



Treating Bruxism-Related Muscle Spasm and Malocclusion: An Intervention Study Using Local Anesthetic without Vasoconstrictor

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ABSTRACT

Background: Hyperactivity of the masticatory muscles and acute malocclusion in bruxism are problematic conditions that are usually misdiagnosed as occlusal disorders. The therapeutic efficacy of local anesthetic infiltration without a vasoconstrictor in local anesthesia cases is still dubious.

Objective: To assess the clinical impact of 2% lidocaine infiltration without vasoconstrictor in bruxism-related masticatory muscle hyperactivity and acute malocclusion, and to determine the impact of dental restorations on the response of treatment.

Methods: The clinical interventional study was carried out in a sample of 20 patients (29-40 years), in the form of two groups: Group A (with restorations) and Group B (without restorations), this time. Everyone was infiltrated at the deepest point of the most tender point of the masseter muscle with 2% lidocaine without vasoconstrictor. The measures were assessed using pain (VAS), muscle tenderness, and occlusal stability at baseline, 30 minutes, 24 hours, and 2-3 months. The chi-square and Fisher's exact tests were used to analyze the data.

Results: In Group B (100%), all patients improved completely and sustained, and none in Group A responded. The difference between them was very high ($\chi^2 = 16.20$, $p < 0.001$; Cramer's $V = 0.90$).

Conclusion: Local infiltration of plain lidocaine is an effective method to decrease the hyperactivity of muscles and restore the occlusal balance in acute malocclusion due to bruxism in the absence of occlusal interferences. This is an easy, inexpensive technique that can be of much use as an addition to the treatment of muscle-origin occlusal disturbances.

Keywords: Bruxism, myofacial pain dysfunction syndrome, acute malocclusion, lidocaine without vasoconstrictor, occlusal interference.

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INTRODUCTION

Bruxism is a repetitive clenching or grinding of the teeth, specific to the jaw muscles, which may take place either during sleep or when awake, being amongst the most widespread parafunctional habits that involve the stomatognathic system [1, 2]. Continued bruxism can result in masticatory muscle hyperactivity and myofascial pain dysfunction syndrome (MPDS), which can present as muscle tenderness, trigger points, headaches, ear pain, and even acute malocclusion [3]. Different forms of treatment modalities, such as behavioral therapy, occlusal splints, physiotherapy, and pharmacological intervention, have demonstrated partial effect on controlling the signs and symptoms of bruxism [4]. Although splints and stretching exercises can improve tenderness, their effects in acute occlusion disturbances are inconsistent [5, 6]. Biofeedback methods are also being studied, and they are yet to be used in clinical practice [6]. Bilateral temporomandibular joint subluxation may cause significant functional impairment, including malocclusion, difficulty chewing, and speech problems [7]. There have been studies indicating that there are possible associations between bruxism and malocclusion, but this has not been conclusive [8, 9]. Although these findings exist, no interventional studies have involved the use of local anesthetic infiltration without vasoconstrictors as a treatment option for muscle hyperactivity and acute malocclusion caused by bruxism in patients with previous restorations and in patients without any previous restorations. In the past, emphasis has been put on systemic safety in hypertensive or diabetic patients [10, 11]. The current study was consequently aimed at evaluating the effect of the local anesthetic injection without a vasoconstrictor on bruxism-induced masticatory muscle hyperactivity and acute malocclusion in patients with or without restorations.

METHODOLOGY

Study Design

This study was a prospective clinical interventional study and was carried out at Dr. Ghassan Al-Khalidi Dental Clinic, Taiz, Yemen, from January 2024 to January 2025.

Study Population

The total number of patients was 20 (10 males and 10 females). Every participant showed signs and symptoms of muscle hyperactivity caused by bruxism, including teeth grinding, ear pains, headaches, tenderness of jaw muscles, and acute instability at the occlusa. The patients were separated into two equally sized clusters:

A 10-patient group (Group A): a group of patients that have had a dental restoration (amalgam or composite fillings, or fixed prosthetic crowns).

Group B (n = 10): Patients who did not have any restorations or any prosthetic work. This grouping was based on the fact that dental restorations can modify occlusal morphology and neuromuscular responses, which may influence bruxism-related muscle activity as reported in the literature [12].

