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Case Report

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Hydrocephalus in a Patient with Alzheimer's Disease

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Conflict of Interest
The authors have no nancial con icts of interest.

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Conceptualization: Kang MJ, Yi SH; Data curation: Kang MJ, Park YH; Formal analysis: Kang MJ, Park YH, Kim SY, Yi SH; Investigation: Park YH, Kim SY; Methodology: Park YH, Kim SY; Project administration: Kang MJ, Yi SH; Resources: Park YH; Software: Yi SH; Supervision: Park YH, Kim SY; Validation: Park YH, Kim SY; Visualization: Kang MJ, Yi SH; Writing - original draft: Kang MJ, Yi SH; Writing - review & editing: Kang MJ, Yi SH.

ABSTRACT

Background: Normal pressure hydrocephalus (NPH) is an etiology of dementia that is reversible following cerebrospinal uid shunt placement, however, surgical intervention not always clinically e ective and the respons to shunt therapy is poorly understood. Furthermore, NPH is a source of comorbidity in diseases with neurodegenerative pathology, such as Alzheimer's disease (AD).
Case Report: A 61-year-old woman presented to the neurology clinic with progressive gait di culties and cognitive impairment over ve years. Nine years a er ventriculoperitoneal (VP) shunt treatment, the patient began to experience frequent falls. There was no improvement in clinical symptoms a er the alteration of valve pressure on the VP shunt. An ^{18}F- orbetaben amyloid positron emission tomography scan showed increased di usion uptake over the bilateral cortices, precuneus, and posterior cingulate cortex.
Conclusions: The patient of NPH was unresponsive to shunt therapy due to the development of AD.

Keywords: Hydrocephalus; Alzheimer Disease; Amyloid

INTRODUCTION

Normal pressure hydrocephalus (NPH), rst reported by Hakim and Adams[1] in 1965, manifests as a triad of gait disturbance, urinary incontinence, and dementia. Clinically, ventricular dilatation and normal cerebrospinal uid (CSF) pressure are observed. NPH is one of the few causes of dementia that is potentially reversible.[2]

NPH can be classi ed into idiopathic and secondary types. Secondary NPH is associated with known etiologies, such as trauma, hemorrhage, infection, mass lesions.[3] The rst line treatment is to reduce CSF pressure via shunt placement, which o en improves symptoms, in both secondary and idiopathic NPH. Furthermore, a reduction of CSF pressure via lumbar puncture can result in temporary improvement in symptoms.[4]

CSF shunt surgery does not always reduce the symptoms of NPH. The variable e ectiveness is poorly understood. However, co-morbid neurodegenerative pathologies, such as Alzheimer's disease (AD), may play an important role.[5] Indeed, several studies have shown the comorbidity of NPH and AD. [5-7]

Fig. 2. Axial images from amyloid imaging with ^{18}F-florbetaben.
^{18}F-florbetaben amyloid positron emission tomography scan showing increased diffusion uptake over the bilateral cortices, precuneus, and posterior cingulate cortex.

with normal-pressure hydrocephalus. The patient showed a marked improvement in gait after 50 mL of CSF was removed. Urinary incontinence and cognitive function showed improvement. Next, a VP shunt was inserted, which led to marked improvement in the patient's gait. Brain MRI was repeated after the operation, and no change in ventriculomegaly was noted.

Nine years after the operation, at the age of 70, the patient began to experience frequent falls and a fracture to the shoulder. She underwent shoulder surgery and experienced post-operative delirium with disorientation and aggression. Following shoulder surgery, her gait, cognitive function, and urinary symptoms worsened over 6 months. There was no change in symptoms after valve pressure alteration on the VP shunt. Brain MRI did not reveal any indication of significant changes in the hydrocephalus (**Fig. 1B**). However, neuropsychological assessment showed deficits in memory, frontal/executive, and language domains with an MMSE score of 26. Furthermore, amyloid imaging with ^{18}F-florbetaben positron emission tomography (PET) showed increased diffusion uptake in the bilateral cortices, precuneus, and the posterior cingulate cortex (**Fig. 2**). A diagnosis of AD was made and the anti-acetylcholinesterase inhibitor, donepezil, was prescribed to the patient.

DISSCUSSION

NPH is an important cause of treatable dementia, however, there is considerable confusion regarding the clinical and radiographic criteria necessary to ensure a satisfactory outcome with CSF shunting procedures.[8,9]

Estimates of the response rate to shunting in NPH vary widely.[10] Disappointing results are often obtained when patients with NPH cannot be differentiated from those with AD, prompting the need for studies to distinguish between NPH and AD.

Non-demented NPH patients show specific patterns of executive impairment in neuropsychological tests, distinct from the deficits observed in patients with mild AD.[11] Brain MRI reveals that the hippocampi are spared in NPH when compared with AD. Therefore,

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hippocampal volume is a potential diagnostic parameter to distinguish NPH from AD.[12] 18- uorodeoxyglucose PET scans show bilateral temporoparietal hypometabolism in patients with AD, while NPH patients show global hypometabolism.[13]

Several studies show patients with comorbid NPH and AD.[5-7] One study reported that 7 (33%) out of 21 patients with NPH exhibited neuropathological changes consistent with AD in cortical biopsy at the time of shunt placement.[6] In another study, 31% - 50% of patients with NPH displayed AD-like neuropathology in cortical samples obtained at shunt implantation.[7] A further study showed that 23 (42%) out of 55 patients who underwent biopsy at the time of shunt placement for NPH met the criteria for AD.[5]

Evidence suggests that CSF secretion and turnover clears toxic molecules, such as amyloid-peptides (Aβ), from the interstitial uid space of the brain into the bloodstream. In NPH, elevated CSF pressure lowers CSF production resulting in less clearance of Aβ.[14]

In conclusion, we found that a lack of shunt response in our patient diagnosed with NPH was accompanied by AD pathology. Furthermore, noninvasive techniques that detect AD pathology, such as amyloid PET, facilitate the evaluation of NPH.

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