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Case report

Diversified psychiatric presentation in a case of progressive supranuclear palsy

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ABSTRACT

Progressive supranuclear palsy (PSP) is an unusual neurodegenerative disorder with variant clinical phenotypes. Accurate diagnosis is challenging in the early stage, especially in psychiatric clinics, where misdiagnoses with psychiatric illness are common. A case of PSP was difficult to differentiate from other Parkinsonian syndromes initially, and the patient's affective symptoms predated the onset of other symptoms. Gaze abnormality and frontal lobe syndromes emerged and she was diagnosed with PSP 5 months after the first psychiatric visit. Heightened awareness of PSP and its diagnosis are important, not only because of prognostic implications, but also because of appropriate interventions and focused therapeutic targets.

1. Introduction

Progressive supranuclear palsy (PSP) is an uncommon, but not rare neurodegenerative disease. Epidemiological studies reported PSP as a frequent cause of atypical parkinsonism with an incidence rate of 5.3/100,000 (in the population aged > 50 years),[1] 5.8/100,000 in Japan,[2] and an age-adjusted prevalence of 6.4/100,000.[3] On review of the literature, PSP is clinically characterized by supranuclear ophthalmoplegia, gait disturbance, postural instability with recurrent falls, rigidity, and frontal cognitive impairment. Pathological characteristics include neuronal loss and neurofibrillary tangles, and gliosis are principally in the basal ganglia, cerebellum, brainstem, and to a lesser extent, the cerebral cortex. It is a distinctive disorder related to tau pathology.

PSP is progressive despite any therapy nowadays. The natural courses that lead to death are usually within 6–12 years of diagnosis.[4] Another investigation published that the median survival from time of disease onset is 5.6 years.[5] In late stage PSP, patients are immobile due to severe akinesia, rigidity, and dystonia, and have difficulty in swallowing with a high risk of aspiration.[6] Therefore the most common causes of death are aspiration pneumonia, primary neurogenic respiratory failure, or pulmonary emboli.[4,5,7]

PSP is not only a movement disorder, but also a disorder with a wide range of neuropsychiatric symptoms.[8] We report the longitudinal course of a patient with PSP, whose psychiatric symptoms were diversified in different periods of the course, and how we distinguish PSP from other movement disorders.

2. Case Report

A 71-year-old married female patient without personal or family history of psychiatric illness presented with abruptly free-floating anxiety, restlessness, irritability, hypervigilance, forgetfulness, insomnia, and multiple somatic discomforts (fatigue, soreness all over, dry mouth, tension headache, back pain, low abdominal fullness) for 2 months. The patient did not have any systemic disease, and she also denied any history of traumatic brain injury or substance use. Although she lost some money in the stock market, her most recent life event occurred over 1 year ago. The event itself did not seem to be related to the prominent symptoms of her current anxiety. The patient was filled with catastrophic thinking and anxiety that something was wrong with her body. She had visited neurology and psychiatric clinics. Neurological examination and blood tests were performed, and

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both physiological and biochemical states were within the normal range. A neurologist and psychiatrist prescribed zolpidem (China Chemical & Pharmaceutical Co., Ltd., Hsinchu County, Taiwan) 5 mg, and escitalopram (Genovate Biotechnology Ltd., Hsinchu County, Taiwan) 10 mg with clonazepam (F. Hoffmann-La Roche, Ltd., Kaiseraugst, Switzerland) 0.5 mg, respectively. Because the patient could not tolerate the side effects of serotonin reuptake inhibitors (SSRI), she only took hypnotics and tripled the dosage by herself. After suffering from the foregoing symptoms, the patient's depressed mood with anhedonia, useless feeling, and slow movement became obvious. For a second opinion, she visited our psychiatric clinic in the latter part of 2nd month of psychiatric disturbance.

The patient was treated with paroxetine controlled-release tablets (Paxil CR; GlaxoSmithKline Inc. Ontario, Canada) 25 mg/d and hypnotics as needed. We started to notice that patient walked with small steps and had some difficulty in rising from her chair during the 2nd visit. Her anxiety and depressed mood were dramatically alleviated in the coming month. However, fluctuating cognition, illusion, transient visual hallucination, and significant akinesia and bradykinesia simultaneously showed up in the 3rd month of illness. Neuroleptic sensitivity and atypical features of "functional depression" might imply that some organic problems were influencing the central nervous system. We advised the patient to complete serial examinations. She was admitted briefly to the neurology ward at another hospital for convenience. Electroencephalography and brain magnetic resonance imaging revealed negative findings. She was diagnosed with extrapyramidal symptoms and was discharged from hospital with the discontinuation of SSRI therapy.

Movement problems were only improved for a while. Affective symptoms subsided spontaneously. Nevertheless, fluctuating cognition persisted and parkinsonism progressed again with remarkable axial rigidity. The patient started to display postural instability, unexplained falls backward, resting tremor over right side limbs, vivid visual hallucinations, and deterioration of executive function in the 4th month of illness. She could not cook or knit as well as usual and sometimes saw a person set the room or bed on fire. After discussing with the patient and family, she took low dose quetiapine (AstraZeneca UK Ltd., Luton, UK) (<25 mg) as needed. We initially referred the patient to our neurology ward under the working diagnoses of dementia with Lewy bodies (DLB). Due to rapid progression of Parkinsonian syndrome, Parkinson's disease (PD) was less likely.

