CLINICAL PRACTICE

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An unusual cause for cerebellar syndrome – case report

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Abstract: *A male from rural area, S.M., aged 77 years, was admitted in our department for discontinuous headache. His medical history was irrelevant. He has been experiencing intermittent right parietal-occipital headaches during the last 3 months. Neurologic exam revealed a slight right limb ataxia. Initial laboratory findings revealed a white blood cell count of 6500/mm^3 with 75% polymorphonuclear leukocytes, 15% lymphocytes and 8% monocytes. His serum glucose was 90 mg/dL. Non Gadolinium CT scan shows rounded, inhomogenous spontaneous hyperdense area (40-45 UH) between 5-12 mm diameter, localized frontal, temporal, occipital and cerebellar bilaterally.*
The question was whether the lesions were metastasis or parasitic infection?

Cerebral MRI showed unenhanced,well defined,multiple lesions between3-17 mm, with iso-hyperintensityT1, T2, and FLAIR, spreadout periventricularly, subcortically, infrontal, temporal, parietal lobes andsubtentorially, right and left cerebellum(figure 2). After serological testsfrom blood and CSF the diagnosticof neurocysticercosis was certified(an enzyme-linked immunosorbentassay of the CSF was positivefor immunoglobulin G cysticercosisantibody, with 1.32 optical densityunits (OD) (positive result > 0.50OD); his serum IgG cysticercosis antibodywas positive with 5.12 OD).

CT findings are depending on the stageof evolution:

- Vesicular stage (viable larva): hypodense, nonenhancinglesions
- Colloidal stage (larval degeneration): hypodense/isodense lesions with peripheral enhancementand edema
- Nodular-granular stage: nodular-enhancinglesions
- Cysticercotic encephalitis: diffuse edema,collapsed ventricles, and multiple enhancingparenchymal lesions
- Active parenchymal stage: the scolex within acyst may appear as a hyperdense dot
- Calcified stage: when the parasite dies, nodularparenchymal calcifications are seen.

Our patient has multiple lesions in differentphases of evolution (active and calcified).

DISCUSSIONS

Neurocysticercosis is a parasitic brain infection,caused by larval cysts of the tapeworm Taenia soliumby accidental ingestion of eggs.

It is the most common parasitic disease of the nervous system and it is themain cause of acquired epilepsy mainly in developingcountries. Once in the human intestine, Taenia eggsevolve to oncospheres

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that cross the intestinal walland lodge in the brain where cysticerci develop.

Time from infestation until first symptoms isbetween days and many years (30).This extremelylong incubation is due to hipnobiosis. They may belocated in subarachnoid space, ventricular system,or spinal cord too, causing a clinical heterogeneity.

Figure 1. Non Gadolinium CT scan shows rounded, inhomogenous spontaneous hyperdense area (40-45 UH) between 5-12 mm diameter, localized frontal, temporal, occipital and cerebellar bilaterally.

