Acute motor neuropathy with reversible conduction blocks and unusual anti-ganglioside complex IgM antibodies: an uncommon Guillain-Barré syndrome case

Aste R.1, Tacconi P.2, Fadda E.3, Salaris E.1, Pische M.1, Floris E.1

Introduction: Motor conduction impairment with conduction blocks (CBs) are typical electrodiagnostic findings in multifocal motor neuropathy (MMN), acute inflammatory demyelinating polyneuropathy (AIDP) variant of Guillain-Barré syndrome (GBS), chronic inflammatory demyelinating polyneuropathy (CIDP), hereditary neuropathy with liability to pressure palsies (HNPP); recently, a new electroclinical entity named “acute motor conduction block neuropathy (AMCBN)” has been proposed as incomplete form of the acute motor axonal neuropathy (AMAN)-GBS variant. Sensory nerve conduction is generally spared in MMN, AMAN and AMCBN. Several IgG or IgM anti-ganglioside antibodies have been found associated to different neuropathies till to become sometimes a serologic hallmark. We report a case of acute motor neuropathy with acute reversible conduction blocks and anti-GM1/GT1A/GT1B IgM complex antibodies.

Case report: A 24-year-old female was admitted to our department complaining of tingling and weakness in both hands. Three months earlier she was diagnosed with type 1 diabetes after a journey to Eritrea, where she complained of fever, diarrhea, weight loss and walking difficulties treated with antibiotics. No traumatic nerve injury nor new gastrointestinal symptoms were reported.

Neurological examination showed mild heel walking impairment, bilateral lower limb hyporeflexia, mild bilateral hand-grip weakness, mild stocking-glove hypoesthesia; cranial nerve exam was normal, nor patient complained about dysphagia or dyspnea.

Serial nerve conduction studies showed a motor polyneuropathy with segmental demyelination and reversible partial CBs. No significant involvement of sensory conduction or reduction of distal compound motor action potential (CMAP) amplitude were observed. CSF examination revealed increased protein content (97.8 mg/dL) with normal cell count; immunoblotting disclosed high titers of anti-GM1, anti-GT1A and anti-GT1B IgM. The patient was treated with IVlg (0.4 mg/kg/day) for 5 days without a significant benefit until two months later when, after an initial clinical and instrumental motor worsening, she reported clinical recovery and nerve conduction improvement occurred. CBs disappeared two months later; mild motor conduction slowing were still present in the distal segments of ulnar and median nerve six months after the onset.

Discussion and conclusions: In our patient the clinical course, the temporal relationship with a new-onset type 1 diabetes and the immunological profile were rather uncommon. The convergence of several pathogenetic mechanisms could explain the unusual clinical-immunological picture.

References