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Multiple Sclerosis's Neuropsychiatric Manifestations and Mental Health Symptoms Linked to it

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Introduction

Multiple sclerosis is the most common chronic neurologic condition affecting young adults in the United States, with a prevalence of approximately 1 in 1000. Previously thought to be an inflammatory, demyelinating disease primarily affecting central nervous system (CNS) white matter, more recent imaging studies have shown that significant damage to cortical gray matter also occurs. Multiple sclerosis affects twice as many women as men, and the prevalence rises as geographical distance from the equator increases. Visual disturbances (diplopia, blurred vision), weakness, gait disturbance, vertigo, fatigue, urinary retention and incontinence, and difficulties with speech and swallowing are all common clinical signs and symptoms of MS. Neuropsychiatric symptoms are also common and may be the first sign of MS. Because many of the typical signs and symptoms are nonspecific and pseudoneurologic, patients are frequently mistaken for having a primarily mental illness, and a diagnosis may be delayed [1].

Supporting laboratory data include the presence of oligoclonal IgG bands on cerebrospinal fluid analysis, abnormalities of visual-evoked potentials, and characteristic MRI lesions corresponding to "plaques" of demyelination.3 There are four MS subtypes that correspond to the course of illness. The clinical diagnosis of multiple sclerosis is based on the presence of neurologic symptoms that are disseminated in space and time relapsing-remitting (66 %), secondary-progressive (16 %), primary-progressive (15 %), and benign MS.8 In relapsing-remitting MS, patients fully recover between exacerbations, whereas primary-progressive MS patients experience accumulating symptoms and disability from the beginning of the disease without going into remission. Mr. A suffers from the secondary-progressive subtype, in which patients experience exacerbations and apparent recovery early in the course of the disease, but symptoms eventually progress and remission is not achieved [2]. A small percentage of patients with benign MS only experience one MS episode and never experience any more exacerbations [3].

Which mental health symptoms are linked to multiple sclerosis?

Mental side effects in MS are profoundly predominant and much of the time neglected in clinical settings. Out of 1 investigation of backsliding transmitting patients with MS going away, 95% detailed critical mental side effects, most often dysphoria (79%), unsettling (40%), tension (40%), and crabbiness (35%).15

Significant burdensome problem (MDD) is especially normal, with a lifetime predominance pace of roughly 50%, when contrasted with a pace of 10% to 15% in everyone. In one study, depression surpassed physical disability and cognitive function as a significant determinant of quality of life13. Several factors related to MS symptomatology, disease course, and treatment may contribute to the exceedingly high rates of depression and its complications seen in MS patients. Suicide rates are also significantly higher in those with MS. However, studies generally support the strikingly high prevalence of depression in MS patients, finding high rates of MS-associated depressive disorders even when somatic complaints are not included in the diagnostic criteria.The burden of accepting a lifelong, progressive, and incurable illness likely plays a role in the development of depression as well. For instance, MS and depression share several neurovegetative symptoms (such as fatigue, poor concentration, and disturbances of sleep and appetite). Depression has been linked to a shorter illness duration (less than one year), which may be due to the difficulty of adjusting to a new diagnosis of this chronic, unpredictable illness; This finding emphasizes the importance of screening MS patients frequently for depression [4]. Reliable with this perception, the patient introduced for the situation vignette, Mr. A, accomplished his most memorable episode of MDD not long after his underlying determination of MS at age 18 years, coming full circle in a fruitless self destruction endeavor. Mr. A was facing a decline in his ability to function without assistance at home, yet he fiercely defended his independence, frequently making statements that hospital and home care made him "more depressed." Although data on the relationship between physical disability and depression are mixed, depressed patients with MS may experience the impact of their disability as greater than nondepressed patients. In addition, physical disability in MS frequently leads to social isolation, loss of independence, and loss of recreational activities, factors that may contribute to the Emotion-centered coping (in contrast to active, problem-centered coping strategies) and feelings of hopelessness are additional psychological characteristics that are predictive of depression in MS patients [5]. Major depressive disorder (MDD) in MS patients has been associated with characteristic CNS changes, including cortical atrophy and lesions in specific regions of the frontal lobes, which may account for some of this observed difference. Major depressive disorder in patients with MS, therefore, is unlikely to represent a simple reaction to the physical disability, uncertainty, and decreased independence that the illness entails. However, given its prevalence, it may be considered a symptom of the illness itself and reflective of CNS cort.

