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Unilateral spastic trismus in brain stem stroke: A hindrance to oral care, mastication, and speech

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Abstract

Spastic trismus (ST) is a rare manifestation of stroke-induced spasticity, characterised by sustained contraction of the masticatory muscles, resulting in restricted jaw opening. It can severely impact oral hygiene, feeding, speech, and access to dental procedures, and may complicate emergency airway management. ST is more commonly observed in bilateral cerebral cortical lesions, while isolated unilateral ST following a brainstem stroke is exceedingly rare. We report a case of unilateral ST in a 57-year-old man with a history of diabetes mellitus and dyslipidaemia who suffered a brainstem stroke affecting the pontomedullary junction and medulla. He developed severe limb incoordination, dysphagia, and dependence on nasogastric tube feeding but remained cognitively intact. He was referred to us two years post-stroke, presenting with severe trismus (interincisal distance of 11 mm) and right jaw hypertrophy. Conservative treatments, including warm compresses and stretching exercises, failed. Botulinum toxin injection was offered, but declined. He was started on oral baclofen (10 mg twice daily), which was later reduced due to drowsiness. After two weeks, his interincisal distance improved to 22 mm, facilitating oral hygiene and speech articulation. Baclofen was discontinued after four weeks as no further improvement was observed, but gains were maintained at three, six, and 12 months post-discharge. This case highlights the challenges in managing post-stroke ST, particularly in a unilateral brainstem lesion. While botulinum toxin remains the treatment of choice, oral antispasmodics may serve as viable alternatives when injections are contraindicated or declined. Further research is needed to establish evidence-based rehabilitation strategies to improve long-term outcomes for ST.

Keywords: Trismus, brainstem stroke, baclofen, botulinum toxin, rehabilitation

INTRODUCTION

Stroke can significantly impact the masticatory muscles, leading to reduced bite force on the hemiplegic side and progressive atrophy due to prolonged consumption of textured-modified diets.[1] Facial palsy may cause food trapping in the buccal sulcus, while tongue paralysis compromises its natural debridement function, worsening oral hygiene.[2] Post-stroke oral health care is often challenging due to hemiparesis, impaired hand coordination, and cognitive deficits.[2] Poor oral hygiene promotes bacterial overgrowth in saliva, which, when aspirated, increases the risk of aspiration pneumonia and systemic infections.[3,4]

These challenges are exacerbated in patients with spastic trismus (ST), a rare condition characterised by sustained spasm of masticatory muscles, primarily the masseter and temporalis, resulting in forced jaw closure.[4] ST interferes with oral feeding, oral hygiene, speech, and dysphagia therapy while also affecting facial appearance and restricting access to dental procedures.[5,6] More critically, restricted jaw opening can be life-threatening in emergencies, particularly when tracheal intubation is required. This case report highlights an unusual case of unilateral ST secondary to a chronic brainstem stroke, successfully managed with a short course of oral antispasmodic therapy.

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CASE REPORT

A 57-year-old man with a five-year history of diabetes mellitus and dyslipidaemia suffered a posterior circulation infarct. Initial stroke management at a government hospital included magnetic resonance imaging (MRI), which confirmed an acute infarction at the pontomedullary junction and medulla. Prolonged intubation during hospitalisation necessitated a tracheostomy tube insertion. The brainstem stroke resulted in severe incoordination of all four limbs, leading to significant imbalance, inability to ambulate, and severe dysphagia. His cognitive function remained relatively intact.

Following the stroke, he underwent rehabilitation at various private institutions, both in inpatient and outpatient settings, including 10 sessions of repetitive transcranial magnetic stimulation. Despite these interventions, his functional recovery remained poor. He remained wheelchair-bound, requiring maximal assistance for bed mobility and transfers. He mobilised primarily with a motorised wheelchair, with family members using a portable hoist for transfers. Two years post-stroke, he was referred to our facility for continuation of rehabilitation, including dysphagia management. On admission, he remained reliant on nasogastric tube feeding, having previously declined percutaneous endoscopic gastrostomy tube insertion.

