

Rabies Encephalitis: follow-up with electroencefalogram

Encefalite Rábica: seguimento com eletroencefalograma

DOI:10.34119/bjhrv6n6-041

Recebimento dos originais: 02/10/2023

Aceitação para publicação: 07/11/2023

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RESUMO

A encefalite rábica é uma encefalite viral rara provocada por um Lyssavirus, possuindo pouca descrição de seus achados no eletroencefalograma e exames de imagem cerebrais. Dessa forma, esse relato tem objetivo de descrever achados no seguimento com eletroencefalograma e ressonância de crânio em um paciente com encefalite rábica, correlacionando com a clínica.

Palavras-chave: Encefalite Rábica, eletroencefalograma, acompanhamento.

ABSTRACT

Rabies encephalitis is a rare viral encephalitis caused by a Lyssavirus, with little description of its electroencephalogram and brain imaging findings. The aim of this report is to describe the electroencephalogram and MRI findings in a patient with rabies encephalitis, correlating them with the clinical picture.

Keywords: Rabies Encephalitis, electroencefalogram, follow-up.

1 INTRODUCTION

Rabies encephalitis is a rare viral encephalitis caused by a Lyssavirus, with few descriptions of its findings on the electroencephalogram and imaging tests. The

electroencephalogram is a functional exam, frequently used in the intensive care environment to: support diagnosis of pathologies, detection of status epilepticus, epileptiform paroxysms, level of sedation, among others. Therefore, this report aims to describe follow-up findings with electroencephalogram and brain MRI in a patient with rabies encephalitis.

2 CASE REPORT

A 2-year-old child presented with fever (39°C), irritability, drowsiness, seizures, ataxia with subacute onset. There are no previous comorbidities. He suffered a scratch on his left leg from a fox two months before the onset of symptoms, without taking anti-rabies prophylaxis. The brain MRI performed on the 15th day after the onset of symptoms and the cerebrospinal fluid analysis performed on the 9th day did not show any abnormalities. The suspicion of rabies encephalitis persisted due to a scar on the left leg by a fox. The cerebrospinal fluid analysis on the 16th day showed: leukocytes 106; polymorphonuclears: 76% proteins:117; glucose: 35mg/dl. The diagnosis of rabies was carried out through a saliva test and PCR in the cerebrospinal fluid.

Serial electroencephalogram (EEG) assessments were carried out, which showed:

On the 15th day, the patient presented with oxygen desaturation and decreased level of consciousness, requiring intubation. The electroencephalogram shows diffuse delta slowing with the presence of asynchronous spindles (Figure 1).

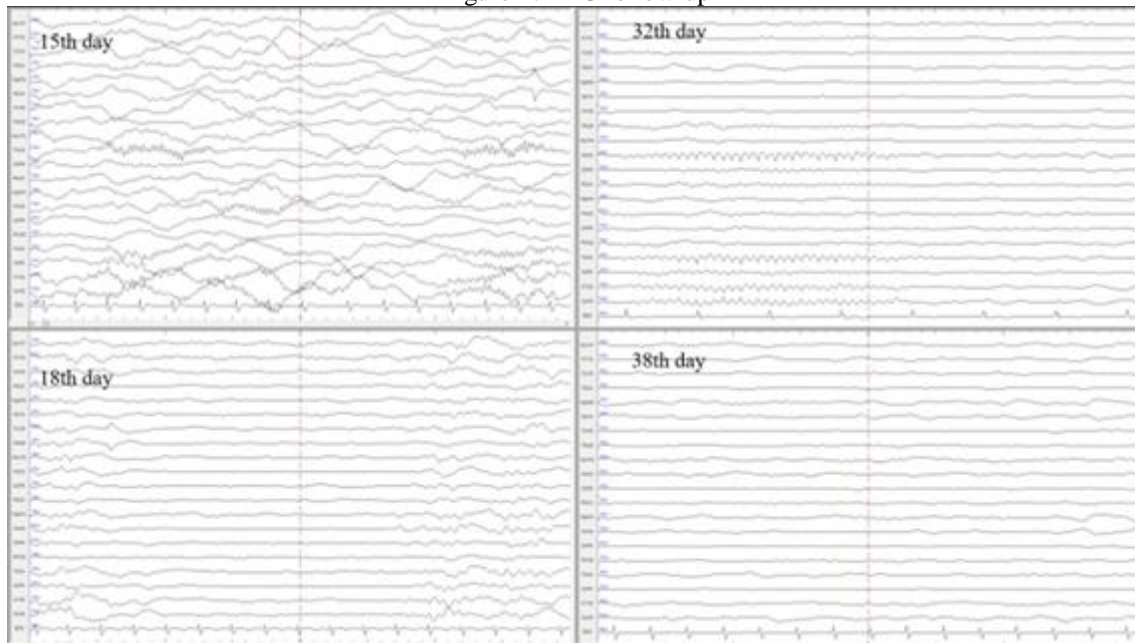
On the 18th day, the patient appeared comatose, with preserved brainstem reflexes. At this moment, the electroencephalogram shows a burst-suppression pattern, with bursts consisting of delta and theta activity (lasting 3 to 5 seconds) and an interburst interval lasting between 4 and 6 seconds (Figure-1).

On the 22nd day, the patient developed an absence of brainstem reflexes.

An electroencephalogram was performed on the 32nd day, which showed a burst-suppression pattern, with the bursts consisting of rhythmic theta activity (6Hz) in the left frontocentral region, lasting up to 4 seconds, and a lack of rhythms in other regions. The interburst interval was up to 15 seconds of activity suppression (Figure-1).

On the 38th day, the electroencephalogram showed suppressed activity, with rare bursts of rhythmic theta activity (below 20uV) in the left central frontal region (Figure-1).

Figure-1: EEG follow-up



Source: Authors

The last brain MRI performed on the 48th day demonstrated: areas of diffuse hyperintensity in the cortex and basal ganglia on T2 and Flair, with no expression on the diffusion sequence. The patient died on the 51st day after the onset of symptoms (Figure-2).

Figure 2: Brain MRI follow-up



Source: Authors

3 DISCUSSION

Rabies is a viral encephalitis caused by a Lyssavirus. It is transmitted through bites or scratches from mammals that carry the disease. It has two forms: the furious form and the paralytic form¹. In the present case, the form was classified as furious. It is characterized by the presence of fever, spasms, altered consciousness and seizures. The incubation time varies from two weeks to 3 months¹.

The clinic is the main tool for diagnostic suspicion. As noted, MRI and cerebrospinal fluid analysis may be normal initially and this does not exclude the diagnosis. Despite reports from survivors, mortality from the disease is still close to one hundred percent².

There are descriptions of multiple EEG patterns of this encephalitis, such as: slowing, periodic discharges³, electrographic crises³, suppression of activity simulating brain death⁴, monorhythmic frontal alpha⁵, among others. None of them are specific to the disease. In the present case, it was possible to monitor, through the electroencephalogram, the catastrophic evolution of the disease, with patterns of progressive hypometabolism⁶, such as: diffuse slowing, burst-suppression and suppression of activity, respectively. This evolution in the electroencephalogram took 40 days and was associated with the patient's clinical worsening.

In one of the few published cases of rabies encephalitis survivors, the improvement in electroencephalogram findings preceded the patient's clinical improvement⁷, reinforcing the role of the electroencephalogram in follow-up. The EEG has no specific findings for rabies encephalitis. However, it serves as a bedside examination with the function of monitoring the level of sedation, identifying subclinical epileptic seizures and monitoring the patient's progress.

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