HHV-7 ENCEPHALITIS AS THE FIRST MANIFESTATION OF IMMUNODEFICIENCY

Joana Rabaçal*,1, Teresa Romão*, Mafalda Teixeira** and Tomas Lamas*

*Hospital Egas Moniz, Lisboa., **Hospital Professor Doutor Fernando Fonseca, Amadora.

ABSTRACT

KEYWORDS Encephalitis, human herpesvirus-7, adult, immunocompetent, glioblastoma multiform

Introduction

Human herpesvirus 7 (HHV-7) is a T lymphotropic herpesvirus first isolated in 1989 [1]. It’s a ubiquitous virus that is present in more than 95%. Infection usually occurs in childhood and peaks at around three years of age, followed by a latent state with possible reactivation in case of immunodeficiency.[2] The entire spectrum of clinical manifestations is yet to be determined, ranging from non-specific febrile illness exanthema subitum to febrile seizures [3,4] This report refers to an immunocompetent 57-year-old man who developed a status epilepticus in association with herpetic encephalitis due to HHV-7.

Learning points

• Human herpesvirus 7 is present in more than 95% of adults, but its role in human illness is yet to be determined, particularly in immunocompetent patients.
• The range of diseases associated with HHV-7 is grand, ranging from febrile illness in children, pityriasis rosea, infectious mononucleosis-like illness and central nervous manifestations.
• Isolating this virus in an apparent immunocompetent individual should prompt a diligent diagnosis investigation.

Case report

A 57 year-old male, with known history of poorly controlled arterial hypertension, chronic obstructive pulmonary disease and an active smoker, was admitted to the emergency department after three generalized tonic-clonic seizures with loss of consciousness. No symptoms were noted in the preceding days namely fever, neurologic deficits, skin rash or respiratory symptoms.

At admission the patient punctuated 3 in the Glasgow Coma Scale (GCS), was febrile (tympanic temperature of 37.9°C), had asymmetrical tetraparesis (predominantly left flaccid hemiparesis with left central facial palsy), conjugate gaze to the right and had no evidence of meningism. Initial complete blood work panel revealed no relevant alterations, including no electrolyte disturbances (tested negative for HIV and for toxic substances as well). Cranial CT scan showed traumatic subarachnoid hemorrhage on the left precentral sulcus and two small parenchymal hemorrhages on the right temporal lobe without arterial or venous lesions on CT angiogram. Triple anticonvulsivant therapy was started. Regarding the inability to maintain airway patency, the patient needed intubation and mechanical ventilation and was later transferred to the Intensive Care Unit (ICU).

A cerebrospinal fluid (CSF) analysis, performed as soon as possible after admission, revealed elevated protein level with 75mg/dL, normal glucose level (96mg/dL; plasma glucose: 126mg/dL), lymphocytes of <1 cell/µL, lactate 2.40mmol/L, albumin quotient pathologically increased, slight cerebral barrier function disorder, normal immunological profile, no intrathecal synthesis detected in the quotient scheme. Acyclovir was therefore started to cover for possible viral encephalitis. Further CSF analysis revealed a positive multiplex polymerase chain reaction (PCR) test for human herpesvirus 7 (HHV-7). The remainder microbiologic assessment of the CSF was negative, including for mycobacteria. Anti-neuronal antibodies were also negative.

Brain MRI performed 3 days after admission showed a right lenticular small acute ischemic lesion and a tumefactive hyperintense lesion on T2 of the right hippocampus and hippocampal gyrus, with a small associated haemorrhage, which was suggestive of herpetic encephalitis (fig.1). Electroencephalography showed focal slowing of the background and focal, scarce parox-
ysmal activity in the right temporal region, but no criteria for status epilepticus. There was favourable neurologic (GCS 14 at discharge from the ICU) and overall clinic progress, and at day seven, he was transferred to a medical ward with the diagnosis of status epilepticus caused by HHV-7 encephalitis in an apparently immunocompetent patient. It was possible to wean down the anticonvulsant therapy.

Figure 1: Brain magnetic resonance imaging showed a right lenticular small acute ischemic lesion and a tumefactive hyperintensive lesion on T2 (arrow) of the right hippocampus and hippocampal gyrus, with associated small haemorrhage (arrowhead).

During the time spent in the ward, a cerebral angiography was performed, showing vasculitic involvement with calibre irregularities of the posterior cerebral arteries, with multi-segment stenosis bilaterally with complete stenosis of the parieto-occipital right branch (fig.2). After 21 days of acyclovir, a second brain MRI was performed and showed persistence of the right temporal lesion, which raised the question of whether a neoplasm of the central nervous system should also be considered (fig.3). The patient was discharged 24 days after admission with a third MRI (fig.4) scheduled to clarify the persistent alterations on the right temporal lobe. Later, after brain surgery, a diagnosis of glioblastoma multiforme was made, making it a case of HHV-7 encephalitis in an immunodeficient host.

Figure 2: Angiography showed calibre irregularities of the posterior cerebral arteries, with multi-segment stenosis bilaterally with complete stenosis of the parieto-occipital right branch.

Figure 3: MRI-FLAIR acquisition showing the persistency of the cortico-subcortical lesion involving the right hippocampus and parahippocampus.

Figure 4: Contrast-enhanced MRI showed the heterogeneous irregular enhancement associated with the mass (glioblastoma multiforme; arrow) with the central non-enhancing area, consistent with necrosis.

Discussion
Because of its ubiquitous nature, it can be challenging to form a disease association for herpesvirus completely. [5] This is an example where the empirical treatment for a rare infection in what was initially interpreted as an immunocompetent adult patient was essential to his survival in order to buy time to the final diagnosis of glioblastoma multiform.

Funding
This work did not receive any grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest
There are no conflicts of interest to declare by any of the authors of this study.

References

