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Peri-implantitis as a potential risk factor for peri-implant oral malignancy

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Abstract (J Korean Assoc Oral Maxillofac Surg 2026;52:27-33)

Peri-implant oral malignancy (PIOM) refers to malignant tumors arising around dental implants and is an increasingly reported complication of implant therapy. PIOM may follow distinct pathophysiological mechanisms, including chronic peri-implant inflammation and implant-related factors that contribute to carcinogenesis. This current review aims to explore the potential role of peri-implantitis (PI) as a risk factor for PIOM, discussing the proposed pathogenic mechanisms, histological findings, and clinical implications. A comprehensive literature search was conducted in PubMed, Scopus, and Web of Science databases. Relevant case reports, clinical studies, and reviews on the keywords "PIOM" and "PI" published from 2019 up to 2025 were included and qualitatively analyzed. Clinicopathologic characteristics are summarized as location and morphology, disease progression, histopathology, and degree of differentiation, and pathophysiological hypotheses involve inflammatory and electrochemical pathways, epithelial barrier dysfunction, molecular alterations, microbiome dysbiosis, and immune dysregulation. Current evidence remains limited and primarily anecdotal. Several studies suggest that chronic inflammation, titanium particle exposure, corrosion byproducts, and sustained tissue damage in peri-implant tissues may contribute to oncogenesis. While a direct causal link between PI and PIOM remains unproven, chronic peri-implant inflammation may contribute to malignancy development in predisposed individuals. Clinicians should consider a biopsy when peri-implant lesions exhibit atypical features, promptly.

Key words: Peri-implant oral malignancy, Peri-implantitis, Current review, Risk factors, Dental implants

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I. Introduction

Dental implants have become a widely accepted and effective solution for oral rehabilitation in patients with partial or complete edentulism. However, the long-term presence of implants, along with chronic inflammation and peri-implant diseases, has raised concerns about the potential development of malignancies in the peri-implant region. The term peri-implant oral malignancy (PIOM) has emerged to describe malignant neoplasms arising in close association with dental implants. Various other terms have also been used in the literature, including oral peri-implant malignancy (OPIM), peri-implant oral squamous cell carcinoma (OSCC), and implant-associated oral cancer[1-3]. Despite differences in terminology, the underlying concern remains the same, distinguishing true malignancies from benign peri-implant pathologies, particularly peri-implantitis (PI). PI refers to chronic inflammation of peri-implant tissue involving the alveolar bone and surrounding soft tissues. Clinical manifestations of PI include gingival ulcers, hypertrophy, hyperplasia, marginal bone loss, and edematous swelling with erythema.

To date, the literature on PIOM remains limited, consisting mostly of isolated case reports and a few systematic reviews that attempt to explore the potential etiological factors and diagnostic challenges[4,5]. Some studies suggest that chronic inflammation induced by PI, mechanical or electrochemical irritation from prosthetic components, and pre-existing mucosal conditions may play a role in malignant transformation[1,2,4,5]. In particular, PI has been proposed as a possible risk factor for PIOM given its characteristic chronic inflammatory environment that may facilitate carcinogenesis in peri-implant tissues[2,4,6]. However, these associations remain largely speculative, as definitive causal relationships have not yet

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been established.

Given the increasing number of implants being placed worldwide, the potential for associated malignancies, although rare, warrants careful investigation. This review aims to summarize and critically discuss the existing literature on PIOM, including proposed pathophysiological mechanisms, reported risk factors with special attention to the potential role of PI, diagnostic challenges, and differential diagnosis. By synthesizing currently available data, we hope to provide clinicians and researchers with a clearer understanding of this emerging phenomenon and underscore areas that require further investigation.

