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CLINICAL AND PATHOGENETIC RATIONALE FOR DIFFERENTIATED TREATMENT OF CHRONIC CATARRHAL GINGIVITIS IN TYPE 2 DIABETES

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Abstract. Objective. To formulate a differentiated approach to chronic catarrhal gingivitis in adults with type 2 diabetes. Materials and methods. A narrative synthesis compared gingivitis definitions, evidence on inflammatory mediators, periodontal diabetes consensus recommendations and adjunctive plaque control research. Findings were assessed by population, clinical endpoint and relevance to treatment selection. Results. The proposed approach separates confirmation of gingivitis from metabolic assessment and links treatment intensity to plaque retention, inflammation, self-care capacity and early response. Three clinical matrices support diagnostic recording, intervention selection and reassessment. Published inflammatory data illustrate the need to distinguish biological association from a treatment indication. Conclusion. Differentiation should strengthen local biofilm control and coordination of diabetes care while reserving additional treatment for a defined clinical need. Persistent inflammation requires renewed assessment of diagnosis and adherence.

Keywords: gingivitis; type 2 diabetes; glycaemic control; biofilm; inflammation; differentiated treatment.

КЛИНИКО ПАТОГЕНЕТИЧЕСКОЕ ОБОСНОВАНИЕ ДИФФЕРЕНЦИРОВАННОГО ЛЕЧЕНИЯ ХРОНИЧЕСКОГО КАТАРАЛЬНОГО ГИНГИВИТА ПРИ САХАРНОМ ДИАБЕТЕ 2 ТИПА

Аннотация. Цель — обосновать дифференцированный подход к лечению хронического катарального гингивита у взрослых с сахарным диабетом 2 типа. Материалы и методы. Проведён аналитический обзор определений гингивита, исследований медиаторов воспаления, рекомендаций по взаимодействию стоматолога и диабетолога и данных о дополнительном контроле биоплёнки. Результаты. Предложено раздельно оценивать состояние пародонта и метаболический статус, а объём лечения определять с учётом налёта, воспаления, возможностей самостоятельной гигиены и раннего ответа. Сформированы три клинические матрицы обследования, выбора вмешательства и повторной оценки. Заключение. Дифференциация лечения предусматривает эффективное удаление биоплёнки и согласование помощи при диабете. Дополнительные средства выбирают по клиническим показаниям; устойчивое воспаление требует пересмотра диагноза и оценки выполнения рекомендаций.

Ключевые слова: гингивит; сахарный диабет 2 типа; гликемический контроль; биоплёнка; воспаление; дифференцированное лечение.

2 ТИП ҚАНДЛИ ДИАБЕТДА СУРУНКАЛИ КАТАРАЛ ГИНГИВИТНИ ТАБАҚАЛАШТИРИБ ДАВОЛАШНИНГ КЛИНИК ПАТОГЕНЕТИК АСОСЛАРИ

Аннотация. Мақсад — 2 тип қандли диабети бўлган катталарда сурункали катарал гингивитни табақалаштириб даволашни асослаш. Материаллар ва усуллар. Гингивит мезонлари, яллиғланиш медиаторлари, диабетда стоматологик ёрдам ва биоплёнка назоратига оид адабиётлар таҳлил қилинди. Натижалар. Пародонт ҳолати ва метаболик назоратни алоҳида баҳолаш, давони эса караш, яллиғланиш, мустақил гигиена имконияти ва дастлабки клиник жавоб асосида танлаш таклиф этилди. Текширув, даво танлаш ва қайта баҳолаш учун учта клиник жадвал шакллантирилди. Хулоса. Табақалаштирилган ёндашув биоплёнкани самарали тозалаш ва диабет бўйича ёрдамни мувофиқлаштиришга таянади. Қўшимча воситалар клиник эҳтиёжга кўра танланади, сақланиб қолган яллиғланишда ташхис ва тавсияларга риоя қилиш қайта баҳоланади.

