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Cerebral and spinal neurocysticercosis with extensive myocysticercosis presenting with new-onset convulsive status epilepticus and myopathic symptoms

Neurocisticercosis cerebral y espinal con miocisticercosis extensa, presentándose con estado epiléptico convulsivo de nueva aparición y síntomas miopáticos

Dear Editor,

Neurocysticercosis is a parasitic infection of the central nervous system caused by the cystic larval stage of the tapeworm *Taenia solium*.[1] Endemic areas include Latin America, Southeast Asia, India, Nepal, China, and Africa, where it is a leading cause of acquired epilepsy.[1] With increasing globalization and international travel, neurocysticercosis is now being reported in many developed countries.[1]

We herein report an adult case of cerebral and spinal neurocysticercosis with extensive myocysticercosis, which presented with new-onset convulsive status epilepticus and proximal muscle weakness masquerading clinically as an acquired myopathy.

A 23-year-old male from rural India was brought by his relatives with an acute new-onset generalized tonic–clonic status epilepticus. He was stabilized at the emergency department with intravenous lorazepam (needed a total of 8 mg in two separate dosages to abort seizure) and a loading dose of phenytoin (700 mg with intravenous normal saline) and shifted to the intensive care unit for close monitoring. His relatives revealed that he complained of progressive generalized weakness, which initially started in the proximal extremities with a visible increase in the bulk of the affected muscles and a bodybuilder-like appearance over the last year associated with easy fatigue and pain. He also complained of persisting headaches with frequent exacerbation and blurred vision for the last two months.

After recovery from the post-ictal state, physical examination revealed that the muscles were firm, tender, and symmetrically hypertrophied in both upper and lower limbs, and weakness, particularly in proximal muscles (MRC 3-/5). He also had multiple soft, non-tender nodules of variable sizes on the forehead and neck.

The complete blood cell count revealed mild anemia (10.1 g/dL, normal 13.5–17.5 g/dL), peripheral eosinophilia (3000 eosinophils/μL, normal absolute eosinophil count 30–350 eosinophils/μL), and a high erythrocyte sedimentation rate (89 mm/h, normal <15 mm/h). Serum creatine phosphokinase (978 U/L, normal <171 U/L) and aldolase (38 U/L, normal <7.6 U/L) levels were raised. The remaining biochemical parameters were within normal levels. Brain magnetic resonance imaging (MRI) revealed the starry sky appearance of neurocysticercosis (Fig. 1). MRI of the neck and limb muscles showed the presence of generalized deposits of cysticercus larvae (Fig. 2). Excision biopsy from a lesion over the left quadriceps muscle was performed, with histopathology revealing cysticercus larvae within the muscle fibers. Diagnosis of neurocysticercosis (including extraneuralcysticercosis) was finally made. Stool samples from the

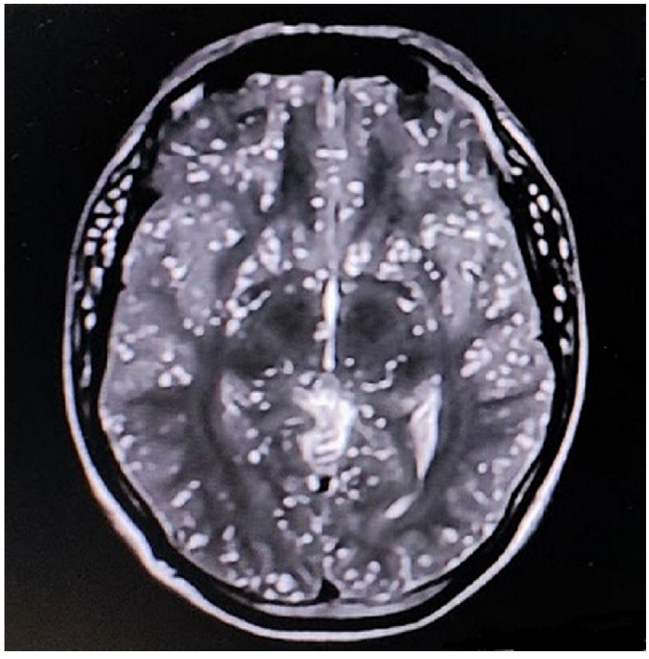

Fig. 1. Brain magnetic resonance imaging reveals the starry sky appearance of neurocysticercosis on axial-T2-weighted imaging (A).

