Patient Preference and Adherence

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ORIGINAL RESEARCH

Teaching Patients to Self-Care for Active, Recurrent Periodontal or Peri-Implant Pockets Guided by the TIME Wound-Healing Model: A Pilot Feasibility Study Based on Clinical and Microbiological Outcomes

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Background: The TIME therapeutic model is used for the management of chronic wounds: Tissue (non-viable); Infection/Inflammation; Moisture (imbalance); Edges (non-advancing). These four components will determine the persistence or the healing of any chronic ulcer on the skin's surface and, by analogy, also those of the ulcerated epithelium at the subgingival level. We aimed to evaluate the clinical and microbiological changes recorded after implementation of this personalized subgingival model.

Methods: Twelve patients with active periodontal or peri-implant pockets were recruited for a feasibility study. Patients were instructed to deeply clean these lesions subgingivally using an angulated interdental brush in a vertical position, twice per day for 15 days. On the first and last days, Löe & Silness gingival index and bleeding on probing (BoP) were recorded and samples were collected using the brush head for the quantitative PCR analysis of 8 bacterial species (commensal and pathogenic).

Results: Severe gingival inflammation with profuse bleeding was present at baseline in ten patients. Eight of them complied and adhered with 100% of the treatment. Following self-treatment at home, ten patients exhibited normal or mildly inflamed gums. Seven patients no longer had bleeding, four had slight bleeding and only one moderate bleeding. Microbiologically, the total bacterial load significantly decreased from 7E07 to 9.39E06 cfu/head.

Conclusion: This proposed conservative cost-effective subgingival model could significantly improve the inflammatory activity of certain recurrent periodontal or peri-implant pockets, stabilize them and thus minimize their progression. The preliminary findings reflected a reduction or absence of bleeding, a relative decrease in pathogenic species, and the restoration of a microbial community in symbiosis with the host.

Keywords: periodontitis, peri-implantitis, chronic wound, dysbiosis, debridement, subgingival healing, ulcerated pocket epithelium, personalized periodontics, self-monitoring, patient empowerment, treatment compliance

Introduction

A wound always involves the loss of anatomical and physiological continuity of the skin or the epithelium. An ulcer is a wound that does not heal in a sequential series of stages or within a normally predictable period, and those that do not heal within a three-month time frame are considered chronic. Chronic skin ulcers are a major cause of morbidity and mortality worldwide, and their aetiology is multifactorial.[1,2] Experts in chronic wound management rely on a model known by the acronym TIME, which stands for: Tissue (non-viable); Infection/Inflammation; Moisture (imbalance);

https://doi.org/10.2147/PPA.S596403
Received: 15 January 2026
Accepted: 24 March 2026
Published: 29 April 2026

Edges (non-advancing). These four components will determine the persistence or the healing of any chronic ulcer on the skin's surface and, by analogy, also those of the ulcerated epithelium at the subgingival level.[1,2]

The pathological deepening of the gingival sulcus intrinsically leads to apical migration of the junctional epithelium and, similarly, to the ulceration of the epithelium of the periodontal pocket. The presence of bloody ulcerated areas in the epithelium of the periodontal and/or peri-implant pockets causes them to behave like chronic open micro-wounds. Through these exposed and discontinuous areas, the contents of the periodontal and peri-implant pockets come into contact with the blood vessels of the gingival connective tissue (Figure 1). This allows for the dispersion of bacteria, bacterial toxins, and inflammatory mediators into the bloodstream.[3–6]

Bleeding on probing (BoP) is an unequivocal indication of inflammation. In fact, BoP allows us to easily and objectively monitor inflammatory activity, associated with tissue damage, inside periodontal pockets.[2,5] It is important to keep in mind that smokers usually have reduced BoP, which may consequently conceal periodontal disease activity.[7–9] The hygienic or causal phase of periodontal treatment involves initial professional mechanical debridement along with meticulous patient care at home, individualised oral hygiene measures, and subsequent surgical treatment according to individual needs in more advanced cases.[10,11]

Scaling and root planing (SRP) is increasingly referred to as periodontal instrumentation, which is considered the gold standard method of mechanical debridement based on the disruption of the biofilm.[12,13] Despite being considered a conventional treatment, it has its disadvantages, as it is a time-consuming and technically demanding procedure that occasionally causes patients discomfort.[14] Furthermore, it has been observed that the result of SRP also depends on the skills of the operator.[15] Research on the non-surgical treatment of periodontitis has revealed highly variable results.[16–19]

Several deep pockets often remain active after non-surgical periodontal treatment (SRP). These residual bleeding pockets are associated with disease progression, and the treatment of these areas is quite unpredictable. Recurrent periodontal pockets can be reduced by resective surgery, but the cost and morbidity are relatively high.[20]

Currently, other means of eliminating subgingival biofilm have evolved, and different technologies and devices have been introduced for its removal, such as vector scraping systems, lasers and an air polishing agent.[12] Recently, Khan et al[21] demonstrated similar reductions in inflammatory parameters when non-surgical treatment was performed using either a chitosan oscillating brush or titanium curettes.

