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Case Report: Botulinum Toxin Type A Injection for Bruxism Following Left Basal Ganglia Hemorrhage

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 40-year-old
Final Diagnosis: Bruxism
Symptoms: Bruxism
Clinical Procedure: —
Specialty: Neurology
Objective: Unusual or unexpected effect of treatment

Background: Post-stroke bruxism is an underrecognized complication that may impair oral function, cause orofacial pain, and negatively impact rehabilitation outcomes. Botulinum toxin type A (BoNT-A) has been reported for bruxism, but its application in post-stroke populations remains sparsely documented. This case describes the assessment and treatment of bruxism following cerebral hemorrhage, utilizing the Standardized Tool for the Assessment of Bruxism (STAB) as a structured multidimensional framework.

Case Report: A 40-year-old man developed bruxism during recovery from a massive left hemispheric intracerebral hemorrhage after decompressive craniectomy and hematoma evacuation. About 3 months after admission, he began to exhibit bilateral masseter muscle hypertrophy with marked tenderness, temporomandibular joint pain, and drooling from the right mouth corner, with no prior history of bruxism. STAB-based evaluation yielded the following Axis A findings: Subject-based assessment revealed nocturnal grinding reported by caregivers. Clinically based assessment revealed bilateral masseter hypertrophy, tenderness on palpation, and dental wear. Instrument-based assessment (surface electromyography used for injection guidance) confirmed increased masseter muscle activity at rest and during clenching. Symptoms were refractory to occlusal splint therapy combined with hot compresses. At 48 hours after electromyography-guided injection into the bilateral masseter muscles (50 U per side, divided into 3 sites per side), muscle tension and tenderness were markedly reduced, and clinical improvement persisted at 6-month follow-up.

Conclusions: In this single case, BoNT-A injection was temporally associated with symptom relief in post-stroke bruxism. STAB provided a structured multidimensional assessment framework. These descriptive findings have limited generalizability. Controlled studies are needed to establish the efficacy and safety of this intervention in the post-stroke population.

Keywords: Botulinum Toxins, Type A • Bruxism • Cerebral Hemorrhage
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Introduction

Bruxism is a manifestation of excessive activity of masticatory muscles, such as the masseter and temporalis muscles, characterized by teeth grinding or jaw compression, occurring during wakefulness or sleep, which affects approximately 10% to 16% of the adult population [1-4]. Long-term bruxism often leads to complications, including tooth wear, tooth loss, temporomandibular joint disorders, jaw pain, and headaches, seriously affecting patients' recovery and quality of life.

Bruxism following cerebral hemorrhage or other central nervous system injuries is increasingly recognized but remains poorly characterized. The pathophysiology is hypothesized to involve disruption of corticobulbar pathways, dysregulation of basal ganglia circuits, or aberrant activation of brainstem masticatory pattern generators [5,6]. Unlike primary bruxism, post-stroke bruxism often presents with distinct features including acute onset during neurological recovery, association with other post-stroke movement disorders, and potential exacerbation by spasticity or altered consciousness [7,8]. Epidemiological data are limited, but 1 study reported bruxism in 8.5% of stroke survivors during inpatient rehabilitation [2].

At present, common treatment methods for bruxism include non-drug interventions such as occlusal plate therapy, reducing the intake of alcohol and caffeine before bedtime, drug interventions such as baclofen, botulinum toxin therapy, and surgery. However, the evidence supporting these interventions is limited, with most studies based on small sample sizes and showing variable treatment effects [2]. Botulinum toxin type A (BoNT-A) has demonstrated efficacy in managing primary bruxism and certain neurological conditions such as oromandibular dystonia [9,10]. However, its use specifically for bruxism following cerebral hemorrhage is sparsely documented. To date, only isolated case reports and small case series have described BoNT-A for post-stroke bruxism, with heterogeneous patient populations, variable injection protocols, and limited objective outcome measures [11-14]. A structured assessment framework incorporating standardized diagnostic criteria remains lacking.

Here, we report a patient with bruxism after cerebral hemorrhage admitted to our department and treated with BoNT-A.

Case Report

A 40-year-old man was admitted to our rehabilitation department 2 months after an initial episode of sudden loss of consciousness. The clinical course and relevant interventions are detailed chronologically below.

