Asian Journal of Periodontics and Orthodontics
2026, Volume 6, Issue 2, Page No:73-84
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Available online at: www.tsdp.net

ISSN: 3062-3499

Original Article

Interpreting Discordant Clinical Signals during Supportive Periodontal Care: An Epistemic Framework for Distinguishing Residual Damage, Recurrent Inflammation, and Evidence Requiring Treatment Escalation

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Received: 29 July 2026; **Revised:** 1 September 2026; **Accepted:**4 September 2026

ABSTRACT

Supportive periodontal care frequently generates clinical findings that do not point in the same direction. Residual probing depths may persist after successful treatment, bleeding may appear without demonstrable structural progression, radiographic defects may record historical tissue loss rather than current destruction, and prognostic tools may indicate elevated future risk despite apparently stable findings at the present visit. Treating any one of these signals as a direct measure of active disease risks both unnecessary intervention and delayed recognition of recurrence. This original non-empirical epistemic and clinical reasoning analysis examines the evidentiary status of clinical, radiographic, biological, and prognostic signals used during supportive periodontal care. It distinguishes observation from inference, residual damage from ongoing inflammatory activity, and risk prediction from evidence of current progression. The analysis indicates that discordant findings are most defensibly interpreted by identifying what each signal can actually establish, locating it within the patient's post-treatment trajectory, examining reproducibility and corroboration, and separating historical disease burden from evidence of current change. Residual abnormalities increase concern under some conditions but do not independently establish renewed destructive disease. Escalation becomes more defensible when persistent or worsening findings survive measurement scrutiny, fit a plausible biological and temporal pattern, and converge with other clinically relevant evidence. Supportive periodontal care requires epistemic adjudication rather than automatic conversion of abnormal findings into treatment indications. Clinical accountability is strengthened when uncertainty and rival explanations remain visible until sufficient evidence supports a change in management.

Keywords: Supportive periodontal care, Periodontitis, Residual periodontal pockets, Bleeding on probing, Periodontal diagnosis, Clinical reasoning

How to Cite This Article: Nielsen F, Holm L, Sørensen M. Interpreting Discordant Clinical Signals during Supportive Periodontal Care: An Epistemic Framework for Distinguishing Residual Damage, Recurrent Inflammation, and Evidence Requiring Treatment Escalation. Asian J Periodont Orthodont. 2026;6(2):73-84. https://doi.org/10.51847/XF6al87wMC

Introduction

Supportive periodontal care is commonly described as surveillance after active treatment, yet the clinical task is more demanding than repeated measurement. At each review, the clinician encounters observations that refer to different biological processes and different periods of the patient's disease history. Probing depth is influenced by current tissue conditions and local anatomy; bleeding reflects inflammatory susceptibility but is not identical to progressive attachment loss; radiography primarily records mineralized structural change; and newer biomarker or digital approaches attempt to provide additional biological or predictive information. Contemporary periodontal diagnosis therefore increasingly depends on combining rather than simply accumulating diagnostic information [1]. The central problem is not scarcity of signals but

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deciding what a signal legitimately permits the clinician to believe.

The contemporary classification of periodontal diseases gives this reasoning a structured background. Experience with the 2018 classification demonstrates both its clinical utility and the practical variability that arises when staging and grading are implemented across settings [2]. Staging describes accumulated severity and treatment complexity, while grading incorporates evidence or expectations concerning progression and risk [3]. These categories organize clinically important information, but neither stage nor grade should be converted into a real-time detector of destructive activity at a particular site. A patient can carry the historical consequences of severe disease while exhibiting good present control, and a patient with apparently reassuring measurements can still have a risk profile that warrants closer longitudinal attention. This distinction becomes especially important in the treated, reduced periodontium. Periodontal health can be defined on a reduced periodontium, including one produced by successful periodontal treatment [4]. Consequently, persistent attachment loss or radiographic bone loss cannot by itself demonstrate recurrence: those findings may simply be durable records of previous disease. Conversely, apparent stability cannot be inferred from the absence of dramatic structural change at a single visit because clinically meaningful progression may be slow, localized, or initially expressed through other signals. Supportive care therefore involves an asymmetry between what is easy to observe and what must be inferred. Historical damage is often directly visible, whereas active destructive change must usually be reconstructed from timing, repetition, corroboration, biological plausibility, and longitudinal comparison.

