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Case Report

Atypical cerebral MRI imaging findings in a patient with isolated neurosarcoidosis ☆,☆☆

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ABSTRACT

Sarcoidosis is a rare, chronic, granulomatous disease of unknown etiology and primarily effects the lymphatic and respiratory systems. The central nervous system (CNS) is unusually implicated in sarcoidosis patients. We describe a rare magnetic resonance imaging (MRI) findings in a case of isolated neurosarcoidosis. The evaluation of suspect patients requires radiological imaging studies, especially MRIs. The diagnosis of neurosarcoidosis is clinically challenging, MRI studies are effective in detecting CNS inflammation but lack specificity.

Introduction

Sarcoidosis is a rare, chronic, granulomatous disease of unknown etiology and primarily effects the lymphatic and respiratory systems. The central nervous system (CNS) is unusually implicated in sarcoidosis patients (5%-13%) or is the only affected system (1%) [1].

We describe a rare magnetic resonance imaging (MRI) findings in a case of isolated neurosarcoidosis (NS). Though this case, we would like to share these rare images with the other practitioners and discuss how to distinguish them from its common mimics.

Case report

A 52-year-old woman was admitted to a teaching hospital with headache, seizures, hallucinations and short-term memory impairment. Her medical history revealed neurosarcoidosis on corticosteroids diagnosed 6 years ago. She never fully recovered from her NS with persistent complaints of episodic seizures.

Conventional brain MRI was performed, revealing left thalamic lesion extended to the internal capsule, lenticular nucleus, mammillary body and the third ventricle, discreetly hypointense on T1-weighted sequences, hyperintense

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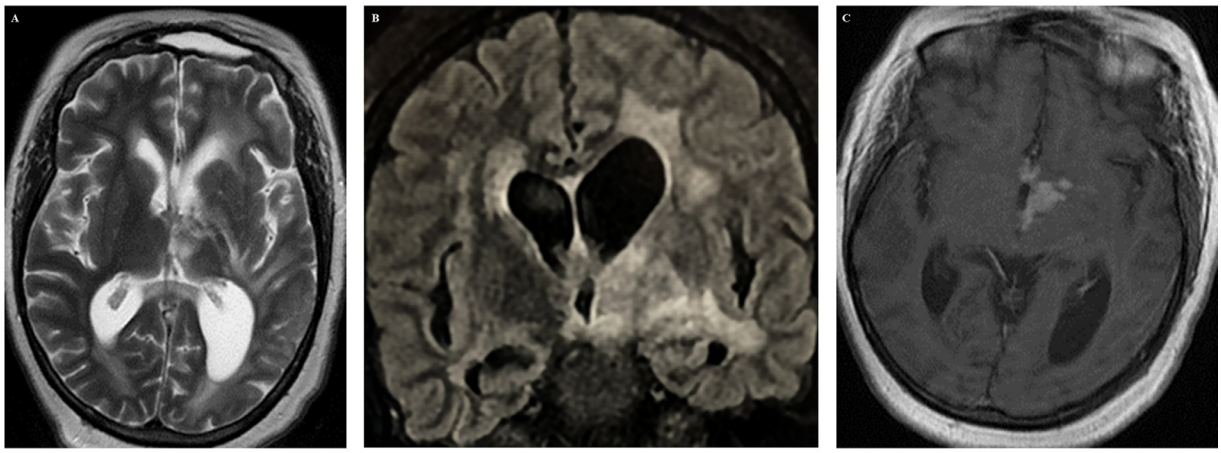

Fig. 1 – Axial T2 (A), coronal T2-FLAIR (B) and postcontrast T1 (C) sequences show left thalamic lesion extended to the internal capsule, lenticular nucleus, mammillary body and the third ventricle, which is hyperintense on T2 and FLAIR and demonstrate enhancement.

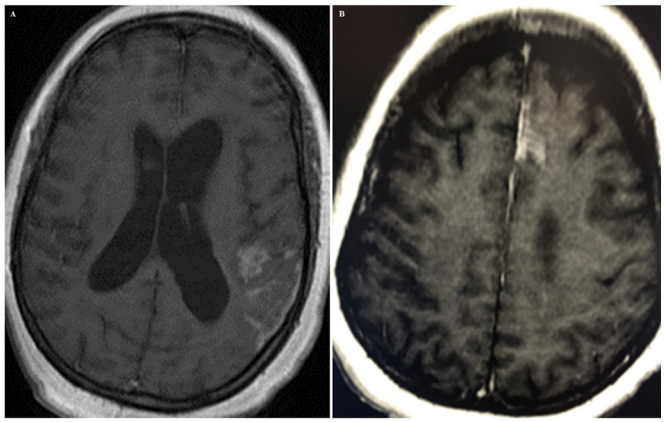

Fig. 2 – (A and B) Postcontrast axial section demonstrating left parietal and left frontal parasagittal diffuse and nodular leptomeningeal thickening and enhancement.

on T2 -weighted and FLAIR sequences, showing heterogeneous enhancement and measuring : 25 × 11mm (Fig. 1). As well as left frontal and parietal leptomeningeal thickening, with nodular gadolinium enhancement on postcontrast T1-weighted (Fig. 2A).

