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*Research Article*

# Does Adjunctive Homecare Sub-Gingival Irrigation Improve the Systemic Inflammatory–Glycaemic Axis? A Randomized Controlled Analysis of C-Reactive Protein and Glycated Haemoglobin in Type 2 Diabetic Patients with Chronic Periodontitis

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## Abstract

**Background:** Type 2 diabetes mellitus (T2DM) and chronic periodontitis are linked by a shared inflammatory pathophysiology in which the systemic dissemination of periodontal pro-inflammatory mediators elevates the acute-phase response and aggravates insulin resistance. C-reactive protein (CRP) is a sensitive marker of this systemic inflammatory burden, whereas glycated haemoglobin (HbA1c) integrates glycaemic control over the preceding two to three months. Whether adjunctive homecare sub-gingival water irrigation, added to non-surgical periodontal therapy (NSPT), produces measurable benefit along this systemic inflammatory–glycaemic axis, over and above conventional oral hygiene, remains poorly defined.

**Objective:** To determine whether adjunctive homecare sub-gingival irrigation, compared with conventional brushing and flossing after NSPT, produces a differential effect on the systemic inflammatory marker CRP and on glycaemic control (HbA1c) in adults with T2DM and chronic periodontitis, and to examine whether the systemic response tracks the resolution of periodontal inflammation.

**Methods:** A single-blind, parallel-group randomized controlled trial enrolled 140 patients with T2DM (baseline HbA1c below 7%) and generalized chronic periodontitis at the Department of Periodontology and Oral Implantology, Yamuna Institute of Dental Sciences and Research, Yamuna Nagar. After Phase I therapy, participants were randomized equally to homecare sub-gingival irrigation plus brushing (Group 1) or brushing and flossing alone (Group 2). Serum CRP (latex agglutination) and HbA1c (liquid chromatography) were measured at baseline and six months;

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periodontal inflammatory parameters were recorded in parallel. Between-group differences were analysed with independent-sample t-tests and within-group change with paired testing, significance at $p<0.05$.

**Results:** CRP fell substantially in both groups but significantly more with irrigation, from 17.06±2.42 mg/L to 6.96±1.21 mg/L in Group 1 versus 18.27±2.38 mg/L to 10.17±1.90 mg/L in Group 2; the six-month value ($p<0.001$) and the magnitude of reduction (9.10 vs 8.10 mg/L, $p=0.048$) both favoured Group 1. HbA1c declined modestly and almost identically in both arms (Group 1: 6.69% to 6.44%; Group 2: 6.76% to 6.52%), with no significant inter-group difference at six months ($p=0.192$) or in magnitude of change ($p=0.182$). The greater systemic anti-inflammatory response in Group 1 paralleled its greater resolution of periodontal inflammation, whereas the glycaemic response did not diverge between arms.

**Conclusion:** Adjunctive homecare sub-gingival irrigation produced a significantly greater reduction in systemic inflammatory burden (CRP) than conventional hygiene, mirroring superior periodontal inflammatory control, but did not confer an additional glycaemic benefit at six months. In well-controlled diabetic patients, the systemic dividend of enhanced home care appears to be anti-inflammatory rather than directly glycaemic, consistent with NSPT itself being the principal driver of HbA1c change.

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**KEYWORDS:** C-reactive protein, glycated haemoglobin, systemic inflammation, type 2 diabetes mellitus, chronic periodontitis, sub-gingival irrigation, non-surgical periodontal therapy.

## 1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder of defective insulin secretion and action that currently affects several hundred million adults worldwide and continues to rise steeply in South Asian populations. Chronic periodontitis, a multifactorial inflammatory disease of the tooth-supporting tissues, is now widely regarded as the sixth complication of diabetes. The two conditions are joined by a bidirectional relationship: hyperglycaemia impairs neutrophil function, alters collagen turnover, and promotes advanced glycation end-product accumulation that accelerates periodontal breakdown, while inflamed periodontal tissue releases pro-inflammatory cytokines such as interleukin-1beta and tumour necrosis factor-alpha into the circulation, where they aggravate insulin resistance and impair glycaemic control.

