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Original

Botulinum toxin type A in trigeminal neuralgia

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Abstract

Objective
To evaluate the efficacy and safety of botulinum toxin type A (BoNT-A) in patients with refractory or inadequately controlled trigeminal neuralgia treated at a specialized outpatient clinic.

Methods
Retrospective, observational, descriptive study was conducted through a review of electronic medical records of patients diagnosed with trigeminal neuralgia and treated at the Neuromuscular Blockade Outpatient Clinic of the Municipal Health Department of Joinville, Santa Catarina, Brazil, between 2019 and 2022. Patients who received BoNT-A injections and had documented clinical follow-up were included. As this was a retrospective review of previously existing medical records, with no prospective intervention or direct patient enrollment, no ethics committee approval was obtained for the present chart-based analysis. Patients who were already receiving pharmacological treatment for pain continued their prescribed medications during follow-up, with BoNT-A administered as an adjunctive treatment. Pain intensity was routinely assessed using the Visual Analog Scale (VAS). The observation period for treatment response and recurrence was 12 months.

Results
Nineteen patients were included in the final analysis. The mean age was 67.9 years (range, 27–87 years). Most patients presented severe baseline pain, with VAS scores ranging from 8 to 10. BoNT-A doses ranged from 20 to 180 U. Following treatment, 18 of 19 patients (94.7%) achieved a clinically significant response, defined as a reduction of more than 50% in pain intensity. Several patients experienced complete or near-complete remission, maintaining VAS scores between 0 and 3 during follow-up. Pain recurrence was observed predominantly in patients in whom reinjection was delayed or follow-up intervals were prolonged. Because the study was retrospective, the precise timing and clinical reasons for delayed reinjection could not always be determined. One patient developed transient facial paresis, which resolved completely without permanent sequelae. No serious adverse events were documented.

Conclusion
In this retrospective case series, BoNT-A was associated with substantial pain reduction and a favorable safety profile in patients with refractory or inadequately controlled trigeminal neuralgia. However, the concomitant use of previous pharmacological treatments, the absence of a standardized injection protocol, and the retrospective nature of the study prevent attribution of the observed clinical response exclusively to BoNT-A. Prospective controlled studies with standardized injection protocols, dilution, injection depth, technique, and predefined reinjection intervals are warranted.

Keywords:
Trigeminal neuralgia
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Introduction

Trigeminal neuralgia (TN) is a neuropathic pain disorder characterized by recurrent episodes of severe unilateral facial pain, typically described as electric shock-like, stabbing, or lancinating pain (1). The pain is usually brief and paroxysmal, although some patients may also experience a persistent background pain between attacks. The condition can substantially impair quality of life and interfere with speaking, oral hygiene, eating, drinking, and other daily activities (2,3). In addition to its physical impact, the unpredictability and severity of pain attacks may contribute to functional limitation, sleep disturbance, social withdrawal, and psychological distress.

Classical TN is commonly associated with neurovascular compression of one or more trigeminal nerve root entry zones, which may lead to focal demyelination and neuronal hyperexcitability (4). However, the pathophysiology of TN is complex and may involve both peripheral and central mechanisms. Structural changes at the trigeminal root entry zone may facilitate abnormal ectopic impulse generation and ephaptic transmission, contributing to the characteristic paroxysmal pain experienced by affected patients. The clinical presentation may vary according to the trigeminal division involved, with the maxillary and mandibular divisions frequently affected.

Although carbamazepine and oxcarbazepine remain the first-line pharmacological treatments, a substantial proportion of patients experience inadequate pain control or adverse effects that limit treatment adherence (5,6). Long-term treatment may also become challenging because of dose-related adverse effects, drug interactions, contraindications, or loss of therapeutic efficacy. In patients with persistent symptoms despite pharmacological therapy, surgical procedures such as microvascular decompression, radiofrequency thermocoagulation, balloon compression, or stereotactic radiosurgery may be considered. Nevertheless, these interventions are not appropriate or acceptable for all patients, particularly those with substantial surgical risk, contraindications, or preference for less invasive treatment options.

