AN ATIPICAL CASE OF GUILLAIN-BARRE'

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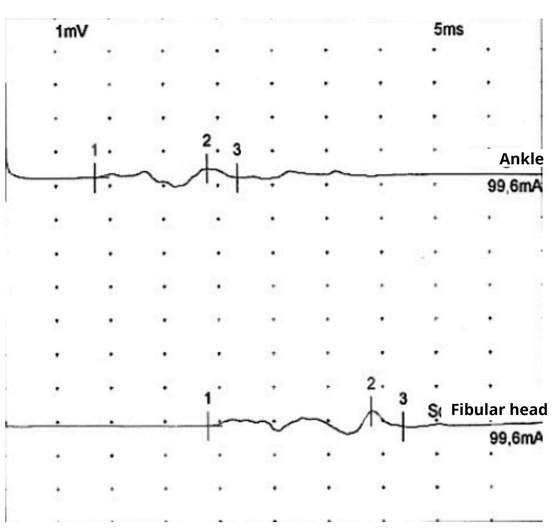
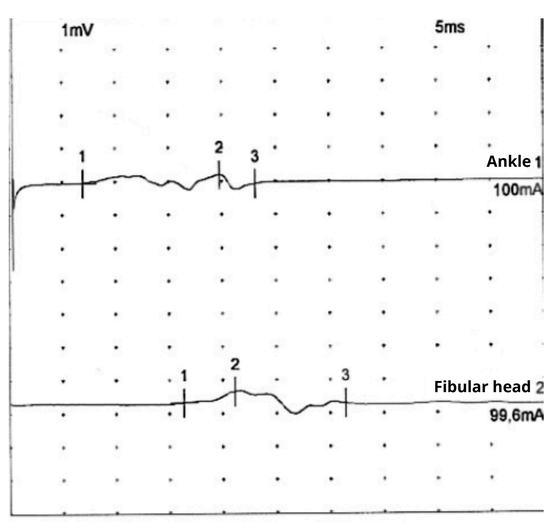
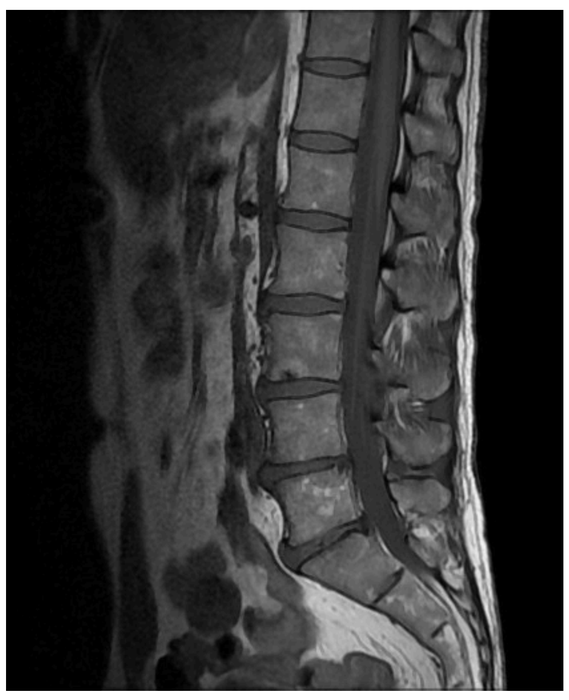
Introduction

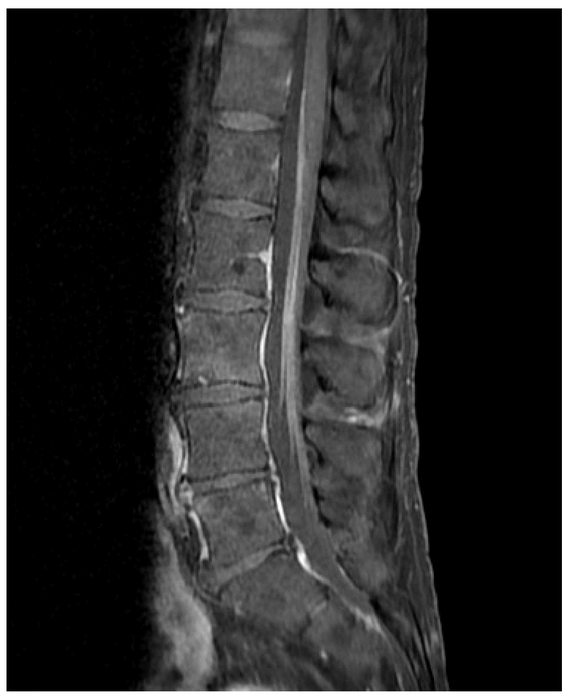
The Guillain- Barré syndrome (GBS) is an acute immune-mediated polyneuropathy and is one of the most common causes of acute, acquired weakness, often provoked by a preceding infection. GBS may be complicated in some cases by autonomic dysfunction or respiratory failure, which can potentially lead to death.







