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NEUROLOGICAL PICTURE

Neurosyphilis with complex partial status epilepticus and mesiotemporal MRI abnormalities mimicking herpes simplex encephalitis

A 48 year old man suffered a 9 h dreamy state with automatisms. The neurological examination revealed disinhibited behaviour, a positive Romberg sign, and absent abdominal and weak tendon reflexes. Neuropsychological examination showed a selective reduction of short term spatial memory. A brain MRI performed after 2 weeks showed a prevalently cortical lesion in the right temporal and basal frontal lobes, which was slight hypointense in T1 weighted images and hyperintense in DP and T2 weighted images (fig 1), without enhancement. Carbamazepine was introduced. About a month later a second brain MRI revealed that the lesion was reduced to a small area in the right mesiotemporal region (fig 2), hypointense in T1 weighted and hyperintense in T2 weighted images, without enhancement after intravenous gadolinium; the right temporal horn was greater than the contralateral. CSF analysis showed normal glucose content, 12 lymphocytes/mm³, slight elevated protein level (63 mg/dL, normal range: 20–40), a positive Link's index (3.5, normal <0.75), 5 IgG oligoclonal bands, a positive VDRL test, a negative microscopic examination for mycobacteria and cryptococcus, and a negative PCR test for herpes simplex virus (HSV) 1 and HSV 2 DNA. Serum VDRL and TPHA were both positive. Serum antibodies to *Borrelia burgdorferi*, *Toxoplasma gondii*, and HIV 1 and HIV 2 were negative. The patient received intravenous penicillin G at a dosage of 4 million IU four times a day for 10 days.



Figure 1 MRI shows an area of high signal on T2 weighted image in right temporal lobe.

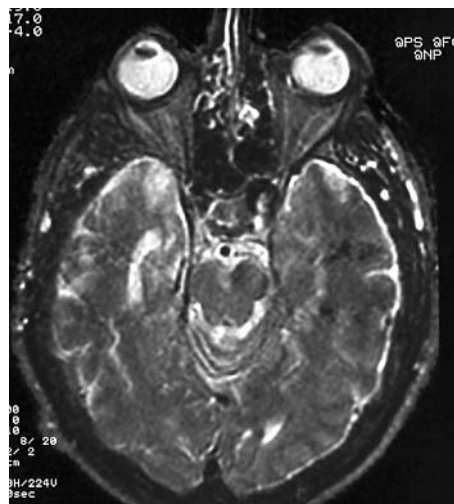


Figure 2 MRI shows a small area of high signal in temporal lobe with atrophic dilatation of temporal horn.

Complex partial status epilepticus (CPSE) has been observed with neurosyphilis.¹ The pathogenesis is thought to be the mesiotemporal localisation of luetic vasculitis. The MRI shows cortical and subcortical hyperintensity in T2 weighted images, probably due to both cytotoxic and vasogenic oedema.² The images are similar to those of HSV encephalitis.³ The T2 hyperintense lesion in the right temporal lobe and fronto-basal region only evident on the first MRI examination and mimicking HSV encephalitis may be due to transient oedema caused by the CPSE⁴ and spontaneously resolved before penicillin treatment. The small right mesiotemporal lesion evident on the second MRI examination may have a vasculitic origin and it may be responsible for CPSE. We recommend short term repetition of brain MRI and CSF examination for differential diagnosis of mesiotemporal syndromes.

E Marano, F Briganti, F Tortora, A Elefante, A De Rosa, F Maiuri, A Filla
Department of Neurological Sciences, Federico II University of Naples,
S Pansini, 5 80131 Naples, Italy; eirtm@tin.it

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