Timeline of Events

On the day of admission (Day 1), the patient experienced a sudden loss of consciousness without any identifiable cause, at home, presenting with coma and unresponsiveness to calls. There were no associated limb convulsions, frothing at the mouth, or incontinence. He was emergently transferred to a local hospital where a head computed tomography (CT) revealed a massive intracerebral hemorrhage in the left cerebral hemisphere with slight rightward shift of midline structures, subarachnoid hemorrhage, and low-density lesions in the right frontal lobe and periventricular area (**Figure 1A**). An emergency decompressive craniectomy with intracerebral hematoma evacuation and tracheostomy was performed. He was subsequently transferred to our rehabilitation department for further recovery. The patient remained comatose until hospital day 66, when he regained consciousness. Two weeks later (Day 80), his tracheostomy tube was removed. Three weeks after that (Day 101), he began to exhibit symptoms of bruxism, characterized by bilateral masseter muscle hypertrophy with marked tenderness and temporomandibular joint tenderness, and drooling from the right corner of the mouth. Further history taking revealed that he had no prior history of bruxism. The bruxism symptoms persisted during assisted feeding and at night during sleep. Concurrent scale assessments showed the following: his Mini-Mental State Examination score was 27, his Generalized Anxiety Disorder-7 (GAD-7) score was 6, and his Patient Health Questionnaire-9 (PHQ-9) score was 4. A repeat head CT was obtained (**Figure 1B**). Despite initial conservative management with an occlusal splint and hot compresses, the bruxism showed no improvement. Fifteen days after the onset of bruxism (Day 116), an electromyography-guided BoNT-A injection was administered (50 U per masseter).

Physical Examination at Admission

Upon admission to our rehabilitation ward, the patient's vital signs were as follows: body temperature 37.6 °C, pulse rate 92 beats per minute, respiratory rate 33 breaths per minute, and blood pressure 134/82 mm Hg. He was unconscious but demonstrated occasional spontaneous eye opening, with Glasgow Coma Scale score of E4 Vt M2 (total score: 4T2), and was transported to the ward on a stretcher. No flexion deformities were noted in the clenched fists, wrists, or elbows; however, ankles were in plantar flexion without inversion. He was unable to speak, follow commands for auditory comprehension, or cooperate with orientation assessment. His pupils were bilaterally equal and round, approximately 3 mm in diameter, with a prompt light reflex. No nystagmus was observed. Cooperation for hearing assessment could not be obtained. Cranial nerve examination showed symmetric bilateral forehead wrinkles, a flattened right nasolabial fold, and deviation of the left mouth corner, while cooperation for tongue

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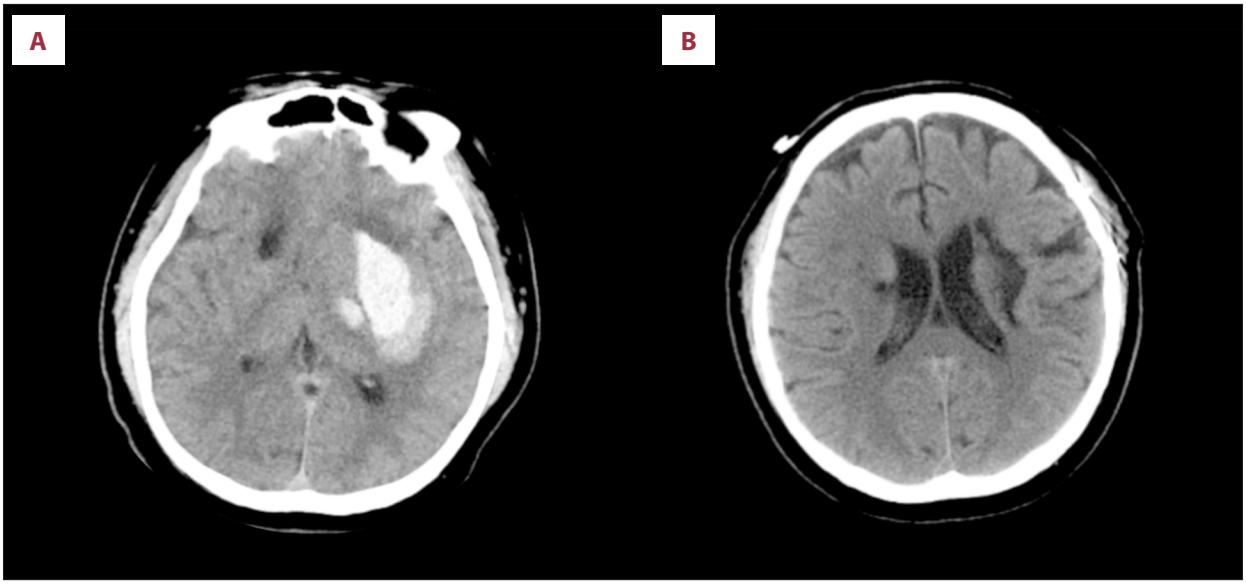