A central node in this cross-talk is the systemic acute-phase response. C-reactive protein (CRP), a hepatically synthesized acute-phase reactant, is elevated in both diabetes and periodontitis and rises further when the two coexist, making it a sensitive, if non-specific, index of the systemic inflammatory burden imposed by periodontal disease. Because circulating inflammatory mediators are mechanistically implicated in insulin resistance, CRP has been proposed as a plausible intermediary linking periodontal inflammation to glycaemic dysregulation. Glycated haemoglobin (HbA1c), by contrast, reflects average glycaemia over the preceding two to three months and is the accepted longitudinal marker of glycaemic control. Examining CRP and HbA1c together therefore offers a window onto the two limbs of the diabetes–periodontitis axis: the inflammatory limb and the metabolic limb.

Non-surgical periodontal therapy (NSPT) reduces this inflammatory burden and, in aggregate across meta-analyses, produces a modest reduction in HbA1c of roughly 0.3 to 0.4 percentage points. What remains far less settled is the incremental role of the homecare maintenance regimen that follows NSPT. Mechanical plaque control by brushing and flossing is standard, but it accesses sub-gingival biofilm poorly, particularly within residual pockets. Powered sub-gingival water irrigation has been proposed as an adjunct capable of disrupting sub-gingival biofilm through pulsatile compression–decompression forces, and several trials report improved periodontal indices with its use. Whether that additional local plaque control translates into a measurable systemic dividend, specifically a greater fall in CRP and, through it, a greater improvement in glycaemic control, has been inconsistently demonstrated, especially in well-controlled diabetic patients whose glycaemic values leave little room for further reduction.

The present analysis was therefore designed to interrogate the systemic inflammatory–glycaemic axis directly. Using a randomized cohort of type 2 diabetic patients with chronic periodontitis, all of whom received NSPT, it asks whether adjunctive homecare sub-gingival irrigation confers a differential effect on serum CRP and on HbA1c compared with conventional homecare, and whether any such systemic response mirrors the degree of periodontal inflammatory resolution achieved.

### 1.1 Objectives

(1) To compare the six-month change in serum C-reactive protein between diabetic patients maintained on homecare sub-gingival irrigation and those on conventional homecare after NSPT. (2) To compare the six-month change in glycated haemoglobin between the two maintenance protocols. (3) To examine whether the between-group difference in systemic inflammatory response corresponds to the between-group difference in periodontal inflammatory resolution. (4) To interpret the relative contribution of the maintenance regimen, as against NSPT itself, to systemic inflammatory and glycaemic outcomes in well-controlled T2DM.

## 2. MATERIALS AND METHODS

### 2.1 Study Design, Setting, and Ethics

This was a single-blind, parallel-group randomized controlled trial conducted at the Post-Graduate Department of Periodontology and Oral Implantology, Yamuna Institute of Dental Sciences and Research, Yamuna Nagar, Haryana, India.

The total study duration from recruitment to final recall was eighteen months. The protocol was approved by the institutional ethical committee of Pandit B. D. Sharma University of Health Sciences, Rohtak, and written informed consent was obtained from every participant before enrolment. The present report is a pre-specified analysis of the trial focused on systemic inflammatory and glycaemic outcomes.

### 2.2 Participants and Eligibility

Patients attending the outpatient department were screened. Inclusion required a diagnosis of T2DM with HbA1c below 7%; generalized chronic periodontitis by the 1999 Armitage classification (probing depth of at least 5 mm and clinical attachment loss of at least 2 mm in more than 30% of teeth); a stable dose and type of anti-diabetic therapy for the preceding six months; and otherwise good systemic health. Patients were excluded for systemic antibiotic use or periodontal treatment within the preceding six months, pregnancy or lactation, immunosuppressive therapy, supplemental ascorbic acid or anticoagulant use, significant cardiovascular, haematological, renal, hepatic, or immunological disease, or a history of smoking or alcohol use. The restriction to HbA1c below 7% and the exclusion of smokers were deliberate, since both hyperglycaemia and tobacco independently elevate CRP and would otherwise confound the systemic read-outs.

