

Obsessive-compulsive Disorders Due to Neuroacanthocytosis Treated with Citalopram

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Habermeyer et al. have reported the interesting case of a 32-year-old man with a 9 year history of chorea, obsessive-compulsive disorder, striatal atrophy and acanthocytosis that they identify as an instance of "neuroacanthocytosis" [1]. Neuroacanthocytosis, as they rightly state, is a heterogeneous syndrome [2,5], which can, however, be clearly split into genetically distinct diseases [3,5].

We would recommend an investigation of X-linked McLeod syndrome by Kell phenotyping, and the performance of a chorein Western blot [4] to evaluate for the alternative diagnosis of autosomal recessive chorea-acanthocytosis. With such additional details, their interesting therapeutic observation will be more valuable for the development of future treatment protocols.

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Erratum

The publishers regret to announce that the Letter to Editor from Walker and Danek was inadvertently omitted in the last issue of *Pharmacopsychiatry*. In this issue we publish both, the letter to the editor from Walker and Danek and the authors' reply from Habermeyer and Fuhr (*Pharmacopsychiatry* 2007; 40: 82–87) again as erratum with the correct received and accepted dates.

Authors' Reply Obsessive-compulsive Disorders Due to Neuroacanthocytosis Treated with Citalopram

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We thank R. Walker and A. Danek for their letter emphasizing that the term neuroacanthocytosis is used for distinct diseases, which can be differentiated by means of genetic tests. Our patient we described [2] suffered from chorea-acanthocytosis. The most prominent differential diagnosis of chorea-acanthocytosis is the McLeod syndrome, which shows a reduction of all Kell antigens and absence of Kx antigen [1,3]. In our patient no abnormalities of Kell antigens was detected; therefore, this important differential diagnosis can be excluded.

In addition to oral and perioral dyskinesias with self-mutilation of mucosa and lips, the patient had additional findings, including atrophy of caudate nuclei in brain MRI, clinical and electrophysiological signs of peripheral neuropathy, an elevated creatinine kinase and liver abnormalities (with normal serologic tests for viral causes and unspecific biopsy result). Lipoproteins were normal as was coeruloplasmin level and copper excretion. The patient had no seizures, and EEG was normal.

While all of this may be found in both chorea-acanthocytosis and McLeod Syndrome, the whole spectrum might be rarely present in other disorders. Serologic exclusion of McLeod syndrome led us to accept the diagnosis of chorea-acanthocytosis, especially, since in our patient the most impressive presenting symptom was his orofacial and lingual dyskinesias [4].

References

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