Figure 1. Head CT findings. (**A**) Pre-admission CT scan showing intracerebral hemorrhage in the left basal ganglia region and subarachnoid hemorrhage. (**B**) Follow-up CT scan after admission, demonstrating postoperative changes following left basal ganglia intracerebral hemorrhage evacuation, with evidence of encephalomalacia formation in the surgical area. CT, computed tomography.

protrusion and gag reflex testing could not be achieved. The palmomental reflex and jaw jerk reflex were both absent, and the neck was supple without resistance. Muscle strength testing could not be performed due to lack of cooperation, and superficial and deep sensation could likewise not be assessed. For the right limbs, the Brunnstrom stages were stage I for the upper limb, hand, and lower limb. Muscle tone, as assessed by the Modified Ashworth Scale, was grade 0 for both the biceps brachii and triceps surae, with no Achilles tendon contracture and an ankle clonus grade of 0. The left Babinski sign was negative. The patient exhibited grade 0 sitting and standing balance, and his Activities of Daily Living ability, as measured by the Barthel Index, was 0 points.

Bruxism Assessment Using the Standardized Tool for the Assessment of Bruxism Framework

Bruxism was systematically evaluated using the Standardized Tool for the Assessment of Bruxism (STAB), a dual-axis framework recommended for clinical and research applications [15]. With regard to Axis A (status assessment), the subject-based assessment confirmed no prior history of bruxism or temporomandibular symptoms. The onset occurred on hospital day 101, with caregiver-reported loud teeth grinding during sleep on a nightly basis, with multiple episodes per night, and awake bruxism was observed as mandible clenching during assisted feeding and daytime rest. For the clinically based assessment, examination revealed bilateral masseter hypertrophy with increased tension on palpation and tenderness at the anterior border and angle of the mandible, rated as 6/10 on the Visual Analog Scale (VAS) on the left and 5/10 on the right. The temporalis muscles exhibited mild tenderness on palpation, and the temporomandibular joints showed mild tenderness without clicking or crepitus. The patient's maximum pain-free, unassisted mouth opening was 39 mm. Oral examination showed no buccal mucosa bite marks, and dental wear, assessed by the Basic Erosive Wear Examination (BEWE) index, yielded a total score of 2. These findings were consistent with a diagnosis of "probable bruxism" per STAB Axis A clinical criteria.

In terms of instrument-based assessment, a Dantec Clavis handheld electromyography device was used solely to guide BoNT-A injection. Auditory feedback confirmed increased masseter muscle activity at rest and during voluntary clenching, localizing the most active regions for targeted injection. Although no quantitative electromyography values were recorded, the combination of clinical examination and electromyography localization fulfilled the STAB Axis A criteria for "definite bruxism" based on the available instrument-based assessment (electromyography for injection guidance); however, we note that polysomnography—the reference standard for sleep bruxism—was not performed.

Regarding Axis B (Etiology and Comorbidities Assessment), the psychosocial evaluation showed a GAD-7 score of 6 (indicating mild anxiety) and a PHQ-9 score of 4 (indicating no clinically significant depression). His caregiver reported that the patient expressed frustration regarding residual hemiparesis and functional limitations. With respect to concomitant

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sleep-related conditions, no snoring, witnessed apnea, or restless legs were reported by caregivers, although polysomnography was not performed; therefore, the diagnosis of bruxism is based solely on caregiver observation. The patient's current medications included amlodipine for hypertension and metformin for type 2 diabetes mellitus, and he reported no alcohol, caffeine, or tobacco use. Other relevant factors included a left basal ganglia hemorrhage affecting corticospinal and corticobulbar tracts, as well as significant medical comorbidities, namely grade 3 (very high risk) hypertension and type 2 diabetes mellitus.