This article treats that inferential task as an epistemic problem in clinical periodontology. Its purpose is not to introduce a numerical decision score or to replace established periodontal guidelines. Instead, it asks how clinicians should interpret discordant evidence while avoiding three recurrent category errors: equating residual abnormality with recurrent disease, equating risk prediction with evidence of current activity, and equating an interpretable abnormal signal with a validated indication for treatment escalation. The analysis first clarifies what common periodontal indicators can and cannot establish, then distinguishes residual damage from current disease activity, and finally examines how disagreement among clinical and prognostic signals should be adjudicated before management is intensified.

Epistemic status of periodontal clinical indicators

Clinical periodontal measurements are essential because they provide direct access to features that cannot be adequately reconstructed from history or imaging alone. Their apparent concreteness, however, can encourage stronger inferences than the measurement supports. Probing depth, clinical attachment measures, bleeding responses, mobility, and related observations are produced through an interaction among anatomy, tissue condition, examiner technique, instrument positioning, local accessibility, and the biological state of the site. Systematic evaluation of periodontal diagnostic methods confirms the continued importance of clinical assessment while also demonstrating method-dependent limitations [5]. A probing measurement is therefore best understood as an observation generated under defined clinical conditions, not as a direct reading of a hidden variable called “active periodontitis.”

This distinction is particularly consequential after periodontal therapy. A deep probing measurement may represent persistent pocket anatomy, altered gingival margin position, incomplete resolution of inflammation, recurrent inflammation, or a site undergoing genuine renewed breakdown. The numerical depth does not specify which interpretation is correct. Bleeding on probing has a different evidentiary character. It can signal local inflammatory activity and may influence concern when persistent, but its biological meaning is not interchangeable with attachment loss or radiographic progression. Absence of bleeding may be reassuring in some contexts, yet it does not erase the patient’s historical susceptibility or guarantee future stability. The epistemic role of the clinical examination is thus to constrain possible explanations rather than to determine the disease state automatically.

Radiographic evidence operates on another temporal and biological layer. Imaging is indispensable for assessing alveolar bone architecture, defect morphology, previous loss of support, and interval structural change. Systematic assessment of ionizing and non-ionizing imaging approaches shows that diagnostic accuracy varies with modality, target condition, and method [6]. A radiograph obtained at one visit is principally a representation of structure at that time. It cannot usually specify when the observed bone loss occurred, whether inflammatory activity is present today, or whether a site with a persistent defect is currently stable. Serial imaging can strengthen inference when technically comparable examinations demonstrate credible change, but even then the image

is evidence of structural change rather than an autonomous explanation of why that change occurred.

Biological markers promise to narrow this gap between observable structure and active pathobiology, but their interpretive reach remains bounded. Host-response markers have shown diagnostic potential across studies, while differences in assays, thresholds, populations, sampling conditions, and reference standards limit direct translation into a universal stand-alone test [7, 8]. Microbial evidence has analogous limitations: detection of organisms or microbial patterns may be biologically relevant without proving that destructive disease is occurring at the sampled site at that moment. These methods can therefore enrich a clinical argument, particularly when they corroborate or challenge conventional observations, but they do not remove the need to reason about temporal sequence and alternative explanations.

Digital technologies add another layer of potential evidence. Imaging analytics, digital measurement systems, and computational approaches may improve screening, reproducibility, pattern recognition, or prognosis, yet their clinical meaning depends on the quality of their reference standards, calibration, external validation, and implementation context [9]. The same caution applies to conventional treatment endpoints. Periodontal treatment success cannot be reduced to a single clinical measure because endpoints such as pocket closure, bleeding control, attachment response, and tooth retention capture different dimensions of outcome [10]. A useful epistemic distinction follows: an indicator should be judged first according to the phenomenon it measures, second according to the time horizon to which it refers, and third according to the strength of inference required to move from that observation to a disease-state claim.

