ANTIBODY NEGATIVE AUTOIMMUNE ENCEPHALITIS: A CASE REPORT

Carolina Sequeira[1*], Pedro Lopes[2]

[1] Ciclo Básico de Medicina, Universidade dos Açores, Ponta Delgada, Azores, Portugal

[2] Department of Neurology, Hospital do Divino Espírito Santo, Ponta Delgada EPE, Azores, Portugal

1 INTRODUCTION

Encephalitis is characterized by a severe inflammation of the brain; literature describes autoimmune as one of the most common etiology of non-infectious encephalitis [1][2].

Autoimmune encephalitis (AEI) may present a range of clinical features, including behavioural changes, impaired memory and cognition, psychiatric symptoms, involuntary movements, altered level of consciousness and seizures. However, AIE was not described until 2007 because of the lack of specific clinical symptoms as well as laboratory and MRI results [3]. Given the similarities in the clinical, imagological and laboratory findings with viral Encephalitis, the diagnosis of AIE is challenging and physicians need an increased awareness and clinical suspicion to make the correct diagnosis.

We present a patient with AIE, whose diagnosis was made when clinical, imagological and laboratory findings were combined, enabling specific therapeutic interventions that significantly improve the outcome, especially if performed at an early stage [5]. Noteworthy, patients with AIE may present a seronegative CSF and therefore a negative result does not exclude this diagnosis.

2 CASE PRESENTATION

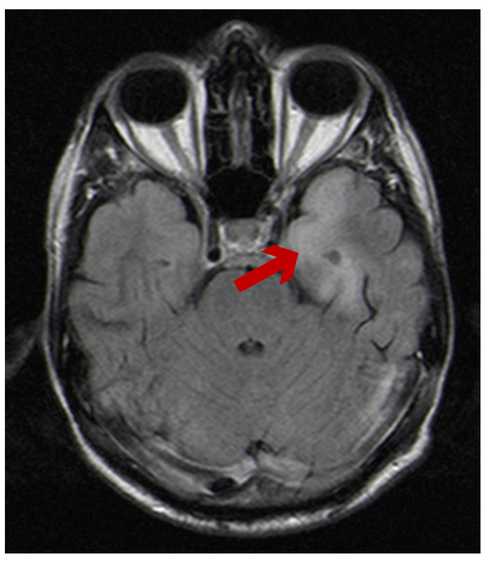
A **35-year-old healthy male**, without any significant past medical history presented at the ED in **January 2019** with:

speech and behaviour disturbances

suspicion of seizure; weight loss of 10kg in 8months

In the last five days: anorexia and vomiting

periods of confusion, sudation and fainting sensation



On neurologic exam he present with global aphasia. CSF showed pleocytosis (47cells). CT was normal, EEG revealed a probable complex partial status epilepticus. MRI showed hyperintense signal on T2/FLAIR on the left temporal lobe (fig.1).

Fig.1 MRI shows hyperintense signal on T2/FLAIR on the left temporal lobe (red arrow)

He received treatment with acyclovir 750mg 8/8 hours and phenytoin 750mg IV. He had a considerable improvement and was released with sporadic speech alterations and without full recovery of cognitive functions.

In **july**, **six months after**, he returns to the ED with:

speech and behaviour alterations; impairment of consciousness (not reactive to stimuli)

right hemiparesis; right arm clonic movements

EEG revealed a probable complex partial status epilepticus, that was refractory to the antiepileptic medication. CSF showed pleocytosis (25 cells).

Brain CT showed a hypodensity in the left frontoparietal region. MRI showed hyperintense T2/Flair signal in the left frontoparietal and occipital region (fig.2).

Negative bacteriology and autoimmune study throughout all the hospital stay.

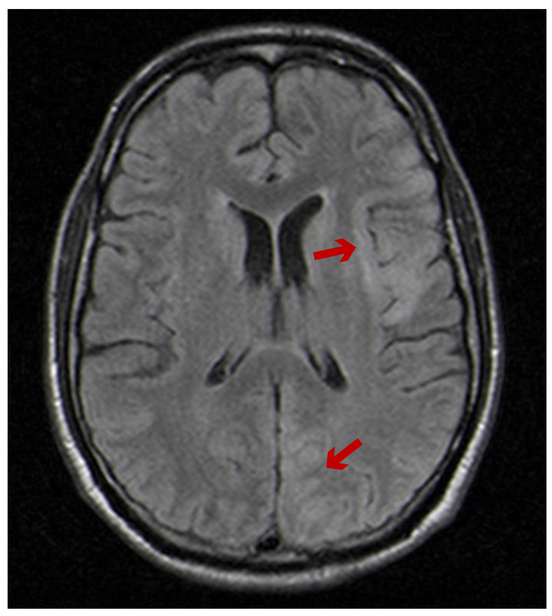
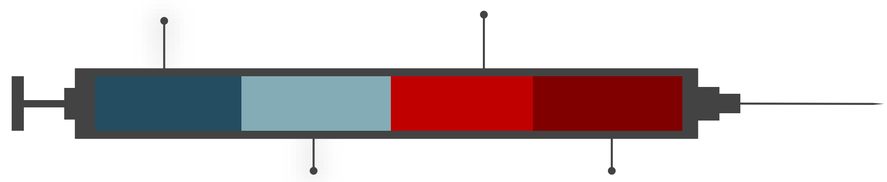


Fig.2 MRI hyperintense T2/Flair signal in in the left frontoparietal and occipital region (red arrow)

He was treated with 21 day cicle of acyclovir

5 days of immunoglobulin



5 days of methylprednisolone

Was **released in August**

In November 2019, although maintaining a **normal neurological exam**, he still had a clear **limitation in time, space and self-orientation** and showed **considerable memory loss** with implications in the daily life activities. He obtained a score of 5 in MoCA [8].

3 DISCUSSION/CONCLUSION

This case presents the challenges of diagnosing AIE, since the patient, despite having the typical clinical presentation and imagological findings consistent with AIE, had negative results for antibodies in the CSF.

Acording to the criteria of Graus et al.(2016), the case presents a probable diagnosis of antibody negative AIE [4]. Which highlights the importance of considering the hypothesis of autoimmune etiology on the differential diagnosis of all patients presenting with clinical and MRI results suggestive of probable encephalitis regardless of the negative antibodies results.

In this case, acyclovir was given empirically for presumed viral encephalitis. The same reasoning can be used, prior to antibody results, with presumed AIE, because clinical presentation and treatment response of antibody negative cases does not differ significantly from definite AIE. Therefore, it is recommended to start corticosteroids, plasmapheresis or first line immunotherapy in suspected patients of AIE based on probable diagnosis. The treatment should not be delayed since evidence suggest early immunotherapy its associated with favourable outcomes and better prognosis [1][6][7].

4 REFERENCES

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