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Case Report

Spontaneous Intracranial Hypotension, Menière's Disease and Secondary Benign Paroxysmal Positional Vertigo: Case Report

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Abstract

Background/Objectives: Spontaneous intracranial hypotension (SIH) is a rare pathology that arises in the context of a known or suspected cerebral spinal fluid (CSF) leak. A key symptom of SIH is an orthostatic headache; however, additional neurological complications are common. This case study not only highlights the co-existence of Menière's disease and SIH but describes a subsequent complication of benign paroxysmal positional vertigo (BPPV) and management thereof. **Case Description:** The patient is a 61-year-old female who presented to the emergency department due to an intractable headache, right sided weakness and aphasia. CT/MRI revealed a subdural hematoma overlying the left cerebral hemisphere measuring up to 8 mm with 4 mm left to right midline shift. Fluoro-guided total spine myelogram, cisternogram, and lumbar epidural blood patch were performed for suspected SIH. As headache, right sided weakness and aphasia resolved, the patient began reporting onset of constant "spinning" dizziness, tinnitus and aural fullness mimicking symptoms of a Menière's attack. The vestibular examination was consistent with compensated bilateral Menière's disease (left > right) and right horizontal canalithiasis BPPV. The patient was treated with Gufoni and Lempert maneuvers with complete resolution of positional dizziness and associated nystagmus along with improved balance and gait. **Discussion/Conclusions:** This case study highlights the importance of multidisciplinary assessment in complex neurological cases and specifically recommends that patients with Menière's disease accompanied by intractable headaches undergo extended neuroradiological examination of the brain to exclude underlying spontaneous intracranial hypotension syndrome.

Keywords: brain; case report; physical therapy; vertigo; vestibular

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1. Introduction

Spontaneous intracranial hypotension (SIH) is a rare but increasingly recognized neurological condition that most commonly results from a spontaneous cerebrospinal fluid (CSF) leak along the spinal axis [1,2]. The condition is characterized by a reduction in intracranial CSF volume and pressure, most often in the absence of antecedent trauma or invasive procedures. A hallmark clinical feature of SIH is an orthostatic headache, typically worsening in an upright position and improving with recumbency [1,3]. However, the clinical presentation of SIH is heterogeneous and may include a wide range of neurological, cognitive, and otologic symptoms which can complicate timely diagnosis. Unfortunately,

the pathophysiological consequences of reduced intracranial pressure extend beyond physical symptoms. Loss of buoyant support to the brain can result in downward displacement of intracranial structures, leading to traction on pain-sensitive meningeal and venous elements. This mechanical strain may cause tearing of bridging veins, predisposing individuals to subdural fluid collections or subdural hematomas (SDH; [2,4]). SDH is a recognized complication of intracranial hypotension and is most often associated with underlying cerebrospinal fluid CSF leakage. Studies have demonstrated a high incidence of spinal CSF leaks in patients presenting with nontraumatic SDH, supporting an underlying hypotensive etiology in this population [5]. Similar findings have been reported in postoperative settings, where CSF loss following spinal procedures can result in intracranial hypotension and subsequent SDH development [6]. Together, these findings emphasize the importance of evaluating for CSF leakage in patients with nontraumatic SDH, as the hematoma may represent a secondary manifestation of an underlying CSF pressure abnormality rather than a primary intracranial process.

In addition to intracranial complications, SIH has been associated with auditory-vestibular symptoms that can closely mimic primary inner ear disorders. These symptoms primarily include fluctuating hearing loss, tinnitus, aural fullness, and episodic vertigo. The proposed mechanism involves altered pressure transmission between the CSF and inner ear compartments, as the perilymphatic space communicates with CSF via the cochlear aqueduct [7,8]. Changes in CSF pressure may disrupt the delicate balance between perilymphatic and endolymphatic pressures, producing symptoms that resemble those of Menière's disease. This overlap presents a diagnostic challenge, particularly when traditional neuroimaging findings are subtle or absent [7,8]. Furthermore, Menière's disease itself has a recognized clinical association with another common condition known as benign paroxysmal positional vertigo (BPPV), reflecting shared inner ear vulnerability or secondary otolithic dysfunction [9,10]. While BPPV is encountered in both idiopathic and secondary forms, its occurrence in the context of intracranial hypotension has received limited attention in the literature. The co-existence of SIH and Menière's disease is exceedingly rare, with only a small number of cases previously reported [1]. Additionally, published cases have focused primarily on overlapping symptomatology rather than downstream vestibular sequelae.

