Background: Neurocysticercosis (NCC) is the most common parasitic disease of the human central nervous system and a major health problem for most developing countries. The most common clinical manifestations of NCC are epileptic seizures. Whenever epilepsy and NCC coexist in the same patient, an uncertainty may rise about a causal relationship between them.

Observation: We described a female patient with disseminated calcified NCC lesions and intractable epilepsy. Her medical history included cysticercotic meningoencephalitis and status epilepticus caused by active NCC. Fundoscopy showed the ocular presence of para- and subcutaneous tissue. Video-electroencephalography and ictal and interictal single-photon emission computed tomography disclosed left mesial temporal lobe epilepsy. The patient underwent left temporal lobectomy and has been seizure free since surgery, for a follow-up of 4 years.

Conclusion: This case report highlights and supports surgical therapy in patients with epileptic seizures and calcified NCC, even when there are several calcifications, provided that clear localization of epilepsy has been determined by means of a presurgical workup.

Arch Neurol. 2004;61:1117-1119

(Reprinted) Arch Neurol. 2004;61:1117-1119

©2004 American Medical Association. All rights reserved.
of reported cases of improved surgical treatment when complete removal of both lesions is performed.8

We herein describe the unique case of a patient with countless calcified NCC lesions who became seizure free after temporal lobectomy. It demonstrates that even extreme dissemination of NCC lesions does not preclude the successful surgical therapy for epilepsy. We believe that this report contributes to the decision-making process about surgical treatment of such patients.

REPORT OF A CASE

A 21-year-old woman with medically intractable complex partial seizures was referred to our center for presurgical evaluation. Medical history included several episodes of cysticercotic meningoencephalitis starting at 6 years of age due to a massive infestation of cerebral parenchyma with cysticerci associated with an intense inflammatory response. These episodes were characterized by headache, vomiting, fever, neck stiffness, and right-sided focal motor status epilepticus. Computed tomography disclosed countless lesions of active (presence of scolex) and transitional forms of NCC. Hydrocephalus and intracranial hypertension due to an intraventricular cyst developed, and a ventriculoperitoneal shunt was then performed. Results of cerebrospinal fluid analysis revealed mononuclear pleocytosis, eosinophilia, reference-range glucose levels, and elevated protein levels. Results of an enzyme-linked immunosorbent assay of the cerebrospinal fluid for cysticercosis were strongly positive. Treatment included oral and intravenous dexamethasone sodium phosphate and phenytoin sodium. The patient also presented with an ocular cysticercus in the subretinal space that caused right monocular blindness. After a silent period of 8 years since her initial seizures, medically intractable complex partial seizures developed.

During the presurgical evaluation, left temporal lobe epilepsy was diagnosed. The CT and x-ray film findings showed disseminated subcutaneous, muscular, and intracranial calcified cysticercotic lesions (Figure, A-C). The magnetic resonance imaging findings also disclosed left mesial temporal lobe atrophy (Figure, D). Results of electroencephalography showed interictal left temporal lobe epileptiform discharges and interictal left temporal intermittent rhythmic delta activity. Results of video-electroencephalographic monitoring showed 100% of interictal spikes on the left temporal lobe and complex partial seizures with ictal electroencephalographic onset in the left temporal region. Results of ictal single-photon emission computed tomography showed a typical hyperperfusion on the left temporal lobe,9 whereas those of interictal single-photon emission computed tomography showed a moderate hypoperfusion on the same region (Figure, E and F).
On the basis of these findings, the patient underwent a left anterior and mesial temporal lobectomy. Results of pathological examination of the surgical specimen revealed hippocampal sclerosis characterized by severe neuronal loss and gliosis in the prosubiculum, CA1 (cornu ammonis 1), and CA4 regions associated with dispersion of granular neurons of the fascia dentata. Multiple calcified nodular lesions with gliosis in the adjacent temporal cortex were also found. Since surgery, she has remained seizure free (Engel class Ia) for 4 years and reports a great improvement in her quality of life.

We described a female patient who presented with a disseminated distribution of calcified NCC lesions and who underwent successful epilepsy surgery. She initially had cystercicercotic meningoencephalitis and focal motor status epilepticus caused by active and transitional forms of NCC. The definitive diagnosis of cystercicercosis was determined on the basis of 2 absolute criteria, ie, the detection of the parasite by funduscopy and evidence of cystic lesions showing the scolex on CT findings.

