

Understanding Peri-implantitis: Disease Burden, Progression, and Clinical Relevance

An evidence-based guide to definitions, epidemiology, disease trajectory and risk interpretation



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

Core message: peri-implantitis is defined by inflammation and progressive loss of supporting bone. Its frequency depends strongly on case definitions and study design, while its course may be early, non-linear and accelerating.

Educational material for dental students, postgraduate trainees and academic teachers

Purpose and learning outcomes

This material explains how peri-implantitis is defined, measured and interpreted. It links population-level evidence with the longitudinal records needed to recognise disease in an individual patient.

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

- Differentiate peri-implant health, peri-implant mucositis and peri-implantitis using contemporary definitions.
- Explain why prevalence and incidence answer different questions and why patient-level and implant-level estimates cannot be used interchangeably.
- Describe the early, non-linear and potentially accelerating pattern of peri-implantitis progression.
- Separate established risk indicators from contextual or uncertain associations.
- Translate epidemiological evidence into baseline documentation, surveillance and risk-based supportive care.

Scope and terminology

The focus is plaque-associated peri-implant disease. Initial physiological bone remodelling after implant placement and prosthetic loading must be distinguished from subsequent pathological bone loss. Mechanical complications, malposition, residual cement, foreign-body reactions and malignancy may produce overlapping findings and require differential diagnosis.

The word risk is used carefully. A risk factor should precede disease and have credible longitudinal evidence. Cross-sectional studies more often identify risk indicators: characteristics associated with disease at one time point. Association does not by itself establish causation.

Population statistics do not diagnose an individual implant. Epidemiology estimates burden and supports planning; diagnosis requires clinical inflammation, longitudinal measurements and radiographic interpretation.

Defining health, mucositis and peri-implantitis

A clear case definition is the starting point for both research and clinical communication. Changing the required probing depth, bleeding threshold or amount of bone loss can substantially change the number of cases counted in a study.

Condition	Clinical findings	Bone-level interpretation	Key distinction
Peri-implant health	No clinical signs of inflammation; no bleeding or suppuration on gentle probing.	No additional loss beyond initial remodelling. Health can exist around implants with reduced support.	There is no single probing-depth range that defines health for every implant.
Peri-implant mucositis	Bleeding on gentle probing is the principal sign; erythema, swelling or suppuration may occur.	No continuing marginal bone loss after initial healing.	The lesion is confined to the soft tissues and is potentially reversible.
Peri-implantitis	Inflammation with bleeding and/or suppuration, often with increasing probing depth or recession.	Progressive loss of supporting bone beyond initial remodelling.	Structural support is being lost; plaque control alone does not recreate lost bone.

Diagnosis with baseline records

When previous measurements and radiographs are available, peri-implantitis is identified by inflammatory signs together with increasing probing depth or recession and bone loss beyond the expected changes after initial healing. Comparable image geometry and stable implant landmarks strengthen the inference that change is real.

Diagnosis without baseline records

The 2018 World Workshop proposed a pragmatic case definition for situations without previous data: bleeding and/or suppuration on gentle probing, probing depth of at least 6 mm and a bone level at least 3 mm apical to the most coronal intra-osseous part of the implant. These thresholds support classification; they do not imply that every shallower inflamed site is healthy.

Measurement principle: a change from a reliable baseline is usually more informative than an isolated threshold.

Measuring disease burden

Epidemiological estimates are not fixed properties of a disease. They reflect who was studied, how disease was defined, how long implants had been in function, whether measurements were made at the patient or implant level and how missing follow-up data were handled.

Measure	Question answered	Essential denominator	Main limitation
Prevalence	How common is disease in a population at a defined time?	People or implants examined at that time.	Combines new and long-standing cases and is sensitive to case definitions.
Incidence	How many disease-free units develop disease during follow-up?	People or implants at risk and disease-free at baseline.	Requires longitudinal data; attrition and unequal follow-up may bias estimates.
Patient-level estimate	How many people have at least one affected implant?	Patients.	A person with one affected implant and a person with several affected implants each count once.
Implant-level estimate	How many implants are affected?	Implants.	Implants are clustered within patients and are not statistically independent.