Lumbo-sacral MRI T1-weighted



Lumbo-sacral MRI T1 fat sat Gad

Case Report

A 53-year-old male was admitted to First Aid Room with mild distal weakness in all four limbs, with impairment of walking and fine hands movements. Some days before he had a slight fever and mild alteration of the bowel; he started to note weakness approximately 1-2 days after beginning of fever. In his past medical history, he reported Hodgkin Lymphoma treated with chemotherapy and lumbar herniectomy. First neurological examination revealed mild symmetric distal weakness in four limbs, no sensibility deficit and normal reflexes, except for absence of Achilles reflexes. The patient promptly underwent a cerebrospinal fluid (CSF) analysis, which showed a mild albumin- cytologic dissociation (protein 0.55 g/L, 0 cells); on the same day, standard therapy with intravenous immunoglobulins (IVIg) was initiated. A neurophysiological study performed the following day documented slightly slowed sensory conductions in the upper limbs and normal conductions in the lower limbs; reduced amplitude and chronodispersion of compound muscle action potentials (CMAPs); slowed motor conduction velocity, more pronounced in the left upper and right lower limbs; slightly increased latency with reduced persistence of F waves; poor muscle recruitment and an absence of spontaneous activity. These findings, although asymmetrical, were classified as acute inflammatory demyelinating polyradiculoneuropathy (AIDP). Over the first four weeks we observed a slow and asymmetrical worsening of strength, with pronounced weakness in right limbs and very mild impairment on the left; he was unable to walk, developed neuropathic pain and urinary retention. Reflexes were reduced but mostly persistent; he did not complain any breath or swallowing dysfunction. Spine MRI showed post-gadolinium enhancement of roots of the cauda without spinal cord involvement. A second neurophysiological study performed approximately two weeks later revealed a worsening of conductions, especially motor ones, with the disappearance of the right common peroneal nerve CMAP, in the absence of spontaneous activity. Antibodies against nerve gangliosides GM1 and GD1b were detected in serum and we ruled out alternative diagnosis with blood test (negative for HIV, TBE, Borrelia and inflammatory markers); Campylobacter was absent on feces. After 4 weeks, a second course of immunoglobulins was administered, while physical therapy was started one week after admission. After 28 days a slow improvement in muscle strength was observed, which continued over time, albeit partially. At the 4-month outpatient follow-up, the patient was able to walk with support and the neurophysiological study documented signs of axonal damage, such as markedly reduced or absent CMAPs, with signs of neurogenic damage in all 4 limbs; the conduction velocities of the main motor nerves remained reduced, with increased latencies.

Discussion

In this case the albumino-cytologic dissociation in the CSF and the NCS results were diagnostic, also supported by MRI for evidence of inflammation of the roots of the cauda and then by the presence of antibodies against gangliosides. We also ruled out alternative diagnosis serology for HIV, TBE, Borrelia, and the absence of inflammatory markers. The presence of IgG anti-GM1 and anti-GD1b antibodies, the partial clinical recovery, and the late finding of axonal damage superimposed on myelin damage led us to hypothesize a possible nodo-paranodopathy. We highlight some clinical aspects, such as the onset of symptoms a few days after the infectious syndrome, the partial preservation of reflexes, the absence of sensory symptoms, and especially the asymmetrical evolution of the weakness, which, although described individually in some cases, are not typical. In the literature, several classification criteria for GBS have been proposed, but our patient does not meet the criteria for the various subtypes, not even for the rarer variants. The atypical course and the absence of major complications could be due to the prompt initiation of therapy.

Conclusions

This case underscores the importance of early recognition of signs and symptoms of GBS Guillain-Barré, as timely initiation of medical therapy and management with physiotherapy can avoid major complications and lead to significant neurological recovery.

References

1. Arends S, Drenthen J, De Koning L et al. Electrodiagnostic subtyping in Guillain–Barré syndrome patients in the International Guillain–Barré Outcome Study. Eur J Neurol 2024;31:e16335
2. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barrè syndrome. The Lancet 2021;397:1214-28
3. Sejvar JJ, Kohl KS, Gidudu J et al. Guillain–Barré syndrome and Fisher syndrome: Case definitions and guidelines for collection, analysis, and presentation of immunization safety data. Vaccine 2011; 29:599-612
4. Uncini A, Vallat JM. Autoimmune nodo-paranodopathies of peripheral nerve: the concept is gaining ground. J Neurol Neurosurg Psychiatry 2017;0:1-9

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