Wernicke's acute encephalopathy due to a B1 deficit secondary to alcohol intake.[5,6]

In terms of prognosis, the MRI findings of laminar cortical necrosis and hyperintensity at basal nodes, together with clinical data, duration of the anoxia, absence of pupil response,[7] absence of motor response[7] and early myoclonic status[8] allowed a poor evolution to be forecast for this patient.

In conclusion, the progression and dissemination of CPR techniques has generated an increase in the number of patients surviving CPR with HIE sequelae, and this has increased the need to determine clinical, electrophysiological or radiological markers allowing short-term and long-term prognoses to be established. NMR imaging provides prognostic information from the acute moment[3,9,10] and it is particularly helpful in cases of patients under sedation, where clinical parameters are less useful.[5]

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# Association of peripheral facial nerve palsy and seropositivity of HTLV 1, a case report

## Asociación entre parálisis facial periférica recurrente y seropositividad HTLV-1: a propósito de un caso

*Dear Editor:*

The aetiological diagnosis of peripheral facial nerve palsy (PFNP) covers a broad spectrum of pathologies; despite this, a highly variable proportion of between 62 and 93% are considered to be idiopathic or Bell's palsy.[1] We must not lose sight of the fact that this is a diagnosis by exclusion, and be alert to the data that lead us to suspect other causes in order to avoid overestimating the figures of idiopathic facial nerve palsy (IPFNP). One of these aetiologies is as yet emerging and still under study: seropositivity for the type 1 human T lymphotropic virus (HTLV-1).

We report the case of a 56-year-old male, originally from Peru and a resident of Spain for the last 18 years. High blood pressure and obesity were the most salient findings in his medical history. He initially presented PFNP on the left side that presented an incomplete response to steroid treatment; the mild erasure of the left nasogenian sulcus and Bell's sign persisted. Approximately 6 months later, the patient presented at the Emergency Department due to contralateral PFNP; the cerebral computerized tomography (CT) performed was normal. The patient was therefore sent home with a decreasing dosing scheme of steroids. One month later, he consulted due to clinical worsening, at which time the examination revealed that he could not close his right eye, Bell's sign, epiphora, and scant mobility in the lower right region of the face, which made it particularly difficult for him to eat and speak. The rest of the neurological examination was normal and there were no signs of lingua plicata or angiooedema. On admission, several complementary studies were carried out but failed to exhibit any significant alteration: blood panel, biochemistry, vitamin $B_{12}$, folic acid, autoimmunity, cell immunity,

immunoglobulins, tumour markers, lupus anticoagulant, ACE, serum blood tests for Borrelia, Brucella, syphilis, HIV, and herpes group viruses, among others, CSF biochemistry, CSF immunological testing, PCR of CSF for EBV, CMV and herpes group viruses, MRI of the brain and auditory canals. The ENG revealed an increased threshold, disintegration and low amplitude of both facial nerves, with a greatly increased latency in the right facial nerve; there were no electromyographic signs of active denervation, although the low number of units observed in the left frontal and orbicular muscles showed polyphasia and low amplitude.

At the present time, in the light of the normal findings on the complementary testing, a more meticulous history was taken, upon which the patient reported a history of probable tropical spastic paraparesis (TSP) in his ex-wife who was living in Peru. With this new information, a blood test for HTLV-1 was ordered that turned out to be positive in serum using enzyme immunoassay techniques (a titre of 62.52 anti-HTLV I/II IgG), western blot (positive for anti-HTLV I IgG), and PCR for HTLV I (677 copies/10E4 cells).

At no time were skin lesions, adenomegaly, hepatomegaly or splenomegaly found on physical examination; nor were analytical alterations revealed (lymphocytosis, hypercalcaemia, or increased levels of lactate dehydrogenase enzyme) which led to the suspicion of adult T-cell leukaemia/lymphoma (ATLL). Similarly, the patient failed to display signs suggestive of myelopathy on neurological examination.

HTLV-1 is a retrovirus that is endemic in Japan, the Caribbean islands, north-eastern South America, and central Africa. In 1980 it was related for the first time to ATLL[2] and later, in 1985, it was associated with TSP;[3] nevertheless, it is currently believed that it might also be involved in the pathogenesis of a wide variety of predominantly motor neurological diseases in the absence of myelopathy.[4,5] The mechanism of transmission is similar to that of other retroviruses, such as the human immunodeficiency virus (HIV), which may account for the fact that both infections often co-exist, demonstrating, in these cases, a higher incidence of myelopathy.[6]

PFNP was described for the first time in this context in 1986, when Bartholomew et al.[7] established the relationship between PFNP and adult T-cell leukaemia, but it was not until 1997 when the same group conducted a study[8] in Trinidad and Tobago that showed a significantly higher prevalence rate of HTLV 1 seropositivity among patients with IPFNP (20.7%) than in the two control groups: the general population (3.5%) and patients hospitalized in various departments for other reasons (5.6%). Although there are other works that corroborate this relationship,[9,10] it cannot be established for certain at the present time.

In spite of the enigmas surrounding this issue, we believe that there is sufficient scientific evidence to recommend HTLV 1 serology when conducting the aetiological study of recurring PFNP also displaying a poor initial response to treatment, particularly in patients who come from endemic areas.

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