

Osteoporosis and Long-Term Implant Stability: From Bone Fragility to Antiresorptive Care

How skeletal fragility, local bone conditions and bone-modifying treatment shape implant planning and surveillance



Core message: osteoporosis and antiresorptive exposure are related but distinct clinical variables. Osteoporosis alone does not automatically contraindicate implant treatment, while medicine indication, dose, schedule, comorbidity and long-term peri-implant inflammation can change the risk profile.

Educational material for dental students, postgraduate trainees and academic teachers

Systemic fragility and local jaw conditions

Osteoporosis is a systemic skeletal disorder characterized by reduced bone strength and increased fracture susceptibility. Diagnosis usually relies on fragility-fracture history, bone mineral density at validated skeletal sites and clinical fracture-risk assessment. These measurements do not directly measure the mechanical or biological properties of an individual implant site.

Domain	What it describes	Implant relevance
Systemic osteoporosis	Whole-skeleton fragility and future fracture risk.	Signals a medical bone disorder, but does not define jaw anatomy or guarantee implant failure.
DXA result	Areal bone mineral density, usually at hip and lumbar spine.	Useful for osteoporosis care; it cannot be transferred directly to a mandibular or maxillary site.
Jawbone anatomy	Available height and width, cortical boundaries and local trabecular architecture.	Guides implant position, osteotomy and augmentation planning.
Bone mechanics	Resistance during preparation, insertion torque and implant stability.	Provides site-specific information during surgery but does not diagnose osteoporosis.
Bone biology	Turnover, mineralization, vascularity and cellular healing.	Influenced by age, disease, nutrition and medication; incompletely visible on imaging.

CBCT does not diagnose osteoporosis

CBCT is valuable for anatomy and qualitative architecture. Grey values vary with device, acquisition and reconstruction and should not be treated as calibrated DXA measurements unless a validated protocol exists.

Age is not the diagnosis

Older age can coexist with osteoporosis, sarcopenia, polypharmacy or reduced self-care, but many older adults remain suitable implant candidates. Functional status, local disease control and the treatment burden are actionable variables.

Separate the questions: fracture-risk assessment belongs to medical osteoporosis care. Implant-site assessment belongs to local clinical, radiographic and surgical evaluation.

Remodelling, fragility and antiresorptive therapy

Bone renews itself through coupled osteoclast resorption and osteoblast formation. Osteoporosis develops when structure and strength decline because remodelling balance, hormonal environment, age-related change or secondary disease favours loss of bone. Antiresorptive medicines reduce fracture risk by suppressing resorption.

Therapy	Main skeletal action	Dental planning point
Oral bisphosphonates	Bind to mineralized bone and reduce osteoclast activity over a prolonged period.	Record agent, duration, adherence and indication; stopping does not rapidly remove skeletal exposure.
Intravenous bisphosphonates for osteoporosis	Provide potent intermittent antiresorptive treatment at osteoporosis dosing.	Do not equate an IV route for osteoporosis with high-frequency oncology regimens.
Denosumab for osteoporosis	RANKL inhibition produces rapid and reversible suppression of osteoclast activity.	Dose timing matters; unplanned delay or cessation can cause rebound bone loss and vertebral fractures.
Romosozumab	Inhibits sclerostin, increasing formation and reducing resorption during a limited course.	Review cardiovascular and bone-treatment context; rare MRONJ has been reported.
Anabolic therapy	Stimulates bone formation through PTH-receptor or related pathways.	The medical treatment sequence and fracture risk remain specialist decisions.

Treatment benefit is substantial

Antiresorptive therapy prevents vertebral, hip and other fragility fractures. The rare possibility of jaw osteonecrosis should be discussed accurately but should not lead patients to abandon effective treatment without medical guidance.

Do not advise an independent drug holiday: bisphosphonate and denosumab interruption have different biological consequences. Any change requires the prescribing clinician.

What implant survival evidence shows

Recent systematic reviews generally report similar implant survival in patients with osteoporosis and controls. Reviews of osteoporosis-dose antiresorptive treatment also do not demonstrate a consistent increase in implant failure. Confidence remains limited by observational designs, heterogeneous definitions and relatively sparse long-term drug-specific data.

