Self-Remitting Limbic Encephalitis

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Introduction

Autoimmune encephalitis is a neurological disorder caused by antibodies reacting against neuronal or glial compounds (1). Autoimmune encephalitis frequently manifests with delusions and psychiatric symptoms, leading to limbic encephalitis. In this disorder IVIG are therapeutic prospects. In limbic encephalitis, focal neurological signs, autonomic instability, and even more epileptic seizures and EEG abnormalities might increase the diagnostic challenge (2). Patients manifesting a similar neurological syndrome, often undergo to a series of long clinical and diagnostic tests. According to our experience, here highlighted, an EEG and a lumbar puncture are extremely useful in limbic encephalitis diagnostic work-up.

Methods

M. G., 58, presented a history of drugs abuse, cirrhotic hepatitis and hepatocarcinoma eradicated with ablation. The patient was admitted to our neurological center, for a gait disorder, involuntary movements of the right upper limb and behavioral and speech abnormalities. Brain MRI revealed a DWI, FLAIR and T2 hyperintensity in left fronto-insular cortex, a T1 hyperintensities in thalamic nuclei, concluding for an encephalopathy on an epileptogenic basis. An EEG showed a continuous 1.5-2 Hz delta potentials of wide voltage with a periodic trend in the left hemisphere, non-responsive to drugs. During the clinical observation, he developed mild ptosis in right eye, nystagmus, ataxia with right and retropulsion, fluctuation of consciousness, behavioral disorder, lateralized focal motor epileptic seizures, with a dystonic posture of the right upper limb. Preliminarily, we required the dosage of vitamins to exclude Wernicke Encephalopathy, and HIV test to exclude HIV-related disorders.

Results

a new standard EEG showed a pattern of lateralized left-sided epileptic anomalies, with delta band rhythm, without a clear modification of the track following the pharmacological infusion, led us to perform a lumbar puncture for the dosage of autoantibodies, suspecting a limbic encephalitis. LP showed RBC: 5920 cells/µL; WBC: 24 cells/µL, IgG: 61.3mg/L. The onconeural antibodies were determined with Anti-CV2.1 positivity (+) and Anti-Yo positivity (+++).

Conclusions

the relevance of our clinical case is related to a challenging diagnosis of limbic encephalitis with atypical presentation. The atypical features of our report are, at first, the clinical history and the neurological slowness of consciousness that might be more characteristic of metabolic encephalopathies. At second, our patient remitted by himself, without any therapies. This report would allow us to identify an internal management protocol and to construct a decision algorithm, which we recommend to use in cases of similar neurological disorders of difficult etiological classification.