Examination revealed right jaw hypertrophy, with a tense and contracted right masseter muscle on palpation. There was no jaw deviation or tightness of the ipsilateral temporalis muscle. His mouth opening was severely restricted, with an interincisal distance of 11 mm, equivalent to the thickness of five tongue depressors. The patient experienced pain during jaw opening. There was no evidence of concomitant spasticity in the upper and lower limbs. The patient and his caregivers reported that the right jaw swelling had been present for several months, though the exact onset was unclear. There were no clinical features suggestive of odontogenic or non-odontogenic infections, malignancy, or temporomandibular joint disorders. Laboratory investigations were unremarkable, and a temporomandibular joint X-ray revealed no abnormalities.

Initial conservative measures, including warm compresses, massage, and stretching exercises, failed to alleviate the symptoms. The patient was offered a botulinum toxin injection but declined for personal reasons. He was subsequently started on oral baclofen at 10 mg twice daily. However, due to drowsiness, the dosage was reduced to 10 mg once daily. After two weeks, his interincisal distance improved to 22 mm, equivalent to the thickness of 14 tongue depressors (Figure 1).

Although a flexible endoscopic evaluation of swallowing confirmed persistent severe pharyngeal phase dysphagia, the increased mouth opening significantly facilitated oral hygiene, speech articulation, and oromotor exercises (Table 1).

The oral baclofen was discontinued after four weeks as no additional improvement in the interincisal distance was observed, and the jaw opening was deemed adequate for oral care and oromotor exercises. The improvement was

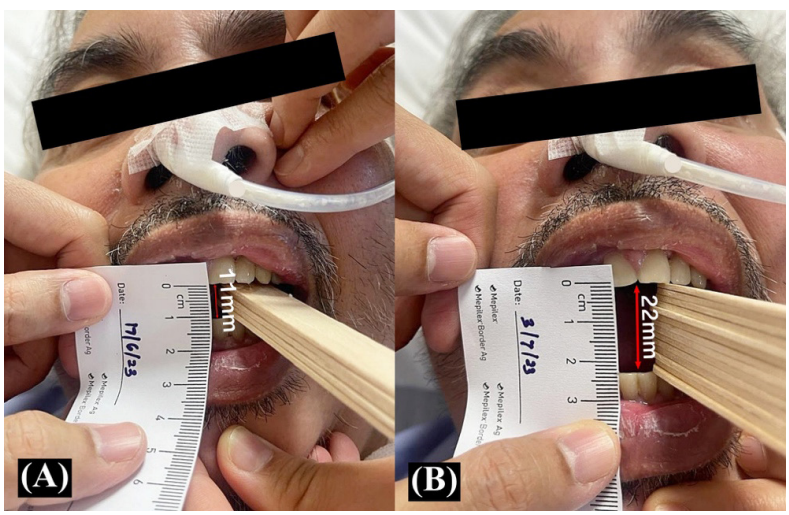

Figure 1. The interincisal distance improvement (double arrow) before (A) and after 2 weeks (B) of baclofen treatment, showing significant improvement.

Table 1: Swallowing and speech-related outcomes at admission and discharge from inpatient rehabilitation

Measure	Admission	Discharge
Interincisal distance	11 mm	22 mm
Penetration-aspiration scale (FEES)	4 (material contacts the vocal folds and is ejected from the airway)	4 (material contacts the vocal folds and is ejected from the airway)
Functional Oral Intake Scale	Level 1 (nothing by mouth)	Level 2 (tube dependent with minimal attempts of food or liquid)
Effortful & dry swallow with cold spoon	Cough in > 2/10 trials	10 repetitions without coughing

Abbreviation: FEES, flexible endoscopic evaluation of swallowing.

maintained at follow-ups at three, six, and 12 months post-discharge (Figure 2), while the patient continued outpatient speech and swallowing therapy.

DISCUSSION

Trismus can arise from a variety of intra- and extra-articular pathologies.[5] However, ST is relatively uncommon. Due to the bilateral cortical innervations of the motor trigeminal nucleus, ST is more frequently observed in bilateral cerebral cortical lesions than in unilateral lesions.[6] Isolated unilateral ST, particularly following a brainstem stroke, is exceedingly rare. In brain stem lesions, trismus occurs due to abnormalities in the programming of the trigeminal motor nucleus.[4] The underlying pathophysiology involves hyperexcitability of motor neurons innervating the masseter muscle, likely resulting from disruption of the descending inhibitory pathways of the dorsal reticulospinal tract, originating from the ventromedial reticular formation in the medulla.[7]