Taken together, the above information indicates that the clinical course and findings were more consistent with secondary bruxism following cerebral hemorrhage than with primary sleep bruxism, medication-induced movement disorder, or oromandibular dystonia. Two weeks of occlusal splint therapy (worn during sleep for approximately 8 hours per night) combined with hot compresses (applied to bilateral masseter muscles twice daily, 20 minutes each session) failed to induce meaningful clinical improvement. The patient continued to exhibit the same frequency of grinding both during sleep and wakefulness (multiple episodes per day and night, per caregiver report), persistent masseter muscle tenderness (VAS unchanged: left 6/10, right 5/10), and no improvement in jaw pain or feeding difficulty. Therefore, treatment with BoNT-A injection was considered.

Botulinum Toxin Type A Injection Treatment and Outcomes

BoNT-A injection was administered after obtaining informed consent from the patient's family. The patient was placed in the supine position, and BoNT-A injection was performed under an electromyographic device with auditory feedback (Dantec Clavis handheld electromyography device). One vial of BoNT-A (100 U) was diluted with 2 mL of 0.9% sodium chloride injection, resulting in a concentration of 50 U/mL. The specific injection sites and dosages were as follows: a total of 50 U divided into 3 injection sites in the left masseter muscle, and a total of 50 U divided into 3 injection sites in the right masseter muscle. The masseter muscle belly is identified by palpation during voluntary clenching. Electromyography confirmed the region of maximal activity at rest and during contraction. The patient was monitored for 30 minutes for immediate adverse effects.

At 24 hours after the BoNT-A injection, the frequency of the patient's bruxism had decreased. At 48 hours after the injection, the bruxism symptoms had nearly completely resolved, accompanied by reduced muscle tone and tenderness in both masseter muscles. Tenderness VAS decreased from 6/10 to 2/10 (left) and 5/10 to 1/10 (right). Maximum mouth opening increased to 42 mm. This clinical improvement persisted at the 6-month follow-up.

Discussion

This case describes a patient who developed bruxism during recovery from left basal ganglia hemorrhage, with onset temporally linked to the subacute phase of stroke recovery. Following failure of conservative measures, electromyography-guided BoNT-A injection resulted in rapid symptom improvement that was sustained for 6 months. The case was systematically assessed using the STAB framework, providing structured documentation of bruxism status and associated comorbidities.

In this patient, bruxism manifested as both sleep-related and awake grinding, with clinical features consistent with previously described post-stroke bruxism [2]. The left basal ganglia hemorrhage provided a plausible neuroanatomical substrate for dysregulation of masticatory motor control, a mechanism distinct from the multifactorial pathophysiology typically implicated in primary bruxism [16].

Pathophysiological Plausibility

The act of grinding is executed by the masticatory muscles, which are regulated by a complex neural network involving the trigeminal motor nucleus, basal ganglia, hypothalamus, amygdala, and mesencephalic reticular formation. Studies have shown that frontal lobe lesions, such as those occurring in frontotemporal dementia or Rett syndrome, can lead to reduced cortical inhibition, affecting the trigeminal motor nucleus located within the brainstem and potentially resulting in bruxism [17,18]. Other research suggests that sleep bruxism may arise from dysfunction of inhibitory neurons within the central masticatory circuitry [19]. Current evidence suggests that bruxism may be associated with an imbalance in basal ganglia circuitry resulting from impaired neuronal plasticity (**Figure 2**). In the present case, the patient's primary intracranial lesions were localized to the basal ganglia region, and hemorrhage likely disrupted the cortico-basal ganglia-brainstem circuits and dopaminergic modulation relevant to masticatory motor control. Within the midbrain, neurons in the substantia nigra pars compacta (A9) project axons via the nigrostriatal pathway to the striatum (caudate nucleus and putamen), globus pallidus, and subthalamus. Separately, dopaminergic neurons in the ventral tegmental area and mesencephalic reticular formation (A10) give rise to mesolimbic projections to the nucleus accumbens, amygdala, and hippocampus, as well as mesocortical projections to the frontal cortex and associated areas. Anxiolytics, analgesics, and antipsychotic drugs act on dopaminergic neurons and receptors within these mesolimbic and mesocortical pathways. The left basal ganglia hemorrhage in

