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Letter to the Editor

Before Attributing Therapy-Refractory Jaw Drop to Botulinum Toxin and Kennedy's Disease, Alternative Causes must be Thoroughly Ruled Out

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Letter to the Editor

We read with interest the article by Ju *et al*. about a 47-year-old man with bulbospinal muscular atrophy (BSMA) due to a CAG repeat expansion of 49 in exon 1 of the androgen receptor gene (AR), who developed jaw drop shortly after the injection of botulinum toxin (BTX) (100 mouse units) into the temporalis and masseter muscles on both sides for head tremors in a dental clinic [1]. Extensive investigations for myasthenia and myasthenic syndrome revealed no concomitant disease that could explain the persistent effect of the BTX injection [1]. The study is remarkable, but some points require discussion.

The first point is that a second problem that could explain the unusual reaction to the BTX injections was not sufficiently ruled out [1]. It is particularly important to rule out the possibility that the patient suffered from congenital myasthenic syndrome (CMS) that occurred in adulthood. Symptomatic or asymptomatic CMS patients often show a decreasing response to repetitive low-frequency nerve stimulation [2]. CMS patients with onset in adulthood may remain asymptomatic for years until they occasionally become symptomatic due to medication. An open mouth is a common feature of CMS [3].

The second point is that myasthenia and myasthenic syndrome were not sufficiently ruled out. To rule out myasthenia, it is recommended that LRP4 and titin antibodies also be tested. To determine whether myasthenic syndrome is present, it is crucial to measure the concentrations of presynaptic voltage-gated calcium channels. It should also be noted that approximately 10–15% of patients with myasthenia are antibody-negative.

The third point is that it is not clear why a dental clinic would treat head tremors [1]. Head tremor is the specialty of a neurologist and should be assessed and treated therapeutically by this specialist. In this context, it is also unclear why BTX was injected bilaterally into the masseter and temporal muscles for the indication of head tremor. Normally, the splenius capitis muscle is injected for the indication of head tremor [4].

The fourth point is that no adequate explanation was given as to why the patient suffered from sensory neuropathy [1]. Although sensory neuropathy has occasionally been described in BSMA [5], other primary and secondary causes of sensory neuropathy should be ruled out before attributing it to BSMA. What concomitant medications did the patient take regularly, and what comorbidities did he have?

The fifth point is that no cerebral MRI results were presented [1]. Were there lesions in the basal ganglia, brain stem, or cerebellum? According to panel B in Figure 1, T1-hyperintensities can be seen in both cerebellar hemispheres. Was this an artifact? It would also be helpful to know the results of other cerebral MRI modalities.

The sixth point is that single-fiber studies are missing. Single-fiber electromyography can be helpful in assessing neuromuscular transmission function, especially when jitter is increased or when the number of blocked responses is increased.

In summary, before attributing persistent jaw drop to a side effect of BTX in BSMA, all other possible causes of jaw drop must be thoroughly ruled out.

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