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Letter to the Editor

The Impact of Comorbid Alzheimer's Disease in a Patient with Normal Pressure Hydrocephalus?

Halil Onder

Neurology Clinic, Yozgat City Hospital, Yozgat, Turkey

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Correspondence to
Halil Onder
Neurology Clinic, Yozgat City Hospital, Erdoğan Akdağ Mah., Viyana Cad., Yozgat 66100, Turkey.
E-mail: halilnder@yahoo.com

ORCID iDs
Halil Onder
https://orcid.org/0000-0002-1823-2278

Conflict of Interest
The author has no financial conflicts of interest.

Dear Sir,

I read with great interest the article by Kang et al.[1] in which they describe a patient with normal pressure hydrocephalus (NPH) who had clinically worsened 9 years after shunt surgery and was evaluated as unresponsive to ventriculoperitoneal shunt (VP shunt) modification. Further investigation of 18F-florbetaben positron emission tomography showed extensively increased diffusion uptake supporting the diagnosis of Alzheimer's disease (AD). Based on this illustration, the authors discuss the importance of differentiating between NPH and AD to avoid disappointing results and remark AD as crucial comorbidity in NPH that can lead to shunt unresponsiveness.

I appreciate the authors for presenting the long-term follow-up of this patient and conducting a clear discussion. However, I would like to comment on the article hoping to provide a better understanding of the report and related issue. Basically, I think that the clinical presentation of the patient should be documented in more detail. The onset time of clinical worsening after nine years from VP shunt surgery is not clearly stated. However, they mention deterioration in gait, cognitive function, and urinary symptoms over 6-months following surgery for a shoulder fracture. I agree with that gait and urinary incontinence are symptoms those can also be encountered in other dementia subtypes like AD,[2,3] other than NPH. However, AD is a disorder primarily characterized by memory loss and disturbances of other cognitive abilities. As I understand from the report, the forefront clinical manifestation in this patient was gait problems including frequent falling. The results of 18F-florbetaben positron emission tomography support the presence of AD pathology. However, it is difficult to prove the presence of symptomatic phase of AD based on the presented clinical data. Thus, the authors should include results of additional cognitive tests to clarify this point. In my opinion, the detailed description of the phenomenology of gait at final presentation (was it characterized by broad-based, short-step gait?) may aid substantially to our understanding the responsible mechanisms of deterioration. Otherwise, still, another crucial possibility may be the malfunction of VP shunt as the responsible mechanism from clinical deterioration. They state that brain magnetic resonance imaging (MRI) did not reveal indications of significant changes in the hydrocephalus. Actually, we expect recovery of hydrocephalus following shunt surgery, also the mechanism of recovery in these patients. Thus, comparing the final MRI (which shows significant hydrocephalus!) with MRI, recorded nine years before shunt surgery, and ignoring the potential impact of hydrocephalus based on this

unremarkable comparison result would be irrational. Hence, I strongly suggest to comparing the final MRI with an MRI recorded on the interval period (at recovery-phase), if available. Besides, the authors state that performing valve pressure alteration on the VP shunt did not yield an improvement in symptoms. However, how they can be sure of that VP shunt is intact. Did they perform a cerebrospinal fluid (CSF)-tap test to confirm the unresponsiveness of CSF diversion? Finally, I wonder if the authors may involve the impact of donepezil treatment in symptoms of the patient one-by-one which would also give crucial perspectives regarding the responsible mechanisms of the clinic in this remarkable patient.

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