Amyotrophic lateral sclerosis and neurocysticercosis

Martínez Héctor R,*,*** Caro Enrique,* Gil-Valadez Alfonso,*,*** Moreno Cuevas Jorge,*** González-Garza María Teresa,*** Molina-López Juan Francisco,*** Treviño-Manllo Sergio A,*** Hernández-Torre Martín****

* Servicio de Neurología, Hospital San José Tecnológico de Monterrey.
** Servicio de Neurocirugía, Hospital San José Tecnológico de Monterrey.
*** Tecnológico de Monterrey, School of Medicine, Servicio de Terapia Celular, CITES, Monterrey N.L., Mexico.
**** Biotechnology and Health, Tecnológico de Monterrey.

RESUMEN
La asociación entre la Esclerosis Lateral Amiotrófica (ELA) y la Neurocisticercosis (NCC) rara vez se ha descrito en la literatura. Presentamos un paciente con ambos trastornos. Se diagnosticó NCC en un paciente de 55 años de edad, masculino con tomografía axial y pruebas de inmunoensayo en líquido cefalorraquídeo (LCR). Después del tratamiento con Praziquantel, desarrolló bradilalia, disartria llegando a lenguaje incomprensible. También mostró sintomatología bulbar y de neurona motora superior e inferior. La electromiografía apoyó el diagnóstico ELA definida, la resonancia magnética de cerebro confirmó la presencia de NCC en motor cortes. Aunque la asociación entre NCC y ALS puede haber ocurrido por azar, se hipotetiza que autoinmune, apoptótico y circulatorio.

ABSTRACT
The association between Amyotrophic Lateral Sclerosis (ALS) and Neurocysticercosis (NCC) has rarely been described in literature. We describe a patient with both disorders. NCC was diagnosed in a 55-year-old male patient with positive CT scan and cerebrospinal fluid (CSF) immunoassay tests. After Praziquantel treatment, he developed slurred speech, bradylalia and periods of speech arrest. He also demonstrated bulbar, upper and lower motor neuron symptomatology. Neurology Service (Clinical)
evaluation showed non-comprehensive speech, sialorrhea, neck weakness, tongue with atrophy and fasciculations. Upper (1/5) and lower (3.5/5) limb weakness with muscular atrophy and fasciculations also presented with generalized hyperreflexia, bilateral Babinski, Chaddock, Hoffman and Trommer signs. Electromyography demonstrated positive sharp waves, fibrillations and fasciculations in four limbs. A nerve conduction study revealed velocities within normal range. Diagnosis of definite ALS in association with NCC was then established. On admission, MRI showed multiple hypointense lesions in frontal lobes including bilateral motor strip (Figure 1).

**DISCUSSION**

The association of ALS and NCC has rarely been described in literature. We describe an NCC patient who presented ALS after receiving cysticidal agent Praziquantel. The cause of ALS remains unknown although the identification of mutations in the SOD1 gene is relevant. Other etiologic hypotheses have been proposed and include exposure to heavy metals, virus, prions, endogenous cytotoxic factors, age, apoptosis, abnormal neurotrophic factors or axonal transport and autoimmunity. None of these mechanisms alone explain the cascade of events that lead to selective motor neuron destruction.

In our patient, NCC and ALS association may have occurred by chance. We consider that inflammatory reaction against the cysticercus promoted the release of substances such as peripherin which is known to cause axonal injury, disorganization of neurofilaments and axonal strangulation. Inflammatory molecule release may have up-regulated transcriptional factors leading to activation of apoptotic paths, thus creating and amplifying cascade of caspases which led to degradation of DNA and cell death. An autoimmune cross reaction between NCC antigens and dystonin may have produced upper motor neuron destruction by losing neuronal cytoskeleton integrity. Adhesive leptomeningitis and meningeal fibrosis in the brain and spinal cord is frequently described in NCC. Perivascular involvement in spinal cord by fibrosis or meningeal inflammation can produce vascular insufficiency with consequent lower motor neuron death.

Since NCC produces a broad spectrum of neurological manifestations, we suggest that ALS patients in developing countries should undergo CSF immunoassay evaluation to corroborate its association with NCC. Autoimmunity, apoptosis and ischemia induced by NCC and/or Praziquantel can play a significant role in the pathogenesis of this ALS patient.

**CONFLICT OF INTERESTS**

The authors declare that they have no conflict of interest.

**REFERENCES**


5. Martínez HR, Rangel GRA, Arredondo EJH, Marfil A, O nofre J. Medical and surgical treatment in neurocysticercosis a magnetic resonance study of 161 cases. J Neurol Sci 1995; 130(1): 25-34.


Corresponding: Héctor R. Martínez MD, FACP
School of Medicine. CITES 3rd floor
Morones Prieto No. 3000
Col. Pte Monterrey
C.P. 64710, Nuevo León, México
Tel.: 52(81) 8888-2177,
Fax: 52(81) 8888-2148
E-mail: hector.ramon@itesm.mx