Калит сўзлар: гингивит; 2 тип қандли диабет; гликемик назорат; биоплёнка; яллиғланиш; табақалаштирилган даволаш.

Introduction

Chronic catarrhal gingivitis describes persistent inflammatory changes of the gingiva, commonly expressed as erythema, oedema and bleeding. In contemporary periodontal terminology, the corresponding plaque-associated condition is assessed within the framework of dental biofilm-induced gingivitis. The term should therefore be linked to a documented periodontal examination rather than used as a diagnosis based solely on visual redness. Establishing whether the supporting tissues are intact determines the subsequent treatment pathway [1].

Type 2 diabetes adds a second dimension to this assessment. The clinician needs to know whether metabolic follow-up is current, whether the patient experiences symptoms that interfere with daily oral care and whether the treatment plan can be integrated into existing medical routines. The diabetes periodontal consensus supports cooperation between dental and medical teams [2]. This cooperation is most useful when the referral conveys a specific clinical question and the response informs a practical decision.

Differentiated treatment is often interpreted as the addition of several products for patients with systemic disease. A more testable interpretation is to identify the factor preventing resolution and choose an intervention that addresses it. A retained deposit, an inaccessible interproximal surface, poor understanding of brushing technique and an unresolved metabolic concern require different actions. Recording these actions makes the clinical rationale visible and permits evaluation at review.

The aim of this article is to develop a clinically grounded treatment framework for chronic catarrhal gingivitis in adults with type 2 diabetes. The specific objectives are to connect pathogenetic evidence with clinical assessment, define the roles of basic and additional interventions, and establish criteria for reassessment without conflating gingivitis with destructive periodontal disease.

Materials and methods

A targeted narrative review was undertaken using PubMed-indexed literature and professional periodontal guidance available by 26 October 2026. Search concepts included gingivitis, type 2 diabetes, inflammatory mediators, glycaemic control, mechanical plaque removal and adjunctive chlorhexidine. Publications were selected for their contribution to a defined decision: establishing the diagnosis, explaining susceptibility, choosing local treatment or interpreting response. The synthesis was designed to formulate a clinical framework rather than to calculate a pooled treatment effect.

Evidence was separated into three categories. Direct evidence concerned gingivitis in people with type 2 diabetes. Indirect clinical evidence concerned gingivitis management in broader populations. Mechanistic literature on diabetes-associated periodontitis was used to frame biological plausibility, with its disease context retained. Findings from these categories were not treated as interchangeable. In particular, a reduction in glycated haemoglobin after periodontal treatment cannot be assumed to describe the effect of treating gingivitis alone.

For each selected publication, the population, periodontal definition, measurement setting and endpoint were considered. Cross-sectional differences were used to describe associations. Intervention estimates were interpreted within the original comparator and follow-up period. Diagnostic thresholds were distinguished from therapeutic targets, and systemic inflammatory markers were distinguished from measures obtained from gingival crevicular fluid.

The resulting matrices were constructed around observable clinical findings. Each proposed action was paired with a reason and an outcome that could be reassessed. Numerical material reproduced from a published study is presented as an illustration of biological association; no new pooled estimate was calculated. This approach allows the framework to be examined clinically while preserving the boundaries of the underlying evidence.

Results

Clinical definition and metabolic context

The first result of the synthesis is a two-part assessment structure. The periodontal record establishes the local diagnosis; the medical record describes the context in which treatment will be delivered. For an intact periodontium, bleeding on probing at 10% or more of sites supports a gingivitis case definition, with 10–30% indicating localized and more than 30% generalized involvement [1]. These percentages do not grade diabetes severity and should not be used to infer attachment loss.

A baseline chart should preserve site-level information. A single whole-mouth score can conceal persistent inflammation around a restoration or in an inaccessible posterior region. Recording the distribution of plaque and bleeding permits the clinician to test whether subsequent improvement follows successful cleaning at the same sites. Probing findings, attachment levels and radiographic assessment when indicated determine whether a periodontitis pathway is required.

