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Case Report

Diagnosing Neurosarcoidosis: When MRI Unveils an Unexpected Pathology

A. El Ouali[1*], A. Merzem[1], A. Orgi[1], H. Belgadir[1], N. Moussali[1], N. El Benna[1]

[1]Radiology Department, 20th August Hospital, Ibn Rochd University Hospital of Casablanca

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Abstract: Neurosarcoidosis is a rare and potentially severe manifestation of sarcoidosis. Its diagnosis is particularly challenging due to its highly variable clinical presentation and its ability to mimic other neurological disorders. This article aims to provide a comprehensive overview of the radiological features of this condition through a case report, while emphasizing the role of advanced imaging techniques (MRI and PET) in diagnosis and follow-up.

Keywords: Neurosarcoidosis, MRI, PET, Radiological features, Granulomatous disease.

INTRODUCTION

Sarcoidosis is a systemic granulomatous disease of unknown etiology, characterized by non-caseating epithelioid granulomas. Neurological involvement remains rare but is considered severe. Its clinical and radiological manifestations are highly variable. MRI and PET play a crucial role in the initial diagnosis, disease monitoring, progression assessment, and evaluation of therapeutic response.

In the absence of histological evidence, a diagnostic strategy based on the detection of latent extra-neurological signs of sarcoidosis makes it possible to establish the diagnosis and avoid nerve tissue biopsy.

We describe the case of neurosarcoidosis in a 27-year-old man with no known history of sarcoidosis, in whom neurological signs on MRI were the first revealing features.

CASE REPORT

A 27-year-old male patient with a history of substance abuse was admitted for neurological symptoms following suspected methadone intoxication. Brain MRI was performed with standard sequences, spectroscopy, and contrast injection.

MRI revealed T2 and FLAIR hyperintense areas in the subcortical white matter, sparing the U-fibers, in bilateral and symmetrical frontal lobes extending to the corpus callosum, periaqueductal region, hypothalamo-pituitary region, optic chiasm, optic tracts, and mammillary bodies (Figure 1). An additional left occipital lesion with similar characteristics was also found. Bilateral T2 and FLAIR hyperintensities were also seen in the posterior limbs of the internal capsules and deep white matter regions (Figure 1).

***Corresponding Author:** A. El Ouali
Radiology Department, 20th August Hospital, Ibn Rochd University Hospital of Casablanca