II. Methods

After Seo et al.[3] coined the term PIOM in 2019, several related articles have been published. A comprehensive literature search was performed on PubMed, Scopus, and Web of Science. The search strategy used in this review consisted of relevant and updated studies from 2019 to 2025 that are associated with PIOM and PI, as a focus question of this review was, "Is PI a potential risk factor for PIOM?" Nine studies consisting of case reports, clinical studies, and review articles were selected for review.(Table 1)

Instead of Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) and other standard criteria, we applied Scale for the Assessment of Narrative Review Articles (SANRA) for a high-quality review process[7]. This tool evaluates narrative reviews based on six key criteria; (1) justification of importance, (2) description of concrete objectives, (3) methodology of the literature search, (4) referencing, (5) scientific reasoning, and (6) adequate data reporting.

III. Results

1. Definition and epidemiology of PIOM

The term PIOM encompasses all malignant tumors arising in close association with dental implants, most frequently OSCC. Various other terms including OPIM, peri-implant OSCC, and implant-associated oral cancer have also been used in the literature[1-3,5]. Epidemiological data remain scarce due to the rarity of the condition, but a previous study estimated that PIOM accounts for approximately 1.5% of all oral cancers[5].

2. Clinicopathologic characteristics

Several studies have described the distinctive but often deceptive clinicopathologic features of PIOM, such as location and morphology, disease progression, histopathology, and degree of differentiation.

1) Location and morphology

Seo et al.[2] reported that most PIOM lesions occurred in the mandible and typically presented as exophytic, ulcerated, or a combination of the two, frequently associated with marginal bone loss on radiographs. Gingival lesions exhibiting hyperplastic papillomatous growths, or presenting with a whitish keratotic surface, should be evaluated by biopsy at an early stage to exclude malignant transformation.(Fig. 1. A) The inflammation-like features of PIOM may lead clinicians to misdiagnose them as PI, which commonly exhibits inflammatory signs such as erythema, hyperplasia, swelling, suppuration, bleeding, and ulceration.(Fig. 1. B)

Table 1. Descriptive table of selected studies on PIOM and PI from 2019 to 2025

Study	Title	Study design	No. of patients
Patil et al.[1] (2024)	Evaluation of risk factors, clinicopathological aspects and implant characteristics in patients of oral peri-implant malignancies	Retrospective	46
Seo et al.[2] (2024)	Clinical retrospective analysis of peri-implant oral malignancies	Retrospective	21
Seo et al.[3] (2019)	Changes in oncogenic protein levels in peri-implant oral malignancy: a case report	Case report	1
Srinivasan et al.[4] (2025)	Peri-implantitis and peri-implant oral malignancies: a systematic review and meta-analysis of diagnostic challenges and potential associations	Systematic review, meta-analysis	161
Limongelli et al.[5] (2025)	Peri-implant OSCC: clinicopathological features and staging issues	Retrospective cohort	21
Špiljak et al.[6] (2024)	Oral microbiome research in biopsy samples of oral potentially malignant disorders and oral squamous cell carcinoma and its challenges	Narrative review	N/A
Łobacz et al.[13] (2025)	Dysregulation of the immune system in advanced periimplantitis: systemic implications and inflammatory mechanisms - a hematological and immunological study	Cross-sectional observational	74
Kim[9] (2023)	Oral galvanism related to dental implants	Review	N/A
Kim et al.[8] (2019)	General review of titanium toxicity	Review	N/A

(PIOM: peri-implant oral malignancy, PI: peri-implantitis, OSCC: oral squamous cell carcinoma, N/A: not available)

2) Disease progression

A key differentiating feature of PIOM is progression. Srinivasan et al.[4] emphasized that lesions unresponsive to conventional treatment for PI and showing rapid progression should raise strong suspicion for PIOM. While PIOM may share clinical and histological features with PI, its lack of response to routine treatment and progressive course are red flags indicating the need for biopsy.

3) Histopathology

Conventional hematoxylin-eosin staining often fails to differentiate PIOM from severe PI. As Limongelli et al.[5] noted, more than 90% of peri-implant OSCC cases exhibited histological PI-like inflammation. Histologically, both PI and PIOM may demonstrate epithelial hyperplasia, ulceration, or peri-implant bone resorption, complicating distinction on morphology alone. As the histology of PIOM is indistinguishable from that of PI, immunohistochemistry (IHC) for cytokeratin is frequently required to confirm malignancy infiltrating peri-implant bone.

