

Peri-implant Tissue Inflammation: Recognising Reversible Mucositis and Progressive Bone Loss

An evidence-based guide to the biological continuum, diagnostic reasoning and preventive care



Conceptual medical illustration. It does not represent an individual diagnostic image.

Core message: peri-implant mucositis involves inflammation without continuing marginal bone loss, while peri-implantitis includes progressive loss of supporting bone. Early detection and control of mucositis form the principal preventive pathway.

Educational material for dental students, postgraduate trainees and academic teachers

Purpose and learning outcomes

This material explains how peri-implant tissues move from health to reversible mucosal inflammation and, in susceptible sites, to destructive disease. It connects tissue biology with the clinical information needed to distinguish peri-implant mucositis from peri-implantitis.

After completing the material, the reader should be able to:

- Differentiate peri-implant health, peri-implant mucositis and peri-implantitis using current case definitions.
- Explain how persistent biofilm and the host inflammatory response contribute to soft-tissue breakdown and osteoclast-mediated bone loss.
- Interpret bleeding, suppuration, probing measurements and radiographic bone levels as parts of one diagnostic process.
- Recognise major patient-related, local and behavioural factors that modify disease risk or complicate plaque control.
- Describe how supportive peri-implant care and early treatment of mucositis contribute to prevention.

Scope

The material addresses biofilm-associated peri-implant diseases. Mechanical complications, implant fracture, foreign-body reactions and oral malignancy may occasionally produce overlapping signs and require a broader differential diagnosis. Laboratory biomarkers remain adjunctive and do not replace clinical and radiographic assessment.

Terminology note

Initial crestal bone remodelling after implant placement and restoration must be distinguished from subsequent pathological bone loss. The diagnostic meaning of any measurement depends on the time point, the reference examination, the clinical inflammatory status and the quality of the radiographic comparison.

Absence of pain does not exclude disease. Peri-implant inflammation may remain clinically quiet until bleeding, suppuration, increasing probing depth or radiographic change becomes evident.

Peri-implant health and disease

Contemporary classification separates three clinically relevant states. They form a useful framework, but diagnosis still requires interpretation of the patient, implant, prosthesis and available longitudinal records.

Condition	Clinical soft-tissue findings	Bone-level findings	Implication
Peri-implant health	No clinical signs of inflammation. No bleeding or suppuration on gentle probing.	No additional loss beyond changes attributable to initial remodelling.	Continue individualised supportive care.
Peri-implant mucositis	Bleeding on gentle probing is the key sign. Erythema, swelling, suppuration or oedema-related increases in probing depth may occur.	No continuing marginal bone loss after initial healing.	Control biofilm, improve access and reassess response.
Peri-implantitis	Inflammation with bleeding and/or suppuration. Increasing probing depth or recession may indicate tissue breakdown.	Progressive loss of supporting bone beyond initial remodelling.	Complete diagnosis and sequential treatment are required.

Peri-implant mucositis

Biofilm accumulation disrupts homeostasis at the implant-mucosa interface. The resulting inflammatory lesion remains confined to the soft tissues and does not include continuing marginal bone loss. Bleeding on gentle probing is the main clinical feature. Tissue redness, swelling, suppuration and an apparent increase in probing depth can occur because oedema increases tissue volume or reduces probing resistance.

The condition can resolve when clinicians and patients successfully control biofilm, although complete resolution does not occur after every intervention. Treatment response should therefore be verified rather than assumed. The EFP guideline recommends reassessment after approximately two to three months.

Peri-implantitis

Peri-implantitis is a biofilm-associated pathological condition in which mucosal inflammation accompanies progressive loss of supporting bone. Disease may begin relatively early and can follow a non-linear, accelerating course. Established bone loss changes the anatomy of the site, complicates decontamination and may reduce access for daily cleaning. Successful control of inflammation does not recreate the original supporting tissues automatically.

From biofilm to supporting bone loss

Biofilm provides the initiating microbial challenge, but tissue destruction reflects the interaction between that challenge and the host response. The process develops through linked biological stages rather than a single event.

