

# Reversible focal encephalic abnormalities in a patient with Guillain-Barré syndrome

Alterações encefálicas focais reversíveis em doente com Síndrome de Guillain-Barré

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**Conflict of interest:** There is no conflict of interest to declare.

Received 11 December 2011; Received in final form 26 December 2011; Accepted 02 January 2012

The most frequent form of the Guillain-Barré Syndrome (GBS) is an acute demyelinating polyradiculoneuropathy. The majority of patients develop an ascending rapidly progressive tetraparesis, which can be associated with respiratory failure. Usually, there is a complete remission of the symptoms. The involvement of the central nervous system (CNS) in the GBS is rare<sup>1</sup>.

## CASE

A 68 year-old male complained of muscle weakness with rostrocaudal progression. The weakness was briefly preceded by

intense headache that started after physical straining. Four days later, the patient had flaccid tetraparesis with mainly crural involvement (grade 2/5 MRC in the lower limbs). He did not present fever or arterial hypertension. The electroneurographic study showed infrequent F waves responses with prolonged latencies. The brain magnetic resonance imaging (MRI) showed cortical serpiginous linear images on the left parietal-temporal-occipital region, suggesting an inflammatory component (Fig A, B and C). Analysis of the cerebrospinal fluid showed albumin-cytologic dissociation, with protein level of 199 mg/dL (reference: 10 to 45 mg/dL). Microbiological (Family *Herpesviridae*, HIV, *Campylobacter jejuni*, *Borrelia burgdorferi*, *Mycoplasma pneumoniae*, *Brucella*,

*Treponema pallidum*) and immunological studies (anti-ganglioside antibodies, ANA, ENA, anti-dsDNA, ANCA, and ACE) were negative in the CSF and in the peripheral blood.

On the seventh day, the patient developed respiratory failure. He had to be put on mechanical ventilation, and intravenous immunoglobulin (IVIG) was started. After a few days, a favorable clinical progression could be seen.

A new electroneurographic study revealed a demyelinating sensory and motor polyradiculoneuropathy.

A second brain MRI, performed one month later, showed the complete resolution of the lesion (Fig D, E and F). The patient started physical therapy and showed complete recovery four weeks later.

## DISCUSSION

This is a case of GBS with an uncommon clinical presentation, characterized by flaccid tetraparesis and reversible cerebral lesion.

The involvement of the CNS in the GBS is rare and often subclinical, but the widespread use of MRI has allowed the identification of different types of lesions<sup>1</sup>. Some cases of GBS

that involved meningoencephalitis, acute disseminated encephalomyelitis, vasculitis and reversible posterior leukoencephalopathy, have been described<sup>2,3</sup>.

In our case, it was not possible to define the exact etiology of the cerebral lesion. It could represent a focal infection secondary to an agent, which was not identified in the performed workup. However, its resolution without any antimicrobial specific therapy makes this hypothesis less probable. An alternative hypothesis is that the deposition of protein material in the cortical sulci, due to the lesion of the blood-brain barrier, may have started an inflammatory process and a self-limited focus of cerebritis. A few similar cases have been described in the literature. It is believed that these protein aggregates may be secondary to increased permeability of the blood-brain barrier and extensive immunological reaction, with protein exudation, which is in agreement with the CSF albumino-cytologic dissociation<sup>4</sup>.

Identical changes may occur at the spinal level, with an accumulation of perineural protein material, leading to the enhancement of the intra-thecal nerve roots<sup>5</sup>.

In our case, the absence of a known etiological agent, the clinical and laboratory responses to IVIG, and the disappearance of lesions without anti-infectious treatment suggest similar etiology.

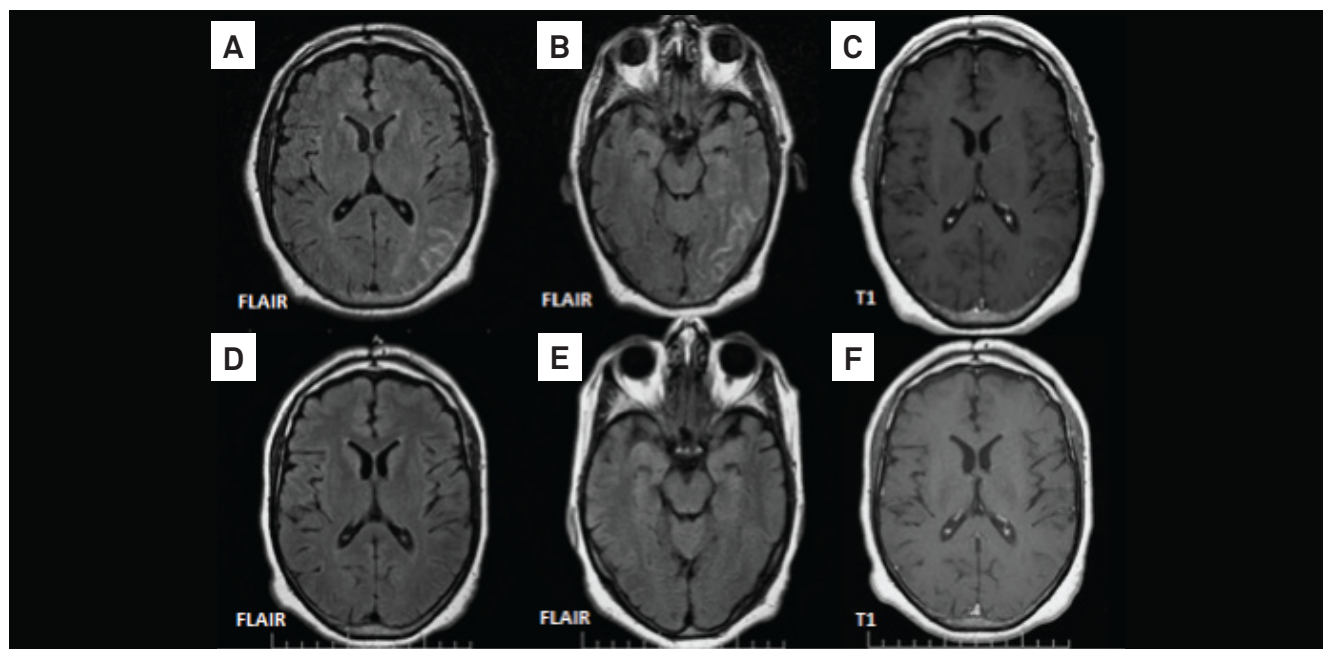


Fig. Brain mri. (A, b, c) first; (d, e, f) second.

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