It became clear from the results of the presurgical workup that she had exclusively left mesial temporal lobe seizures, despite the presence of multiple cerebral lesions. The natural history of her epilepsy was indistinct from typical cases of mesial temporal lobe epilepsy, including the presence of an initial precipitating insult, a silent period without seizures, and the late onset of complex partial seizures confirmed by results of video-electroencephalographic monitoring and a seizure-free outcome after surgery. In this case, 2 possible initial precipitating insults were implicated in the pathogenesis of the hippocampal sclerosis, ie, the status epilepticus and/or the meningoencephalitis. The main dilemma during the decision-making process of this case was obviously whether the NCC was related or unrelated to the epilepsy. This definition affected the decision of eligibility or noneligibility for surgical treatment. If the conclusion was that cystercicercotic lesions were a coincidental finding, the patient would be a candidate for surgery. On the other hand, if NCC was thought to be causally related to the epilepsy, surgery would not be feasible, considering that removal of all lesions was not possible.

We believe that this case highlights the possibility of successful surgical treatment when patients present with well-defined localization-related epilepsy, despite the existence of disseminated calcified NCC lesions. It is possible that NCC had played a role early as an initial precipitating insult, leading to the development of hippocampal sclerosis. In recent years, however, the presence of calcified NCC and chronic epilepsy was considered to be unrelated in this patient, and calcified NCC per se did not represent an absolute contraindication for the surgical treatment.

Accepted for publication December 12, 2003.

Author contributions: Study concept and design (Dr Wichert-Ana, Walz, Bianchin, Leite, and Sakamoto); acquisition of data and administrative, technical, and material support (Dr Wichert-Ana, Velasco, Terra-Bustamante, Alexandre, Bianchin, Leite, Assirati, Carlotti, Araújo, Santos, Takayanagui, and Sakamoto); analysis and interpretation of data (Dr Wichert-Ana, Terra-Bustamante, Alexandre, Araújo, and Sakamoto); drafting of the manuscript (Dr Wichert-Ana, Carlotti, and Sakamoto); critical revision of the manuscript for important intellectual content (Dr Wichert-Ana, Velasco, Terra-Bustamante, Alexandre, Walz, Bianchin, Leite, Assirati, Carlotti, Araújo, Santos, Takayanagui, and Sakamoto); statistical expertise (Dr Wichert-Ana, Velasco, and Walz); obtaining funding (Dr Wichert-Ana, Walz, Leite, Carlotti, and Sakamoto); study supervision (Dr Wichert-Ana, Terra-Bustamante, and Alexandre).

This study was supported by Programa de Capacitação de Recursos Humanos para Atividades Estratégicas from the Conselho Nacional de Desenvolvimento Científico e Tecnológico, Brasília, Brazil (Drs Walz and Bianchin); Programa Temático Multinstitucional em Ciência da Computação (TeleMed/04-2000) from the CNPq, Brasília (Drs Wichert-Ana and Walz); Fundação de Amparo à Pesquisa do Estado de São Paulo, São Paulo, Brazil (Drs Walz, Bianchin, and Leite); Coordenação de Aperfeiçoamento de Pessoal de Nível Superior, Brasília (Dr Wichert-Ana); Fundação de Apoio ao Ensino, Pesquisa e Assistência do Hospital das Clínicas da Faculdade de Medicina de Ribeirão Preto da Universidade de São Paulo, Ribeirão Preto (Dr Wichert-Ana), and Volkswagen Stiftung für Latein Amerika, Hannover, Germany (Drs Walz and Bianchin).

We thank Electra Greene for her professional review of the English version.

Correspondence: Lauro Wichert-Ana, MD, CIREP–Centro de Cirurgia de Epilepsia, Hospital das Clínicas da Faculdade de Medicina de Ribeirão Preto, Av Bandeirantes, 3900, Campus Universitário, Monte Alegre, Ribeirão Preto, SpCeP, 14.048-900, Brazil (lwichert@rnp.fmrp.usp.br).

REFERENCES