

regimen. One month later (after a *grand-mal* seizure), she was diagnosed with syndrome of inappropriate antidiuretic hormone secretion (SIADH), that resolved after the discontinuation of the amantadine (association of amantadine with SIADH has been reported in 2 patients⁴). Ropinirole was added at a dose of 0.75 mg per day. For more than 3 years, the patient has remained on the same regimen with over 90% improvement in her OBL dyskinesias while displaying a mild extrapyramidal syndrome consisting of mild bradykinesia and minimal postural tremor.

There are a very few reports covering the medical therapies for CAHD. Liver transplantation,⁵ lactulose diet,² and branched-chain amino acid therapy³ have been associated with neurological improvement in CAHD. There is also very limited information on symptomatic therapy for the movement disorder component of the CAHD. Successful use of levodopa with for unilateral rest tremor, bradykinesia, and rigidity has been reported in a patient that subsequently developed dyskinesias.² In a report on 11 patients with Parkinsonism due to CAHD, only 2 were treated with L-dopa with relative improvement.⁶

There is no information regarding symptomatic treatment of hyperkinetic manifestations—other than tremor—of CAHD. Our own experience suggests that use of potent dopamine blockers such as haloperidol would be fraught with eventual re-emergence of OBL dyskinesias in addition to induction or exacerbation of parkinsonian features.

We decided to use tetrabenazine, a monoamine depletor, because of its reported success in the treatment of a various hyperkinetic movement disorders including Huntington's disease, Tourette syndrome, and dystonia.⁷ Although tetrabenazine proved effective in the treatment of our patient's OBL dyskinesias, our patient experienced mild parkinsonian side effects (well described in the literature⁷) successfully treated with both amantadine and later ropinirole. L-Dopa was not tried, to minimize the chance of exacerbation of the OBL dyskinesias.

In summary, we report the successful use of tetrabenazine combined with a dopamine agonist for the symptomatic treatment of CAHD and suggest it could be considered in lieu of L-dopa to avoid triggering or exacerbating dyskinesias.

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References

1. Victor M, Adams RD, Cole M. The acquired (non-Wilsonian) type of chronic hepatocerebral degeneration. *Medicine (Baltimore)* 1965; 44:345–396.
2. Jog MS, Lang AE. Chronic acquired hepatocerebral degeneration: case reports and new insights. *Mov Disord* 1995;10:714–722.
3. Ueki Y, Iozaki E, Miyazaki Y, et al. Clinical and neuroradiological improvement in chronic acquired hepatocerebral degeneration after branched-chain amino acid therapy. *Acta Neurol Scand* 2002;106: 113–116.
4. van Laar T, Lammers GJ, Roos RA, Gerritsen JJ, Meinders AE. Antiparkinsonian drugs causing inappropriate antidiuretic hormone secretion. *Mov Disord* 1998;13:176–178.
5. Lewis M, Howdle PD. The neurology of liver failure. *QJM* 2003; 96:623–633.
6. Burkhard PR, Delavelle J, Du Pasquier R, Spahr L. Chronic parkinsonism associated with cirrhosis: a distinct subset of acquired hepatocerebral degeneration. *Arch Neurol* 2003;60:521–528.
7. Jankovic J, Beach J. Long-term effects of tetrabenazine in hyperkinetic movement disorders. *Neurology* 1997;48:358–362.

Erratum

Retrospective Evaluation of the Dose of Dysport and BOTOX in the Management of Cervical Dystonia and Blepharospasm: The REAL DOSE study

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In the article by Marchetti and colleagues, Dr. Evžen Růžička's name was misspelled. It is reprinted correctly here.

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