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Psychiatry Complications of Multiple Sclerosis: A Case Review

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ABSTRACT

Multiple Sclerosis is the commonest cause of neurological disability in young and middle aged adults. While it increases vulnerability to psychiatric illnesses, only very few of these patients receive any form of help. There is an increasing recognition and an attempt to elucidate the role of psychiatric symptoms in the course of the disease. The most debilitating symptoms that have been reported are changes in mood, personality and cognitive functioning. Patients with MS an increase in extrapyramidal side effects to antipsychotics including second generation antipsychotics, probably due to an underlying organicity. We present a patient with multiple sclerosis and an array of neuropsychiatric symptoms/syndromes with attendant difficulty in management.

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INTRODUCTION:

Multiple Sclerosis [MS] is a progressive disease of the central nervous system [CNS] characterized mainly by a recurrent or chronic immune mediated inflammatory and demyelinating changes [1,5]. These often lead to a pattern of acute relapses and remissions of neurological deficits which are distressing to the individual. Over the long-term course of the illness, there is also a progressive accrual of neurological and cognitive deficits. It is typified by an array of symptoms ranging from severe neurological symptoms and somatic signs to behavioral disturbances. Its course could be unpredictable and has a disparate clinical presentation which may follow a relapsing and remitting or progressive course [1,5,8].

There is an increasing recognition and an attempt to elucidate the role of psychiatric symptoms in the course of the disease. The most debilitating symptoms that have been reported are changes in mood, personality and cognitive functioning [4,6]. Patients with multiple sclerosis have been shown to have an increased vulnerability to psychiatric illness, however only very few of these patients receive any form of help [1,4,5].

We present a patient with multiple sclerosis and a wide array of neuropsychiatric symptoms with attendant difficulty in management. The onset of psychiatric symptoms was prior to the diagnosis of MS.

CASE DESCRIPTION:

Patient is a 19-year-old Caucasian female who presented to the inpatient unit from the emergency room for suicidal ideation and responding to internal stimuli. Per patient's mother: she began noticing changes in her daughter starting 6 months prior to presentation, just after she graduated high school. Changes included patient talking to self, sexual promiscuity, laughing inappropriately, hypersociability, poor hygiene, hyper-religiosity, compulsive lying, wandering away from home and eating large amounts of food. Patient reported that she has had a boyfriend of 5 years, has been arrested for breaking and entering, grand theft auto (stealing a car), uses various psychoactive substances including "kush" (high-grade marijuana), cigarettes, alcohol, and "rollos" (methylenedioxymethamphetamine). She also reported "doing everything you can think of" whenever she got them. She stated she has 6 children, having had 2 sets of triplets, all of whom are in foster care. She reported also that one of her daughters died about 1 month prior, and that her mother physically abused her since she was a child. Mother denied all the claims regarding having children. Current and

past emergency department notes showed negative alcohol and toxicology screening. She endorsed hearing voices of several people talking to her and among themselves but could not elaborate further. Family history was positive for bipolar disorder in patient's mother and "extensive history" of bipolar and schizoaffective disorders on patient's mother's side of family.

Patient stated she was diagnosed with multiple sclerosis about 1 month prior to presentation. She received this diagnosis after she noticed symptoms that waxed and waned over the last 3-4 months of numbness and tingling in her hands, pain down her spinal cord, memory impairment and visual changes. Patient was not receiving treatment at that time or at the time of this encounter. Magnetic Resonance Imaging (MRI) of the brain without contrast shows an extensive abnormal white matter lesion noted in the periventricular, bi-frontal, bi-parietal and bi-temporal gray-white junctions [Figure 1] and the bilateral centrum semiovale and the higher convexity. The lesion also involves the corpus callosum [Figure 2] with mild dilatation of the ventricles. There is no focal mass and the brainstem and cerebellum appears spared.

