Neurocysticercosis: Factors predicting seizure recurrence

Carpio and Hauser found a high risk for seizure recurrence (40.3%) following a first seizure in people with newly identified neurocysticercosis. Among several variables, including antihelminthic treatment, only persistent lesions on CT predicted seizure recurrence. Antiepileptic drug should be continued until lesions clear on CT.

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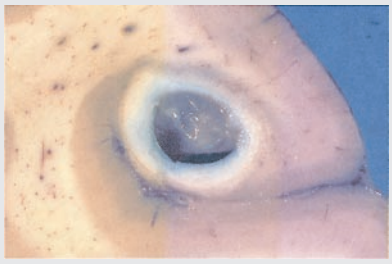
Neurocysticercosis and seizures: Avoiding the cost of antihelminthic treatment

Commentary by Larry E. Davis, MD

The prevalence of neurocysticercosis in the United States is rising as a result of increasing immigration from Latin America where neurocysticercosis is endemic, particularly in rural areas. The disease results from consumption of eggs (ova) of the tapeworm *Taenia solium* that are shed in the stool of individuals infected with the tapeworm.[1,2] Following consumption of uncooked egg–contaminated food, the ova transform in the gastrointestinal tract to a oncospheres that penetrate the gastrointestinal wall, circulate in blood, usually lodge in small vessels near the cerebral gray-white matter junction, and mature into cysticercosis cysts. For 2 to 10+ years, the viable cyst produces little inflammation around the cyst bladder wall and only rare clinical symptoms. As the cyst degenerates, proteins leak from the bladder, eliciting an inflammatory response. Degenerating cysts produce simple partial or secondarily generalized seizures in 50% to 90% of patients, presumably triggered by the gray matter inflammation.[1] Inflammation subsides when the cyst completely dies, becoming a



small, fibrotic, often calcified nodule. The time from degeneration to complete cyst death takes up to 2 years. Following death of all cysts, provoked seizures disappear, leaving a smaller percent of patients with residual epilepsy.

A controversy exists in patients with symptomatic neurocysticercosis: whether treating the degenerating cysts with antihelminthic drugs reduces the incidence of recurrent seizures or shortens the duration of provoked seizures. The argument for antihelminthic treatment is based on shortening the time to cyst disappearance on CT and that all cysts simultaneously die, preventing prolongation of brain inflammation due to multiple cysts degenerating at different times.

The Carpio and Hauser prospective study of patients with a first seizure and neurocysticercosis on CT showed that recurrent seizures were associated with persistence of active brain lesions seen on follow-up CT. However, treating patients with albendazole, an effective antihelminthic drug, did not reduce the risk of seizure recurrence. A recent Cochrane review also found that antihelminthic drugs did not significantly improve seizure outcome.[3] Hence, this and other studies support avoiding the cost of treating all symptomatic neurocysticercosis patients with antihelminthic drugs.

References

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Antibody-negative MG

Plested et al. show that 8 of 12 AChR antibody-negative plasmas from patients with MG transiently inhibit AChR function, probably via an indirect mechanism that involved AChR phosphorylation and desensitization. The inhibitory activity may be an IgM antibody and does not correlate with the IgG antibodies to MuSK recently described by the same group.

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Lambert-Eaton myasthenic syndrome

The Nakao et al. paper is remarkable for the number of cases (110) since LEMS is only one tenth as frequent as MG. Eighty-five percent of LEMS cases are seropositive. Passive transfer studies with serum that does not contain antibodies to the P/Q voltage-gated calcium channel support a still-to-be defined antibody as the cause of LEMS.

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The editorial by Abicht and Lochmüller considers the implications of these two papers on seronegative but presumably antibody-mediated neuromuscular junction disorders. MG is associated with antibodies to AchR in 80% of cases, antibodies to MuSK in 10% to 15% and this new factor—possibly an IgM antibody. Early evidence suggests that treatment (e.g., thymectomy) may be different depending on which antibody is present.

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Restless legs syndrome after spinal anesthesia

Högl et al. evaluated 202 consecutive patients undergoing various types of surgery under spinal anesthesia and of these patients, 8.7% developed transient RLS. Low MCV and MCH were associated with the occurrence of new-onset RLS.

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Sturge-Weber syndrome: Hemispherectomy for intractable epilepsy

Kossoff et al. studied 32 children worldwide who underwent hemispherectomy for Sturge-Weber syndrome. These children had intractable epilepsy starting at a mean age of 4 months. Currently 81% are seizure-free and 52% are off anticonvulsants. Unlike prior case series, performing hemispherectomy at a later age did not adversely affect seizure control or cognitive outcomes.

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Cognitive impairment after subarachnoid hemorrhage

Mayer et al. found that cognitive impairment is associated with diminished functional status, emotional health, and quality of life after subarachnoid hemorrhage. The Telephone Interview of Cognitive Status, a simple test of global mental status, detected disabling cognitive impairment nearly as effectively as more detailed neuropsychological tests.

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Driving safety in Parkinson's disease

Zesiewicz et al. found that using a driving simulation as the method of testing, Parkinson's disease patients sustained "more "motor vehicle accidents" than normal control subjects. The number of "motor vehicle accidents" correlated with disease stage and cognitive impairment.

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New artistic skill and severe frontotemporal dementia (FTD)

Antérion et al. describe a patient without previous artistic interest/expression in whom a new artistic skill emerged despite advanced frontotemporal dementia. He began to draw crude self-portraits despite being completely mute, akinetic, and unresponsive to examiners.

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