WriteClick: Editor's Choice

Editors' Note: In WriteClick this week, the authors of "Evidence-based guideline: Treatment of tardive syndromes," Bhidayasiri et al., address the concerns of Lerner et al. regarding inclusion of articles in evidence-based guideline analysis and further clarify the American Academy of Neurology's classification scheme for rating therapeutic articles. Brenner and author DiMauro discuss mitochondrial DNA dysfunction in neurodegenerative disorders without typical mendelian inheritance patterns.

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

EVIDENCE-BASED GUIDELINE: TREATMENT OF TARDIVE SYNDROMES: REPORT OF THE GUIDELINE DEVELOPMENT SUBCOMMITTEE OF THE AMERICAN ACADEMY OF NEUROLOGY

Vladimir Lerner, Chanoch Miodownik, Be'er Sheva, Israel: We read the article by Bhidayasiri et al.[1] with interest. We found some inaccuracies. The authors cited only one of our articles regarding treatment of tardive dyskinesia (TD).[2] Even though the authors performed a search from 1966 to 2011, they did not include 2 studies published in 2007 that could broaden the knowledge about new options of TD management.[3,4] The first deals with vitamin B_6 and the other with piracetam. Both studies include greater samples (50 and 40 subjects, respectively) and could be considered Class I evidence according to the authors' classification. Inclusion of this information could positively influence the weight and emphasize the significance of these medications. Our experience shows that different types of TD react uniquely to different types of medications.

Author Response: Roongroj Bhidayasiri, Bangkok, Thailand; Stanley Fahn, New York; Gary S. Gronseth, Kansas City, KS; Kelly L. Sullivan, Theresa A. Zesiewicz, Tampa, FL: We thank Lerner et al. for their comments and interest in our article.[1] We agree that various forms of tardive syndromes (TDS) can respond differently to medications or interventions. Well-designed randomized controlled trials with specific TDS inclusion criteria/subtypes are necessary to determine the most effective interventions for TDS symptoms. As Lerner et al. noted, 2 studies on vitamin B_6 and piracetam were not included in our original analysis.[3,4] The first study, a double-blind, placebo-controlled trial on vitamin B_6 treatment in 50 inpatients with schizophrenia/schizoaffective disorders, was rated Class III for no allocation concealment and a >20% (14/50) dropout rate.[3] The second study, involving 40 patients with schizophrenic/schizoaffective disorders who received piracetam or placebo over 4 weeks, was rated Class III for dropout rate.[4] After applying the AAN's classification scheme for rating therapeutic articles,[5] the data are still insufficient to support or refute use of vitamin B_6 and piracetam for TDS. Although the AAN endeavors to find all pertinent literature,[5] no literature search is 100% effective, and we thank the authors for alerting us to these 2 studies. We corrected errors for an article cited in table e-1 and this table is updated online: http://www.neurology.org/content/81/5/463/suppl/DC1.[1]

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2. Lerner V, Miodownik C, Kaptsan A, et al. Vitamin B6 in the treatment of tardive dyskinesia: a double-blind, placebo-controlled, crossover study. Am J Psychiatry 2001;158:1511–1514.
3. Lerner V, Miodownik C, Kaptsan A, et al. Vitamin B6 treatment for tardive dyskinesia: a randomized, double-blind, placebo-controlled, crossover study. J Clin Psychiatry 2007;68:1648–1654.
4. Libov I, Miodownik C, Bersudsky Y, Dwolatzky T, Lerner V. Efficacy of piracetam in the treatment of tardive dyskinesia in schizophrenic patients: a randomized, double-blind, placebo-controlled crossover study. J Clin Psychiatry 2007;68:1031–1037.
5. Gronseth GS, Woodroffe LM, Getchius TSD. Clinical Practice Guideline Process Manual. St. Paul, MN: The American Academy of Neurology; 2011.

MITOCHONDRIAL ENCEPHALOMYOPATHIES—FIFTY YEARS ON: THE ROBERT WARTENBERG LECTURE

Steven R. Brenner, St. Louis: I read the article on mitochondrial diseases with interest.[1] Multiple sclerosis,[2] Alzheimer disease,[3] and Parkinson disease[4] have an epidemiologic maternal effect. However, the cause is still unclear except in a minority of familial cases with mendelian inheritance. Perhaps the

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