Stage	Biological process	Clinical meaning
Biofilm accumulation	Microorganisms colonise the implant and prosthetic surfaces, especially where contours or thread geometry limit cleaning.	Persistent plaque around a non-shedding surface sustains exposure.
Dysregulated inflammation	Neutrophils and macrophages respond to the microbial challenge. IL-1 beta, IL-6 and TNF-alpha amplify cell recruitment and inflammatory signalling.	Bleeding, oedema and suppuration may emerge before structural bone change is recognised.
Matrix degradation	Proteolytic enzymes, including MMP-8 and other matrix metalloproteinases, participate in collagen breakdown and tissue remodelling.	The mucosal barrier becomes less stable and probing measurements may increase.
Osteoclast activation	A shift in the RANKL/OPG balance favours osteoclast differentiation and resorptive activity.	Inflammation begins to affect supporting bone.
Progressive structural loss	Repeated or persistent inflammatory activity produces a bone defect around the implant.	Radiographic bone loss and changing probing measurements support a diagnosis of peri-implantitis.

RANKL and OPG

Receptor activator of nuclear factor kappa-B ligand supports differentiation and activation of osteoclasts. Osteoprotegerin acts as a soluble decoy receptor that limits RANKL signalling. An inflammatory environment that favours RANKL activity shifts bone turnover towards resorption. This pathway explains how a mucosal inflammatory lesion can become connected with structural loss, but routine diagnosis does not require measurement of RANKL or OPG.

Matrix metalloproteinases and biomarkers

Matrix metalloproteinases reflect tissue remodelling and collagen degradation. Peri-implant crevicular fluid can contain cytokines, enzymes and bone-related mediators, while blood tests reflect systemic rather than site-specific inflammation. Biomarker research may eventually improve activity assessment or risk stratification. Current evidence does not support replacing probing and radiographic comparison with a single molecular or blood-derived marker.

Why peri-implant tissues respond differently

An osseointegrated implant lacks cementum and a periodontal ligament. Supracrestal connective-tissue fibres do not insert into the implant surface as periodontal fibres insert into a natural tooth. The local vascular arrangement and the orientation of the tissue barrier also differ. These features do not independently cause disease, but they shape how inflammation spreads and how the site responds to sustained biofilm.

Feature	Natural tooth	Dental implant
Attachment apparatus	Periodontal ligament and cementum form a specialised attachment.	Direct bone-to-implant contact with no periodontal ligament.
Connective-tissue fibres	Fibres insert into cementum.	Fibres generally run parallel or circumferentially and do not insert into titanium.
Vascular supply	Periodontal and suprapariosteal sources contribute to the tissues.	The peri-implant barrier has a different and relatively reduced vascular arrangement near the implant surface.
Clinical warning	Pain, mobility or periodontal symptoms may occur, although disease can also be silent.	Inflammation and bone loss may progress with little or no pain; mobility usually appears late and may indicate loss of osseointegration.

Host susceptibility

Patients do not respond uniformly to the same biofilm exposure. Previous periodontitis identifies a susceptible phenotype and remains an important risk signal. Poor plaque-control skills and absence of regular maintenance also have strong support as risk indicators. Poorly controlled diabetes, smoking, medication exposure and other systemic conditions can modify healing or inflammatory regulation, but their meaning should be interpreted within the complete clinical context.

Local environment

Implant position and prosthetic design influence whether the patient and clinician can reach the peri-implant sulcus. Excessive contour, deep restoration margins, residual cement and unfavourable implant position can create plaque-retentive areas or reduce diagnostic access. These factors are clinically important because some can be modified during planning, prosthetic delivery or maintenance.

Risk is not diagnosis. A patient can have several risk indicators without peri-implantitis, and peri-implantitis can occur without every recognised risk indicator. Diagnosis rests on tissue findings and bone-level change.

A structured diagnostic pathway

Reliable diagnosis combines medical context, a reproducible clinical examination and radiographic comparison. No single probing depth separates health from disease in every implant.

Step	What to assess	Interpretive principle
1. History	Previous periodontitis, smoking, diabetes control, medication exposure, recent symptoms and maintenance attendance.	Risk information modifies probability and care planning; it does not establish disease alone.
2. Clinical examination	Plaque, erythema, swelling, bleeding, suppuration, probing depth, mucosal margin and prosthetic cleansability.	Use gentle probing at reproducible sites and record limitations caused by restoration contours.
3. Baseline comparison	Measurements and a radiograph obtained after completion of the prosthetic phase.	Change over time provides stronger evidence than a single measurement.
4. Radiographic assessment	Marginal bone level relative to stable implant landmarks using comparable projection.	Two-dimensional radiographs show mesial and distal bone best and may underestimate buccal or lingual defects.
5. Integrated diagnosis	Inflammatory signs plus longitudinal soft-tissue and bone-level change.	State uncertainty when baseline records, image quality or probing access are inadequate.