Evidence question	Current direction	Remaining uncertainty
Does osteoporosis alone reduce survival?	Meta-analyses generally show no statistically significant difference from patients without osteoporosis.	Studies vary in diagnostic criteria, follow-up and control of age or comorbidity.
Does osteoporosis increase marginal bone loss?	Most pooled analyses do not show a consistent clinically important difference.	Measurement protocols and maintenance exposure differ.
Do low-dose bisphosphonates cause implant failure?	Evidence is comparatively reassuring for osteoporosis regimens.	Rare MRONJ and late events require larger, longer studies.
What about osteoporosis-dose denosumab?	Recent consensus considers placement possible in appropriately selected patients.	Fewer studies and medication-timing complexity limit precision.
Can oncology evidence be extrapolated?	No. High-dose cancer treatment belongs to a different risk category.	Cancer, chemotherapy, antiangiogenic therapy and systemic burden confound outcomes.

Survival is not the only outcome

A functioning implant may still develop peri-implant inflammation, marginal bone loss or a rare medication-related complication. Patient-level survival should be complemented by implant-level health, prosthetic maintainability and quality-of-life outcomes.

Evidence-based interpretation: reassuring average survival does not make every patient low risk. It supports individualized treatment rather than automatic exclusion.

Primary stability and surgical adaptation

Systemic osteoporosis does not provide a direct map of jawbone density. Local cortical thickness, trabecular structure, implant geometry and osteotomy technique determine primary stability. The surgical protocol should respond to the site rather than to the diagnostic label alone.

Finding	Possible adaptation	Boundary
Thin cortex or low mechanical resistance	Select implant geometry and osteotomy preparation that improve engagement without excessive compression.	High insertion torque is not a substitute for viable bone.
Limited ridge dimensions	Consider narrower or shorter implants, augmentation or a staged plan according to prosthetic needs.	Do not increase surgical burden automatically in a medically complex patient.
Low initial stability	Use a protected healing period and delay loading when indicated.	One stability value cannot guarantee or rule out osseointegration.
Need for augmentation	Compare regenerative benefit with additional surgical trauma, exposure and healing demand.	Evidence under antiresorptive therapy remains less certain than evidence for straightforward placement.
Existing periodontal disease	Stabilize infection and establish maintainable hygiene before implantation.	Systemic bone treatment cannot compensate for uncontrolled local inflammation.

Avoid overcompression

Aggressive undersizing or excessive torque can impair perfusion and produce microdamage. In low-density bone, the goal is adequate stability with preservation of tissue vitality, not the largest possible torque value.

Loading is a biological decision

Immediate or early loading should reflect primary stability, implant distribution, prosthetic control and patient factors. Osteoporosis alone does not dictate one loading protocol, but lower local stability or extensive regeneration may justify delayed loading.

Site-specific planning: adapt the osteotomy to local bone mechanics and the prosthetic plan, then verify stability rather than assuming it from DXA or CBCT grey values.

Osteoporosis-dose therapy and MRONJ risk

MRONJ is rare in patients receiving antiresorptive treatment for osteoporosis, and its absolute risk is far lower than in patients receiving high-dose oncology regimens. Nevertheless, implant surgery injures bone and a functioning implant can later become a site of inflammation, so informed consent and long-term surveillance remain appropriate.

Risk dimension	Lower-concern pattern	Higher-concern pattern
Indication	Osteoporosis or low bone mass.	Metastatic bone disease, multiple myeloma or another malignant indication.
Dose and schedule	Standard osteoporosis regimen.	Frequent high-dose parenteral oncology regimen.
Local tissue	Healthy mucosa, controlled periodontium, cleanable prosthesis.	Active periodontitis, suppuration, mucosal trauma or poor access.
Additional treatment	No major immunosuppression.	Corticosteroids, chemotherapy, antiangiogenic therapy or combined exposure.
Procedure	Straightforward placement with limited trauma.	Extensive augmentation, multiple sites or repeated bone surgery.
Follow-up capacity	Reliable maintenance and early reporting.	Irregular review or limited ability to maintain hygiene.

No validated chairside predictor

CTX and other bone turnover markers do not predict individual MRONJ risk sufficiently for implant clearance. Treatment duration, comorbidity and local inflammation matter, but no validated score combines them into a precise probability.

