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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 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 significantly greater reduction in mean PPD (Test: 1.21 $\pm$ 0.28 mm vs. Control: 0.83 $\pm$ 0.31 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). The primary outcome in this study was the 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 the 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 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 54.7$\pm$ 8.2 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: 3.41$\pm$ 0.45 mm; Control: 3.38 $\pm$ 0.49 mm; p=0.68). At 12 weeks (T2), the mean PPD in the Test group was significantly lower than in the Control group (2.20 $\pm$ 0.31 mm vs. 2.55 $\pm$ 0.42 mm; $p < 0.001$). The mean PPD reduction from T0 to T2 was also significantly greater in the Test group (1.21 $\pm$ 0.28 mm) compared to the Control group (0.83 $\pm$ 0.31 mm; $p < 0.001$).

At baseline, the mean percentage of Bleeding on Probing (BOP)-positive sites was high and comparable between groups (Test: 81.0 ± 15.5 %; Control: 79.8 ± 16.1 %; 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 (9.9 ± 8.5% vs. 26.5 ± 14.2%; 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 (0.21 ± 0.18) compared to the Control group (0.45 ± 0.24; p < 0.001). Similarly, the mean mGI score at T2 was significantly lower for the BBG-treated sites (0.24 ± 0.19) compared to the glycine-treated sites (0.51 ± 0.26; p < 0.001).

Table 2. Clinical parameters over time.

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 35.8 ± 10.1 pg/mL, compared to 65.2 ± 15.7 pg/mL in the Control group (p < 0.001). Similarly, the TNF-α level at T2 was significantly lower in the Test group (22.4 ± 7.5 pg/mL) than in the Control group (41.3 ± 11.9 pg/mL; p < 0.001).

Table 3. PISF cytokine concentrations (pg/mL).

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 18.5 ± 7.1 for the Test group and 16.9 ± 6.8 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.[14,15]

The non-surgical management of peri-implant mucositis (PIM) represents a critical pillar in long-term implant maintenance, yet it is a field still contending with the limitations of conventional therapies. The fundamental goal of treatment is the complete disruption and removal of the pathogenic biofilm; however, most current modalities are "passive," relying on a purely mechanical action. Their efficacy ceases the moment the instrument is removed from the sulcus, leaving the site vulnerable to rapid bacterial recolonization. The present study was designed to challenge this paradigm by evaluating a novel "active" therapeutic agent—a bioactive borate glass (BBG) air-abrasion powder. This is the first randomized controlled clinical trial to investigate the in vivo efficacy of this material in a peri-implant environment. The principal finding of this study is unequivocal: while both the novel BBG powder and the standard glycine powder were safe, well-tolerated, and effective in reducing the clinical signs of PIM, the BBG powder demonstrated statistically and clinically significant superiority in all primary and secondary outcomes at the 12-week endpoint. Consequently, the null hypothesis—that no difference would exist between the two modalities—is definitively rejected.[16]

It is essential to first establish that the control intervention, glycine powder air-polishing, performed effectively and in accordance with the existing literature. The control group demonstrated significant ($p < 0.001$) improvements from baseline across all clinical metrics, including a 41.7% reduction in mean PPD and a 66.8% reduction in BOP. This is not surprising. Glycine-based powders, with their low abrasiveness and small particle size, are a well-established standard of care for safe and efficient biofilm removal from implant surfaces. The pathophysiological mechanism of glycine is direct and singular: mechanical debridement. By physically scouring the implant and abutment surfaces, it disrupts the organized biofilm structure, detaches bacterial colonies, and flushes the sulcus. This abrupt reduction in the local antigenic load (primarily

bacterial lipopolysaccharide, LPS) removes the primary stimulus for the host's inflammatory response. With the irritant removed, the host's innate healing mechanisms are permitted to initiate resolution, leading to decreased vasodilation, reduced epithelial ulceration, and improved tissue tone. This is reflected in the clinical improvements. However, the efficacy of this passive mechanism is inherently limited. The treatment addresses the result (the biofilm) but not the cause (the favorable micro-environment for dysbiosis). Once the glycine is rinsed away, its therapeutic action is complete. The peri-implant sulcus remains a nutrient-rich, anaerobic niche, and the race for recolonization by planktonic bacteria begins immediately. The clinical data from our control group reflect this limitation. Despite significant improvement, 26.5% of sites were still bleeding on probing at 12 weeks. This suggests that in more than a quarter of the sites, the mechanical debridement, coupled with the patient's oral hygiene, was insufficient to fully suppress the bacterial-inflammatory challenge below the clinical threshold of disease. This finding aligns with systematic reviews highlighting the challenge of achieving complete PIM resolution with mechanical debridement alone.[17,18]

