



Hallucinating presentation of multiple sclerosis: A case report

Rashida Jabeen¹, Rukhsana Kousar¹, Narjis Raza Jafree², Humaira Iftikhar²

¹ University of Lahore, Lahore, Pakistan

² University of Health Sciences, Lahore, Pakistan

Abstract

There is variety of discriminating myelin nerve tissue ailments presented as a series of inflammatory demyelinating disorders including disseminated encephalomyelitis, neuromyelitis optica and Multiple sclerosis which do not occur immediately. Although the disease is not most common however multiple sclerosis has been found to be communal among inflammatory demyelinating diseases which may lead to serious disabilities as is presented a case of multiple sclerosis with exhibition of dementia primarily in this study. A woman aged to 37 years was referred to _____ with acute symptoms of drowsiness, aggression and departing monotonous activities, presented neurological discrepancies after 2-3 days. On further investigation through clinical and radiological concluded the diagnosis of multiple sclerosis however a massive variety of differential diagnosis to confirm the diagnosis of multiple sclerosis was ignored. Appropriate neurological investigation and proper imaging empowered the timely diagnosis of multiple sclerosis with dementia allowed to provide suitable treatment to the patient.

Keywords: dementia, multiple sclerosis, plasmapheresis

Introduction

There is variety of discriminating myelin nerve tissue ailments presented as a series of inflammatory demyelinating disorders including disseminated encephalomyelitis, neuromyelitis optica and Multiple sclerosis (MS) which do not occur immediately (Rahmlow *et al*, 2013)^[6]. Another monophasic form of inflammatory demyelinating disorders (IDDS) usually includes acute disseminating encephalomyelitis which is common in children after viral infection or vaccination (Scolding, N. 2014)^[7]. Neuromyelitis optica affects optic nerve considerably however also target the aquaporin-4 which is a channel protein of spinal cord (Weinshenker, *et al*, 2008)^[10]. The most communal IDD i.e. MS often progress chronically which may lead to number of disabilities (Rahmlow *et al*, 2013)^[6]. Sometimes IDDS mimic clinical orientation of infections like bacterial meningitis and herpes encephalitis (Martins, *et al*, 2004 & Wang *et al*, 2013)^[4, 9]. Determinations of therapeutic consequences are dependent on accurate diagnosis therefore para-clinical and clinical aspects of these patients must be essentially considered for definite diagnosis to save time (Karussis, D. 2014)^[2]. This case report is presented to demonstrate a young lady showing unexpected inception of dementia as primary sign of MS with steady progression to neurological discrepancies.

Case Report

A woman aged to 37 years formerly healthy was admitted to _____ hospital in December 2017 with current occurrence of progressive psycho-syndrome with symptoms of dysarthria and bilateral numbness of hand. Previous history of patient revealed hospital admission due to acute psychiatric signs comprising loss of aggression & energy, drowsiness, normal delivery four weeks before admission, gestational hypertension

and minor hypothyroidism. History of smoking, drug abuse, alcohol or any other addiction was absent. A history of lungs or colon cancer was also present in her family tree. Current admission was sought with detection and progression of dementia with uncertain fundamental etiology. Vital signs were found to be normal and a comprehensive neurological investigation was undertaken which showed dysarthria, vertical nystagmus and sustainable gaze-evoked, pre-umbilical hypoesthesia of both upper and lower extremities pre-dominantly on left side, lack of abdominal reflex and spinal ataxia. Nuchal rigidity, Brudzinski's and Kernig's signs were absent with weakness of musculoskeletal was revealed in physical examination. Drowsiness, reduced incitement, slower formal thought and Mnestic deficiency were observed in Psychopathology. An antipsychotic haloperidol 5mg once a day was used to control aggression.

Physical findings of patient were suggestive of central lesion while cognition involving disorders appear as slow activity was showed by electroencephalogram. Magnetic resonance imaging (MRI) at A 1.5-T showed various periventricular hyper-intensities. Consequently MRI of spinal cord showed a central lesion of 14x3 mm between C3 and C4 vertebra. The sense evoked potentials found to be normal. Patient was further tested for infectious diseases, complete antibody assay and C-reactive protein which found to be negative or normal further lacking signs of paraneoplastic syndrome. Cytology of cerebrospinal fluid showed 196 lymphocyte/ μ l, chemistry high protein level of 115mg/dl and measles rubella-varicella zoster test was positive. On the basis of results antibiotic and antiviral therapy was started immediately. Further results excluded lyme, herpes encephalitis and other bacterial or viral infections and patient was started with administration of methylprednisolone 2g for 5 days along proton

inhibitor pump and injections of anti-thrombosis hence antibiotics and antiviral were stopped. Although symptoms were improving with treatment but new abrasions were diagnosed in fresh MRI. Keeping in view McDonald criteria a diagnosis of MS was confirmed subsequently plasma-apheresis was started, subside both the symptoms and lesions in five cycles of well tolerated procedures of apheresis. Further therapy plan omitted antipsychotic treatment. Cytology and chemistry of cerebrospinal fluid became normal and patient was discharged with extended disability status scale of 1.5. An aggressive course with natalizumab was given for 2 weeks and control dose was administered for 28 months follow up and no episode of flare up was observed.