4) Degree of differentiation

Many cases of PIOM are well-differentiated SCC, further complicating the differential diagnosis with similar clinical features[2].

These findings suggest that PIOM often mimics benign peri-implant disease both clinically and radiographically, leading to delayed diagnosis.(Table 2) Importantly, a systematic review and meta-analysis revealed that almost half of PIOM cases were initially misdiagnosed as PI, underscoring the significant diagnostic challenge posed by their overlapping clinical presentations[4]. Retrospective analyses from Korea and India highlight the frequent misinterpretation of PIOM as PI. Seo et al.[2] reviewed 21 patients and found that nearly all had a prior record of PI before malignant transformation, with most cases presenting as exophytic or ulcerated mandibular lesions. Similarly, Patil et al.[1] analyzed 46 cases and emphasized that all cases were previously treated for PI.

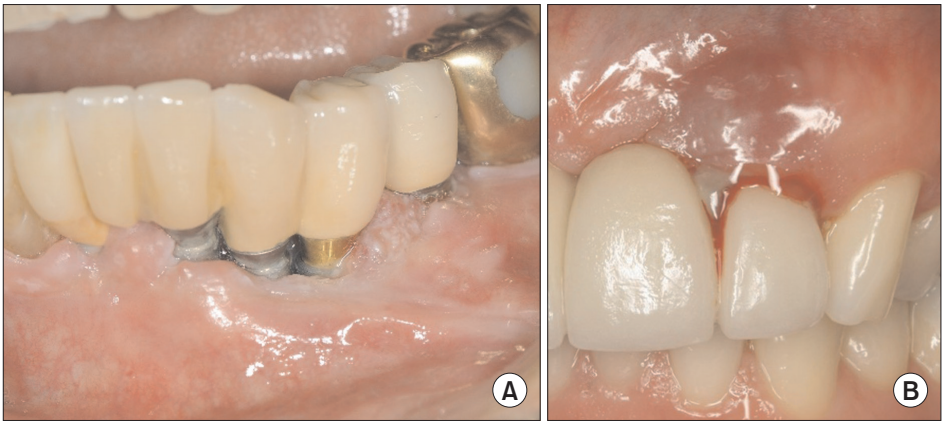

Fig. 1. A. Clinical features of peri-implant oral malignancy showing hyperplastic papillomatous gingival growths with prominent ulceration and swelling and severe plaque deposition around implant fixtures. B. In peri-implantitis, erythema, swelling, suppuration, bleeding, and ulceration are observed.
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Table 2. Clinical and histopathological characteristics of PI versus PIOM

Feature	PI	PIOM	Reference
Typical location	Both maxilla & mandible	Predominantly in mandible	2,5
Clinical appearance	Erythema, swelling, suppuration, hyperplasia/hypertrophy, ulceration, bleeding, pain, increased probing depth	Exophytic, ulcerated, or combined lesions; sometimes whitish gingival/alveolar covering; papillomatous growths	1,2,4
Radiographic findings	Horizontal or vertical bone loss, crater-like defects	Marginal bone loss with irregular, destructive pattern	2
Histology	Inflammatory infiltrate, epithelial hyperplasia, ulceration	Indistinguishable from PI on H&E; SCC confirmed by IHC	5
Degree of differentiation	Not applicable	Often well-differentiated SCC	2
Response to treatment	Improves with debridement, antimicrobial therapy, surgical management	Persistent despite PI therapy; rapid progression	4
Risk factors	Plaque, poor oral hygiene, prosthetic misfit	Prior PI history, occlusal/prosthetic irritation, galvanic current; OPMDs in some cases	1,5
Diagnostic challenge	Diagnosed clinically and radiographically	Frequently misdiagnosed as PI (≈50%); requires biopsy/IHC	4

(PI: peri-implantitis, PIOM: peri-implant oral malignancy, H&E: hematoxylin and eosin, SCC: squamous cell carcinoma, IHC: immunohistochemistry, OPMD: oral potentially malignant disorder)
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The most common clinical appearance of PIOM was exophytic-ulcerative growth. This overlap explains the frequent delay in diagnosis, as clinicians must initially manage the lesion as PI before suspecting malignancy.

