SALIVARY RISK MARKERS IN THE PERSONALIZED ASSESSMENT OF DENTAL CARIES IN CHILDREN WITH DIABETIC POLYNEUROPATHY

Teshayeva Nozigul Hamidullayevna
Assistant at the Department of Dentistry, Asia International University
ORCID: 0009-0008-9884-8531 |
E-mail: nozigulteshayeva@gmail.com
https://doi.org/10.5281/zenodo.22702353

Relevance

Saliva is a major determinant of the oral environment. It clears fermentable substrates, dilutes and buffers bacterial acids, lubricates mucosal and dental surfaces, and supplies calcium and phosphate for enamel repair. When flow is reduced or buffering is insufficient, the period during which plaque pH remains below the critical range may lengthen. These changes do not cause caries independently, but they may shift the balance toward net mineral loss when active biofilm and frequent carbohydrate exposure are also present [2,6].

Keywords: saliva, dental caries, diabetic polyneuropathy, children, pH, buffering capacity, Streptococcus mutans

Studies in children with type 1 diabetes have reported changes in salivary flow and composition, yet the direction and magnitude of these findings are not fully consistent [3,7]. Hydration, recent food intake, time of day, medication, metabolic control and sample stimulation can all alter a result. Consequently, a salivary value is clinically meaningful only when pre-analytical conditions are standardized and the result is linked with lesion stage, oral hygiene, diet and the child's medical history.

A personalized risk profile should not depend on a single marker. Physical parameters describe the quantity and handling characteristics of saliva; chemical variables describe acid neutralization and mineral availability; microbiological tests estimate the cariogenic challenge; and immune or oxidative markers characterize the local response. Their combined pattern can explain why apparently similar enamel lesions behave differently over time.

Aim

To define a standardized panel of salivary variables and an integrated interpretation pathway for individualized caries-risk assessment in children with diabetic polyneuropathy.

Materials and methods

The prospective controlled study includes 130 children aged 7-15 years examined in Bukhara during 2022-2026: 85 children with dental caries associated with diabetic polyneuropathy and 45 healthy controls. The clinical

examination includes DMFT/DMFS, OHI-S and ICDAS II. The saliva panel is used to characterize the biological environment of the lesions and not as an independent diagnostic substitute.

Samples are collected between 08:00 and 11:00. For at least two hours before collection, children do not eat, drink, brush their teeth or use a mouth rinse. The child sits quietly with the head slightly lowered. Unstimulated whole saliva is expectorated into a sterile graduated tube at one-minute intervals for 5-10 minutes. Flow rate is calculated as collected volume divided by collection time. Stimulated saliva, when required, is obtained and interpreted separately because the two procedures are not interchangeable.

Table 1. Salivary variables included in the personalized risk panel

Domain	Variables	Clinical meaning	Main source of variation
Physical	Flow rate, viscosity	Clearance and lubrication	Hydration, stimulation, time of day
Acid-base	pH, buffering capacity	Neutralization of plaque acids	Recent diet and collection conditions
Mineral	Ionized Ca, inorganic P, Ca/P	Potential for enamel remineralization	Diet, pH and analytical method
Enzymatic/oxidative	ALP, ACP, MDA, SOD, catalase	Tissue turnover and redox response	Storage and processing delay
Microbiological	S. mutans, Lactobacillus, CFU/ml	Cariogenic biological challenge	Sampling site and recent hygiene
Immune	Secretory IgA	Local immune defence	Age, infection and assay variability

Chairside measurements of pH, viscosity and buffering capacity are performed without unnecessary delay. Samples intended for laboratory analysis are centrifuged at 3000 rpm for 10-15 minutes. The supernatant is used for

biochemical and immunological measurements, while the sediment may be used for microbiological assessment. The time from collection to processing, temperature and storage conditions are documented because uncontrolled handling can obscure real biological differences.

The biochemical panel comprises ionized calcium, inorganic phosphorus, the Ca/P ratio, alkaline and acid phosphatases, malondialdehyde, superoxide dismutase and catalase. Streptococcus mutans and Lactobacillus are quantified as colony-forming units per millilitre, and secretory IgA is determined by immunoassay. Each result is interpreted against the method-specific laboratory range and in relation to the child's clinical findings; universal thresholds should not be assumed where the study method has not validated them.

Integrated interpretation

Reduced flow weakens clearance and prolongs substrate contact. Low pH and limited buffering reduce the capacity to neutralize acids formed in the biofilm. An unfavourable calcium-phosphate pattern may restrict remineralization, whereas elevated cariogenic bacterial counts indicate a stronger biological challenge. None of these findings is specific in isolation. Their importance increases when they occur together and coincide with active ICDAS 1-2 lesions or unsatisfactory oral hygiene.

A relatively low-risk pattern may include stable or inactive early lesions, satisfactory OHI-S, adequate flow and buffering, and no marked microbiological burden. A moderate-risk pattern is considered when one or two biological domains are altered but lesion activity remains limited. A high-risk pattern is suggested by clustering: active noncavitated lesions, poor plaque control, reduced flow, acidic pH, weak buffering and increased cariogenic microorganisms. The risk category should remain open to revision after improved hydration, hygiene instruction or repeat standardized collection.

