HERPES SIMPLEX ENCEPHALITIS

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ABSTRACT

Herpes simplex encephalitis (HSE) is a serious disease, with increased risk of morbidity and mortality. We present the case of a 55 years old male who was admitted in our clinic with the suspicion of encephalitis with a MRI scan highly suggestive for HSE.
Key Words: herpes simplex, encephalitis.

BACKGROUND

The first pathological description of the herpes simplex encephalitis (HSE) was made in 1944 in an infant, and shortly afterward in an adult by Zarafonetis (1).

HSE is caused by herpes simplex virus type 1 (2). It is a serious disease, with increased risk of morbidity and mortality, despite the advances of the antiretroviral therapy (2). In the absence of effective antiretroviral therapy, the mortality is about 70%, with only 2,5% of patients regaining normal neurological functions (3).

HSE is an acute or subacute illness, causing general and focal neurological signs. There are no pathognomonic clinical findings that can reliably differentiate HSE from other neurological diseases with similar presentations (e.g. other encephalitis, brain abscess or tumors) (4), although headache, fever, confusion, behavioral changes, focal neurological signs and abnormal cerebro-spinal (CSF) fluid are suggestive for HSE. Brain imaging studies with localized temporal abnormalities are highly suggestive for HSE. Magnetic resonance imaging (MRI) is preferred because computer tomography (CT) is less sensitive, approximately one third of the patients having normal CT scans at presentation.

CASE STUDY

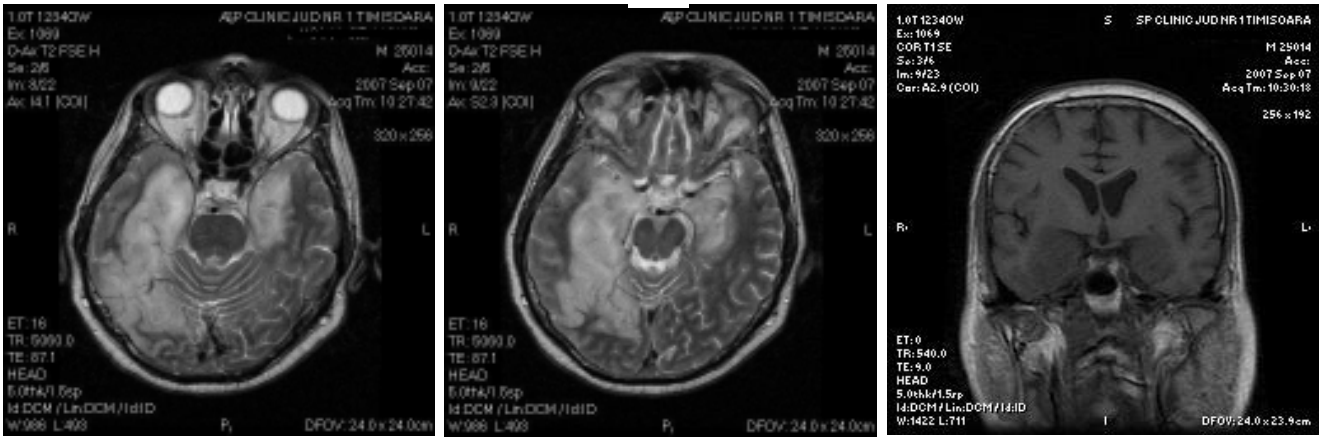
We present the case of a 55 years old male who was admitted in our Neurology Department with the suspicion of encephalitis. The patient was hospitalized in the Neurology Clinic from Resita for 10 days because he had an anamnestic episode of loss of consciousness followed by headache, confusion and vertigo. The CT scan performed was normal and the CSF analysis showed a negative Pandy reaction, 18 lymphocytes/mm^3 with no detectable microbial agents. Three days after, he was released, his status deteriorated, presenting Jacksonian motor seizures, fever (38-40°C) and confusion and he was sent to the Clinic of Infectious Diseases from Timisoara. Here, another LCR study was performed revealing: proteins: 0,32 mg/dl, glucose: 62 mg/dl, lymphocytes: 10/mm^3 and erythrocytes: 5/mm^3. He received antibiotherapy, antitermic and antiepileptic drugs and vitamins, but he was still confused, with repeated focal motor seizures and became aggressive. He was sent for a consult to our Neurology Department and we decided to transfer him in our clinic for further investigations.

The neurological examination revealed a conscious, hardly cooperant and temporo-spatial disoriented patient, without meningeal signs. Gait was possible but hesitant. At the cranial nerves examination there was a horizontal nystagmus. Otherwise his neurological examination was normal.

In our clinic, MRI scan performed showed extended hyperintense signal in T2-weighted images, hypointense in T1, without contrast enhancing, deep in the temporal lobes, bilaterally. The lesion was extended at the right cortico-subcortical occipital areas. The images did not correspond to an arterial territory, suggesting an encephalitis, very probable herpetic.

The patient received therapy with Acyclovir, antiepileptic and neurotrophic drugs, but despite adequate medication, his status started to deteriorate. Firstly, the focal seizures became more

Figure 1
T2 and T1 brain MRI sequences showing a focal inflammatory process located on the mesial part of temporal lobes, bilaterally.



frequent, the next day he presented generalized tonico-clonic seizures, and soon he become unconscious, with respiratory failure and pneumonic bloc. He was transferred to the Intensive care unit where he was mechanically ventilated. Ten days later, he was transferred to the clinic from Rest to continue his treatment, being stabilized, conscious, breathing through a T tube and starting to recover.

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