Drug holidays remain uncertain

Evidence is insufficient to recommend routine interruption before implant surgery. Denosumab timing requires particular caution because delayed or stopped treatment can cause rebound vertebral fractures.

Proportional consent: explain that risk is low but not zero, evidence is incomplete for rare late events and medicine changes must be coordinated with the prescriber.

Implant surgery and implant presence are different pathways

Implant-associated MRONJ can appear soon after surgery or years after successful osseointegration. The timing and surrounding inflammatory condition help frame the differential diagnosis.

Pattern	Typical context	Clinical interpretation
Surgery-triggered event	Non-healing wound, exposed or probeable bone, pain or infection after implant placement or augmentation.	Consider MRONJ alongside infection, wound dehiscence, thermal injury and early implant failure.
Presence-associated event	Previously integrated implant becomes surrounded by progressive inflammation, sequestration or exposed bone after antiresorptive treatment begins or continues.	Peri-implant infection may precipitate osteonecrosis in susceptible tissue.
Routine peri-implantitis	Biofilm-associated inflammation with progressive supporting-bone loss.	Medication history and healing trajectory remain part of the differential diagnosis.
Mechanical complication	Overload, fracture, loosening or loss of osseointegration without diagnostic MRONJ features.	Do not label every implant failure as osteonecrosis.

Red flags that exceed routine peri-implantitis

- Persistent exposed or probeable bone, especially beyond eight weeks.
- Pain, suppuration, paraesthesia, fistula or progressive sequestration.
- Failure of expected healing after local infection control or peri-implant therapy.
- Radiographic change that extends beyond the pattern expected from marginal peri-implantitis.
- A relevant history of antiresorptive, antiangiogenic or immune-modifying treatment.

Case-series percentages are not incidence

Reports of implant-associated MRONJ contain selected patients with established disease. Frequencies of pain, exposed bone, mandibular location or surgical treatment within those series cannot be used to calculate the probability for an implant patient.

Escalation rule: abnormal healing or exposed bone requires early specialist assessment rather than repeated routine peri-implantitis treatment.

Augmentation, prosthetic design and maintenance

Straightforward implant survival data should not be assumed to prove the safety of every regenerative procedure. Bone grafting, membrane exposure and repeated surgery add tissue trauma and infection opportunity. Evidence in patients receiving antiresorptives remains comparatively sparse.

Stage	Risk-reduction focus	Practical approach
Before surgery	Control oral infection and confirm a maintainable treatment plan.	Treat periodontitis, remove plaque-retentive factors and review the medicine history.
Surgical planning	Limit unnecessary trauma while achieving prosthetically correct placement.	Compare short, narrow or staged solutions with the burden of augmentation.
Prosthetic phase	Provide access for home care and professional instrumentation.	Avoid deep overcontours and cement remnants; verify tissue response.
Early healing	Detect wound dehiscence, infection and non-progressive integration promptly.	Schedule review according to procedure complexity and medication context.
Long-term maintenance	Prevent late inflammatory triggers around a stable implant.	Monitor probing findings, suppuration, plaque control and radiographic change against baseline.

Maintenance protects more than implant survival

A cleanable restoration and reliable recall reduce peri-implant inflammation and may lower the chance that an integrated implant becomes a focus for late complications. Recall frequency should reflect periodontal history, hygiene, smoking, diabetes, antiresorptive exposure and prosthetic access.

Instrumentation should preserve surfaces and tissues

Professional care should remove biofilm effectively without unnecessary soft-tissue or implant-surface damage. Persistent suppuration or progressive bone loss requires diagnostic reassessment rather than repeated superficial debridement alone.

Long horizon: successful early osseointegration does not end risk assessment. Medication can begin after implant placement, and late inflammation can change the local environment.

A decision pathway for clinical practice

Step	Clinical action	Decision supported
1. Confirm osteoporosis context	Record diagnosis, fracture history, DXA context, secondary causes and treating clinician.	Distinguishes systemic fracture care from local implant assessment.
2. Reconcile medicines	Record agent, indication, route, dose, schedule, duration, last dose and concurrent corticosteroid or cancer therapy.	Separates low-dose osteoporosis treatment from oncology exposure.
3. Assess local readiness	Evaluate periodontal health, infection, anatomy, soft tissue, hygiene and prosthetic access.	Identifies direct modifiable risks.
4. Match procedure burden	Define number of implants, osteotomy, augmentation and loading strategy.	Supports proportional adaptation or referral.
5. Discuss benefits and uncertainty	Explain expected function, alternative rehabilitation, low but non-zero MRONJ risk and evidence limitations.	Creates informed shared decision-making.
6. Plan surveillance	Establish baseline findings, early healing checks and long-term maintenance with red-flag escalation.	Targets both early failure and late presence-associated complications.