Due to limited focal efficacy and systemic side effects, localised spasticity, such as ST, is difficult to manage with oral antispasmodics alone. Evidence supporting the use of oral antispasmodics in ST is sparse. Ungar *et al*. reported a patient with severe traumatic brain injury who developed generalised spasticity and ST with frequent teeth chattering.[8] The patient showed only modest improvement with the maximum tolerable dose of oral baclofen. However, significant resolution of ST occurred following intrathecal baclofen therapy. Forty-five days after the initiation of treatment, the patient exhibited no teeth chatter, had near-normal tone in his masticatory muscles, and regained the ability to speak. In contrast, our patient experienced substantial improvement with oral baclofen alone, with an increase in interincisal distance from 11 to 22 mm. Interincisal distance

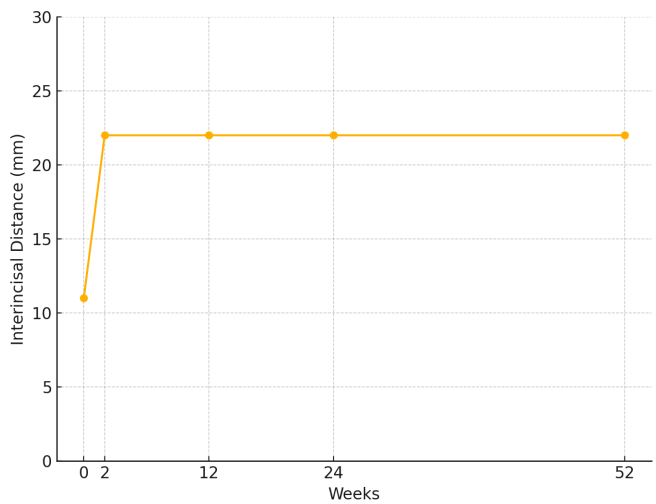

Figure 2: Interincisal distance from inpatient through one-year follow-up.

is the distance between the lower border of the upper incisors and the upper border of the lower incisors.[4] The normal range of mouth opening varies between 40 and 60 mm.[5] Although oral baclofen improved jaw opening, the low dosage likely constrained further gains, which could have potentially improved with botulinum toxin injections.

The efficacy of botulinum toxin in treating ST is well-supported in the literature. Kobal *et al*. reported a case involving a 3.5-year-old girl hospitalised for viral encephalitis who experienced improvement in interincisal distance from 0 to 10 mm over two months following botulinum toxin injections to the bilateral masseter and temporalis muscles.[4] Similarly, Seo *et al*. documented a case of ST secondary to brainstem stroke where interincisal distance improved from 2 to 14 mm after injection into the left masseter and temporalis.[6] In a randomised controlled trial, Fietzek *et al*. demonstrated a statistically significant improvement in the interincisal distance among patients who received botulinum toxin in the masseter muscle compared to the placebo group.[9] Despite its proven efficacy, botulinum toxin use may be limited by cost, access to trained personnel, or patient preference, as was the case in our patient.

While ST has improved, the patient's dysphagia persists, underscoring the multifactorial nature of post-stroke dysphagia. Long-term management of ST requires an interdisciplinary approach, incorporating pharmacological, physical, and, when feasible, procedural interventions.[5] Physical modalities and interventions such as warm compresses, stretching, and massage may provide temporary relief but are often insufficient as standalone treatments.[9] Therapeutic exercise involving jaw opening, closing, and lateral excursions may facilitate recovery.[5] Besides baclofen, muscle relaxants such as benzodiazepines and analgesics like aspirin may be used in the acute phase.[5] In more resistant and complicated cases, physical therapy may be combined with jaw opening devices such as stacked tongue depressors, corkscrews, TheraBite, and Dynasplint.[10]

Due to its rarity, the natural history of ST remains poorly understood, rendering prognostication difficult. While botulinum toxin has demonstrated efficacy in various cases, long-term outcomes and optimal rehabilitation strategies remain under-investigated.

In conclusion, ST is a complex and functionally debilitating neurological disorder. Management requires a patient-centred approach, balancing treatment efficacy, patient preferences, and resource availability. While botulinum toxin remains the preferred treatment, oral antispasmodics such as baclofen are viable alternatives in selected cases, particularly when injections are contraindicated or declined. Given the limited evidence, further research is needed to establish effective rehabilitation interventions for this enigmatic neurological phenomenon.

DISCLOSURE

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