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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.

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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 extra-

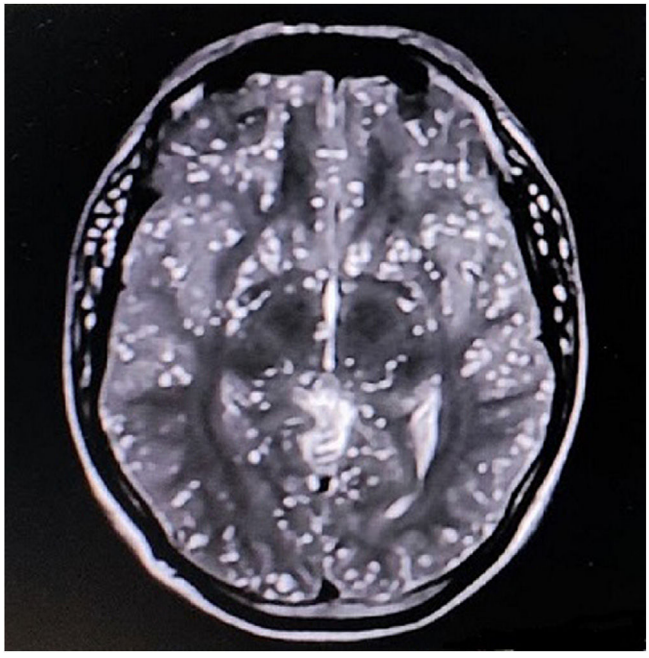

Fig. 1. Brain magnetic resonance imaging reveals the starry sky appearance of neurocysticercosis on axial-T2-weighted imaging (A).

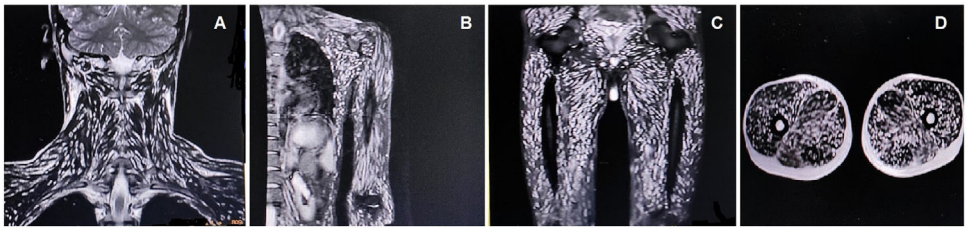

Fig. 2. Magnetic resonance imaging of the neck and limb muscles shows the presence of generalized deposits of cysticercus larvae (A–D).

neuralcysticercosis) was finally made. Stool samples from the 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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Delayed diagnosis of HIV in migrant patient suffering from opportunistic imported infection

Diagnóstico tardío de VIH en paciente migrante con infección oportunista importada

Through the report of a case treated in our department, we wanted to draw the attention of readers to current problems involved in the initial care of HIV infection in the migrant patient group.

This was a 53-year-old trans-sexual woman, a sex worker, originally from Colombia, who had been living in Spain for a year before admission. Six months before admission, she had been diagnosed with HIV infection following screening in primary care, but had not started antiretroviral treatment due to logistical difficulties in being able to join a specialised unit and due to poor understanding of the implications of the disease.

She was admitted with a three-month history of abdominal pain associated with a one-month history of diarrhoea with no blood or mucus and weight loss. On admission, she had a low-grade fever and a painful mass on her right side. She also had enlarged axillary lymph glands and odynophagia. Chest and abdominal X-rays were normal. Bloods showed an increase in acute phase reactants, $CD4^+$ T lymphocyte count 46 cells/μl (CD4/CD8 ratio 0.08) and HIV viral load of 220,000 copies/ml. The initial microbiological study was negative for mycobacteria in urine, blood and faeces, cryptococcal antigenaemia, beta-D-glucan and galactomannan, stool culture, *C. difficile* toxin, parasite study and rectal PCR for chlamydia-LGV and gonococcus. Plasma CMV viral load <34.5 IU/ml.

As infectious ileocolitis was suspected, the patient was started on empirical antituberculosis treatment and prophylaxis with cotrimoxazole. One week later, she was started on antiretroviral therapy with bictegravir, emtricitabine and tenofovir. As an extension study, an abdominal CT scan was requested, which showed extensive inflammation in the stomach and small intestine with predominance in the ileocolic area. Colonoscopy showed a tumour-like ulcerated lesion from the distal ileum to the ascending colon, from which biopsies were taken: negative for Ziehl–Neelsen staining and cytopathic signs compatible with cytomegalovirus colitis, and immunohistochemistry was positive. Treatment was started with ganciclovir.

After a week on antiretroviral treatment and ganciclovir, the patient developed intestinal subocclusion and dysphagia for which she required parenteral nutrition. A repeat CT scan showed worsening of the mural thickening at the ileocolic level, causing a decrease in the intestinal lumen and retrograde dilation. At the same time, the patient developed a high fever and acute respiratory failure. Chest X-ray showed a miliary pattern (Fig. 1B). Bloods showed a marked elevation of acute phase reactants and bicytopenia not present on admission (red blood cells 3.42×10^{12}/l, haemoglobin 70 g/l, leucocytes 2.96×10^{9}/l).

With the deterioration in the patient's clinical condition and multiorgan involvement, an immune reconstitution syndrome was suspected due to the unmasking of a probable opportunistic infection. Between the differential diagnosis and taking into account the patient's origin and the additional tests, we considered the possibility of disseminated histoplasmosis. The colon biopsies were reviewed and periodic acid-Schiff and silver staining identified yeasts compatible with *Histoplasma* spp (Fig. 1C). The patient was started on liposomal amphotericin B and a bronchoalveolar lavage was performed, which revealed the presence of small yeasts. After incubation, *Histoplasma capsulatum* was isolated (Fig. 1D). Fibre-optic gastroscopy showed a thickening of gastric folds and biopsies taken for molecular biology also showed the presence of *H. capsulatum*. Tests were completed with a spinal aspirate, which did not show bone marrow infiltration. After two weeks of treatment, the patient showed clinical improvement and had good oral tolerance, so consolidation treatment with isavuconazole was started. At discharge, the same treatment was maintained and the patient was linked up with a pilot programme of close multidisciplinary follow-up: specialised nursing, social work and medical team.

In Spain, endemic mycoses such as histoplasmosis are increasing due to migratory movements and tourism in endemic areas, especially in areas of Latin America. Molina-Morant et al. studied 286 cases of histoplasmosis requiring hospitalisation in the period 1997–2014 in Spain, with the most affected regions being Madrid, Catalonia and Andalusia. Up to 50% of patients with histoplasmosis had HIV infection. These patients had higher mortality rates and longer hospital stays.[1–5]

With the migrant population comes an added situation of vulnerability, which can make it more difficult for them to access the healthcare system. Ndumbi et al. report that the most common barriers to access that migrant patients in Spain perceive are the procedures associated with obtaining healthcare coverage and the

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