Electrophysiological abnormalities in iatrogenic botulism: two case reports and review of the literature.
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Therapeutic use of botulinum toxin type A (BoNT/A) is effective, and generally safe. Nevertheless, iatrogenic Botulism (IB) is rarely reported as a result of systemic spread of the BoNT/A, causing general weakness, bulbar symptoms and dysautonomia. Suggestive clinical feature are decisive to raise the diagnostic suspicion, which however needs a confirmation in the electrodiagnostic study, above all to exclude other treatable diseases. In this study, we report 2 patients who developed IB after receiving therapeutic doses of BT/A for spasticity.

Conclusions:

• There is no highly specific pattern of electrophysiological abnormalities in IB.
• Literature review and our findings suggest both decrement and increment as unexpected changes in IB.
• If there is clinical suspicion, the lack of altered response after RNS in weak muscle, represents a highly suggestive clue for the diagnosis of IB.

Patient 1
A 48-year-old Caucasian woman was initially treated with 400 U of BoNT-A (Botox®) for lower limb spasticity due to Multiple Sclerosis, in another clinic. A second treatment with BoNTs-A was performed two months later, at a higher dosage of 600 U. Two weeks after injection, the patient complained of double vision, dysphonia, dysphagia, generalized weakness, and dry mouth. Based on a suspected diagnosis of seronegative myasthenia gravis, a treatment with pyridostigmine was initiated without benefit. Five months later, following a third treatment with 400 U of Botox®, the patient referred to our ER for the appearance of general weakness and bulbar symptoms. At neurological examination, she presented bilateral diplopia, bilateral ptosis, slow reactive pupils, accommodation deficit, dysphonia, dysphagia, and tetraparesis prevalent proximally and in the upper limbs. Nerve conduction study (NCS) demonstrated a diffuse mild reduction of cMAP amplitude, with normal conduction velocity and no sensory abnormalities. Low frequency repetitive nerve stimulation (RNS) at 3 Hz in the right deltoid and the right orbicularis oculi showed no decrement (Fig 1). Neither decremental response, nor facilitation were observed at 3Hz and 30 Hz RNS respectively. Sympathetic skin reflex test showed normal response (Fig 1). The symptoms and the neurophysiological alterations fully resolved over three months, with no treatment.

Patient 2
A 32-year-old Caucasian man with lower-limb spasticity due to infantile cerebral palsy, was treated at our clinic with a dose of 2000 U of BoNT-A (Dysport®) divided between quadriceps and adductors muscles bilaterally. Ten days later, he complained of difficulty swallowing, hoarse voice, blurred vision, and generalized weakness. At neurological examination, he showed multi-directional diplopia, impaired accommodation, bilateral facial weakness, and proximal tetraparesis. Neurophysiological assessment was consistent for diffuse small amplitude cMAP, abnormal spontaneous activity (PSWs) and fibrillations, small and brief, but not polyphasic, MUAPs, with impaired recruitment. SFEMG of right extensor digitorum communis showed abnormal jitter (75.6 µsecs) and blocks occurrence respectively. Symptomatic skin reflex test showed normal response (Fig 1). The symptoms and the neurophysiological alterations fully resolved over three months, without treatment.