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A CASE OF THE FIRST EPISODE OF MULTIPLE SCLEROSIS

General Medicine

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ABSTRACT

Multiple sclerosis is characterised by chronic inflammation and selective destruction of CNS myelin. The lesions are termed as plaques. Onset is in early to middle adulthood and women are affected three times more commonly than men. The most common symptom in these patients is recurrent attacks of focal neurological dysfunction lasting for weeks or months. This may be followed by variable recovery. Here I present a case which was the first presentation of the multiple sclerosis and was diagnosed on MRI

KEYWORDS

INTRODUCTION:-

Multiple sclerosis may present as slowly progressive neurological deterioration. Fatigue, stress, exercise or heat may worsen the symptoms. The manifestations of multiple sclerosis may include weakness or sensory symptoms involving a limb, visual difficulties, abnormalities of gait and coordination, urinary urgency or frequency and abnormal fatigue. Motor involvement can present as heavy, stiff, weak or clumsy limb. Localised tingling, pins and needles and dead sensation are common.[1] Blurring of vision especially in central visual fields associated with retro-orbital pain accentuated by eye movements may be present. Brainstem involvement may result in diplopia, nystagmus, vertigo, facial pain, numbness, weakness, hemispasm or myokymia. Cerebellar involvement involvement may present as ataxia, tremor, dysarthria. Cervical spine involvement may present with Lhermitte's sign. Physical examination usually shows visual field defects, loss of visual aquity, disturbed colour perception, optic pallor/papillitis, afferent papillary defect, nystagmus, internuclear ophthalmoplegia, facial numbness or weakness, dysarthria, weakness and spasticity, hyperreflexia, ankle clonus, ataxia, extensor plantars, sensory abnormalities.[2]

Case Report:-

A 32 years old male presented to the emergency with chief complaints of-
Weakness in both the lower limbs for the past 1 week
Difficulty in vision for the past 1 week

In the history of present illness, patient was alright 1 week back when he started experiencing weakness in both the lower limbs. It started with tingling sensation in both the lower limbs which progressed over the next day to in ability to lift the leg off the bed. This stayed for the next 2 days and then the weakness progressed furthur and the patient was not able to move the lower limbs in the bed at all in the next few days.

There was no history of any sensory involvement as the patient was able to feel the clothes he was wearing and the bed he was lying on.

The patient also had difficulty in seeing for the past 1 week. Initially, the patient could not see in the peripheral parts of the visual field which then progressed to involve the whole visual field.

There was no history of any weakness in the upper limbs.

There was no history of any involuntary movements.

There was history of bladder involvement in the form of urinary incontinence.

There was no history of any other cranial nerve involvement.

In the past history, there was no history of any similar complaints in the past. There was no history of weakness in any other part of the body in the past. There was no history of diabetes mellitus, hypertension, coronary artery disease or tuberculosis.

The family history showed nothing significant.

In the personal history, the patient was a chronic smoker, non alcoholic, bowel and bladder was normal and the sleep was normal. The patient was vegetarian by diet.

The patient belonged to the middle class.

On general physical examination, the patient was conscious, cooperative and lying comfortably in the bed. The BMI of the patient was 24. The patient was afebrile, pulse rate was 84/min, regular, normal volume, normal character, no radiofemoral delay, all peripheral pulses well felt, condition of the vessel wall was normal, equal both the sides. Blood pressure was 120/70 mmHg in the right arm, supine position. There was no pallor, icterus, cyanosis, clubbing, edema or lymphadenopathy. The JVP was not raised.

On examination of the CNS, the higher mental functions were normal. The Cranial Nerves examination showed the IInd cranial nerve involvement in the form of visual field defect causing bilateral temporal hemianopia. Other cranial nerve examination was normal. The motor system examination showed that there was weakness in both the lower limbs where the power was 2/5 in both the lower limbs. Tone was increased in both the lower limbs. There was no wasting of the muscles, no involuntary movements. Gait could not be tested due to the same reason. Coordination in the upper limb could not be tested as there was defect in the vision. The deep tendon reflexes were exaggerated in the lower limbs and were normal in the upper limbs. The superficial reflexes showed that the plantar reflex was extensor. The lower part of the abdominal reflex was lost and the upper part was preserved. The cremastric reflex was lost. The sensory system examination was normal. There was no signs of meningeal irritation.
The probable differential diagnosis kept was-
Multiple Sclerosis 1st episode
Transverse Myelitis
NMOSD
NMO
Compressive Myelopathy
Spinal cord tumour with metastasis in the brain

The investigations done showed-

Hb of 11.4 gm%, TLC- 6300/mm^3, Platelet count of 1.75 lac/mm^3,RFT was normal, LFT was normal, RBS was 126mg%, ECG was normal, Urine examination was normal, Chest X-ray was normal.

MRI Brain done showed- involvement in the form of demyelination at the level of both Optic nerves and the Optic chiasma.

MRI Spine showed demyelination at the level of T7 vertebral spine and T10 spinal cord level.

The patient was started on treatment as the MRI reports were suggestive of Multiple Sclerosis.

The patient was given treatment in the form of IV fluids, Inj Methylprednisolone 1 gm iv for 5 days, Inj Pantoprazole, Inj Multivitamin and Physiotherapy was given. The patient improved slightly with the improvement of power from grade 2 to grade 4 with pulse steroid therapy. He was then referred to the neurology department at PGI Chandigarh for furthur management as our institute did not have a neurology department.

DISCUSSION-

There are four general categories of Multiple Sclerosis-

Relapsing-Remitting Multiple Sclerosis
Secondary Progressive Multiple Sclerosis
Primary Progressive Multiple Sclerosis
Progressive Relapsing Multiple Sclerosis

Most of the patients require assistance with ambulation later on.

MRI usually shows thw evidence of bright areas on T2 weighted sequences often in the periventricular location.

CSF shows mild lymphocytic pleocytosis, oligoclonal bands, elevated IgG and normal protein levels.

Other tests that can be done are visual, auditory and somatosensory evoked response tests and urodynamic studies.[3]

Treatment of the condition involves the disease modifying therapies for the relapsing forms of the multiple sclerosis. The drugs that can be used are-

1. Interferon-β1a 30mg im once a week
2. Glatiramer acetate 12mg sc per day
3. Natalizumab 300mg iv every 4 weeks
4. Fingolimod 0.5mg per week
5. Mitoxantrone 12mg per m^2 iv every 3 months
6. Cladribine 3.5mg per kg orally every year

These treatments prevent the annual exacerbation rates by 30% and also reduces development of new MRI lesions.

Plasma exchange (7 exchanges; 40-60ml/kg every other day for 14 days) may benefit the patients with fulminant attacks of demyelination not responding to glucocorticoids. Other drugs that can be used are Methotrexate, Azathioprine, Cyclophosphamide.[4]

Somptomatic therapy involves-

For spasticity- Physical therapy, Baclofen, Diazepam, Tizanidine, Dantrolene, Cyclobenzaprine.

For dysesthesia- Carbamazepine, Phenytoin, Gabapentine, Pregabalin, Amitryptyline.

For bladder dysfunction- treatment based on underlying pathophysiology. Oxybutynin is used for bladder hyper-reflexia. Bethanechol is used for bladder hypo-reflexia.

Depression associated with the disease has to be treated aggressively.[5]

CONCLUSION-

The present case was the classical example of the first episode of the multiple sclerosis. The point favouring the diagnosis of multiple sclerosis was that the symptoms, examination and the MRI showed the multifocal involvement in the CNS. The patient was given the guideline directed therapy.

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