Therefore, the development of more effective methods for non-surgical periodontal treatment is of great interest and remains an area of active research. However, in general, they have not been found to be significantly better than conventional mechanical approaches such as SRP.[7,10,19]

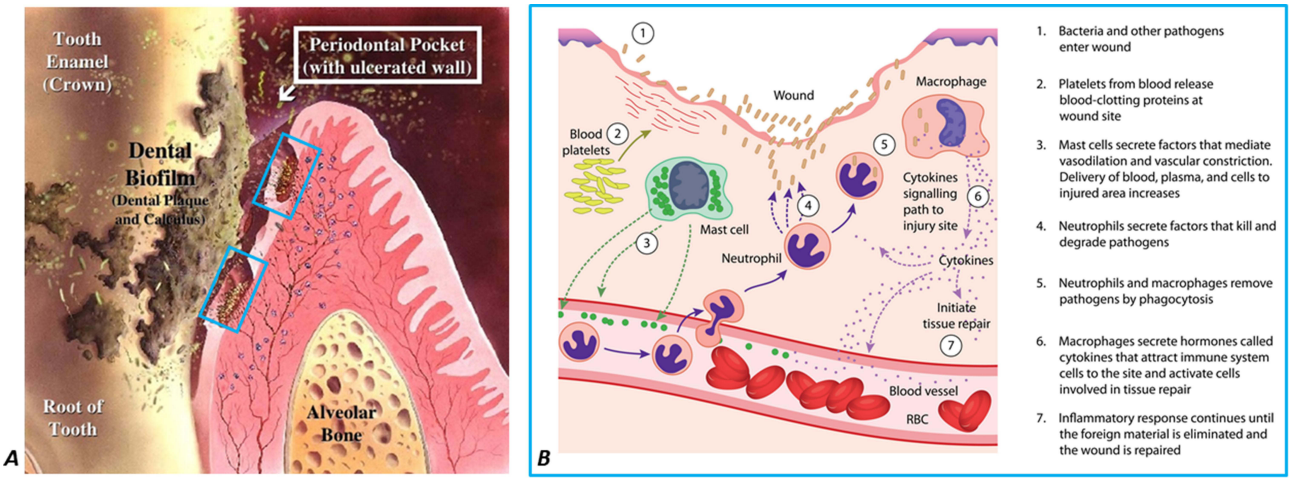

Figure 1 (**A**) Ulcerated periodontal pocket epithelium. Note also the proximity of the chronic ulcers to the connective blood vessels. From Offenbacher S. Scientific American 2006 (October); Special Supplement, pp. 24–29. (**B**) Inflammation and tissue repair. Cellular-chemical factors involved in the inflammatory response to tissue damage and healing. Source: Dreamstime.com LLC. USA. License for use - ID 62002173.

https://doi.org/10.2147/PPA.S596403

The accumulation of biofilm as a consequence of the establishment of a dysbiotic microbial community is considered the main aetiological factor of the inflammatory response in both periodontal and peri-implant soft and hard tissues.[22–24] Treatment focuses on controlling inflammation by reducing the bacterial load and helping return the microbiota around the infected tooth root or implant to a eubiotic state.[24,25] The development of effective non-surgical treatment methods is essential for treating patients for whom surgical treatment is contraindicated or for patients who are not willing to undergo surgery. Consequently, and based on the TIME therapeutic model, for chronic skin wounds, it seems appropriate to establish analogies to heal the micro-wounds of the ulcerated epithelium located inside these subgingival lesions. The objective of this pilot feasibility study was to conduct an initial assessment of the implementation of this home-based protocol. This evaluation was done by analysing the clinical and microbiological changes after home treatment of bleeding periodontal and/or peri-implant pockets using a conventional angled interdental brush, in vertical positioning, to subgingivally clean them.

Methods

Patients in the periodontal maintenance phase and who had active residual lesions were recruited to carry out this study at the Clinicians Associates dental practice (Terrassa, Barcelona, Spain), by a senior periodontist and consultant.

The study adheres to the ethical principles of the Declaration of Helsinki for research involving human subjects. It is part of a broader study on periodontal health approved by the clinical research and ethics committee of the Sant Joan de Déu Foundation (Internal code. PIC 26-18/187-21). All volunteers were patients in the periodontal maintenance phase who freely chose to participate and gave their written consent after being duly informed about the procedure.

One recurrent periodontal or peri-implant pocket was selected with a probing depth of between 5 and 9 mm per patient. In total, 12 motivated patients, who regularly attended supportive periodontal therapy (SPT) visits, were non-smokers and had no medical or pharmacological history were recruited. Clinical parameters were recorded at baseline (T0) and at completion (T1) of the home-based treatment. They were individually trained to clean these subgingival lesions on their own, twice per day, for 15 days. To carry out home care, they were provided with sufficient interproximal brushes of the appropriate diameter for the morphology of each lesion.

The degree of gingival inflammation was recorded using the Löe and Silness[26] gingival index: (0) no inflammation; (1) mild inflammation: slight colour changes and little change in texture; (2) moderate inflammation: reddening, oedema and moderate overgrowth as well as bleeding when pressure is applied; (3) severe inflammation: marked reddening and swelling; tendency toward spontaneous haemorrhage; ulceration.

BoP was evaluated by counting the time elapsed from the removal of the brush inserted into the lesion for sample collection until the appearance of bleeding. With this, the level of bleeding was classified: (0) no bleeding; (1) slight bleeding with the presence of a single spot of blood after more than 5 s; (2) moderate bleeding with the presence of a line of blood after 2–5 s; and (3) severe and immediate, with profuse, persistent bleeding. The periodontist was in charge of performing the clinical examination and instructing the patients personally to implement this home-based protocol. First, patients were taught with the help of a handheld mirror how dentists gently explore the area under the gums using a periodontal probe. Then the similar shape of the manual probe and the angled interdental brush was explained with the intention of showing that wound cleaning should be an atraumatic process. The angled interproximal brush selected, which resembled a periodontal probe, had a diameter that was suited to the size of the lesion (Figure 2). Each patient was trained to insert the brush deep into the subgingival lesion and wiggle it for five seconds. Finally, a schedule was given out for patients to record the date and the frequency with which home treatments were performed (stipulated frequency: every 12 hours - morning/night - for 15 days). In case of missing a treatment, importance was given to not marking that task on the record sheet so as to not falsify the result. Before and after this home treatment, clinical variables were recorded to evaluate the healing process, and microbiological samples were taken to examine changes in the microbial community of this niche. On day 0, aside from the clinical record, the same periodontist collected a subgingival sample from the lesion selected for microbiological analysis using an interproximal brush, like a flocked broom. The brush head was inserted directly into a sterile 1.5 mL tube, after cutting it with sterile pliers, and frozen at −80°C. On day 15, following the last home treatment, the same clinical record and sample collection were repeated using the same procedure.

