Parkinson's Disease in a Chronic Alcoholic: A Case Report

Abstract:

A Parkinson's disease (PD) is a neurological condition that worsens over timeand is mostly defined by motor symptoms such stiffness, bradykinesia,tremors, and unstable posture. The pathology of PD is predominantly associated with the loss of dopaminergic neurons in the substantia nigra pars compacta and the presence of Lewy bodies, which are intracellular inclusions containing alpha-synuclein protein. The quality of life is greatly impacted by non-motor symptoms, which frequently appear before motor manifestations. These symptoms are autonomic dysfunction, depression, sleep difficulties, and cognitive impairment. The current treatments are symptomatic, with the goal of employing drugs such levodopa, dopamine agonists, and MAO-B inhibitors to mimic or replenish dopamine. Research on alpha-synuclein aggregation, neuroinflammation, and mitochondrial dysfunction is underway in an effort to identify disease-modifying therapies. We present a case of a 50-year-old male witha history of chronic alcoholism and smoking, who presented with shiveringof hands and legs, along with general weakness and sleep disturbances.Understanding the underlying mechanisms, looking into novel therapytargets,and enhancing early detection techniques are the main goals ofemerging research. An overview of Parkinson's disease (PD) is given in thisabstract, with a focus on the need for ongoing research to better comprehendand treat this complex neurological disorder. This case report offersinteresting details about the difficultiesin identifying and treatingParkinson's disease in individuals with a history of long-term alcohol abuse.It also emphasizes how crucial a multidisciplinary strategy is to providingpatients with the best care possible. For PD to be fully managed, a multidisciplinary strategy is needed to address both motor and non-motor symptoms. This case report aiming to improve patient outcomes and quality of life.

- **Keywords**: Parkinson's disease, chronic alcoholism, resting tremors,bradykinesia, rigidity, monoamine oxidase.

Introduction:

A neurodegenerative condition that progresses over time and always involves gastrointestinal problems is Parkinson's disease (PD). Gastrointestinal symptoms, like as constipation, are frequently seen in PD patients before the first manifestation of the disease 1.Parkinson's disease is characterized by slowness, stiffness, trembling, and poor movement. Physical symptoms of the disorder include tremor when at rest, rigidity during passive movement, bradykinesia (slow movement), and hypokinesia (poor movement). Parkinson's disease is a degenerative neurological illness resulting from nerve cell loss in the substantia nigra, the area of the brain responsible for controlling movement. These nerve cells stop functioning or degenerate, losing their capacity to make the crucial neurotransmitter dopamine.As the illness worsens, these initially unilateral traits turn bilateral. Later on, dementia, orthostatic hypotension, and postural instability and accidents may occur 2.

Parkinson's disease is thought to be largely caused by the degeneration of dopaminergic neurons in the brain. Dopaminergic neurons are also involved in pacemaking. Given that they are consistently rhythmically discharging, this suggests that they require a significant amount of energy to recharge. Neurons degenerate when their energy supply is out. By reducing their energy requirements, research is currently being done to protect these neurons and prevent Parkinson's disease from progressing3. Numerous extranigral regions are affected by the neuropathology that underlies the neurodegeneration in Parkinson's disease (PD), which jeopardizes the dopaminergic, serotonergic, adrenergic, and cholinergic

neurotransmission systems. People with Parkinson's disease (PD) who have a variety of non-motor symptoms (NMS) are closely associated with dysregulation and neurodegeneration of associated neurons, owing to the importance of such systems in modulating functions such as cognitive function, emotional processing, circadian rhythm, and reward 4.

DA neurons are traditionally divided into three subtypes. Three subgroups of DA neurons are traditionally recognized. A8 neurons are found in the retrorubral field, A10 neurons in the ventral tegmental area, and A9 neurons in the substantia nigra pars compacta (SNpc). They demonstrate basic disparities in vulnerability to cell death in Parkinson's disease (PD), as well as varied susceptibility to disease processes and different roles and axonal innervations.While A10 neurons are mostly untouched by Parkinson's disease (PD) and their malfunction is linked to neuropsychiatric diseases, the A9 DA neuron subtype, which regulates motor function, is mostly degraded in the disease. As of right now, topographical characteristics—such as anatomical placement in the midbrain and projection targets in the forebrain—are the only ways to properly classify DA neurons 5.