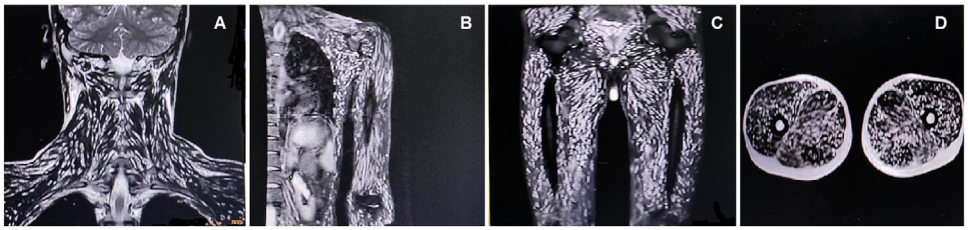

Fig. 2. Magnetic resonance imaging of the neck and limb muscles shows the presence of generalized deposits of cysticercus larvae (A–D).

patient and family members were negative for *Taenia solium* infestation. MRI screening of the family members was negative for extra-intestinal infestation, too. However, serological tests could not be done due to a lack of specificity/sensitivity and the inadequate laboratory setup required for those tests.

The patient was put on phenytoin (300 mg/day in three divided doses) and levetiracetam (2000 mg/day in two divided doses) for seizure prevention, together with dexamethasone (24 mg/day in three divided doses). Unfortunately, the patient abruptly discontinued antiseizure medication (without supervision) after two months and again came with severe convulsive status epilepticus, and this time, could not be saved despite all possible medical efforts.

Our case highlights the importance of including neurocysticercosis in the differential diagnosis of new-onset convulsive status epilepticus or myopathic pictures, especially in patients from endemic areas.

The decision not to start antiparasitic therapy (albendazole and praziquantel to albendazole) in our patient aligns with our current understanding and recommendations for managing neurocysticercosis, particularly in cases with encephalitis or when there is a risk of exacerbating cerebral edema and brainstem compression (sometimes, this edema can cause blindness if there are lesions near optic nerves).[2] Antiparasitic drugs can worsen cerebral edema and potentially trigger new seizures or worsen existing ones.[2] In such instances, the priority is stabilizing the patient with antiseizure medications and corticosteroids like dexamethasone to control inflammation.[2] This approach aims to manage the acute symptoms and prevent complications associated with increased intracranial pressure and seizure activity. Once the patient is stabilized and the risks of cerebral edema are mitigated, a careful reassessment can be made regarding the timing and safety of introducing anthelmintic therapy.[2] Our cautious approach reflects the necessity of balancing the benefits of eradicating the parasite with the immediate risk of precipitating severe complications (i.e., cerebral edema leading to refractory status epilepticus/brainstem compression and visual threat).[2]

The patient's death after discontinuing antiseizure medication also highlights the critical nature of seizure control in the management of neurocysticercosis. Ensuring patient adherence to prescribed medication regimens is a significant aspect of long-term disease management to prevent such tragic outcomes.

Neurocysticercosis diagnosis depends on multiple factors, including symptoms, imaging with computed tomography and MRI, and serology. MRI provides more detailed images of active parenchymal stages and non-parenchymal disease within the brainstem, subarachnoid, and intraventricular regions and can confirm the diagnosis of neurocysticercosis if a scolex is identified within the cyst (a pathognomonic finding) as well as identify disease outside of the parenchyma.[3]

The most common presentation of parenchymal neurocysticercosis is focal and brief seizures.[1] Convulsive status epilepticus is a rare presentation of all the evolutionary stages of neurocysticercosis.[4,5] Perilesional factors like perilesional edema and gliosis have also been implicated in neurocysticercosis-associated epileptogenesis,[4,5] but the effects of cystic factors, including lesion load and location, seem not to play a role in the development of epilepsy.

Neurocysticercosis is a multifaceted disease with a broad spectrum of clinical and radiographic features, whose medical management is complex and needs to be individualized. Early recognition of this clinical-parasitic-radiographically setting is essential for achieving a proper treatment to reduce morbidity and mortality. Further studies are needed to elucidate the exact epileptogenic mechanisms in neurocysticercosis.

Ethical statement

Written informed consent was obtained from the patient participating in the study (consent for research). We also confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

Authors' contributions

All authors contributed significantly to the creation of this manuscript; each fulfilled criterion as established by the ICMJE.

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Conflict of interest

The authors declare that they have no conflict of interest.

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Diagnóstico tardío de VIH en paciente migrante con infección oportunista importada

Delayed diagnosis of HIV in migrant patient suffering from opportunistic imported infection

A propósito de un caso atendido en nuestro servicio, queríamos llamar la atención de los lectores respecto a la problemática de actualidad que implica la atención inicial de la infección por VIH en el colectivo de pacientes migrantes.