Inclusion Criteria

Eligible participants are adults aged 18 to 45 years who present with clinical manifestations of bruxism, such as self-reported or clinically observed teeth grinding. Additionally, they must exhibit tenderness upon palpation of the masseter or temporalis muscles (Figure 1) and the existence of trigger points associated with referred pain to areas such as the ear, temple, or neck. Finally, inclusion requires normal panoramic radiographs, with no evidence of joint pathology, bone lesions, or gross dental abnormalities.

Exclusion Criteria

Participants were excluded if they met any of the following conditions: a documented history of hypersensitivity or allergic reaction to lidocaine or other local anesthetic agents; presence of systemic neuromuscular or neurological disorders; current or recent use of sedatives, anxiolytics, or muscle-relaxant medications; history of recent trauma, surgical intervention, or diagnosed temporomandibular joint (TMJ) disorders; pregnancy or breastfeeding at the time of enrollment; or presence of severe systemic disease classified as American Society of Anesthesiologists (ASA) physical status III or higher.





Figure 1: Palpation of tenderness

Intervention Procedure

Each of the patients was infiltrated with non-vasoconstrictor 2% lidocaine (1.8 mL per carpule, Septodont, France).

Injection site: The tenderest point of the masseter muscle was found by palpation.

Technique: 1.5-1.8 mL of lidocaine was injected in the perimuscular site blindly and slowly with a 27-gauge short needle at a depth of about 8-10 mm, perimuscular (Figure 2).

Laterality: In bilateral cases injections were made on both sides (Figures 3 and 4).

Consistency of the operators: The operator used made all the injections, thereby standardizing the injections.

Aseptic: Aseptic solution 70 percent isopropyl alcohol was applied to the area of insertion beforehand [13, 14].



Figure 2: Injection in per muscular

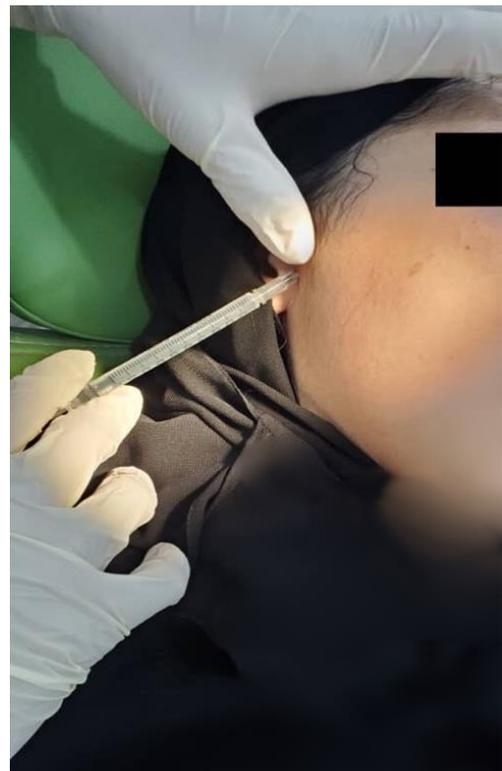


Figure 3: Injection on right side of muscle



Figure 4: Injection on left side of muscle

Clinical Assessment

The standardized parameters were used in the evaluation of the patients at baseline and during follow-up [12, 15, 16]:

- 1. Pain:** was assessed on a 10-point Visual Analog Scale (VAS) (0 no pain, 10 worst pain imaginable).
- 2. Muscle tenderness:** the masseter and temporalis muscles are examined by palpation, at which 5 seconds of approximately 1 kg of digital pressure is used. It was noted to be tender or not.
- 3. Trigger points:** the location of a trigger point is detected by response to palpation reaction to local pain.
- 4. Occlusion stability:** it is tested by asking patients to bite into maximum intercuspation. Premature contact, mandibular shift, or occlusal interference was put on record.

Follow-Up Protocol

Thirty minutes after injection: measurement of short-time effect on pain and occlusion. 24 hours after injection: re-evaluation of the strength of pain and stability of the occlusion.

2-3 months follow-up: final assessment of long-term effect. Improved outcome was determined as reduction in VAS score, no muscle tenderness and trigger points, and no premature contacts with stable occlusion. In addition, not improved outcome was referred to as persistence of symptoms or occlusal disturbances [12, 15, 16].