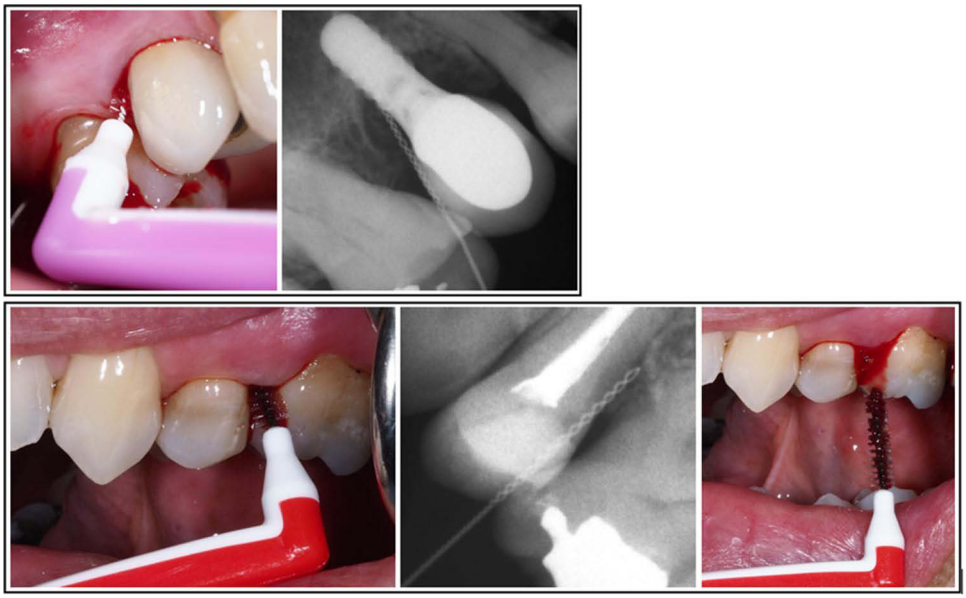

Figure 2 Participants were personally given instructions to insert the brush all the way to the bottom of the subgingival lesion and wiggle it for five seconds. The X-ray shows how the interdental brush reaches the bottom of the periodontal/peri-implant lesion.

Microbiological analysis was performed as follows: each head was resuspended in tubes with 800 µL of phosphate buffered saline (PBS), and tubes were vortexed for 5 minutes to release the bacteria. The brush was then removed with sterile forceps, and cells were pelleted by 5 min of centrifugation at 8,000 x *g*. After removing the supernatant, DNA was extracted with a QiAamp DNA Mini kit (Qiagen) following the manufacturer's instructions.

For each sample, quantitative PCR (qPCR) was used to quantify 8 bacterial species (*Actinomyces naeslundii, Streptococcus gordonii, Streptococcus oralis, Filifactor alocis, Porphyromonas endodontalis, Porphyromonas gingivalis, Prevotella intermedia* and *Tannerella forsythia*) as well as the total number of bacteria.

The oligonucleotides, probes and conditions used can be found in Àlvarez et al[27,28] except for *A. naeslundii*, whose sequence is TCGGGTTGTGAACCTCTTTC (forward), AGAGGATTTCACGA-CAGACG (reverse) and FAM-CAGTGAAGCAGGC-MGB (probe). Quantification was performed by converting Crossing Point (Cp) values into colony-forming units (cfu) using species-specific standard curves. These curves were generated by correlating the Cp values from serial dilutions of template DNA with the cfu/mL counts obtained from the original cultures, as previously described.[27] The statistical analysis of the differences between baseline and day 15 was performed using the Wilcoxon signed-rank test for paired data.

Results

Of the 12 participants, 9 were women whose average age was 57 years (range 29–78 years), and of the lesions monitored, two were affecting implants. The average probing depth of the lesions at baseline was 7.5 mm. The gingival index was grade 3 in 10 patients associated with severe, immediate, profuse and persistent bleeding (grade 3 bleeding); and in the other two patients, the gingival index was grade 2 with moderate bleeding (grade 2 bleeding).

The degree of compliance with home treatment after 15 days was very high. Eight patients complied with 100% of the treatments and of the twelve, only one had more erratic compliance with the home-based treatment (Table 1).

After the home treatments, all subgingival lesions scraped by deep and vertical brushing improved. As expected, periodontal probing did not show any variations compared to the initial recording. After the 15-day treatment, all patients expressed a feeling of clinical improvement including cessation of bleeding in most of the cleaned pockets, and also a reduction in the initial discomfort or even pain described. The gingival index significantly reduced in 5 patients to grade 0 (normal gingiva), another 5 patients had mild inflammation, and the remaining two patients had moderate inflammation.