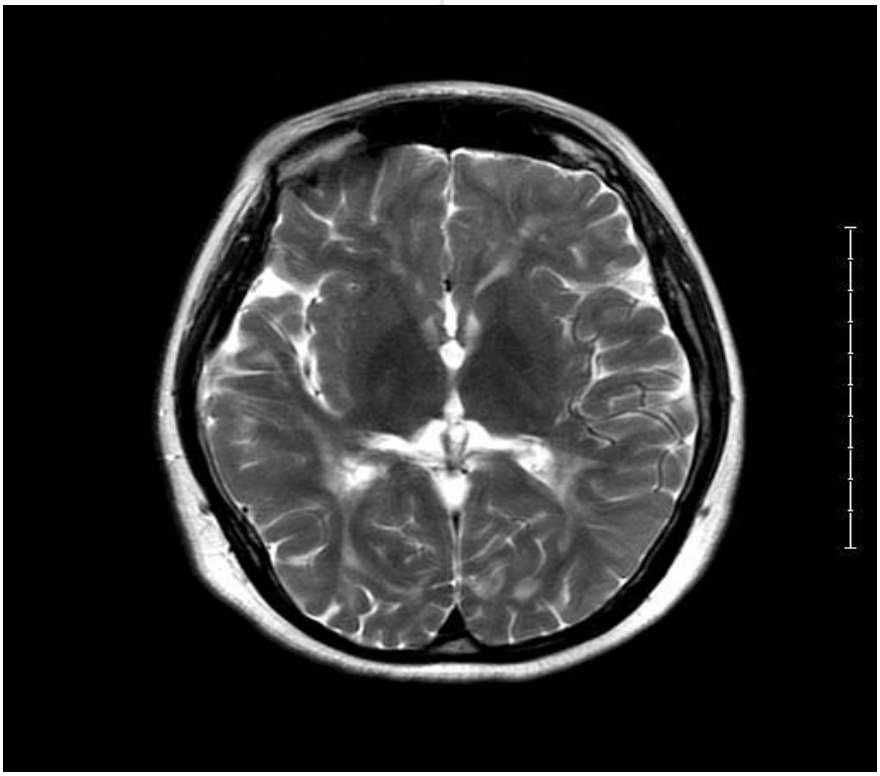

Figure 1

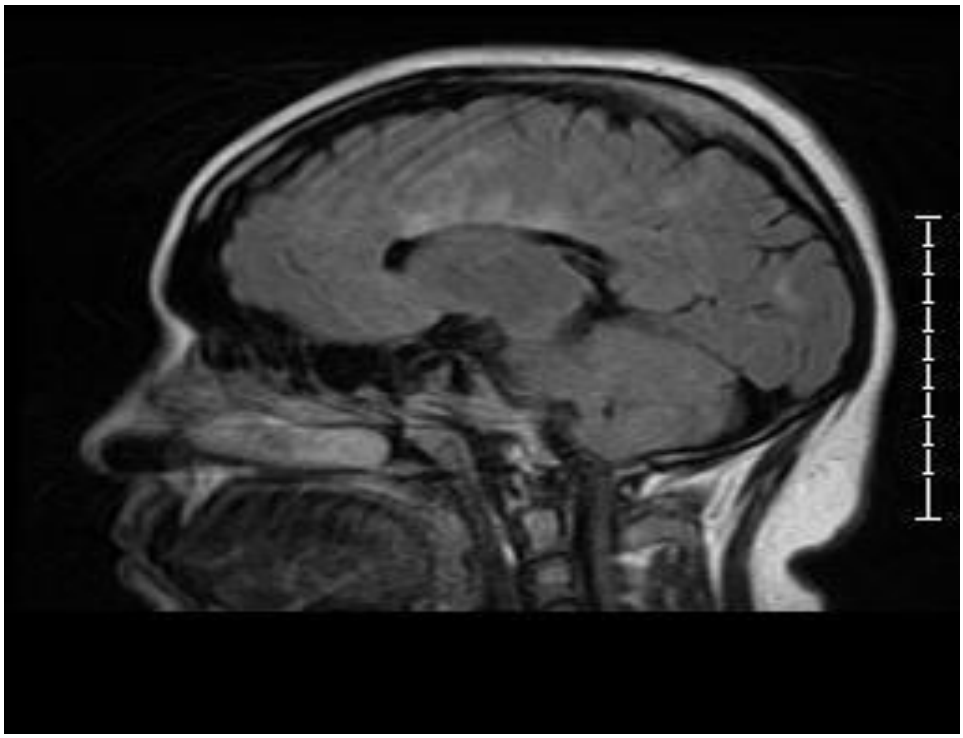

Figure 2:

Mental status examination revealed a young female, awake and alert, very dismissive and disinterested in the conversation. Patient was also irritable, talkative, restless, childish, had tangential and circumstantial speech, making inappropriate jokes, laughing to herself at random times, talking to herself, making clanging associations like "I am a boss like ross," internally preoccupied, socially disinhibited, over-inclusive, reported auditory hallucinations, suicidal ideation, and thoughts of harming her mother. Appeared to have grandiose delusions with a religious theme. Mood was "I am blessed" with an expansive, inappropriate and mood incongruent affect. She had poor judgment and lacked insight. Patient was uncooperative during a Mini Mental State Examination (MMSE), however, stated she didn't know what the date was and didn't have to know who the current president of the United States was. Physical examination yielded a wide based ataxic gait. She was not cooperative with respect to the rest of the neurological examination. Laboratory investigations including CBC, CMP, thyroid panel and urinalysis were all within normal limits. Pregnancy test was negative.

An assessment of MS induced mood disorder bipolar type with psychotic features was made based on the temporal relation. Also noted was the fact that patient had several behavioral syndromes including, pseudobulbar affect, euphoria, psychosis, and personality change.

Patient was started on Risperidone, initially at 1mg bid then increased to 2mg twice daily [BID]. Benztropine was added at 1mg BID to alleviate extrapyramidal symptoms in the form of stiffness and bradykinesia. Risperidone has adjusted to 1 mg in the morning and 3mg at bedtime [HS] on day 4 to decrease daytime sedation and Lithium 300mg BID was added and titrated to 600 mg BID over 8 days. Side effects to Risperidone continued to be an issue, patient also continued to be very disinhibited and expansive. She would go into other patients' room asking to pray for them. She continued