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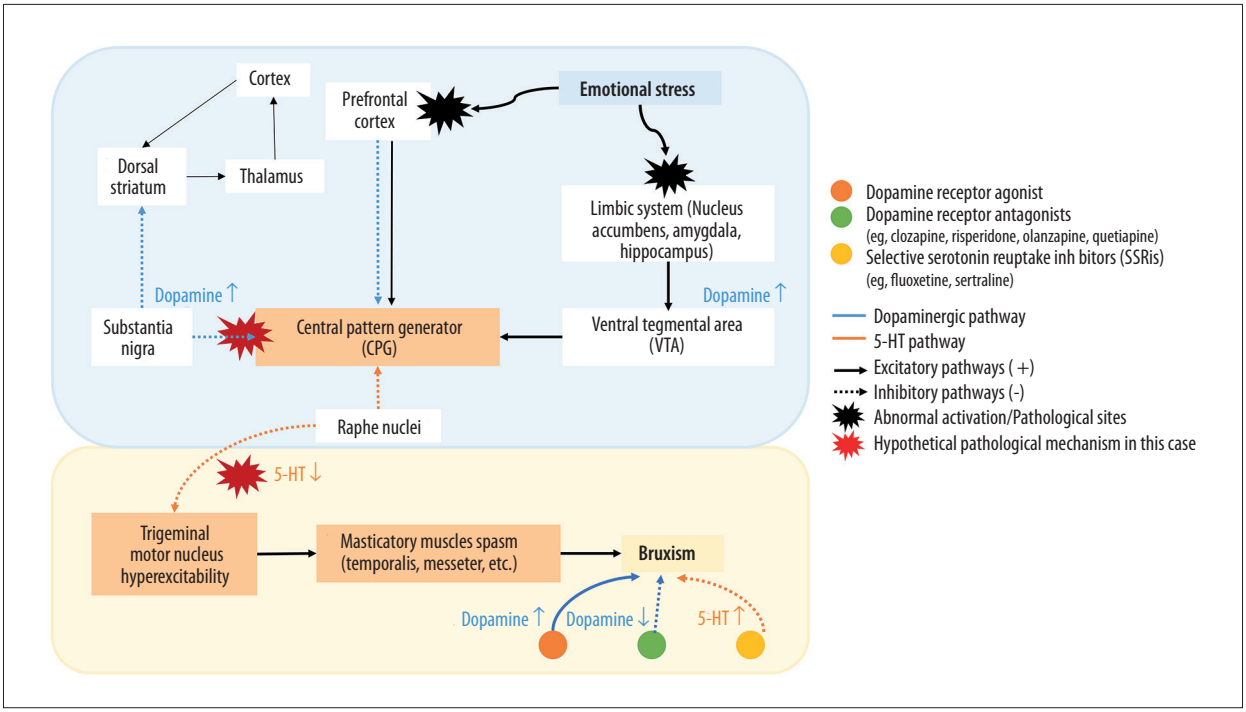

Figure 2. Proposed mechanism of bruxism and the effects of different classes of drugs.

this patient provides a plausible neuroanatomical substrate for the development of bruxism. Consequently, bruxism is hypothesized to result from reduced prefrontal dopaminergic transmission. This hypothesis is supported by a trial investigating levodopa plus benserazide for sleep bruxism, which demonstrated reduced masseter muscle activity on electromyography in 70% of patients [20]. This finding further corroborates the potential role of diminished dopaminergic signaling in the prefrontal cortex.