Table 1 Clinical Values of Participants

ID	♀/♂	Age	Staging & Grading Periodontitis	Location	Probing Depth	Compliance	GI T0	BoP T0	GI T1	BoP T1
1	♀	53	II–III / B	# 37 M-L	9mm	100%	3	3	0	0
2	♀	29	I–II / A	# 33 D-V	5mm	25%	3	3	2	1
3	♂	72	III / B	i 26 M-V	9mm	76%	3	3	0	0
4	♂	70	III / A	i 15 M-V	5mm	100%	3	3	1	1
5	♂	67	I–II / A	# 27 M-V	9mm	100%	3	3	1	0
6	♀	71	IV / B	# 21 M-V	9mm	100%	3	3	1	0
7	♀	45	IV / C	# 46 Furca-V	9mm	85%	3	3	2	2
8	♀	66	III / B	# 16 D-V	9mm	100%	3	3	1	1
9	♀	49	I / A	# 46 D-V	9mm	100%	3	3	0	0
10	♀	40	III / B	# 22 D-V	6mm	100%	2	2	0	0
11	♀	45	III / B	# 32 M-V	6mm	100%	2	2	0	0
12	♀	78	II / A	# 15 D-V	6mm	50%	3	3	1	1
Avg.	**NA**	**57**	**NA**	**# (10) & i (2)**	**7.5mm**	**86.30%**	**2.83**	**2.83**	**0.75**	**0.5**

Notes: Bold values in the last row indicate the mean parameters of the participants.
Abbreviations: #, tooth; i, implant; M, mesial; D, distal; V, vestibular; L, lingual; Avg, Average; NA, Not applicable; T0, first day home treatment; T1, after 15 days home treatment; GI, Gingival index; BoP, Bleeding on probing.

Regarding gingival bleeding after taking the second sample on Day 15, seven of these patients no longer showed bleeding on probing, while four had slight bleeding and only one patient had moderate bleeding. The worst clinical results were associated with a lower degree of compliance in the home treatments carried out (Table 1).

These clinical changes were supported by a significant decrease in the number of periodontopathogens studied. In fact, the total bacterial load recovered from the interdental brush heads decreased significantly, going from an average of 7E07 cfu after the first use to 9.39E06 cfu after 15 days (Figures 3 and 4).

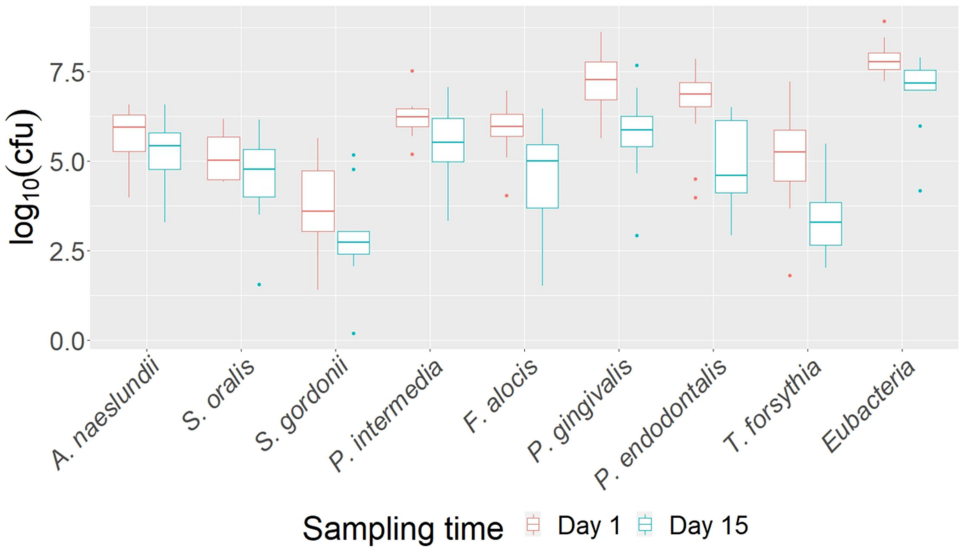

Figure 3 Bacterial load recovered from the periodontal pocket on days 1 and 15 of treatment. Quantitative PCR was used to quantify 8 species, in addition to the total bacterial load (eubacteria).

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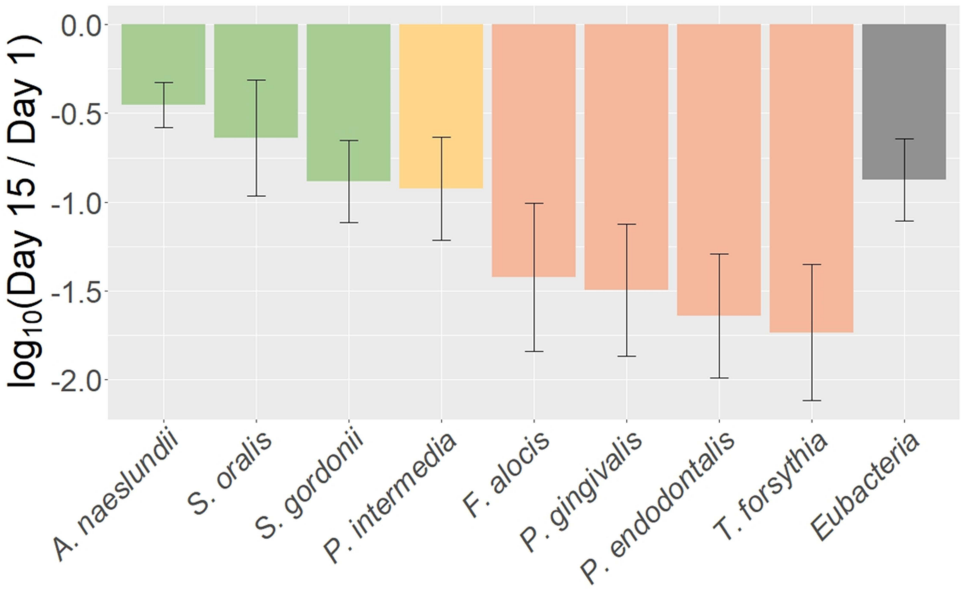

Figure 4 Mean proportion of bacteria recovered on day 15 compared to day 1, expressed on a logarithmic scale. Whiskers represent standard error. Primary colonisers, secondary colonisers, pathobionts and total bacteria are represented in green, yellow, salmon and grey, respectively.

