A 61-Year-Old Woman with Motor and Behavioral Disturbances

J. Christopher Buckley, MD; Julie Bernstein, DO; Brytney Cobia, MD; and W. Bogan Brooks, MD

A 61-year-old woman presented to our community mental health outpatient center for evaluation after being hospitalized 2 months earlier because of a suicide attempt and homicidal ideation toward her spouse. Her diagnosis at discharge was major depressive disorder and psychotic disorder. The patient described herself as a relatively happy person with no psychiatric or non-psychiatric medical issues until the 4 years prior to her first appointment with us.

Her family medical history was significant for a sister with depression and a brother with early-onset dementia. There were no suicide attempts or death by suicide in her family members.

In the 4 years prior to her presentation, the patient noted episodes of difficulty moving her arms followed by difficulty moving her legs a few months later. She was given a tentative diagnosis of Parkinson’s disease and prescribed a combination of carbidopa and levodopa. Initially she improved, but her motor symptoms soon returned. Her medications were adjusted over the years, but were only partially effective. She later developed episodic stiffness in her feet, poor vision, and loss of coordination, resulting in frequent falls. In addition to her motor symptoms, the patient demonstrated emotional lability with uncontrollable crying spells several times per day accompanied by disarticulated speech that worsened despite speech therapy.

Psychotic symptoms began to emerge in the 4 years after the initial motor symptoms, manifested by thoughts that her spouse was cheating on her despite evidence to the contrary. These delusions of infidelity culminated in the patient pointing a gun at her spouse and twice attempting suicide by overdose. Ingestion of 50 1-mg clonazepam tablets in her most recent suicide attempt led to intubation in the intensive care unit of a local hospital for several days before her condition improved enough for her to be discharged to a freestanding psychiatric inpatient facility.

By the time of her outpatient assessment, the patient denied thoughts of suicide or homicide, hallucinations, symptoms of depression or mania, and no longer voiced delusional thoughts. She reported adequate sleep and appetite with 50 mg of quetiapine at bedtime. The patient also was taking 50 mg/day of desvenlafaxine, which she reported was less effective than sertraline for treatment of her depressive symptoms. Her regimen of 25 mg of carbidopa and 100 mg of levodopa 4 times daily no longer helped with her parkinsonian symptoms. The patient restarted 1 mg of clonazepam every night to decrease episodes of irritability in the evenings and yelling in her sleep.

The neurologist reported that the patient exhibited a blunted affect, mild psychomotor retardation,
More than a makeover.
See what’s new!

Explore the redesigned Healio – now faster than ever with exciting enhancements to improve usability and your overall experience. You’ll find a clean new look, easier navigation, plus more personalized content and expanded features. Driven by user data and feedback, the all-new Healio is designed around you.

The online home of PSYCHIATRIC ANNALS
stooled posture, shuffling gait, upper extremity rigidity, and disarticulated, interrupted speech lacking in fluency. Although judgment had been impulsive per her history, it was appropriate during the outpatient assessment. Thought process and content, cognitive status, and mood were also unremarkable at the outpatient assessment.

Given that the patient had vision and ambulation problems as well at least four nonpsychiatric hospitalizations for falls, her outpatient neurologist diagnosed her with progressive supranuclear palsy (PSP).

**DISCUSSION**

PSP is a rare neurologic disorder, colloquially referred to as a “Parkinson-plus” syndrome by the medical community. Age at symptom onset is typically in the mid-50s to early 60s, and initial presentation is usually characterized by bradykinesia, rigidity, and gait and postural instability resulting in frequent falls. The disease progression is insidious, and patients gradually develop supranuclear ophthalmoplegia, resulting in impairment of vertical gaze. The disease is also marked by a labile affect and inappropriate emotional outbursts, with seamless transitions from laughter to tears.

The pathophysiology is thought to be due to the accumulation of neurofibrillary tangles comprised of tau protein within the basal ganglia, subthalamic nuclei, superior colliculus, and the frontal lobe. Deposition of tau protein in different neuroanatomic locations accounts for the variability in both neurologic and psychiatric manifestations across the spectrum of the disease.

