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NEUROMYELITIS OPTICA SPECTRUM DISORDER: A CASE REPORT

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ABSTRACT

Neuromyelitis optica spectrum disorder (NMOSD), formerly known as neuromyelitis optica (NMO) or Devic's syndrome or Devic's disease, was initially considered as part of multiple sclerosis (MS) because the symptoms were considered overlapping. But now, it is known that the pathophysiology of these two diseases is different.[2] Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune disease that causes severe demyelination, especially in the optic nerve and spinal cord with typical clinical manifestations of acute optic neuritis and transverse myelitis. The symptoms can occur simultaneously or separated by a variable period. NMOSD is associated with serum aquaporin antibodies 4 immunoglobulin G (AQP4-IgG).[4]

KEYWORDS: Neuromyelitis optica spectrum disorder, Aquaporin antibodies 4 immunoglobulin G.

INTRODUCTION

Neuromyelitis optica spectrum disorders (NMOSDs) are autoimmune diseases that manifest clinically with 6 core symptoms, which include acute optic neuritis, transverse long segment myelitis, and acute brainstem syndrome.[1] The first episode generally includes one or more core symptoms but does not involve pathological changes in peripheral nerves.[1]

It is more common in the form of polyphasic (90%) such as optic neuritis or myelitis, or both occurring together. The monophasic form has only occurred in 10% of cases.[6]

CASE REPORT

35 yrs old female, presented in Gastroenterology OPD with complaints of nausea, vomiting and hiccups for last 3 weeks. She has recently undergone LSCS and sterilization in October, 2019. Her pregnancy period was uneventful. She gave birth to a female child. On 13/12/2019, she developed nausea and vomiting, she was admitted in a local private hospital for about 1 week. She was treated symptomatically. Her vomiting episodes decreased, but during this time she developed hiccups which lasted for several days .After a week, her vomiting aggravated along with persistent intermittent but less severe hiccups. She had difficulty to take any food orally because of intractable nausea and vomiting and also had light headedness. She was then referred to the Gastroenterologist for further evaluation and

assessment. No h/o fever/burning micturation/abdominal pain/hematemisis. No history of blurred vision, dysarthria, dysphonia or limb weakness/ unsteadiness of gait. She denied any significant neurological symptoms in the past.

Her neurological examination revealed mild finger nose in-cordination on left side. In view of features suggestive of a central cause of vomiting, MRI Brain +MRV+MRA were done. MRI Brain showed hyperintense area with minimal peripheral enhancement in left dorsa medullary region in the area postrema extending caudally. Rest of the brain and spinal cord did not show any significant pathology. Lumbar puncture was done on 1/1/2020.CSF microscopy (TC/DC), sugar/proteins, TB PCR/HSV PCR and NMOSD, OCBs work up was sent. Vasculitis work up and lyme antibodies also were negative except pANCA which is mildly positive (needs to be checked again). She was treated with IV methyl prednisolone 1 gm in 100ml NS over 2 hrs. Serial electrolyte monitoring was done .On the next day, she improved symptomatically, her vomiting subsided. She had complaints of swallowing difficulty, for which ENT opinion was also sought. On examination, she had pooling of saliva in Oropharynx. 70 degree laryngoscopy showed pooling of saliva in both pyriform fossa and cricopharynx with trickling into the laryngeal inlet. Both vocal cords moving and airway is adequate. Speech and language therapy consultation was sought, advised exercises to improve safe swallowing and started to give liquid diet. Her vasculitic work up reports were negative except for elevated p - anca, NMOSD reports were negative in Serum and CSF. However in view of her clinical symptoms, she was diagnosed with Serum Negative Neuromyelitis Optica .A cardiology consultation was sought for ECG variations (t wave inversions), advised to do TMT. At the time of discharge, she is hemodynamically stable, tolerating oral soft diet with no neurological deficits.

DISCUSSION

NMOSD is an autoimmune disease that causes severe demyelination, especially in the opticnerve and spinal cord with typical clinical manifestations of acute optic neuritis and transverse myelitis. It has been associated with serum AQP4-IgG.[7]

NMO IgG represents a highly specific diagnostic marker for NMO but also for NMO spectrum disorders (NMO-SDs), which include recurrent longitudinally extensive myelitis or recurrent optic neuritis.NMO and NMO-SD are rare pathological entities with serious neurological manifestations.[3]

Eugene Devic (1858-1930) who first introduced the French term acute neuromyelitis optic “neuro-myélite optiquë aiugë” to show a new syndrome characterised by myelitis and acute optic neuritis. Lennon and Wingerchuck (2004) detected the presence of IgG-NMO or IgG-AQP4, the specific antibodies that distinguish NMOSD from MS.[5]

CONCLUSION

In the case of relapsing NMOSD patient, combination therapy of immunosuppressants, corticosteroids, and TPE was used. This case is an illustration of a long diagnostic process in this rare form of disease. The study presents the possibilities of treatment in a severe case, basedon a multidisciplinary clinical team. The most important problem of NMO patients is limited activity of daily living (ADL), so one goal of this article was also to present the important role ofrehabilitation, as a support, in the independent character of the disease.

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