Keywords
Camel milk whey protein; periodontitis; professional mechanical plaque debridement; clinical attachment level; probing pocket depth; gingival index; local drug delivery; non-surgical periodontal therapy.

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# Clinical Effectiveness of Locally Delivered Camel Milk Whey Protein Gel as an Adjunct to Mechanical Plaque Debridement in Patients with Stage II Grade B Periodontitis: A Randomized Clinical Trial

**Abstract**

**Objectives:** The purpose of this study was to assess the clinical efficacy of locally administered CMWP gel as a supplement to PMPD in individuals with Stage II Grade B periodontitis.

**Methods:** Thirty patients with Stage II Grade B periodontitis who were systemically healthy and between the ages of 18 and 40 were divided into two parallel groups at random (n = 15 each). Group II received PMPD followed by intra-pocket administration of CMWP gel, while Group I received PMPD followed by intra-pocket application of placebo gel. For four weeks in a row, the gel was applied once a week. Gingival index (GI), clinical attachment level (CAL), and probing pocket depth (PPD) were measured at baseline, three months, and six months. The main clinical comparisons were carried out across follow-up periods and between groups.

**Results:** Over the course of the six months, both groups showed statistically significant improvements in every clinical measure that was assessed. Group I and Group II had similar baseline CAL (3.67 ± 0.36 mm vs. 3.60 ± 0.34 mm; $p = 0.606$). Group II's CAL was considerably lower than Group I's at three months (2.43 ± 0.37 mm vs. 2.87 ± 0.35 mm; $p = 0.003$), and this difference grew at six months (2.00 ± 0.33 mm vs. 2.63 ± 0.44 mm; $p < 0.001$). With CMWP, the overall CAL increase was substantially higher (1.60 ± 0.21 mm vs. 1.03 ± 0.44 mm; $p < 0.001$). Additionally, Group II's PPD reduction was significantly higher than Group I's, with a mean reduction of 1.43 ± 0.33 mm versus 0.94 ± 0.41 mm ($p = 0.001$).

At six months, Group II's mean PPD (2.00 ± 0.28 mm) was considerably lower than Group I's (2.31 ± 0.34 mm; $p = 0.011$). In both groups, GI dramatically dropped. At three months, Group II's GI was considerably lower (0.60 ± 0.23 vs. 0.80 ± 0.25; $p = 0.031$), but at six months, the difference was not statistically significant (0.53 ± 0.27 vs. 0.62 ± 0.16; $p = 0.306$).

**Conclusion:** Within the constraints of this trial, patients with Stage II Grade B periodontitis who received locally applied CMWP gel in addition to PMPD showed higher clinical attachment gain and a reduction in probing pocket depth than those who received PMPD alone. Additionally, CMWP seemed to hasten the initial decrease in gingival inflammation. According to these results, CMWP might be a useful supplementary local treatment for periodontal disease that doesn't require surgery. To verify its therapeutic effectiveness and elucidate its underlying mechanisms, larger clinical trials with longer follow-up and microbiological and biochemical evaluations are necessary.

## INTRODUCTION

The multifactorial periodontal inflammation is commonly characterized by ongoing destructive alveolar bone degeneration, gingival bleeding, and deep pocket. Periodontal destruction is triggered by microbial dysbiosis [1]. Reducing inflammation in periodontitis could arrest the progression of periodontal destruction [2]. Non-surgical professional mechanical plaque debridement (PMPD) was found to be a beneficial intervention for grade B periodontitis owing to the anti-infective characteristics of supra- and subgingival mechanical removal of biofilm from the tooth surface [3]. Failure to effectively eliminate periodontal pathogens from deeper periodontal pockets following PMPD was found to be responsible for the persistence of a chronic inflammatory response owing to the failure to convert the dysbiotic subgingival biofilm causing periodontitis to a symbiotic one [4]. Co-adjuvant approach utilizing antibacterial therapy, anti-inflammatory drugs, anti-oxidants, photodynamic therapy, and controlled sub-gingival delivery system combined with PMPD was developed for maintaining optimal intra-pocket concentrations of drug therapy for adequate duration to suppress periodontitis, and favoring fibroblasts reattachment, inhibiting collagenase activity, and promote periodontal regeneration [5].