Quality control and statistical analysis

The pre-analytical checklist is completed for every sample. If clinical and laboratory findings are discordant, the first step is to examine collection time, fasting interval, hydration, stimulation, tube identification and processing delay. Recollection under the same conditions is preferable to building a clinical decision on a doubtful specimen. Duplicate measurements and periodic calibration are appropriate for variables with substantial analytical variation.

Data are processed in SPSS 23. Distributional assumptions are checked before choosing parametric or non-parametric comparisons. Continuous salivary variables are summarized with means and standard deviations or medians and

interquartile ranges, as appropriate. Associations with ICDAS II, DMFT/DMFS and OHI-S can be assessed with correlation or regression methods suited to the scale and distribution of the variables. Multivariable interpretation is preferable because age, sex, hygiene and metabolic factors may confound simple bivariate associations.

Discussion

Salivary diagnostics are attractive because collection is non-invasive and can be repeated during follow-up. However, ease of collection should not be confused with ease of interpretation. The same pH value may have different meaning in a child with adequate flow and good hygiene than in a child with low flow, active white-spot lesions and frequent fermentable carbohydrate intake. For that reason, the proposed panel is organized by functional domain and connected to the clinical examination.

Oxidative and immune markers may provide insight into the local response to systemic disease, but they are best treated as complementary research variables until robust age- and method-specific thresholds are established. In everyday clinical care, flow rate, pH, buffering, plaque status and lesion activity are likely to carry the clearest immediate implications. The broader panel can then clarify mechanisms and support individualized monitoring.

Practical significance

A standardized salivary protocol helps the dentist distinguish a transient laboratory fluctuation from a coherent high-risk pattern. It also makes repeat measurements at 3, 6 and 12 months more comparable. The resulting profile can guide the intensity of hygiene support, dietary counselling, remineralization and recall without labelling every child with diabetic polyneuropathy as uniformly high risk.

Interpretive safeguards

Risk classification should be reviewed whenever the clinical situation changes. Fever, acute infection, dehydration, a new medication or a marked change in glycaemic management can influence saliva and may justify postponing collection. Recording these circumstances prevents a temporary systemic event from being interpreted as a stable oral characteristic. The same principle applies to recent professional cleaning or antimicrobial use, which may alter microbiological counts.

Laboratory precision and clinical usefulness are related but not identical. A statistically different biomarker may have little practical value if the ranges of individual children overlap substantially or if the result does not change

management. Therefore, the analytical plan should report effect size and uncertainty in addition to significance testing, and it should assess whether a combined profile improves classification beyond readily available clinical indicators.

The child and caregiver should receive a short explanation of the findings in practical language. Instead of presenting isolated biochemical values, the dentist can explain whether saliva currently provides adequate clearance and acid neutralization and which daily action is expected to improve the risk profile. This translation from laboratory data to behaviour is essential if salivary assessment is to support prevention rather than remain only a research procedure.

Conclusion

A combined salivary panel can strengthen personalized caries-risk assessment in children with diabetic polyneuropathy when collection and processing are standardized. Flow, pH, buffering, calcium-phosphate balance, oxidative and enzymatic markers, cariogenic microorganisms and secretory IgA should be interpreted together with ICDAS II, DMFT/DMFS, OHI-S and longitudinal lesion behaviour. The most reliable conclusion arises from a consistent clinical-biological pattern rather than any isolated result.

References:

1. World Health Organization. Oral Health Surveys: Basic Methods. 5th ed. Geneva: WHO; 2013.
2. Pitts NB, Zero DT, Marsh PD, et al. Dental caries. Nat Rev Dis Primers. 2017;3:17030. doi:10.1038/nrdp.2017.30.
3. Ismail AF, McGrath CP, Yiu CKY. Oral health of children with type 1 diabetes mellitus: a systematic review. Diabetes Res Clin Pract. 2015;108(3):369-381.
4. Wang Y, Xing L, Yu H, Zhao L. Prevalence of dental caries in children and adolescents with type 1 diabetes. BMC Oral Health. 2019;19:213.
5. Pitts NB, Ekstrand KR. ICDAS and ICCMS: methods for staging and managing the caries process. Community Dent Oral Epidemiol. 2013;41(1):e41-e52.
6. Alamoudi A, Alamoudi R, Gazzaz Y, Alqahtani AM. Role of salivary biomarkers in diagnosis and detection of dental caries. Diagnostics. 2022;12(12):3080.
7. Ferizi L, Dragidella F, Spahiu L, et al. The influence of type 1 diabetes mellitus on dental caries and salivary composition. Int J Dent. 2018;2018:5780916.
8. American Academy of Pediatric Dentistry. Caries-risk assessment and management for infants, children, and adolescents. The Reference Manual of Pediatric Dentistry. Chicago: AAPD; 2025:325-331