With type I error of 0.05 and power of 80%, the minimum sample to detect the pre-specified periodontal difference was 19 per group; to strengthen comparison and accommodate the systemic analyses, 140 patients were enrolled and randomized equally into two groups of 70.

### 2.3 Randomization, Blinding, and Intervention

Eligible patients were allocated to Group 1 or Group 2 using research randomizer software, with single blinding of participants to allocation. All 140 patients first underwent Phase I therapy (supra-gingival scaling and root planing), and baseline parameters were recorded one week later. Group 1 (n=70) used a powered oral irrigator with a sub-gingival tip after brushing, twice daily, with a full reservoir of water per manufacturer instructions; correct technique was demonstrated at baseline and reinforced at each recall. Group 2 (n=70) brushed twice daily and flossed with commercially available floss, without irrigation. Both groups used standard dentifrices throughout.

### 2.4 Systemic Outcome Measures

The primary outcomes of this analysis were serum CRP and HbA1c. Because the biological half-lives of both markers make short-interval re-testing uninformative, each was assessed only at baseline and at six months. HbA1c was measured by liquid chromatography and CRP by a latex agglutination-based assay, both in a certified laboratory. To contextualize the systemic findings, periodontal inflammatory parameters, the Gingival Index (Loe and Silness, 1963), Plaque Index (Silness and Loe, 1964), Oral Hygiene Index-Simplified (Greene and Vermillion, 1964), Gingival Bleeding Index (Ainamo and Bay, 1975), probing depth, and clinical attachment loss, were recorded at baseline and at one, three, and six months, with probing performed using a calibrated Michigan probe with William's markings at approximately 0.25 N.

### 2.5 Statistical Analysis

Analyses were performed in SPSS version 25.0 and MedCalc. Continuous variables were summarized as mean and standard deviation. Independent (unpaired) Student's t-tests compared the two groups at baseline and at six months and compared the magnitude of change from baseline; paired testing assessed within-group change over time. For the periodontal context parameters, repeated-measures ANOVA with post-hoc Bonferroni correction was applied across the four time-points. To address the third objective, the between-group systemic responses were interpreted alongside the between-group periodontal responses to assess whether the marker that resolved differentially (CRP) tracked the arm with superior periodontal inflammatory control. A two-sided p-value below 0.05 with a 95% confidence interval was considered significant.

## 3. RESULTS

### 3.1 Cohort

All 140 randomized patients, 70 per group, completed the six-month follow-up, and no adverse events attributable to either homecare protocol were reported. The two arms were comparable at baseline for both systemic markers, with no significant difference in CRP (p=0.096) or HbA1c (p=0.081), supporting valid between-group comparison of the six-month response.

### 3.2 Systemic Inflammatory Response (C-Reactive Protein)

CRP fell markedly in both arms but significantly more with adjunctive irrigation. In Group 1, mean CRP decreased from 17.06±2.42 mg/L at baseline to 6.96±1.21 mg/L at six months, a reduction of 9.10 mg/L (t=16.810, p=0.001). Group 2 also improved, from 18.27±2.38 mg/L to 10.17±1.90 mg/L, a reduction of 8.10 mg/L. Although baseline values did not differ (p=0.096), the six-month CRP was significantly lower in Group 1 than Group 2 (6.96 vs 10.17 mg/L, p<0.001), and the magnitude of reduction was significantly greater in Group 1 (9.10 vs 8.10 mg/L, p=0.048), indicating a superior systemic anti-inflammatory effect associated with the irrigation protocol (Table 1).

**Table 1:** Inter-group comparison of C-reactive protein (*p<0.05)

| Time-point | Group 1 Mean±SD (mg/L) | Group 2 Mean±SD (mg/L) | p-value |
|---|---|---|---|
| Baseline | 17.06 ± 2.42 | 18.27 ± 2.38 | 0.096 |
| 6 months | 6.96 ± 1.21 | 10.17 ± 1.90 | <0.001* |
| Baseline–6-month reduction | 9.10 ± 2.42 | 8.10 ± 2.24 | 0.048* |

### 3.3 Glycaemic Response (Glycated Haemoglobin)

In contrast to the inflammatory marker, HbA1c improved only modestly and to a near-identical degree in both arms. Mean HbA1c fell from 6.69±0.20% to 6.44±0.19% in Group 1 (a reduction of 0.25 percentage points) and from 6.76±0.19% to 6.52±0.17% in Group 2 (a reduction of 0.24 percentage points). Neither the six-month value (p=0.192) nor the magnitude of

reduction (p=0.182) differed significantly between the two protocols, and the baseline values were also statistically comparable (p=0.081). Thus, the enhanced homecare regimen produced no additional glycaemic benefit over conventional hygiene despite its clear inflammatory advantage (Table 2).