One of the main pathogenic features of Parkinson's disease (PD) is the loss of dopaminergic (DA) neurons in the substantia nigra (SN). Movement, learning, reward, and addiction are all regulated by DA neurons. In Parkinson's disease (PD), the loss of DA neurons and other cell types results in motor symptoms as well as potential psychological problems. Though their susceptibility in Parkinson's disease (PD) has been explained by a number of theories, including dopamine toxicity, iron overload, autonomous pace-making, and axonal arborization, the molecular mechanisms behind the death of DA neurons in the SN are still poorly understood. To comprehend the underlying causes of DA neuron degeneration, however, a thorough molecular and histological dissection of human DA neurons is required.Neuromelanin (NM), or dark pigment, is found in human DA neurons and is produced by the body from l-3,4-dihydroxyphenylalanine, a precursor to DA. Its concentration rises with age. It has long been known that PD is associated with the loss of melanin-containing DA neurons in the SN 6.

this is because the brain's nerve cells are gradually degenerating, which lowers dopamine levels. One substance that facilitates message transmission between cells is dopamine. With the exception of emergencies, the majority of Parkinson's patients are elderly and are less inclined to choose dental care. Xerostomia is a side effect of parkinsonism medication. Anxiety increases the Parkinson's symptoms. It is important that patient should remain calm during dental treatment. It is essential to make the environment stress-free and as serene as possible. Here is one of such cases of a partiallyedentulous Parkinson's patient successfully treated with an immediate7.The secondary, avoidable consequences of immobility coexist with the primary impairments linked to Parkinson's disease. For those in the early or intermediate phases of the condition, a community-based fitness program may help them preserve function and raise their level of activity 8.

The presence of the cardinal motor signs—tremor, rigidity, bradykinesia, andpostural instability—has historically been used to diagnose Parkinson's disease (PD). But as the disease progresses, a growing amount of data suggests that nonmotor symptoms such autonomic insufficiency, cognitive decline, melancholy, olfactory deficiencies, psychosis, and sleep disturbance are frequently encountered. Motor dysfunction is believed to result from a progressive loss of dopamine cells in the substantia nigra pars compacta, which is manifested at a loss of about 50% of nigral neurons and 80% of striatal dopamine.Deep brain stimulation, amantadine, monoamine oxidase (MAO) inhibitors, anticholingergics, and dopamine agonists are some other therapy choices.5. The objective of neuroprotective therapy, which stops or delays neurodegeneration in Parkinson's disease, has not yet been achieved 9.

It Is increasingly evident that there are multiple diseases that share clinical, pathological, and maybe biochemical end points, rather than just one Parkinson's disease. The only environmental factor known to date that can cause parkinsonism (and to do so within 14 days of exposure) is the neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP). However, other environmental factors, such as the use of pesticides and herbicides, have been linked to an increased risk of disease10.The eye movement dysfunction typical of progressive supranuclear palsy, as well as signs and symptoms of autonomic failure and cerebellar impairment typical of multiple system atrophy, may not be noticeable in the early stages of these illnesses. While not very specific or commonly available, PET imaging with '8F-dopa and other ligands can distinguish classic

Parkinson's disease from other Parkinson disorders in about 80% of patients 11.For Parkinson's disease patients who are not responding to medication, deep brain stimulation, or DBS, is a recognized therapy option. Due to the therapy's effectiveness, more patients are now getting DBS implants. It is crucial that medical professionals treating patients with implanted neurostimulators understand the recommended course of treatment for these patients, which includes the application of therapeutic ultrasound, which is diathermy, and imaging tests like magnetic resonance imaging (MRI) 12.

Case report..

Early Onset Parkinson's Disease in a Chronic Alcoholic: A Case Study

A 50-year-old male presented to hospital with chief complaints of shivering of hands and legs for the past week, along with a lack of sleep and general weakness for 15 days. The patient had a 30-year history of chronic alcoholism and was a smoker. On examination, he displayed resting tremors of both upper and lower limbs, bradykinesia, and rigidity, characteristic of PD. His neurological examination was otherwise unremarkable. His vital signs were within normal limits. Neurological examination revealed cogwheel rigidity in both upper and lower limbs, along with a resting tremor that was more pronounced at rest and reduced with movement, bradykinesia and short steps. The patient's gait was slow with reduced arm swing bilaterally.

The patient Is conscious and afebrile with a pulse rate of 94 beats per minute. Their blood pressure is measured at 130/90 mmHg. Cardiovascular system examination reveals normal heart sounds (S1S2+), and the abdomen is soft to palpation.

Clinical investigations complete blood count Indicates a white blood cell count (WBC) of 5.91 x 10^3/uL, red blood cell count (RBC) of 4.66 x 10^6/uL, and hemoglobin levels at 14.7 g/dL. Platelets are within the normal range at 228 x 10^3/uL, and the red cell distribution width coefficient of variation (RDW CV) is 12.8%, also normal. Neutrophil count is 5.4 x 10^3/uL, and eosinophil count is 2.7 x 10^3/uL. Mild normocytic anemia (Hemoglobin: 11 g/dL)Leukocytosis with neutrophil predominance.

