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CASE REPORT

Delirium Unmasking A Case Of Progressive Supranuclear Palsy- A Case Report.

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1 | INTRODUCTION

Progressive supranuclear palsy (PSP) is a type of neurodegenerative extrapyramidal syndrome. The prevalence of PSP in studies is found to be 5.8-6.5 per 100,000 individual with a slightly male preponderence. (1) Onset is usually between 55 and 70 years of age and median survival is around 6 years after the onset of symptomatology. (2) It is characterized by motor symptoms, such as postural instability, rigidity, akinesia, supranuclear gaze palsy, pseudobulbar palsy; levodopa-unresponsive parkinsonism, and behavioral and cognitive symptoms. Early PSP is also difficult to differentiate from PD; as it shares many features like abnormalities in gait, speech, and eye movements. Neuropsychiatric symptoms commonly observed in patients with PSP are dementia, apathy, depression, anxiety, disorders of sleep and disinhibition. (3) Approximately 20% of patients initially present with cognitive dysfunction and behavioral changes in the first 2 years which often leads to misdiagnosis. (4) The aim of this paper is to highlight a case of previously diagnosed Idiopathic Parkinson's Disease who presented with delirium and on further evaluation the diagnosis was reviewed to Progressive Supranuclear Palsy.

2 | CASE DESCRIPTION

A 69 year old man presented with impaired memory, disturbed sleep and restless behavior for 2months in geriatric mental health outpatient department. He had a history of tremors on both the hands and stiffness of body which was gradually progressive for last 4 years. They consulted a neurologist 1.5 years back as his symptoms were severe enough to hamper in day to day functioning. He was diagnosed as a case of idiopathic Parkinson's Disease and was prescribed Tab Levodopa 100mg and Carbidopa 25mg combination preparation thrice daily. On medication within 2months, family members reported decrease in his symptoms. For last 6months new symptoms

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like frequent falls during walking without any precipitant developed. Falls usually occurred within a few steps of walking mostly towards the left side and following which he sustained an injury over his left eye leading to lower lid ectropion. The activity of the patient became restricted and he started to remain confined to his bed for the fear of falls since last 3months. They consulted a physician again and he was prescribed Tab Trihexyphenydyl 2mg three times a day and dose of Tab Levodopa 100mg and Carbidopa 25mg combination increased to four times a day and Tab Levodopa 100mg and Carbidopa 25mg combination contolled relase preparation was added at bedtime. After taking those medications the frequency of falls increased, forgetfulness appeared, sleep got disturbed, at night he becomes restless and some confusion in behavior like pointing things, picking bed linen were noticed during evening hours.

On examination the patient had prominent vertical wrinkling of forehead, wide opened eyes with reduced blinking, 'procerus sign' was present. His speech revealed hypophonia and reduced fluency. On the first two days his orientation was fluctuating; from the third day no abnormality was noticed in his mental state. Neurological examination revealed wide based festinating gait, intention tremor in upper limb, lead pipe rigidity in all four limbs bilaterally(right>left), axial rigidity and impaired balance and coordination; and supranuclear gaze palsy.

During the hospital stay the anticholinergic drug (trihexyphenydyl 2mg) and Tab Levodopa 100mg and Carbidopa 25mg combination contolled relase preparation was withheld; Tab Melatonin 5mg was added along with other drugs were continued. Within 2days of hospitalization, his sleep improved, restlessness and confusion in behavior decreased, also his cognition was improved.

On assessing the overall cognition, HMSE score was found to be 25, impairment was seen in the component of attention and visuospatial ability. On neuropsychiatric evaluation, NPI was applied and score was 36, impairment was present in domains of sleep disturbances, anxiety, dysphoria/depression, agitation/aggression, apathy, irritability/lability. For assessing depression HAM –D was applied and the score was 7; suggesting few depressive symptoms not amounting to diagnosis of depressive disorder.

Routine blood and urine examination revealed no abnormality. On neuroimaging MRI brain with MRA revealed atrophy of superior and lateral aspect of midbrain, chronic infarct in right basal ganglia region and diffuse cerebral and cerebellar atrophy. Typical 'hummingbird sign' and 'morning glory sign' was present in sagittal and axial section of MRI brain respectively.

From the above mentioned history of gradually progressive postural instability, examination and neuroimaging findings a diagnosis of Progressive Supranuclear Palsy with Mild cognitive impairment was established.

3 | DISCUSSION

Progressive supranuclear palsy (PSP) is often difficult to discriminate clinically from idiopathic Parkinson's disease (PD). Diagnosis in early stage is important for maximal benefit from disease modifying treatment for PSP. Delirium is defined as an acute organic brain syndrome which presents with decreased level of consciousness, varied psychomotor activity, wake-sleep rhythm disorders, cognitive impairment and attention deficits. High dose of Levodopa in Parkinson's disease is reported to cause confusion and psychosis. (5) Moreover dopaminergic drug in Progressive Supranuclear Palsy has response in only 20-40% cases. (2) Anticholinergic drugs in ageing brain with comorbidities are known to cause confusion and delirium. (6)

The patient mentioned were on high doses of dopaminergic and anticholinergic drugs which might be the cause of delirium. Deficits in the neurotransmitters like dopamine and acetylcholine, and systemic inflammation have also been proposed in the pathophysiology of delirium in PD which can also be an explanation for Progressive Supranuclear Palsy. (5)

In the first two years Progressive Supranuclear Palsy are characterized by asymmetric

onset, predominant bradykinesia or tremor and a moderate initial therapeutic response to