Ethical Considerations

The Medical Ethics Committee at the University of Science and Technology, Aden, Yemen, has approved the study [MEC/AD0110]. Written informed consent was obtained from all participants. The study followed the principles of the Declaration of Helsinki.

Statistical Analysis

Data were analyzed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as frequencies and percentages. Intergroup comparisons were performed using the chi-square test and Fisher's exact test where appropriate. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. A p-value < 0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

Twenty patients were recruited, half of them in Group A (with restorations, n=10) and the other half in Group B (without restorations, n=10). Participants were aged 29 to 40, with a mean age of 34.5 +3.2 (10 males and 10 females per group). Group A patients primarily complained of teeth grinding, headache, and cervical pain. Group B, on the other hand, mostly reported ear pain as related to headache and masticatory muscle tenderness. All patients had trigger points in the masseter muscle, and no pathological changes were found in the panoramic radiographs in either group (Table 1).



Table 1: Demographic and Clinical Characteristics of Populations

Variable	Group A	Group B
Age (years)	34.8 ± 3.1	34.2 ± 3.3
Male	5 (50%)	5 (50%)
Female	5 (50%)	5 (50%)
Main Complaint	Headache, pain around jaw during movement, neck pain, teeth grinding	Headache, pain around jaw during movement, ear pain, neck pain
Previous Treatment	Dental fillings or prosthetic restorations	No dental fillings or prosthetic restorations
Clinical Examination	Trigger points	Trigger points
Radiographic Examination	Panoramic X-ray (normal)	Panoramic X-ray (normal)
Diagnosis	Myofascial Pain Dysfunction Syndrome	Myofascial Pain Dysfunction Syndrome
Treatment	Local anesthetic without vasoconstrictor	Local anesthetic without vasoconstrictor
Follow up	2-3 months	2-3 months

Treatment Outcomes

The results of infiltration of the 2% lidocaine solution in the absence of a vasoconstrictor were significantly different: Group A (where restorations were made): No patients (0/10; 0%) showed any improvement in clinical terms. Follow-up did not show any changes in persistent pain, hyperactivity of the muscles, or occlusal disturbances. Group B (no restorations): All subjects (10/10; 100 percent) reported a significant and persistent improvement, such as the decrease in pain intensity, the disappearance of trigger points, and the achievement of stable occlusion, which was followed until 2-3 months.

Table 2: Treatment Outcomes Following Lidocaine Injection without Vasoconstrictor

Outcomes	Group A	Group B
Improved	0 (0.0%)	10 (100.0%)
Not improved		10 (100.0%) 0 (0.0%)
Total (n)		10 10

Statistical Analysis

Improvement rate: 0% in group A vs 100% in group B. Chi-square test: $\chi^2 = 16.2$, $p = 5.7 \times 10^{-5}$. Exact test

of Fisher: Odds ratio $p = 1.08 \times 10^{-5}$. The calculated odds ratio (OR) was infinite ($OR \rightarrow \infty$; 95% CI not estimable), reflecting the fact that all patients in Group B improved, whereas none in Group A did. When applying the Haldane–Anscombe correction to adjust for zero cell counts, the OR was estimated at 0.0 with a 95% CI of 0.0–0.35. Effect size: Cramer’s V = 0.90.

Table 3: Statistical Analysis of Treatment Outcomes

Test	Value	p-value
Chi-square test (χ^2)	16.2	< 0.001
Fisher’s exact test	-	< 0.001
Odds ratio (uncorrected)	$OR \rightarrow \infty$	
Odds ratio (corrected)	$OR = 0.0$ (95% CI: 0.0–0.35)	< 0.001
Cramer’s V (effect size)	0.90	-

DISCUSSION

A study was conducted to evaluate plasma dopamine levels in patients with myogenous temporomandibular disorders (M-TMD) and to examine their association with clinical pain manifestations. The findings indicated that bruxism was present in 44% of patients, while morning jaw stiffness and stress-related jaw tension were reported in 40% and 56% of cases, respectively. Based on these observations, the authors concluded that elevated plasma dopamine levels in M-TMD patients may play a significant role in pain modulation and stress-related pathophysiology, suggesting its potential utility as a biomarker for these conditions [17]. The current research examined the impact of the local anesthetic infiltration in the absence of a vasoconstrictor on the masticatory muscle hyperactivity and acute malocclusion associated with bruxism. The results showed a rather impressive difference between patients who had restorations (Group A) and patients who had no restorations (Group B). None of the patients who had undergone restorations were clinically improved, and all the patients who had no restorations were completely and permanently cured of the symptoms after the infiltration.

