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Omega-3 with guided biofilm therapy in periodontitis: Case report

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ABSTRACT

Stage IV periodontitis requires sustained biofilm and inflammatory control. Evidence for omega-3 adjuncts is heterogeneous, and their use within a structured Guided Biofilm Therapy (GBT) pathway for Stage IV disease has not been evaluated. This case report describes a 54-year-old man recorded as having generalised Stage IV, Grade C periodontitis who received GBT, site-specific ultrasonic and hand instrumentation, and adjunctive omega-3 supplementation. NOW Ultra Omega-3 was prescribed as one 1,000-mg softgel once daily before breakfast for 90 days; each softgel provided 500 mg eicosapentaenoic acid and 250 mg docosahexaenoic acid. From baseline to 6 months, full-mouth plaque and bleeding-on-probing scores decreased from 100% to 28%, mean probing depth from 4.8 to 3.2 mm, and mean clinical attachment level from 5.6 to 4.7 mm. Total leukocytes decreased from 8.87 to 6.70 × 10^3/µL and C-reactive protein from 4.5 to 3.9 mg/L. The periodontal changes are compatible with a favourable short-term response to comprehensive non-surgical care, whereas the laboratory shifts remain descriptive. Because behaviour change, air polishing, instrumentation, supportive care, and omega-3 were delivered concurrently in one patient, neither component-specific efficacy nor causality can be inferred. A placebo-controlled randomised trial using identical periodontal care in both arms is required to isolate omega-3 effects; a factorial design would additionally test the GBT pathway. This case therefore provides a detailed hypothesis-generating observation rather than evidence of efficacy.

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1. INTRODUCTION

Periodontitis is a biofilm-associated, host-mediated inflammatory disease causing progressive periodontal-attachment and alveolar-bone loss (Papapanou et al., 2018). Staging describes severity and treatment complexity, whereas grading estimates progression and future risk (Tonetti et al., 2018). Untreated disease can cause tooth loss and impair function and oral-health-related quality of life (Tonetti et al., 2018). Associations with systemic disorders vary by condition (Oliveira et al., 2020); although periodontal therapy may modify systemic inflammatory burden, responses require interpretation within the patient's broader medical context (Mainas et al., 2022).

Contemporary care uses stepwise behavioural support, control of risk factors and supragingival biofilm, subgingival instrumentation, reassessment, and supportive periodontal care (Sanz et al., 2020). Stage IV additionally requires management of functional and rehabilitative complexity (Herrera et al., 2022). Deep pockets, furcations, and root concavities can limit instrumentation access (Tomasi et al., 2023); adjunctive-device benefits are inconsistent and do not replace meticulous mechanical debridement (Gheorghiu et al., 2021).

Air polishing targets supra- and subgingival biofilm. Omega-3 eicosapentaenoic and docosahexaenoic acids may act through pro-resolving mediators that limit neutrophil recruitment and promote macrophage efferocytosis and resolution (Donos et al., 2020; Jentsch et al., 2020; Serhan, 2017). Omega-3 trials have accompanied conventional therapy, whereas GBT studies have focused on biofilm control; their combination in Stage IV disease remains untested. This report documents the 6-month periodontal and laboratory response while defining the causal limits of a single-case observation.

2. RESEARCH METHODS

This case involved a 54-year-old systemically healthy non-smoker with no history of professional periodontal therapy, presenting with generalised inflammation and heavy plaque. Baseline assessment included disclosed-biofilm photographs (Figure 1), panoramic radiography (Figure 2), full-mouth periodontal measurements, and blood tests. The record classified generalised Stage IV, Grade C periodontitis based on generalised bone loss, reported periodontitis-related loss of multiple mandibular posterior teeth, and probing depths of at least 6 mm. Exact Stage IV complexity and Grade C progression criteria could not be independently verified from functional or longitudinal data or a quantified bone-loss-to-age ratio.

The Guided Biofilm Therapy protocol comprised biofilm disclosure, individualised oral-hygiene instruction, supra- and subgingival air polishing, and site-specific ultrasonic and hand instrumentation for residual calculus. NOW Ultra Omega-3 was prescribed as one 1,000-mg softgel once daily before breakfast for 90 days; each softgel provided 500 mg eicosapentaenoic acid and 250 mg docosahexaenoic acid. The patient reported adherence to the prescribed regimen, good tolerability, and no adverse events; adherence was not objectively verified. Reviews at 3 and 6 months reinforced self-performed plaque control and supportive care. The 6-month endpoint captured short-term response after active therapy and early supportive care beyond initial healing; it did not establish long-term stability. Outcomes were summarised descriptively without inferential analysis because no comparator existed.

