Letter to the Editor

Cerebral ring enhancing lesion with diffusion restriction in a South American patient

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Cerebral ring enhancing lesions
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Dear Editor,

The differential diagnosis of cerebral ring enhancing lesions is usually challenging and involves the use of multiple paraclinical tools such as central nervous system (CNS) imaging and cerebrospinal fluid (CSF) studies [1,2]. These lesions can be the manifestation of a variety of pathologies, including infection, primary or secondary neoplastic processes and demyelinating diseases, amongst others. By using conventional magnetic resonance imaging (MRI), an accurate diagnosis of ring enhancing lesions is often difficult. Therefore, the use of novel imaging techniques plays a crucial role in fully characterizing and identifying the underlying pathology of such ring enhancing lesions.

A 23-year-old man from the south of Chile was admitted to our Department of Neurology in Santiago, with a history of 2 weeks of multiple self-limiting episodes of involuntary clonic movements of his right face and arm, and one single episode of generalized tonic–clonic seizure. On admission, his vital signs and general examination were unremarkable. Initial neurological examination revealed mild right hemiparesis (MCR score 4) with symmetrical tendon reflexes and an ipsilateral up-going plantar reflex. Cranial nerves and sensory function were normal. Complete blood count, erythrocyte sedimentation rate and C-reactive protein were normal, together with negative antibody testing for HIV, Hepatitis B and C, and VDRL. CSF analysis showed 4.8 leukocytes per microliter, protein of 0.27 g/L, normal glucose levels, and a negative Gram stain and culture. The brain MRI showed one cortico-subcortical lesion in the left precentral frontal region (Fig. 1), mildly hyperintense to CSF on T1-weighted image, hyperintense on T2-weighted image and fluid-attenuated inversion recovery (FLAIR) image, with perilesional vasogenic oedema (hypointense on T1 and hyperintense on T2/FLAIR). The three-dimensional constructive interference in steady state (3D-CISS) sequence demonstrates a cystic lesion a small eccentric scolex (Fig. 1C). In addition, the lesion showed a thick, well-enhancing capsule following contrast, which was hypointense in T2 and 3D-CISS. The corresponding diffusion-weighted image (DWI) revealed restricted diffusion at the centre of the lesion (Fig. 1E and F). Given the epidemiological background and clinical presentation, parenchymal neurocysticercosis (NCC) in a colloid vesicular stage was suspected. A CSF immunodiagnostic study (Western blot) was done, which resulted as positive for Taenia solium (IgG). The patient started on valproic acid and continued to be free of seizures during the follow-up.

NCC is the most common parasitic disease of the CNS and one of the world’s leading causes of acquired epilepsy, especially in South America, India, Africa and China. NCC is characterized by the presence of encysted larvae of the cestode Taenia solium in CNS tissue, developing after the ingestion of pork tapeworm eggs (faecal–oral route) [3]. Nowadays, NCC diagnosis relies on diagnostic criteria based on clinical, radiologic, immunologic and epidemiological data and thus, the development of neuroimaging techniques is crucial for the visualization of the parasites [4].

From the radiological point of view, four stages of parenchymal NCC can be recognized using computed tomography (CT) or MRI, which resemble the histopathological stages of parasitic cyst degeneration [5]. These stages are: (i) vesicular, (ii) colloid vesicular, (iii) granular nodular and (iv) nodular calcified, representing the natural progression of the cyst from acute to chronic form. The stage of cyst degeneration depends on multiples factors, including intrinsic features of the parasite, immune response of the host and presence of cyst complications. The MRI is considered a superior modality than CT to assess the different stages and location of the cysts. Also, the use of additional MRI sequences, such as 3D-CISS, is strongly recommended to increase scolex detection, a crucial clue for making a definitive diagnosis of NCC [6].

Commonly, larval cysts do not present with diffusion restriction in MRI studies, which is a key finding to differentiate between larval cysts and pyogenic abscesses. In this
context, diffusion restriction has shown to have a high sensitivity and specificity to differentiate pyogenic abscesses from other cystic lesions [7]. However, its positive predictive value has not been evaluated in endemic areas for NCC. Of further relevance for this case, Santos and colleagues have described an eccentric dot-like or curvilinear area of diffusion restriction within NCC lesions, most commonly seen in the parenchymal forms [8]. Specifically, this imaging pattern was described in up to 29% of vesicular and 19% of colloidal vesicular stage lesions and it has been interpreted as the cyst scolex in the vesicular stage and as a sign of scolex degeneration in the colloidal vesicular stage. Moreover, a small fraction of colloidal vesicular stage lesions presented a different imaging pattern with total or subtotal diffusion restriction without a clearly identifiable scolex. In this particular scenario, the use of additional MRI sequences such as 3D-CISS could increase the detection of the scolex. Finally, one study described decreasing mean diffusivity values and increasing fractional anisotropy progressively from vesicular to granular nodular stages [9], which is in keeping with a higher frequency of lesions with diffusion restriction in early stages of the disease.

Overall, from a clinico-radiological point of view, NCC should be considered in the differential diagnosis of parenchymal lesions with reduced diffusion and ring enhancement, specifically in endemic areas. The combination of multiple MRI techniques may assist with correct differentiation between ring enhancing lesions.

Conflict of interest

None declared.

Authors contribution

JM undertook conception, design, and writing the first draft. RF, AJ, GC and RS undertook review and critique. All authors read and approved the final manuscript.
References


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