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Spontaneous CSF Leakage Leading to Symptomatic Improvement in a Patient with Normal Pressure Hydrocephalus: A Case Report

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ABSTRACT

Normal Pressure Hydrocephalus (NPH) is a reversible cause of dementia that often responds to Cerebrospinal Fluid (CSF) drainage. Spontaneous CSF leakage is rare in this setting. We report a 79-year-old man with urinary incontinence, gait disturbance, and cognitive decline, diagnosed with probable NPH. Serial lumbar punctures led to marked improvement. During a treatment delay due to the COVID-19 pandemic, he developed spontaneous CSF rhinorrhea, which was followed by further symptomatic relief. This case suggests that spontaneous CSF leakage may temporarily replicate the therapeutic effects of CSF drainage, offering insight into symptom fluctuation and potential diagnostic markers in NPH.

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Abbreviation

CSF: Cerebrospinal fluid
Hrs: Hours
MoCA: Montreal Cognitive Assessment
WBC: White Blood Cell
RBC: Red Blood Cell
GLU: Glucose
PR: Protein
OP: Opening Pressure

Introduction

Normal Pressure Hydrocephalus (NPH) is a potentially treatable cause of dementia characterized by the triad of gait disturbance, cognitive impairment and urinary incontinence. Typically associated with normal Cerebrospinal Fluid (CSF) pressure and ventriculomegaly out of proportion to cortical atrophy. Diagnosis can be guided by the Relkin criteria, classifying cases as probable, possible, or unlikely [1].

Therapeutic lumbar drainage can transiently improve symptoms and help identify candidates for permanent CSF diversion. Moreover, serial lumbar puncture can serve as a therapeutic alternative for patients with NPH who are not ideal candidates for surgical intervention. Spontaneous CSF leakage, though rare, has been observed in a variety of conditions, including hydrocephalus, tumors, elevated intracranial pressure, and congenital anomalies [2,3].

This report presents a unique case of NPH with spontaneous CSF rhinorrhea leading to substantial clinical improvement, a phenomenon not previously reported in idiopathic NPH (iNPH).

Case Presentation

A 79 years-old male was referred in December 2019 with a one-year history of urinary incontinence, followed by progressive gait disturbance and cognitive decline. His family reported increasing forgetfulness, repeated questions and statements, word finding difficulties, and disorientation to time and place over the preceding months. Additional features included visual hallucination and symptoms suggestive of Rapid Eye Movement (REM) sleep behavior disorder.

His medical history was significant for diabetes mellitus, hypertension, and cardiac arrhythmia managed with a permanent pacemaker. Current medications included rivaroxaban, insulin, bisoprolol, valsartan, simvastatin, clonazepam and duloxetine.

Neurologic examination revealed a positive Myerson sign, absent ankle reflex, stocking-glove hypoesthesia, and a mild tremor in the left hand. There was no rigidity, bradykinesia, ideomotor or ideational apraxia, or grasp and snout reflexes. He exhibited a slightly stooped posture with prominent postural instability. His gait was wide-based, shuffling, and markedly effortful, requiring assistance, characteristics consistent with frontal lobe gait. Neuropsychological testing revealed deficit in memory, attention, and visuospatial domains, with Mini-Mental State Examination (MMSE) and Montreal Cognitive Assessment (MoCA) scores of 19 and 14 out of 30, respectively.

A non-contrast brain CT demonstrated significant ventriculomegaly, including dilation of the third and fourth ventricles, with an Evans index of 0.37, which was disproportionate to the degree of cortical atrophy (Figure 1). Other laboratory investigations including complete blood count, thyroid function tests, HIV serology and vitamin B12 level, were within normal limits.

The clinical picture was consistent with iNPH. The patient underwent four lumbar punctures over the course of several weeks, with each session involving the removal of approximately 30 mL of CSF. Notably, two of the four opening pressure measurements were mildly elevated (up to 21 cmH_2O), while the other two fell within the normal range. All four procedures resulted in marked clinical improvement, particularly in gait function, enabling the patient to walk independently after each intervention.

In February 2020, due to the onset of COVID-19 pandemic, planned serial taps were suspended. In late March, the patient developed sudden-onset CSF rhinorrhea following several sneezing episodes, expelling approximately 30-40 ml of CSF. Family members observed marked improvement in both gait and cognition, described it as “miracle” lasting three months before symptoms gradually return.