3. RESULTS AND DISCUSSION

At baseline, the full-mouth plaque and bleeding-on-probing scores were both 100%, mean probing depth was 4.8 mm, and mean clinical attachment level was 5.6 mm (Figure 3). Baseline intraoral photographs documented extensive disclosed biofilm and inflamed gingival tissues (Figure 1), while panoramic radiography demonstrated generalised alveolar bone loss and multiple missing mandibular posterior teeth (Figure 2). At 6 months, the full-mouth plaque and bleeding-on-probing scores were 28%, mean probing depth was 3.2 mm, and mean clinical attachment level was 4.7 mm, corresponding to a mean probing-depth reduction of 1.6 mm and a mean clinical attachment gain of 0.9 mm (Figure 3). The intraoral photographs also showed less visible plaque and gingival inflammation (Figure 1). Total leukocytes decreased from 8.87 to 6.70 × 10^3/µL, the neutrophil proportion from 73% to 51%, and quantitative C-reactive protein from 4.5 to 3.9 mg/L; the lymphocyte proportion increased from 17% to 39% (Figure 4). These laboratory changes are descriptive and cannot be attributed to either omega-3 supplementation or periodontal therapy in the absence of repeated controls and a comparator.

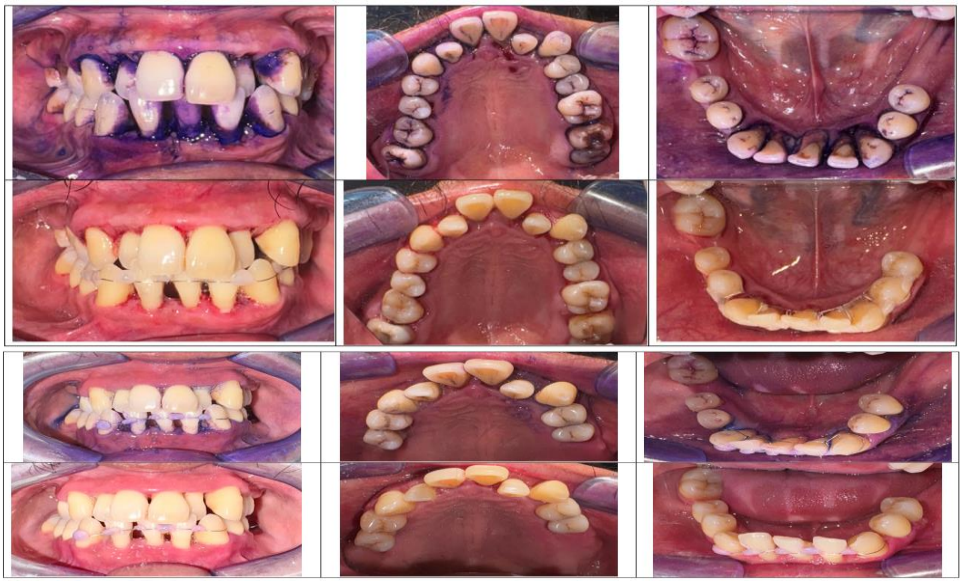

Figure 1. Intraoral conditions at baseline (upper panel) and 6 months after non-surgical periodontal therapy (lower panel)