Management of vestibular disorders such as Menière's disease and BPPV requires a multidisciplinary approach, often including physical therapy (PT). In the United States (U.S.), physical therapists play a key role in the evaluation and management of vestibular disorders in both acute inpatient and outpatient settings. Physical therapists in the U.S. can be expected to perform at an advanced beginner to intermediate level in vestibular assessment and rehabilitation techniques upon graduation [11]. This is not the case in other areas of the world despite the education recommendations from the Barany Society [12]. In Europe, it is reported that only 19% of clinicians practicing vestibular rehabilitation have received formal training in this area during their entry-level education. Most respondents indicated that they acquired vestibular knowledge through postgraduate courses or workplace experience [13].

Asian and Middle Eastern regions report similar findings as their European counterparts. Sung (2020) reported that more than 75% of physical therapists in South Korea had not received vestibular education during their professional training. Despite this lack of formal instruction, most respondents recognized the clinical value of vestibular rehabilitation and expressed interest in receiving additional education [14]. Similarly, Al Shammari et al. (2025) found that many physical therapists in Saudi Arabia reported limited knowledge of vestibular assessment techniques, despite acknowledging the importance of vestibular rehabilitation in clinical practice [15].

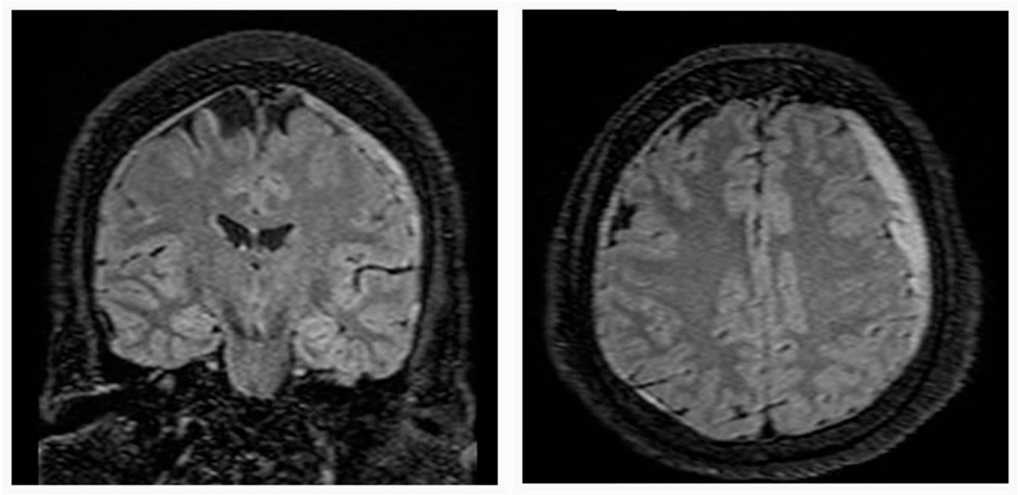
In addition to vestibular assessment and rehabilitation techniques, physical therapists have the potential to address the more typical functional consequences of vestibular dysfunction, including impaired balance, gait instability, and fall risk. This traditional PT role becomes particularly important in medically complex presentations such as spontaneous intracranial hypotension, where vestibular symptoms may evolve, overlap, or change over time, requiring ongoing clinical reasoning and reassessment [16]. Treatment of Menière's disease is typically aimed at symptom management and may include dietary modification, medication, and/or procedural interventions such as this case. Despite these options, management can be challenging due to symptom fluctuation, variable disease progression, and overlap with other vestibular conditions. In contrast, BPPV is most effectively treated with canalith repositioning maneuvers when the involved canal is accurately identified [10,17].