Metabolic information should include the date of the most recent HbA1c result and the target agreed with the treating clinician. An undated value has limited value for present-day planning. Diabetes duration, medication history, hypoglycaemic episodes and the practical demands of treatment help explain how a patient can participate in care. The proposed record therefore documents both biological context and the ability to carry out the oral hygiene plan.

Table 1. Baseline information and its role in treatment planning

Assessment	Record	Decision supported
Periodontal status	Bleeding sites, probing, attachment	Confirm gingivitis or redirect care
Biofilm retention	Plaque distribution and local obstacles	Select sites for professional cleaning
Metabolic context	Dated HbA1c and medical follow-up	Coordinate diabetes care
Self-care capacity	Demonstrated technique and access	Choose feasible cleaning aids
Symptoms	Bleeding, discomfort and dryness	Identify barriers and differential diagnoses

Inflammatory pathways and their clinical meaning

Direct evidence supports consideration of the host response. In a cross-sectional study of lifelong non-smokers, Andriankaja and colleagues examined gingivitis and type 2 diabetes alongside local and systemic inflammatory mediators [3]. Within the diabetic group, mean serum IL-6 was higher in participants with gingivitis than in those with gingival health. Figure 1 presents the reported means. These observations concern an association; they do not establish an IL-6 threshold for prescribing a particular drug.

Mechanistic literature identifies advanced glycation end products and their receptor pathway, altered inflammatory signalling and impaired repair as plausible contributors to diabetes-associated periodontal disease [4]. Much of this evidence concerns periodontitis. Its contribution to the present framework is to explain why a similar local challenge may coexist with a different host response, while avoiding the assumption that every inflamed gingival site is undergoing irreversible destruction.

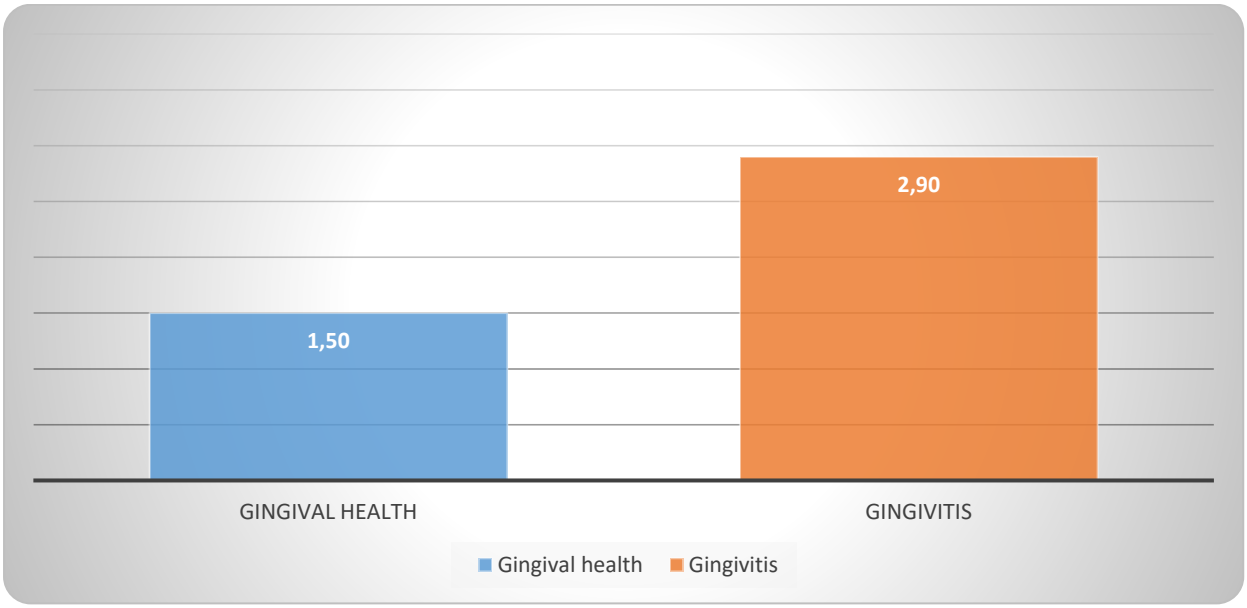


Figure 1. Serum IL-6 in non-smoking participants with type 2 diabetes by gingival status. Published means were 1.5 and 2.9 pg/mL; corresponding standard deviations were 1.4 and 3.2 pg/mL [3]. Bars display means only. The comparison is cross-sectional and is not a before-and-after treatment result.

