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A Rare Case of Relapsing Multiple Sclerosis with Multifocal Brain and Spinal Cord Demyelinating Lesions in a Middle-Aged Male: A Case Report

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Abstract: Multiple sclerosis is an autoimmune inflammatory demyelinating process that affects the central nervous system. It is characterized by inflammatory demyelination and neurodegeneration leading to involvement of the brain, spinal cord, and optic nerves. Relapsing-remitting multiple sclerosis (RRMS) is the most frequent type of MS and involves recurrent episodes of neurological dysfunction followed by partial or full recovery. Multifocal involvement of the central nervous system and numerous brain and spinal cord lesions constitute a difficult clinical condition necessitating early diagnosis and adequate therapy. A patient of middle age demonstrated signs and symptoms of neurological impairment related to central nervous system involvement. Brain and spinal cord magnetic resonance imaging (MRI) showed multiple demyelinating hyperintensive lesions in periventricular, juxtacortical, and infratentorial regions with concomitant spinal cord lesions. Taking into consideration the pattern and characteristics of the lesions, a diagnosis of multiple sclerosis could be established in this case. The patient was diagnosed as having relapsing multiple sclerosis. He was prescribed appropriate immunomodulatory therapy and supportive treatment. In this case, the significance of detecting multifocal demyelinating lesions in the presence of recurrent neurological manifestations is emphasized. Proper diagnosis based on MRI assessment and adequate treatment could reduce the number of relapses and the occurrence of neurological disability in the future.

Keywords: *Multiple Sclerosis, Demyelination, CNS Lesions, Spinal Cord Involvement.*

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I. INTRODUCTION

Multiple sclerosis (MS) is an inflammatory autoimmune disease of the central nervous system (CNS), associated with immune-mediated damage of myelin, oligodendrocytes, and neurodegeneration. The disease affects about 2.8 million people around the world and is one of the major causes of neurological disability in young and middle-aged patients.[1]

It predominantly affects the white matter in the brain, spinal cord, and optic nerves and is associated with diverse neurological symptoms, including sensory problems, motor deficits, vision loss, cognitive impairments, and walking difficulties.[2] Pathological changes observed in MS include formation of demyelinated lesions due to infiltration of T lymphocytes, B cells, macrophages, and inflammatory factors leading to loss of myelin integrity and axon damage.[3]

MS can be manifested in several forms, including clinically isolated syndrome (CIS), relapsing-remitting MS (RRMS), secondary progressive MS (SPMS), and primary progressive MS (PPMS). About 85–90% of patients with MS are diagnosed with RRMS, which is associated with recurrent attacks of neurological symptoms followed by remissions.[4] Prolonged inflammatory processes lead to axonal damage and neurological disability over time.[5]

MRI holds an important place in diagnosing MS and following up on the disease. The McDonald criteria of

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diagnosis include both clinical and MRI criteria that include dissemination of lesions both in space and time. The common MRI findings of multiple sclerosis are found in periventricular, juxtacortical, infratentorial regions, and spinal cord.[6] The presence of spinal cord lesions indicates greater disease severity and increased chances of developing disability.[7]

While MS is mostly seen in younger patients, the atypical presentation and occurrence of the disease in middle-aged patients might complicate the diagnosis process, as well as its differentiation from other inflammatory, vascular, or infectious conditions.

This report describes a rare case of relapsing MS in a middle-aged man with multifocal demyelinating lesions in the brain and spinal cord.

II. CASE PRESENTATION

A 43-year-old male patient coming under neurology department with the complaints of numbness and paraesthesia over right leg and band like tight sensation over lower abdomen for approximately 2 weeks. He has past medical history of dyslipidaemia. He was receiving Tab. Atorvastatin 10mg orally once daily at night.

On admission, the patient was conscious, alert, and oriented with stable vital signs. Her blood pressure was 120/80 mmHg, pulse rate was 81 beats/min, respiratory rate was 24 breaths/min, and oxygen saturation was 98% on room air. Findings on cardiovascular examination were normal S1 and S2 without murmurs. Respiratory examination was normal air entry bilaterally with no additional sounds. Findings on abdominal examination included a soft and non-tender abdomen with no organomegaly. Findings on neurological examination were sensory loss of the right half of the body, without motor deficits.

