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Parkinsonism in a young adult after ventriculoperitoneal shunt procedure for tri ventricular obstructive hydrocephalus – A case report

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ABSTRACT

This case report centers on a 19-year-old male diagnosed with tri-ventricular obstructive hydrocephalus due to aqueductal stenosis. Following a right ventriculoperitoneal (VP) shunt procedure, the patient initially exhibited marked improvement but later developed Parkinsonian symptoms. Extensive investigations meticulously ruled out shunt malfunction and phenytoin toxicity. The suspicion of Parkinsonism secondary to VP shunt emerged, leading to the initiation of anti-Parkinsonian medications. The patient underwent a personalized treatment regimen with anti-Parkinsonian drugs, which yielded substantial clinical improvement. This case report underscores the importance of identifying unusual neurological presentations post-VP shunt. The critical role of early diagnosis and precise pharmacological intervention in the patient's recovery cannot be overstated, and it emphasizes the potential for rare complications like post-shunt Parkinsonism.

Key words: Tri-ventricular obstructive hydrocephalus; Right ventriculoperitoneal; Post-shunt Parkinsonism

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INTRODUCTION

Tri-ventricular obstructive hydrocephalus is a rare neurological condition characterized by the blockage of cerebrospinal fluid (CSF) flow within the ventricular system, typically caused by aqueductal stenosis. Clinically, it presents with symptoms related to increased intracranial pressure, such as headaches, nausea, and visual disturbances. The standard treatment for hydrocephalus involves ventriculoperitoneal (VP) shunts, which divert excess CSF from the brain's ventricles into the peritoneal cavity. While these shunts are generally effective, they can occasionally lead to unexpected complications, such as infection, intracerebral hemorrhage, and CSF leak.

One such rare complication is the development of Parkinsonism following a VP shunt procedure. Parkinsonism encompasses a range of motor symptoms, including bradykinesia (slowness of movement), tremors, rigidity, and postural instability.[1] Although Parkinson's disease (PD) is the most common cause, secondary Parkinsonism can be due to several other causes, such as drug-induced, toxin-induced, and trauma, malignancies.[2-6] However, the development of Parkinsonism after a VP shunt surgery is exceptionally rare, and the underlying mechanisms remain poorly understood.

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CASE PRESENTATION

A 19-year-old male patient presented with a four-day history of double vision in May 2023. Imaging studies revealed tri-ventricular obstructive hydrocephalus secondary to aqueductal stenosis. Following the diagnosis, the patient underwent a right VP shunt procedure to alleviate the hydrocephalus-related symptoms. The patient initially showed significant improvement post-surgery, and his hydrocephalus-related symptoms resolved. He was discharged with a prescription for anti-epilepsy drugs and scheduled for regular shunt evaluations to monitor his condition. Serum phenytoin levels were within the normal limits (Table 1).

Approximately 1 month after the VP shunt procedure, the patient's clinical course took an unexpected turn. He began experiencing generalized slowness of activities, starry gaze, generalized tremors, and disturbances in his sleep patterns. Given his medical history, shunt malfunction was the initial suspicion; however, a comprehensive evaluation, including magnetic resonance imaging scans, consistently ruled out any evidence of hydrocephalus recurrence or shunt-related complications (Figures 1 and 2).

Table 1: Biochemical parameters of the patient

Investigations	Level
WBC	8.1 C/cu.mm
Hemoglobin	13.6 g/dl
Sr. Sodium	141 mEq/L
Sr. Potassium	4.22 mEq/L
Sr. Bicarbonate	22.8 mEq/L
Sr. Creatinine	0.8 mg/dL
Sr. Phenytoin	<0.8 µg/mL

WBC: White blood cell

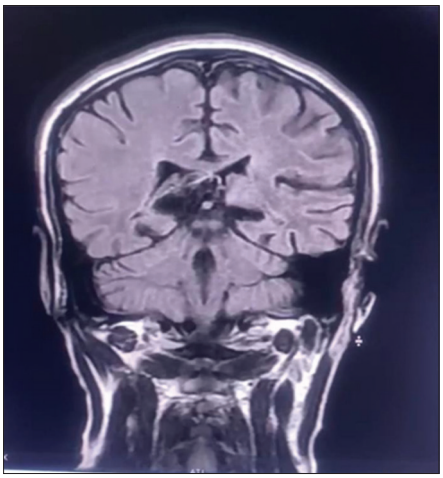

Figure 1: Magnetic resonance imaging brain showing the ventriculoperitoneal shunt *in situ* with its tip in the left lateral ventricle and normal third, fourth and lateral ventricular spaces bilaterally

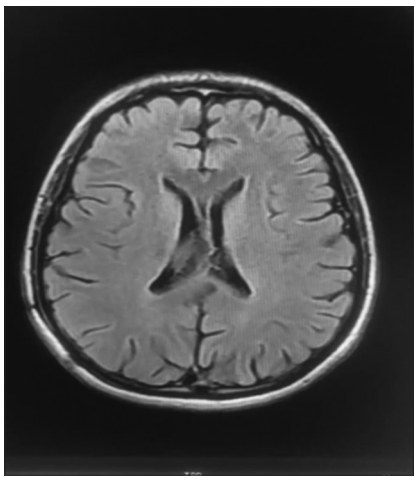

Figure 2: The ventricular spaces with no significant dilatation and no evidence of hydrocephalus

DISCUSSION

The emergence of Parkinsonian symptoms in a patient with a history of hydrocephalus presented a diagnostic challenge for the medical team. Parkinsonism is caused by dopaminergic neuronal loss in the midbrain due to neuronal degeneration, and this results in a decrease in dopamine levels, especially in the post-commissural putamen and other regions of the basal ganglia.[1] The absence of shunt malfunction and normal phenytoin levels led to exploring alternative explanations for the patient's condition. Parkinsonism, a rare complication of the VP Shunt procedure, was suspected.[7] In this case, the medical team initiated a carefully tailored pharmacological regimen for the patient. Medications included levodopa[8], trihexyphenidyl hydrochloride, amantadine hydrochloride, and pramipexole dihydrochloride. This approach alleviated the patient's Parkinsonian symptoms while minimizing potential side effects.

Remarkably, the therapeutic strategy yielded significant clinical improvements. The patient's tremors subsided, and his overall clinical status improved, reinforcing the hypothesis that Parkinsonism was related to hydrocephalus and its surgical treatment. Two proposed mechanisms underpinning this phenomenon were of particular interest. The first centers around dysfunction in the presynaptic nigrostriatal dopaminergic pathway, which regulates motor control. In cases where Parkinsonism emerges, high doses of dopaminergic drugs may be necessary to alleviate symptoms.[9] The second mechanism involves the corticobasal ganglia loop. Some evidence suggests that the endoscopic third ventriculostomy, which diverts CSF differently than VP shunts, may be more effective in addressing this aspect of Parkinsonism following hydrocephalus surgery.[10]

CONCLUSION

This case report highlights the importance of recognizing atypical neurological presentations in patients with a history of hydrocephalus, even when surgical interventions initially appear successful. It underscores the potential for rare complications, such as Parkinsonism, following VP shunt procedures.

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