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A CASE REPORT ON PARKINSONISM-PORENCEPHALIC CAVITY WITH GLIOSIS

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ABSTRACT

Porencephalic cavities or cysts are rare cerebrospinal fluid-filled spaces that result from the destruction or loss of brain tissue, often due to ischemic events, hemorrhages, or congenital malformations. Gliosis, a reactive process which is marked by the proliferation of the glial cells in the brain on response to brain injury, which typically accompanies with these structural lesions. When combined, these abnormalities can lead to motor dysfunction resembling Parkinson's disease (PD), which is a neuro degenerative disorder predominantly affecting dopaminergic neurons in the substantia nigra of the brain. In the case of this 67-year-old patient, the

presentation of parkinsonism alongside porencephalic cavities and gliosis suggests a potential disruption in the basal ganglia circuitry, a critical network involved in motor control. Neuroimaging revealed a porencephalic cavity in the right fronto-parietal region with surrounding gliosis with hemosiderin residue, which may contribute to impaired neural connectivity and exacerbation of parkinsonism symptoms such as bradykinesia, rigidity, and tremor. The age of the patient further worsens the complexity, as the degenerative changes associated with both age-related Parkinson's disease and the structural brain abnormalities could interact, complicating the diagnosis and its treatment. The patient also mentions about disease history of type 2 Diabetes Mellitus and Hypertension which further worsens his condition. Conventional dopaminergic therapy are often only the mainstay for Parkinsons Disease, may be less effective due to the involvement of structural brain damage, thus requiring a tailored and patient centered treatment approach. This case highlights the complex interplay between vascular risk factors and structural brain damage in the pathogenesis of parkinsonism. It shows the importance of a multidisciplinary approach combining neurologic, metabolic, and rehabilitative strategies.

Keywords: Porencephalic cavities, Gliosis, Cerebrospinal Fluid, Parkinsons Disease, Dopaminergeic

INTRODUCTION

Parkinsonism is a clinical syndrome characterized by bradykinesia, resting tremor, rigidity, and postural instability. While idiopathic Parkinson's disease (PD) is the most common cause of parkinsonism, secondary or atypical parkinsonism can arise from structural brain lesions, infections, toxins, or vascular insults. Porencephaly, a rare condition characterized by cystic cavities within the brain parenchyma, is typically congenital but can also result from perinatal or postnatal insults. Gliosis, a reactive process involving astrocyte proliferation, often accompanies such lesions. The association between porencephalic cavities with gliosis and parkinsonism is exceedingly rare, with limited cases reported in the literature. This case report presents a 67-year-old male with parkinsonism secondary to a porencephalic cavity with gliosis. The report highlights the clinical presentation, diagnostic workup, imaging findings, and management, followed by a detailed discussion of the pathophysiology and implications of such a rare association.

CASE PRESENTATION

Clinical History

A 67-year-old male presented with slurring of speech, dysarthria and walking difficulty and intermittent tremors in his right hand. He also reported frequent falls, mild memory difficulties, and a soft, monotonous voice. There was no history of fever, head trauma, or toxin exposure. He has personal medical history of parkinsonism, Hypertension and Type 2 Diabetes mellitus and CVA with right hemiplegia.

Physical Examination

On examination, the patient exhibited bradykinesia, and resting tremor. His gait was shuffling, and he had difficulty initiating movements. Postural instability was evident. Facial expression was reduced, and his speech was slurred in nature. Cognitive assessment revealed mild in dysfunction.

Laboratory Investigations

Routine blood tests, including complete blood count, renal and liver function tests, thyroid function tests, and vitamin B12 levels, were within normal limits. Serological tests for syphilis, HIV, and autoimmune encephalitis were negative. HbA1c level was elevated (10.9%), Cerebrospinal fluid (CSF) was found to be normal.

Imaging Studies

1. MRI Brain: Revealed Hemosiderin residue noted
2. CT Brain: Porencephalic cavity with surrounding gliosis in fronto parietal region and volume loss. Ex Vacuo dilation of atrium, occipital and temporal horns of right lateral ventricle.

Diagnosis

Based on the clinical presentation and imaging findings, the patient was diagnosed with parkinsonism secondary to a porencephalic cavity with gliosis.

Treatment

The patient was treated with T. LEVODOPA+CARBIDOPA (100/25 mg 1-1-1). T ROPINIROLE (3mg HS), T RASAGILINE (1mg 1-0-0), T AMANTADINE (100mg PO 1-0-1), T TRIHEXYPHENIDYL (500mg PO 1-0-0), T DOSULEPIN (25mg HS), INJ THIAMINE (200mg BD), T CLOPIDOGREL+ASPIRIN (75mg 0-1-0), T TELMISARTAN (40mg 1-0-0), T TENOFOVIR (400mg 0-1-0),T RIFAXIMINE (400mg 1-0-1), SYP LACTULOSE (15ml HS), INJ HUMAN ACTRAPID (12U-12U-10U). Physical therapy was initiated to improve mobility and reduce fall risk. Cognitive training and speech therapy were also recommended.

DISCUSSION

Parkinsonism secondary to structural brain lesions, often termed "vascular parkinsonism" or "secondary parkinsonism," is well-documented. Lesions affecting the basal ganglia, thalamus, or frontal-subcortical circuits can disrupt dopaminergic pathways, leading to parkinsonian symptoms. In this case, the porencephalic cavity in the left frontoparietal region likely disrupted cortico-striato-thalamo-cortical loops, contributing to the clinical presentation. Gliosis, a reactive process to brain injury, may exacerbate neuronal dysfunction by promoting neuroinflammation and altering synaptic plasticity. The presence of gliosis around the cavity suggests chronicity and may have contributed to the progressive nature of the symptoms.

From previous case reports on the above heading it was observed as: Vascular lesions in the basal ganglia or thalamus caused contralateral parkinsonism, often levodopa-resistant. Unlike our case, these patients lacked cystic formations but shared disconnection of motor circuits. A young adult with a post-traumatic porencephalic cyst developed hemiparkinsonism, improving with dopaminergic therapy. A delayed presentation of motor symptoms due to compensatory mechanisms failing in adulthood.

CONCLUSION

This case highlights a rare association between parkinsonism and a porencephalic cavity with gliosis in a 67-year-old male. The clinical presentation, imaging findings, and response to treatment underscore the importance of considering structural brain lesions in the differential diagnosis of parkinsonism, particularly in cases with atypical features. The presence of gliosis suggests a chronic, progressive process, emphasizing the need for comprehensive management, including pharmacological and non-pharmacological interventions. Further research is needed to elucidate the mechanisms linking porencephaly and gliosis to parkinsonism and to explore potential therapeutic targets. This case adds to the limited literature on this rare association and underscores the importance of a thorough diagnostic workup in patients with atypical parkinsonism.

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