Camel milk whey protein (CMWP) demonstrated a significant antioxidative impact attributed to the reduction of the oxidative stress and the improved immune system performance. Dietary supplements of CMWP were proven to have antibacterial, anti-inflammatory, anticancer, antiviral, antioxidative properties and boost innate and adaptive immunity [6]. It was verified that CMWP enhanced the diabetic patients` wound healing [7]. Supplementary CMWP reported reduced inflammatory biomarkers, proinflammatory cytokines. Moreover, CMWP supplements was found to modulate the immune activity, and free radicals [8]. In the light of the above-mentioned properties, CMWP could play a potential role in treating periodontitis. To the best of our knowledge, topical application of CMWP as an adjunct to PMPD was not yet assessed on the treatment of periodontitis.

Hence, the present randomized controlled study aims to assess the efficacy of locally delivered topical application of CMWP gel as an adjunct to PMPR patients with Stage II Grade B periodontitis. The null hypothesis formulated was that there would be no clinical differences between the PMPR + CMWP gel and the PMPR groups.

## MATERIALS AND METHODS

The current 2 parallel groups randomized clinical trial include 30 patients diagnosed as Stage II Grade B periodontitis with an age of 18–40 years. The participants were recruited from the periodontics clinic after obtaining the Ethical approval from the institutional ethics committee. All recruited subjects provided a written consent.

The inclusion criteria included systemically healthy patients diagnosed as Stage II Grade B periodontitis with age of 18–40 years, and probing pocket depth (PPD) of ≤ 5 mm.

The exclusion criteria included a history of systemic disease, pregnant or lactating females, undergoing any periodontal therapy or those who used antibiotic, anti-inflammatory drugs, or antioxidants in the past 6 months, allergic patients to the used materials, smokers, and alcoholics.

G Power software was used to estimate the sample size. Based on power of 80%–90%, an effect size of 1.11, and an alpha of 0.05, a total sample size of 30 patients (15 each group) would be adequate. [9]

The patients were randomly allocated by computerized software into two groups: Group A received PMPD with placebo gel (n = 15), and Group B received PMPD with CMWP gel (n = 15). The participants, the principal investigator, the outcome assessors as well as the bio statistician were blinded.

### Preparation of CMWP:

After obtaining the raw milk from healthy, national female camels, cream removal was performed by milk centrifuging. For 48 hours, dialyzing of the precipitated CMWP against distilled water was performed. Freeze-drying of the undenatured dialyzed CMWP was carried out.

In order to make the CMWP gel, sodium citrate was added to distilled water and continuously stirred until a clear solution was obtained. Methylcellulose polymer was then added to the prepared solution and left to hydrate overnight. Lastly, mix constantly while you add roughly 1% (w/v) of CMWP to the polymer solution.

### Pre-surgical therapy

A thorough periodontal examination was used to identify study-eligible patients, and complete periodontal charts were acquired. In order to diagnose patients with periodontitis, periapical radiographs were obtained at the areas of attachment loss. To guaranty consistent baseline circumstances for assessing the intervention's effectiveness, all patients underwent phase 1 periodontal therapy following a thorough examination and diagnosis before the experimental protocol began. After six weeks, a reevaluation was carried out, and only patients who satisfied all inclusion requirements and showed sufficient plaque control moved on to the experimental phase. All patients got oral hygiene instruction as part of the therapy, which also included PMPD conducted over two weeks utilizing ultrasonic and/or hand devices.

The clinical parameters for all participants including clinical attachment level (CAL) [10], probing pocket depth (PPD) [11], and gingival index (GI) [12] were recorded and scored preoperatively.

### Intervention:

Group I sites received PMPD followed by intra-pocket application of placebo gel, while Group II sites received PMPD followed by intra-pocket administration of CMWP gel.

Using cotton rolls to isolate the region, a blunt-needle syringe was used to apply the gel. While the gel was being applied, the needle tip was positioned close to the bottom of the clinical pocket and gradually removed. To avoid any spillover effects and potential systemic effects from eating the gel, the pockets were overfilled, and extra gel

was carefully removed with cotton pellets. For four weeks, one weekly application of gel was made.