Laboratory analyses including differential blood counts, electrolytes, hepatic enzymes, creatinine, urea nitrogen, creatine kinase, thyroid hormones, cortisol, urine analysis, and cerebrospinal fluid tests, were all within normal limits, as were plain films of the chest and electrocardiogram. Creutzfeldt–Jakob disease and viral/bacterial encephalitis could be excluded primarily. Unexpectedly, neurological examination revealed that the patient had difficulty in looking up. Computed tomography (CT) of the brain revealed the initial change of brainstem atrophy related to PSP,[9,10] in which midbrain atrophy was prominent with a relatively preserved pons (Figures 1 and 2). The 18-Fluoro-deoxyglucose positron emission tomography (F-18 FDG PET-CT) brain scan showed multiple metabolic/perfusion lesions over the cerebral cortex, including typical PSP lesions at midline frontal regions (Figure 3).[11] Dopamine transporter scan by single photon emission CT examination was performed with the ligand Tc-99m TRODAT-1. Results showed moderate decreased uptake of radioactivity involving the bilateral striatum. The images showed predominant loss of radioactivity in the putamen, especially in the left side. Under the impression of presynaptic dopaminergic lesions with rapid onset course, multiple system atrophy (MSA) and PSP should be considered. On the basis of these findings and clinical presentation, PSP was confirmed 5 months after the emergence of first psychiatric symptoms.

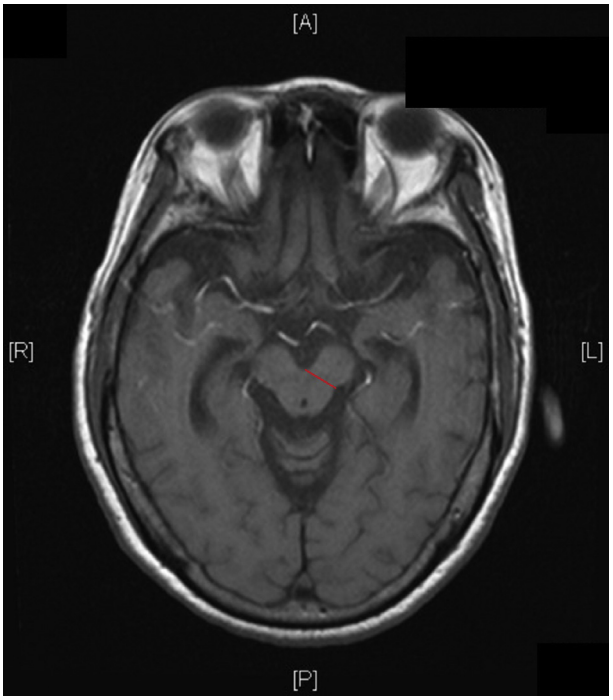

Figure 1. Mickey mouse sign. Patient's axial midbrain diameter at superior colliculus level reduced. Interpeduncular fossa to intercollicular groove (red line) measures 11.7 mm.[9]

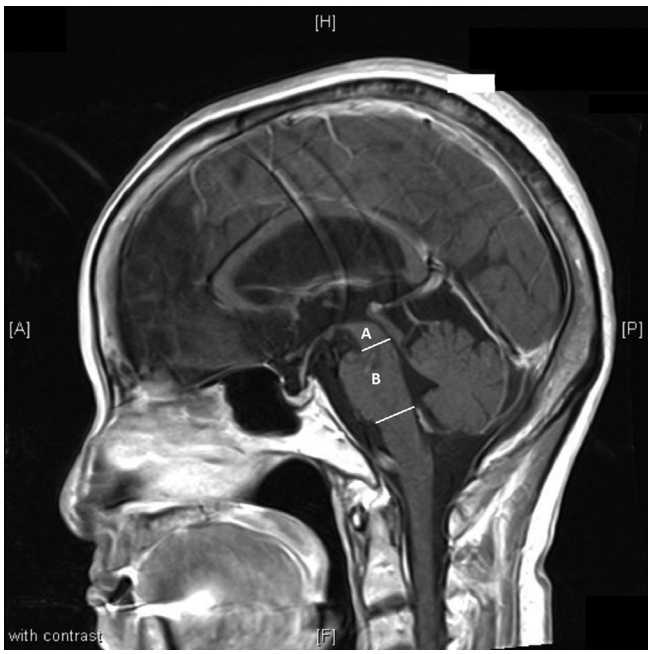

Figure 2. Initial change related to progressive supranuclear palsy was shown in the brain computed tomography. Midbrain (A) to pons area (B) ratio reduced on the midline sagittal plane from 0.147 to approximately 0.12.[10]

A neurologist prescribed Sinemet (carbidopa 25 mg–levodopa 100 mg; Mylan Pharmaceuticals Inc., West Virginia, America) four pills per day, Madopar HBS (levodopa 100 mg–benserazide 25 mg;

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