These findings indicate that the occlusal interferences that are linked to dental restorations could be a decisive factor in either the preservation or increase of the hyperactivity of the masticatory



muscles. It has been previously noted that there is an association between occlusal discrepancies and elevated electromyographic (EMG) levels of masticatory muscles when patients of bruxism are involved [18, 19]. This justifies the fact that patients with no restorations (and hence, probably no occlusal interferences) react more to lidocaine infiltration.

The process of this effect might come through the temporary neuromuscular modulating. Local anesthetics work by inhibiting sodium channels in the peripheral nerves, causing temporary inhibition of muscle hyperactivity and nociceptive input [20, 21]. Without a vasoconstrictor, infiltration gives faster systemic absorption, with a reduced anesthetic duration but possibly a higher degree of muscle relaxation at the beginning stages following the injection as a result of this rapid absorption [11]. This momentary inhibition of hyperactivity has the potential of breaking the pain-spasm-pain loop typical of the myofascial pain dysfunction syndrome (MPDS) [22].

In contrast, when restorations or occlusal interferences are present, the continuous proprioceptive input from altered occlusal contacts may perpetuate muscle hyperactivity, negating the transient benefit of anesthetic infiltration. Various researchers have emphasized that wrong occlusal morphology or 8-10 high points in restorations may stimulate or sustain bruxism-related symptoms and TMJ dysfunction [23-25]; this is in agreement with the total failure of improvement in group A of the present study.

Interestingly, the findings are in line with other studies that have revealed that local anesthetic infiltration of trigger points or masticatory muscles can result in a considerable reduction in pain in the short term and improved functioning in patients with myofascial pain disorders [26,27]. This is, however, the first research to our knowledge to assess the impact of restorations as a variable with respect to its impact on the response to infiltration therapy. It leads to a significant clinical issue to take into consideration: before proceeding with the pharmacological or injection-based interventions, priority must be given to the occlusal adjustment and restoration correction.

Considering the whole picture, these results emphasize the interdependence of the management of bruxism that is a combination of the prosthodontic,

occlusal management, and pain modulation. The findings also cast doubt on the formulation of the best anesthetics applicable to muscle-related conditions. Although vasoconstrictors extend anesthesia, they can also decrease vascular supply to the tissue and slow down recovery, compared to basic lidocaine that can trigger the development of faster metabolism and muscle normalization [28].

Most importantly, the limitations of the study should be mentioned. The sample was also small, and only one clinical center was sampled, which could restrict generalizability. Moreover, the study did not include a comparison group receiving lidocaine with a vasoconstrictor, as this would not align with the therapeutic goal of transient neuromuscular relaxation. The electromyographic test was not conducted to objectively measure the changes of muscle activity. However, the consistency of the results and high level of statistical correlation between the lack of restorations and the improvement (Cramer's V = 0.90) confirm the validity of the results. These findings need to be validated by future research that would use larger randomized controlled trials and EMG assessment to understand the underlying neurophysiological mechanisms.

CONCLUSION

This clinical interventional research revealed that 2% lidocaine without a vasoconstrictor, locally infiltrated around mucous membranes, is capable of reducing masticatory hyperactivity and acute malocclusion in patients, but not those who have dental restoration or occlusal interferences. Lack of improvement in patients whose restorations occurred highlighted the critical role of occlusal factors in the perpetuation of muscular dysfunction, and it justifies the importance of careful consideration and correction of occlusal discrepancies before administering any therapeutic intervention.

The results imply that plain lidocaine infiltration could be an easy, safe, and cost-effective adjunctive treatment in muscle-related occlusal disorders, especially in a low-resource clinical facility. More extensive electromyographically controlled and large-scale studies are, however, justified to prove these initial findings and to understand the specific neuromuscular mechanisms that are involved.



Author's Contributions

Khalidon Al-Buriah contributed to the conceptualization and methodological design of the study, supervised the research process, and was responsible for data collection and data review. Raef Ali Alkhulidi performed data curation and data analysis, conducted the literature review, and led the manuscript writing, revision, and editing. Both authors reviewed and approved the final version of the manuscript.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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