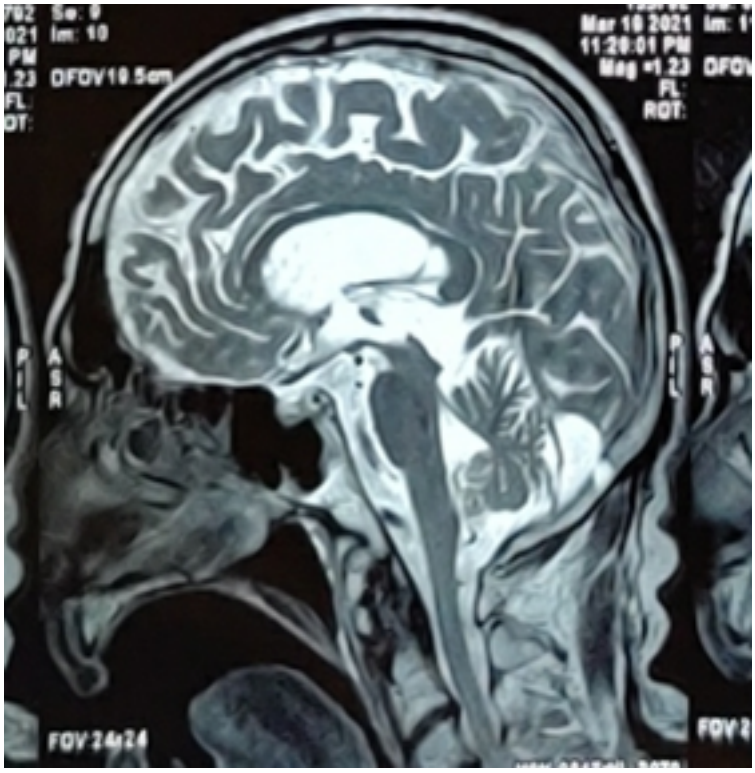

FIGURE 1: Axial T2 weighted MRI image of brainshowing distinct `morning glory' signAxial T2 weighted MRI image of brainshowing distinct `morning glory' sign

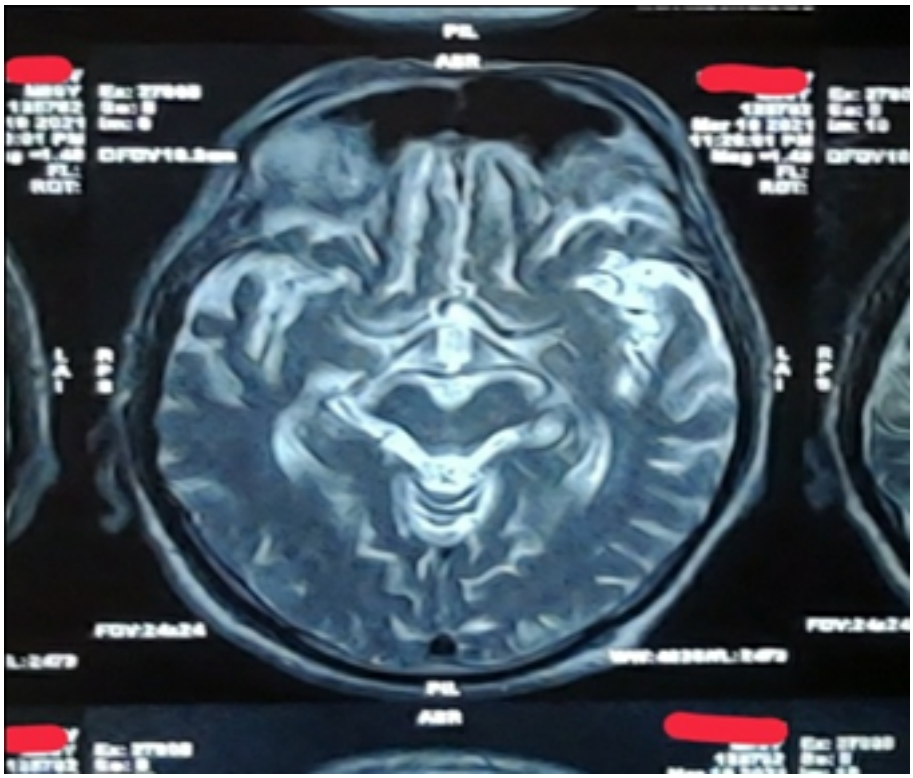

FIGURE 2: Mid-sagittal T2 weighted MRI image of brainshowing distinct `humming bird' sign

levodopa which is indistinguishable from Idiopathic Parkinson's Disease. The symptoms which might help in differentiation are early onset of postural instability and falls, vertical supranuclear gaze palsy and frontal behavioural dysfunction which was evident in our case after resolution of delirium. (7) So, meticulous examination, frequent follow up of a case with Parkinsonism, evidence of any change of symptomatology will help in diagnosis of Progressive Supranuclear Palsy. Early diagnosis might prevent inadvertent medication use and its side effects and direct the treatment course towards disease modifying agent for treatment of Progressive supranuclear Palsy.

4 | CONCLUSIONS

With regard to this patient the cause of delirium in Progressive Supranuclear Palsy treated as Idiopathic Parkinson's Disease with high dose of dopaminergic medication and anticholinergics is an important point to be highlighted. Another contribution from this report is the importance of recognition of change or appearance of new symptomatology in the diagnosis Progressive Supranuclear Palsy.

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