What recent pooled evidence suggests

A 2025 AO/AAP systematic review of 102 studies estimated patient-level prevalence at 46% for peri-implant mucositis and 21% for peri-implantitis. The weighted mean patient-level incidence within up to 20 years of function was 53% and 22%, respectively. These pooled estimates communicate substantial disease burden, but they should not be treated as universal probabilities for every clinic or patient population.

Why estimates vary

- Different thresholds for bleeding, probing depth and radiographic bone loss.
- Convenience samples, specialist cohorts and community populations with different baseline risk.
- Different implant systems, restorative designs, function times and maintenance exposure.
- Patient-level versus implant-level reporting and failure to account for clustering.
- Exclusion of failed implants, non-attenders or participants lost to follow-up.

Interpretation rule: always read the case definition and denominator before comparing two prevalence estimates.

Onset and progression are not necessarily linear

Peri-implantitis can begin relatively early after restoration and may remain unnoticed without baseline documentation. Longitudinal evidence indicates that progression is often non-linear and can accelerate. A stable early interval therefore does not guarantee lifelong stability, and a single cross-sectional image cannot reveal the speed of previous change.

Clinical phase	What may be observed	Interpretive consequence
Initial remodelling	Expected crestal bone adaptation after placement and prosthetic loading.	Document a post-restorative reference rather than labelling all early bone change as disease.
Mucosal inflammation	Bleeding, erythema, swelling or suppuration without continuing bone loss.	Treat biofilm-associated inflammation and verify resolution.
Early peri-implantitis	Inflammation with new bone-level change or increasing probing depth.	Confirm progression, identify modifiable contributors and intervene promptly.
Accelerating destruction	Increasing defect depth, recession, suppuration or rapid radiographic change.	Treatment complexity rises as anatomy and cleansability deteriorate.

The value of a reference examination

A baseline clinical chart and radiograph obtained after completion of the prosthetic phase allow later observations to be interpreted as change rather than isolated measurements. Record probing depth, bleeding, suppuration, mucosal margin position, plaque and prosthetic access at reproducible sites. Use a radiographic projection that can be repeated.

Why early recognition preserves options

Peri-implant mucositis can often resolve after effective biofilm control and correction of accessible local factors. Once supporting bone is lost, decontamination becomes more difficult, defect morphology influences treatment and regeneration is not predictable in every case. Earlier recognition therefore reduces biological and technical complexity even when implant survival is not immediately threatened.

Surveillance is longitudinal: compare each implant with itself over time, not only with a universal probing-depth threshold.

Interpreting determinants and risk indicators

Evidence strength is uneven. Some determinants recur across consensus reports and longitudinal studies, while others are plausible but heterogeneous or dependent on context. Risk assessment should reflect this hierarchy.

Evidence category	Examples	Clinical interpretation
Established signals	History of periodontitis; inadequate plaque control; absence of regular supportive care.	Increase surveillance intensity, reinforce maintenance and ensure periodontal stability.
Supported patient-level association	Diabetes or hyperglycaemia, especially when glycaemic control is poor.	Coordinate medical care and improve modifiable control; do not infer disease from diagnosis alone.
Contextual local contributors	Implant malposition; excessive prosthetic contour; poor cleansability; residual cement; difficult access for examination.	Assess whether the design or position sustains plaque retention or limits prevention and treatment.
Heterogeneous or uncertain indicators	Smoking and several systemic, behavioural or implant-related variables for which estimates differ by study design.	Use calibrated language and avoid presenting association as proof of causation.

Prior periodontitis

A history of periodontitis is a consistent risk signal and may reflect both biological susceptibility and cumulative behavioural exposure. Implant therapy should follow control of active periodontal disease, and maintenance should include teeth and implants within the same longitudinal plan.

Plaque control and supportive care

Plaque is the initiating exposure, while prosthetic access and patient dexterity influence how effectively it can be controlled. Regular supportive care creates opportunities to reinforce self-care, remove deposits, detect inflammation and identify changes before extensive bone loss occurs. Recall intervals should be individualised rather than identical for every patient.

Systemic and behavioural factors

Diabetes and hyperglycaemia have growing evidence of association with peri-implantitis, particularly when metabolic control is poor. Evidence for smoking and other exposures varies across analyses. These factors should be recorded, managed where possible and integrated with local findings, not used as stand-alone diagnostic labels.

