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Letter to the Editor

Letter regarding the article entitled 'Is frontal gait a myth in normal pressure hydrocephalus?'

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Letter to Editor,

I read with great interest the article by Morel et al. in which they compare the specific gait parameters in patient groups of idiopathic normal pressure hydrocephalus (iNPH) and its mimics [1]. In the result of their study, they emphasized that frontal gait was not the most prevalent gait abnormality in iNPH and it could not discriminate iNPH from its mimics. I appreciate the authors for conducting this original study and remarking crucial conclusions which may give important applications in the clinical grounds. However, there may be some additional points to be discussed for a better understanding of the study results.

Remarkably, a major question may be that if there was a prognostic difference including treatment (shunt) responsiveness between iNPH patients with frontal gait abnormality, and iNPH patients presenting with other gait features? In a crucial report by Bugalho et al., frontal signs and disequilibrium were found to be more frequent in iNPH patients those nonresponsive to shunt-surgery, whereas hypokinesia responded significantly to cerebrospinal fluid (CSF) diversion [2]. In conclusion of their study, they suggested the existence of two principal components of gait disturbance in iNPH: hypokinesia, possibly related to basal ganglia dysfunction (reflecting parkinsonian characteristics), and disequilibrium, probably related to frontal disturbance [2]. Ergo, I wonder if the authors might give further data regarding the follow-up of these patients with iNPH, particularly including the comparisons between distinct subgroups according to the characteristics of gait. I think that these data might provide contributions regarding our understanding of distinct symptoms of iNPH and their pathophysiology.

Another critical point may be that the authors state that they had excluded the effect of comorbidities using logistic regression models. However, this method can be interrogated in multiple aspects. For instance, while comparing the symptoms between the groups of iNPH and vascular dementia (that has been emphasized to be frequently overlapping with vascular parkinsonism [3]), excluding the effect of subcortical lesion burden which is a known as an etiological agent inducing the clinic of vascular parkinsonism, would be irrational. Besides, I am not sure that the effect of a previous stroke in these patients may be excluded in these analyses, as clinical output may be completely different according to the function of regions, affected by the ischemic lesion (while some of them including other than motor areas might not influence locomotion, the lesions of the motor areas and basal ganglia would result in severe disturbances in locomotion). At this point, it would be remarkable to state that the most prevalent rate of gait in iNPH group was the subgroup of others (30%), consisting of hemiparetic gait, ataxic gait, which potentially might be caused by a possible stroke. Taken together, I wonder if the authors would include the results of analyses comparing gait of iNPH mimics with at least iNPH patient subgroup without a previous stroke. I think that this comparison, allowing a stronger exclusion of the possible effect of the previous strokes in the current study results, would surely provide crucial contributions in this regard.

The authors also mention the absence of data about the rate of patients treated with shunt [1] and they refer to a previous study reporting that two-thirds of the patients diagnosed with iNPH at baseline tend to be diagnosed with an alternate condition in the three following years [4]. In my opinion, this point may be strictly a major limitation of the study. Neurodegenerative comorbidities have recently been reported frequently in patient with NPH [4]. Such that, Espay et al. suggested a terminology as 'neurodegenerative normal pressure hydrocephalus' to define these patient subgroups. At this point, I think that the exclusion of NPH patients with neurodegenerative pathologies is strictly important for obtaining reliable results for further deliberations. The authors state that they have made the diagnosis according to the international NPH consensus guidelines criteria, however, this is certainly insufficient to exclude these variants. Moreover, they do not indicate if the group consisted of the diagnosis of probable or possible iNPH? Besides, in this classification, response to CSF TAP test is not included which has been emphasized as the gold standard diagnostic method for NPH in the recent publications [4]. Therefore, the diagnostic accuracy of the patients included may be interrogated in light of our current knowledge. Another remarkable point was that there was an extremely high rate of patients which were evaluated as having normal gait in iNPH group (29%) as well as mimics group (which was interpreted as 'minority of patients' by Morel et al.?). The authors mention that they have included patients at a very early stage to improve the early management of these conditions. However; gait impairment, besides being a cardinal finding, is generally postulated as the initial manifestation of iNPH [5]. At this point, I wonder how the authors explain these high rates of evaluation results of normal gait? Might subtle changes, which had been stated by the patients, be missed

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by the clinicians? If so, it would be also irrational to emphasize a conclusion like that the clinical gait examination can be easily performed during the standard neurological assessment and better fits to the daily reality of clinicians (they also emphasize to the disadvantages of a spatiotemporal gait analysis requiring a time-consuming assessment and expansive equipment).

Remarkably, there was also no a difference in terms of cognition and urinary incontinence scales between patient groups of iNPH and mimics ($p = .319$, $p = .577$; respectively) which might be another devastating result of this study. I think these results with nonsignificant analyses results of frontal gait in discriminating iNPH from its mimics further draw attention to the challenges regarding the clinical diagnosis of these patients.

In conclusion, although the discussion above need to be clarified, the results of this study may provide crucial perspectives about the clinical evaluation processes of patients with iNPH. However, these discussions are surely warranted to be clarified in the large-scale future reports. The confirmation of these results in future reports may support a consideration of the need for new paraclinical tests to overcome the diagnostic challenges of iNPH. The results of these studies may also provide substantial contributions regarding the unknown aspects of iNPH pathophysiology.

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Declaration of Competing Interest

None.

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Halil Onder
Neurology Clinic, Yozgat City Hospital, Yozgat, Turkey
E-mail addresses: halilnder@yahoo.com, halil.onder@hacettepe.edu.tr.