It also shows left frontal parasagittal nodular enhancement with a major axis of 9 mm and bilateral cerebellar lesions showing contrast enhancement (Figs. 2B and 3).

Additionally, it demonstrates an hyperintense patchy lesion on T1, T2-weighted and FLAIR sequences in the left cerebral peduncle (Fig. 4).

It revealed as well moderate hydrocephalus with hyperintense confluent lesions on T2-weighted and FLAIR sequences in the periventricular and centrum semiovale white matter (Fig. 5). No DWI or GRE signal abnormalities were detected.

Discussion

Neurosarcoidosis remains a diagnostic challenge. The evaluation of suspect patients requires radiological imaging studies, especially MRIs. Mass lesions are frequently reported in patients with neurosarcoidosis and they may mimic primary or metastatic tumors. However, it is common to observe adjacent leptomeningeal involvement, a discovery that could aid in making a correct diagnosis [2]. These individuals frequently present with seizure, which was a clinical characteristic of our patient, who also had leptomeningeal involvement. On gadolinium-enhanced T1-weighted MRI, NS in the basal leptomeninges manifests as thickening and enhancement (either localized, multifocal, or diffuse). It is usually indistinguishable from the pattern seen in CNS lymphoma or

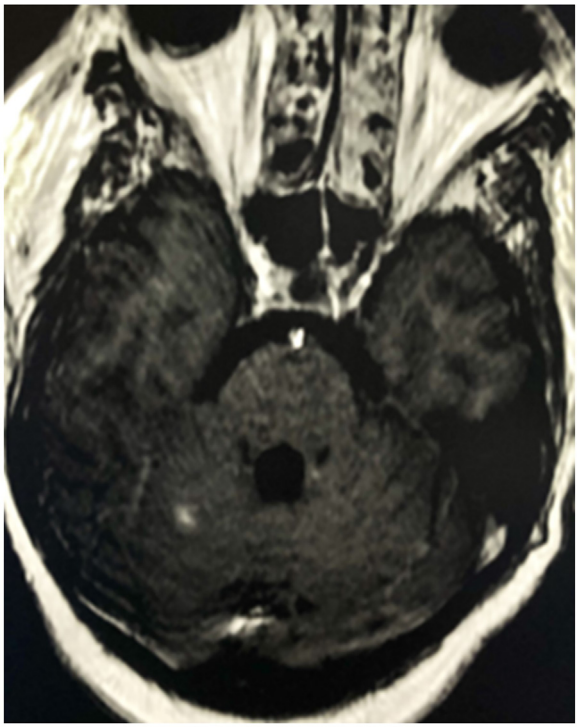

Fig. 3 – Axial postcontrast T1 weighted sequence showing a small well-defined round enhancing lesions in the right cerebellum.

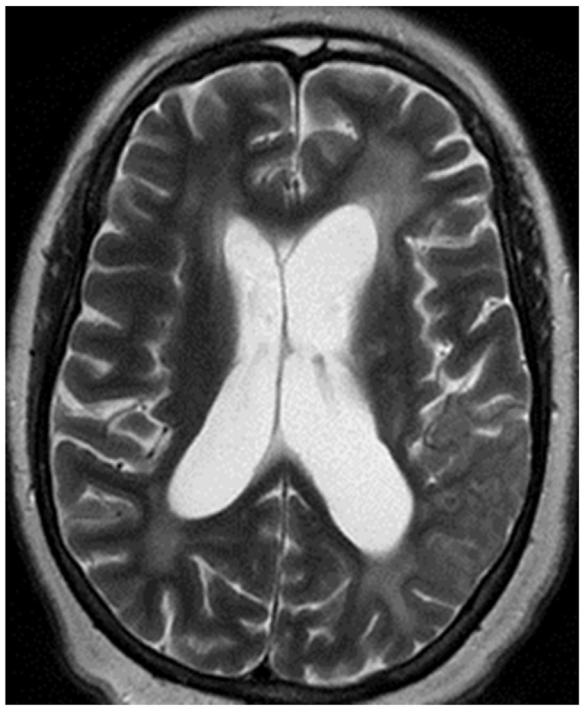

Fig. 5 – Axial T2-weighted sequences at the level of lateral ventricles showing dilatation of these structures.

