Acute disseminated encephalomyelitis associated with Cytomegalovirus infection: a case report

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Introduction
ADEM is an immune-mediated inflammatory and demyelinating disorder of central nervous system (CNS), usually preceded by an infection, or rarely by a vaccination. It is more frequent in children and adolescents, but it may present at every age. Spinal cord MRI may show confluent intramedullary lesion, usually longitudinally extensive in addiction to abnormal brain, while a predominant involvement of spinal cord is rare. Gadolinium enhancement is variably present. The clinical presentation must be polysymptomatic and often includes encephalopathy; fever and meningeal signs are frequently reported.

Case report: A 36-year-old man was admitted to our hospital for persistent headache for ten days, fever (38°C), retroorbitary pain with blurred vision and vomiting.
Brain CT: normal.
Laboratory tests: mild leucocytosis and increase in alanine aminotransferase (ALT).
Neurological examination: no focal deficits, neither meningeal signs, while the patient complained urinary retention, hands hypoesthesia and numbness. The following day he developed encephalopathy with drowsiness, paraplegia and hypoesthesia with a D6 sensory level.
Brain and spine MRI: multiple lesions on T2/FLAIR sequences both in brain and mainly in spine, with no Gd-enhancing lesions (fig. 1 a, b, c)
Lumbar puncture: mild proteinorrachia and pleocytosis, there were no oligoclonal bands.
Serological evaluation for neurotropic viruses and bacteria: negative, except for a recent Cytomegalovirus (CMV) infection: positive IgM and IgG, and PCR. Culture of CSF was sterile and PCR for viruses resulted negative.

Clinical findings and course, neuroradiological features and serological tests were suggestive for ADEM associated with CMV infection.

Treatment and follow-up
Antiviral therapy with gancyclovir was administered for 17 days. High-dose intravenous methylprednisolone was started simultaneously (1 gr for 7 days, 500 mg for 3 days) and two plasma exchange were performed followed by intravenous immunoglobulins (80 gr for 3 days, repeated after 30 and 60 days).
On follow-up examination after five months the patient is fully ambulatory and only bladder dysfunction persists.
A brain and spinal cord MRI performed after five months shows resolution of both brain and spinal cord lesions (fig. 1 d, e, f)

Conclusions
To our knowledge, this is the fifth reported case of ADEM associated with CMV infection and shows some different findings compared to the previously described cases. The frequent occurrence of infections preceding ADEM supports the hypothesis of molecular mimicry. It has been shown that the human CMV major capsid protein shares sequences similarity with a myelin/oligodendrocyte glycoprotein (MOG 34-56). This structural homology provides the foundation for a putative autoimmune mechanism linking CMV infection to ADEM.
CMV infection must be considered as the specific infectious trigger in this case of ADEM; early treatment and resolution of viral replication positively affected the prognosis. The association between CMV and ADEM and other demyelinating inflammatory CNS disorders warrants further studies.

References