CASE REPORT

Atypical Onset Of Primary Progressive Multiple Sclerosis: Emotional Lability As The Initial Manifestation

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ABSTRACT

Multiple Sclerosis (MS) is a chronic autoimmune disorder characterized by demyelination of axons in the central nervous system. While it commonly presents with physical symptoms, psychiatric manifestations such as emotional lability or delirium can be the first indicators in rare cases. These early symptoms are often overlooked until significant physical disability develops. We report a case of a young woman who initially experienced unexplained bouts of crying, laughing, and emotional instability, which were misdiagnosed as psychiatric illness. Her condition remained undiagnosed until she developed spastic paraplegia, leading to an extensive workup that confirmed primary progressive multiple sclerosis (PPMS). This case highlights the importance of considering organic causes when evaluating psychiatric symptoms, ensuring timely diagnosis and appropriate management.

Keywords: *Multiple sclerosis, autoimmune disease, psychiatric symptoms, emotional lability, primary progressive MS*

INTRODUCTION:

Multiple sclerosis (MS) is an inflammatory, demyelinating, and neurodegenerative disease with a diverse clinical presentation. Traditionally, MS has been diagnosed based on its classical neurological symptoms and disease course, typically relapsing-remitting or progressive disability patterns1. However, in rare instances, MS can manifest initially with psychiatric symptoms such as emotional lability or delirium, which can delay diagnosis2. Mahboobi et al. were among the first to report MS presenting as delirium rather than with neurological deficits3.

This case report describes a young woman whose initial symptoms of emotional instability were dismissed as a psychiatric disorder until she developed profound neurological deficits, ultimately leading to a diagnosis of primary progressive multiple sclerosis (PPMS).

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A 26-year-old woman presented with a two-year history of unexplained emotional outbursts, including frequent bouts of crying, weeping, and laughing without an identifiable trigger. She consulted multiple physicians, who attributed her symptoms to a psychiatric disorder, leading to her referral to a psychiatrist. Despite receiving psychiatric treatment, for which no medical records were available, her condition did not improve. Due to the social stigma surrounding mental illness, her family discontinued psychiatric consultations and sought alternative treatment from faith healers.

Approximately six months prior to her presentation, she began experiencing progressive lower limb weakness, initially as painful difficulty in walking and performing daily tasks. Over time, her condition

deteriorated, and she became completely bedridden one month before seeking medical attention.

On neurological examination, she exhibited increased muscle tone in the lower limbs, exaggerated knee reflexes (+++), diminished ankle reflex (+), bilateral Babinski signs, and pes cavus deformity. In the upper limbs, deep tendon reflexes were markedly exaggerated (++++) with positive Hoffman's sign and inversion of the biceps and brachioradialis reflexes. She had scanning speech but no other cerebellar signs. Additionally, right-sided seventh cranial nerve palsy and left hypoglossal nerve involvement were noted, while other cranial nerves and sensory functions were intact.

Given her presentation, a differential diagnosis of inflammatory demyelinating diseases such as MS, Friedrich's ataxia, hereditary spastic paraplegia, and motor neuron disease was considered. Brain and spinal cord MRI revealed hyperintense lesions on T2-weighted and FLAIR sequences in the bilateral parietal white matter, some perpendicular to the corpus callosum, consistent with Dawson's fingers. No perilesional edema, mass effect, or midline shift was observed. Spinal MRI was unremarkable.

Cerebrospinal fluid (CSF) analysis revealed the presence of oligoclonal bands, confirming the diagnosis of primary progressive multiple sclerosis.

DISCUSSION:

Psychiatric or cognitive symptoms as initial manifestations of MS are rare and often overlooked. Physicians frequently rely on physical neurological signs for MS diagnosis, which can delay early intervention. Mahmoud et al. reported a similar case of a female initially diagnosed with schizoaffective disorder, which an MRI later confirmed as multiple sclerosis following neurological deficits4. Likewise, Enderami et al. documented an MS patient presenting with psychosis linked to left temporal lobe lesions5. A review by Sabe et al. stated that any delay or poor response to antipsychotic treatment should warrant a need to dig deeper into other differentials like multiple sclerosis6.

In our case, the patient experienced two years of misdiagnosed emotional instability before developing neurological deficits. The diagnosis was ultimately confirmed based on progressive symptoms, MRI findings of periventricular and high parietal lesions, and CSF analysis showing oligoclonal bands. Similar to the case reported by Mahmoud et al., our patient underwent an extensive diagnostic workup only after significant physical symptoms emerged.

This case underscores the necessity of considering organic causes in patients with unexplained psychiatric symptoms. If psychiatric symptoms persist despite appropriate treatment, further neurological evaluation, including imaging and CSF analysis, should be performed to rule out underlying neurological disorders.

CONCLUSION:

This case highlights the need for increased awareness of atypical MS presentations, particularly psychiatric symptoms. Physicians should remain vigilant in evaluating patients with unexplained emotional instability, ensuring that MS and other neurological conditions are considered before attributing symptoms solely to psychiatric disorders. Early recognition and diagnosis can lead to timely intervention, potentially improving patient outcomes.

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