Role of botulinum toxin in the management of hand ulceration due to post-stroke spasticity among aged care residents

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Background
Individuals with significant post-stroke impairments often move to residential care. The prevalence of spasticity is high among these residents. Palmar ulceration is an under-recognised complication of unmitigated post-stroke hand spasticity. The ulcerations are painful, emit offensive smell and cause significant suffering for the individuals and the carers.

Objective
The aim of this article is to discuss the healing of these ulcerations by reducing spasticity in the hand muscles with botulinum toxin A (BoNTA) injection followed by dressing, splinting and hand therapy.

Discussion
This article discusses the epidemiology, anatomy and clinical presentations of spastic hand ulcerations and mechanisms of action of BoNTA in alleviating the ulcers and related symptoms such as pain, sweating, offensive smell, and lessening the carer burden. The primary neuromuscular blocking action of BoNTA results in the opening of the hand, allowing dressing and ease of care. The toxin provides pain relief, inhibits excessive sweat production and causes vasodilatation, ultimately resulting in healing of the ulcerations.

Epidemiology of upper limb PSS in the RACS
There is a paucity of data regarding the prevalence and management of spasticity among individuals residing in the RACS.4,5 Stroke is one of the common causes of spasticity and is reported to be present in 22% of residents examined by a neurologist.6 In a study conducted in the USA and UK, approximately 15–18% of nursing home residents...
>65 years have had a stroke. The study also reported that PSS is underdiagnosed and inadequately treated in individuals living in the RACS. In an Australian study, researchers found 19.3% of all individuals living in the Australian RACS had previously had a stroke. The same group reported that approximately 70% of individuals living in the RACS with a history of stroke had upper limb spasticity. This study also found upper limb PSS was associated with pain and significant passive care difficulties. The prevalence data are similar to results from a cross-sectional study performed by Meijer et al, reporting spasticity among 73% of a mixed cohort of patients with central nervous system disorders in a nursing home. Individuals with past ischaemic stroke constituted the most common group. Meijer et al studied a population group of 850 patients in three nursing homes.

**PSS and the hand**

The human hand has a complex anatomy, and it is instrumental in multiple grips and postures resulting from highly mobile fingers and thumb. The hand is a multipurpose body part involved in reaching, grasping, manipulating objects and feeling. It is also an important conduit to our rich tactile and communicating world. The evolution of human hand dexterity became possible to a significant extent because of the highly mobile thumb.

PSS commonly affects the hand; the thumb loses its mobility and gets drawn into the palm in what is known as the thumb-in-hand deformity, and the fingers flex unbidden as a result of the hyperexcitability of stretch reflexes, giving rise to a permanently clenched fist. The hand is no longer capable of conducting active tasks such as feeding, dressing, grooming and other activities of daily living. Moreover, individuals with unmitigated upper limb spasticity develop pain, deformity, contracture and, in some cases, serious complications such as palmar ulcerations in the hand. Family members and carers find it exceedingly difficult to clean the individual’s hands and maintain hand hygiene.

Besides stroke, individuals with upper motor neurological disorders such as brain injury, multiple sclerosis and cerebral palsy are also vulnerable to develop similar conditions.

**Functional anatomy and clinical presentation**

PSS in the upper limb manifests as clenched fist, flexed wrist and pronated forearm. The elbow is flexed, and the shoulder joint remains adducted and internally rotated.

The clenched fist is due to spasticity in the spastic flexor digitorum superficialis and flexor digitorum profundus muscles. The thumb-in-hand deformity is caused by spastic flexor pollicis longus, flexor pollicis brevis, adductor pollicis and the first dorsal interosseous muscles. Intrinsic plus hand deformity is caused by spastic lumbricals and interossei muscles. Wrist flexion is due to spastic flexor carpi ulnaris and flexor carpi radialis muscles, with secondary contribution from the long finger flexors. Forearm pronation is caused by spasticity in the pronator teres and pronator quadratus muscle. These specific muscles causing various deformities, and the objective assessment of the spastic hand pre- and post-BoNTA injection have been described elsewhere.