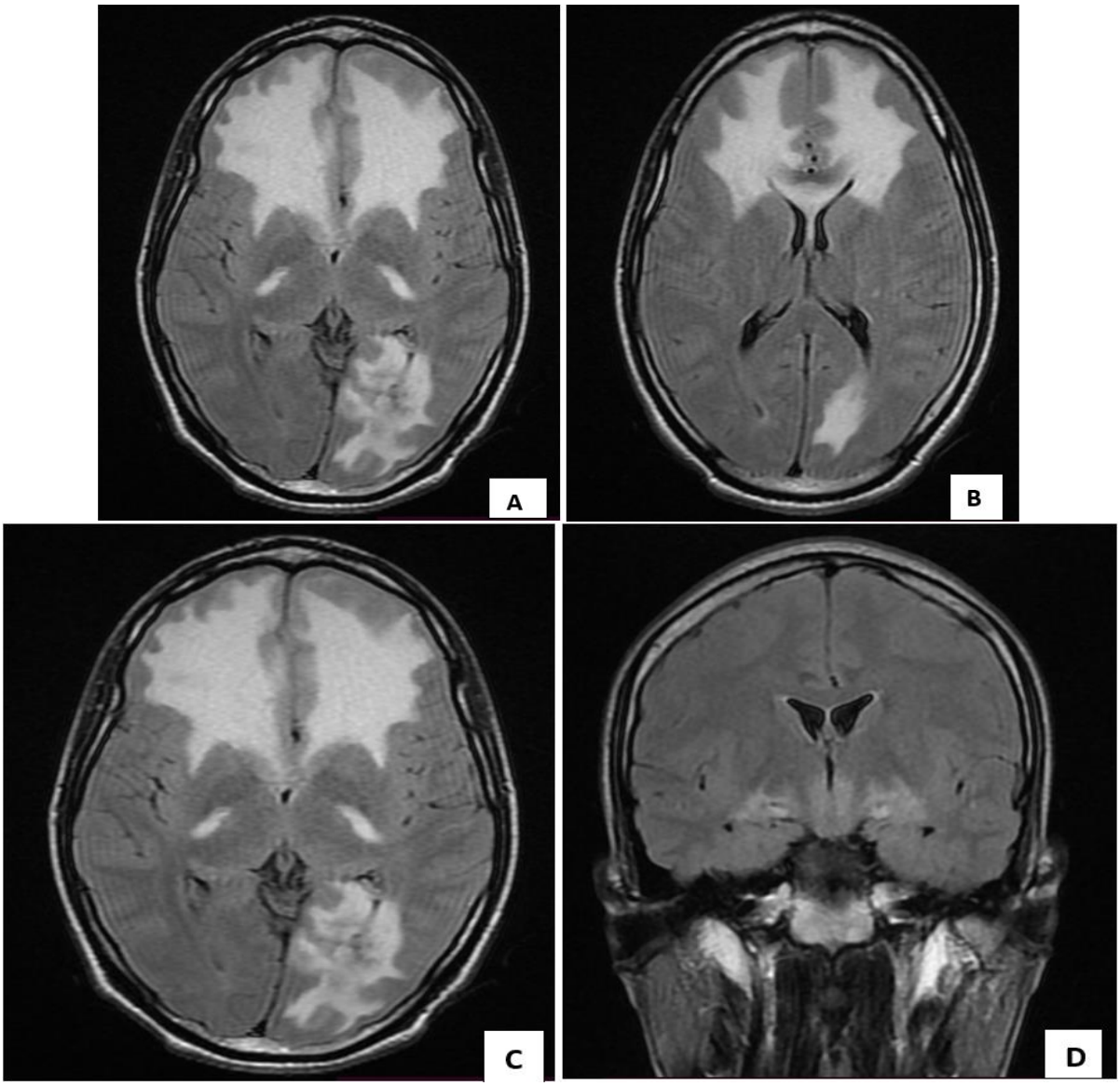

Figure 1: MRI sequence T2 FLAIR, axial (A, B, C) and coronal (D) slices showing hyperintensities in the subcortical white matter, sparing the U-fibers, in a bilateral symmetrical frontal distribution extending to the corpus callosum, periaqueductal region, hypothalamo-pituitary region, optic chiasm, optic tracts, and mammillary bodies. A second lesion with similar characteristics was found in the left occipital region. Additionally, hyperintensities were observed in both posterior limbs of the internal capsules and deep white matter regions

Post-contrast sequences revealed multiple punctate and nodular enhancements within these lesions, along with pachymeningitis in the bifrontal area and at the skull base along the midline (Figure 2). Diffusion-weighted imaging showed no abnormal signal, and spectroscopy revealed a lactate peak (Figure 3).

Associated findings included pansinusitis and osseous abnormalities with T2 hyperintensity and marked enhancement of the skull base bones, as well as enhancement of the right masticatory muscles without a detectable mass (Figure 2).