The etiology is unclear, but is hypothesized as viral or toxin in origin. A definitive diagnosis can only be reached through histologic examination, so the diagnosis of probable PSP is generally a clinical one and requires vertical supranuclear gaze palsy, prominent postural instability, and no evidence of another disease that could explain these features. Hypothalamic dysfunction is a potential cause of PSP and differentiates it from Parkinson's disease. Treatment for PSP is through dopamine agonists. In this case, the patient was given carbidopa and levodopa, with an initial improvement in her symptoms for several years. Although passive death wishes and suicidal actions are exceedingly rare, this patient was also notable for endorsing Parkinson's disease, a common misdiagnosis due to the similarity of presenting symptoms. Although PSP eventually reveals characteristic features that distinguish it from Parkinson's disease, another way to differentiate between the two diseases is by evaluating a patient's response to dopamine analogs. In this case, the patient was given carbidopa and levodopa, with an initial improvement in her symptoms for several years before she noticed a gradual decline in the effectiveness of the medication, which is a typical response curve in a PSP patient.  

Psychiatric manifestations of PSP have been well-documented since the 1964 article by Steele et al. that first identified the disease. In their modest but pioneering study of nine patients, Steele et al. recorded six of the patients as having personality changes in the form of irritability, suspiciousness, depression, and apathy. Our patient exhibited both typical and atypical psychiatric manifestations of PSP as documented in the literature. The most cited psychiatric features in recent years are apathy and disinhibition, with psychosis and depression being relatively rare findings. Although neither she nor her family reported that the patient exhibited apathy, the patient clearly demonstrated symptoms of disinhibition as evidenced by non-bizarre delusions of infidelity that were quickly followed by impulsive homicidal and suicidal actions.

Although passive death wishes have been reported in a small number of cases, active suicide attempts are exceedingly rare. This patient was also notable for endorsing...
symptoms of depression. Although it has not historically been a common finding, a 2013 cohort found that depression may occur in up to 60% of patients with PSP.

Although not well-documented and considered rare, findings of impulsivity and/or disinhibition in one-third of PSP patients may lend credence to the possibility that homicidal actions or suicide attempts in PSP patients with active psychosis are more common than previously thought. More research is warranted in the area of homicidal and suicidal behavior in patients who have PSP and who exhibit psychotic symptoms.

Although 50 years have passed since PSP was first identified as a neurologic disorder, the treatment options are still limited to symptomatic and supportive care. Even with effects that are short lived, the combination of carbidopa and levodopa remains an acceptable option for alleviation of symptoms, as evidenced in this patient. Selective serotonin reuptake inhibitors and tricyclic antidepressants are generally effective in treating symptoms of depression. Antipsychotics can be effective in treating psychotic symptoms associated with PSP, and those least likely to induce extrapyramidal symptoms, such as quetiapine, should be considered for this patient population.

Our patient also suffered from chronic insomnia, a common problem in PSP patients, and she was treated with clonazepam for sleep. Although clonazepam can be used successfully as a sleep aid, any benzodiazepine use increases the risk of dependence, depression, memory problems, disinhibition, and falls. In a patient like the one discussed here, with a history of dangerous impulsivity, trazodone might be a better soporific choice.

CONCLUSION
This case highlights how the psychiatric manifestations of PSP nearly caused a middle-aged woman to commit suicide and to fatally harm her husband. Like many patients with PSP, her course was plagued by misdiagnosis in the medical, neurologic, and psychiatric setting. She underwent several years of inpatient and outpatient evaluation before the correct diagnosis was reached. Despite having an understanding of the poor prognosis of this progressive disease, the patient was comforted by finally having an explanation for her symptoms and she remained hopeful for a cure. Literature searches yield few matches with regard to studies of PSP and the risk of suicide or homicide secondary to increased impulsivity and psychosis, indicating that more research into this area is warranted.

REFERENCES