Last but not least, the effects of MS treatment, particularly interferon, may occasionally aggravate depressive symptoms. Bipolar disorder is twice as common in MS patients as it is in the general population and frequently presents later in the course of MS, as in the case of Mr. A, who presented with his first episode of mania at the age of 41 [6,7]. Although studies have not demonstrated a well-defined relationship between treatment with interferon and depression, such studies are plagued by methodological flaws and results that are

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inconsistent. Glatiramer acetate has generally not been associated with deterioration in mood. Mr. A's late onset of manic symptoms would be highly atypical for primary bipolar disorder and suggests that MS lesions in critical brain regions may be a substantial contributing factor to his presentation. For example, lesions along the orbitofrontal prefrontal cortex circuit lead to impulsivity, mood lability, and personality changes, symptoms frequently seen in acute mania. On the other hand, a history of manic or hypomanic symptoms in Mr. A may have been long overlooked given the complexity

How can multiple sclerosis's neuropsychiatric symptoms be evaluated and treated?

All MS patients should be routinely screened for neuropsychiatric symptoms due to the high prevalence of mood disorders, psychosis, and cognitive impairment [8]. If it is found, these patients' quality of life can be greatly improved with prompt intervention.

Simply asking patients about their moods when screening for depression ("Are you depressed?") furthermore, anhedonia ("Do you actually find delight in things you used to appreciate?") can quickly identify patients who might benefit from a more in-depth assessment. When MS patients have prominent somatic complaints, it can be particularly helpful to identify true depression by asking them about their capacity for pleasure and hope for the future. In addition, as with other depressed patients, assessing a patient's sense of hope for the future is crucial in determining their risk of suicide. This is a useful tool for screening for depression. Alternately, a nine-item Patient Health Questionnaire-929, a brief self-report survey for depression, can be completed in the waiting room for review during the appointment. To tailor the approach to treatment, physicians should attempt to identify any contributing factors (such as the stress of the diagnosis, disability, or treatment with steroids or interferon) after depressive symptoms are detected. Cognitive-behavioral therapy is the only psychological intervention that shows modest benefit for the treatment of depression in MS.31 It may be especially helpful for improving functional outcome by coaching the patient to actively re-engage in daily activities despite fatigue, low energy, and lack of motivation. Each patient's individual factors that contribute to depression must be carefully considered for optimal treatment [9]. A peer support group or individual psychotherapy, for instance, may be beneficial to a patient who develops depression shortly after a new MS diagnosis, whereas antidepressant medication may be most beneficial to a patient who develops depression shortly after beginning interferon therapy. The multifactorial nature of depression in many patients necessitates a tailored and adaptable treatment plan.

Antidepressants may precipitate manic, hypomanic, or rapid cycle of mood episodes in patients with underlying bipolar disorder, so assessing patients for current or past manic symptoms is also essential when considering treatment for depression [10-12]. A few straightforward screening questions (such as "Have you ever felt so good or so "hyper" that people thought you were not your normal self, or were you so "hyper" that you got into trouble?") are similar to those used for depression. Have you ever experienced a time when you slept in significantly less than usual but found that you didn't really miss it?") can identify patients who require additional investigation of manic symptoms.

It is reasonable to anticipate the effects that commonly used treatments for managing neurologic MS symptoms can have on a patient's mood and behavior. Clinicians who use corticosteroids to treat acute MS exacerbations need to be especially careful for neuropsychiatric sequelae like depression, mania, and psychosis. Even though these side effects can happen to anyone, it's important to look at a patient's family history to see if they are more likely to get them because they could be a result of a genetic predisposition [13]. Pre-morbid depression and a family history of mood disorders may predispose a patient to the development of depression with interferon treatments, just as they do with corticosteroid treatment. As with corticosteroid treatment, available data generally do not support an association between treatment with interferon and the onset of depression. However, idiosyncratic reactions are possible. For patients with a known history of significant burdensome episodes, clinicians ought to think about inception of treatment with a SSRI preceding starting interferon. Core MS treatments may also reduce psychiatric symptoms. Healthcare providers should also keep in mind that interferon treatment that delays physical disability may improve self-esteem and increase independence, lowering the risk of depression.

Conclusion

Multiple sclerosis is a disease of the central nervous system with protean manifestations. Affective and cognitive neuropsychiatric changes are common in multiple sclerosis and are frequently regarded as the primary symptoms of this prevalent neurologic disorder. When predicting quality of life, these symptoms may be just as important as physical disability. As a result, providing patients with MS with the best possible care necessitates that healthcare providers carry out the appropriate screening and thoughtful management of neuropsychiatric sequelae.

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