This case report describes a patient with SIH presenting with Menière's disease-like symptoms who subsequently developed BPPV as a secondary complication. This presentation adds to the limited body of literature describing the interplay between altered CSF dynamics and peripheral vestibular dysfunction, particularly in cases where symptoms evolve over time. The clinical course highlights how vestibular manifestations may shift or overlap, requiring careful longitudinal assessment to accurately identify emerging conditions such as BPPV. Beyond the clinical presentation, this case highlights important considerations for care delivery across healthcare systems. While the amount of vestibular education in the U.S. varies between institution, physical therapists are routinely involved in vestibular assessment, diagnosis and management. In other areas around the world, the role of PT may be limited or not present at all. The evolution of symptoms observed here underscores the potential value of integrating vestibular-specific assessment with ongoing functional management. Broader incorporation of advanced vestibular training within physical therapy practice may support earlier recognition of secondary conditions and improve management of complex, evolving vestibular presentations. This case report was written according to CARE guidelines. The CARE checklist is in the Supplementary Materials (Supplementary File S1).

2. Case Description

A 61-year-old female with a complex medical history presented to a large tertiary care hospital with progressive neurological and vestibular symptoms. Her past medical history was significant for bilateral Menière's disease, including a left-sided vestibular neurectomy performed in the 1990s, recurrent pulmonary emboli previously managed with warfarin, common variable immunodeficiency treated with monthly intravenous immunoglobulin infusions, insulin-dependent diabetes mellitus, Sjogren's syndrome, chronic pain syndrome, and multiple prior lumbar spine surgeries, including a recent lumbar fusion revision.

The patient initially presented after approximately two weeks of worsening headache and subsequent acute right-sided upper and lower extremity weakness, aphasia, nausea, vomiting, and two syncopal episodes. In the emergency department, she was assessed by stroke neurology and assigned a National Institutes of Health Stroke Scale (NIHSS) score of three. Non-contrast computed tomography of the brain revealed a left convexity SDH measuring up to 8 mm in thickness with approximately 3 to 4 mm of left-to-right midline shift. Computed tomography angiography of the head and neck and perfusion imaging revealed no large vessel occlusion, hemodynamically significant stenosis, or perfusion deficits. Magnetic resonance imaging of the brain revealed bilateral convexity subacute SDH, left greater than right, with diffuse pachymeningeal enhancement, narrowing of the pontomesencephalic angle, and decreased fluid within the optic nerve sheaths (Figure 1). These findings were consistent with intracranial hypotension. No acute ischemia or intracranial mass was identified.



(**a**) (**b**)

Figure 1. *Brain MRI demonstrating findings consistent with intracranial hypotension.* Axial (**a**) and coronal (**b**) magnetic resonance imaging of the brain demonstrate bilateral convexity SDH, greater on the left, with associated mild left-to-right midline shift. There is diffuse pachymeningeal enhancement, along with imaging features of intracranial hypotension including narrowing of the pontomesencephalic angle and reduced cerebrospinal fluid surrounding the optic nerve sheaths. No evidence of acute ischemia or intracranial mass is identified.

Given concern for SIH in the context of prior lumbar surgery and reported intermittent clear right-sided otorrhea during a recent hospitalization, the patient underwent a fluoroscopy-guided total spine myelogram and cisternogram. Although no discrete cerebrospinal fluid leak was visualized, a lumbar epidural blood patch was performed due to high clinical suspicion. Following the procedure and prescribed bedrest, the patient demonstrated progressive resolution of headache, aphasia, and right-sided weakness. Her NIHSS score improved to zero. Despite this favorable response, it should be acknowledged that clinical and imaging findings may be due to another unknown etiology. As her focal neurological deficits improved, the patient reported new-onset vestibular symptoms, including constant spinning dizziness, tinnitus, and aural fullness. She described these symptoms as similar to prior Menière's disease episodes. The symptoms gradually improved with continued bedrest; however, she continued to experience severe positional vertigo described as spinning, most notable with rolling to her right side in bed. This positional dizziness persisted and prompted referral to PT for vestibular evaluation.

