Neurocysticercosis: new insight into an old pathology

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DESCRIPTION
A female in her thirties presented with recent onset of left focal seizures without secondary generalisation. There were no other neurological findings except mild confusion. Other investigations including CSF analysis were normal. MRI showed a solitary enhancing lesion (SEL) in right parietal lobe and diagnosed as degenerating neurocysticercosis (NCC) (figure 1). Antiepileptics, methyl prednisolone and antihelminthic treatments were started. She was seizure free and the cyst healed with calcification on a 2-year follow-up (figure 2).

NCC is caused by larva of Taenia solium and is the most common cause of new onset seizures in developing world with poor socioeconomic status.1 NCC is increasingly diagnosed in developed countries due to greater international migration and travel. Having reached a dead end of life cycle in brain, the scolex retracts into a protective capsule to evade immune attack. During invagination, it drags a layer of capsule (tegumen) along with it, thus forming two chambers. The protoscolex lies within the inner chamber as a curled structure. The histopathology of cysticercus cyst is well described in the literature.2 We obtained a representative histopathological section from an autopsy specimen harbouring multiple neurocysticerci. Paraffin-embedded 3 micron sections of a parasite with grossly identifiable features were stained with H&E, mounted and studied at 40× magnification. The larva was located within the inner chamber of the cyst. The larval head contained four suckers and hooklets around a rostellum (not shown). A neck and rudimentary body containing alimentary tract were attached to the cyst wall (figure 3).

Neuroimaging is accurate in showing viable parasite as a cyst with eccentric nodule within it representing scolex (‘dot within a hole’). This morphology and direct demonstration of parasite or its characteristic three-layered wall on histology are considered major criteria for diagnosis of NCC.3 On imaging, scolex of the parasite appears as a hypointense structure on T2 and susceptibility weighted images, while it is hyperintense on T1, fluid-attenuated inversion recovery and diffusion-weighted images. CT reveals it as a hyperdense intracystic structure. Imaging studies after contrast administration may show SEL or multiple ring or disc enhancing lesions. Modern imaging using heavily T2-weighted sequences like constructive interference in steady state (CISS) and fast imaging employing turbo (FIESTA) acquisitions have markedly improved the image resolution,4 and provides submillimetre isotropic volume acquisition, facilitating accurate reconstructions. SEL on imaging can mimic tuberculoma in endemic areas and neoplasms in non-endemic areas. We believe that the potential
of imaging is underused and understanding the anatomy of the cyst can lead to correct diagnosis and management.

On reconstructed CISS images, the head of the parasite remarkably resembled that of the parasites in the intestine (Figure 1). It bears four suckers as knob-like structures and a long neck attached to cyst wall. Such morphology on imaging has not been described in the literature. We believe understanding and appreciating the cystic morphology in such details will increase the confidence for imaging diagnosis of NCC.

**Patient’s perspective**

It was a relief to know that the cause of convulsions in my case was treatable and there was no need to undergo any brain surgery. I was completely cured by medicines only.

**Learning points**

- Understanding the anatomy of neurocysticercosis.
- High-resolution MRI allows more confidence in diagnosing neurocysticercosis.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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**REFERENCES**