**Pathophysiology of palmar ulceration due to hand PSS**

Spasticity is the most common cause of difficult-to-treat pressure ulcers in atypical locations, such as the hand. In the spastic hand, fingers can neither be actively nor passively extended to facilitate cleaning of the hand. In some cases, the spastic and distorted fingers, often with overgrown nails, press unremittingly into the palm. It is recognised that ‘external pressure of more than 33 mmHg occludes capillary perfusion so that the

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**Figure 1. Examples of spastic hand with complications successfully treated**

A. Pressure ulcerations in the proximal and distal palmar creases and metacarpophalangeal crease of the ring finger caused by spasticity in the flexor digitorum superficialis and flexor digitorum profundus muscles; B. Hand ulcerations involving the distal palmar crease and metacarpophalangeal crease of the ring and middle finger caused by spasticity in the flexor digitorum superficialis muscle; C. A spastic hand with longstanding finger flexion deformity and excessive sweating causing build-up of debris and ulceration underneath; D. Significant improvement following treatment with botulinum toxin A injection into the spastic hand muscles followed by dressing, splinting and therapy.
underlying and surrounding tissues become anoxic and if the pressure continues for a critical duration, cell death will occur, resulting in soft tissue necrosis and eventual ulceration. Over time, pressure from the spastic fingers with overgrown nails causes skin breakdown, resulting in atypical pressure ulceration in the palm of the hand, commonly around the proximal palmar, distal palmar and metacarpophalangeal creases. It becomes difficult to dress these ulcers and maintain general cleanliness and hygiene because the hand cannot be opened. These painful and apparently untreatable ulcers of the hand and fingers cause intense suffering for the patient and stress for families and carers. Spasticity thus creates a powerful...
Barrier as the ulcers become inaccessible to treatment. Ulcerations are more common on the ulnar side of the hand (Figure 1A and 1B). It is reported that the ulnar two digits play a significant part in hand grip. The overall grip strength decreases to 34–67% (with a mean decrease of 55%) without the contribution of the ulnar digits. It is hypothesised that the stronger ulnar flexor muscles become more spastic than the muscles on the radial side, giving rise to ulcerations occurring commonly on the ulnar side of the hand. In the wrist, flexor carpi ulnaris is the commonly deforming force and, coupled with weak extensors and radial deviators, causes wrist flexion and ulnar deviation.

There is excessive sweating in the spastic hand (hyperhidrosis), which remains closed most of the time. Hyperhidrosis is the result of sympathetic hyperactivity of eccrine sweat glands in the palms. It is common for the untreated spastic hand to build up debris and become foul smelling from excessive sweating because of biotransformation of odourless natural secretions into odorous molecules (bromhidrosis).

**Mechanisms of BoNTA action in the healing of hand ulcerations and associated symptoms**

BoNTA is useful in many disorders of the hand, such as spasticity, dystonia, hyperhidrosis, tremor and Raynaud’s phenomenon. Interestingly, several mechanisms of BoNTA are likely to be operational in the healing of hand ulcerations.

Injection of the toxin relaxes the spastic muscles by blocking the presynaptic release of neuromuscular transmitter acetylcholine and deactivating soluble N-ethylmaleimide-sensitive factor attachment protein receptor (SNARE) protein. This facilitates opening of the hand, which allows dressing and maintenance of hygiene.

BoNTA also inhibits excessive sweating by blocking the release of acetylcholine in the postganglionic sympathetic fibres to the eccrine glands, which are responsive to cholinergic stimulation, unlike the apocrine glands. Studies have shown that BoNTA is used to inhibit palmar hyperhidrosis. Reduction of palmar sweating is helpful in lessening the build-up of debris and alleviating the offensive smell. This makes it easier to dress these wounds and promotes healing of the wounds (Figure 2).

BoNTA has been shown to be effective in vasospastic conditions such as Raynaud’s phenomenon by relieving pain and improving perfusion. The mechanism of its function is not fully elucidated. The putative mechanisms are inhibition of the activity of sympathetic and sensory hyperactivity of eccrine sweat glands in the palms. It is common for the untreated spastic hand to build up debris and become foul smelling from excessive sweating because of biotransformation of odourless natural secretions into odorous molecules (bromhidrosis).

**Figure 4.** Mechanism of botulinum toxin A injection in pain reduction involves 1) retrograde axonal transport of toxin, 2) inhibition of neuropeptides such as substance P, calcitonin gene-related protein and glutamate and 3) deactivation of sodium channels. These mechanisms of action reduce peripheral and central sensitisation.
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The role of botulinum toxin (BoNTA) in the management of hand ulceration due to post-stroke spasticity among aged care residents is discussed. BoNTA injection into the spastic hand muscles – along with other adjuvant measures such as dressing, splinting and therapy – can alleviate the distressing complications of hand ulcerations. BoNTA is also useful in the treatment of other symptoms related to the ulcerations, such as pain, sweating and offensive smell. The injection of BoNTA into spastic muscles followed by dressing, splinting and therapy improves patient outcomes and lessens the carer burden. Patients in the RACS who are likely to develop spasticity-related complications should be referred early to a multidisciplinary spasticity clinic in tertiary hospitals.

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References

Conclusion
PSS is common among residents living in the RACS. As there is no multidisciplinary management of spasticity in the RACS, the residents are prone to develop...
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