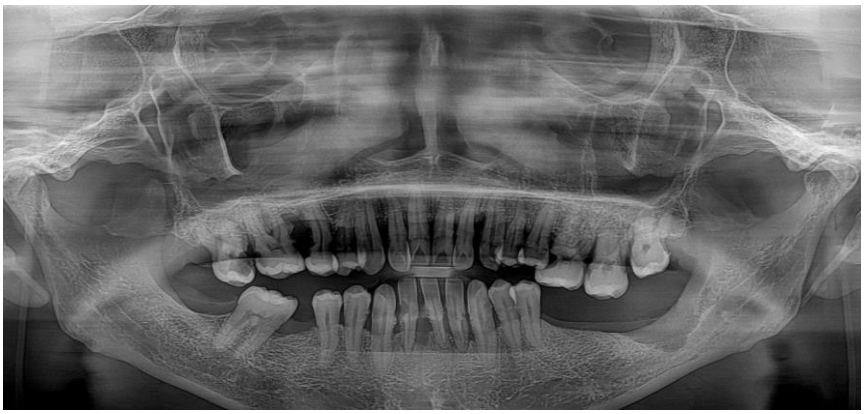

Figure 2. Baseline panoramic radiograph

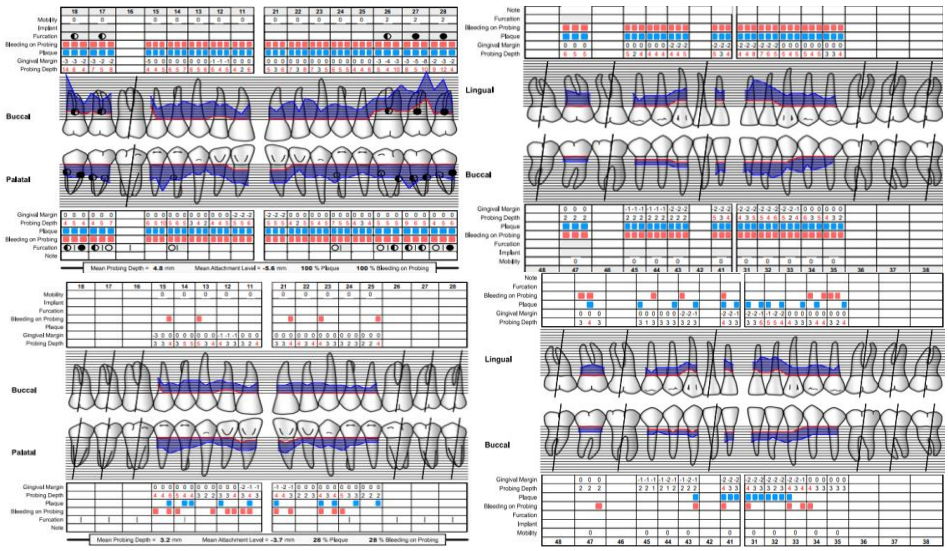

Figure 3. Full-mouth periodontal charting at baseline (left) and 6 months (right)

Figure 4. Laboratory blood test results at baseline and at the 6-month follow-up

Omega-3 polyunsaturated fatty acids have been investigated as adjunctive host-modulatory agents because eicosapentaenoic and docosahexaenoic acids can alter eicosanoid synthesis and serve as substrates for specialised pro-resolving mediators. Resolvins, protectins, and related mediators can limit excessive neutrophil recruitment, promote macrophage efferocytosis, and support a return to tissue homeostasis (Bannenberg & Serhan, 2010; Serhan, 2017). In periodontal tissues, the proposed consequence is attenuation of dysregulated, neutrophil-driven collateral injury while preserving the active resolution of inflammation. This biological plausibility does not establish that omega-3 caused either the periodontal response or the leukocyte and C-reactive-protein changes in this patient.

Published randomised trials have evaluated omega-3 as an adjunct to conventional non-surgical periodontal therapy and have reported additional probing-depth reductions or clinical-attachment improvements in some settings (Deore et al., 2014; El-Sharkawy et al., 2010; Stańdo et al., 2020). Interpretation remains limited because some protocols also used low-dose aspirin and because supplement composition, dose, duration, patient populations, periodontal protocols, and outcome definitions varied. Consistent with this uncertainty, the EFP Stage I-III guideline did not recommend omega-3 polyunsaturated fatty acids as an adjunct to subgingival instrumentation because the evidence was insufficient (Sanz et al., 2020). Subsequent evidence syntheses reported a possible modest added benefit but noted heterogeneous regimens and relatively small trials, limiting regimen-specific inference (Kruse et al., 2020). A later meta-analysis estimated adjunct-specific mean benefits of 0.39 mm in probing-depth reduction and 0.41 mm in clinical-attachment gain (Van Ravensteijn et al., 2022). These pooled between-group effects are not directly comparable with the total within-patient response observed here. Observational evidence in smokers and non-smokers likewise cannot establish causality (Savran & Sağlam, 2024). None of these designs isolates omega-3 within a GBT pathway or establishes whether the two adjunctive concepts interact in Stage IV periodontitis. The present report therefore contributes a clinically documented hypothesis, not an estimate of omega-3 efficacy.

Guided Biofilm Therapy is a proprietary, structured workflow combining biofilm disclosure, patient education, low-abrasive air polishing, and selective calculus instrumentation and should not be interpreted as a biologically independent alternative to mechanical therapy (Savran & Sağlam, 2024). Mechanical subgingival instrumentation remains the evidence-based foundation of non-surgical periodontal treatment (Suvan et al., 2020). Air polishing can improve biofilm disruption and patient comfort, but its incremental clinical effect depends on powder, device, pocket morphology,

maintenance interval, and comparator (Braun et al., 2007; Gheorghe et al., 2023; Stein et al., 2021). In this case, disclosure, instruction, and air polishing plausibly supported biofilm control, whereas ultrasonic and hand instruments were used for residual calculus and deposits not removed by air polishing. Because these components were intentionally combined and no GBT-only or conventional-instrumentation comparator was used, their separate contributions cannot be quantified.

