytic damage after they are attached to the extracellular domain of the AQP4 receptor. As a result, the astrocyte is rendered helpless, ultimately leading to the loss of support for neighboring cells, including oligodendrocytes and neurons. Following granulocyte infiltration, oligodendrocyte destruction and demyelination occur.

In contrast to MS, the demyelination found in NMOSD is a secondary event that results from primary astrocyte injury.

In NMOSD, clinical syndromes like bilateral optic neuritis, longitudinally extensive transverse myelitis, region postrema syndrome, acute brain stem syndrome, diencephalic syndrome, and symptomatic cerebral syndrome were seen.

In this case our patient fulfilled the diagnostic criteria for "NMOSD" as evidenced by the presence of core clinical characteristics such as optic neuritis and acute myelitis with the serum NMO-IgG was found to be positive.

Corticosteroids, immunosuppressant therapy, Therapeutic Plasma Exchange (TPE), immunomodulatory therapy, and other novel medicines are available as treatment options for preventing relapse.

Our patient was initially treated with corticosteroids, Rituximab infusion as per protocol after premedication followed by Plasmapheresis. She became neurologically stable.

Neuromyelitis optica spectrum disorder (NMOSD) is a relapsing central nervous system disease associated with aquaporin-4 antibodies. Common presentations include longitudinally extensive myelitis, severe optic neuritis, and area postrema syndrome.

Prompt and aggressive treatment of relapses with high dose steroids, immunomodulators and +/- plasma exchange improves outcomes. All patients with aquaporin-4 antibodies should be immunosuppressed indefinitely to prevent further attacks.

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