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# Hypothyroidism and Peri-Implantitis: A Hidden Risk

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**Abstract**

Thyroid diseases—including hypothyroidism, hyperthyroidism, and autoimmune thyroid conditions—have increasingly been associated with peri-implantitis, a severe inflammatory condition affecting tissues surrounding dental implants. Clinical evidence suggests that individuals with thyroid dysfunction are at a heightened risk of peri-implant inflammation, delayed healing, and unfavorable therapeutic outcomes. In addition, thyroid hormone imbalance can disrupt bone metabolism, thereby influencing osseointegration and long-term implant stability. Considering the rising global prevalence of thyroid disorders, their role must be carefully evaluated during dental implant planning and postoperative management.

This review examines the multifaceted relationship between thyroid pathology and peri-implantitis, emphasizing the importance of interdisciplinary collaboration in patient care. A deeper understanding of how thyroid disorders affect peri-implant health may contribute to improved treatment strategies and enhanced patient outcomes.

**Keywords:** thyroid disorders, hypothyroidism, autoimmune thyroid disease, peri-implantitis, dental implants, inflammation, bone metabolism, risk factors, treatment strategies

## 1. Introduction

The thyroid gland is an essential endocrine organ responsible for producing hormones that regulate numerous metabolic activities throughout the body. The primary thyroid hormones—triiodothyronine (T3) and thyroxine (T4)—are fundamental to skeletal development, bone maintenance, and remodeling processes.[1,2,3] Adequate thyroid hormone levels are necessary for normal bone growth, whereas hormonal imbalance may result in abnormal skeletal development, reduced bone mass, and increased fracture susceptibility.[4]

Both hyperthyroidism and hypothyroidism exert significant effects on bone physiology. Hyperthyroidism, characterized by suppressed thyroid-stimulating hormone (TSH) levels and elevated T3 and T4 concentrations, accelerates bone turnover and reduces bone mineral density. Common etiologies include Graves' disease, toxic multinodular goiter, and toxic adenoma. Conversely, hypothyroidism presents with elevated TSH and reduced thyroid hormone levels, leading to decreased bone turnover and impaired bone maintenance [3.] Hashimoto's thyroiditis, thyroid ablation, radiation exposure, and certain pharmacological agents are among its principal causes.

Given the strong association between thyroid function and bone health, concerns have been raised regarding the potential influence of thyroid disorders on dental implant osseointegration. Osseointegration depends on bone quality, density, and metabolic activity, all of which may be altered in patients with thyroid dysfunction.[7] Although dental implants demonstrate high survival rates in systemically healthy individuals, the extent to which thyroid disorders affect implant success remains uncertain. [3]

Further investigation is required to clarify whether thyroid hormone imbalances directly modulate osteoclastic activity and bone resorption, thereby influencing implant stability and long-term outcomes.[2,3,8] Understanding these mechanisms is essential for optimizing implant therapy in patients with thyroid disease.[3]

## 2. Clinical Implications

Thyroid disorders may significantly affect the clinical course of dental implant therapy. Patients with thyroid dysfunction are more prone to peri-implant inflammation, delayed tissue repair, and compromised healing following implant placement. Additionally, thyroid disease may intensify established risk factors for peri-implantitis—such as smoking, diabetes, and inadequate oral hygiene—thereby increasing the likelihood of implant-related complications. Consequently, thorough assessment of thyroid status should be incorporated into peri-implant disease prevention and management strategies. [11]

### 2.1 Thyroid Disorders

Thyroid disorders comprise a diverse group of conditions that impair the normal function of the thyroid gland, which plays a critical role in metabolic regulation, growth, and development. The most prevalent disorders include hypothyroidism, hyperthyroidism, and autoimmune thyroid diseases such as Hashimoto's thyroiditis and Graves' disease.

Hypothyroidism results from insufficient thyroid hormone production, leading to generalized metabolic slowing and symptoms such as fatigue, weight gain, cold sensitivity, dry skin, and constipation. In contrast, hyperthyroidism is caused by excessive hormone secretion, accelerating metabolic processes and manifesting as weight loss, palpitations, tremors, heat intolerance, and anxiety. The underlying mechanisms of thyroid dysfunction are complex and involve genetic susceptibility, environmental influences, and immune system dysregulation. [12,13,14]

### 2.2 Peri-Implantitis

Peri-implantitis is a progressive inflammatory condition affecting the soft and hard tissues surrounding dental implants, ultimately leading to peri-implant bone loss. The disease is multifactorial, arising from interactions between microbial biofilms, host immune responses, environmental influences, and implant-related factors. Bacterial plaque accumulation on implant surfaces plays a central role in disease initiation, with pathogens such as Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans frequently implicated. Additional contributing factors include smoking, poor plaque control, diabetes mellitus, genetic predisposition, and implant surface characteristics. Peri-implant mucositis often precedes peri-implantitis, underscoring the importance of early diagnosis.