Physical therapy evaluation occurred after medical clearance for mobilization. At the time of the PT evaluation, the patient returned to her auricular baseline. The patient denied tinnitus, aural fullness, and acute hearing changes. Vestibulo-ocular examination revealed smooth pursuits, saccades, and convergence within normal limits. No spontaneous nystagmus or end-gaze nystagmus was observed. Vestibulo-ocular reflex testing revealed gaze instability without associated dizziness. VOR cancelation was within normal limits. Head impulse testing was positive bilaterally, evidenced by corrective catch-up saccades. Vestibular-ocular examination findings were consistent with compensated bilateral vestibular hypofunction, greater on the left, aligning with her known history of bilateral Menière's disease and prior left vestibular neurectomy. From a functional standpoint, the patient demonstrated impaired balance and gait instability. The patient's mobility was assessed using the Functional Gait Assessment (FGA) for discharge recommendations with the patient scoring 18 out of 30 points, indicating an increased risk for falls [18]. Following functional testing, the patient underwent positional testing for BPPV beginning with the

supine roll test. Traditional Dix–Hallpike assessment was deferred due to excessive cervical mobility requirements that were deferred at the time of assessment.

Positional testing via the supine roll test revealed geotropic nystagmus lasting less than 30 s with greater intensity on the right side versus the left side, suggesting right horizontal canalithiasis. Canalithiasis is characterized by free floating otoconial debris within the endolymph of an affected semicircular canal. This results in transient deflection of the cupula and brief episodes of positional vertigo. In contrast, cupulolithiasis involves otoconia adherent to the cupula of the affected semicircular canal. This causes the cupula to become gravity-sensitive and produces sustained nystagmus during certain positions [19]. Based on these findings, the patient was treated with two Gufoni maneuvers for right geotropic horizontal canalithiasis (Figure 2). The Gufoni maneuver was initially selected over the more common Lempert maneuver due to the ease of the maneuver, as the patient had various lines and wires requiring careful navigation [20].

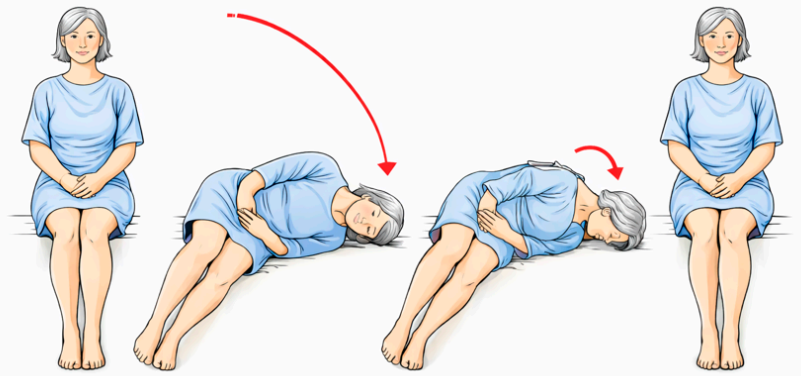


Figure 2. *Gufoni maneuver for right geotropic horizontal canalithiasis. OpenAI. (2026) [AI-generated].* Schematic illustration of the Gufoni maneuver performed to treat right-sided geotropic horizontal canal benign paroxysmal positional vertigo (BPPV). Starting from an upright seated position, the patient is rapidly brought down onto the left (unaffected) side. After a brief pause, the head is then turned approximately 45–60° downward toward the bed (nose-down position) and held for 1–2 min to facilitate movement of otolith debris within the right horizontal semicircular canal. The patient is subsequently returned to the seated position. This sequence may be repeated as clinically indicated.