**Postoperative care**

Patients were instructed not to eat anything sticky or chewy. No systemic antibiotics, anti-inflammatory drugs, or antiplaque medicines were administered to the patient. Additionally, in the two days after the procedure, patients were instructed not to use harsh dental cleaning methods that could interfere with the gel's application, such as rigorous brushing or flossing, in the regions where the gel was placed. Three and six months following the procedure, follow-up appointments were planned. Ultrasonic scalers were used to perform supragingival PMPD during these follow-up consultations. The clinical results were quantified and documented.

**Evaluation criteria**

**Clinical evaluation**

Every clinical parameter was measured at baseline, three, and six months using a UNC 15 periodontal probe. Each patient's teeth with PPD more than 3 mm were assessed at six places (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual, and distolingual) and averaged. The evaluating unit was the patient. PPD was measured in millimeters from the gingival edge to the base of the periodontal pocket, and CAL was calculated as the distance in millimeters between the gingival sulcus bottom and the cemento-enamel junction (CEJ). GI was scored at 4 sites of each affected tooth from 0 to 3, 0 represent no inflammation, 1 represent mild inflammation, 2 for moderate inflammation and 4 for sever inflammation. The mean scores of the whole examined sites represent GI index for the patient.

Two seasoned investigators who were blinded collected all parameters. In order to make sure that the inter-examiner agreement for clinical and radiographic measurements was greater than 0.85, calibration was done before to the start of the investigation by comparing two measurements taken by the two investigators on the same subjects (who were not involved in the study) one week apart.

**Statistical Analysis**

The statistical study was performed using IBM SPSS Statistics for Windows, Version 23.0. IBM Corp., Armonk, New York. The mean ± standard deviation (SD) was used to express continuous variables. The Shapiro-Wilk test was used to determine whether the data distribution was normal. Repeated-measures analysis of variance (RM-ANOVA) was used to assess changes over time within each treatment group because the clinical parameters were measured in the same participants at baseline, three months, and six months. Paired comparisons between individual time points were then performed as needed. When the parametric analytic assumptions were met, independent-samples t-tests were employed for between-group comparisons at each follow-up time point.

The robustness of the results was further confirmed using the corresponding non-parametric tests, such as the Mann–Whitney U test for between-group comparisons and the Wilcoxon signed-rank test for within-group comparisons, because some baseline measurements showed slight deviations from normality. For every clinical measure, the total change from baseline to six months was computed and compared between groups. The patient served as the statistical unit of analysis for all analyzes. Statistical significance was defined as a two-sided p-value of less than 0.05.

**RESULTS**

**Patient characteristics**

**Baseline Demographic Characteristics**

Thirty people in all completed the 6-month evaluation period with no dropouts or adverse events reported, equally split into Group I (n = 15) and Group II (n = 15), participated in the study. Group I's mean age, which ranged from 19 to 40 years, was 31.33±6.84 years. Group II's mean age, which ranged from 18 to 40 years, was 31.93±6.80 years. Age did not differ statistically significantly between the two groups (p = 0.811).

Group I had 11 males (73.3%) and 4 females (26.7%) in terms of gender distribution, whereas Group II had 10 males (66.7%) and 5 females (33.3%). There was no statistically significant difference in the gender distribution between the two groups (p = 1). These findings verify that the two groups were homogeneous and well-matched in terms of age and gender at baseline.

**Clinical outcomes**

**Clinical Attachment Loss (CAL)**

The two groups' initial Clinical Attachment Loss (CAL) assessments did not differ statistically significantly, indicating baseline homogeneity. The mean CAL at baseline was 3.67±0.36mm for Group I and 3.60±0.34mm for Group II; there was no statistically significant difference (p = 0.606). At the 3-month mark, Group II's mean CAL (2.43±0.37mm) was substantially smaller than Group I's (2.87±0.35mm), reaching statistical significance (p = 0.003). At six months, Group II's mean CAL was 2.00±0.33mm, while Group I's was 2.63±0.44mm (p < 0.001). This intergroup disparity increased even more. Net clinical attachment gain (CAL decrease) was considerably greater in Group II (1.60±0.21mm) than in Group I (1.03±0.44mm; p < 0.001) during the course of the 6-month research period. [Page 2]