Botulinum toxin type A (BoNT-A) has emerged as a promising therapeutic option for severe neuropathic pain syndromes. Beyond its well-established neuromuscular blocking effects, experimental evidence suggests that BoNT-A modulates the release of nociceptive neurotransmitters, including substance P, glutamate, and calcitonin gene-related peptide (CGRP), thereby reducing peripheral and central sensitization (7,8). This proposed multimodal analgesic effect provides a biological rationale for the use of BoNT-A in pain disorders in which conventional analgesic strategies may be insufficient.

In trigeminal neuralgia, BoNT-A is generally administered locally in the painful facial region, allowing treatment to be directed toward the anatomical distribution of symptoms. Randomized controlled trials have reported significant reductions in pain intensity and attack frequency following BoNT-A treatment, with a generally favorable safety profile (9–11). Systematic reviews and meta-analyses have also reported potential therapeutic benefits of BoNT-A in TN (6,7,12). Nevertheless, the available evidence remains limited by relatively small sample sizes, differences in doses and injection techniques, heterogeneous patient populations, and differences in outcome assessment.

Real-world clinical data may therefore contribute to understanding how BoNT-A performs in routine specialist practice, particularly among patients with refractory or inadequately controlled symptoms who may differ from participants enrolled in randomized clinical trials. Observational data can also provide information regarding treatment tolerability, recurrence of pain, the need for repeated administration, and the practical challenges associated with long-term follow-up.

The present study aimed to describe the clinical experience of a referral center in the treatment of refractory or inadequately controlled trigeminal neuralgia using BoNT-A and to characterize treatment response, adverse events, pain recurrence, and long-term follow-up outcomes. We hypothesized that BoNT-A treatment would be associated with clinically meaningful reductions in pain intensity and an acceptable safety profile in patients with TN treated in a specialized outpatient setting.

Methods

Study design and setting

This retrospective, observational, descriptive study was conducted through a review of electronic medical records of patients treated at the Neuromuscular Blockade Outpatient Clinic of the Municipal Health Department of Joinville, Santa Catarina, Brazil.

Patients treated between 2019 and 2022 were evaluated. The outpatient clinic provides specialized treatment for patients referred for therapeutic use of botulinum toxin in neurological and neuromuscular conditions.

The retrospective design allowed the evaluation of treatment response and adverse events under routine clinical conditions rather than under a predefined experimental protocol. All clinical information was obtained exclusively from previously existing medical records. No prospective intervention, randomization, or additional procedure was performed for research purposes.

Because this investigation consisted exclusively of a retrospective review of existing medical records and did not involve prospective patient recruitment or additional interventions, no ethics committee

approval was obtained for this chart-based analysis. Patient information was analyzed from the clinical documentation available in the medical records.

Patients with trigeminal neuralgia were evaluated clinically by the medical team, and treatment was prescribed according to the severity and distribution of symptoms and the clinical response to previous therapies.

Participants

Eligible participants were patients diagnosed with trigeminal neuralgia by a neurologist according to established clinical criteria who underwent treatment with BoNT-A and had documented clinical follow-up after treatment.

Patients with alternative diagnoses of facial neuropathic pain, incomplete follow-up data, or insufficient clinical documentation were excluded from the analysis. The diagnosis was based primarily on the clinical characteristics of the pain and the neurological evaluation documented in the medical records.

Baseline clinical information, including age, sex, affected trigeminal branch, disease duration, previous treatments, and baseline pain intensity, was collected when available. The affected trigeminal division was classified according to the ophthalmic (V1), maxillary (V2), and mandibular (V3) branches.

The study population consisted of patients who had been referred for specialized treatment because of persistent or inadequately controlled symptoms. Previous pharmacological treatment was documented when available, allowing characterization of the clinical context in which BoNT-A was administered.

