



Myasthenia Gravis after Botulinum Toxin Type A Injection

Botulinum Toksin Tip A Enjeksiyonu Sonrası Açığa Çıkan Myasthenia Gravis Olgusu

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Keywords: Myasthenia gravis, Botulinum toxin A, dystonia
Anahtar Kelimeler: Myasthenia gravis, Botulinum toksin tip A, distoni

Introduction

Botulinum toxin type A (BoNT type A) is a neurotoxin produced by *Clostridium botulinum*, which causes paralysis by presynaptically binding to the cholinergic nerve terminals at the neuromuscular junction and decreasing the release of acetylcholine (1). BoNT type A, first used to treat strabismus, is now widely used to treat dystonia, spasticity, and other movement disorders, and also autonomic nervous system disorders including hyperhidrosis and sialorrhea (2). As with the effects of the toxin, the adverse effects are also reversible. The most common and severe adverse effects of the toxin are unwanted and excessive weakness of muscle after injection, paralysis of adjacent muscles caused by diffusion of the toxin and iatrogenic botulism characterized by widespread paralysis including the bulbar muscles.

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction, which is caused by antibodies produced against acetylcholine receptors. Fluctuating weakness is the main characteristic of the disease.

A woman aged 80 years who presented with forceful contractions of the jaw and tongue, which caused difficulty in opening the mouth, was diagnosed as having oromandibular dystonia. Ten MU (mouse units) of BoNT type A was administered to each hypoglossal muscle once every 3 months. The patient was admitted to the emergency service with bilateral dropping of the eyelids, diplopia, dysphagia, general weakness, and shortness of breath, ten days after the 9th injection. Neurologic examination

showed right dominant bilateral ptosis, mild restriction of lateral gaze of the right eye, isochoric pupils, and weakness of eye tightening. Her speech had a nasal quality. The masseter muscles, tongue, and the flexors of neck were weak. Motor examination of other muscles was normal. Generalized botulism, a rare adverse effect of BoNT type A injection, was considered in the patient. Supportive treatment and pyridostigmine 60 mg 5 times per day were given. Worsening of the symptoms, fluctuating weakness, and inadequate response to pyridostigmine suggested the diagnosis of MG. Repetitive nerve stimulation performed 6 weeks after BoNT type A injection revealed dysfunction of neuromuscular junction and autoantibodies against acetylcholine receptors were found positive in serum. Thorax computerized tomography performed for diagnosis of thymoma was normal. Treatment of 0.5 mg/kg/d prednisolone following 0.4 g/kg/d intravenous immunoglobulin was given because dysphagia and shortness of breath worsened. In time, necessity for pyridostigmine decreased and a significant improvement in neurologic status was achieved. Neurologic examination after 6 months was normal except bilateral mild ptosis. Unfortunately, the patient died of acute myocardial infarction 3 weeks after her last follow-up examination.

After BoTN type A injection, symptoms including unilateral ptosis, diplopia, and dysphagia can occur and mimic MG (3). Patients with MG are rarely reported to show movement disorders including blepharospasm and cervical dystonia in the literature (4). The occurrence of symptoms of MG after BoTN type A injection

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Received/Geliş Tarihi: 27.04.2016 **Accepted/Kabul Tarihi:** 25.05.2016

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in our patient suggests that BoTN type A injection may unmask subclinical MG, as reported previously (5).

Ethics

Peer-review: Internal peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: Hacer Durmuş Tekçe, Yeşim Parman, Piraye Oflazer-Serdaroğlu, Feza Deymeer, Concept: Hacer Durmuş Tekçe, Yeşim Parman, Piraye Oflazer-Serdaroğlu, Feza Deymeer, Design: Hacer Durmuş Tekçe, Yeşim Parman, Piraye Oflazer-Serdaroğlu, Feza Deymeer, Data Collection or Processing: Hacer Durmuş Tekçe, Analysis or Interpretation: Hacer Durmuş Tekçe, Yeşim Parman, Piraye Oflazer-Serdaroğlu, Feza Deymeer, Literature Search: Hacer Durmuş Tekçe, Writing: Hacer Durmuş Tekçe, Yeşim Parman, Piraye Oflazer-Serdaroğlu, Feza Deymeer.

*Conflict of Interest: No conflict of interest was declared by the authors.
Financial Disclosure: The authors declared that this study has received no financial support.*

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