This distinction matters operationally because supportive care routinely places unlike forms of evidence beside one another. A residual probing depth, absent bleeding, unchanged radiographic architecture, elevated historical risk, and a model-generated prognosis are not five independent votes on a common scale. They refer to different constructs. The clinician should therefore resist a simple accumulation model in which the largest number of abnormal signals determines the conclusion. The relevant question is whether the observed findings converge on the same biological and temporal explanation.

Table 1 organizes this difference between the clinical signal that is observed and the stronger conclusion that would require additional evidence.

Table 1. Evidentiary reach of common periodontal signals during supportive care

Clinical or investigative signal	What the observation can reasonably establish	What it cannot establish by itself	Important source of interpretive variation	Longitudinal evidence that would strengthen interpretation	Principal epistemic error to avoid
Probing depth	Current measurable sulcus/pocket depth at an examined site	Whether the depth represents residual anatomy, active recurrence, or future progression	Probe angulation, force, local anatomy, inflammation, access, examiner variability	Reproducible change across comparable examinations, particularly with corroborating attachment or inflammatory findings	Treating one depth measurement as direct evidence of active destruction
Bleeding on probing	A bleeding response associated with local inflammatory conditions	Definite attachment loss, bone loss, or inevitable progression	Probing technique, tissue condition, smoking-related response modification, local plaque control	Persistence or change when interpreted with probing, attachment, hygiene and structural information	Equating inflammation with demonstrated destructive progression
Clinical attachment level	Current relationship between attachment reference points and the gingival margin	Timing or cause of previously accumulated attachment loss	Gingival-margin change, measurement reproducibility, tooth anatomy	Credible interval deterioration on standardized reassessment	Treating historical attachment loss as present activity
Radiographic bone loss	Existing mineralized structural loss and defect morphology visible with the selected method	Whether the defect is currently inflamed or when historical loss occurred	Projection geometry, image quality, modality, detectability of small changes	Technically comparable serial evidence of new structural loss	Reading a static structural defect as a real-time activity marker

Plaque or local self-care findings	Current local exposure relevant to inflammatory control	That destructive recurrence has already occurred	Accessibility, behavior, restorations, dexterity, recall interval	Persistent exposure accompanied by inflammatory or structural deterioration	Converting a risk exposure into a disease diagnosis
Host-response biomarker	Biological information associated with inflammatory or destructive processes	A universally validated site-level diagnosis or treatment threshold	Assay choice, sampling, thresholds, patient phenotype, systemic context	Reproducible validated change with clinical concordance	Treating assay positivity as an autonomous treatment indication
Microbial marker	Presence or relative abundance of selected microbial features	That detected organisms are causing current tissue breakdown	Sampling location, analytical method, ecological variation, prior treatment	Persistent clinically concordant microbial change in validated settings	Equating microbial detection with active destructive disease
Digital or algorithmic output	A derived classification, pattern, or prediction within the model's validated task	Causation, universal transportability, or automatic need for escalation	Training data, calibration, reference standard, case mix, external validation	Stable performance in relevant populations plus concordant longitudinal clinical evidence	Transferring clinical judgment to an unvalidated model

Distinguishing residual damage from active disease

The most consequential interpretive error in supportive periodontal care is to treat persistent abnormality as synonymous with recurrent disease. Periodontitis leaves durable anatomical consequences even when inflammatory control has been achieved. A successfully treated patient may therefore continue to have reduced attachment support, altered bone architecture, recession, furcation involvement, or sites that remain deeper than those in an intact periodontium. Evidence synthesized across supportive-care cohorts shows that clinical stability and long-term tooth retention are achievable after treatment, while also confirming substantial variation among patients and sites [11]. Stability is consequently not the restoration of an undamaged anatomy; it is a conditional post-treatment state in which previous damage persists without sufficient evidence of continuing destructive change.

Time is critical to this distinction. Post-treatment endpoints have prognostic relevance over extended supportive-care follow-up, but their interpretation depends on when and how they are measured. Long-term observational evidence, systematic assessment of re-evaluation timing, and pooled evidence concerning pocket closure and residual pockets collectively show that post-treatment findings should be interpreted as trajectories rather than isolated labels [12–14]. A residual pocket observed soon after instrumentation, a persistent pocket that remains clinically unchanged over repeated maintenance visits, and a pocket that becomes progressively deeper while bleeding and attachment findings deteriorate are not epistemically equivalent. They may share a numerical probing depth while representing different biological histories.