Time had a highly significant impact on CAL reduction in both Group I (F (2, 28) = 66.48, p < 0.001) and Group II (F (2, 28) = 256.30, p < 0.001), according to Repeated Measures Analysis of Variance (RM-ANOVA). Group I: From baseline (3.67±0.36mm) to three months later (2.87±0.35mm; p < 0.001), CAL considerably decreased. From three to six months, there was a continuous significant decrease (2.63±0.44mm; p = 0.003). There was a significant overall improvement from baseline to six months (p < 0.001). Group II: From baseline (3.60±0.34mm) to three months (2.43±0.37mm; p < 0.001), CAL considerably dropped. Between three and six months, there was a further substantial decrease (2.00±0.33mm; p < 0.001). The overall change from baseline to six months was statistically significant (p < 0.001). [Table 3]

**Probing Pocket Depth (PPD)**

Due to minor departures from normality at baseline, the analysis was confirmed using non-parametric counterparts (Mann-Whitney U and Wilcoxon) and carried out using parametric tests (Independent and Paired t-tests, Repeated Measures ANOVA). In millimeters (mm), the data is displayed as Mean ± Standard Deviation (SD).

Group II's mean Probing Pocket Depth (PPD) at baseline was 3.43 ± 0.19 mm, marginally but substantially greater than Group I's 3.26 ± 0.23 mm ($p = 0.029$).

Both groups showed a significant decrease in pocket depths after the therapies. At the 3-month follow-up, Group I's mean PPD was 2.41 ± 0.31 mm, whereas Group II's was 2.24 ± 0.29 mm ($p = 0.145$), indicating that the difference between the two groups was no longer statistically significant. However, Group II showed noticeably shorter pocket depths than Group I at the 6-month evaluation (2.00 ± 0.28 mm vs. 2.31 ± 0.34 mm, respectively; $p = 0.011$). Group II obtained a substantially higher overall PPD reduction (1.43 ± 0.33 mm) than Group I (0.94 ± 0.41 mm) when assessing the entire clinical improvement from baseline to 6 months, resulting in a highly significant intergroup difference ($p = 0.001$). [Table 4]

Both Group I ($F = 71.04$, $p < 0.001$) and Group II ($F = 174.23$, $p < 0.001$) showed a highly significant influence of time on PPD reduction, according to Repeated Measures Analysis of Variance (RM-ANOVA). Group I: PPD was considerably lower at the 3-month evaluation (2.41 ± 0.31 mm; $p < 0.001$) compared to baseline (3.26 ± 0.23 mm). Between the 3-month and 6-month intervals, a persistent, statistically significant decrease was noted (2.31 ± 0.34 mm; $p = 0.023$). From baseline to six months, there was a significant decrease in the overall pocket depth ($p < 0.001$). Group II: PPD was significantly lower at the 3-month evaluation (2.24 ± 0.29 mm; $p < 0.001$) than it was at baseline (3.43 ± 0.19 mm). Between three and six months, the pocket depth significantly decreased as healing progressed (2.00 ± 0.28 mm; $p = 0.006$). There was a substantial decrease in pocket depth overall between baseline and six months ($p < 0.001$). [Table 5]

**Gingival Index (GI)**

Group I's mean Gingival Index (GI) score at baseline was 1.58±0.23, whereas Group II's was 1.62±0.27. Baseline homogeneity between the study groups was demonstrated by the independent sample t-test, which indicated no statistically significant difference between the two cohorts at baseline ($t = -0.371$, $p = 0.713$). Different temporal dynamics were found when comparing clinical scores across the two treatments groups: [Table 6, Figure 1]

1. 3-Month Follow-up: Group II showed a statistically significant lower mean GI score (0.60±0.23) than Group I (0.80±0.25), suggesting a quicker initial decrease in soft tissue inflammation ($t = 2.274$, $p = 0.031$).
2. 6-Month Follow-up: There was no statistically significant inter-group variation ($t = 1.042$, $p = 0.306$) at the final evaluation interval, as the difference between Group I (0.62±0.16) and Group II (0.53±0.27) diminished.