Discussion

Presentation of MS is rarely presented in studies as in this case of a young lady who was healthy earlier before sudden occurrence dementia. Symptoms of MS characteristically start with optic nerve, brainstem or spinal cord lesions with mood cognition lately (Messina, *et al*, 2014). Psychiatric disorders among MS patients have been scarcely presented in studies as Tapos *et al*, presented MS in a male patient aged to 16 years who was originally identified as schizoaffective disease and an MRI of brain was found to be shown the development of neurologic deficits confirming MS (Tapos, *et al*, 2013). Yadav, *et.al*, on the other hand presented a defined MS case in 2010 that showed psychosis and observed with temporal lesions (Yadav, *et al*, 2010). Kosmidis, *et.al*, in a study on psychotic topographies of MS reported that temporal abrasions on left side and treatment with corticosteroid may resolve the psychopathy of MS despite uncertain etiology (Kosmidis, *et al*, 2010). Presently definite diagnosis of MS was not an easy task as dementia is not a common primary feature of such patients, further lymphocytes of the patients were also almost double than the described values with high protein levels are not expected in MS patients challenged the diagnostic approach. Studies however have shown promising effect of pregnancy on MS and relapse over initial perinatal peak, though mechanism of action is still unknown (Hughes *et al*, 2014).

Presently dementia as the primary presentation of MS may be triggered by rapid hormonal variations at pre and post-partum. Though temporal lesions confirmed on MRI may result to develop confusion. However serial extreme therapeutic variances during propagation of MS have caused no impediment to date.

Conclusion

Complete physical examination and aggressive neurological investigations with apposite MRI enabled the definite diagnosis of MS to set aside delirium followed by effective treatment approach considerably help the patient to revive.

Conflict of Interest

None

Acknowledgements

References

- Hughes SE, Spelman T, Gray OM, Boz C, Trojano M, Lugaresi A, Grammond P. Predictors and dynamics of postpartum relapses in women with multiple sclerosis. *Multiple Sclerosis Journal*, 2014;20(6):739-746.
- Karussis D. The diagnosis of multiple sclerosis and the various related demyelinating syndromes: a critical review. *Journal of Autoimmunity*, 2014;48:134-142.
- Kosmidis MH, Giannakou M, Messinis L, Papathanasopoulos P. Psychotic features associated with multiple sclerosis. *International Review of Psychiatry*, 2010;22(1):55-66.
- Martins HM, Teixeira-Jr AL, Lana-Peixoto MA. Acute hemorrhagic leukoencephalitis mimicking herpes simplex encephalitis: case report. *Arquivos de Neuro-Psiquiatria*, 2004;62(1):139-143.
- Messina S, Patti F. Gray matters in multiple sclerosis: cognitive impairment and structural MRI. *Multiple Sclerosis International*, 2014.
- Rahmlow MR, Kantarci O. Fulminant demyelinating diseases. *The Neurohospitalist*, 2013;3(2):81-91.
- Scolding N. Acute disseminated encephalomyelitis and other inflammatory demyelinating variants. In *Handbook of Clinical Neurology*, 2014;122:601-611. Elsevier.
- Tapos D, Sivaswamy L. Mood disorder as the presenting manifestation of demyelination. *Case Reports in Neurology*, 2013;5(2):104-109.
- Wang JY, Wang K, Chen XW, Wang JW, Zhang K. Meningoencephalitis as an initial manifestation of neuromyelitis optica spectrum disorder. *Multiple Sclerosis Journal*, 2013;19(5):639-643.
- Weinshenker BG, Wingerchuk DM. Neuromyelitis optica: clinical syndrome and the NMO-IgG autoantibody marker. In *Advances in Multiple Sclerosis and Experimental Demyelinating Diseases*. Springer, Berlin, Heidelberg, 2008, 343-356.
- Yadav R, Zigmond AS. Temporal lobe lesions and psychosis in multiple sclerosis. *Case Reports*, 2010, bcr0120102651.