A significantly lower load was also observed for 7 of the 8 bacterial species analysed (*S. oralis* was the exception). On day 0, the highest loads observed were of periodontal pathogens of the genus *Porphyromonas* (1.7E07 and 9.39E06 cfu/head of *P. gingivalis* and *P. endodontalis*, respectively) and *P. intermedia* (1.68E06 cfu/head). Around 1E05 cfu/head were recovered from each of the other species (Figure 3). After 15 days of treatments, the number of cells of each of the four pathobiont species studied (*F. alocis, P. gingivalis, P. endodontalis* and *T. forsythia*) had reduced by more than 10 times, while the loads of typically commensal species, considered primary and secondary colonisers of the oral biofilm, reduced by less than 10 times (Figure 4).

Discussion

Conceptually, healing is an endogenous process that occurs to repair tissue and involves the interaction between numerous types of cells. One of the primary objectives of some of these cells is to try to remove from the wound all debris that can hinder the progress of the healing stages. Debris or detritus is an ideal substrate for biofilm to grow. If not removed regularly, they will delay the healing process of chronic ulcers.[29] Additionally, this debris can become an appropriate substrate that promotes the development of complex phenomena associated with the bacterial burden: local infection, presence of biofilm, etc.[30]

Gums act as a protective barrier and have a sealing function around the teeth, but disease promotes the formation of periodontal pockets. These lesions are, ultimately, reservoirs of bacteria that will elicit an immune-inflammatory response from the host. Certain conditions promote the development of a dysbiotic microbiota, leading to an increase in the presence of pathobionts and their performance as pathogens.[24] These pathobionts release pro-inflammatory signals, which locally trigger the synthesis of cytokines and prostaglandins, favouring the destruction of the periodontal tissues themselves and increasing oxidative stress with consequent tissue damage.[3]

Although SRP is considered the gold standard method of mechanical debridement, it also has disadvantages.[16,31–33] Periodontal instrumentation has traditionally focused on the root surface. But today, periodontal medicine highlights the importance of curettage and debridement, disrupting the subgingival biofilm and promoting the healing of the ulcerated epithelium, thus providing a more curative approach to the hygienic phase,[34] which could also be based on the TIME strategy. This model is intended to be introduced as part of the conservative home management of selected active recurrent periodontal and peri-implant lesions, which in our study is carried out autonomously by the patients themselves.

In conclusion, a certain analogy can be established between chronic skin ulcers and those present in the epithelium of the periodontal or peri-implant pocket.

Methods currently used for non-surgical debridement of implants include titanium curettes, plastic or carbon fibre curettes, ultrasound, air polishing, and laser. However, no specific non-surgical treatment for peri-implantitis that produces superior results is supported by sufficient scientific evidence,[35,36] to the point of suggesting that some procedures may become iatrogenic and cause more complications, rather than improving peri-implant health.[37,38] What is imperative is to intervene and treat the inflammatory activity without causing further problems that would contribute to the progression of peri-implant attachment loss. In a multicentre study on the early stages of peri-implantitis, implant sockets were debrided with a chitosan brush placed on an oscillating dental bur for 3 minutes followed by irrigation with sterile saline solution.[39] Chitosan is a completely biocompatible biopolymer that has also been shown to be bacteriostatic and have anti-inflammatory properties.[40–42] In this series of multicentre cases of implants affected by mild peri-implantitis, significant reductions in clinical inflammation parameters were demonstrated at all time points following initial treatment with a chitosan brush.

This same research group carried out a 6-month multicentre, randomised, examiner-blinded clinical trial with seventy-eight patients with periodontitis. Patients were selected with a periodontal probing depth (PPD) of between 5 mm and 7 mm after previous active periodontal treatment. Patients were assigned subgingival treatment with curettes or an oscillating chitosan brush. Changes in BoP and PPD were evaluated between baseline and final evaluation at 6 months. The study's conclusions indicated that the chitosan brush demonstrated significantly better PPD reductions up to 6 months after baseline, compared to conventional treatment. An improvement in BoP was observed in both groups, and no adverse effects were observed.[7]

Our pilot study design was based on an analysis of clinical practice with exploratory and hypothesis-generating purposes. In our feasibility study, we instructed patients to use a conventional angled interproximal brush so that they could use it vertically, parallel to the axis of the tooth and do so independently at home. After 15 days, a clinical improvement in BoP was obtained, similar to the studies described above. Furthermore, we observed a significant decrease in the total bacterial load (eubacteria), which could be considered a measure of the effect of the treatment on the subgingival dysbiotic community. Since the aetiology of periodontitis and peri-implantitis depends on the homeostatic state of the microbial community,[24] we continued the microbiological analysis by quantifying three commensal species associated with health (*A. naeslundii, S. gordonii* and *S. oralis*) and five pathobiont species (*F. alocis, P. endodontalis, P. gingivalis, P. intermedia* and *T. forsythia*).[43,44] This way, we discovered that the significant decrease in bacterial load was greater in pathobionts than in commensals, indicating a transition towards a eubiotic community in symbiosis with the host, where pathobionts (harmful pathogens) are found in low relative abundances and commensals predominate.[24] This transition could have been produced by a disruption of the dysbiotic biofilm and the mechanical release of part of it, enhancing the performance of the immune system, and promoting recolonisation of the area under treatment, which would be carried out by the primary colonisers.