The classification of periodontitis reinforces this need to preserve temporal distinctions. Case definitions identify periodontitis through accumulated evidence of attachment loss and its distribution [15]. Once that historical disease state has been established, supportive-care interpretation should not repeatedly "rediscover" active disease merely because the patient still exhibits its structural consequences. The stage I–III treatment guideline instead places reassessment and supportive care within a stepwise therapeutic process in which response to treatment and residual conditions influence subsequent management [16]. The relevant question is therefore not whether the patient still looks as though periodontitis once occurred—the answer often remains visibly yes—but whether current evidence indicates inadequate control, renewed inflammatory activity, measurable progression, or a risk state that justifies modifying care.

A similar distinction is needed in more complex disease. Stage IV periodontitis may involve severe attachment loss, tooth loss, functional problems, migration, occlusal consequences, and multidisciplinary treatment needs [17]. These features increase the consequences of interpretive error, but they do not justify lowering the evidentiary standard until every residual abnormality becomes proof of recurrence. A heavily reduced dentition can be clinically stable; conversely, apparently modest inflammatory findings may deserve heightened attention when they occur within a vulnerable functional configuration. Complexity therefore

changes the consequences and context of interpretation without eliminating the distinction between historical destruction and current activity.

An epistemically defensible assessment should consequently ask several questions in sequence. Is the finding new, persistent, or merely newly observed? Was it measured comparably to previous examinations? Does it correspond to current inflammatory evidence? Is structural deterioration demonstrable rather than presumed? Does the pattern fit local anatomy or known post-treatment morphology? Is there a plausible alternative explanation, such as measurement variation, transient inflammation, restorative access difficulty, or inconsistent self-care? Only after these possibilities are addressed should the clinician infer that a residual finding represents recurrent destructive disease.

The practical consequence is that abnormality should be treated as an interpretive prompt, not an automatic treatment trigger. Some abnormalities warrant observation because the most plausible explanation remains residual damage. Others warrant closer reassessment because the evidence is insufficiently coherent to classify. Still others become more concerning because persistence is accompanied by corroborating inflammatory, structural, or longitudinal change. **Table 2** summarizes these patterns without assigning numerical treatment thresholds.

Table 2. Interpreting persistent post-treatment findings without equating residual abnormality with recurrent disease

Post-treatment pattern	Most defensible initial interpretation	Findings that would favor residual damage or stable treated anatomy	Findings that would increase concern for recurrent activity	Appropriate reasoning response before escalation	Interpretation that should be avoided
Persistent deep probing site with little or no inflammatory change	Residual anatomy remains plausible	Long-term measurement stability; absent or minimal inflammatory change; unchanged attachment and structural findings	Increasing depth, repeated bleeding, attachment deterioration or credible structural change	Verify measurement comparability and examine trajectory before changing disease-state classification	"Deep pocket equals active periodontitis"
Bleeding at a previously reduced site without demonstrated structural change	Current local inflammation is present; destructive progression is not yet established	Transient or isolated bleeding with otherwise stable measurements and corrected local exposure	Persistent bleeding with worsening probing/attachment pattern or other concordant evidence	Correct modifiable local factors and reassess persistence and concordance	"Bleeding equals new attachment loss"
Historical radiographic defect with stable current clinical findings	Residual structural record of previous disease is likely	Comparable serial images without new loss; stable clinical trajectory	Reproducible interval bone loss accompanied by compatible clinical deterioration	Confirm technical comparability and integrate serial clinical data	"Bone loss visible today means bone loss is occurring today"
Residual pocket plus unfavorable historical risk profile	Elevated prior concern requiring longitudinal attention	Stable repeated measurements and satisfactory maintenance response	Progressive site change, deteriorating inflammatory control, repeated recurrence pattern	Use risk information to modify vigilance, not to replace current evidence	"High risk means active disease is already present"
Apparent improvement despite inconsistent maintenance	Present measurements may be reassuring but longitudinal confidence is reduced	Stable findings sustained after continuity of care is restored	Recurrent inflammation, missed assessments, or deterioration during/after gaps in care	Re-establish comparable observation points before assuming durable stability	"One good visit proves successful long-term control"
Discordant clinical and radiographic findings	Evidence refers to different biological or temporal layers	Clinical stability plus structurally unchanged historical defects	Concordant serial deterioration across clinical and structural measures	Identify which observation is time-sensitive and seek longitudinal corroboration	Forcing all modalities to imply the same current state