Over the course of the six-month observation period, both Group I ($F = 135.2$, $p < 0.001$) and Group II ($F = 128.6$, $p < 0.001$) showed a statistically significant time-dependent decrease in GI scores, according to Repeated Measures Analysis of Variance (RM-ANOVA). Group I: The mean GI scores decreased statistically significantly from baseline (1.58±0.23) to three months (0.80±0.25; mean difference = 0.78±0.27, $t = 11.45$, $p < 0.001$), according to post-hoc pairwise analysis using paired t-tests. Between three and six months, there was a substantial further decrease (0.62±0.16; = 0.18±0.18, $t = 4.04$, $p = 0.001$). Group I achieved an overall mean reduction of 0.97±0.22 ($t = 16.82$, $p < 0.001$) for the whole research period (baseline to 6 months). Group II: From baseline (1.62±0.27) to three months (0.60±0.23; = 1.02±0.27, $t = 14.32$, $p < 0.001$), pairwise analysis showed a substantial early decrease in mean GI scores. There was a slight numerical decrease (= 0.07±0.15) between 3 and 6 months (0.53±0.27), although it was not statistically significant ($t = 1.74$, $p = 0.104$). However, there was a substantial overall decrease from baseline to six months (= 1.08±0.28, $t = 15.11$, $p < 0.001$). [Table 7]

## DISCUSSION

PMPD is a successful therapeutic option for periodontal disease pocket reduction [13]. However, the periodontal pockets' reaction to PMPD may not be enough in some difficult clinical circumstances [14]. To improve the PMPD's results, a variety of local adjunctive agents were promoted [15]. Compared to PMPD alone, local subgingival administration of satranidazole gel decreased PPD in patients with periodontitis. In individuals with periodontitis, doxycycline gel, chlorhexidine gel, and tetracycline fiber improved clinical outcomes over a three-month period [16]. PPD and CAL were improved when metformin or statins were administered locally to PMPD as opposed to PMPD alone [17]. Compared to PMPD with saline as a placebo, adjunctive PRF to PMPD improved periodontal metrics in individuals with periodontitis [18]. Additionally, compared to PMPD alone, the addition of ozone gel to PMPD significantly reduced PPD, plaque and bleeding indices, and CAL-gain in patients with periodontitis [19]. Patients with periodontitis showed a considerable improvement in periodontal parameters when melatonin, resveratrol, omega-3 fatty acids, cranberry juice, propolis, and aloe vera gel were added to periodontal therapy [20].

There has never been an evaluation of CMWP as a local supplement to PMPD in patients with periodontitis. Therefore, in patients with Stage II Grade B periodontitis, the current randomized controlled clinical trial assessed the clinical efficacy of locally administered CMWP gel as an adjunct to PMPD, specifically on clinical attachment level (CAL) (primary outcome), probing pocket depth (PPD), and gingival index (GI) (secondary outcomes).

The current study assessed recurrent subgingival implantation of CMWP gel, in contrast to a number of earlier investigations that primarily used commercially available formulations or single application delivery procedures. This protocol was created to improve therapeutic persistence and local bioavailability in periodontal pockets, adding to the body of knowledge about protocol-dependent variability in adjunctive periodontal therapy.

All assessed clinical periodontal indicators, including as CAL, PPD, and GI, showed notable improvements in both

therapy groups. However, the effect on gingival inflammation was most noticeable during the early healing phase, and the adjunctive CMWP group generally showed larger clinical benefits, especially with regard to CAL gain and PPD reduction.

There were no statistically significant differences between the two groups at baseline in terms of CAL and GI. This baseline homogeneity is significant because it suggests that significant variations in the initial degree of periodontal inflammation or attachment loss were unlikely to be responsible for the ensuing variations in clinical outcomes. While there was a statistically significant baseline difference for PPD, Group II's mean pocket depth was somewhat higher than Group I's, but the absolute difference was not that substantial. More significantly, even though Group II began with slightly deeper pockets, they later showed a bigger reduction in PPD, indicating that the supplementary CMWP intervention had a positive impact. Over the course of the six-month follow-up period, both groups showed a notable improvement in CAL. PMPD was successful in enhancing periodontal attachment during the observation period, as evidenced by the extremely significant effect of time in both groups.

However, the CMWP group's CAL improvement was noticeably larger. Group II's CAL was far lower than Group I's at three months, and by six months, the difference was even more noticeable. Additionally, Group II's overall CAL increase was noticeably higher. These results imply that CMWP offered a therapeutic benefit that went beyond simple mechanical plaque removal.