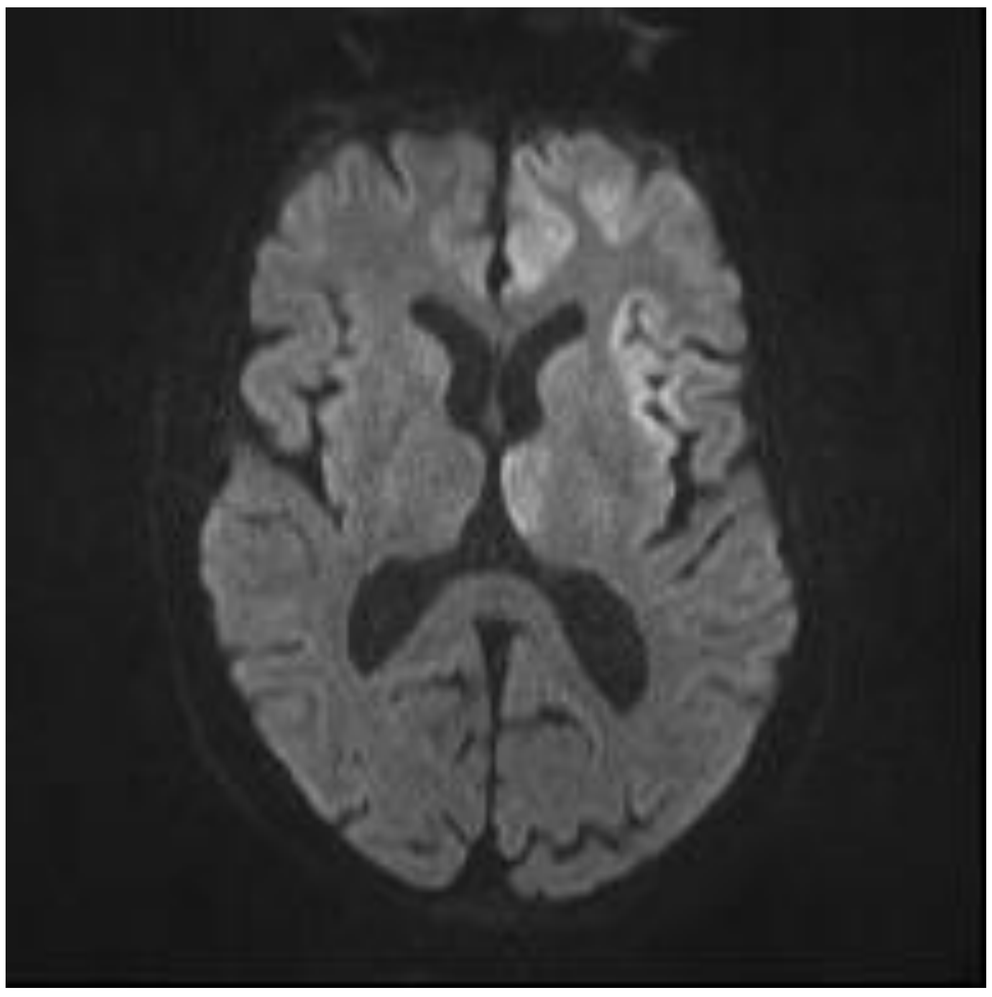

Fig.1. Brain MRI DWI hyperintensity in left fronto-insular cortex and thalamic nuclei.

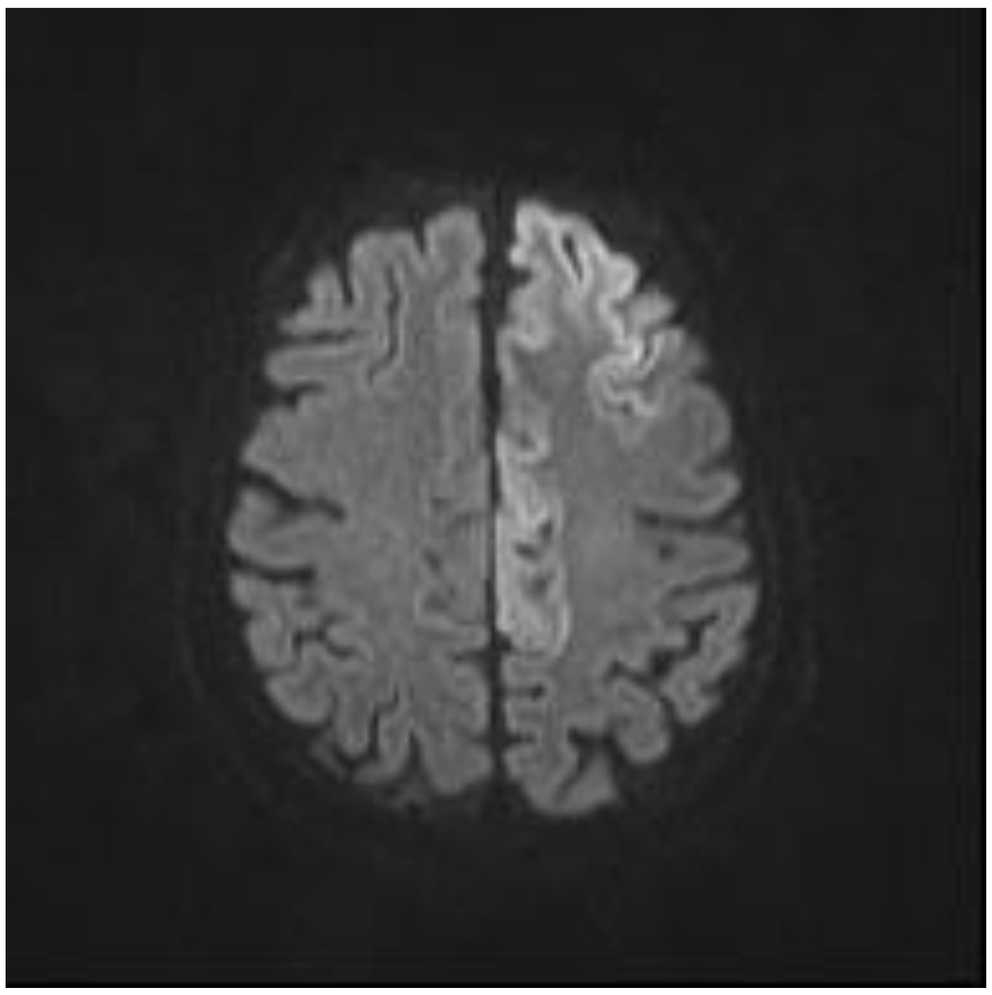

Fig. 2. Brain MRI DWI hyperintensity left fronto-insular cortex

Bibliografia

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