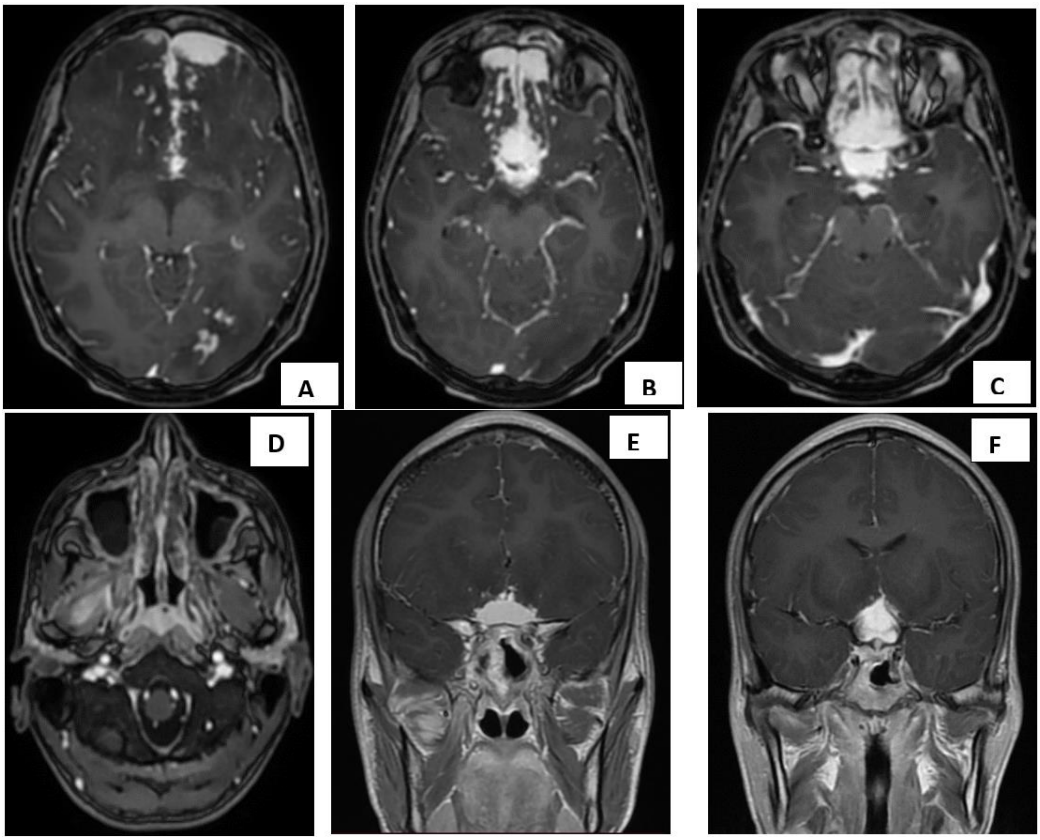

Figure 2: MRI 3D T1 sequence with gadolinium injection, axial (A, B, C, D) and coronal (E, F) slices demonstrating multiple punctate and nodular contrast enhancements within the lesions, along with pachymeningitis in the bifrontal area and at the skull base along the midline. There is also exaggerated enhancement of the skull base bones and the right masticatory muscles, without any detectable mass.

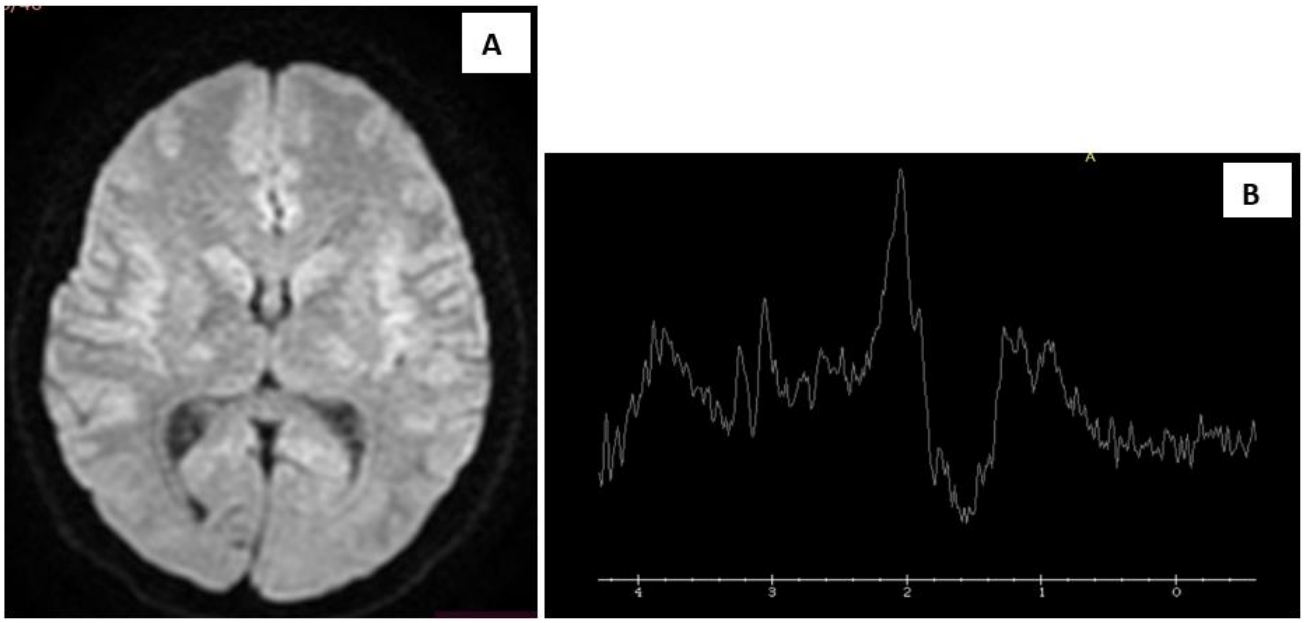

Figure 3: Diffusion MRI sequence (A) and spectroscopy (B): No abnormalities observed on the diffusion sequence (A), lactate peak detected on spectroscopy (B)

These MRI findings were compatible with granulomatous involvement of the brain, bones, sinuses, and soft tissues, highly suggestive of neurosarcoidosis.