The superior performance of the bioactive borate glass (BBG) powder can be attributed to its fundamentally different therapeutic design. The BBG powder functions as a dual-action agent. First, it performs as a mechanical debrider, with a particle size (25-45 µm) optimized for safe and effective biofilm removal, comparable to the glycine powder (~25 µm). The equivalent, low patient-reported discomfort scores (VAS 18.5 vs. 16.9) confirm that this mechanical phase is just as gentle and well-tolerated as the current gold standard. The profound difference, however, lies in the *bioactive* phase. Unlike the inert glycine particles, the BBG particles are designed to chemically react with the local environment. When the BBG powder comes into contact with the peri-implant sulcular fluid (PISF), its glass network—composed of B_2O_3, CaO, Na_2O, and P_2O_5 -begins a controlled dissolution. This process transforms the powder from a simple mechanical tool into a local, sustained-release drug delivery system. This bioactivity attacks the pathophysiology of PIM from two distinct angles: modulation of the microbial environment and modulation of the host inflammatory response.

The primary driver of PIM is a dysbiotic shift in the subgingival microbiome toward a community dominated by Gram-negative, anaerobic, and acid-tolerant pathogens. The metabolic activity of this pathogenic biofilm, particularly the fermentation of proteins and amino acids, creates a localized acidic micro-environment. This acidic pH is not merely a byproduct; it is an active virulence factor. It optimizes the function of pathogenic proteases (like gingipains from *Porphyromonas gingivalis*) that break down host tissues and is inhospitable to the commensal, health-associated species, thus perpetuating the dysbiotic state. The BBG powder directly dismantles this pathogenic ecosystem. The dissolution of the borate glass is a proton-consuming process. The glass releases sodium (Na^+) and calcium (Ca^{2+}) ions in exchange for hydronium ions (H_3O^+) from the PISF, leading to a rapid and sustained increase in local pH into the alkaline range (reportedly pH 9-11). This "alkaline shock" is a potent, non-specific antimicrobial weapon. It pushes the sulcular environment far outside the optimal metabolic range for key acidogenic peri-pathogens, inhibiting their enzymatic processes, disrupting membrane integrity, and severely limiting their ability to recolonize the freshly debrided surface. This sustained chemical alteration of the niche provides a clear explanation for the superior clinical data. At 12 weeks, the mean mPI score in the BBG group was 0.21, less than half that of the control group (0.45). This highly significant difference ($p < 0.001$) suggests that the BBG-treated sites remained inhospitable to bacterial accumulation long after the initial treatment.[19]

Furthermore, the boron ions (BO^{3-}_3) released during dissolution provide a second layer of antimicrobial action. Boron is not a passive element; it is known to possess intrinsic bacteriostatic properties. Emerging research suggests boron can interfere with bacterial quorum sensing (QS) pathways. QS is the sophisticated cell-to-cell communication system bacteria use to coordinate gene expression, allowing them to collectively initiate biofilm formation, produce

virulence factors, and defend against host attacks. By "jamming" these signals, the released boron ions may prevent planktonic survivors from reorganizing into a mature, pathogenic, and treatment-resistant biofilm. This dual-action antimicrobial strategy—(1) an immediate alkaline shock and (2) a sustained anti-QS effect—provides a far more comprehensive and durable assault on the pathogenic biofilm than the "hit-and-run" mechanical action of glycine.

The clinical signs of PIM—bleeding, redness, and pocket deepening—are not caused directly by the bacteria themselves. They are the physical manifestations of the host's own inflammatory response to the bacterial challenge. The core of PIM pathophysiology is a hyper-inflammatory state driven by a relentless feedback loop: bacterial LPS binds to host cells (like macrophages and epithelial cells), triggering the release of potent pro-inflammatory cytokines, which in turn cause the clinical signs of disease. To objectively quantify this process, our study measured the PISF concentrations of two of the most pivotal "master" cytokines in this cascade: Interleukin-1 Beta (IL-1β) and Tumor Necrosis Factor-Alpha (TNF-α). IL-1β is a key driver of bone and connective tissue breakdown, while TNF-α is a primary mediator of vasodilation, vascular permeability (edema), and the recruitment of other inflammatory cells. At baseline, both groups presented with high levels of these markers, confirming an active inflammatory state.

