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Parkinson's Disease A Case Report

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ABSTRACT

A 53 years old female patient is a known case of Parkinson's disease was admitted in neurology department, KIMS, Seunderabad with complaints of deviation of mouth to the right side. It was transient only for a few minutes associated with paresthesia of mouth. No history of paresthesia/weakness in limbs. She has a past history of Parkinson's disease, hypothyroidism, CHD and S/P closure of ASD. On examination patient was conscious and obeying commands. Investigations (MRI Brain, 2D Echo, Carotid doppler) were done. During hospital stay she was treated with dopamine agonists, lipid lowering agents, dual antiplatelets, multivitamins, and other supportive medications. Patient developed new onset of macular rash over hands, stopped ecosprin and reviewed in dermal OPD. Patient is stable, there was no further episode of deviation of mouth and is being discharged. This case report discusses about the PD, it's history, epidemiology, causes, symptoms, pathophysiology, appropriate investigations, it's management and preventive measures.

KEYWORDS

PD, dopamine, MPTP, SNc neurons, Lew pathology.

INTRODUCTION

Parkinson's disease (PD) is a slow-progressive neurological disorder affecting the extrapyramidal motor system. Dopamine neurons in the substantia nigra are largely damaged, and degeneration of these neurons disrupts the ability to initiate movement in the muscles. The clinical condition consists of four cardinal features:

1. Bradykinesia
2. Muscular rigidity
3. Resting tremor
4. Postural imbalance

HISTORY

In AD 175, the physician Galen referred to it as "shaking palsy" in western medical literature. But it wasn't until 1817 that London physician James Parkinson published a thorough medical study. "An essay on the Shaking Palsy" was the title of the publication. The chemical alterations in Parkinson's disease patients' brains were discovered in the 1960s. In the substantia nigra, a region of the brain, low dopamine levels lead to the degeneration of nerve cells. Levodopa was initially used to treat the symptoms in the 1960s and has since emerged as the "gold standard" in medicine.

EPIDEMIOLOGY

The age of onset of PD varies, ranging from 50 to 80 years, with a mean of 55 years. The prevalence of PD is 100 cases per 100,000 people. The incidence is 20 cases per 100,000 persons each year. In India, more than one million cases are reported each year. Men are impacted slightly more often than women. Despite the availability of great symptomatic therapies that enhance both quality of life and life expectancy, there is no cure for the disease, which can lead to increased morbidity and death.

ETIOLOGY

1. Idiopathic PD (80%)
2. Secondary PD (20%)

Most cases of PD are of unknown cause, and referred as idiopathic parkinsonism.

- Environmental factors: chronic exposure to pesticides, carbon monoxide, methanol, hydrogen sulfide, petrochemicals & heavy metals like manganese, copper, iron, rural living, drinking well water, xenobiotics, herbicides.
- Neurotoxins: MPTP (1-methyl-4-phenyl-1,2,3,6-tetrapyridinine). In human's administration of this compounds results in irreversible PD
- Genetics: It plays an important role, particularly if PD develops before age 50.

Autosomal dominant forms: Mutations of PARK1, LRRK2 genes.
Autosomal recessive forms: Mutations of parkin, PINK1 genes.

- Oxidative stress: Mitochondrial dysfunction and autooxidation of dopamine
- Rare causes: Drugs (Antiemetics, antipsychotics), Wilsons disease, Trauma, Viral inflammation.

PATHOPHYSIOLOGY

The hallmark histopathological features of PD are:

1. Depigmentation of dopamine producing neurons (loss of SNc neurons)
2. Presence of Lewy bodies in the remaining SNc neurons.

Lew pathology has been proposed to predict anatomical distribution within the parkinsonian brain.

- Pre-clinical stages: Lewy bodies are found in medulla oblongata, locus coeruleus, raphe nuclei, olfactory bulb. This correlates with anxiety, depression and impaired olfaction.
- Clinical stages: Ascends to midbrain and develops motor features
- Advanced stages: Spreads to cortex, correlates with behavioral and cognitive changes.

Decrease of neurotransmitters in basal ganglia correlates with non-motor symptoms.