All routine lab workup was found to be mostly normal. CBC values were recorded as follows: haemoglobin content of 14.8 g/dL, leukocyte count of 7,190 cells/mm³, platelet count of 2.6 lakh/mm³, red blood cell count of 4.93 million/mm³, PCV of 42.8%, MCV of 86.8 fL, and MCH of 30 pg. In the differential count, there was only a mild increase in eosinophils at 6% with normal values for all other parameters. LFTs were found to be normal with total bilirubin being 0.70 mg/dL, AST being 30 U/L, ALT being 29 U/L, and alkaline phosphatase being 49 U/L. The lipid profile showed total cholesterol to be 200 mg/dL, with elevated LDL cholesterol of 129mg/dL, consistent with dyslipidaemia.

MRI of CE- Brain with bilateral Orbits demonstrate multiple rounded to oval shaped plaques in the cortical, juxta cortical, deep and periventricular white matter, callososeptal interface with few of the lesion involving the U fibres involving the superior tentorial parenchyma with lesion in right frontal and occipital lobe showing enhancement, indicating active inflammatory plaques consistent multiple sclerosis.

MRI of Lumbar Spine demonstrated mild degenerative disc disease in cervical spine, small focal ill-defined intramedullary T2/STIR hyperintensity in central and peripheral portions of cervical and thoracic cord at C4-C5, C5-C6, C6-C7, T10-T11 and T12-L1 showing ill defined patchy heterogenous enhancement suggestive of active demyelination.