**Table 2:** Inter-group comparison of glycated haemoglobin

| Time-point | Group 1 Mean±SD (%) | Group 2 Mean±SD (%) | p-value |
|---|---|---|---|
| Baseline | 6.69 ± 0.20 | 6.76 ± 0.19 | 0.081 |
| 6 months | 6.44 ± 0.19 | 6.52 ± 0.17 | 0.192 |
| Baseline–6-month reduction | 0.25 ± 0.16 | 0.24 ± 0.24 | 0.182 |

### 3.4 Systemic Response in Relation to Periodontal Inflammatory Resolution

The systemic findings were interpreted against the periodontal response of the same cohort. The arm that achieved the greater systemic anti-inflammatory effect, Group 1, was also the arm that achieved significantly superior periodontal inflammatory control at six months: lower Gingival Index (0.48±0.36 vs 0.78±0.32, p=0.009), shallower probing depth (2.24±0.48 mm vs 3.60±0.51 mm, p<0.001), less residual clinical attachment loss (0.63±0.60 mm vs 1.01±0.57 mm, p=0.049), and significantly lower gingival bleeding at three and six months (Table 3). The direction of the CRP response therefore tracked the direction of periodontal inflammatory resolution: the group with more complete disruption of sub-gingival biofilm and less bleeding on probing carried the lower circulating acute-phase burden. The glycaemic response, by contrast, did not follow this gradient, remaining essentially equal between arms despite the divergent periodontal and inflammatory outcomes.

**Table 3**: Periodontal inflammatory context at six months (*p<0.05)

| Parameter at 6 months | Group 1 | Group 2 | p-value |
|---|---|---|---|
| Gingival Index | 0.48 ± 0.36 | 0.78 ± 0.32 | 0.009* |
| Probing depth (mm) | 2.24 ± 0.48 | 3.60 ± 0.51 | <0.001* |
| Clinical attachment loss (mm) | 0.63 ± 0.60 | 1.01 ± 0.57 | 0.049* |

## 4. DISCUSSION

This analysis of 140 well-controlled type 2 diabetic patients with chronic periodontitis addressed a specific question: whether adjunctive homecare sub-gingival irrigation moves the systemic inflammatory–glycaemic axis differently from conventional homecare when both follow NSPT. The answer was dissociated across the two limbs of that axis. The inflammatory limb responded: CRP fell significantly more, and to a significantly lower six-month value, with irrigation. The glycaemic limb did not: HbA1c improved only modestly and equally in both arms.

The differential CRP response is mechanistically coherent. A pulsating stream of water generates alternating compression and decompression forces within the periodontal pocket, disrupting sub-gingival biofilm to a depth that brushing and flossing cannot reach. Sustained twice-daily reduction of sub-gingival bacterial load lowers the local inflammatory drive, and because CRP is a hepatic acute-phase reactant responsive to circulating pro-inflammatory cytokines of periodontal origin, a lower local burden is reflected in a lower systemic one. That the arm with the greater CRP reduction was also the arm with lower gingival bleeding, shallower pockets, and less residual attachment loss reinforces the interpretation that the systemic anti-inflammatory dividend was a downstream consequence of superior periodontal inflammatory control rather than an independent effect of irrigation. This is consistent with earlier reports of reduced serum inflammatory markers following successful periodontal therapy in diabetic populations, and with observations that reductions in CRP after periodontal intervention are closely tied to the degree of inflammatory resolution achieved.