Se trata de una mujer transexual de 53 años, trabajadora sexual, natural de Colombia, residiendo en España desde un año antes del ingreso. Seis meses antes del ingreso había sido diagnosticada de una infección por VIH a raíz de un cribado en atención primaria pero no había llegado a iniciar tratamiento antirretroviral a causa de dificultades logísticas para poder vincularse a una unidad especializada y por escasa comprensión de las implicaciones de la enfermedad.

Ingresó por dolor abdominal de 3 meses de evolución asociado a deposiciones diarreicas sin productos patológicos de un mes de evolución y pérdida de peso. Al ingreso destacaba febrícula y una masa dolorosa en flanco derecho. Además, presentaba aumento de adenopatías axilares y odinofagia. Radiografías de tórax y abdomen normales. En la analítica destacaba aumento de reactantes de fase aguda, recuento de linfocitos T $CD4^+$ 46 cél/μl (cociente CD4/CD8 0,08) y carga viral para VIH de 220.000 cp/ml. El estudio microbiológico inicial fue negativo para micobacterias en orina, sangre y heces, antígenemia criptococo, beta-D-glucano y galactomanano, coprocultivo, toxina *C. difficile*, estudio de parásitos y PCR rectal para clamidia-LGV y gonococo. Carga viral plasmática de CMV < 34,5 UI/ml.

Ante la sospecha de ileocolitis infecciosa se inició tratamiento empírico tuberculostático y profilaxis con cotrimoxazol. Una semana después se inició terapia antirretroviral con bictegravir, emtricitabina y tenofovir. Como estudio de extensión se solicitó una TC abdominal que evidenció extensa inflamación en estómago e intestino delgado con predominio de zona ileocólica. La colonoscopia evidenció una lesión ulcerada seudotumoral desde íleon distal a colon ascendente, de la cual se tomaron biopsias: negatividad para tinción Ziehl-Neelsen y signos citopáticos compatibles con colitis por citomegalovirus, así como positividad de técnica inmunohistoquímica. Se inició tratamiento con ganciclovir.

Tras una semana bajo tratamiento antirretroviral y ganciclovir la paciente desarrolló un cuadro de suboclusión intestinal y disfagia que obligó a iniciar nutrición parenteral. Una nueva TC evidenció empeoramiento del engrosamiento mural a nivel ileocólico, condicionando una disminución de la luz intestinal y dilatación retrograda. Paralelamente presentó un cuadro de fiebre elevada e insuficiencia respiratoria aguda. La radiografía de tórax mostró un patrón miliar (fig. 1B). Los análisis revelaron elevación marcada de reactantes de fase aguda y una bicitopenia no presente al ingreso (hematíes $3{,}42 \times 10^{12}$/l, hemoglobina 70 g/l, leucocitos $2{,}96 \times 10^{9}$/l).

Ante el empeoramiento clínico con afectación multiorgánica, se sospechó un síndrome de reconstitución inmune por el desenmascaramiento de probable infección oportunista. Entre el diagnóstico diferencial y teniendo en cuenta el origen de la paciente y las exploraciones complementarias se planteó la posibilidad de histoplasmosis diseminada. Se revisaron las biopsias colónicas y en la tinción de PAS y argéntica se identificaron levaduras compatibles con *Histoplasma* spp (fig. 1C). Se inició anfotericina B liposomal y se realizó un lavado broncoalveolar que objetivó la presencia de levaduras de pequeño tamaño. Tras la incubación se aisló *Histoplasma capsulatum* (fig. 1D). La fibrogastroscopia mostró un engrosamiento de pliegues gástricos en cuyas biopsias también se evidenció la presencia de *H. capsulatum* mediante biología molecular. Se completó el estudio con un aspirado medular que no mostró infiltración de médula ósea. Tras 2 semanas de tratamiento la paciente presentó una mejoría clínica y tolerancia correcta a vía oral, por lo que se inició tratamiento de consolidación con isavuconazol. Al alta se mantuvo el mismo tratamiento y se vinculó a la paciente a un programa piloto de seguimiento estrecho multidisciplinar: enfermería especializada, trabajo social y equipo médico.

En España las micosis endémicas como la histoplasmosis se encuentran en aumento debido a los movimientos migratorios y el turismo en zonas endémicas, especialmente en zonas de Latinoamérica. Molina-Morant et al. estudiaron 286 casos de histoplasmosis que requirieron hospitalización entre los años 1997-2014 en España, siendo las comunidades más afectadas Madrid, Cataluña y Andalucía. Hasta un 50% de los pacientes afectos de histoplasmosis presentaban infección por VIH. Estos pacientes asociaban mayor mortalidad y estancias hospitalarias más prolongadas[1–5].