The biological rationale changes how persistent inflammation is investigated. When bleeding remains after the initial intervention, the clinician can compare the residual plaque pattern, the accuracy of the original diagnosis and the current metabolic information. Automatically escalating antimicrobial treatment would leave these alternative explanations untested. Conversely, attributing all bleeding to diabetes could allow a correctable local factor to persist.

Laboratory markers may help investigate mechanisms in a structured research setting, but a proposed clinical panel needs a decision attached to each test. A result that does not alter diagnosis, management or monitoring adds complexity without clarifying treatment. Local sampling also requires control of collection site and contamination. The framework therefore retains clinical endpoints as the principal measures of resolution and assigns biomarkers a supporting explanatory role.

Differentiation of the initial intervention

Mechanical control remains the foundation of the treatment plan. The prevention consensus places sustained plaque removal at the centre of gingival care [5]. Professional guidance similarly recommends personalized instruction, removal of deposits where needed and correction of plaque-retentive factors [6]. In the proposed framework, differentiation determines where effort is concentrated and which obstacles are addressed first.

For a patient with accessible deposits and a workable home-care technique, the main task is to remove local obstacles and consolidate the routine. Where plaque persists interproximally, the clinician should observe actual use of the chosen aid. A verbal statement that the patient cleans daily cannot reveal whether the device reaches the affected surface. Demonstration and return demonstration provide a clearer basis for modifying the plan.

If the patient reports difficulty incorporating oral care into diabetes treatment routines, the plan should be simplified around achievable actions. Selecting multiple devices at the same visit may make adherence harder to evaluate. The proposed approach starts with a feasible routine, verifies the technique and adds another component only when a specific access problem remains. This is a practical implementation choice rather than a separate pharmacological treatment category.

Table 2. Proposed differentiation according to the dominant barrier

Clinical pattern	Priority action	Review question
Plaque at accessible sites	Demonstration and targeted cleaning	Has the same-site plaque burden fallen?
Retention around restorations	Correct the local obstacle	Can the patient clean the margin?
Difficulty with daily care	Adapt tools and simplify the routine	Can the technique be demonstrated?
Unclear metabolic follow-up	Communicate with the treating clinician	Is an updated medical plan available?
Persistent bleeding after care	Reassess diagnosis and site conditions	Is there attachment loss or another lesion?

Selection of additional treatment

Additional plaque control should answer a defined need. Antiseptic use can be considered when effective mechanical care is temporarily limited, but the decision should specify the intended purpose and review date. A Cochrane review found a small adjunctive Gingival Index reduction with chlorhexidine in populations with mild inflammation; the reviewers judged this change not clinically relevant [7]. These results do not establish a diabetes-specific response or justify treating an elevated HbA1c value with a mouthrinse.

Short-term prescribing guidance is relevant when chlorhexidine is selected for an appropriate indication [8]. Product choice should account for tolerability, the ability to rinse safely and the instructions applicable to the formulation. A planned endpoint prevents an initial short course from becoming an unreviewed permanent part of care. Persistent symptoms after the course call for examination of the underlying problem.

The same reasoning applies to proposed antioxidant or host-modulating preparations. A plausible pathway is a rationale for investigation, but it does not identify a clinically effective product, dose or duration. The present framework therefore does not create a routine multi-drug regimen from mechanistic findings. Systemic antimicrobial therapy is not part of the proposed pathway for uncomplicated plaque-induced gingivitis. A separate infectious diagnosis would require its own assessment and treatment justification.