The flat glycaemic response merits equal attention, because it clarifies where the maintenance regimen does and does not act. Several considerations converge to explain why HbA1c did not diverge between arms. First, meta-analytic evidence indicates that the glycaemic benefit of periodontal care, typically a reduction of roughly 0.3 to 0.4 percentage points, derives predominantly from the mechanical debridement of NSPT itself rather than from the particular homecare regimen that follows; since both arms received identical NSPT, the principal glycaemic lever was shared, leaving little for the maintenance protocol to add. Second, HbA1c integrates glycaemia over two to three months and is shaped by diet, physical activity, medication adherence, and psychosocial stress, none of which was controlled here, so any small incremental glycaemic effect of superior plaque control could readily be diluted by these unmeasured influences. Third, and most importantly, enrolment was restricted to patients with baseline HbA1c below 7%; in this well-controlled range a floor effect operates, and a further statistically demonstrable reduction is inherently difficult to achieve. The dissociation observed here, a moving CRP alongside a static inter-group HbA1c, is therefore not contradictory but expected: the inflammatory marker is more proximate and more responsive to incremental local plaque control, whereas the glycaemic marker is more distal, more buffered, and already near its floor.

These findings sit comfortably within the wider literature. Studies dissecting the CRP–HbA1c relationship after periodontal therapy have suggested that improvements in glycaemic control are most evident precisely in those patients whose CRP falls, implicating amelioration of the inflammatory state as the mediating step. In the present well-controlled cohort, the inflammatory step was achieved differentially by irrigation, yet the glycaemic step did not follow, most plausibly because the metabolic reserve for improvement had already been narrowed by the sub-7% entry criterion. The results thus refine rather than contradict the inflammation-mediated model: reducing systemic inflammation is necessary but not, on its own and within a short window, sufficient to shift HbA1c in patients who are already well controlled.

The clinical implication is that homecare sub-gingival irrigation should be valued in diabetic periodontal maintenance chiefly for its anti-inflammatory and periodontal benefits. Lowering the systemic inflammatory burden is itself worthwhile in a

population at elevated cardiometabolic and renal risk, independent of any measurable movement in HbA1c. Clinicians should not, however, expect enhanced homecare to deliver additional glycaemic improvement in patients whose diabetes is already well controlled, where NSPT remains the operative intervention for whatever glycaemic benefit is attainable.

## 5. CONCLUSION

In this randomized controlled analysis of 140 well-controlled type 2 diabetic patients with chronic periodontitis, adjunctive homecare sub-gingival irrigation produced a significantly greater reduction in systemic inflammatory burden, measured as serum CRP, than conventional homecare, mirroring its superior control of periodontal inflammation. It did not, however, confer any additional benefit for glycaemic control, with HbA1c improving modestly and equally in both arms. The systemic dividend of enhanced homecare in this population is therefore anti-inflammatory rather than directly glycaemic, and NSPT itself appears to be the principal driver of HbA1c change. Homecare sub-gingival irrigation is best positioned as a beneficial adjunct for lowering periodontal and systemic inflammation in diabetic maintenance, while larger trials enrolling patients across a wider glycaemic range and incorporating a broader biomarker panel are needed to determine whether its inflammatory advantage can be converted into a demonstrable metabolic one.

### 5.1 Limitations

The cohort was drawn from a single centre and a single geographic region, limiting generalizability, and the six-month horizon precluded assessment of durability. CRP is a non-specific acute-phase reactant influenced by intercurrent conditions, and no additional cytokines, such as interleukin-1beta or tumour necrosis factor-alpha, were measured to trace the mediating pathway. Confounders of both CRP and HbA1c, including diet, physical activity, body-mass index, and comorbidity, were neither controlled nor recorded, and the restriction to baseline HbA1c below 7% imposed a floor effect that constrained the detectable glycaemic response.

### 5.2 Scope for Future Research

Future work should enrol diabetic patients across a broader glycaemic spectrum, including moderately and poorly controlled individuals in whom greater metabolic reserve may allow an inflammatory improvement to translate into a glycaemic one; extend follow-up beyond six months; and incorporate a wider panel of inflammatory mediators together with formal mediation analysis to test directly whether CRP lies on the causal path between periodontal resolution and glycaemic change in the diabetes–periodontitis axis.

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