Imaging of Herpetic Meningoencephalitis. A Short Illustrated Focus

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Introduction

Herpetic meningoencephalitis (HME) is a sporadic, non-epidemic acute disease with an incidence of approximately 1 to 3 cases per million [1,2]. 95% of fatal cases in adults are due to Herpes Simplex Virus type 1. In neonatal period and children Herpes Simplex Virus type 2 is most often the culprit (80 to 90%).

The virus reaches the nervous system via the respiratory tract and then the trigeminal ganglion where it becomes latent in non immunocompromise patients [1,3]. The reactivation of the virus would be secondary to a decrease immunity due to genetic mutation on neurons and oligodendrocytes altering interferon production which normally inhibits viral replication [3,4]. This situation probably explains the preferential involvement of internal temporal lobe (early in the course) because of the anatomical proximity with the meckel’s cavum that houses the trigeminal ganglion (Figure 1) [1,5]. Extratemporal involvement only is also possible [2].

HME is an infectious, necrotizing and haemorrhagic (Figure 2) involvement of the brain parenchyma that is classically bilateral and asymmetrical. It may be associated with a meningeal involvement [2] (Figure 3). The usual clinical presentation associates acute or subacute behavioral disturbances, focal or generalized convulsions, focal neurological signs, cognitive disorders with headaches and infectious syndrome [2,3,6]. Aseptic meningitis can also be revealing. Signs of bradycardia, hypotension, syncope and hypothermia are related to hypothalamic involvement [5]. MRI is the most sensitive imaging current technique and plays a key role in diagnostic and follow-up. MRI imaging can guide the diagnosis early. It typically shows bilateral, asymmetrical involvement of the brain predominantly in the limbic system (medial temporal lobe, insular cortex, cingular gyrus, infero-lateral frontal lobe), with or without extension to the basal ganglia and the other lobes (Figure 3). Diencephalon and brain stem involvement are possible [1] Bonnici-Malia reported a case of hypothalamic and optochiasmatic involvement. Extra temporal involvement only can also be observed. CT is normal or the early course in 19% and shows the same aspects with a lower resolution than the MRI [2]. Due to the high mortality rate despite the treatment (20 to 30%), the diagnosis must be rapidly evoked in the presence of cytotoxic (early) or vasogenic edema associated with hemorrhage and meningeal enhancement [7]. According to some authors, MRI can distinguish an HSV lesion from that caused by HHV6 or Japanese encephalitis [5,8].
Herpetic meningoencephalitis due to HSV is rapidly progressive compared to HHV6 lesions. This distinction is important because HHV6 is not sensitive to acyclovir. Japanese encephalitis involves the thalamus firstly. However, aetiologies of limbic infectious lesions are numerous, apart from the HSV1 and 2; such as listeriosis, tuberculosis, varicella zoster virus, mycoplasma pneumoniae, enteroviruses, HHV6, Creutzfeldt-Jacob disease, West Nile virus, Influenza virus, Rocky Mountain spotted fever [2,8]. The gold standard diagnosis examination is virus detection in the cerebrospinal fluid by PCR (Polymerase Chain Reaction) with a high sensitivity and specificity; 96 and 99% respectively. False negatives are possible early in the course. Repetition of the exam is then recommended. Mendes et al. reported a case with a repeated false negative PCR examination. Diagnosis was confirmed at autopsy. This illustrates the value of the associated clinical presentation [9,10] in the management. Acyclovir treatment initiated early is the only prognosis factor that can improve the prognosis of this condition. Survival is usually at the cost of significant cognitive sequelae. Complete recovery without sequelae is also possible [2].

References