Concomitant pharmacological treatment

An important characteristic of this real-world cohort was that BoNT-A was used as an adjunctive treatment rather than necessarily as a replacement for previous pharmacological therapy.

Patients who were already receiving medications for pain control or pain modulation at the time of BoNT-A treatment continued their prescribed medications during the observation period according to routine clinical practice. These medications were not systematically discontinued following BoNT-A administration.

Therefore, the clinical response observed during follow-up reflects the effect of BoNT-A within the context of the patients' existing therapeutic regimens. Because the retrospective medical records did not allow standardized assessment of medication adherence, dose adjustments, or the independent contribution of each concomitant medication, the effect of BoNT-A cannot be isolated from the effects of concurrent pharmacological treatment.

Botulinum toxin treatment

BoNT-A (Dysport®, Ipsen Pharma) was administered according to an individualized clinical approach based primarily on the distribution and intensity of pain and the affected trigeminal branch.

Injection sites included areas corresponding to the ophthalmic (V1), maxillary (V2), and mandibular (V3) divisions of the trigeminal nerve, as well as adjacent symptomatic areas when clinically indicated. The anatomical distribution of injections was determined according to the patient's predominant painful area and the clinical assessment performed during the outpatient visit.

The total BoNT-A dose ranged from 20 to 180 U.

Because this was a retrospective study based exclusively on medical records, detailed information regarding the preparation and administration of BoNT-A was not consistently available for all patients. In particular, the exact dilution used, the final concentration of the toxin, the injection depth, needle positioning, number of injection points, injection volume per site, and specific injection technique could not be reliably reconstructed from the available medical records.

Therefore, although the treatment was individualized according to the clinical presentation, it was not possible to establish a standardized dilution, injection depth, or technical protocol applicable to the entire cohort. These characteristics should be considered important sources of heterogeneity and limitations of the present study.

Repeated treatment and follow-up

Repeated treatment was performed according to the clinical response observed during routine outpatient follow-up. Patients were reassessed periodically, and subsequent BoNT-A administration was considered according to the recurrence or persistence of symptoms and the treating physician's clinical assessment.

The observation period analyzed in the present study was 12 months following treatment. During this period, clinical records were reviewed for changes in pain intensity, recurrence of symptoms, subsequent BoNT-A administration, and adverse events.

Outcome assessment

Pain intensity was assessed using the Visual Analog Scale (VAS), which was routinely recorded during outpatient follow-up visits. The VAS was used as the principal clinical measure of pain severity and allowed comparison between baseline and subsequent assessments.

Treatment response was evaluated by comparing baseline and post-treatment VAS scores. A clinically significant response was defined as a reduction of more than 50% in pain intensity compared with baseline.

Complete or near-complete clinical remission was characterized by very low post-treatment pain scores, with several patients achieving VAS scores between 0 and 3 during follow-up.

Pain recurrence and the need for subsequent BoNT-A administration were also recorded when documented in the medical records. Recurrence was assessed clinically based on the return or worsening of facial pain after an initial response to treatment.

Patients were followed for approximately 3 years, with monthly outpatient follow-up visits. This extended observation period allowed assessment of the persistence of treatment response, recurrence of symptoms, need for repeated treatment, and adverse events over time.

Results

Initially, 22 medical records of patients treated at the referral center were identified. After application of the eligibility criteria, 19 patients with a confirmed diagnosis of trigeminal neuralgia were included in the final analysis.

The patients' ages ranged from 27 to 87 years, with a mean age of 67.9 years. The clinical characteristics of the disease, including the affected trigeminal branch and disease duration, are summarized in Table 1. Most patients presented unilateral disease.

At baseline, VAS scores ranged from 8 to 10, indicating severe pain in the analyzed population. The high baseline pain scores reflected the clinical severity of the population treated at the specialized outpatient service and the indication for BoNT-A in patients with refractory or inadequately controlled symptoms.

