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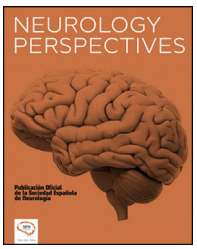

A case of primary HIV infection presenting as Guillain-Barré syndrome: Could it be a separate entity?

Un caso de primoinfección VIH debutando como un síndrome de Guillain Barré. ¿No será una entidad propia?

We present the case of a 63-year-old man who visited the emergency department due to neuropathic pain and sensory alterations in the abdominal region and the tips of the toes; symptoms had progressed over 12 days, ultimately hindering his ability to walk. The neurological examination revealed mild dysarthria, bilateral hip flexion strength of 4+/5 on the Medical Research Council (MRC) scale, with preserved distal strength, absent reflexes in the lower limbs and hypoactive reflexes in the upper limbs, hypoaesthesia from T4 to T12, and tingling in a glove-and-stocking pattern. Over the following days, the patient developed right-sided peripheral facial palsy (House-Brackmann grade III–IV). Neuraxis neuroimaging ruled out structural lesions, laboratory analyses yielded normal results, and CSF analysis revealed a protein level of 153 mg/dL, with 8 cells/mm^3. Glucose levels, cytology results, and multiplex PCR for central nervous system pathogens were normal or negative. As the patient met the criteria for motor-sensory Guillain-Barré syndrome (GBS), treatment was started with intravenous immunoglobulins (2 g/kg body weight over 5 days). Pain improved only partially, despite high doses of neuromodulators. At the peak of weakness, on day 4 of immunoglobulin treatment (day 19 after weakness onset), the patient presented asymmetrical facial diplegia and bilateral hip flexion weakness of 3/5 (MRC sum score of 54/60). Electroneurography studies performed on days 18 and 25 from symptom onset identified no alterations; the patient declined further studies in the following months. HIV serology tests yielded positive results, with a viral load of 1,820,000 copies/mL and a CD4+ count of 820 cells/mm^3; treatment was started the same day with dolutegravir and emtricitabine/tenofovir. This led to a marked clinical improvement, with hypoaesthesia restricted to the umbilical region and a Guillain-Barré Syndrome Disability score of 2 at discharge, and 0 at 6 months. No relapses were reported over the subsequent 3 years of follow-up.

Discussion

We present a case of GBS with atypical progression, characterised by predominantly sensory involvement, an atypical muscle weakness pattern, and delayed clinical improvement. Neuropathic pain was a predominant symptom and was resistant to neuromodulatory treatment. Interestingly, the patient presented hypoaesthesia in a "bathing suit" pattern, a distribution not previously described in the context of GBS secondary to primary HIV infection, but a typical pattern in polyradiculopathy secondary to acute intermittent porphyria. In our patient, weakness affected the facial muscles bilaterally and asymmetrically, as well as the pelvic girdle muscles. This pattern was also observed in the largest published series of HIV-associated GBS (n = 10), with facial muscle involvement reported in 60% of patients, while the lower limbs were similarly or more severely affected than the upper limbs in 80%.[1] While most cases of classic GBS peak within 2 weeks of symptom onset,[2] weakness in our patient was most severe on day 19 after symptom onset (coinciding with day 4 of immunoglobulin therapy and day 1 of antiretroviral treatment). This trend towards a delayed peak is also reported in the literature on HIV-associated GBS, occurring between days 11 and 60.[1,3,4] Our case reinforces the available evidence supporting the good functional prognosis of this entity when timely treatment with antiretrovirals and intravenous immunoglobulins is administered.[5] The clinical differences observed between HIV-associated GBS and classic GBS, as well as the differences in treatment response following the introduction of antiretrovirals, may be due to the disruption of HIV–peripheral nerve molecular mimicry and the indirect toxicity of HIV proteins. Genetic susceptibility has also been proposed as a pathogenic factor.[6] In conclusion, our case underscores the need for further research into the differences between classic GBS and HIV-associated GBS. The latter may constitute a distinct entity characterised by a longer disease course, a weakness pattern involving the muscles of the face and pelvic girdle, sensory alterations

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following a "bathing suit" pattern, and severe neuropathic pain, and may require treatment with antiretrovirals and immunoglobulins.

Patient consent (informed consent)

Written consent was obtained by the patient following institution's rules.

Ethical considerations

The authors declare that for this article, there was no animal or human experimentation.

Authors declare that all legal procedures of our own institution have been followed.

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Conflict of interest

There is no conflict of interest among authors.

The article is original.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.neurop.2025.100201.

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