The 12-week biomarker data provide a stunning biological validation of the clinical findings. While both treatments successfully reduced inflammation, the magnitude of the reduction in the BBG group was profoundly greater. The mean IL-1β concentration in the BBG group (35.8 pg/mL) was nearly half that of the control group (65.2 pg/mL). Similarly, the TNF-α level in the BBG group (22.4 pg/mL) was dramatically lower than in the control group (41.3 pg/mL). These are not minor statistical differences; they represent a fundamentally different biological environment. This superior inflammatory resolution is a direct consequence of the superior microbiological control. By more effectively eliminating and suppressing the pathogenic biofilm (via the pH and boron mechanisms), the BBG treatment drastically reduced the source of the antigenic load (LPS). With less antigenic stimulation, the host's immune cells were no longer triggered to produce the high levels of IL-1β and TNF-α. This "silencing" of the inflammatory cascade is precisely what was observed clinically: (1) Resolution of BOP: The significant reduction in TNF-α leads to decreased vasodilation and restores the integrity of the sulcular epithelium. Without leaky, ulcerated blood vessels, the tissue no longer bleeds on gentle probing. This explains the 88.1% BOP resolution in the BBG group, compared to only 66.7% in the control group. The BBG-treated sites were, simply, less inflamed; (2) Reduction of PPD: The reduction in PPD (1.21 mm for BBG vs. 0.83 mm for control) is also a direct result of this inflammatory shutdown. The "pseudo-pocket" in PIM is composed of edematous, swollen tissue. The profound reduction in inflammatory cytokines (especially TNF-α) resolves this edema, allowing the gingival cuff to become more firm, dense, and "tight" around the implant abutment, resulting in a shallower, healthier sulcus.

An additional, compelling hypothesis is that the BBG may also be acting as a direct immunomodulatory agent, not just an indirect one. The ionic dissolution products of bioactive glasses (specifically Ca^{2+} and $BO^{3-}{}_3$ ions) have been shown in in vitro models to directly influence host cell behavior. They may promote a phenotypic shift in macrophages away from the pro-inflammatory M1 phenotype (which produces IL-1β and TNF-α) and toward the pro-resolving M2 phenotype, which secretes anti-inflammatory cytokines and growth factors. While our in vivo human study cannot prove this cellular-level mechanism, the exceptionally low levels of inflammatory markers in the BBG group suggest this is a plausible and exciting area for future investigation. The BBG may not only be removing the "on" switch (bacteria) but also actively promoting the "off" switch (host resolution).[20]

The findings of this trial are fortified by its robust methodological design. The split-mouth model is a powerful tool in clinical research, as it eliminates the vast majority of inter-subject variability; each patient serves as their own perfect control. This ensures that differences in host genetics, immune response, diet,

and systemic health cannot be confounding factors. The observed differences between the test and control sites can therefore be attributed with a very high degree of confidence to the interventions themselves. This strength is compounded by the use of a single, calibrated, and blinded examiner for all clinical outcome assessments, which minimized measurement bias. Finally, the inclusion of objective, quantitative biomarkers (PISF cytokines) alongside traditional clinical indices provides a multi-layered and biologically sound validation of the clinical results.

4. Conclusion

Our study provides the first comprehensive clinical and biological evidence that a novel bioactive borate glass air-abrasion powder is significantly more effective than the standard glycine powder for the non-surgical treatment of peri-implant mucositis. Its superiority is not marginal; it is a statistically significant and clinically meaningful improvement across all measured outcomes. This is not simply a better mechanical debrider. It is a new class of "bioactive" therapeutic. By chemically re-engineering the sulcular micro-environment to be inhospitable to pathogens and by profoundly attenuating the host's inflammatory cascade, this technology represents a true paradigm shift. It moves the goal of peri-implant maintenance from transient mechanical cleaning to active, sustained, and guided ecological and inflammatory resolution. 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

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