Following BoNT-A administration, 18 of the 19 patients (94.7%) experienced a reduction of more than 50% in pain intensity. This reduction was observed during clinical follow-up and represented the principal measure of treatment response in the present study.

Table 1. Clinical characteristics of the disease

Patient	**n**
Female	13
Mean age	67,9
Age range	27-82
Baseline VAS	Predominantly 8-10
Responders (>50% pain reduction)	18/19 (95,7%)
Non-responders	1/19 (5,3%)
Dysport dose range	20-180 U
Serious adverse events	0
Transient facial paresis	1 (5,3%)

Several patients achieved complete or near-complete symptom remission, with VAS scores between 0 and 3 during follow-up. The observed reduction in pain was maintained in patients who continued regular outpatient follow-up and received subsequent treatment when clinically indicated.

The BoNT-A dose ranged from 20 to 180 U. Because treatment was individualized, the dose and injection sites varied according to the distribution of pain and the affected trigeminal branch. This variation reflected the clinical approach used at the specialized outpatient clinic, in which treatment was adapted to each patient's symptoms rather than administered according to a single fixed injection protocol.

Patients were followed for approximately 3 years, with monthly outpatient follow-up visits. This longitudinal follow-up permitted repeated clinical assessment of pain intensity and treatment response and allowed documentation of symptom recurrence and adverse events over an extended period.

Pain recurrence was observed predominantly in patients in whom reinjection was delayed, or in those with prolonged intervals between scheduled outpatient visits. Among patients with adequate follow-up and reinjection according to the treating physician's planned schedule, pain control was generally maintained. Because the study was retrospective, the exact contribution of treatment interval to recurrence could not be quantitatively established.

Regarding safety, one patient (5.3%) developed transient facial paresis following treatment. The deficit resolved completely without permanent sequelae. No serious adverse events were documented during the follow-up period.

Overall, the findings indicate a high proportion of patients with clinically meaningful pain reduction following BoNT-A administration, together with a low frequency of documented adverse events during the long-term observation period.

Discussion

Trigeminal neuralgia is a highly disabling neuropathic pain disorder characterized by recurrent episodes of severe facial pain that may substantially impair quality of life. Although carbamazepine and oxcarbazepine remain the first-line pharmacological treatments, a proportion of patients experience inadequate pain control, treatment intolerance, or loss of efficacy over time (5,6). These limitations have stimulated interest in alternative therapeutic strategies, particularly for patients with persistent symptoms despite conventional pharmacological treatment.

In this context, BoNT-A has emerged as a minimally invasive therapeutic option for patients with refractory or inadequately controlled TN. In the present study, 18 of 19 patients (94.7%) achieved a reduction of more than 50% in pain intensity, while several patients experienced complete or near-complete remission during follow-up. The high proportion of responders observed in this cohort suggests that BoNT-A may provide clinically meaningful analgesia in selected patients treated in specialized outpatient settings.

The response rate observed in this study should, however, be interpreted cautiously because of the retrospective design, small sample size, lack of a control group, and individualized treatment protocol. The observed response rate is therefore not directly comparable with randomized controlled trials in which patients were selected according to predefined eligibility criteria and treated using standardized protocols. In addition, the present cohort represents a real-world population referred to a specialized service, which may differ from populations included in controlled clinical trials.

Previous randomized controlled trials have nevertheless demonstrated clinically relevant benefits of BoNT-A in TN. Wu et al.(11) reported significant reductions in pain intensity and attack frequency following BoNT-A treatment in a randomized, double-blind, placebo-controlled trial. Similarly, Zhang et al.(10) demonstrated significant improvements in pain outcomes in a randomized, double-blind, placebo-controlled study evaluating two BoNT-A doses. These findings provide clinical support for the analgesic effects observed in the present cohort.

A systematic review and meta-analysis by Morra et al.(6) also reported significant improvements in pain-related outcomes with BoNT-A compared with placebo in randomized controlled trials, although the authors emphasized the limitations of the available evidence, including small sample sizes and heterogeneity among studies. More recently, Hu et al.(12) published an updated systematic review and meta-analysis reporting reductions in pain intensity and attack frequency following BoNT-A treatment in patients with TN.