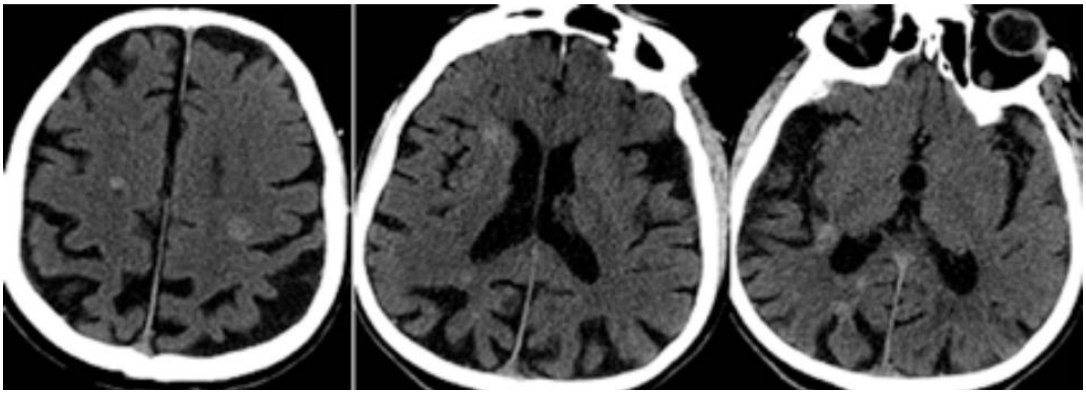
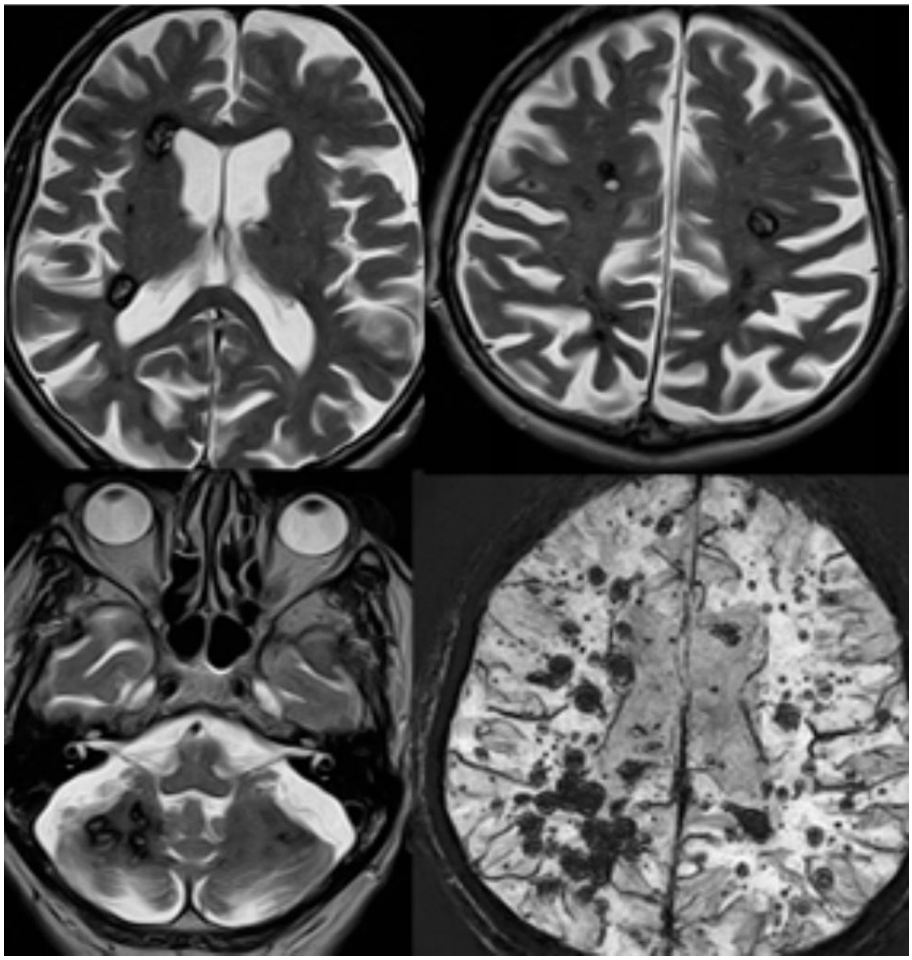


Figure 2. Cerebral MRI showed unenhanced, well defined, multiple lesions between 3-17 mm, with iso-hyperintensity T1, T2, and FLAIR, spread out periventriculary, subcortically, in frontal, temporal, parietal lobes and subtentorially, right and left cerebellum



Onset of most symptoms is usually insidiousto chronic, with seizures (most common presentation),headache, dizziness, stroke, neuropsychiatricdysfunctions. Almost every neurological sign orsymptom may be present but physical findings occurin less than 20% of the cases.

There is a pleomorphism of the immune responseagainst Taenia solium. In some cases,cysticerci are destroyed by immunological attack,while in others, parasites may live unchanged foryears. CT scan shows a rounded, homogeneous hyperdensearea with no enhancement with contrastmedium. This phase corresponds to the inactive parenchymalform of the disease.

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