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Depression in Multiple Sclerosis: A Case Report and Review of Clinical Considerations

J. Salim^{1*}, W. Mansouri¹, Y. Amara¹, K. Benallel¹, K. Mouhadi¹, M. Kadiri¹

¹Psychiatry Department, Mohammed V Military Teaching Hospital, Rabat

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*Corresponding author: J. Salim

Psychiatry Department, Mohammed V Military Teaching Hospital, Rabat

Abstract		Case Report
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Multiple sclerosis (MS) is a chronic demyelinating disease of the central nervous system that can result in various neuropsychiatric manifestations, particularly mood disorders. Depression is the most prevalent psychiatric comorbidity in MS, often preceding the neurological diagnosis. We present the case of a 23-year-old female referred for depressive symptoms including low mood, tearfulness, asthenia, anorexia, and insomnia. She responded favorably to Sertraline and Alprazolam, but two months later developed progressive vision loss. Brain MRI revealed demyelinating lesions, and a diagnosis of MS was established. Corticosteroid therapy followed by interferon beta-1a led to visual recovery and further improvement of mood. This case illustrates how depression can precede or mask the neurological and neurobiological mechanisms, including cytokine imbalances and lesion localization. While interferons may trigger or worsen depressive symptoms in susceptible individuals, their role remains controversial. The overlap of MS symptoms with those of depression complicates diagnosis, underscoring the importance of comprehensive clinical and neurological evaluation. Early psychiatric intervention and careful monitoring during immunomodulatory therapy are critical to improving outcomes and quality of life in patients with MS.

Keywords: Multiple sclerosis; Depression; Mood disorders; Neuropsychiatry; Psychoneuroimmunology, Demyelination;.

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INTRODUCTION

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease that primarily affects the white matter of the central nervous system (CNS) and also leads to neuro-axonal loss. This widespread involvement of the CNS can result in psychiatric disorders, particularly mood disorders, with depression being the most common.

CASE DESCRIPTION

A 23-year-old female patient was referred to us for depressive syndrome by her gastroenterologist, who had been managing her for constipation. The psychiatric assessment revealed a sad mood with crying episodes, irritability, asthenia, anorexia, and sleep-onset insomnia. The patient was prescribed Sertraline 50 mg and Alprazolam 0.5 mg daily. One month later, an improvement in mood and appetite was noted. Two months after the initiation of treatment, she developed progressive blindness. A brain MRI revealed demyelinating lesions in the central nervous system, indicative of multiple sclerosis. She was treated with corticosteroid pulses followed by Beta-1A interferon, which led to the resolution of visual disturbances after one month and improvement in psychiatric symptoms.

DISCUSSION

Mood disorders and depression affect more than half of MS patients. They are detected at all stages of the disease and interact with other symptoms such as fatigue and cognitive impairment. The prevalence of depression in MS is well established, and despite the usual variations in studies on depressive manifestations in CNS disorders, approximately one in two patients experiences depression [1], with a suicide rate 7.5 times higher than that of the general population [2].

Diagnosing depression in MS is challenging due to the frequent occurrence of emotional lability (see below), overlapping symptoms such as fatigue and cognitive disturbances, and the difficulty some patients experience in verbalizing their emotions. Other studies have also highlighted the role of brain lesions in fostering

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depression: while depression is not more frequent in MS than in patients with traumatic spinal cord injuries, it tends to be more severe [3].

Certain medications seem to exacerbate an underlying depressive state, particularly interferon therapy. The onset of depressive syndrome, as assessed by the Beck Depression Inventory, appears to correlate with elevated interferon-gamma levels [4]. Researchers also emphasize that this biological marker is reversible with antidepressant treatment. However, establishing a causal relationship remains complex.

The CHAMPS study demonstrated a significant incidence of depressive syndrome (20%) in patients treated with intramuscular interferon beta-1a after a first demyelinating event, compared to 13% in the placebo group [5]. Conversely, in the PRISMS study, which compared subcutaneous interferon beta-1a ($22 \mu g$, $44 \mu g$) with placebo, no difference was observed in depression frequency or severity [6].

Nonetheless, researchers recommend caution when prescribing interferons to patients with a history of depressive syndrome, as they may be more vulnerable to developing depression. In such cases, prescribing an antidepressant before initiating interferon therapy may be advisable [7].

CONCLUSION

Depression is common in MS. Its varied symptomatology, both somatic and cognitive, overlaps

with MS symptoms, making it difficult to determine whether depression is an intrinsic manifestation of the disease or a complication.

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