3. Pathophysiological hypotheses

Multiple pathogenic mechanisms have been proposed for PIOM, including inflammatory and electrochemical pathways, epithelial barrier dysfunction, molecular alterations, microbiome dysbiosis, and immune dysregulation.

1) Inflammatory, mechanical, and electrochemical pathways

Both clinical studies and systematic reviews indicate that persistent peri-implant inflammation may drive malignant transformation, even in the absence of classical risk factors such as smoking or alcohol. Persistent inflammation can promote carcinogenesis by enhancing cellular proliferation and survival through oncogene activation and tumor suppressor gene inactivation. In addition, peri-implant OSCC has been reported in association with conventional oral cancer risk factors, as well as implant-specific contributors such as the presence of titanium implants, chronic inflammatory conditions, and prolonged immunosuppressive therapy[2,4].

Retrospective analyses report frequent associations of prosthetic rehabilitation, occlusal trauma, and galvanic currents, suggesting these as potential cofactors of PIOM[1,2]. Although the titanium or titanium alloys of an implant surface form an oxide layer, mechanical force or exposure to chemicals and other factors could cause galvanic corrosion in the saliva-filled oral environment.

We propose novel inflammatory, mechanical, and electrochemical mechanisms through which PI may drive progression to PIOM.(Fig. 2) Chronic inflammation in PI promotes long-term degradation of implant surfaces, increasing galvanic intensity. Corrosion and wear of implants release of titanium and titanium alloy particles and ions, which can generate reactive oxygen species (ROS) through NADPH oxidase[8,9]. The resulting oxidative stress induces DNA damage, providing a plausible pathway for carcinogenesis[4]. These galvanically generated agents may also stimulate proliferation of precancerous, leukoplakia cells, and induce apoptosis. Consistently, overexpression of the p53 tumor suppressor protein in PIOM tumor tissue and patient serum suggests mutations that compromise DNA repair and apoptosis regulation, further supporting a mechanistic link between chronic peri-implant inflammation and malignant transformation[3].

Mechanical stress, such as implant loading, may be a potential risk factor for medication-related osteonecrosis of the jaw, suggesting that prosthetic and occlusal irritation may cause peri-implant bone diseases[10]. Mechanical forces have been shown to regulate YAP/TAZ signaling[11], a key pathway