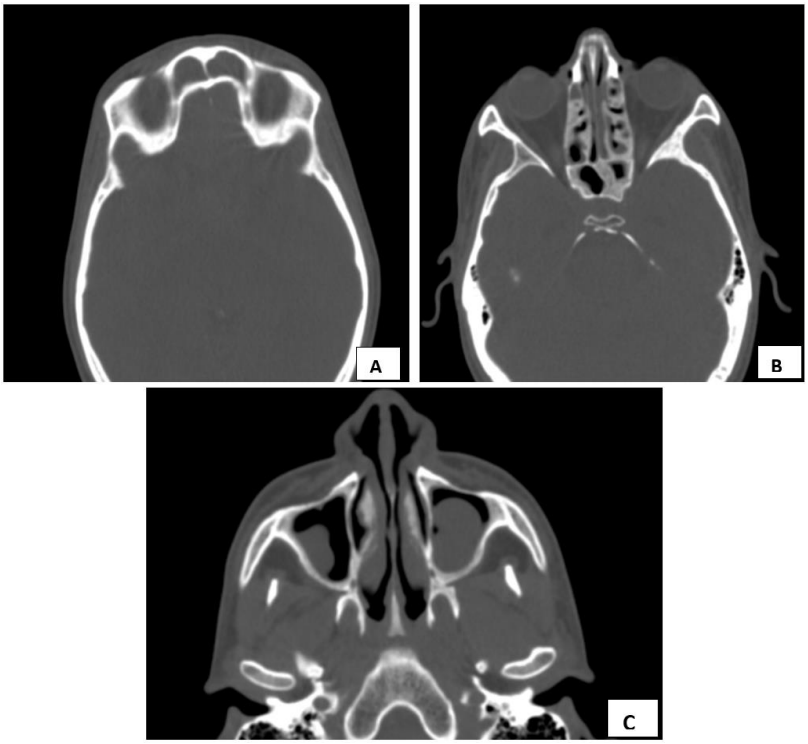

Figure 4: Non-contrast CT scan, axial slices (A, B, C): Pansinusitis

Further CT imaging of the sinuses and thoracoabdominopelvic region revealed mediastinal and deep abdominal lymphadenopathy and pansinusitis, consistent with systemic sarcoidosis (Figure 5).