Research has indicated that dopamine receptor agonists can induce bruxism by modulating dopaminergic and serotonergic pathways [21,22]. Conversely, the use of dopamine receptor antagonists such as clozapine, risperidone, olanzapine, quetiapine, and haloperidol has been associated with tardive bruxism, which may manifest as grinding and/or clenching of teeth, or as an incomplete form of tardive oromandibular dystonia. These movements often subside during sleep. Selective serotonin reuptake inhibitors (SSRIs), including venlafaxine, fluoxetine, and sertraline, have also been implicated in bruxism [21]. Excessive serotonergic neuron activation may inhibit dopaminergic neurons in this pathway, reducing dopamine release and leading to bruxism [23,24]. In the present case, we ruled out the effects of these medications. Pharmacological evidence has linked both dopaminergic agonists and antagonists to bruxism, suggesting that basal ganglia lesion-inducing dopaminergic imbalance—rather than simple deficiency—may disinhibit brainstem central pattern generators (**Figure 2**). However, the precise mechanisms underlying bruxism in the context of acute neurological conditions remain unclear and elucidation of these mechanisms warrants further investigation. Alterations in dopaminergic neurotransmission represent a promising avenue for future research.

Comparison With Existing Literature

Current treatment strategies for bruxism include non-pharmacological approaches such as sleep hygiene education and reduction of alcohol and caffeine intake. However, a randomized controlled trial found that a 4-week program combining sleep hygiene instruction and progressive muscle relaxation did not significantly improve sleep bruxism [25]. Occlusal splints and stabilization appliances are recommended as first-line treatments due to their affordability, convenience, and ability to reduce bruxism frequency in the short term. However, they do not address the underlying etiology and may not improve long-term quality of life [26,27].

A meta-analysis by Fernández-Núñez et al demonstrated that BoNT-A is an effective treatment for bruxism, significantly reducing both the frequency of grinding episodes and pain levels compared with traditional therapies and placebo [11]. To our knowledge, no prior report has systematically applied the STAB framework to assess post-stroke bruxism, nor provided 6-month follow-up with structured documentation of injection protocol and response. Typical BoNT-A injection doses for bruxism range from 20 to 60 U per masseter muscle and 15 to 40 U per temporalis muscle, with total doses varying between

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30 and 200 U [28]. In the present case, given the clinically observed higher tension in the masseter muscles compared with the temporalis, a dose of 50 U per masseter muscle was administered. A reduction in bruxism frequency was noted 24 hours post-injection, with near-complete resolution of symptoms by 48 hours. This clinical improvement was sustained at the 6-month follow-up. The clinical improvement observed within 24-48 hours is earlier than the typical 48-72 hour onset cited for BoNT-A in skeletal muscle [12,29]. However, this rapid response has been previously reported in bruxism: Ivanhoe et al noted resolution 2 days post-injection [13], and Mallaguti et al reported improvement as early as day 1 [30]. Possible mechanisms include activity-dependent toxin uptake (facilitated by persistent bruxism) and a direct analgesic effect via inhibition of pain-related neuropeptides (eg, substance P, calcitonin gene-related peptide) [31,32]. These observations are consistent with prior reports but warrant further investigation.

This case does not represent the first report of bruxism following central nervous system injury or its treatment with BoNT-A. Rather, it illustrates a well-documented, illustrative case in a challenging population, with emphasis on standardized assessment and long-term follow-up.

Limitations

However, there are some limitations in this case; in particular, the fact that quantitative surface electromyography and polysomnography were not performed. In addition, this is a single case, and large studies are needed. Several potential confounders were considered. No new medications associated with bruxism (eg, SSRIs, antipsychotics) were introduced prior to symptom onset. The patient's anxiety (GAD-7 score 6) may have contributed to awake bruxism, but nocturnal symptoms persisted during sleep, independent of conscious anxiety. Concurrent rehabilitation interventions (physical therapy, occupational therapy) remained unchanged before and after BoNT-A injection, minimizing the likelihood that functional improvement resulted from non-pharmacological factors.

Conclusions

BoNT-A injection can alleviate bruxism symptoms in patients following cerebral hemorrhage, but further cases are needed for validation. The STAB framework provided a structured, multidimensional approach to assessment, facilitating clear documentation of bruxism status and associated comorbidities, along with a systematic clinical characterization. Although botulinum toxin has been previously reported for post-stroke bruxism, this case contributes a well-characterized example with long-term follow-up and standardized assessment. Controlled studies are needed to establish efficacy, optimal dosing, and long-term outcomes in this population.

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Institution Where Work Was Done

Ningbo Rehabilitation Hospital, Ningbo, Zhejiang, PR China.

Informed Consent

Written informed consent for publication of this case report was obtained from the patient.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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