Initial Gufoni maneuvers resulted in the conversion of horizontal geotropic nystagmus to horizontal apogeotropic nystagmus, which increased the duration of dizziness and produced persistent, non-fatiguing nystagmus. The conversion from geotropic to apogeotropic horizontal nystagmus during treatment represented the migration of otoconial debris from the non-ampullary arm into the ampullary arm of the horizontal semicircular canal. In this case, the conversion most likely occurred during the return to the upright sitting position following the Gufoni maneuver. During this transition, the head and trunk rapidly change orientation relative to gravity. This change creates inertial forces that may allow the otoconia to continue accelerating within the canal even after body movement decelerates. Rather than progressing toward the utricle, the debris was redirected toward the ampullary portion of the canal. This shift in particle location would alter the direction of endolymphatic flow generated during subsequent positions and produce an apogeotropic nystagmus pattern. The case illustrates that repositioning maneuvers are dynamic procedures and require ongoing interpretation of nystagmus by trained clinicians. Continuous observation of eye movements is critical for recognizing and selecting appropriate treatment. The Lempert maneuver was then used to promote movement of the debris away from the ampullary arm and toward the utricle (Figure 3) [19].

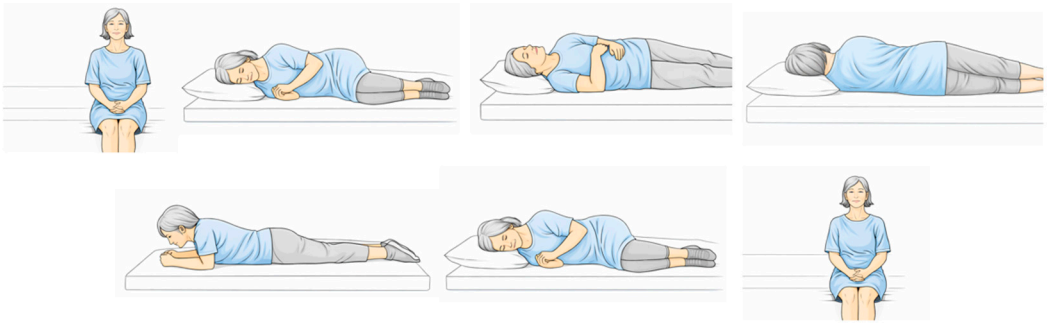


Figure 3. *Lempert maneuver for right horizontal cupulolithiasis. OpenAI. (2026) [AI-generated].* Schematic illustration of the Lempert maneuver performed to treat right-sided horizontal canal cupulolithiasis following conversion from canalithiasis. Beginning in an upright seated position, the patient is brought into a supine position with the head in neutral alignment. The head is then sequentially rotated in 90° increments toward the unaffected (left) side, progressing through supine, left side-lying, prone (face-down), and right side-lying positions. Each position is maintained for approximately 15–30 s or until nystagmus subsides, facilitating detachment and repositioning of debris from the cupula. The maneuver concludes with return to the seated position.

Following one Lempert maneuver, the patient was reassessed and demonstrated complete resolution of positional nystagmus and subjective positional vertigo. Typically, the Gufoni maneuver is considered an incomplete maneuver for the apogeotropic nystagmus variant and has been found to not be as effective as other maneuvers such as the Lempert maneuver [21]. Furthermore, utilizing the Gufoni maneuver for the apogeotropic nystagmus variant is more complicated to manage and requires conversion of the nystagmus back to geotropic first, followed by additional maneuvers. This attempted reconversion would likely have involved a number of transitions from seated to/from side-lying for successful nystagmus conversion as well and therefore the Gufoni for the apogeotropic variant was deferred.