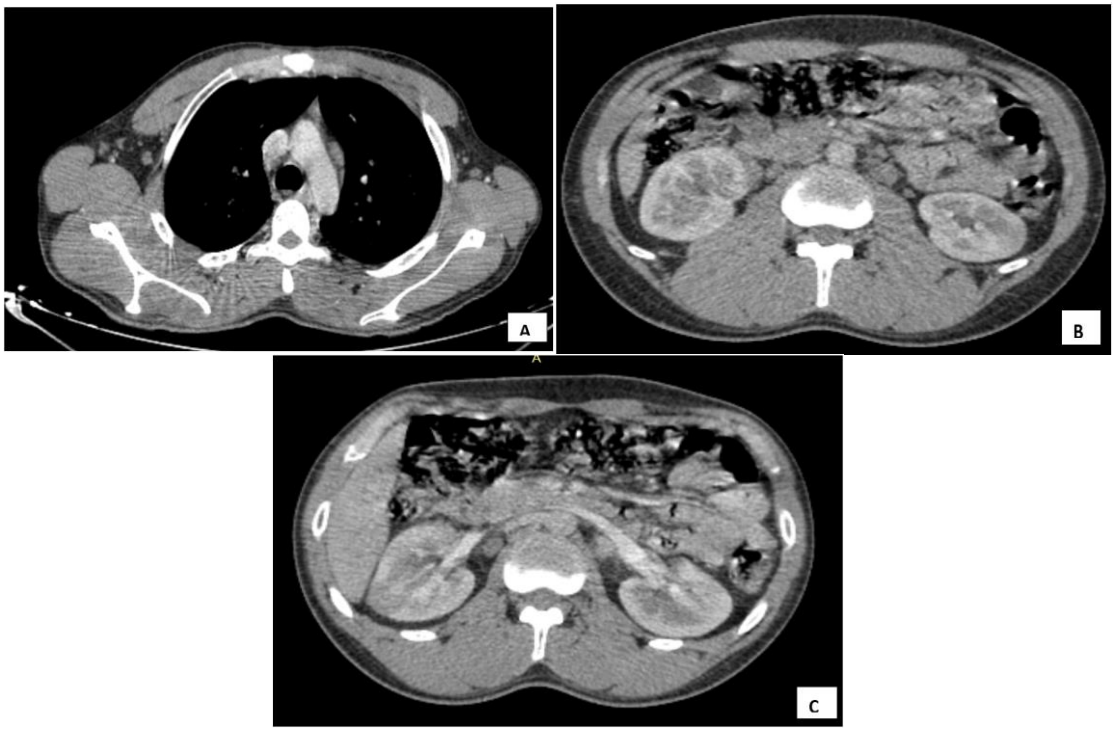

Figure 5: Thoracic CT scan with mediastinal window (A) and abdominal CT scan with contrast injection (B, C): Mediastinal and deep abdominal lymphadenopathy

Standard treatment includes corticosteroids, and in some cases, immunosuppressants to control systemic inflammation.

DISCUSSION

Sarcoidosis is a complex granulomatous disease that can affect multiple organs [1]. Neurological

involvement is a rare but severe manifestation, associated with significant morbidity and mortality [2].

Neurological involvement occurs in 5 to 10% of cases according to various series, and may reach up to 20% in autopsy studies. In 48 to 66% of neurosarcoidosis cases, sarcoidosis has not yet been diagnosed, and neurological signs are the revealing features [3].

Due to the variability in symptoms and imaging, diagnosis is often difficult or uncertain, especially in isolated neurological involvement [4]. The challenge of neurosarcoidosis, often called the “great imitator,” lies in the need to consider it early, prompting appropriate investigations to gather sufficient diagnostic evidence [1].

Diagnosis remains difficult despite advances in complementary investigations. Only histological confirmation from neural tissue can definitively establish the diagnosis. In other cases, diagnosis relies on the combination of clinical and/or paraclinical evidence, and if possible, histological findings from another organ [1].

Neurological involvement may affect the nervous system or its coverings, including the dura mater (29–50%), leptomeninges (31%), subarachnoid/perivascular spaces (preferential involvement of basal cisterns, optic chiasm, hypothalamus, pituitary stalk), cranial nerves (34–50%), optic nerves (28%), brain parenchyma (22%), and spinal cord (25%) [5].

Morphologically, the disease consists of localized or diffuse infiltrative granulomas, and the absence of abnormal enhancement does not exclude the diagnosis [5].

The key to diagnosis is the presence of solitary or multifocal CNS masses associated with an abnormal chest radiograph. MRI remains the modality of choice for diagnosing neurosarcoidosis. Its sensitivity for detecting lesions, whether cerebral or spinal, is superior to that of CT.

On T2/FLAIR sequences, the lesions typically appear hyperintense, although they may initially be T2 hypointense. The most common T1 findings after gadolinium injection include leptomeningeal, dural (diffuse or nodular), or parenchymal enhancement [2].

Multiple masses are observed in 35% of cases and solitary in 15%. Central necrosis is rare. Occasionally, perilesional edema, calcifications, or mass effect with intense contrast enhancement may mimic tumors, particularly meningiomas, gliomas, lymphomas, or metastases.

In cases where cerebral vasculitis is suspected, intracranial vascular sequences such as time-of-flight (TOF) and 3D-TOF can help detect the so-called "string of beads" appearance [1]. If suspicion remains high and the imaging is negative, cerebral angiography, which has greater sensitivity for small-caliber vessels (<500 µm), should be considered.

During follow-up, the disappearance of contrast enhancement is a sign of favorable evolution, whereas persistence or new enhancement should raise concern and prompt a reassessment of the therapeutic strategy [1].

First-line treatment includes corticosteroids. In refractory cases, immunosuppressants and TNF inhibitors may be used [1]. This disease requires regular evaluations of disease activity and monitoring for treatment-related side effects, hence the need for close collaboration among radiologists, neurologists, and other specialties involved in patient care.

CONCLUSION

Although rare, neurosarcoidosis diagnosis relies on a multimodal approach combining clinical, imaging, and histopathological data when available. Radiologists play a crucial role in identifying typical imaging patterns and facilitating early and accurate diagnosis. Optimal management of this complex and debilitating condition requires close interdisciplinary collaboration.

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