to report experiences of auditory hallucinations, delusions of having a family and suicidal ideation without an accompanying affective response to feeling suicidal. Antipsychotic was switched to Olanzapine and then Quetiapine over the course of several weeks with no response from patient. She was eventually switched to Paliperidone at 3mg HS and titrated to 9mg at bedtime in addition to long acting Paliperidone at 234 mg monthly. Lithium was also switched to Oxcarbazepine (Trileptal®) titrated up to 900 mg BID because of poor response, marked fluctuating plasma lithium levels and excessive sedation. Response to this was minimal, there was a decrease in intrusiveness, improved sleep and "shaky" delusion of having a boyfriend and children. At this point, she stated she had 3 children. She remained very suggestible with variable response to being suicidal and having auditory hallucinations. She continued to laugh inappropriately and somewhat expansive. At this point, she had not commenced any medication specific for MS. Patient's mother had expressed her concern over patient being at risk of wandering away from home and suggested she be transferred to an Adult Foster Care program.

DISCUSSION:

MS is the commonest cause of neurological disability in young and middle aged adults, it is reportedly due to acute and/or chronic lesions in the CNS characterized by cellular infiltrates of T- cells/macrophages and patchy multifocal loss of myelin with axonal damage respectively [1,3,5]. CNS involvement is reportedly concentrated in the frontal lobe as seen in our patient's MRI. This is the region associated with cognitive, temperament and affective functioning. However, the periventricular region is the most common site for abnormalities in patients with MS, in contrast, lesions are more frequently located in the temporal lobes when MS is associated with a psychiatric disorder. This patient has extensive white matter disease in the temporal region too [3,5]. Clinical symptoms are vast and include but are not limited to diplopia or visual loss, weakness, ataxia, and sensory deficit as seen in our patient [1,5].