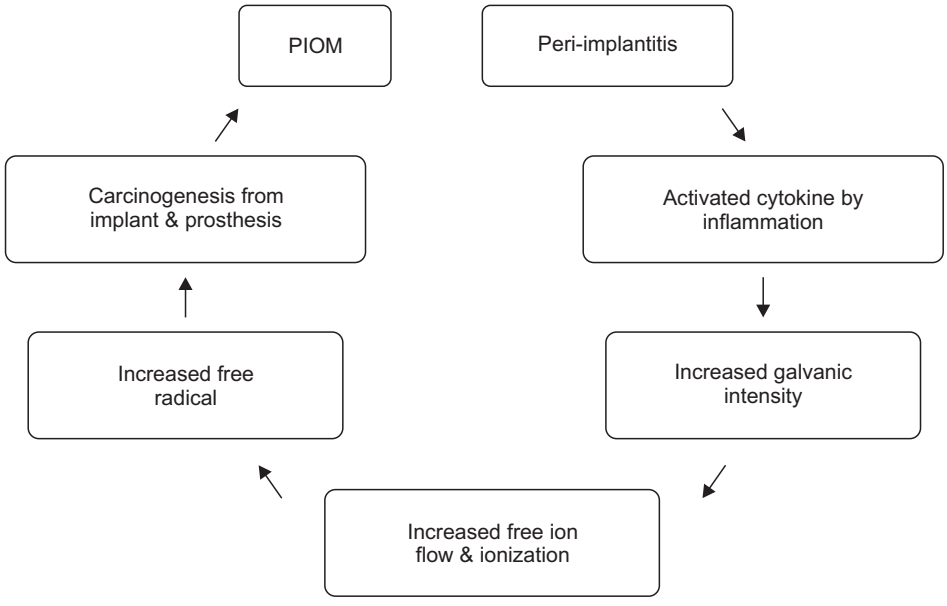

Fig. 2. Schematic representation of the progression from peri-implantitis (PI) to peri-implant oral malignancy (PIOM), involving cytokine release as an inflammatory response, galvanic intensity increases to generate free ion flow and ionization, and consequent increase in free radicals around implants and prostheses. These pathways may induce carcinogenesis and progression from PI to PIOM.
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implicated in OSCC progression[12]. Because dental implants transmit occlusal forces directly to peri-implant tissues, excessive or chronic mechanical loading may promote aberrant mechanotransductive signaling. Although direct evidence linking YAP/TAZ activation specifically to implant loading remains limited, it represents a biologically plausible mechanism connecting mechanical stress, refractory peri-implant lesions, and potential malignant transformation.

2) Epithelial barrier dysfunction

Chronic inflammation and sustained metallic particle exposure may compromise the implant sulcus epithelial barrier, facilitating the penetration of bacterial toxins and corrosion byproducts into peri-implant tissues. This breakdown might act as a gateway for malignant transformation[3,4].

3) Molecular alterations

A single-patient case report demonstrated fluctuations of oncogenic proteins (p53, HER2, β-catenin, etc.) following tumor resection, suggesting potential biomarker roles for these molecules in PIOM progression[3].

4) Microbiome dysbiosis

The review by Špiljak et al.[6] emphasized that peri-implant and OSCC tissues harbor distinct microbial communities. Pathogens such as *Fusobacterium nucleatum* and *Porphyromonas gingivalis* promote inflammation, immune modulation, and direct carcinogenic signaling.

5) Immune dysregulation

Łobacz et al.[13] reported that advanced PI patients exhibited systemic immune dysfunction, characterized by T-cell exhaustion (PD-1/PD-L1 upregulation) and reduced NK cell activity. These findings suggest that chronic peri-implant inflammation may induce systemic immunosuppression, potentially facilitating malignant progression.

Limongelli et al.[5] further demonstrated that peri-implant OSCCs were predominantly associated with oral premalignant diseases or prior OSCC, with worse prognosis in patients harboring these predisposing conditions. Peri-implant OSCCs may not represent a de novo malignancy but rather an exacerbation or recurrence arising within high-risk mucosa, contributing to poorer clinical outcomes. Taken together, these mechanisms support the hypothesis that PIOM may be a multifactorial disease involving persistent inflammation, surface degradation, epithelial barrier breakdown, microbial dysbiosis, host immune dysfunction, and molecular changes.

IV. Discussion

PIOM constitutes a rare but increasingly recognized clinical entity, and the nine most recent studies strongly suggest that PIOM might be not merely an extension of PI but an independent malignant lesion involving complex pathophysiology with systemic and molecular mechanisms. PI can establish a pro-carcinogenic microenvironment through immune suppression, tissue damage, and molecular and microbial alterations, functioning as a precursor or significant risk factor for PIOM development. Recognition of this distinction is essential, as delayed diagnosis is directly associated with adverse prognosis.

Persistent or refractory peri-implant lesions that remain unresponsive to both non-surgical and surgical conventional periodontal therapy for at least 3 months should undergo early biopsy reviewed by a multidisciplinary team including periodontists, pathologists, and surgical oncologists. Integration of histopathological evaluation, IHC, serum biomarkers, and molecular profiling may facilitate early detection, risk stratification, and prognostic assessment. The rarity of PIOM highlights the need for international collaborative research networks to enable long-term follow-up studies, standardized sampling, and molecular and microbiome analyses. Understanding the multifactorial risk factors, including the preneoplastic potential of PI, immune dysregulation, microbial dysbiosis, and mechanical or electrochemical stimuli from prosthetic components, can inform implant treatment planning and postoperative management.

