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Atypical Psycho-neurological Presentation of Multiple Sclerosis: A Case Report

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ABSTRACT

Background: Multiple sclerosis (MS) is a very common immune-mediated inflammatory demyelinating disorder of the central nervous system (CNS). Psychiatric and behavioral manifestations are well-documented but rarely dominate the initial sign, often adjourning diagnosis.

Case Presentation: We report the case of a 32-year-old male with a history of polysubstance abuse who presented with bizarre behavior, inappropriate laughter, and tremors, primarily suggesting a psychiatric disorder. Detailed assessment, including magnetic resonance imaging (MRI) and cerebrospinal fluid (CSF) analysis, showed findings consistent with MS, in accordance with the revised McDonald criteria. Exclusion of metabolic and genetic conditions strengthened the diagnosis. The patient was initiated on fingolimod 0.5 mg daily, ensuing in significant improvement of neuropsychiatric symptoms and tremors at two-month follow-up.

Conclusion: This case underlines the importance of preserving diagnostic vigilance for MS in patients presenting with atypical psychiatric or behavioral symptoms. Early recognition and timely initiation of disease-modifying therapy can improve both neurological and psychiatric outcomes.

Keywords: Multiple sclerosis; psychiatric onset; atypical presentation; fingolimod; case report

INTRODUCTION

Multiple sclerosis (MS) is an immune-mediated demyelinating disease of the CNS affecting over 2.8 million people globally.[1] Classically, MS presents in young adults with focal neurological deficits such as optic neuritis, motor weakness, sensory disturbances, gait imbalance, and bladder dysfunction.[2] Although psychiatric and behavioral disturbances such as depression, anxiety, and cognitive decline are highly predominant over the disease course, their occurrence as initial manifestations is infrequent.[3,4]

Neuropsychiatric onset complicates the diagnostic pathway, as patients are often misclassified as having primary psychiatric illness. This diagnostic delay may postpone initiation of disease-modifying therapy (DMT),

with adverse long-term effects.[5] The revised McDonald criteria emphasize dissemination of lesions in space and time, reinforced by paraclinical evidence such as CSF oligoclonal bands.[6] These principles are critical in distinguishing MS from imitating metabolic, genetic, and psychiatric conditions.

We present a case of a young man who originally exhibited bizarre behavior and tremors, prompting psychiatric evaluation before an eventual diagnosis of MS. This case highlights the clinical complexity of psychiatric-dominant MS onset and emphasizes the role of complete diagnostic workup and early therapeutic intervention.

CASE PRESENTATION

A 32-year-old Caucasian male with a past medical history of polysubstance abuse was brought to the emergency department for bizarre behavior at home, including urinating and defecating on the floor. He carried a prior diagnosis of Parkinson's disease made at a local clinic but was non-compliant with prescribed medications. The patient refuted recent illicit drug usage.

On examination, his vital signs were stable (BP 118/73 mmHg, HR 65 bpm, RR 12/min). He was thin, with inappropriate laughter and slow speech but no hallucinations. Ocular findings included right eye esotropia and marked horizontal and vertical nystagmus. Tremors were noted in all four extremities at rest, without cogwheeling. Psychiatry was consulted and excluded an acute psychiatric disorder.

Investigations:

- MRI brain (without contrast): suggested demyelinating or metabolic disorder.
- Copper studies: unremarkable.
- Lumbar puncture: 6 oligoclonal bands, elevated IgG index (1.03), and IgG synthesis rate (8.1).
- EEG: mildly abnormal with disorganized background and excessive beta activity.
- Metabolic/Genetic testing: very long chain fatty acids, arylsulfatase A, and C9ORF72 DNA were normal.
- MRI brain (with contrast): demonstrated Dawson's fingers protruding perpendicular into gray matter, highly suggestive of MS.

Differential Diagnosis:

Adrenoleukodystrophy, Wilson's disease, dystonia/Parkinsonism, spinocerebellar ataxia, Huntington's disease, vitamin E deficiency, essential tremor, and polysubstance abuse were considered but excluded based on investigations.

Management and Follow-up:

The patient was diagnosed with MS based on clinical, radiological, and CSF findings in accordance with the revised McDonald criteria.[6] He was initiated on fingolimod 0.5 mg daily. After 10 days of inpatient care, he was discharged to an acute rehabilitation facility for ongoing therapy. At two-month follow-up, the patient demonstrated improvement in bizarre behavior and tremor, and remained adherent to fingolimod therapy.

DISCUSSION

This case illustrates a diagnostically challenging presentation of MS dominated by psychiatric and behavioral disturbances. Although psychiatric symptoms occur in up to 95% of MS patients during their lifetime, psychosis, inappropriate behaviors, or bizarre conduct are rarely the first manifestations.[7]

Neuropsychiatric Spectrum in MS:

Cognitive impairment and depression are the most common neuropsychiatric features, with psychosis being far less frequent.[8] Bader et al.[9] reported a case where psychotic symptoms revealed underlying MS, paralleling our patient's initial psychiatric presentation. Pathophysiologically, psychosis in MS may be linked to lesion burden in temporal and periventricular regions, white matter disconnection, and neuroinflammatory cytokines.[10]

Diagnostic Considerations:

The McDonald criteria (2017 revision) integrate MRI evidence of dissemination in time and space with CSF markers.[6] Our patient's MRI showed Dawson's fingers—classic radiological features of MS—alongside positive CSF oligoclonal bands, fulfilling diagnostic requirements. Differential diagnoses including metabolic (Wilson's disease, adrenoleukodystrophy) and genetic disorders (spinocerebellar ataxia, Huntington's disease) were appropriately excluded.

Therapeutic Implications:

Fingolimod, an oral sphingosine-1-phosphate receptor modulator, has demonstrated efficacy in reducing relapse rates and delaying disability progression in relapsing MS.[11] Recent studies suggest potential benefits of fingolimod in mood regulation, with reduced odds of depression among stressed MS patients.[12] While evidence regarding psychotic presentations remains sparse, our case supports fingolimod's potential in ameliorating both neurological and psychiatric symptoms.

Literature Context:

Neuropsychiatric onset of MS has been described in several case reports.[9,13] These cases consistently emphasize delayed recognition due to initial psychiatric labeling, underscoring the need for heightened clinical suspicion. A systematic review of psychiatric manifestations in MS highlighted that psychiatric symptoms can precede neurological deficits in 2–3% of cases.[14] Early initiation of DMT is critical, as delays are associated with poorer long-term outcomes.[5]

Lessons for Clinical Practice:

1. Psychiatric or behavioral changes in young adults warrant neurological evaluation, particularly if atypical in onset or course.
2. MRI and CSF studies remain pivotal for differentiating MS from primary psychiatric disorders.
3. Disease-modifying therapy such as fingolimod may be effective even when psychiatric

manifestations dominate the clinical picture.

CONCLUSION

This case highlights an atypical psycho-neurological presentation of MS, where bizarre behavior and tremors preceded classic neurological findings. Clinicians should maintain a high index of suspicion for MS in patients with atypical psychiatric symptoms, particularly when accompanied by subtle neurological signs. Early recognition and initiation of DMTs such as fingolimod can significantly improve outcomes.

DECLARATIONS

- **Patient Consent:** Written informed consent was obtained from the patient for publication.
- **Ethics Approval:** Not applicable.
- **Funding:** None.
- **Conflicts of Interest:** The author declares no conflicts of interest.

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