MS is often progressively disabling physically and increases vulnerability to psychiatric illnesses, about two-thirds of patients with psychopathology would require treatment as did our patient [5]. Underlying inflammation, demyelination and axonal loss are hypothesized to drive the neuropsychiatry manifestations of MS [1,3]. The prevalence of psychiatric symptoms is higher compared to the general population and in some studies, it is as high as 95% [75%-95%] of cases and doesn't appear to be related to the extent of physical disability [3,4]. Pseudobulbar affect [PBA] characterized by a disconnect between mood and affect is found in about 10% of patients with MS, they are more likely to have cognitive deficit and extensive frontal lobe affectation as seen in our patient. Some patients respond well to Nuedexta (a combination of dextromethorphan and quinidine), which was not tried in this patient because she had declined taking it [6,10]. Affective/mood disorders and cognitive manifestations are common in the course of MS [3,10,12]. Psychotic disturbances have often been described too however, rarely do they present at the onset of the illness compared to other neuropsychiatric complications of MS. Major Depressive Disorder and Bipolar disorder [prevalence of 50% and 13% respectively] are the mood disorders with increased frequency in patients with MS, affective disorders with increased frequency in these group of patients are pathological laughing and crying [PLC] and euphoria [prevalence of 25% and 10% respectively] [3,4,6,10,12]. The syndrome of euphoria is typified by a shallowness of affective response especially in patients with advanced disease and severe cognitive decline as seen in our patient. The euphoric patient has a fixed sense of well-being despite the presence of significant physical morbidity and linked with heavy lesion load with extensive frontal load involvement as seen in our patient [1,3,4]. The prevalence of psychotic disorder ranges between 2-4% [9,11], a rate greater than that in the general population and reflects a regional demyelination especially in the left temporal lobe notably in the periventricular area, two-thirds of cases of psychotic disorder in MS are women as seen in our patient [1,2,3,7]. Personality changes have been reported in patients with MS and some studies report a prevalence of up to 40% characterized by behavioral changes similar to what one would find in euphoria, however they are more extensive and diverse based on the localization of the lesion [2,3,4,8]. These changes ranges from profane, disinhibited and socially inappropriate behaviors as seen in our patient and linked to lesions in the orbitofrontal subcortical circuits, to the apathetic and indifferent clinical picture clearly evident in our patient and linked to lesions in the anterior cingulate subcortical pathways. About 45-65% of patients with MS will have cognitive dysfunction, affected domains include memory, learning, attention and information processing as seen in our patient [2,3,4]. Suicide is twice as common compared

to the general population, however, in these group of patients with pathological expression of affect, they may not experience the distress, functional impairment nor suicidal risk that characterize mood disorders [1,2,3,4,8]. This was as seen in our patient.

There may be a shared genetic predisposition to bipolar disorder and MS, however, no specific gene has been implicated.

There is a reported increase in extrapyramidal side effects to second generation antipsychotics as seen in our patient (may need to elaborate a bit on the reasons for increased vulnerability to EPSE) [2,10,11].

Day-to-day care alongside pharmacotherapy is an integral part of management of patients with neuropsychiatric manifestations of MS.

CONCLUSION:

Presence of these constellation of neuropsychiatric symptoms and poor responses to medications necessitates further studies to determine a better treatment protocol while attempt is being made to elucidate the exact pathophysiology of these neuropsychiatry disorders associated with MS. Onset of behavioral syndromes could occur way before the diagnosis of MS is made.

The authors have no conflict of interest to declare.

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