1. Role of PI as a potential risk factor

The relationship between PI and PIOM is not definitively established; however, multiple reports support its role as a significant risk factor. Retrospective cohorts consistently note that most PIOM patients had a prior history of PI[1,2]. Moreover, cases have been reported in patients lacking traditional carcinogenic exposures such as tobacco, alcohol, and human papillomavirus (HPV), suggesting that peri-implant inflammation alone may initiate malignant transformation in predisposed individuals[4]. Histological analyses of PI biopsies have occasionally revealed dysplastic or atypical epithelial changes, further reinforcing the potential for malignant evolution[3]. When viewed alongside immunological and microbiome studies, PI emerges not merely as a local inflammatory disease but as a potential precursor environment for carcinogenesis through sustained cellular stress, dysregulated immune

surveillance, and altered microbial diversity[6,7].

2. Diagnostic considerations for PIOM

Given the frequent clinical and histological overlap, timely and accurate diagnosis remains the central challenge. Persistent peri-implant lesions that fail to respond to conventional periodontal treatment should always prompt biopsy. Srinivasan et al.[4] found that half of PIOM cases were misdiagnosed as PI at first presentation, delaying definitive treatment. Limongelli et al.[5] similarly noted that clinical and radiological signs mimicking PI accounted for the majority of diagnostic pitfalls.

Biopsy with histopathological evaluation remains the gold standard, but IHC staining and adjunctive biomarker assays may provide additional discriminatory value. Clinically, patients with risk factors such as oral potentially malignant disorders, previous OSCC, or refractory PI should be subject to heightened surveillance, shorter recall intervals, and a low threshold for tissue sampling.

3. Application of the SANRA criteria

The use of the SANRA criteria provided a structured framework for assessing clarity, objectivity, and comprehensiveness in this narrative review. The review was designed to clearly justify its rationale and objectives, describe the literature search methodology, and present scientific evidence supported by appropriate references. Applying this standardized tool helped minimize subjective bias and ensured that the review met accepted standards for scientific validity and transparency.

Given that this review is based on nine recent studies about PIOM, the body of evidence is limited and should be interpreted with appropriate caution. The few numbers of included studies and the heterogeneity in study design and outcome measures constrain the overall methodological rigor and limit the generalizability of the findings. Nevertheless, these limitations are partially offset by the inclusion of higher-level evidence, including retrospective and cross-sectional observational studies as well as a systematic review/meta-analysis, which extend the evidence base beyond isolated anecdotal observations. Furthermore, the included articles represent the most contemporary studies available, thereby reflecting current clinical practice and emerging research directions. Collectively, these factors provide a more comprehensive and up-to-date overview of the existing literature, while emphasizing that the findings remain largely exploratory and warrant confirmation through well-designed prospective studies.

V. Conclusion

Conclusively, diagnostic challenges, optimal biopsy timing, and systemic and microbial imbalance provide critical evidence to support early detection, identification of high-risk patients, and development of individualized therapeutic strategies for PIOM. The insights from nine recent studies underscore the necessity of early diagnosis, a multidisciplinary clinical approach, and global research collaboration to improve patient outcomes and advance understanding of the emerging entity of PIOM.

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Authors' Contributions

Y.L. participated in data collection and writing the manuscript. K.R.M., S.M.K., and E.M.Y. participated in the study design. Y.J.C. participated in the coordination and assisted with formatting the figures. All authors read and approved the final manuscript.

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Ethics Approval and Consent to Participate

The study protocol complied with the principles of the Declaration of Helsinki and was approved by the Seoul Na-

tional University Institutional Review Board (S-D20170026). All methods were performed in accordance with the relevant guidelines and regulations. The patient was informed of the surgical procedure with the potential risks and benefits, and an informed consent was obtained to receive the treatment and to be included in the study.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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