In studies like this one, which aim to determine the benefits of a treatment, the conclusions drawn from the total count of bacteria or from a particular genus are limited, since oral microorganisms are capable of rapidly colonising oral surfaces.[45] Therefore, it is important to analyse bacterial species with different functions in the microbial community and in the aetiology of periodontal diseases, in order to determine the level of dysbiosis of the community, which is closely related to the pathogenesis of periodontitis.[24] However, we have not been able to find such an evaluation in other studies similar to ours, such as the case of non-surgical treatment with the chitosan brush, which is the device most similar to the one we used in the present study. These studies show an improvement in BoP but none of them determine or quantify whether there is a change in the presence of periodontopathogens before and after such therapy.[7,21,38]

The care of chronic wounds contaminated by biofilm is one of the greatest challenges today for healthcare professionals. Biofilm promotes an unfavourable microenvironment that leads to a delay in the evolution and healing of wounds, highlighting the importance and impact of various therapeutic strategies on biofilm disruption.[30] Undoubtedly, mechanical debridement of ulcerated epithelial surfaces in periodontal or peri-implant pockets will also facilitate the disruption and loss of mass of the subgingival biofilm. Consequently, a temporary reduction in bacterial load

will occur, and microbiota will recolonise the lost area and form new biofilm. This role belongs to colonising species resulting in a decrease in the relative load of pathobionts.[24]

Patient empowerment involves a shift in mindset regarding the approach to treating chronic conditions such as periodontitis. In our case, it is the periodontal patients who must decide whether they want to actively participate in the healing of their own subgingival lesions from home, following instruction and with professional supervision. Patients will discover that they too can evaluate inflammatory activity by inserting a brush to the bottom of the lesion and observing the degree of bleeding and discomfort. If these chronic subgingival wounds do not heal, progressive periodontal or peri-implant tissue destruction will come into play, leading to loss of attachment. In any case, once the epithelium has healed, we advise examining the lesion twice per week to check stability and the absence of bleeding. We are currently working on the design of a prototype to facilitate these home treatments as well as their monitoring by the patients themselves.

In view of the limitations detected during the study, we stress the importance of both selecting motivated and committed patients and choosing accessible lesions. Specifically, some patients demonstrated less dexterity in accessing very deep lesions in the posterior areas, especially lingually or palatally. Another aspect to consider is the discomfort some patients experience, particularly in the first few days. It is worth noting, however, that this discomfort gradually diminishes, which is associated with rapid clinical improvement. In extreme cases, initially, it may be advisable to use topical anaesthetics combined with an oral analgesic (ibuprofen) one hour before treatment and to start implementing this procedure only on alternate days. We stress that our participants did not require either of these measures.

The healing process of the ulcerated periodontal or peri-implant epithelium is complex and requires personalized instructions and monitoring by a professional. By carrying out our proposed procedure, we may also be oxygenating this ecological niche and at the same time acting on the debris generated by the wound itself, which hinders healing and may favour the growth of biofilm, especially in the deeper pockets with a higher component of anaerobic periodontopathogens. This, in turn, could ultimately contribute to reduced tissue oxidative stress and promote the improvement of these subgingival chronic wounds. In this context, the message is simple: touch the wound to restore balance and foster repair.

Many clinicians would like patients themselves to be able to monitor the inflammatory activity of certain localized lesions. Educating patients on this proposed home-based treatment could boost the resilience of subgingival oral biofilm associated with periodontal health.

Future research is needed to study this home-based periodontal treatment, distinguishing between active recurrent periodontal and peri-implant pockets. In addition, a larger sample size with a control group would allow for performing linear models and crossing clinical variables with microbiological ones. Lastly, it is worth mentioning that in subsequent studies we will consider the option of introducing some subgingival devices made from materials that facilitate their handling and are comfortable for patients.

Conclusion

The implementation of this subgingival home-based treatment model for active recurrent periodontal or peri-implant pockets, using angled interdental brushes in a vertical position, is inexpensive, appears to be efficient, and could enhance the resilience of oral biofilm associated with periodontal health. The greater relative decrease in pathogenic species compared to commensals suggests the recovery of a microbial community in symbiosis with the host. This conservative procedure reflects a commendable interest in promoting self-care strategies in supportive periodontal therapy. This work also considers the cross-cutting nature of periodontology in the health sciences, particularly in relation to medicine and nursing.

Data Sharing Statement

The data that support the findings of this study are available from the last author lluis.brunet@sjd.es and the corresponding author jmiranda-rius@ub.edu upon reasonable request.

Ethics Approval and Consent to Participate

Ethical approval for the study was obtained by the clinical research and ethics committee of the Sant Joan de Déu Foundation: Internal code. PIC 26-18/187-21. Informed consent was also taken from all participants.

Consent for Publication

All participants provided written informed consent to participate in the study and to have their anonymised data published.

Acknowledgments

We would like to thank Ann Bangle for the English revision of the manuscript.

Author Contributions

All authors made significant contributions to the study, either in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas. All authors took part in drafting, revising or critically reviewing the article and gave final approval of the version to be published. They have all agreed on the choice of the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

Funding

This manuscript received financial support to cover open access publication fees from the competitive 2025 call of the University of Barcelona, "Ajuts Àrees Singulars" (UB-AS-2025-01). The funding was awarded to a research project on periodontal biomarkers led by Prof. Jaume Miranda-Rius (PI) and Prof. José Luis Rosa (co-PI). We have also received funding under call 2026 from the Vice-Rectorate for Research at the University of Barcelona to publish in open-access scientific journals, as part of its policy to support the free dissemination of knowledge.

Disclosure

Drs. Gerard Àlvarez, Vanessa Blanc, and Rubén León are researchers at the Dentaid Research Center. Although they are affiliated with the company DENTAID SL, the manuscript does not mention or depict any specific products. The three authors affirm that DENTAID SL had no influence on the design, data collection, analysis, interpretation or publication of this study. Dr. Vanessa Blanc (Translational Research Director) and Dr. Rubén León (Basic Research Director) report the incurring of departmental costs during the study. The authors declare that they have no competing interests and that no additional financial or non-financial conflicts relevant to this study exist.