The patient was re-evaluated the following day by the same PT as before and reported complete resolution of positional dizziness, nystagmus and no recurrence of neurological symptoms. Repeat vestibulo-ocular examination revealed smooth pursuits, saccades, convergence, vestibulocular reflex, and vestibulocular cancelation within normal limits. No spontaneous nystagmus or end-gaze nystagmus was observed. Head impulse testing was negative bilaterally, with no evidence of corrective catch-up saccades. Functionally, her FGA score improved to 23 out of 30 points, reflecting improved dynamic balance, gait stability and minimal fall risk. At the time of discharge from the hospital, she was ambulating without positional vertigo and was referred to the outpatient vestibular PT clinic connected to her neuro-otolaryngologist for follow up.

3. Discussion

This case highlights a rare but clinically significant cascade in which spontaneous intracranial hypotension altered inner ear pressure regulation, produced Menière's-like hydropic symptoms, and subsequent onset of horizontal canal BPPV. The progression from central CSF pressure dysregulation to peripheral otolithic dysfunction illustrates the interconnected nature of intracranial and labyrinthine physiology. Recognition of this cascade was essential to guiding appropriate vestibular testing modifications and effective canalith repositioning despite recent neurologic complications.

The audiovestibular symptoms observed in this patient can likely be explained by altered cerebrospinal fluid dynamics and their downstream effects on inner ear pressure regulation. The endolymphatic and perilymphatic compartments exist in a pressure-dependent equilibrium that is mediated by CSF through the cochlear aqueduct and endolymphatic

sac. According to the hydromechanical hypothesis, reductions in intracranial pressure are transmitted to the inner ear through passive fluid shifts, with the rate of transmission dependent on cochlear aqueduct patency [7]. In the setting of SIH, a rapid reduction in perilymphatic pressure can disrupt endolymphatic pressure homeostasis, resulting in secondary endolymphatic hydrops and symptoms that clinically resemble Menière's disease. Although she had a known history of bilateral Menière's disease, the onset of persistent positional vertigo following resolution of her acute neurologic symptoms suggested an additional peripheral vestibular process. Importantly, her positional symptoms were mechanically provoked, direction-specific, and reproducible, features that are more consistent with benign paroxysmal positional vertigo than with fluctuating Menière's-related vertigo [8,22]. This distinction was critical in supporting clinical decision making and targeted vestibular intervention despite the presence of significant neurologic comorbidities.

The development of BPPV in this case may not be a stand-alone idiopathic event. Secondary BPPV has been well described following inner ear insults such as head trauma, vestibular neuritis, labyrinthine ischemia, and otologic surgery [23]. However, additional variables including but not limited to vitamin D deficiency, hypertension, persistent inflammation, and prolonged bedrest may have contributed to BPPV onset as well [24]. In this patient, endolymphatic hydrops related to intracranial hypotension may have contributed to utricular dysfunction, increasing the likelihood of otoconial detachment. In Menière's disease, the inner ear repeatedly swells due to fluid buildup, a process known as hydropic distension [25]. Theories suggest that this swelling can cause otoconia to detach, increasing the likelihood of BPPV after a Menière's attack. Repeated episodes of inner ear swelling may also change the mechanical properties of the membranous labyrinth, making it less elastic. Additional proposed mechanisms include enlargement of the utricle and adherence of otoconia to the membranous labyrinth, both of which may further obstruct normal otoconia repositioning techniques [22]. These partial obstructions may persist even when Menière's symptoms are not active. In the presence of partial obstruction, canalith repositioning maneuvers become more difficult, though still possible. This may explain why BPPV associated with Menière's disease often responds more slowly or incompletely to standard repositioning treatments and tends to be more persistent than idiopathic BPPV [22,26]. This proposed mechanism is consistent with the observed horizontal canal involvement, which is more frequently reported in secondary BPPV than in idiopathic cases [22]. Recognition of secondary BPPV mechanisms is essential, as these presentations often occur in medically complex patients and may co-exist with central vestibular or neurologic pathology.