When to seek specialist coordination

Consultation becomes particularly important with malignant-disease dosing, combined antiresorptive and antiangiogenic treatment, established MRONJ, major augmentation, unexplained jaw symptoms or uncertainty about denosumab timing. The question should describe the planned procedure and the specific information required.

When elective treatment should pause

Active oral infection, unresolved exposed bone, unstable systemic disease, current high-risk oncology treatment or inability to provide safe maintenance should be addressed before elective implantation. Urgent infection still requires timely care in an appropriate setting.

Decision principle: combine fracture-treatment benefit, local feasibility, procedural burden and maintenance capacity. The word osteoporosis alone should neither authorize nor prohibit implant treatment.

Clinical synthesis and selected references

Clinical synthesis

- Separate systemic osteoporosis from local jaw anatomy and mechanical bone conditions.
- Do not use hip or spine DXA values as direct measurements of an implant site.
- Recognize that osteoporosis alone generally does not reduce implant survival in current meta-analyses.
- Interpret antiresorptive therapy by indication, dose, route, schedule and duration.
- Do not extrapolate reassuring osteoporosis-dose evidence to high-dose oncology treatment.
- Never advise unsupervised discontinuation of bisphosphonates or denosumab.
- Do not use CTX as an individual MRONJ clearance test.
- Balance primary stability with preservation of vascular and cellular bone vitality.
- Treat persistent exposed bone or abnormal healing as a reason for specialist evaluation.
- Maintain implants long term because late inflammation may trigger complications around previously integrated implants.

Selected references

1. Kim SY, et al. Impact of Osteoporosis on Dental Implant Survival, Failure, and Marginal Bone Loss: A Systematic Review and Meta-analysis. *J Clin Med*. 2025;14:6719.
2. Lemos CAA, et al. Do dental implants placed in patients with osteoporosis have higher risks of failure and marginal bone loss? A systematic review and meta-analysis. *Clin Oral Investig*. 2023.
3. de Medeiros F, et al. Dental implants in patients with osteoporosis: a systematic review with meta-analysis. *Int J Oral Maxillofac Surg*. 2018;47:480-491.
4. Ali DS, et al. Antiresorptive Therapy to Reduce Fracture Risk and Effects on Dental Implant Outcomes in Patients With Osteoporosis: Systematic Review and ONJ Taskforce Consensus Statement. *Endocr Pract*. 2025.
5. Mirza R, et al. Dental Implant Failure and Medication-Related Osteonecrosis of the Jaw in Patients Receiving Antiresorptive Therapy for Osteoporosis. *Endocr Pract*. 2025.
6. Ruggiero SL, et al. AAOMS Position Paper on Medication-Related Osteonecrosis of the Jaws - 2022 Update. *J Oral Maxillofac Surg*. 2022;80:920-943.
7. Stavropoulos A, et al. The effect of antiresorptive drugs on implant therapy: systematic review and meta-analysis. *Clin Oral Implants Res*. 2018;29 Suppl 18:54-92.
8. Nisi M, et al. Implant-associated medication-related osteonecrosis of the jaw: a review of reported cases and clinical patterns.
9. American Dental Association. Osteoporosis Medications and Medication-Related Osteonecrosis of the Jaw. Updated clinical resource.
10. International Osteoporosis Foundation. Denosumab: recommendations and safety.

Educational use and limitations

This material supports implant risk assessment and interdisciplinary communication. It does not replace osteoporosis diagnosis, fracture-risk treatment, specialist MRONJ management or individualized medical care.

Funding statement

This work was co-funded by the European Union under grant agreement No. 2024-1-PL01-KA220-HED-000251208. Views and opinions expressed are those of the author(s) only and do not necessarily reflect those of the European Union or the Foundation for the Development of the Education System (FRSE). Neither the European Union nor FRSE can be held responsible for them.