SYMPTOMS

The symptoms of PD progress, and most PD patients become completely immobile within 10 to 20 years. Patients with PD exhibit both motor and nonmotor symptoms. Non-motor symptoms may precede major complaints.
Motor Symptoms:

- Rigidity
- Tremor at rest

- Akinesia or bradykinesia
- Postural instability
- Gate abnormalities

Non-motor Symptoms:

- Sleep disturbances (insomnia, rapid eye movement sleep behavioral disorder, restless leg syndrome)
- Psychological symptoms (anxiety, psychosis, depression, cognitive impairment)
- Autonomic symptoms (constipation, drooling, sweating, dysphagia, urinary problems, sexual dysfunction, orthostatic hypotension)
- Other symptoms (fatigue, nausea, pain, problems with speech, vision, seborrhea).

CASE REPORT

A 53 years old female patient was admitted in the neurology department of KIMS (KRISHNA INSTITUTE OF MEDICAL SCIENCES)-Secunderabad, with her chief complaints of deviation of mouth to the right side. Patient is a known case of PD was apparently asymptomatic, then she had deviation of mouth to the right side. It was transient only for a few minutes associated with paresthesia of mouth. No history of paresthesia or weakness of limbs. She has a past history of PD, Hypothyroidism, CHD, S/P closure of ASD (2006). No family history of PD, and no known allergies. Her appetite, sleep, bowel and bladder habits are in normal condition. On examination patient was conscious and obeying commands. Her vitals were found to be PR – 80/min, B.P – 140/80 mm hg, SPO2 – 96% at room air, RR – 16/min, and Temp – 98.6 F. Her laboratory investigation showed slightly increase in RBC count (4.80 million/cumm). MRI brain shows multiple patchy and punctate foci of T2/FLAIR hyperintensity involving periventricular and deep white matter of both frontal and parietal lobes. 2D Echo shows post ASD surgical closure, no RWMA with no PE/clots. Carotid doppler shows no evidence as stenosis or plaques. During hospital stay she was treated with dopamine agonist, lipid lowering agents, dual antiplatelets, multivitamins and other supportive medications. Patient developed new onset macular rash over hands, stopped ecosprin and advised for dermal review in OPD. Patient was stable, there was no further episode of deviation of mouth. Hence, discharged in hemodynamically stable condition with following medications:

- Tab PAN 40 mg once daily at 7am before food
- Tab ATARAX 10 mg at night, for 3 days
- Tab OPTINEURON once daily at 2pm
- Tab ATORVA 80 mg at bedtime
- Tab SYNDOPA CR at bed time
- Tab THYRONORM 50 mg once daily before food
- Tab SYNDOPA PLUS four times a day before food
- Tab CLOPITAB 75 mg once daily at 2pm
- Tab PACITANE 2 mg twice daily at 8am,8pm
- Cap AMNATROL 100 mg thrice daily at 8am,2pm,8pm
- Tab SIBELIUM 5mg once daily at 9pm

DERMAT review for macular rashes and review after 2 weeks/SOS in neurology OPD.

DISCUSSION

The elderly female patient is a known case of PD. No history of paresthesia / weakness in limbs. She also has history of hypothyroidism, CHD and S/P closure of ASD (2006). She has been admitted with the complaints of deviation of mouth to the right side. Laboratory investigations were done and treated with the medications. PREVENTIVE MEASURES – The patient and attendants have been explained about the nature of the disease, causes, and future Course of illness. Also explained about the lifestyle modifications like:

- Ensuring proper diet, physical health, and social relationships.
- Avoid drugs that inhibit central dopamine.
- A multidisciplinary approach involving dietitians, speech therapists, physical therapists, occupational therapists, and social workers can improve care.
- Consistently see ophthalmologists and dentists
- Get adequate sleep and manage stress

- Since these lifestyle changes may enhance ADL, gait, balance, and mental health, they must to be initiated early and maintained throughout treatment.

The patient will need proper follow up with the neurologist, has to have a regular usage of medications and intermittent investigations to see the status of the disease control.

CONCLUSION

Thus, the main motive of this written report is to create awareness to the patient and the people around about the Parkinson's disease. The treatment of PD mainly focuses on to improve patient independence, ADL, and quality of life by treating symptoms, decreasing the development of response fluctuations, and limiting medication-related adverse effects. This can be accomplished via a combination of drugs, treatments, and surgical procedures.

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