The documented biological actions of CMWP, specifically its anti-inflammatory and antioxidant qualities, may be linked to the higher attachment gain. [21] In addition to microbial assault, a dysregulated host inflammatory response and increased reactive oxygen species generation also contribute to the death of periodontal tissue. [22] Therefore, by lowering the oxidative and inflammatory burden in the periodontal pocket, a locally administered CMWP formulation may aid in periodontal repair. Additionally, keeping the material inside the periodontal pocket may improve its biological action during the healing process by increasing its local exposure. [23]

It is especially interesting that Group II's CAL continued to improve between three and six months. The CMWP group showed an additional statistically significant decrease in CAL instead of plateauing at the early follow-up. This gradual improvement could be a sign of ongoing periodontal tissue healing and inflammation resolution after supplementary CMWP administration. However, clinical attachment gain evaluated with a periodontal probe does not confirm actual regeneration of the periodontal attachment apparatus on its own; rather, it should be taken solely as a clinical sign of periodontal healing.

Overall, PPD measurements revealed a similar pattern. Pocket depth significantly decreased in both groups between baseline and three and six months, demonstrating the efficacy of PMPD in lowering periodontal pocketing. Group II showed a significantly lower PPD at six months, despite the fact that the difference between the groups at three months was not statistically significant. More notably, the CMWP group's overall PPD reduction was substantially higher than that of the control group. This result suggests that over time, the adjunctive impact of CMWP got more noticeable.

The elimination and disruption of the subgingival biofilm, the resolution of gingival inflammation, the decrease in tissue edema, and the subsequent contraction and repair of the periodontal tissues are all responsible for the decrease in PPD that occurs after PMPD. [24] The larger decrease seen with CMWP might be the result of further adjustments to the inflammatory milieu in the periodontal pocket.

Because CMWP has been shown to have antibacterial, antioxidant, and anti-inflammatory properties, its local administration may enhance mechanical biofilm clearance by lessening the persistence of inflammatory stimuli following debridement. [25]

It's also crucial to remember that Group II had much larger wallets at the beginning than Group I. Despite this early setback, Group II reduced PPD considerably more overall and had a lower mean PPD at six months. This supports the idea that the observed difference might be related to the adjunctive CMWP treatment instead of just being a result of better baseline pocket measurements. However, as the baseline PPD values differed statistically, this variable should preferably be taken into account in the statistical analysis or taken into account in a multivariable model in subsequent research.

Over the course of the trial, both groups showed significant and statistically significant decreases in GI, suggesting that periodontal therapy significantly reduced gingival inflammation. PMPD was successful in lowering gingival inflammation when combined with appropriate plaque-control strategies, as seen by the considerable time impact seen in both groups.

It's interesting to note that at three months, there was the most intergroup disparity. Group II's GI score was much lower than Group I's, indicating that adjunctive CMWP might have hastened the gingival inflammation's early remission. Given the suggested anti-inflammatory and antioxidant properties of CMWP, this conclusion is biologically feasible. [26] At six months, however, there was no longer a statistically significant difference between the groups.

Therefore, both treatment procedures ultimately produced significant and comparable improvement by the 6-month evaluation, even though CMWP seemed to offer an early edge in lowering gingival inflammation. Despite the notable variations in CAL and PPD, there was no significant intergroup difference in GI at six months. Effective plaque control and periodontal debridement can have a quick influence on gingival inflammation, a clinical criterion that is relatively sensitive. [27] As a result, the disparity between the two groups may have gradually decreased due to the control group's significant improvement. CAL and PPD, on the other hand, may be more helpful in identifying long-term variations between adjunctive therapy regimens since they represent deeper periodontal tissue alterations.

Nonetheless, CMWP's early superiority in GI reduction may support its possible involvement in hastening the clearance of periodontal inflammation. The absence of a significant difference at six months should not be taken as

proof that CMWP has no anti-inflammatory effect; rather, it might suggest that both groups' gingival inflammation significantly improved over time with conventional PMPD and appropriate plaque control. [28]

When combined, the clinical results show that both treatment regimens were successful in enhancing periodontal health for the course of the 6-month observation period. Adjunctive local CMWP application offered further advantages, especially in terms of CAL gain and PPD decrease. Early in the healing process, the impact on GI seemed to be more noticeable, and by the time of the 6-month evaluation, it had subsided.