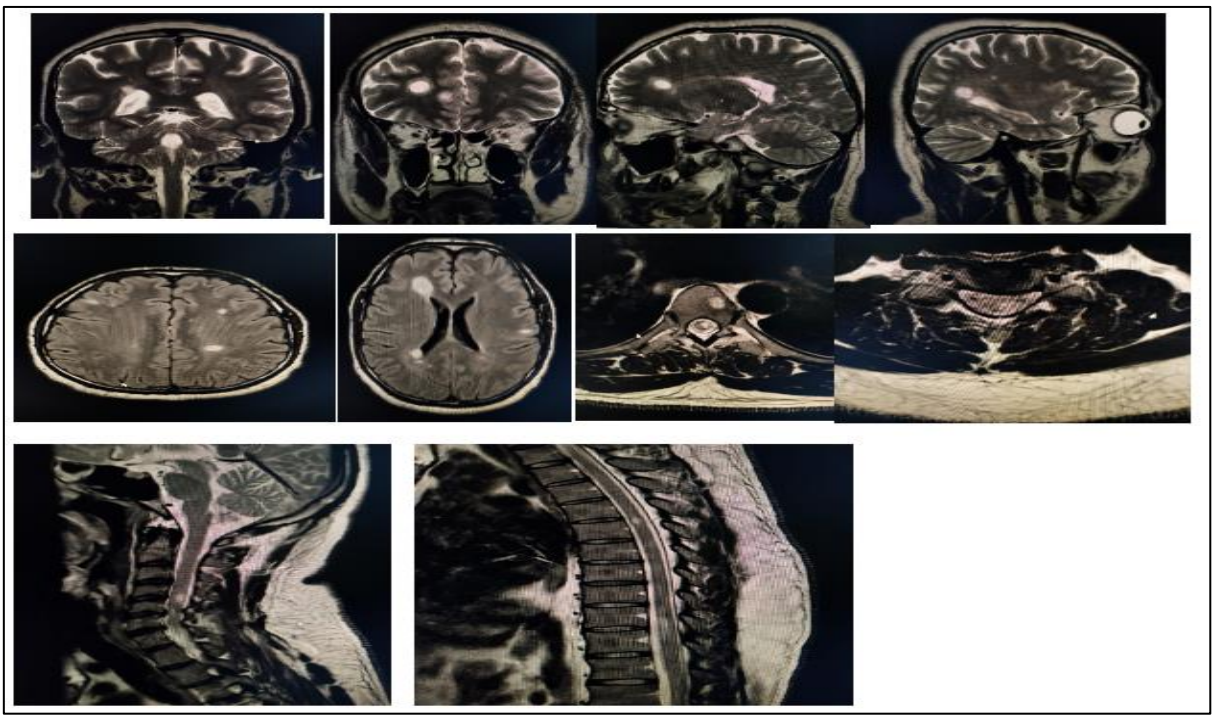

Fig 1 Magnetic Resonance Imaging (MRI) of Brain and Spine Showing Multiple Discrete Ovoid Lesions in the Juxtacortical Periventricular and Brainstem and Spinal Cord with Few Ovoid Block Spots in Periventricular Regions with Contrast Enhancement Suggestive of Active Lesions and Burnout Lesions Fitting Dissemination in Time and Space.

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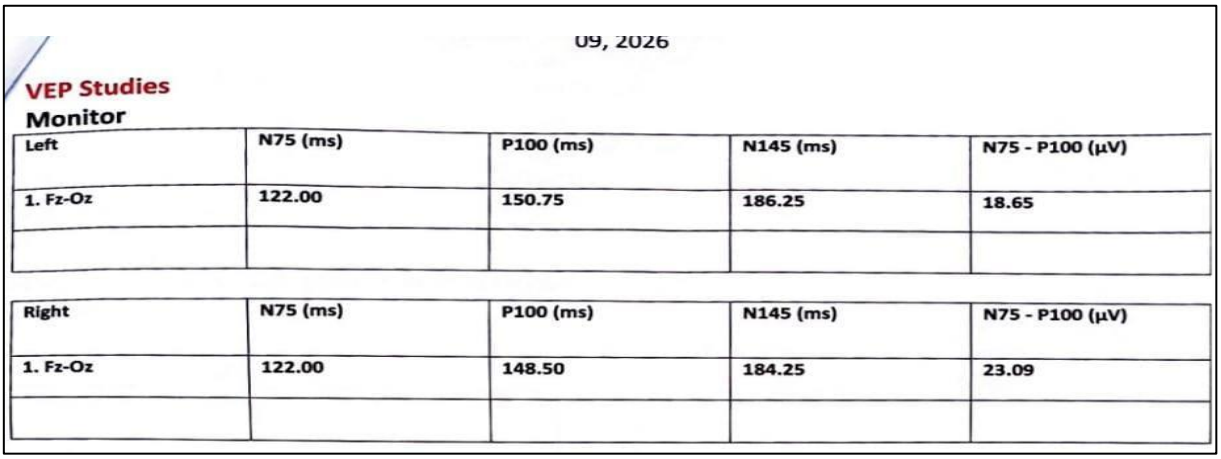
The procedure of lumbar puncture was done with the help of stringent aseptic measures and collection of cerebrospinal fluid samples was done for analysis. Analysis of cerebrospinal fluid showed total cell count of 12 cells, predominating lymphocytic picture (97% lymphocytes, 3% polymorphs). CSF was clear, and results of biochemical analysis showed protein 52 mg/dL, glucose 51 mg/dL, albumin 21.4 mg/dL, CRP 2.9 mg/L, and LDH 46 U/L. According to cytology report there were some scattered mature lymphocytes with few neutrophils, without any malignant cells detected. Gram-staining showed no organisms, and CSF was sterile by culture after 6 days of cultivation, decreasing the probability of bacterial CNS infection.

The concentration of CSF adenosine deaminase (ADA) was 4.2 U/L, which is within the described normal reference range (0-9 U/L), and did not give any laboratory evidence for tuberculous meningitis. In particular, the detection of CSF oligoclonal bands shows that there is the process of intrathecal immunoglobulin synthesis. This finding along with the patient's clinical presentation and MRI lesions is consistent with the diagnosis of MS.

