CASE STUDY

Objective Tinnitus from Palatal Myoclonus. Use of Botulinum Toxin: A Case Report

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Abstract  Objective tinnitus can have many different etiologies, palatal myoclonus being one of the less frequent. This type of tinnitus is generated by involuntary rhythmic contraction of the soft palate, which generates an audible click for the patient and for the explorer.

Botulinum toxin achieves temporary muscle paralysis through presynaptic inhibition of the acetylcholine level at the neuromuscular union.

We present a patient with long-term objective tinnitus, along with this patient’s response to botulinum toxin injection.

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Case Report

We present the case of a 78-year-old female patient with a history of arterial hypertension and depressive symptoms, in the context of chronic objective tinnitus.

We performed oropharyngeal inspection, otoscopy, nasofibroscopy, liminal tone audiometry, tinnitusmetry, tympanometry and distortion-product otoacoustic emissions (DPOAE), leading to the diagnosis of palatal myoclonus. The patient had previously been examined by computed tomography (CT) and magnetic resonance imaging (MRI) tests at the Neurology Service, and both assessments were within normal limits.


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The patient was intervened under local anaesthesia and direct vision with a 0° rigid endoscope. We injected 10 units (U) of botulinum toxin, subtype A (5U in the tensor of the soft palate [tensor veli palatini] and 5U in the elevator of the soft palate [levator veli palatini], bilaterally) (Figs. 1 and 2). We performed weekly controls during the first month and then after 3 and 6 months. Tinnitus disappeared after 7 days. On the 15th day we observed bilateral paralysis of the tensor and levator veli palatini muscles, noting only a slight contraction of the left soft palate, without associated symptoms. As a side effect, we observed open rhinolalia during the first week of treatment, which generated nasal fluid reflux on 2 occasions. There was no evidence of otalgia or increase of hearing loss (bilateral, moderate, and mixed hearing loss). Neither did we observe episodes of dysphagia or aspiration. Correct laryngeal function after injection was also observed during follow-up.

Tinnitus reappeared after 5 months, although its intensity was less than the initial, so we decided to administer a second dose of type-A botulinum toxin (30U).1,2

Discussion

Objective tinnitus can present various aetiologies, with palatal myoclonus being one of the most unusual. This rare pathology is generated by a rhythmic, involuntary contraction of the soft palate.

Botulinum toxin subtype A is indicated in all those pathologies resulting from muscular hyperfunction and autonomic dysfunction. It binds to cholinergic nerve endings, resulting in flaccid muscular paralysis.3 Its peak effect occurs within 7–14 days, and muscular function is re-established within 3–6 months. Its repeated use does not result in muscle atrophy or permanent degeneration.3

In the present case, the patient suffered objective tinnitus which had intensified to the point of significantly affecting her daily activities. In the diagnosis, we highlighted the repeated presence of a notch in the impedance curve which corresponded to the tinnitus observed in the patient. Oropharyngeal examination and nasofibroscopy were also consistent with the diagnosis. In order to calculate the dose, we began by using the lowest effective dose published in the literature,5–9 noting minimal side effects. We subsequently decided to increase the dose (30U), without observing a significant increase in its duration of effect.

The use of botulinum toxin for the treatment of palatal myoclonus in our case obtained optimal results with minimal side effects. The only inconvenient was the temporary effect of the toxin, which required repeated injections every 5–6 months.

Conflict of Interests

The authors have no conflict of interests to declare.

References