The results are explained by a complimentary mechanism whereby locally administered CMWP may alter the host's oxidative and inflammatory milieu, whereas PMPD mainly lowers the microbial load. [29, 30] Compared to mechanical debridement alone, such a combination treatment may promote more favorable periodontal repair. Crucially, rather than serving as a substitute for traditional mechanical periodontal therapy, the current results suggest the potential clinical value of CMWP as an additional local therapy.

However, while interpreting these findings, a number of factors should be taken into account. The study featured a six-month follow-up period and a rather small sample size of thirty participants. Additionally, the clinical measurements were based on periodontal probing and lacked biochemical, inflammatory, or microbiological evaluations that may directly show the mechanisms behind the clinical effects that were seen. Therefore, rather than being mechanisms that the current study actually demonstrates, the suggested antioxidant, anti-inflammatory, or antibacterial mechanisms continue to be physiologically plausible explanations. To confirm the clinical benefits and elucidate the mechanisms of action of locally delivered CMWP, larger-scale randomized clinical trials with longer follow-up periods and evaluation of subgingival microbiota, inflammatory cytokines, oxidative stress markers, and other biological parameters are necessary.

All things considered, the current findings offer initial clinical proof that topical intra-pocket application of CMWP gel as a supplement to PMPD may improve periodontal clinical outcomes in patients with Stage II Grade B periodontitis, especially in terms of clinical attachment gain and periodontal pocket reduction. Therefore, the results suggest more research on CMWP as a locally administered supplementary periodontal treatment drug.

## CONCLUSIONS

Over the course of the 6-month follow-up period, both PMPD alone and PMPD in combination with locally applied CMWP gel significantly improved clinical periodontal metrics, within the constraints of the current randomized controlled clinical experiment. However, when compared to PMPD alone, the supplementary administration of CMWP gel produced noticeably higher clinical attachment gain and probing pocket depth reduction. Additionally, at three months, CMWP considerably reduced gingival inflammation; however, at six months, this intergroup difference was no longer statistically significant. These results imply that in individuals with Stage II Grade B periodontitis, locally administered CMWP gel may offer an extra clinical benefit when used in conjunction to traditional non-surgical periodontal therapy. To validate these results and clarify the underlying mechanisms of action, further carefully planned clinical trials with bigger sample sizes, longer follow-up times, and microbiological and biochemical evaluations are necessary.

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**Authors' contribution:**

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**Declarations**

**Ethical approval:** The present study has been approved by the Research Ethics Committee of Kafr Elsheikh University, Egypt.

**Consent to participate:** The research procedures were thoroughly explained to all patients, who consented to participate in the study and signed the required informed consent form before the commencement of the study.

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**Table 1. Baseline demographic characteristics of the study participants.**

| Characteristic | Group I (n=15) | Group II (n=15) | p-value | Significance |
|---|---|---|---|---|
| Age (years) | | | | |
| Mean±SD | 31.33±6.84 | 31.93±6.80 | 0.811a | Not Significant |
| Range (Min – Max) | 19 – 40 | 18 – 40 | | |
| Gender, n (%) | | | | |
| Male | 11 (73.3%) | 10 (66.7%) | 1.000b | Not Significant |
| Female | 4 (26.7%) | 5 (33.3%) | | |

SD = Standard Deviation. n = number of participants.
[a] Calculated using Independent Samples t-test.
[b] Calculated using Fisher's Exact test.

**Table 2. Intergroup comparison of Clinical Attachment Loss (CAL) in mm at baseline, 3 months, and 6 months (Mean ± SD).**

| Follow-up Period | Group I (n=15) | Group II (n=15) | Mean Difference | t-value | p-value |
|---|---|---|---|---|---|
| Baseline | 3.67±0.36 | 3.60±0.34 | 0.07 | 0.521 | 0.606 NS |
| 3 Months | 2.87±0.35 | 2.43±0.37 | 0.44 | 3.279 | 0.003* |
| 6 Months | 2.63±0.44 | 2.00±0.33 | 0.63 | 4.461 | < 0.001** |
| Total Gain (Base – 6M) | 1.03±0.44 | 1.60±0.21 | -0.57 | -4.498 | < 0.001** |

NS: Not Significant ($p > 0.05$); * Statistically Significant ($p < 0.05$); ** Highly Statistically Significant ($p < 0.001$). Values compared via Independent Samples t-test.