The high response rate observed in our sample is therefore broadly consistent with the direction of findings reported in randomized studies and systematic reviews, although the magnitude of the response observed in this retrospective cohort should not be interpreted as an estimate of comparative treatment efficacy. The present findings should instead be considered complementary real-world evidence supporting further investigation of BoNT-A as a treatment option for selected patients.

The analgesic mechanism of BoNT-A appears to extend beyond inhibition of acetylcholine release at the neuromuscular junction. Experimental evidence suggests that BoNT-A can modulate nociceptive transmission by reducing the peripheral release of neurotransmitters and neuropeptides, including substance P, glutamate, and CGRP (7,8). These effects may contribute to reductions in peripheral and central sensitization and may partially explain the analgesic effects observed in neuropathic pain conditions.

The modulation of peripheral nociceptive signaling may be particularly relevant in TN, in which abnormal trigeminal activity plays a central role in the generation and maintenance of pain. By reducing the release of pain-related mediators at peripheral nerve terminals, BoNT-A may decrease nociceptive input and consequently reduce the amplification of pain signaling. Although the precise mechanism responsible for the clinical response remains incompletely understood, the available experimental and clinical evidence provides a plausible biological basis for its use.

In the present study, the injection sites and doses were individualized according to the distribution of pain and the affected trigeminal branch. This individualized approach may have facilitated treatment adaptation to the clinical characteristics of each patient. However, the absence of a standardized injection protocol also represents an important source of heterogeneity and limits the reproducibility and interpretation of the findings.

The dose range observed in this cohort, from 20 to 180 U of Dysport®, also illustrates the variability in treatment requirements among patients. Differences in dose may reflect differences in the anatomical distribution of pain, number of affected trigeminal divisions, severity of symptoms, and clinical response to previous treatment. Because the present study was observational, it was not designed to determine an optimal dose or to establish a dose-response relationship.

The long-term follow-up of approximately 3 years, with monthly outpatient assessments, represents an additional characteristic of

this cohort. Extended follow-up allowed the clinical team to monitor the persistence of treatment response and to identify recurrence of pain over time. Longitudinal observation is particularly relevant for TN because treatment response may vary over time and repeated interventions may be necessary in patients experiencing recurrence.

Pain recurrence appeared to be associated with delayed reinjection or prolonged intervals between outpatient visits. However, this observation should be interpreted cautiously because the retrospective design did not allow us to establish a causal relationship between treatment interval and pain recurrence. Other factors, including individual disease characteristics, baseline severity, duration of treatment response, and variations in adherence to follow-up, may also have influenced recurrence.

The observation that pain control was generally maintained among patients receiving regular follow-up and reinjection when clinically indicated may support the potential role of repeated treatment in maintaining symptom control. Nevertheless, prospective studies are required to determine the optimal interval between BoNT-A administrations and to establish whether scheduled reinjection or symptom-driven treatment provides better long-term outcomes.

The safety findings were generally favorable. One patient developed transient facial paresis, which resolved completely without permanent sequelae. This adverse event is consistent with the potential local effects of BoNT-A when injections are performed near motor branches. The absence of serious adverse events during the follow-up period is consistent with the generally favorable safety profile reported in previous studies.

The occurrence of transient facial paresis also highlights the importance of accurate anatomical localization and careful selection of injection sites. Because the trigeminal branches are anatomically close to muscles responsible for facial expression and other motor functions, diffusion of the toxin may produce temporary weakness in adjacent muscles. Such effects are generally reversible, as observed in the present cohort.

Another relevant aspect of this study is that treatment was performed in a specialized outpatient setting under routine clinical conditions. The real-world nature of the cohort may increase the clinical relevance of the findings because patients treated in specialist services may present with greater disease complexity or previous treatment failure than patients recruited into controlled trials. At the same time, this characteristic may introduce selection bias and limits the generalizability of the results.