Abnormal prognostic or algorithmic output with clinically stable examination	Future-risk signal, not proof of present recurrence	Stable repeated clinical trajectory and absence of corroborating change	Subsequent clinically demonstrable deterioration consistent with predicted risk	Review calibration, patient similarity, and the model's intended prediction target	"Predicted risk is equivalent to a present diagnosis"

Interpreting discordant evidence

Discordance becomes clinically important when two or more credible observations support different interpretations of the same patient or site. The clinician may encounter a persistent deep pocket without bleeding, bleeding without demonstrable attachment change, a radiographic defect without convincing clinical deterioration, or a high prognostic risk estimate in a patient who appears stable. These are not necessarily failures of diagnosis. They often arise because the signals refer to different phenomena. Risk factors documented during periodontal maintenance are associated with progression and tooth loss, but they modify the background probability against which current findings are interpreted rather than directly identifying active breakdown [18]. Similarly, long-term site-level evidence indicates that characteristics of residual pockets can help predict later worsening [19], yet predictive association does not mean that every residual pocket currently contains an active destructive process.

The interpretive challenge is therefore to distinguish discordant evidence from contradictory evidence. Two observations are contradictory only if they purport to measure the same construct over the same time window and produce incompatible results. Many periodontal findings are instead discordant because they measure different constructs. A residual radiographic defect and absence of bleeding, for example, can both be accurate: one records past structural loss, while the other provides information about current inflammatory expression. Persistent residual pockets have nevertheless been associated with later tooth loss, so their prognostic significance should not be dismissed simply because residual anatomy is a plausible explanation [20]. Baseline stage and grade can also modify the observed benefits associated with regular supportive periodontal care [21]. The correct inference is neither that residual findings are harmless nor that they automatically demonstrate recurrence; it is that their meaning depends on convergence, persistence, and context.

Longitudinal information becomes especially valuable when cross-sectional signals disagree. A single examination offers a state description. Repeated examinations provide evidence about direction. When probing measurements, attachment findings, bleeding patterns, and imaging remain stable under comparable conditions, the residual-damage interpretation becomes more credible. When deterioration is reproducible across time or appears in more than one biologically coherent domain, the active-disease interpretation gains weight. The evidentiary strength arises from the pattern of change, not merely from the number of abnormal measurements. A newly worse value that returns to baseline at the next carefully performed examination has a different meaning from a monotonic or recurrent adverse trajectory.

Formal prognostic models illustrate both the attraction and the limits of evidence integration. External validation of a 10-year periodontal tooth-loss nomogram shows that multiple clinical variables can be combined into structured prediction rather than interpreted in isolation [22]. At the same time, systematic evaluation of periodontal prognosis models demonstrates heterogeneity in predictors, modelling methods, validation, and performance [23]. A model can therefore estimate a future outcome within a defined population without answering the distinct question of whether a particular residual site is actively deteriorating today. Prediction and diagnosis occupy related but non-identical epistemic roles.

The same caution applies to simpler risk scores. Tooth-level periodontal risk models demonstrate that clinically available variables can be integrated into formalized estimates [24], while direct comparison of prognostic systems indicates that different approaches do not perform identically across patient populations [25]. Disagreement among models is not merely a technical nuisance; it exposes uncertainty in how clinical variables are weighted, which outcomes are predicted, and how well the development population resembles the patient being assessed. A prognostic output should therefore modify the clinician's prior concern, not erase discordant clinical evidence or serve as an independent treatment mandate.

