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ACUTE ONSET QUADRIPARESIS AS MYELIN OLIGODENDROCYTE GLYCOPROTEIN(MOG) ASSOCIATED DISEASE WITH ADEM (ACUTE DISSEMINATED ENCEPHALOMYELITIS) AFTER CHICKEN POX- A RARE CASE PRESENTATION

Clinical Research

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ABSTRACT

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory disease of central nervous system characterized by attack of immune mediated demyelination. This disease entitiy is seronegitive for AQP-4 and Oligoclonal band and positive for anti MOG. We report a case of post chicken pox quadriparesis which was ultimately diagnosed as MOGAD with ADEM. This is rare post chicken pox complication.

KEYWORDS

MOGAD, Multiple sclerosis, NMO spectrum disorder, plasma pharesis

INTRODUCTION:

The most common chronic inflammatory demyelinating disease of the nervous system is multiple sclerosis (MS). After so many years MS is extensively studied. The currently used McDonald criteria for MS have been modified several times and do not seem definitive [1]. Patients who presented with symptoms of optic neuritis and acute transverse myelitis were managed as a variant of MS–Devic's disease, which is currently named as neuromyelitis optica (NMO). An antibody against the water channel aquaporin-4 (AQP-4 IgG), clinical course, neuroimaging and neuropathological findings, all led to the recognition of NMO as an independent disease entity [2,3]. However, in recent years it was found that in 10–25% NMO patients, AQP-4 antibodies were not detected and of such AQP-4 seronegitive population in almost 21% patients, antibodies against myelin oligodendrocyte glycoprotein (MOG) were identified [4]. Antibodies against MOG have recently been recognized in a clinical syndrome that is likely a CNS demyelinating disorder different from multiple sclerosis (MS), acute demyelinating encephalomyelitis (ADEM), and neuromyelitis optica spectrum disorder (NMOSD). However these MOG antibodies have been mentioned in the literature for the last 30 years, but their role in demyelinating disease is still remains controversial (5,6). Recurrent optic neuritis, myelitis, brainstem encephalitis, and ADEM-like presentation such as encephalomyelitis have all been described in MOGimmunoglobulin (IgG)-positive patients (7–9). However, the complete clinical profile of this disease entity remain undetermined. Here we report a case of post chicken pox MOG-IgG-positive patient who did not fulfill the NMOSD criteria, and dofferent from clinical spectrum of this disease.

Case report:

A 17 year old female patient presented to emergency department of SMS Hospital,Jaipur with history of acute onset rapidly progressive quadriparesis and altered sensorium since one day.with loss of sensatioan and band like sensation at the level of nipple with retention of urine since one day. Attandants gave history chicken pox infection 15 days ago of presentation.

On neurological examination motor power of upper limb was 0/5 on both side and in lower limb 0/5.Deep tendon refelxes were absent in both upper limb and lower limb (ankle, knee, supinator, biceps and triceps reflexes) and plantar reflexes were mute. On sensory examination patients complains of loss of sensation below nipples and feeling of band like sensation at the level of nipples.(as patients gained consciousness after pulse therapy of steroid after 2 days of admission). Sensory level was present at T4 level.

On physical examination BP was 110/70 mm of hg pulse rate 90 per minute and SPO2 was 98%.

ECG suggestive of normal sinus rhythm with normal axis and chest x ray was normal. Serum antibody profile for varicella zoster virus (VZV) was also done which was positive for IgG and IgM. This was suggestive of recent past infection of chicken pox.

Contrast enhanced MRI brain reveals multiple, discrete & confluent, intra axial areas of altered signal intensity in bilateral cerebral hemisphere appears hyperintense on T2W & FLAIR image, hypo to isointense on T1W image and no restricted diffusion on DW images, no obvious contrast enhancement on post contrast T1W images. Imaging findings represents most likely inflammatory pathology (Fig.A).

Contrast MRI cervical spine reveals abnormal elongated intra medullary areas of altered signal intensity in spinal cord parenchymal opposite C2 to D12 vertebral level,appearhyperintense on T2W & STIR images and iso to hypointense on T1W images and no obvious contrast enhancement on post contrast T1W images without evidence of cord expansion or enlargement represent diffuse spinal cord edema findings s/o viral myelitis.(Fig.B)

On CSF examination it was clear in appearance, proteins 57.4 mg/dl(normal 15-45 mg/dl), glucose 55mg/dl(normal 50-70 mg/dl), total count 18 cell/cumm(normal 0-5 cells/cumm), lymphocyte/ neutrophil ratio 90/10 % and ADA 2.11 U/L . Overall CSF findings were suggestive of viral pathology.

Laboratory studies reveals a random blood sugar 104 mg/dl, Haemoglobin 10.0 gm/dl, total leucocyte counts 5950/cumm, platelets 1.35 lakhs/ml, Urea/creatine 38/0.52 mg/dl, sodium/potassium 144/3.66 mMol/L, SGOT/SGPT 50/94 md/dl.

Special investigation –As patient present with post viral quadriparesis with whole spine myelitis so patient evaluated for NMOSD/MOG Antibody Associated Disease(MOGAD) and patient was found MOG Antibody positive. CSF was negative for AQP-4 and oligoclonal bands.

Along with intravenous methylprednisolone 7 cycles of plasmapheresis (on alternate day) was done. Motor power of both upper limbs improved from grade 0/5 to 5/5. However improvement in motor power of lower limb was not significant. Power of lower limb

was improved from 0/5 to 2/5. For further rehabilitation patient is shifted to PMR (physical medicine and rehabilitation) department.

DISCUSSUION:

Myelin oligodendrocyte glycoprotein antibody-associated disease is an inflammatory disease of central nervous system characterized by attack of immune mediated demyelination.

Clinical symptoms –Optic neuritis is the most common clinical sympotom of MOGAD at presentation of disease and it is more common in relapse .

ADEM and ADEM like presentation is also manifest as an acute-onset encephalopathy associated with polyfocal neurologic deficit and typically self limiting .

Transverse Myelitis-inflammation across the spinal cord parenchyma leading to neurologic dysfunction. Neurologic symptom in transverse myelitis usually develop over hours or days and reaches maximum within 21 days. Its important to be noted that spinal cord involvement in MOGAD may occur as an isolated attack or CNS involvement may be seen along with ADEM,Optic neuritis. MOGAD may also present as subacute onset paraperesis or parapersis with sensory motor loss with frequent autonomic bladder and bowel involvement and erectile dysfunction.

Diagnosis- Along with different clinical presentations serum MOG –IgG antibody remains the diagnostic criteria. MRI brain and spine should always be done.

Clinical	Investigations
Optic neuritis	MRI brain/MRI Spinal cord/orbits
ADEM	CSF study
Transverse myelitis	Serum MOG antibody
Brain or brainstem demyelination syndrome	

Treatment: Choice of the therapy – adult dose is high dose intravenous methylprednisolone 1 gm/day or 20-30 mg/kg for 5 days.

Second line of treatment-patients who fails to respond to intravenous methylprednisolone second line of treatment is IVIG 2 gm/kg total dose should be given over 5 days with continuous infusion or plasma exchange every other day total 5 to 7 cycles.

CONCLUSION:

MOGAD can occur after any viral infection. Every clinician should suspect about MOGAD in patient with quadriparesis/paraparesis with or without optic neuritis and such patient should always be worked up for anti MOG antibody.

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