This study has several limitations. First, its retrospective design introduces the possibility of selection and information bias. Second, the sample size was small, limiting the generalizability of the findings. Third, there was no control group, preventing direct comparison with placebo or conventional pharmacological treatment. Fourth, the injection protocol was individualized, resulting in heterogeneity in dose and injection sites. Fifth, the study relied on information recorded in medical records, which may have resulted in missing or inconsistently documented clinical variables.

Another limitation is that the study was not designed to evaluate specific predictors of treatment response. Therefore, it was not possible to determine whether age, affected trigeminal division, disease duration, baseline pain intensity, previous treatments, or BoNT-A dose influenced the probability or magnitude of clinical response.

Finally, although the patients were followed for approximately 3 years with monthly visits, the observational design did not allow a standardized assessment of the exact duration of pain relief after each injection. The timing of recurrence and subsequent treatment was determined according to routine clinical practice. Future prospective studies should incorporate standardized intervals for pain assessment and predefined criteria for reinjection.

Despite these limitations, the present study provides real-world clinical data from a specialized outpatient service and suggests that BoNT-A may be a useful treatment option for selected patients with refractory or inadequately controlled TN, particularly when conventional pharmacological treatment is insufficient or poorly tolerated. The high proportion of patients achieving clinically meaningful pain reduction, together with the low frequency of documented adverse events, supports the need for further prospective investigation.

Prospective studies using standardized injection protocols, predefined follow-up intervals, validated outcome measures, and larger patient populations are warranted. Future research should also investigate optimal dosing, anatomical injection patterns, duration of analgesic benefit, predictors of response, and the most appropriate strategy for repeated treatment.

The retrospective nature of this study represents both a methodological characteristic and an important limitation. The analysis was based exclusively on information already documented in medical records, without prospective intervention or additional procedures performed for research purposes. Consequently, no ethics committee approval was obtained for the present chart review.

This design allowed evaluation of BoNT-A use under routine clinical conditions but limited the availability and standardization of important variables. In particular, information regarding medication adherence, changes in concomitant pharmacological

treatment, exact BoNT-A preparation, dilution, injection depth, technical administration, and precise timing of symptom recurrence was not consistently available.

Therefore, the findings should be interpreted as descriptive real-world clinical evidence rather than as evidence derived from a controlled experimental protocol.

Conclusion

In this retrospective case series, botulinum toxin type A was associated with a substantial reduction in pain intensity and a favorable safety profile among patients with refractory or inadequately controlled trigeminal neuralgia treated at a specialized referral center.

Most patients achieved a reduction of more than 50% in pain intensity, and several experienced complete or near-complete symptom remission during follow-up. One patient developed transient facial paresis, which resolved completely without permanent sequelae, and no serious adverse events were observed.

Patients were followed for approximately 3 years, with monthly outpatient assessments, allowing longitudinal evaluation of treatment response, pain recurrence, and safety. Pain recurrence was observed predominantly in association with delayed reinjection or prolonged intervals between outpatient visits; however, the retrospective design prevents establishing a causal relationship.

The individualized administration of BoNT-A allowed treatment to be adapted to the distribution and severity of symptoms and the affected trigeminal branch. However, this same characteristic introduces heterogeneity and prevents determination of an optimal dose or standardized injection protocol from the present data.

Given the small sample size, lack of a control group, retrospective design, and heterogeneity of the treatment protocol, these findings should be interpreted as observational evidence rather than definitive evidence of treatment efficacy. Nevertheless, the observed clinical response and favorable safety profile provide supportive real-world data regarding the potential role of BoNT-A in the management of selected patients with TN.

Prospective controlled studies with standardized injection techniques, predefined follow-up intervals, validated pain outcomes, and longer follow-up are needed to further establish the efficacy, safety, optimal dosing, treatment interval, and durability of BoNT-A in trigeminal neuralgia.

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