Cosmetic botulinum toxin A (Botox) injections and neuromuscular monitoring – a case report

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Background

Neuromuscular blocking agents are commonly used during general anaesthesia to facilitate endotracheal intubation and optimise surgical conditions. Ensuring adequate reversal is essential in order to minimise residual post-operative weakness and its complications[1]. The AAGBI 2015 *Recommendations for standards of monitoring during anaesthesia and recovery* mandate the use of a peripheral nerve stimulator whenever neuromuscular blocking agents are used[2]. The degree of neuromuscular blockade is typically assessed by applying a supramaximal stimulus to a suitable peripheral nerve and observing or measuring the associated muscular response. The AAGBI guideline advises that ulnar nerve stimulation is preferable, but that facial nerve stimulation may be used if this is not possible[2].

Case review

We present the case of a 39 year-old woman who underwent emergency laparoscopic oophorectomy to treat an ovarian torsion. She had a background of well-controlled asthma, was otherwise fit and well and had previously undergone an uneventful general anaesthetic. She received 70mg of rocuronium (1.2mg/kg) as part of a rapid sequence induction. Train-of-four and tetanic stimulation of the facial nerve intra-operatively failed to elicit a muscular response. In contrast, ulnar nerve stimulation at this time demonstrated four twitches with no fade (via qualitative monitoring). Extubation was uneventful. Subsequent discussion with the patient revealed that she had undergone cosmetic botulinum toxin (Botox) treatment three weeks previously.

Patient consent for this report was gained and documented.

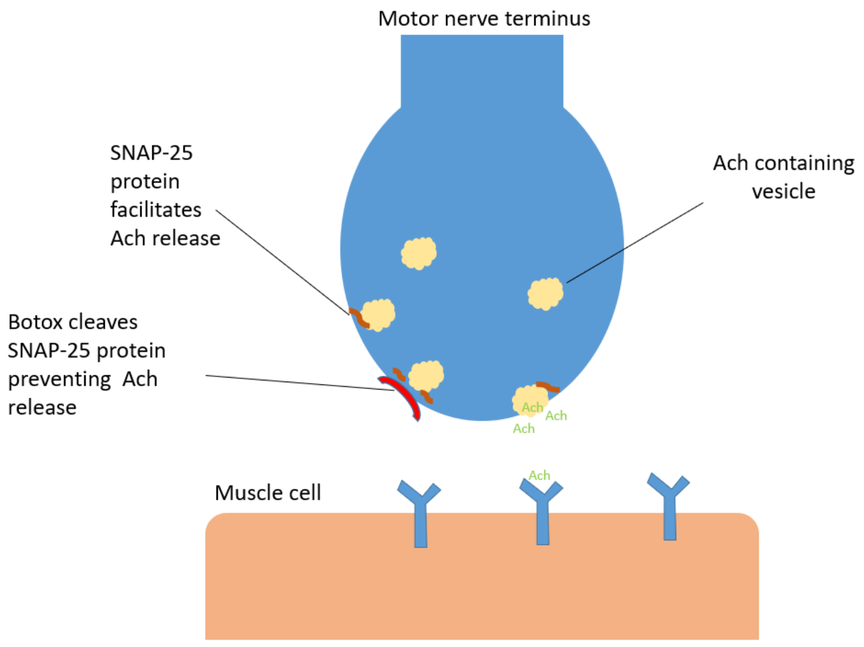

Figure 1: mechanism of action of Botulinum toxin A at the neuromuscular junction

Cosmetic use of Botulinum toxin A

Botulinum toxin is produced by the anaerobic, Gram-positive bacterium *Clostridium botulinum*. Several serotypes of botulinum toxin have been identified. Type A (BoNTA) is the only serotype to be approved for the cosmetic treatment of facial lines.

In normal physiology, voluntary muscle contraction occurs in response to the arrival of an action potential at the motor end plate of a muscle fibre, via an efferent nerve and the neuromuscular junction. The depolarisation of a motor nerve causes voltage-gated calcium channels at the presynaptic membrane to open, allowing influx of calcium to the nerve terminal. This triggers release of acetylcholine into the synaptic cleft. Acetylcholine binds with nicotinic acetylcholine receptors on the motor end plate causing local depolarisation of the muscle fibre, resulting in contraction.

Botulinum toxin A inhibits the SNAP-25 protein (Synaptosomal-Associated Protein-25) at the motor nerve terminal, preventing the discharge of acetylcholine into the synapse (Figure 1). Binding of the toxin is irreversible, so recovery of muscle function occurs by proliferation of axonal nerve buds to the target muscle. Maximal effect of botulinum toxin A occurs at 2 weeks, and some effect may continue for up to 6 months. Common injection targets include frontalis, corrugator supercilii and orbicularis oculi[3].

Discussion

Little definitive information is available about the size of the cosmetic procedures market, of which cosmetic Botox forms a major part. However, one estimate for the UK market in 2015 (including surgical and non-surgical procedures) was £3.6 billion – a 500% increase over a 15-year period[4].

Cosmetic Botox is increasingly common in adult patients. Recent treatment to the upper facial muscles clearly makes the facial nerve an unsuitable site for assessing neuromuscular blockade, however cosmetic Botox may not be disclosed or recognised at pre-operative assessment. Literature review reveals a small number of previously-reported cases, affecting both men and women[5,6].

This case suggests that anaesthetists should consider explicitly asking about facial Botox at pre-operative assessment. It also highlights an important limitation of monitoring the facial nerve, lending further weight to the assertion that the ulnar nerve should be the first choice of monitoring site wherever possible.

References

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