References

1. Zhao R, Liang H, Clarke E, Jackson C, Xue M. Inflammation in Chronic Wounds. *Int J Mol Sci*. 2016;17(12):2085. doi:10.3390/ijms17122085
2. Harries RL, Bosanquet DC, Harding KG. Wound bed preparation: TIME for an update. *Int Wound J*. 2016;13(Suppl 3):8–14. doi:10.1111/iwj.12662
3. Torumtay G, Kırzıoğlu FY, Öztürk Tonguç M, Kale B, Calapoğlu M, Orhan H. Effects of periodontal treatment on inflammation and oxidative stress markers in patients with metabolic syndrome. *J Periodontal Res*. 2016;51(4):489–498. doi:10.1111/jre.12328
4. Madianos PN, Bobetsis YA, Offenbacher S. Adverse pregnancy outcomes (APOs) and periodontal disease: pathogenic mechanisms. *J Periodontol*. 2013;84(4 Suppl):S170–S180. doi:10.1902/jop.2013.1340015
5. Ide M, Papapanou PN. Epidemiology of association between maternal periodontal disease and adverse pregnancy outcomes--systematic review. *J Periodontol*. 2013;84(4 Suppl):S181–S194. doi:10.1902/jop.2013.134009
6. Van Dyke TE, van Winkelhoff AJ. Infection and inflammatory mechanisms. *J Clin Periodontol*. 2013;40(Suppl 14):S1–S7. doi:10.1111/jcpe.12088
7. Hussain B, Karaca EO, Kuru BE, Gursoy H, Haugen HJ, Wohlfahrt JC. Treatment of residual pockets using an oscillating chitosan device versus regular curettes alone-A randomized, feasibility parallel-arm clinical trial. *J Periodontol*. 2022;93(5):780–789. doi:10.1002/JPER.21-0496
8. Papapanou PN, Sanz M, Buduneli N, et al. Periodontitis: consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol*. 2018;89 Suppl 1:S173-S182. doi:10.1002/JPER.17-0721
9. Lang NP, Adler R, Joss A, Nyman S. Absence of bleeding on probing. An indicator of periodontal stability. *J Clin Periodontol*. 1990;17 (10):714–721. doi:10.1111/j.1600-051x.1990.tb01059.x
10. Sanz I, Alonso B, Carasol M, Herrera D, Sanz M. Nonsurgical treatment of periodontitis. *J Evid Based Dent Pract*. 2012;12(3 Suppl):76–86. doi:10.1016/S1532-3382(12)70019-2
11. Graziani F, Karapetsa D, Alonso B, Herrera D. Nonsurgical and surgical treatment of periodontitis: how many options for one disease? *Periodontol*. 2017;75(1):152–188. doi:10.1111/prd.12201

