PSYCHOTIC DEPRESSION: A PECULIAR PRESENTATION FOR MULTIPLE SCLEROSIS

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Multiple sclerosis (MS) is frequently associated with a number of different psychiatric syndromes. Solely psychiatric syndrome may be the first clinical presentation of multiple sclerosis. We report a patient whose first attack was psychotic depression. The present case emphasizes that psychiatric symptoms can occur at any time during the course of the disease and, moreover, may be the presenting feature.

Keywords attack, depression, imaging, interferon, multiple sclerosis, psychosis

INTRODUCTION
Multiple sclerosis (MS) is frequently associated with a number of different psychiatric syndromes. These psychiatric syndromes are broadly divided into...
two categories: 1) those related to mood, affect, and behavior and 2) those impairing cognition (Feinstein, 2007). In MS, admission to psychiatry prior to neurology is not uncommon. Approximately 2%–9% of all MS patients, especially those with vague symptoms, had a psychiatric diagnosis before the diagnosis of MS was made (Skegg, Corwin, & Skegg, 1988; Stenager & Jensen, 1988). Several types of psychiatric problems can be seen at any stage of the course of MS (Diaz-Olavarrieta, Cummings, Velazquez, & Garcia de la Cadena, 1999). However, psychiatric onset of MS is not frequent (Jongen, 2006). The present case demonstrates that the psychiatric symptoms preceding the neurologic findings is possible in MS patients.

CASE REPORT

In 1999, a previously healthy 34-year-old lecturer in fine arts was admitted to a psychiatrist and diagnosed as major depression with psychotic features. Antidepressant and antipsychotic medications were initiated. In 2000, she presented with loss of weight, loss of appetite, insomnia, anhedonia, and hallucinations. Antipsychotic treatment with zuclopentixol and sulpride was given. Until 2004 she had one more major depressive episode with psychotic features treated with sertraline, maprotilin, and thioridazine. In 2004, she developed new psychiatric features consisting of mood instability composed of emotional lability and euphoric and manic episodes of affective dysregulation. Lithium treatment was started. Soon after this episode, she presented with left retrobulber optic neuritis. During this attack she was put on pulse methyl prednisolone for three days at the ophthalmology clinic where she had been followed. Cranial magnetic resonance imaging performed during this period showed hyperintense lesions less than five in number and located periventricularly (Figure 1). In 2005, she complained of sleep disturbances, frequent awakenings, and loss of energy. Depression with anxiety was the diagnosis. She was put on risperidone treatment by her psychiatrist. She first encountered a neurologist in 2005. Complete diagnostic workup was done to differentiate vasculitis and demyelinating disorders. Somatosensory and visual evoked responses were abnormal on the left side. Cerebrospinal fluid analysis showed oligoclonal bands.

One year later, while she was taking antipsychotics, she had a control cranial MRI to determine the follow-up of the above described lesions (Figure 2). At that time her psychiatric picture was resistant to antipsychotics and psychiatric diagnosis was psychotic depression. The new MRI exposed an increase in white matter lesions without gadolinium enhancement. And
the neurologist accepted the diagnosis of definite MS and decided to initiate interferon beta 1a intramuscularly once a week. Her expanded disability status scale (Kurtzke, 1983) was zero and cognitive function evaluation did not demonstrate any pathology on executive functions. After four months, before she developed right-sided pyramidal tract involvement, her psychiatric state worsened again and diagnosed as relapsing psychotic depression. Brain imaging was repeated and a dramatic increase in lesion volume and gadolinium enhancement was observed (Figure 3). Her EDDS (Kurtzke,
1983) was one (pyramidal = 1) and she received pulse methyl prednisolone for seven days in the neurology clinic. Both neurological and psychiatric statuses were resolved in the following weeks. The patient asked to visit our outpatient clinic at three-month intervals and during the last visit performed 12 months after the last attack her neurologic and psychiatric evaluations were normal. She is still on antidepressant (fluoxetine HCl 20 mg/day) and interferon beta 1a treatment.

**DISCUSSION**

Individuals living with MS have increased vulnerability to psychiatric syndromes as high as 95% (Diaz-Olavarrieta et al., 1999). Depression by
far is the most common outcome (79%) (Diaz-Olavarrieta et al., 1999). In some MS patients depression periods will come even prior to and together with the demyelinating attacks. In this group of patients, the exact diagnosis is one of the most challenging issues for clinicians. In the reported case, there were two episodes of depression prior to the development of frank neurological symptoms. At that time, her psychiatric symptoms were resistant to pharmacologic treatment modalities. The last two depression episodes preceded the neurological deterioration and subsided as the neurological findings faded. Psychiatric symptoms alone could represent an MS attack if the patient has the diagnosis of definite MS. However, if the psychiatric features were experienced before the neurological symptoms, clinicians need very convincing paraclinical laboratory findings to diagnose the patients as definite MS. In the history of our patient, she had two psychotic depression episodes within a one-year interval and four years later she had retrobulbar neuritis. She had abnormal oligoclonal bands in CSF, progressively increasing lesion load in her cranial MRI. During follow-up of the patient, we observed the fluctuation of psychiatric symptoms and especially relapses in last two before the neurological deterioration occurred. Similar results have been mentioned in the literature. Kroencke, Denney, and Lynch (2001) discussed the fluctuations of psychiatric symptoms, namely depression, in accordance with neurologic findings and the fact that these psychiatric symptoms could resolve as the neurologic attack subsides. However, it is hard to make the decision to begin steroids for attack treatment, since the steroid itself can induce a variety of psychiatric symptoms varying from a sense of well being to hypomania and even to frank psychosis (Rome & Braceland, 1952). The present case experienced two attacks with abnormal neurological findings preceded by major depressive episodes and was successfully treated with pulse MP.

There are plenty of studies of the neuroanatomical correlates of depression in MS; however, there is no consensus between the authors. Disconnection in the projections between cortico-subcortical and limbic regions (Berg et al., 2000), in the temporal lobe (Zorzon et al., 2001), arcuate fascicule in the dominant hemisphere (Pujol, Bello, Deus, Martí-Vilalta, & Capdevila, 1997) frontal especially the medial inferior prefrontal cortex, parietal and dominant hemisphere temporal lobe (Feinstein et al., 2004) are claimed areas. Different studies emphasize for depression of MS patients involvement of the left anterior temporoparietal region (Siegert & Abernethy, 2005). In our case, lesions on right superior frontal gyrus and left supramarginal gyrus were most prominent. However, it is hard to make a speculation about the lesion localization and
psychiatric symptoms other than a kind of disconnection syndrome since arcuate fasciculus was affected.

Patients with psychiatric onset MS have been rarely been described in the literature (Jongen, 2006). Our case has no neurological signs until she developed retrobulber optic neuritis. Extensive work-up to diagnose MS whenever suspected must take place even when the patient has little or no neurological signs (Jongen, 2006). Sometimes a very resistant psychiatric symptom group can cause a suspicion of organicity and need to be recorded. The present case emphasized that psychiatric symptoms can occur at any time during the course of the disease and moreover may be the presenting feature.

REFERENCES


