Letters to Editor

The effect of lie on the perceived therapeutic response is undeniable. Lying by the patient about improvement or deterioration in symptoms often misguides the clinician. The clinician needs to consider this factor while judging the therapeutic response, though it may not be applicable in every case.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

Sujita Kumar Kar

Department of Psychiatry, King George’s Medical University, Lucknow, Uttar Pradesh, India

Address for correspondence: Dr. Sujita Kumar Kar
Department of Psychiatry, King George’s Medical University, Lucknow, Uttar Pradesh, India.
E-mail: skkar1981@yahoo.com

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Neuropsychiatric Symptoms as Early Manifestation of Progressive Supranuclear Palsy

Sir,

Progressive supranuclear palsy (PSP) is a neurodegenerative extrapyramidal syndrome characterized by motor symptoms such as postural instability, rigidity, akinesia, supranuclear gaze deficits, and behavioral and cognitive symptoms, which usually occurs from 55 to 70 years of age.[1] The neuropsychiatric symptoms of PSP include apathy, depression, sleep disturbances, personality changes, disinhibition, and cognitive impairment.[2-4] There have been very few reports of PSP cases who presented with neuropsychiatric symptoms. Here, we report a case of a 60-year-old patient, who initially presented with depression and behavioral symptoms and was later diagnosed with PSP. The novelty of this case report is the rarity of presentation and importance of neurologist–psychiatrist team approach in the diagnosis as well as treatment of such cases.

CASE REPORT

A 60-year-old female, with no past or family history of psychiatric problems, presented to the outpatient psychiatric clinic with persistent and pervasive sadness of mood, loss of interest in daily activities, decreased sleep and appetite, easy fatigability, ruminative thoughts, anxiety symptoms, and death wishes for the past 18 months. The patient did not have a
history of any chronic medical illness, traumatic brain injury, or substance use. After a detailed history and mental state examination (MSE), she was diagnosed with major depressive disorder without psychotic features (as per International Classification of Diseases, Tenth Edition)\(^\text{[3]}\) and was started on tablet escitalopram 5 mg and tablet clonazepam 0.5 mg per day. After 1 week, the dose of escitalopram was hiked to 10 mg/day. After 1 month, the patient reported 80%–90% improvement in her depressive symptoms, and sleep pattern was restored to normal.

Nine months after the presentation of depression, she started having tremors, weakness of body, backward falls, giddiness, tightening of the body parts, and difficulty in mouth opening, slurred speech, and difficulties in swallowing. Gradually, she also developed forgetfulness and problematic inattention. Subsequently, the above symptoms progressively increased and became obvious. On repeat MSE, she was alert and oriented, and her affect was blunt. Her Mini–Mental Status Examination (MMSE) score was 18. Her neurological examination showed bilateral resting tremors, mild cogwheel rigidity, decreased arm swing, postural instability, dysphagia, dysarthria, jaw dystonia, and vertical gaze deficit with slow saccades. Her thyroid, kidney, and liver function tests as well as complete blood count and blood sugar were found to be normal. Magnetic resonance imaging (MRI) brain revealed cerebral and midbrain atrophy.

Neurologist consultation was sought for the above findings. She was diagnosed with PSP and started on tablet syndopa125 mg (a combination of levodopa 100 mg and carbidopa 25 mg) BD. Later, syndopa dose was hiked to 125 mg TDS and tablet baclofen 20 mg was added for spasticity, with continuation of escitalopram 10 mg and clonazepam 0.5 mg per day. At present, she is maintaining well. Her MMSE score improved from 18 to 22 (in the domain of immediate recall and attention). She also reported significant improvement in depression and moderate improvement in PSP symptoms.

**DISCUSSION**

In the index case, depression developed around 9 months before the onset of PSP symptoms, although we could not clearly answer whether depression was the first symptom of PSP or depression was concurrent with PSP. In literature, there have been few reports of cases with depression as an antecedent symptom of PSP\(^\text{[6,7]}\) similar to the index case. Unlike the reports which indicated apathy as the dominant behavioral change in PSP\(^\text{[1,8]}\), the main behavioral symptom, in this case, was depression. The index case initially had more psychiatric symptoms such as depression, anxiety, and sleep problems but later also developed mild cognitive problems in accordance with the available literature.\(^\text{[9]}\)

PSP is diagnosed on the basis of clinical criteria and neurological examination. Because the index patient met the mandatory criteria, she was diagnosed with probable PSP as per National Institute of Neurological Disorders and Stroke and the Society for PSP.\(^\text{[10]}\) Imaging techniques are useful only for supporting the diagnosis of PSP. MRI brain of the index case showed cerebral atrophy along with midbrain atrophy but no hummingbird sign. However, this sign may occur later during the disease evolution, so the hummingbird sign is, therefore, not always present in early presentations of PSP.\(^\text{[11]}\)

The treatment for PSP is to identify the target symptoms so that conservative management can be carried out. As the index case presented with depression, anxiety, and sleep disturbances, it was initially treated with escitalopram and clonazepam by a psychiatrist and later treated by a neurologist for the core features of PSP with combined levodopa and carbidopa therapy on which she reported significant improvement in depression and moderate improvement in core features of PSP. Available literature also reported that antidepressants help to improve mood\(^\text{[5,12]}\) and 40%–50% of patients treated with levodopa show modest improvement.\(^\text{[8]}\) The cognitive functions somewhat improved with antidepressant and without any specific treatment.

Due to clinical-pathological heterogeneity and atypical presentations, a diagnosis of PSP can be missed. Hence, psychiatrists should be aware of such clinical scenarios and should keep this differential diagnosis after a thorough evaluation of patients. The index case also shows that a neurology–psychiatry team approach would help in timely diagnosis and appropriate management.

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Nil.
Marchiafava–Bignami Disease Presenting as Acute Psychosis

Sir,

Marchiafava–Bignami disease (MBD) is a rare neurological disorder of unknown etiology, afflicting mostly middle or elderly males with a history of chronic alcohol consumption or chronic malnutrition.[1,3] There are <300 described cases in the literature and many cases go undiagnosed.[4] Its pathological hallmark is symmetrical demyelination and necrosis of the central part of the corpus callosum.[3,4] The disease has varied neuropsychiatric manifestations which include agitation, confusion, delirium, rigidity, dysarthria, mutism, split-brain syndrome, incontinence, dementia, psychotic symptoms, and gaze palsy.[6,7]

We report a rare presentation of MBD in a 32-year-old male having a history of heavy alcohol consumption who presented with persecutory delusion, auditory hallucinations, dysarthria, and urinary incontinence and whose psychotic symptoms responded to olanzapine.

Navratan Suthar, Naresh Nebhinani1, Karandeep Paul1

Department of Psychiatry, Dr. S.N. Medical College, 1Department of Psychiatry, All India Institute of Medical Science, Jodhpur, Rajasthan, India

Address for correspondence: Dr. Naresh Nebhinani
Department of Psychiatry, All India Institute of Medical Science, Jodhpur - 342 005, Rajasthan, India. E-mail: drnaresh_pgi@yahoo.com

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