Discussion

Spontaneous CSF rhinorrhea is most often reported in the setting of elevated intracranial pressure, hydrocephalus, or congenital anomalies [1]. In this case, mild elevation in opening pressure was noted during lumbar taps, but the clinical context was consistent with idiopathic NPH.

Ommaya et al. first classified CSF leaks into traumatic and spontaneous subtypes, with non-traumatic leaks further categorized into congenital, tumoral, infectious, and vascular causes [2,3]. While hydrocephalus-related CSF leaks are rarely spontaneous, there have been reports in patients with aqueductal stenosis [4,5]. Our case appears to be the first report of spontaneous CSF leakage leading to temporary symptomatic relief in iNPH.

The presumed mechanism is transient reduction in CSF pressure, mimicking the effect of therapeutic lumbar drainage. Similar symptomatic relief has been documented in idiopathic intracranial hypertension following CSF leak [6,7]. In our case, sneezing may have precipitated the leak via transient ICP elevation opening a dural defect.

These observations support the hypothesis that temporary CSF loss can relieve symptoms in NPH and highlight the complex dynamics of CSF physiology.

Conclusion

Spontaneous CSF leakage may result in temporary symptomatic relief in patients with Normal Pressure Hydrocephalus. This rare phenomenon underscores the therapeutic effect of CSF pressure modulation and may provide insight into CSF dynamics in neurodegenerative conditions.

Acknowledgement

This study was performed under the supervision of Firoozgar Clinical Research Development Center (FCRDC), affiliated by Iran University of Medical Sciences. In addition, we kindly appreciate our patient and his son that allow us to share our outstanding experience in this study with the other researchers and clinicians.

Author Contribution

Conceptualization: MAD; Data Curation: MAD, SF, TK, SNT; Investigation: MAD, SF, TK, SNT; Methodology: SNT; Project administration: MAD; Supervision: MAD, SNT; Validation: MAD, TK, SNT; Writing – original draft: SF, TK; Writing – review & editing: MAD, SNT.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Funding Statement

There are no funding resources for this study.

Conflict of Interest Disclosure

There is no conflict of interest to disclose related to this study.

Ethics Approval Statement

This study was performed under the supervision of Firoozgar Clinical Research Development Center (FCRDC) which is an academic research center affiliated by Iran University of Medical Sciences.

Patient Consent Statement

Written informed consent was obtained from the patient’s son to publish this report anonymously in accordance with the journal's patient consent policy.

Table 1: The Characteristics of Gait Indices, Moca Score and CSF Markers Before and After Four Trials of Lumbar Tap.
***Unable to stand and walk**
ND: No data present.

	Gait indices								MoCA score		CSF analysis	CSF OP (cmH_2O)
	CADENCE (step/min)				SPEED (cm/sec)							
	Before Tap	2 hrs after Tap	6 hrs after Tap	24 hrs after Tap	Before Tap	2 hrs after Tap	6 hrs after Tap	24 hrs after Tap	Before Tap	24 hrs after Tap		
1st TAP 2019.23.12	*	*	77.7	71.11	*	*	18	22	14	14	WBC:0 RBC:0 GLU:37 PR:49	13
2nd TAP 2019.29.12	71.11	80	72	72.85	22	16	28	28	14	14	WBC: 0 RBC:100 GLU:88 PR:68	ND

3rd TAP 2020.21.1	83.75	84	82.5	76	20.8	20	20.8	16	10	11	WBC: 0 RBC:100 GLU:65 PR:50	22
4th TAP 2020.19.8	75.6	78.8	ND	ND	19	20	ND	ND	10	11	ND	21

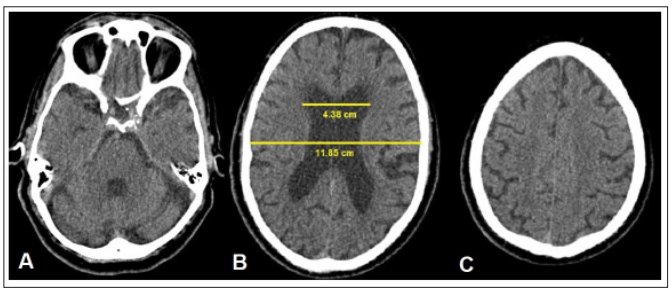

Figure 1: Brain CT Scan, Axial View, Revealed Increased the Size of 4th (A), 3rd (not shown) and Lateral Ventricles (B) with the Evans index of 0.37 (B). The Ventriculomegaly is Disproportionate to the Amount of Cortical Atrophy (C). These Findings are Compatible with the Diagnosis of NPH.

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