Additionally, visual evoked potentials (VEPs) were investigated in order to evaluate the integrity of visual pathways. Latency of P100 in left eye was 150.75 ms, in right eye - 148.50 ms. The amplitudes of N75-P100 waves in left eye were 18.65 µV, in right eye - 23.09 µV. Relatively symmetrical VEPs gave some more information about visual pathways' conduction and were taken into account.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VEP Studies

Monitor

Left	N75 (ms)	P100 (ms)	N145 (ms)	N75 - P100 (µV)
1. Fz-Oz	122.00	150.75	186.25	18.65

Right	N75 (ms)	P100 (ms)	N145 (ms)	N75 - P100 (µV)
1. Fz-Oz	122.00	148.50	184.25	23.09

Fig 2 Visual Evoked Potential (VEP) Showing Bilateral N75–P100–N145 Waveforms with P100 Latencies of 150.75 MS in the Left Eye and 148.50 Ms in the Right Eye. The Responses Were Recordable in Both Eyes, Demonstrating Visual Pathway Conduction with a Relatively Symmetrical Latency Between the Two Sides.

The patient's clinical manifestations, neurological assessment, CSF analysis, VEP findings, MRI indicating presence of multiple active lesions within the central nervous system and past medical history were considered for the final diagnosis which turned out to be relapsing multiple sclerosis (acute demyelinating relapse) with dyslipidaemia.

The patient was provided with 1g intravenous Methylprednisolone once a day for 3 days consecutively, which was the standard treatment for acute exacerbation of multiple sclerosis. Pantoprazole IV 40 mg was prescribed twice a day to protect the patient's gastrointestinal tract in connection with steroid therapy. Gabapentin 300 mg orally was prescribed for pain and paraesthesia management. Atorvastatin 10 mg once a day was prescribed for dyslipidaemia management. No adverse effects were noted in the process of therapy.

III. CLINICAL OUTCOME & FOLLOW-UP

The patient had a stable state throughout his admission in the hospital. There were signs of improvement with regards to numbness and paresthesia after high-dose corticosteroid therapy. No new neurological deficit has been observed following the treatment regimen. The procedure for the lumbar puncture has been successful without any complications. The patient is discharged in a stable condition with orders to take their medications on time, observe strict compliance in neurological follow-up as well as seek medical help immediately when experiencing new neurological symptoms.

IV. DISCUSSIONS

Multiple sclerosis (MS) is a disease of demyelination caused by inflammation and occurs in the form of multiple lesions in various parts of the central nervous system. Clinical importance of the current case is attributed to the fact that it presents with predominantly sensory manifestations even with multifocal involvement of both brain and spinal cord. This illustrates the significant clinical diversity associated with MS in which the localization of lesions and the number of symptoms do not always correlate. The case is especially informative since clinical, imaging, cerebrospinal fluid oligoclonal bands, and visual evoked potentials are used to prove an active demyelination process [1,2,3,4,5].

The presence of numbness and tingling sensations on the right side over the lower abdomen in the patient is a vital aspect to consider since they are an indication that central

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sensory pathways are affected. Paresthesia is one of the clinical signs of MS and can arise due to the inability of the nerves to conduct impulses through demyelinated nerves. Despite the absence of motor impairment, significant disease process cannot be ruled out; sensory symptoms can present as the first or primary symptoms of lesions in the ascending sensory pathways. The location of symptoms including the truncal sensory symptom can suggest the involvement of the spinal cord. This presentation is in line with the clinical aspects of the disease explained by Lublin et al. and Oh et al [4,13].

The MRI results offer an anatomical explanation to the patient's neurological symptoms. Imaging revealed multifocal lesions located within periventricular, cortical/juxtacortical and deep white matter areas as well as multiple intramedullary lesions located within cervical and thoracic segments. Dissemination within various brain regions is one of the features of MS. Enhancement of certain lesions indicates the presence of inflammation, while non-enhancing lesions may indicate old lesions with demyelination. Filippi and Rocca stressed the significance of the lesion localization and characteristics in the diagnosis of MS, while Bot et al. paid attention to the diagnostic value of lesions in spinal cord [7,12].

