HEAD NODDING SEIZURES AND O.VOLVULUS INFESTATION

Head nodding (HN) syndrome, a new epilepsy disorder in sub-Saharan Africa, is described in 62 patients studied prospectively at the University of Ulm, Germany; Haydom Lutheran Hospital, Tanzania; and other centers in Austria, Tanzania, and Canada. The onset of HN attacks was at 6 to 10 years in 50% cases, and at 11-15 years in 37%. At the time of diagnosis and evaluation, most patients were between 11 and 15 years of age. Twenty-eight (45%) patients had only HN attacks, and 28 had HN plus one other type of seizure, usually generalized or partial complex. HN was associated with loss of neck tone, and 37 (60%) patients had additional loss of tone of upper extremities. Consciousness was impaired in 11 (18%). Food was a provoking factor in 9 patients, and bathing in cold water caused HN in 2. A family history of epilepsy was present in 90%. EEGs in 10 patients were normal in 4 and showed abnormal slowing in 6, with sharp waves in 2. MRIs in 12 patients were normal in 4, and showed hippocampal sclerosis in 5 and gliotic changes in 5. Thirty-one (61%) of 51 patients had microfilariae visible on microscopic examination of the skin. Traces of Onchocerca volvulus DNA in the skin were identified by PCR in 12 of 20 without microscopically visible microfilariae. Skin PCR positivity was significantly associated with MRI abnormalities. Neutrophil counts were elevated in 14 (27%) patients and eosinophils in 28 (55%). O. volvulus serum ELISA test was positive in 44 (86%). CSF PCR was negative in all patients. HN seizures were 50% controlled by conventional antiepileptic drugs. (Winkler AS, Friedrich K, Konig R, et al. The head nodding syndrome – clinical classification and possible causes. Epilepsia Dec 2008;49:2008-2015). (Respond: Dr Andrea S Winkler, Department of Neurology, University of Ulm, Oberer Eselsberg 45, 89081 Ulm, Germany. E-mail: drawinkler@yahoo.com.au).

COMMENT. The prevalence of epilepsy is reportedly higher in areas where onchocerciasis is endemic, eg Mexico, Sudan, Uganda, Tanzania, and S America, but meta-analysis fails to show a significant association between O. volvulus and epilepsy (Druet-
The cause of the head nodding epilepsy syndrome in Tanzania and Sudan remains unclear. Alternative explanations offered include hippocampal sclerosis, and genetic susceptibility. Evidence of CNS invasion by *O. volvulus* was not supported by CSF PCR tests.

Onchocerciasis (River Blindness, Filariasis) involves skin, subcutaneous tissue, lymphatic vessels and the eyes. The AAP Red Book, 27th ed. 2006 makes no mention of CNS invasion or epilepsy as a complication of onchocerciasis. Ivermectin, a microfilarial agent, and doxycycline are the drugs of choice for treatment of the infestation. Only anticonvulsant drug treatment is discussed in the article on HN syndrome. The effect of treatment with ivermectin on seizure frequency might be of interest.

**CARBAMAZEPINE-INDUCED HYPERSENSITIVITY SYNDROME AND ROLE OF HHV-6 REACTIVATION**

A 14-year-old Japanese boy with localization-related epilepsy and carbamazepine (CBZ)-induced hypersensitivity syndrome is reported from Ehime University School of Medicine, Japan. He developed a maculopapular rash and low-grade fever after 3 weeks of CBZ therapy. CBZ was discontinued and systemic corticosteroid (1 mg/kg/day) started. The rash spread to become diffuse, WBC increased with 19% atypical lymphocytes and 24% eosinophils. Improvement started on day 11, but relapse followed on day 15 with high fever, purpura, abdominal discomfort and liver dysfunction. AST and ALT were markedly elevated. On day 19, blood PCR was positive for HHV-6 DNA, and HHV-6 was isolated from peripheral blood mononuclear cells. On day 26, the anti-HHV-6 immunoglobulin G (IgG) titer was increased by 5,120-fold. Symptoms gradually subsided, and corticosteroid was discontinued without sequelae. Seizures did not recur during a 6-month follow-up and alternative anticonvulsant therapy was not required. (Suzuki Y, Fukuda M, Tohyama M, Ishikawa M, Yasukawa M, Ishii E. Carbamazepine-induced drug-induced hypersensitivity syndrome in a 14-year-old Japanese boy. *Epilepsia* Dec 2008;49:2118-2121). (Respond: Dr Yuka Suzuki, Department of Pediatrics, Ehime University School of Medicine, Shitsukawa, Toon, Ehime 7910295, Japan. E-mail: yusuzuki@m.ehime-u.ac.jp).

COMMENT. The treatment of anticonvulant drug-induced hypersensitivity syndrome (DIHS) is controversial, except for the discontinuance of the drug. The association with HHV-6 reactivation may discourage the use of immunosuppressive therapy that may worsen the infectious complications of DIHS. Alternative treatments, especially in patients with liver dysfunction, include immunoglobulin and plasmapharesis. The successful use of N-acetylcysteine and intravenous immunoglobulin is reported in an adult with DIHS and liver dysfunction induced by phenytoin. (Cumbo-Nacheli G, Weinberger J, Alkhalil M, Thati N, Baptist AP. Anticonvulsant hypersensitivity syndrome: Is there a role for immunomodulation? *Epilepsia* Dec 2008;49:2108-2112).