A defensible interpretation can be organized around four questions. First, what phenomenon does each signal actually measure? Historical structure, present inflammation, current anatomy, longitudinal change, and predicted future risk should not be collapsed. Second, what is the temporal relationship among the

observations? A stable residual defect and a newly emerging inflammatory pattern may legitimately support different conclusions. Third, which rival explanations remain plausible? Measurement variability, access difficulty, transient inflammatory exposure, post-treatment anatomy, inconsistent maintenance, systemic influences, and genuine recurrence may produce superficially similar findings. Fourth, what additional observation could discriminate among those explanations? Sometimes the appropriate response to discordance is not immediate escalation but a more informative reassessment.

The resulting epistemic position is deliberately conditional. Evidence requiring treatment escalation is not defined by a single abnormal sign or a numerical score. Concern strengthens when an adverse finding is reproducible, persists despite correction of obvious local causes, fits a biologically plausible pattern, demonstrates credible longitudinal deterioration, or converges with independent evidence referring to the same pathological process. Concern weakens when discordance is explained by historical anatomy, non-comparable measurements, transient inflammatory conditions, or a predictive tool operating outside its validated role. **Figure 1** represents this adjudication as a bounded landscape rather than a one-direction treatment algorithm.

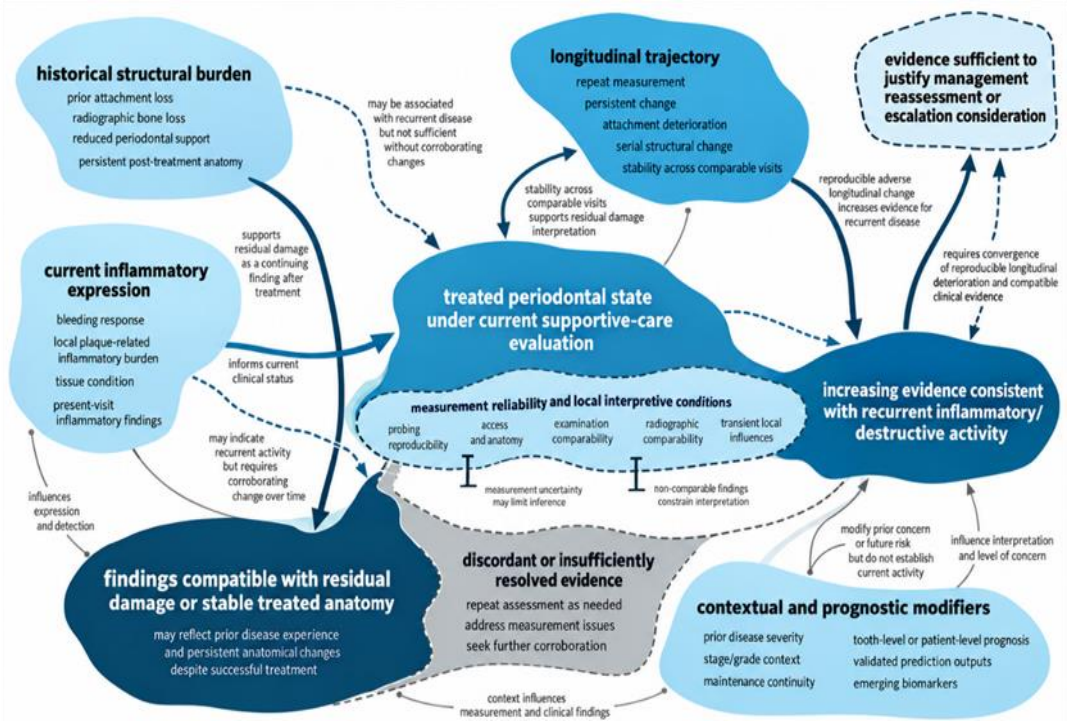

Figure 1. Adjudicating discordant periodontal signals across historical damage, current inflammation, and longitudinal escalation evidence

A framework for evidence integration and escalation

The preceding analysis suggests that treatment escalation should not begin with the question, "Which abnormal finding requires intervention?" A more defensible starting point is whether the observation has been sufficiently characterized to support a disease-state inference. Supportive periodontal care itself can be evaluated prospectively, and randomized evidence demonstrates that different maintenance approaches can be subjected to clinical testing rather than assumed to be equivalent [26]. More recent randomized evaluation of structured supportive-care protocols similarly confirms that maintenance is an active clinical process involving repeated assessment, prevention, professional instrumentation, and adaptation to patient and site conditions [27]. These findings support continued surveillance and intervention when indicated, but they do not establish a universal numerical threshold at which every residual abnormality should trigger escalation.