12. Shrivastava D, Natoli V, Srivastava KC, et al. Novel Approach to Dental Biofilm Management through Guided Biofilm Therapy (GBT): a Review. *Microorganisms*. 2021;9(9):1966. doi:10.3390/microorganisms9091966
13. Lasserre JF, Brecx MC, Toma S. Oral Microbes, Biofilms and Their Role in Periodontal and Peri-Implant Diseases. *Materials*. 2018;11(10):1802. doi:10.3390/ma11101802
14. Fleischer HC, Mellonig JT, Brayer WK, Gray JL, Barnett JD. Scaling and root planing efficacy in multirooted teeth. *J Periodontol*. 1989;60 (7):402–409. doi:10.1902/jop.1989.60.7.402
15. Boyd LD, Mallonee LF, Wyche CJ. *Wilkins' Clinical Practice of the Dental Hygienist*. 13th ed. Philadelphia, PA, USA: Lippincott Williams and Wilkins; 2021.
16. Lindhe J, Westfelt E, Nyman S, Socransky SS, Heijl L, Bratthall G. Healing following surgical/non-surgical treatment of periodontal disease. A clinical study. *J Clin Periodontol*. 1982;9(2):115–128. doi:10.1111/j.1600-051x.1982.tb01227.x
17. de Oliveira RR, Schwartz-Filho HO, Novaes ABJ, Taba Jr M. Antimicrobial photodynamic therapy in the non-surgical treatment of aggressive periodontitis: a preliminary randomized controlled clinical study. *J Periodontol*. 2007;78(6):965–973. doi:10.1902/jop.2007.060494
18. Serino G, Rosling B, Ramberg P, Socransky SS, Lindhe J. Initial outcome and long-term effect of surgical and non-surgical treatment of advanced periodontal disease. *J Clin Periodontol*. 2001;28(10):910–916. doi:10.1034/j.1600-051x.2001.028010910.x
19. John MT, Michalowicz BS, Kotsakis GA, Chu H. Network meta-analysis of studies included in the Clinical Practice Guideline on the nonsurgical treatment of chronic periodontitis. *J Clin Periodontol*. 2017;44(6):603–611. doi:10.1111/jcpe.12726
20. Graziani F, Karapetsa D, Mardas N, Leow N, Donos N. Surgical treatment of the residual periodontal pocket. *Periodontol 2000*. 2018;76 (1):150–163. doi:10.1111/prd.12156
21. Khan SN, Koldsland OC, Roos-Jansåker AM, et al. Non-surgical treatment of mild to moderate peri-implantitis with an oscillating chitosan brush or a titanium curette-12-month follow-up of a multicenter randomized clinical trial. *Clin Oral Implants Res*. 2023;34(7):684–697. doi:10.1111/clr.14078
22. Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. *Clin Oral Implants Res*. 1992;3(1):1–8. doi:10.1034/j.1600-0501.1992.030101.x
23. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(Suppl 20):S286–S291. doi:10.1111/jcpe.12957
24. Radaic A, Kapila YL. The oralome and its dysbiosis: new insights into oral microbiome-host interactions. *Comput Struct Biotechnol J*. 2021;19:1335–1360. doi:10.1016/j.csbj.2021.02.010
25. Renvert S, Hirooka H, Polyzois I, Kelekis-Cholakis A, Wang HL. Working Group 3. Diagnosis and non-surgical treatment of peri-implant diseases and maintenance care of patients with dental implants - Consensus report of working group 3. *Int Dent J*. 2019;69(Suppl 2):12–17. doi:10.1111/idj.12490
26. Löe H, Silness J. Periodontal disease in pregnancy. I. Prevalence and severity. *Acta Odontol Scand*. 1963;21:533–551. doi:10.3109/00016356309011240
27. Àlvarez G, González M, Isabal S, Blanc V, León R. Method to quantify live and dead cells in multi-species oral biofilm by real-time PCR with propidium monoazide. *AMB Express*. 2013;3(1):1. doi:10.1186/2191-0855-3-1
28. Àlvarez G, Arredondo A, Isabal S, et al. Association of nine pathobionts with periodontitis in four South American and European countries. *J Oral Microbiol*. 2023;15(1):2188630. doi:10.1080/20002297.2023.2188630
29. Pastar I, Stojadinovic O, Yin NC, et al. Epithelialization in Wound Healing: a Comprehensive Review. *Adv Wound Care*. 2014;3(7):445–464. doi:10.1089/wound.2013.0473
30. Perdomo P, Pérez MF, Benítez MD, Ruiz C. The detritus in the healing process and its elimination for a correct preparation of the wound bed. *Gerokomos*. 2018;29(3):141–144.
31. Sultan DA, Hill RG, Gillam DG. Air-polishing in subgingival root debridement: a critical literature review. *J Dent Oral Biol*. 2017;2:1065.
32. Rabbani GM, Ash Jr MM, Caffesse RG. The effectiveness of subgingival scaling and root planing in calculus removal. *J Periodontol*. 1981;52 (3):119–123. doi:10.1902/jop.1981.52.3.119
33. Eaton KA, Kieser JB, Davies RM. The removal of root surface deposits. *J Clin Periodontol*. 1985;12(2):141–152. doi:10.1111/j.1600-051x.1985.tb01373.x
34. Shrivastava D, Srivastava KC, Dayakara JK, et al. Bactericidal Activity of Crevicular Polymorphonuclear Neutrophils in Chronic Periodontitis Patients and Healthy Subjects under the Influence of Areca Nut Extract: an In Vitro Study. *Appl Sci*. 2020;10(14):5008. doi:10.3390/app10145008
35. Villa O, Lyngstadaas SP, Monjo M, et al. Suture materials affect peri-implant bone healing and implant osseointegration. *J Oral Sci*. 2015;57 (3):219–227. doi:10.2334/josnusd.57.219
36. Larsen OI, Enersen M, Kristoffersen AK, et al. Antimicrobial Effects of Three Different Treatment Modalities on Dental Implant Surfaces. *J Oral Implantol*. 2017;43(6):429–436. doi:10.1563/aaid-joi-D-16-00147
37. Roos-Jansåker AM, Renvert H, Lindahl C, Renvert S. Submerged healing following surgical treatment of peri-implantitis: a case series. *J Clin Periodontol*. 2007;34(8):723–727. doi:10.1111/j.1600-051X.2007.01098.x
38. Wohlfahrt JC, Aass AM, Koldsland OC. Treatment of peri-implant mucositis with a chitosan brush-A pilot randomized clinical trial. *Int J Dent Hyg*. 2019;17(2):170–176. doi:10.1111/idh.12381
39. Wohlfahrt JC, Evensen BJ, Zeza B, et al. A novel non-surgical method for mild peri-implantitis- a multicenter consecutive case series. *Int J Implant Dent*. 2017;3(1):38. doi:10.1186/s40729-017-0098-y
40. Costa EM, Silva S, Pina C, Tavaria FK, Pintado MM. Evaluation and insights into chitosan antimicrobial activity against anaerobic oral pathogens. *Anaerobe*. 2012;18(3):305–309. doi:10.1016/j.anaerobe.2012.04.009
41. Choi BK, Kim KY, Yoo YJ, Oh SJ, Choi JH, Kim CY. In vitro antimicrobial activity of a chitooligosaccharide mixture against *Actinobacillus actinomycetemcomitans* and *Streptococcus mutans*. *Int J Antimicrob Agents*. 2001;18(6):553–557. doi:10.1016/s0924-8579(01)00434-4
42. Sarasam AR, Brown P, Khajotia SS, Dmytryk JJ, Madihally SV. Antibacterial activity of chitosan-based matrices on oral pathogens. *J Mater Sci Mater Med*. 2008;19(3):1083–1090. doi:10.1007/s10856-007-3072-z
43. Abusleme L, Dupuy AK, Dutzan N, et al. The subgingival microbiome in health and periodontitis and its relationship with community biomass and inflammation. *ISME J*. 2013;7(5):1016–1025. doi:10.1038/ismej.2012.174
44. Lafaurie GI, Sabogal MA, Castillo DM, et al. Microbiome and Microbial Biofilm Profiles of Peri-Implantitis: a Systematic Review. *J Periodontol*. 2017;88(10):1066–1089. doi:10.1902/jop.2017.170123
45. Kolenbrander PE, London J. Adhere today, here tomorrow: oral bacterial adherence. *J Bacteriol*. 1993;175(11):3247–3252. doi:10.1128/jb.175.11.3247-3252.1993