Clinically, peri-implantitis presents with erythema, swelling, bleeding on probing, suppuration, increased probing depths, and radiographic evidence of progressive bone loss. Advanced disease stages may result in implant mobility and eventual failure. Effective management relies on early detection, risk factor control, and tailored therapeutic interventions. [15,16]

### 2.3 Hormonal Imbalances and Immune Dysregulation

Thyroid hormone disturbances can significantly influence immune competence and inflammatory responses, thereby affecting susceptibility to peri-implant disease. Hypothyroidism may impair immune surveillance, increasing vulnerability to infections and chronic inflammation. In contrast, hyperthyroidism can provoke excessive immune activation, contributing to persistent inflammation and tissue breakdown. [17]

Autoimmune thyroid disorders further complicate this relationship, as immune-mediated mechanisms may intensify peri-implant tissue destruction. Emerging evidence indicates that thyroid hormones directly interact with immune cells via specific transporters, receptors, and deiodinases, highlighting their role in immune regulation. [23]

***Key Points:***

- Thyroid hormone imbalance alters immune function
- Hypothyroidism is associated with increased infection risk
- Hyperthyroidism may promote excessive inflammatory responses
- Autoimmune thyroid disease can aggravate peri-implant inflammation [17]

**2.4 Altered Bone Metabolism**

Thyroid hormones are key regulators of skeletal remodeling, influencing bone formation and resorption.[18.] Variations in thyroid hormone levels—even within the normal reference range—have been associated with changes in bone mineral density and fracture risk. Thyrotoxicosis is a recognized cause of high-turnover osteoporosis, while subclinical hyperthyroidism and long-term TSH suppression therapy may also contribute to bone loss, particularly in postmenopausal women. Although anti-resorptive therapies such as bisphosphonates have been proposed to mitigate thyroid hormone–related bone loss, robust prospective clinical trials are lacking. Further research is needed to establish evidence-based therapeutic guidelines for managing skeletal complications in patients with thyroid dysfunction.[21]

## 3. Impact on Peri-Implant Healing and Treatment Outcomes

Thyroid disorders can adversely affect peri-implant wound healing and therapeutic success. Altered thyroid status may impair angiogenesis, soft tissue repair, and bone regeneration, increasing the risk of postoperative complications. Thyroid hormones act as powerful regulators of angiogenesis through both genomic and non-genomic pathways, influencing endothelial cell activity, vascular growth, and tissue remodeling.

Hypothyroidism is associated with delayed granulation tissue formation and prolonged wound closure, whereas hyperthyroidism or localized thyroid hormone application may enhance vascularization and accelerate healing.[22] Novel approaches involving topical hormone delivery, biomaterial scaffolds, and combination therapies with platelet-rich plasma or stem cells show promise, although further investigation is necessary to optimize clinical protocols.[18]

## 4. Preventive Measures

Preventive strategies are essential for reducing peri-implantitis risk in patients with thyroid disorders.

**4.1 Key Preventive Approaches**

**Oral hygiene optimization**: Emphasize effective plaque control through regular brushing, flossing, and interdental cleaning

**Smoking cessation**: Encourage smoking cessation to improve peri-implant tissue health

**Routine dental monitoring**: Schedule regular professional cleanings and examinations for early disease detection

**Metabolic control**: Maintain optimal glycemic control in diabetic patients to support healing and immune function [18]

**5. Therapeutic Considerations**

**5.1 Non-Surgical and Surgical Management**

Peri-implantitis treatment in patients with thyroid disease requires an individualized approach that may include both non-surgical and surgical modalities. [19,20]

**5.2 Treatment Options**

**Non-surgical therapy:** Mechanical debridement and biofilm removal to reduce inflammation

**Surgical therapy:** Open flap debridement, guided bone regeneration, and implantoplasty to manage advanced defects

**5.3 Adjunctive Therapies**

Adjunctive treatments may enhance conventional therapy outcomes and include:

**Antimicrobial agents**: Local or systemic antimicrobials to control bacterial load

**Host modulation therapy**: Anti-inflammatory agents to regulate immune responses

**Photodynamic therapy**: Light-activated antimicrobial strategies to disrupt biofilms

A comprehensive, multidisciplinary treatment approach combined with long-term maintenance is essential for preserving implant stability in this patient population. [19,20]

**Conclusion**

Thyroid hormones are key regulators of wound healing and angiogenesis, profoundly influencing tissue repair and vascular development. Reduced thyroid function is associated with delayed and incomplete healing, whereas adequate thyroid hormone activity supports effective tissue regeneration. Advances in localized thyroid hormone delivery and combination therapies offer promising avenues for improving surgical recovery and chronic wound management. Future research should focus on refining therapeutic protocols, identifying suitable patient populations, and ensuring long-term safety. A deeper understanding of thyroid-implant interactions will ultimately enhance clinical outcomes and patient care.

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