This case additionally highlights the importance of safely adapting vestibular examination procedures in the presence of medical precautions. Due to the patient's recent SDH and intracranial hypotension, Dix–Hallpike testing was deferred to minimize symptoms associated with rapid head and trunk movements. Instead, supine roll testing was selected as a less aggressive, more tolerable assessment to evaluate horizontal canal involvement while respecting medical restrictions. Conservative management of SIH typically includes strict bedrest and avoidance of prolonged upright positioning. Although the risk associated with rapid positional transitions between sitting and supine in individuals with SIH has not been established in the literature, the treating PT deferred these positions as a symptom mitigation strategy [27,28]. Furthermore, the conversion from geotropic to apogeotropic nystagmus during repositioning maneuvers required additional management and clinical decision making while still considering medical restrictions. These modifications underscore the importance of clinical reasoning and risk stratification in vestibular management, particularly when standard positional tests are not appropriate or demonstrate limited efficacy. Furthermore, they highlight the skill set of U.S.-trained physical therapists in the management of complex vestibular disorders.

The patient's functional impairments further supported the need for PT intervention. Her initial FGA score of 18/30 indicated impaired dynamic balance and elevated fall risk, consistent with her reports of positional vertigo and gait instability. In patients with vestibular pathology, an FGA score less than 22/30 is strongly associated with high risk of falling [29]. Following canalith repositioning maneuvers, her FGA score improved five points to 23/30 within one day, exceeding minimal clinically important difference values of four points reported in the literature and reflecting a meaningful improvement in functional mobility [18]. Although spontaneous neurologic recovery cannot be fully excluded given her recent intracranial pathology, the rapid resolution of positional symptoms and associated nystagmus following vestibular intervention supports a peripheral vestibular contribution that was responsive to PT intervention and indicates a favorable prognosis.

The clinical demands of this case highlight the importance of advanced vestibular training in the management of complex and evolving presentations. Accurate identification of secondary pathology, appropriate modification of positional testing, and safe implementation of canalith repositioning required integration of vestibular-specific knowledge with ongoing assessment of medical risk. While physical therapists routinely address balance, gait, and fall risk across healthcare systems, the addition of targeted vestibular training may enhance the ability to recognize and manage overlapping or sequential vestibular conditions. Given that this foundational skillset is already present in many settings, expansion of vestibular-specific competencies represents a practical opportunity to improve clinical decision making in similarly complex cases. These considerations also underscore the importance of integrating vestibular findings within the context of systemic and neurological pathology. Patients presenting with vestibular symptoms accompanied by intractable headache, recent neurologic change, or atypical symptom progression warrant heightened vigilance and interdisciplinary collaboration, including physical therapy involvement. While vestibular rehabilitation plays a critical role in managing peripheral dysfunction, timely recognition of red flags and appropriate referral remain essential components of comprehensive vestibular management.

4. Conclusions

This case highlights a unique occurrence of SIH, Menière's disease, and subsequent BPPV. This report demonstrates how SIH can generate a sequence of neurologic and vestibular findings that directly affect physical examination and management. The emergence of Menière's-like symptoms following SIH has rarely been reported in the literature, less so describing subsequent secondary BPPV in this context. The progression from altered CSF dynamics to hydropic symptoms and then positional vertigo highlights the need for careful, ongoing vestibular assessment in medically complex patients. Treating clinicians, whether physical therapists or other clinicians, must be prepared to modify positional testing when precautions are present and to distinguish fluctuating inner ear symptoms from mechanically provoked vertigo. Early identification and treatment of secondary BPPV in this setting led to rapid symptom resolution and measurable functional improvement, reinforcing the value of targeted vestibular intervention even when the underlying driver was of central origin.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/ohbm7010019/s1, CARE Checklist of information to include when writing a case report.

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Informed Consent Statement: Written informed consent has been obtained from the patient to publish this paper.

Data Availability Statement: The original contributions presented in this study are included in the article material. Further inquiries can be directed to the corresponding author.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

SIH	spontaneous intracranial hypotension
CSF	cerebrospinal fluid
SDH	subdural hematoma
U.S.	United States
BPPV	benign paroxysmal positional vertigo
PT	physical therapy
NIHSS	National Institutes of Health Stroke Scale
FGA	Functional Gait Assessment

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