The proposed integration framework therefore begins with verification. A concerning finding should first be assessed for technical comparability, reproducibility, local anatomical explanation, and temporal relationship to previous therapy. The second operation is classification of evidentiary meaning: structural findings should be identified as primarily historical or longitudinal; inflammatory findings as evidence of current inflammatory expression; risk factors as modifiers of prior concern; and prognostic outputs as

estimates of future outcomes rather than diagnoses of current destruction. The third operation is corroboration. Confidence in recurrent activity increases when independently meaningful findings converge—for example, persistent inflammatory evidence accompanied by reproducible probing deterioration or credible interval structural change. Conversely, uncertainty should remain visible when findings refer to different biological processes or cannot be reproduced.

Only after these interpretive operations should evidence for treatment modification be considered. Adjunctive approaches during supportive periodontal therapy have been evaluated systematically, but their incremental benefits vary according to intervention, study design, indication, and outcome [28]. Evidence concerning local interventions for residual pockets likewise demonstrates heterogeneity rather than a single superior escalation pathway [29, 30]. The existence of an available intervention should therefore not influence the preceding diagnostic inference by making an equivocal finding appear more certain than it is.

Specific trials illustrate the same boundary. Adjunctive indocyanine-green photodynamic therapy has been evaluated in residual periodontal pockets during maintenance [31], while a polynucleotide and hyaluronic-acid gel has been tested as an adjunct to subgingival re-instrumentation [32]. Such studies provide evidence about particular interventions under specified conditions. They do not establish that the presence of any residual pocket automatically justifies those treatments, nor do short-term changes in periodontal parameters prove durable disease control. Evidence of intervention efficacy is downstream from the question of whether escalation is indicated.

Accordingly, the framework proposed here does not generate a numerical score or binary treatment output. Its endpoint is a reasoned level of actionability. At one end are abnormalities adequately explained by stable post-treatment anatomy or measurement conditions. An intermediate state contains discordant findings requiring correction of modifiable factors, closer observation, or more informative reassessment. At the opposite end are reproducible, temporally coherent findings that increasingly support recurrent inflammatory or destructive activity and make management reassessment more defensible. The distinction is deliberately conditional: the evidentiary threshold for concern may be influenced by the consequences of delayed action, but greater clinical consequence should increase the care devoted to evidence gathering rather than justify uncritical interpretation of weak evidence.

This approach also preserves a necessary separation between evidence of disease and evidence for a particular intervention. A clinician may reasonably conclude that recurrence is increasingly likely while remaining uncertain about the most appropriate escalation strategy. Conversely, an intervention with supporting trial evidence should not be used merely because it is available. The analytical sequence is therefore observation, verification, interpretation, corroboration, disease-state inference, and only then consideration of management modification. These are reasoning functions rather than validated algorithmic stages, and prospective testing would be required before the framework could be treated as a formal clinical decision instrument.

Clinical accountability, limitations, and future refinement

Clinical uncertainty does not disappear when evidence is organized more carefully. Instead, the responsibility for handling that uncertainty becomes clearer. Continuity of supportive care is especially important because interpretation depends on comparable observations across time. Discontinuation of supportive periodontal therapy has been associated with deterioration in periodontal status [33]. Yet absence from care creates an additional epistemic problem: it removes observations that might otherwise distinguish gradual recurrence from a newly detected but longstanding abnormality. Clinicians should therefore document not only current measurements but also gaps in follow-up, changes in instrumentation or technique, treatment received elsewhere, and other factors that affect longitudinal comparability.