When baseline information is available

Peri-implantitis is supported by clinical inflammation, an increase in probing depth or apical migration of the mucosal margin, and radiographic bone loss beyond the changes expected after initial healing. Consistent measurement sites and image geometry increase confidence in the comparison.

When baseline information is unavailable

The 2018 World Workshop case definition identifies peri-implantitis when bleeding and/or suppuration on gentle probing occurs together with a probing depth of at least 6 mm and a bone level at least 3 mm apical to the most coronal intra-osseous portion of the implant. These thresholds provide a pragmatic classification when previous records are absent. They should not be used to redefine every shallower inflamed site as healthy.

Diagnostic uncertainty and differential diagnosis

Implant fracture, technical complications, residual cement, foreign material, medication-related osteonecrosis and oral malignancy can occasionally share features with biofilm-associated disease. Atypical lesions, persistent ulceration, unexplained rapid progression or failure to respond to appropriate treatment should widen the differential diagnosis and prompt referral or additional investigation.

Prevention and supportive peri-implant care

Prevention begins during treatment planning and continues for as long as the implant remains in function. The EFP S3 guideline places prevention before implant placement, during surgical and prosthetic treatment, and throughout maintenance.

Phase	Priority actions
Before implant placement	Stabilise periodontal disease, assess patient-level risk, establish effective oral hygiene and plan a position that supports biological and prosthetic maintenance.
During restoration	Provide contours and contact relationships that permit cleaning and clinical access. Avoid residual cement and document baseline tissue and bone levels.
Healthy implant in function	Provide supportive peri-implant care with professional biofilm removal, oral-hygiene coaching and periodic tissue assessment.
Peri-implant mucositis	Disrupt biofilm professionally, improve daily plaque control, correct modifiable plaque-retentive factors and reassess after approximately two to three months.
Peri-implantitis	Begin with risk-factor control and nonsurgical infection control, then select further therapy according to residual disease, defect anatomy and patient factors. Continue supportive care after treatment.

Patient education

Patients should understand that bleeding during cleaning is a reason to request assessment, not a reason to stop cleaning the implant area. Home-care devices must match the prosthesis and the patient's dexterity. Instructions should be demonstrated at the individual site rather than limited to generic advice.

Recall interval

Supportive care should follow an individualised schedule based on disease history, plaque control, prosthetic cleansability and systemic context. Recent consensus supports closer surveillance after peri-implantitis treatment and continued reassessment of the interval. A fixed schedule for every patient cannot account for differences in susceptibility or treatment history.

Why mucositis treatment matters

The 2025 AO/AAP consensus identified peri-implant mucositis as a predisposing condition for peri-implantitis. This supports early intervention, while not implying that every mucositis site inevitably progresses. Effective prevention depends on recognising inflammation, correcting accessible causes and confirming that tissue health has been restored.

Interpretation pitfalls

Common error	Why it is misleading	Better approach
No pain means no disease	Peri-implant inflammation can progress without pain.	Ask about bleeding during cleaning and perform structured surveillance.
Bleeding alone equals peri-implantitis	Bleeding identifies inflammation but does not demonstrate progressive bone loss.	Differentiate mucositis from peri-implantitis through bone-level assessment.
One deep probing measurement proves disease	Healthy implants can have different soft-tissue dimensions.	Interpret depth together with bleeding, recession and change from baseline.
One radiograph proves progression	Projection differences and initial remodelling can mimic change.	Compare standardised images and stable landmarks over time.
A biomarker establishes the diagnosis	Local and systemic markers overlap across diseases and remain influenced by confounders.	Use biomarkers only as adjuncts to established clinical criteria.
A systemic condition automatically contraindicates implants	Risk depends on disease control, treatment exposure and the whole clinical context.	Use individual risk assessment and interdisciplinary consultation when indicated.

Key learning points

- Peri-implant mucositis affects the mucosal tissues without continuing marginal bone loss after initial healing.
- Peri-implantitis combines peri-implant inflammation with progressive loss of supporting bone.
- Biofilm initiates the lesion, while tissue anatomy, local conditions and host susceptibility influence severity and progression.
- Longitudinal records strengthen diagnosis. Gentle probing and comparable radiographs provide complementary information.
- The 2018 threshold-based definition supports diagnosis when baseline records are missing, but clinical judgement remains necessary.
- Early control of mucositis, cleanable prosthetic design and structured supportive care form the central preventive pathway.

Clinical reasoning summary: detect inflammation, compare with baseline, verify bone-level change, identify modifiable factors and reassess the response to care.

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