There were several other causes that might present with similar clinical and imaging findings and hence had to be ruled out. Neuromyelitis optica spectrum disorder (NMOSD), MOG-antibody-associated disease (MOGAD), transverse myelitis, neurosarcoidosis, infection involving the central nervous system, deficiency in certain nutrients, autoimmune diseases, and vascular white-matter disease were some of the differential diagnoses. There was a slight lymphocytic response to inflammation in the CSF of the patient; however, the lack of organisms seen in Gram stain, the negative culture, and the CSF ADA of 4.2 U/L failed to support the presence of bacteria or tuberculosis infection.

The diagnosis was further confirmed by the 2017 McDonald criteria that use both clinical and paraclinical parameters to prove the dissemination in space and time. In this case, the lesions in different CNS areas provide the evidence for dissemination in space, whereas the presence of enhancing and non-enhancing lesions is the evidence of the lesions in different phases of inflammation. Also, the positive CSF oligoclonal bands indicate the intrathecal production of immunoglobulin and may serve as the evidence of the dissemination in time. Thompson et al. proved the importance of specific CSF oligoclonal bands for increasing the sensitivity of the revised criteria [6].

The VEP results give additional information about the neurological status. The P100 latencies were 150.75 ms and 148.50 ms in the left eye and right eye correspondingly, with bilateral presence of response. It shows that there is a preserved conduction of the visual pathways, while the abnormality in the latency has to be considered in the light of the laboratory specific norm. The VEP is important as the demyelination may influence the visual pathways even in the absence of clear visual manifestations.

Compared with previously published literature, this case demonstrates the typical radiological principle of multifocal CNS dissemination described in MS, but its clinical presentation is noteworthy for the prominence of sensory manifestations. Compston and Coles, Reich et al., and Dobson and Giovannoni have described MS as a disorder with substantial variation in clinical expression and lesion distribution [2,9,11]. The combination of brain and spinal cord lesions in the present case is also consistent with the radiological characteristics described by Filippi and Rocca and the spinal cord involvement reported by Bot et al [7,12]. Thus, the case reinforces existing knowledge while demonstrating how extensive radiological disease can present clinically with relatively focused sensory complaints.

For the acute relapse, intravenous methylprednisolone 1 g once daily for three consecutive days was administered. High-dose corticosteroids are recommended for clinically significant MS relapses because they shorten the duration and severity of acute inflammatory symptoms. Gabapentin was used for symptomatic management of neuropathic sensory symptoms, while pantoprazole was administered for gastrointestinal protection during corticosteroid treatment. Atorvastatin was continued for the patient's dyslipidaemia. Importantly, corticosteroid treatment addresses the acute relapse but does not prevent future disease activity; appropriate long-term disease-modifying therapy and continued neurological monitoring are therefore essential [8,10].

After treatment, the patient was stable in terms of his condition, with resolution of the presented sensory symptoms and absence of any new neurologic deficits in the hospitalization period. The successful performance of CSF analysis without any problems and discharge have enabled the patient to continue the examination by follow-up neurological evaluation. Clinical and MRI follow-up examinations are necessary to establish if there is any further inflammatory process and choose disease modifying therapy.

The most important aspect about this case is the clinical and radiological correlation between predominantly sensory symptoms and diffuse demyelination in the brain and spinal cord. The right-sided paraesthesia and the abnormalities in the abdominal sensation give a clue to the disturbance in the sensory pathways, whereas the multiple demyelinating lesions in the neck and thoracic region provide an appropriate substrate for such clinical features. The occurrence of typical lesions in the brain, spine, positive oligoclonal bands in the CSF and the supporting visual evoked potential test highlights the need for a multi-modal diagnostic approach. It shows that rather than being an isolated sensory problem, this case represents the clinical manifestation of a disseminated demyelinating disorder.

V. CONCLUSION

This case demonstrates the significance of the VEP test as an auxiliary investigation in the assessment of suspected multiple sclerosis. There was evidence of prolonged P100 latency bilaterally, suggesting delayed conduction along the

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visual pathways. Nevertheless, VEP results need to be considered in light of the clinical picture and the MRI, not as the sole basis for a diagnosis of MS.

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