Accountability also requires resistance to technological overconfidence. Genetic biomarkers may eventually improve periodontal risk stratification, but the available literature remains heterogeneous in the markers studied, populations sampled, analytical methods used, and strength of replication [34]. Genetic information may modify background susceptibility; it does not currently determine whether a particular periodontal pocket is actively deteriorating during a supportive-care visit. The same principle applies to host-response and microbial markers discussed earlier: greater biological sophistication does not automatically confer greater clinical decisiveness.

Artificial intelligence and multivariable predictive systems may offer more efficient integration of longitudinal information. Recent longitudinal modelling demonstrates the feasibility of developing

AI-based predictions of periodontitis progression [35]. Such approaches may eventually detect combinations or trajectories that are difficult to identify through unaided clinical reasoning. Their appropriate role, however, depends on external validation, calibration in the intended population, transparent definition of predicted outcomes, and evidence that their use improves clinical decisions rather than merely statistical discrimination. A probability of future progression is not identical to evidence of current progression, and a model trained on one case mix may perform differently when maintenance access, disease severity, examination protocols, or population characteristics change.

Several limitations therefore apply to the framework developed here. First, it is a non-empirical synthesis and has not been prospectively validated as a decision rule. Its proposed relationships should be tested for reproducibility, clinical usability, inter-examiner agreement, and consequences for overtreatment and undertreatment. Second, much of the evidence concerning residual pockets, tooth loss, maintenance adherence, and prognosis is observational. Associations may be influenced by treatment selection, patient adherence, unmeasured susceptibility, and differences in maintenance systems. Third, the evidentiary meaning of a clinical signal is context dependent. Findings from treated stage I–III periodontitis may not transfer unchanged to stage IV configurations, medically complex populations, or patients receiving care under substantially different maintenance conditions.

Fourth, the framework intentionally avoids fixed cutoffs for escalation. This limits its immediate use as a protocol but protects against unsupported precision. A future decision instrument would require independently derived and externally validated thresholds linked to clinically important outcomes, not thresholds created from conceptual convenience. Fifth, periodontal disease may progress episodically or locally, meaning that repeated stability does not guarantee indefinite future stability. Longitudinal evidence should therefore change the degree of confidence assigned to an interpretation rather than convert uncertainty into certainty.

Future refinement should focus on the relationship between measurement trajectories and clinically consequential outcomes. Studies designed around repeated standardized observations could examine how combinations of probing change, bleeding patterns, attachment measurements, imaging, maintenance adherence, biomarkers, and patient-level susceptibility alter the probability of subsequent deterioration. Model development should distinguish prediction of future events from classification of present disease state. External validation should evaluate performance across different periodontal-care systems and patient populations. Most importantly, clinical-impact studies should test whether structured handling of discordant evidence improves decision quality, reduces unnecessary escalation, identifies recurrence earlier, or improves long-term tooth retention.

The clinician's responsibility remains central throughout these developments. A transparent statement such as “the finding is abnormal but presently insufficient to distinguish residual anatomy from recurrent activity” may be more scientifically defensible than an unjustifiably certain diagnosis. Clinical accountability consists not only of acting when evidence is strong but also of identifying what remains unknown, specifying what additional evidence could change the interpretation, and ensuring that uncertainty is reassessed rather than ignored.

Conclusion

Discordant clinical signals are an expected feature of supportive periodontal care because probing findings, inflammatory signs, radiographic structures, historical disease classifications, biomarkers, and prognostic models describe different aspects and time horizons of periodontal health and disease. Their disagreement should therefore prompt interpretation rather than automatic escalation.

The central distinction is between an abnormal observation and evidence that the treated periodontal state has changed. Residual attachment loss, bone defects, or persistent pocket anatomy may coexist with stability, while recurrent disease becomes increasingly plausible when adverse findings are reproducible, temporally coherent, biologically compatible, and corroborated by independent evidence of deterioration. Prognostic factors and predictive technologies can modify concern but should not be treated as direct measurements of current disease activity.

A defensible clinical response consequently proceeds from verification and interpretation to corroboration and only thereafter to management reconsideration. This preserves sensitivity to recurrence without converting every residual abnormality into treatment failure. The proposed framework is an evidence-bounded reasoning structure rather than a validated clinical algorithm; its value lies in making the boundaries between observation, inference, prediction, and intervention explicit enough to be tested and refined.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

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