At the patient level, the absolute periodontal changes were substantial: mean probing depth decreased by 1.6 mm, mean clinical attachment level improved by 0.9 mm, and plaque and bleeding scores each decreased by 72 percentage points. These changes indicate substantial short-term improvement, but residual plaque and bleeding scores of 28% show that biofilm control and inflammatory resolution were incomplete. Moreover, full-mouth means do not disclose residual deep pockets, pocket closure, tooth-level prognosis, or functional recovery. The most plausible contributors to plaque reduction were biofilm disclosure, individualised oral-hygiene instruction, improved self-performed plaque control, and reinforcement at the 3- and 6-month reviews. Reduced bleeding and probing depth additionally reflect professional supra- and subgingival biofilm disruption and calculus removal. These components occurred together, so their relative importance cannot be ranked. Consistent with this interpretation, trials comparing full-mouth instrumentation concepts show that substantial improvement can follow thorough mechanical therapy and maintenance without identifying one adjunct as the decisive factor (Stein et al., 2021). The C-reactive-protein reduction was only 0.6 mg/L, while total leukocytes decreased by $2.17 \times 10^3/\mu L$ and the neutrophil and lymphocyte proportions shifted by 22 percentage points in opposite directions. With one measurement at each time point, no comparator, and no control of intercurrent influences, these laboratory changes cannot be interpreted as a clinically established systemic anti-inflammatory effect.

The principal limitations are the single-patient design, concurrent interventions, lack of a control or withdrawal phase, absence of calibrated-examiner data and microbiological outcomes, and follow-up limited to 6 months. Although the product and prescribed regimen were documented, adherence remained self-reported and was not verified by capsule counts, pharmacy records, or plasma fatty-acid measurements; actual exposure and the exposure-response relationship therefore remain uncertain. The recorded Grade C estimate could not be independently verified from longitudinal measurements or a quantified bone-loss-to-age ratio. The report also lacks residual-pocket distributions, pocket-closure data, tooth-level prognosis, masticatory and occlusal outcomes, definitive rehabilitation, oral-health-related quality of life, and longer-term tooth retention. Accordingly, neither the periodontal nor laboratory response can be attributed to omega-3, GBT, air polishing, mechanical instrumentation, or any interaction among them.

For Stage IV periodontitis, the practical implication is that rigorous infection control and early supportive care should precede reassessment and any definitive functional or rehabilitative phase; this case demonstrates short-term inflammatory improvement but not comprehensive Stage IV rehabilitation or stability and does not justify routine omega-3 prescribing. To test an omega-3-specific effect, a multicentre, double-blind, placebo-controlled randomised trial should provide identical evidence-based periodontal therapy and supportive care to both groups while varying only a standardised omega-3 regimen. Allocation concealment, calibrated blinded examiners, adherence assessment, adverse-event monitoring, prespecified patient- and site-level periodontal outcomes, systemic biomarkers, patient-reported outcomes, and at least 12 months of follow-up are needed. If the incremental GBT contribution and a possible omega-3-by-GBT interaction are also study questions, a sufficiently powered 2×2 factorial design would be preferable.

4. CONCLUSION

This case documents a marked 6-month periodontal response after a structured GBT-based non-surgical programme in which omega-3 was prescribed adjunctively for a patient recorded as having

Stage IV periodontitis. Its principal contribution is a detailed, hypothesis-generating description of this combined pathway and its unresolved causal questions. Clinically, the findings reinforce, but cannot independently validate, the established priority of intensive biofilm control, behavioural reinforcement, site-specific instrumentation, and early supportive care before reassessment and subsequent Stage IV functional and rehabilitative planning. They do not demonstrate efficacy of either GBT or omega-3 because the interventions were concurrent and there was no comparator. An omega-3-specific effect requires a double-blind, placebo-controlled randomised trial with identical periodontal care, a standardised and verifiable supplement exposure, blinded outcome assessment, and longer follow-up; a factorial design is required if the GBT pathway and its interaction with omega-3 are also to be isolated. Patient consent and ethics, written informed consent was obtained from the patient for treatment and for publication of de-identified clinical information, periodontal charts, radiographs, and clinical photographs. The report was prepared in accordance with the CARE case-report guideline.

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