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LETTER TO THE EDITOR

Idiopathic bilateral facial palsy: is a causative role of anti-GM1 ganglioside and herpes simplex type 1 possible?

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Dear Sir,

Bilateral facial nerve palsy (FP) is a rare clinical entity often idiopathic or secondary to several diseases [1]. A role of Herpes simplex virus type 1 (HSV-1) in idiopathic FP is under debate [2]. We report here on a case of bilateral FP associated with anti-GM1 ganglioside antibodies and cerebrospinal HSV-1 DNA, who recovered after therapy with intra-venous immunoglobulins (IVIg).

A 68-year-old woman presented a sudden complete right sided FP that, despite treatment (methylprednisolone 40 mg/day i.m.), became bilateral in 1 week. The clinical history was devoid of relevant pathological events. At day 10 from the onset of symptoms, a neurological examination showed bilateral complete lower motor neuron FP (House-Brackmann grading system V on right and IV on left side). Meanwhile, there was no evidence of motor or sensory impairment of other cranial nerves and limbs, and deep tendon reflexes were normal. Neurophysiology, which included nerve conduction studies, motor, somato-sensory and auditory evoked potentials, was normal. Bilateral stapedial muscle and blink reflexes were absent in keeping with the diagnosis of bilateral facial palsy. MRI showed signs compatible with inflammation in both the facial nerves (Fig. 1). Routine blood investigations, including serological tests, paraneoplastic markers, CSF analysis and neuroimaging studies ruled out several known aetiologies. CSF nested PCR was positive for HSV-1 DNA and negative for other neurotropic viruses. To confirm the CSF positivity and the presence of an active infection, an aliquot of residual frozen CSF sample was thawed and used as inoculum on HSV-1 susceptible HEP cell lines. Though the freeze-thawing procedure may impair the viability of viral particles in biological samples, a cytopathic effect was observed after a 10-day culture (Fig. 2). However, no viral DNA was detected in cell lysates by nested PCR analysis from the same culture. Serum analysis revealed a past infection by HSV-1 since anti-HSV-1 IgG titre was 1:9,900 (v.n. $<$ 1:230) and the research for anti-HSV-1 IgM was negative.

As Guillain–Barre' (GBS) and Miller Fisher syndrome are likely causes, a serum anti-GM1 and anti-GQ1b antibody (Ab) assay was performed by ELISA. Increased level of anti-GM1 IgG Ab was found (0.083 OD at a dilution 1:640) since absorbance was more than three standard deviations above the mean levels found in serum of normal subjects (normal values $<$0.050 OD). Anti-GQ1b Ab was negative. Anti-GM2 Ab assay was not performed. No Ab for *Borrelia burgdoferi* and *Campylobacter jejuni* was detected in the blood.

IVIg therapy (400 mg/kg/day for 5 days) was immediately started, with a prompt improvement. Considering the amelioration of symptoms, the lack of evidence of active infection, and the current orientation for the treatment of facial palsy [3], we avoided the use of antiviral. After 6 months the patient showed a marked clinical recovery (House and Brackmann grade II, residual deficit more pronounced on the right side); serum anti-GM1 Ab was not detectable.

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