Risk is probabilistic. It changes the likelihood of disease and the intensity of prevention; it does not replace examination.

Why peri-implantitis matters clinically

The clinical significance of peri-implantitis extends beyond the binary question of whether an implant remains in place. Disease alters tissue architecture, compromises cleansability, increases treatment burden and can affect comfort, aesthetics, confidence and long-term restorative planning.

Domain	Potential consequence
Biological support	Progressive bone loss reduces the supporting envelope and may ultimately compromise osseointegration.
Tissue architecture	Recession, pocket formation and defect morphology can create sites that are difficult to clean and decontaminate.
Treatment complexity	Non-surgical access becomes limited; surgical therapy, prosthetic modification or implant removal may be required.
Restorative function	Changes around one implant can affect multi-unit prostheses and future rehabilitation choices.
Patient experience	Aesthetic change, bleeding, suppuration, repeated procedures, cost and uncertainty may affect quality of life.

A distinct tissue environment

An implant lacks cementum and a periodontal ligament. Connective-tissue fibres generally run parallel or circumferentially rather than inserting into titanium, and the vascular arrangement near the crestal barrier differs from that around a tooth. These features do not cause disease, but they shape lesion development and access for repair.

Inflammatory lesion characteristics

Peri-implantitis lesions may extend apically and include plasma cells, lymphocytes, neutrophils and macrophages. Compared with periodontitis lesions, they can be larger and may lack a protective connective-tissue zone between the inflammatory infiltrate and the bone crest. These observations help explain why persistent inflammation deserves prompt attention.

Implant survival is not the only endpoint

An implant can remain functional while disease progresses. Evaluation should therefore include tissue stability, patient-centred outcomes, maintainability and the likely burden of future intervention, not survival alone.

Clinical relevance: preserving maintainable tissues and restorative options is a more useful goal than waiting for mobility or implant failure.

From epidemiology to surveillance

Disease burden justifies structured follow-up, but surveillance should remain individualised. A practical pathway integrates baseline records, modifiable risk, prosthetic cleansability and evidence of change.

Step	Action	Reason
1. Establish reference	Record probing, bleeding, suppuration, mucosal margin and a post-restorative radiograph.	Creates the comparator needed to detect progression.
2. Assess context	Review periodontitis history, plaque control, supportive-care attendance, glycaemic control and prosthetic access.	Identifies modifiable factors and informs recall intensity.
3. Detect inflammation	Use gentle, reproducible probing and examine all accessible aspects of the restoration.	Bleeding or suppuration is an early clinical signal, even without symptoms.
4. Verify change	Compare measurements and radiographs using stable landmarks and similar projection.	Distinguishes disease progression from anatomy, remodelling or measurement variation.
5. Act and reassess	Control biofilm and local contributors; escalate when bone loss or persistent inflammation is present.	Earlier action preserves more treatment and restorative options.

Common interpretation errors

- Comparing prevalence estimates without checking whether the denominator is patients or implants.
- Using a cross-sectional association as proof that an exposure caused peri-implantitis.
- Assuming a single deep probing depth establishes disease without inflammation or bone-level change.
- Treating the absence of pain, mobility or obvious radiographic change as proof of health.
- Failing to distinguish initial remodelling from progressive loss after the restorative baseline.
- Applying one recall interval to all patients regardless of history, cleansability and current findings.

Key learning points

- Peri-implantitis combines clinical inflammation with progressive supporting bone loss.
- Prevalence, incidence, patient-level estimates and implant-level estimates describe different quantities.
- Case definitions, function time and follow-up quality strongly influence measured disease burden.
- Progression may begin early and follow a non-linear, accelerating pattern.
- History of periodontitis, plaque control and supportive care have the most consistent clinical relevance.
- Baseline documentation and risk-based surveillance translate epidemiology into prevention.

Selected references

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Educational use and limitations

This material is intended for education and does not replace individual clinical assessment, diagnostic imaging selected for a specific patient, or specialist consultation. Prevalence and incidence estimates are pooled research findings and should be interpreted in relation to the case definitions, populations and follow-up methods used in the underlying studies.

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