idiopathic intracranial hypertension (IIH). We would like to add the following case to reports of patients presenting chronic daily headache with elevated lumbar puncture (LP) opening pressure without papilledema.[2,3]

We saw an obese woman with global headache associated with nausea and back pain that began 1 month earlier. Brain MRI/venography was normal, papilledema was not detected, and optic/ocular examinations were normal. LP revealed an opening pressure of 177 mm with normal CSF contents. Amitriptyline was initiated for chronic daily headache, the patient was advised to lose weight, and improvement was noted.

Six months later, the patient complained of severe, global, nonsteroidal anti-inflammatory drug–resistant headache taking place over the previous 4 weeks. She showed a 12-pound weight gain. The repeated brain MRI/venography was normal and we did not detect papilledema or optic/ocular abnormalities. LP documented an opening pressure of 313 mm with normal CSF contents. Treatment with acetazolamide was initiated and the symptoms resolved.

The revised criteria[1] did not support an IIH diagnosis; yet it is possible that our patient could have had IIH with a progressive evolution. Development of papilledema is possible. We suggest that patients presenting clinical conditions similar to our case could be diagnosed with probable IIH requiring vigilant follow-up.

Author Response: Deborah Friedman, Dallas; Kathleen Digre, Salt Lake City; Grant Liu, Philadelphia: We thank Dr. Liguori et al. for their comments and appreciate that there will always be occasional exceptions to any rule. However, in our experience, patients who are diagnosed with pseudotumor cerebri syndrome (PTCS) on the basis of CSF pressure alone—without other objective findings—rarely have the disorder. We have seen devastating consequences in patients who were diagnosed incorrectly and subjected to unnecessary medications and surgical procedures that carry significant risk. The LP opening pressure is a snapshot in time and may be influenced by the presence of cephalgia and other technical factors. The therapeutic response to CSF removal and acetazolamide are not specific to PTCS.

1. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 2013;81:1159–1165.
2. Marcelis J, Silberstein SD. Idiopathic intracranial hypertension without papilledema. Arch Neurol 1991;48:392–399.
3. Wang SJ, Silberstein SD, Patterson S, et al. Idiopathic intracranial hypertension without papilledema: a case-control study in a headache center. Neurology 1998;51:245–249.

CORRECTION

***C9orf72* expansions are the most common genetic cause of Huntington disease phenocopies**

In the article "*C9orf72* expansions are the most common genetic cause of Huntington disease phenocopies" by D.J. Hensman Moss et al. (*Neurology*® 2014;82:292–299), there is an omission in the Methods section under the subheading "Case ascertainment." The third paragraph should read "All *C9orf72*-positive cases were given a modified Goldman score,[21,22] which was used to quantify the strength of the autosomal dominant family history (scoring was modified to give a score of 0 for no data, 4 for definitely no family history, and 4.5 for unknown or undescribed family history)." The full scoring system has been added to the supplemental data. The authors regret the omission.

Author disclosures are available upon request (journal@neurology.org).

C9orf72 expansions are the most common genetic cause of Huntington disease phenocopies

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