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Case Report

Underestimated and Unappreciated PSI

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Abstract

Over the last decade or so, the demand for so-called quick-fix treatments such as Botulinum toxin injections has continued to increase exponentially, on a par with the demand of cosmetic procedures. The benefit of having a non-surgical, non-invasive procedure which patients can have during their lunch-breaks is an appealing one. In the UK, nurse practitioners, GPs, dermatologists and other medical practitioners as well as plastic surgeons are using Botulinum toxin in their daily practice for a myriad of disorders ranging from cervical dystonia, facial nerve palsy and multiple sclerosis as well as in private practice. Increasing numbers of individuals both medically and non-medically qualified are attending facial rejuvenation courses and are as a result, administering it regularly. Once candidates have been assessed on the course, the individuals are deemed competent to administer Botulinum toxin and practice freely as non-surgical aesthetic practitioners, but are these individuals really aware of the potential fatal consequences of inappropriate use of this naturally occurring neuro-toxic substance?

Introduction

The demand for Botulinum toxin is well known, but Botulinum toxin complications can arise if improperly administered [1]. Factors cited for the increasing demand in cosmetic procedures include, increased competition for corporate jobs, media pressure as well as vanity, in addition to quicker, cheaper, less invasive procedures when compared with cosmetic surgery [2]. The reasoning behind this increasing demand for looking good is that men and women are living more health-conscious lifestyles, and procedures such as Botulinum toxin injections are a way for individuals to maintain the same physical energy and youthful feeling on the outside that they feel on the inside [2]. In the US alone last year 16 people suffered were reported to have died to the sequelae of Botulinum toxin injections. More regulations are necessary in regard to its competent and safe use particularly in the UK where anyone can administer this potentially fatal toxin.

Case Report

Botulinum toxin® was being used 'off-label' by the end of the 1990s for wrinkle reduction but was Food and Drug Administration (FDA) approved for aesthetic uses in 2002 [3,4]. Since then, Botulinum toxin® has become one of the most popular cosmetic procedures for both women and men. In 2003, 2.56 million Botulinum toxin® procedures were performed on women and nearly 334,000 on men. After Botulinum toxin® use increased remarkably following FDA approval, the FDA was concerned with the toxin being abused and the risk of Botulinum toxin® complications suffered [3]. Similar trends were witnessed in the UK and cited by the British National Formulary (BNF) [5].

Although millions of people have been safety treated with Botulinum toxin® for a variety of medical conditions, patient safety is a prime concern because of the potential for Botulinum toxin® complications to arise. Recently, an increasing number of patients have suffered fatal consequences of Botulinum toxin® injections [5,6]. The growth of the entire cosmetic surgery field has been in response to demand, but some under-qualified healthcare professionals have been practicing Botulinum toxin® treatment without understanding and appreciating the pitfalls of this neurotoxin.

The Natural history of botulinum Toxin

Botulinum neurotoxin is produced by the gram-negative anaerobic bacterium *Clostridium botulinum*. There are eight known serologically different neurotoxins namely, A, B, C1, C2, D, E, F, and G, of which seven exhibit properties paralysis. Types A, B, E and, rarely, F and G are associated with human botulism [7].

The first case of botulism, a bilaterally symmetric, descending neuroparalysis caused by the botulinum neurotoxin was first described by the German physician Justinus Kerner in 1817-1822 [7]. He observed the food-borne botulism variety which was seemingly endemic in Napoleonic Germany during 1790-1820 and largely attributed to the poor hygiene in food production and handling [7,8]. Amongst the people of the time, the 'sausage poisoning endemic' as it was known was given this title as the illness became evident after several witnessed cases of spoiled sausage ingestion [9]. Kerner subsequently deduced that the toxin acts by interrupting synaptic transmission within the peripheral and sympathetic nervous system, leaving sensory transmission intact, and also postulated hypotheses with respect to the possible therapeutic uses of the toxin [8]. Botulinum toxin blocks acetylcholine release, causing a chemical denervation and typically the clinical effect following an injection is on average 2-6 months with usual restoration of pre-botulinum nerve function [10].

Pathophysiology of Botulinum Toxin

When foods tainted with neurotoxin are ingested, the neurotoxin is metabolised and absorbed haematogenously to peripheral cholinergic nerve endings, where it blocks the release of the neurotransmitter acetylcholine [7-9]. Being heat labile, the neurotoxin is denatured by cooking [9]. Historically, sporadic outbreaks of botulism in the Western World have occurred but are now largely rare in view of the

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irradiation that takes place in preparation of canned foods such as tinned meat and fish products [9].

With respect to infant botulism, *C. botulinum* colonises the gut which leads to production and absorption of the toxin [9]. Consumption of tinned and canned products such as honey has been implicated in these cases as evidenced by microbiological studies typically showing type B spores [9].

Botulism exhibits clinical signs including progressive muscle weakness typically affecting the extra-ocular or pharyngeal muscles becoming generalised, gastrointestinal disturbances, dilated unreactive pupils, dry and erythematous mucous membranes are often dry and erythematous [5,6]. Sensory signs are uncommon with conscious level generally maintained as long as respiration is adequate [5,6].

Botulinum toxin type A was initially isolated by Schantz amongst others in the late 1940s, which led to its use in treating ocular muscle over and under action in monkeys by Scott in the late 1970s [4,7-9]. Continuing this work, a group from Columbia University serendipitously witnessed the cosmetic improvement after it use for facial dystonias [1,4]. Since then, the toxin was experimented to treat various neuromuscular disorders and is now licensed for the conditions including, strabismus, spasticity such as in multiple sclerosis, hemifacial spasms, focal dystonias including blepharospasm, torticollis, spasmodic dysphonia, limb dystonia, benign essential tremor, tics, hyperhidrosis, and oesophageal dysmotility disorders including achalasia and sphincter dysfunction [1,5,6,10,11,12]. Current work is being carried out to evaluate the treatment of headaches and complex regional pain syndromes [1,11]. Although there are wide-ranging medical uses for botulinum toxin, the vast majority of its use is 'off-label' and used for facial aesthetics in the cosmetic setting [1,12].

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