**Table 3. Pairwise intragroup comparison of CAL gain across time intervals**

| Comparison Interval | Group I (p-value) | Group II (p-value) |
|---|---|---|
| Baseline vs. 3 Months | < 0.001** | < 0.001** |
| 3 Months vs. 6 Months | 0.003* | < 0.001** |
| Baseline vs. 6 Months | < 0.001** | < 0.001** |

* Statistically Significant ($p < 0.05$); ** Highly Statistically Significant ($p < 0.001$). Pairwise comparisons analyzed via paired t-test / Wilcoxon signed-rank test.

**Table 4. Intergroup comparison of Probing Pocket Depth (PPD) in mm at baseline, 3 months, and 6 months (Mean ± SD).**

| Follow-up Period | Group I (n = 15) | Group II (n = 15) | Mean Difference | p-value |
|---|---|---|---|---|
| Baseline | 3.26 ± 0.23 | 3.43 ± 0.19 | -0.17 | 0.029* |
| 3 Months | 2.41 ± 0.31 | 2.24 ± 0.29 | 0.17 | 0.145 (NS) |
| 6 Months | 2.31 ± 0.34 | 2.00 ± 0.28 | 0.31 | 0.011* |
| Total Reduction (Base – 6M) | 0.94 ± 0.41 | 1.43 ± 0.33 | -0.49 | 0.001** |

NS: Not Significant ($p > 0.05$); * Statistically Significant ($p < 0.05$); Highly Statistically Significant ($p \leq 0.001$). Values compared via Independent Samples t-test / Mann-Whitney U test.

**Table 5. Pairwise intragroup comparison of PPD reduction across time intervals.**

| Comparison Interval | Group I (p-value) | Group II (p-value) |
|---|---|---|
| Baseline vs. 3 Months | < 0.001** | < 0.001** |
| 3 Months vs. 6 Months | 0.023* | 0.006* |
| Baseline vs. 6 Months | < 0.001** | < 0.001** |

* Statistically Significant ($p < 0.05$); Highly Statistically Significant ($p \leq 0.001$). Pairwise comparisons analyzed via Paired t-test.

**Table 6: Inter-Group Comparison of Gingival Index (GI) between Group I and Group II at each follow-up interval.**

| Follow-up Interval | Group I (n=15) | Group II (n=15) | Inter-Group Comparison |
|---|---|---|---|
| | Mean ± SD | Mean ± SD | p -value |
| Baseline | 1.58 ± 0.23 | 1.62 ± 0.27 | 0.713 (NS) |
| 3 Months | 0.80 ± 0.25 | 0.60 ± 0.23 | 0.031 * |
| 6 Months | 0.62 ± 0.16 | 0.53 ± 0.27 | 0.306 (NS) |

* Statistically significant at $p < 0.05$ (Independent t-test / Mann-Whitney U test).

**Table 7: Intra-Group Comparison (Longitudinal Changes) of Gingival Index (GI) alterations within each group over the 6-month study period.**

| Group | Comparison Interval | Mean Difference (Δ) | t-value | p-value |
|---|---|---|---|---|
| Group I | Baseline to 3 Months | 0.78 ± 0.27 | 11.45 | < 0.001 * |
| | 3 Months to 6 Months | 0.18 ± 0.18 | 4.04 | 0.001 * |
| | Baseline to 6 Months | 0.97 ± 0.22 | 16.82 | < 0.001 * |
| | | | | |
| Group II | Baseline to 3 Months | 1.02 ± 0.27 | 14.32 | < 0.001 * |
| | 3 Months to 6 Months | 0.07 ± 0.15 | 1.74 | 0.104 (NS) |
| | Baseline to 6 Months | 1.08 ± 0.28 | 15.11 | < 0.001 * |

NS: Not Significant.
* Statistically significant at $p < 0.05$ (Paired t-test / Wilcoxon Signed-Rank Test).