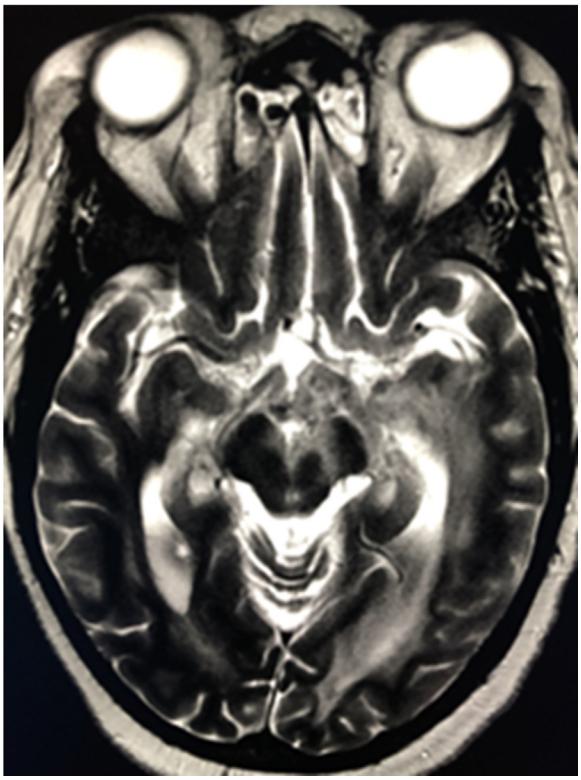

Fig. 4 – T2- weighted image show patchy hyperintensity at the level of the left cerebral peduncle.

tuberculous meningitis, which both might present clinically in a comparable way [3].

Leptomeningeal enhancement in sarcoidosis, frequently with a nodular component, may be disproportionately worse on MRI than clinical symptoms might indicate, which is the case of our patient. Leptomeningeal disease normally responds favorably to immunosuppressive therapy, however it may be worsened by seizures or cranial nerve damage; communicating hydrocephalus is another defining feature, which commonly develops after chronic meningitis. Sarcoidosis less frequently causes pachymeningitis that must be separated from other causes of hypertrophic pachymeningitis [4].

Brain parenchymal neurosarcoidosis is characterized by contrast-enhancing lesions, T2 hyperintense and T1 isointense lesions that may or may not enhance (6%-37%).

In patients with NS, subcortical encephalopathy, including dementia, may be present. Nonspecific white matter T2/FLAIR hyperintense lesions without enhancement are frequent and may be small and focal or larger, more diffuse lesions that resemble chronic vascular disease; their correlation to NS is undetermined as they are usual even in the absence of NS [5].

Many of the imaging findings in neurosarcoidosis can be caused by its mimics. For Leptomeningeal enhancement we can see it in the cases of Tuberculosis, Cryptococcus and Primary Angiitis of the Central Nervous System, rarely we can see it in granulomatosis with polyangiitis and IgG4-related disease causing pachymeningitis. Isolated enhancement is rare in cases of primary central nervous system lymphoma, but enhancement with mass lesions is common [6].

Glucocorticoids are typically used as the initial line of treatment. For patients who are more severely affected, initial IV or PO bioequivalent pulse dosing (for example, 1 g IV methylprednisolone for 3-5 days) would be considered. This would be followed by a few month oral glucocorticoid taper (for example, prednisone 1 mg/kg/d and tapering lower), with speed and taper duration being adjusted based on severity, clinical, and imaging response [7,8]. Unfortunately, the patient was lost from sight and further explorations could not be performed.

What makes our case uncommon is the association of multiple lesions seen on MRI within the same patient. As far as we know this is the first reported case of such a diversity of cerebral lesions seen in 1 case of neurosarcoidosis.

Conclusion

The diagnosis of neurosarcoidosis is clinically challenging, MRI studies are effective in detecting CNS inflammation but lack specificity. Additionally, the low prevalence of the disease makes clinical trials difficult and more studies involving detailed pathological analyses and longer follow-up periods are required. The main purpose of our work is to think about neurosarcoidosis whenever similar lesions are found on MRI. Practitioners should be aware of the possibility of having those neurosarcoidosis MRI images and know how to manage it.

Patient consent

Consent for publication has been obtained from the patient.

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