Reassessment and continuing care

Reassessment should distinguish three outcomes: the local clinical response, the feasibility of daily self-care and the status of medical coordination. Improvement in one domain does not prove improvement in the others. The patient may report easier cleaning before all bleeding resolves, while a newer HbA1c

result may arrive after the gingival review. Keeping these observations separate helps avoid inaccurate attribution of benefit.

A planned early review after completion of the initial phase can use the same recording method and comparable sites. The precise timing should reflect symptoms, the intervention delivered and the patient's ability to maintain care. Continuing recall is then individualized according to residual inflammation and ongoing treatment need, consistent with risk-based periodontal planning [9]. A fixed interval selected solely because diabetes is present would disregard these clinical differences.

Table 3. Interpretation of findings at reassessment

Observed response	Interpretation to examine	Management implication
Plaque and bleeding decrease	Local control is improving	Reinforce the feasible routine
Plaque persists with bleeding	Access or technique remains inadequate	Repeat demonstration and correct obstacles
Low visible plaque with bleeding	Residual deposits or an alternative diagnosis	Repeat site-level examination
New attachment loss	Disease extends beyond gingivitis	Reclassify and plan periodontal treatment
Local improvement with unchanged HbA1c	Oral and metabolic outcomes differ	Continue medical follow-up separately

Treatment records should specify what changed between visits. If the technique, cleaning device, professional intervention and medical therapy all change simultaneously, the contribution of any one component becomes difficult to interpret. Routine care can still improve, but claims about a particular adjunct should remain proportionate to the information collected. Recording treatment exposure and adherence is therefore part of the evaluation rather than an administrative addition.

Discussion

The proposed framework organizes treatment around a clinical question: what prevents this patient's gingival inflammation from resolving? This question connects pathogenesis to action without converting every biological association into a prescription. Diabetes may influence susceptibility and repair, whereas plaque retention and daily cleaning determine the local challenge that can be addressed directly. A useful differentiated plan records both dimensions and revisits their interaction after treatment.

The diagnostic boundary is especially consequential. A swollen gingival margin may increase probing depth without equivalent loss of attachment, while a patient with a history of periodontitis may have a reduced periodontium even when current probing depths are shallow. Neither situation can be adequately described by the word catarrhal alone. Clinical classification should therefore be stated alongside the traditional terminology used in the presenting diagnosis.

Another issue is the interpretation of inadequate response. An apparent failure of standard care may reflect an intervention that was not delivered consistently, an aid that the patient cannot use, or a local factor that remained untreated. The matrices make these alternatives reviewable. They also provide a reason to change the plan when a new finding emerges rather than repeating an identical intervention indefinitely.

The framework is a narrative clinical synthesis. It does not estimate the comparative effectiveness of the proposed branches, and the selected evidence differs in population and design. The inflammatory comparison is cross-sectional, the adjunctive mouthrinse evidence is not specific to type 2 diabetes, and mechanistic studies often concern periodontitis. These differences delimit the conclusions: the synthesis

supports a reasoned structure for care, while the superiority of the complete algorithm requires prospective comparison.

For such a comparison, an informative design would predefine the periodontal case definition, record metabolic context and compare clinically feasible care pathways using the same follow-up methods. Bleeding and plaque should be primary oral outcomes; patient-reported comfort and ability to maintain hygiene would complement them. HbA1c could be recorded as a separate metabolic outcome, with changes in medical treatment documented. This design would evaluate whether differentiation improves care rather than merely increasing the number of interventions.

Conclusion

Differentiated treatment of chronic catarrhal gingivitis in type 2 diabetes should begin with a verified periodontal diagnosis and a dated assessment of metabolic context. The proposed framework links local therapy to plaque retention, self-care capacity and the response observed at review. Mechanical biofilm control remains the common foundation, while additional measures are selected for a documented clinical purpose. Persistent inflammation prompts reassessment of site conditions, adherence and diagnosis. The framework preserves the distinction between local resolution, biomarker associations and metabolic outcomes.

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