WriteClick: Editor's Choice

Editors' Note: In this week's WriteClick, O'Callaghan and Hornberger point out that there are studies that contradict the findings of authors Weintraub et al. that dopaminergic therapy in Parkinson disease (PD) is the driver behind impulse control disorders rather than the disease itself. They suggest a compromise theory and more objective screening. The authors disagree on both points.

Megan Alcauskas, MD, and Robert C. Griggs, MD

SCREENING FOR IMPULSE CONTROL SYMPTOMS IN PATIENTS WITH DE NOVO PARKINSON DISEASE: A CASE-CONTROL STUDY

Claire O'Callaghan, Michael Hornberger, Sydney, Australia: Weintraub et al.[1] found that de novo PD itself does not confer an increased risk for impulse control disorders (ICDs). This implies that dopaminergic therapy is the critical factor driving ICDs in PD.

However, other studies have shown that patients with PD—even without ICDs—perform more impulsively than controls on laboratory-based tasks.[2,3] Similarly, gray matter atrophy in impulse-control regions (nucleus accumbens, orbitofrontal cortex) correlates with inhibitory-control failures in PD,[4] suggesting that increased impulsivity may not only be dependent on medication status but also on neuroanatomical abnormalities intrinsic to PD. It is possible that there is a clinical PD subgroup with an impulsivity endophenotype[5] due to neuroanatomical/neurochemical abnormalities in impulse-control brain regions, which could be further aggravated via dopamine therapy.

Identifying this potential clinical subgroup should be the target of future investigations of patients with de novo PD. In particular, targeted imaging of impulse-control brain regions and employment of more objective impulse-control measures should be investigated because these can be more sensitive than self-completed screening questionnaires. This would allow identification of patients who are more vulnerable to developing ICDs later in the disease course or with initiation of dopaminergic therapy.

Author Response: Daniel Weintraub, Andrew Siderowf, Kimberly Papay, Philadelphia: We thank Drs. O'Callaghan and Hornberger for their interest in our recent article, where we demonstrate that clinical impulse disorders are not more common in patients with untreated de novo PD than in controls.

We agree that identification of a biological substrate for ICDs is an important area for research and that structural changes on MRI are a potential biomarker for ICD risk. We also agree that some studies show abnormalities in laboratory-based tests of impulsiveness in patients with untreated PD. However, this finding is not consistent across all studies.[6] In addition, changes within laboratory-based tasks will not always predict the occurrence of ICD behaviors that are important to patients with PD.

Based on our study, and prior research,[7] we believe that treatment with dopamine agonist medications is the most important risk factor for development of ICDs in PD and that clinically meaningful ICDs occur in patients with untreated PD as frequently as they occur in the general public. Moreover, the results of validation studies support the Questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease (QUIP) as a useful screening tool for clinically relevant ICDs. The QUIP is highly sensitive and brief enough to be administered in a busy office practice.[8]

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CLINICAL COURSE OF IDIOPATHIC INTRACRANIAL HYPERTENSION WITH TRANSVERSE SINUS STENOSIS

Francesco Bono, Aldo Quattrone, Catanzaro, Italy: Riggeal et al.[1] found no correlation between the degree of transverse sinus stenosis (TSS) and the clinical course in idiopathic intracranial hypertension (IIH). There is no direct evidence of normalization of the CSF pressure in this series and this demonstration is needed to support their observation. The authors did not cite our studies[2,3] that may confirm their findings. We studied 14 consecutive patients with IIH over a 6-year period. At presentation and during follow-up, patients underwent CSF pressure measurements and magnetic resonance venography. TSS persisted after normalization of the CSF pressure in 9 patients with a good clinical course, suggesting the lack of a direct relationship between the caliber of TSS and CSF pressure in IIH. Moreover, unilateral TSS was observed in 30% of 111 subjects with normal CSF pressure, whereas bilateral TSS occurred in only 1.8% of individuals.[3] Our observations provide evidence that bilateral TSS is one of the factors contributing to IIH. Our findings support the observations of Riggeal et al. and suggest that both clinical course and CSF pressure should determine the management of patients with IIH in clinical practice.

Author Response: Beau B. Bruce, Nancy J. Newman, Valerie Biousse, Atlanta: We appreciate the contributions of Drs. Bono and Quattrone and their support of our conclusions. We also found that bilateral TSS occurs rarely in patients with normal CSF opening pressure.[4] Concerning IIH, clinical management in our practice is guided by visual function and symptom severity; we usually obtain lumbar puncture only at the time of diagnosis unless there is diagnostic uncertainty or evidence of worsening. Therefore, it is likely that our results will be useful to others who practice in a similar fashion, even though we did not demonstrate normalization of the CSF opening pressure. In addition, as we reported,[1] there was a trend toward higher initial CSF opening pressure predicting both a poor clinical course and greater visual field loss, even though there was no association between TSS and CSF opening pressure and no association between TSS and clinical course. The association between CSF opening pressure and clinical course in our data reinforces the suggestion by Drs. Bono and Quattrone that CSF opening pressure should be considered in the management of patients with IIH.

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Screening for impulse control symptoms in patients with de novo Parkinson disease: A case-control study

Claire O'Callaghan, Daniel Weintraub, Michael Hornberger, et al.

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