Moving to the kidney function tests, urea is 20 mg/dL, and creatinine is 0.97 mg/dL, both within normal limits. Total protein is 6.9 g/dL, with albumin at 4.35 g/dL and globulin at 2.55 g/dL. Liver function testsshow normal values for alanine aminotransferase (ALT) at 14 U/L, aspartate aminotransferase (AST) at 22 U/L, and total bilirubin at 1.22 mg/dL. Elevated liver enzymes:AST: 120 U/L (Normal: 5-40 U/L), ALT: 90 U/L (Normal: 7-56 U/L), Alkaline Phosphatase: 200 U/L (Normal: 40-150 U/L) and elevated Alkaline Phosphatase: 200 U/L (Normal: 40-150 U/L).Finally, electrolytes are balanced with sodium at 140 mmol/L, potassium at 3.5 mmol/L, and chloride at 105 mmol/L electrolytes are within normal limits.

Diagnosis:

Based on the clinical presentation and Many symptoms such as bradykinesia (slowness of movement), stiffness, tremors, and postural instability. A comprehensive medical history, a neurological examination, and a medical examination of these symptoms are usually used to make the diagnosis. the patient was diagnosed with early-onset Parkinson's disease. The patient's history of chronic alcoholism raised suspicion for a potential link between alcohol consumption and the development of PD.

Treatment: management

The patient was started on the following treatment regimen:

Carbidopa/Levodopa 25/100 mg three times daily for management of motor symptoms.

Thiamine supplementation to address possible thiamine deficiency associated with chronic alcoholism.

Pantoprazole for gastroprotection due to long-term nonsteroidal anti-inflammatory drug use for arthritis.

Discussion:

This case presents a 50-year-old male with a history of chronic alcoholism and smoking, now presenting with complaints of shivering of hands and legs, lack of sleep, and general weakness. Parkinson's disease (PD) was diagnosed based on clinical symptoms of resting tremors, bradykinesia, and rigidity. The patient's lab results showed a relatively normal blood count and renal function, but liver function tests showed mild abnormalities 13.The patient's chronic alcoholism Is significant as chronic alcohol consumption has been associated with an increased risk of developing PD. Ethanol and its metabolites can lead to neurotoxicity and oxidative stress, which are implicated in the pathogenesis of PD. Furthermore, alcohol can interfere with nutrient absorption, potentially contributing to thiamine deficiency, which is also common in alcoholics and can exacerbate neurological symptoms14.

PD is a progressive neurodegenerative disorder characterized by dopaminergic neuron loss in the substantia nigra, leading to motor and non-motor symptoms. The patient's symptoms of resting tremors, bradykinesia, and rigidity align with classic PD presentations. The treatment plan includes Carbidopa/levodopa to increase dopamine levels in the brain, alleviating motor symptoms. Thiamine supplementation is essential, especially given the patient's history of alcoholism, to prevent or treat potential thiamine deficiency 15.

The results of the hepatic function tests revealed moderate variations in total bilirubin, AST, and ALT. Alcoholic liver damage can result from long-term alcohol use and can show up as high bilirubin and liver enzyme values. The liver enzymes are just slightly increased in this particular case, though, which raises the possibility of early liver dysfunction. This case study provides valuable insight into the challenges associated with diagnosing and treating Parkinson's disease in people who have a history of chronic alcohol misuse. It also highlights how important a multidisciplinary approach is to provide patients the best treatment possible 16.Alcohol's primary site of action is the brain. Extended and excessive alcohol use raises the risk of neuroimmune response, glutamate excitotoxicity, and oxidative stress, all of which interact to cause irreversible brain damage 17. Control over alcohol intake, avoiding obesity and eating too many long-chain fatty acids (or substituting them with medium-chain fatty acids), and utilizing PPC to restore S-adenosylmethionine and PCs. There is hope for improved prevention and treatment of alcoholic fatty liver due to advances in our understanding of the pathophysiology of the condition and how it progresses to inflammation and fibrosis 18.

Conclusion:

This case highlights the complexity of managing a patient with Parkinson's disease and a history of chronic alcoholism. Chronic alcoholism increases the risk of developing PD and it can also worsen its symptoms and complicate treatment due to potential nutrient deficiencies and liver dysfunction. Early recognition of PD symptoms, appropriate diagnosis, and relevant treatment are crucial in managing the disease. In this case, the patient was started on Carbidopa/levodopa and thiamine supplementation. Regular monitoring of liver function tests is recommended due to the patient's history of chronic alcoholism. It Is important for healthcare providers to consider the effect of alcoholism on neurological disorders like PD and to add both the neurological and systemic effects of chronic alcohol consumption in treatment plans.

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