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Review article

Peri-implantitis and peri-implant oral malignancies: A systematic review and meta-analysis of diagnostic challenges and potential associations☆

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ABSTRACT

Objective: This systematic review and meta-analysis aimed to investigate the potential association between peri-implantitis (PI) and peri-implant oral malignancies (PIOM).
Data and Sources: Systematic searches were conducted in Medline (PubMed), Embase, CENTRAL, and Web of Science up to December 10, 2024. Gray literature and reference lists were also searched. The protocol was registered in PROSPERO (CRD42024622566). Data synthesis involved descriptive analyses, Kaplan–Meier survival estimates, and a meta-analysis of proportion.
Study Selection: Studies were eligible if they reported PIOM cases. Animal studies, in vitro studies, reviews, and conference proceedings were excluded. A meta-analysis included retrospective studies reporting more than five PIOM cases. Inter-investigator reliability was assessed using Cohen's kappa statistic.
Conclusions: Fifty-seven studies (51 case reports/case series, 6 retrospective studies) involving 161 patients were included. Although the current evidence, limited to retrospective analyses and case reports, was insufficient to establish a definitive link, this review synthesized available data to provide preliminary insights and highlight directions for future research. The meta-analysis revealed that 50 % of PIOM cases were initially misdiagnosed as PI, with squamous cell carcinoma being the most common final diagnosis (81 % in case reports and 97 % in retrospective studies). The median time from implant placement to PIOM diagnosis was 5 years, irrespective of traditional risk factors such as smoking, alcohol consumption, or previous cancer history. Although the evidence suggests a potential link between chronic peri-implant inflammation and malignant transformation, the limited nature of the available data highlights the need for prospective studies to establish causality and improve diagnostic protocols.
Clinical significance: PIOM can mimic peri-implantitis, leading to misdiagnosis and delayed treatment. Clinicians should maintain a high index of suspicion for malignancy in persistent peri-implant lesions unresponsive to conventional therapy, emphasizing the need for timely biopsy and histopathological evaluation, even in patients without traditional risk factors.

1. Introduction

Peri-implant disease is a broader term encompassing both peri-implant mucositis (PIM) and peri-implantitis (PI). PIM is limited to soft tissue inflammation without radiographic bone loss, whereas PI involves both soft tissue inflammation and progressive peri-implant bone loss [1]. A recent systematic review reported the prevalence of PI is approximately 20 % at the patient level and 13 % at the implant level

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[2]. Patient-level factors, including poor oral hygiene, smoking, diabetes, and history of periodontitis, contribute to the development of PI [3]. The clinical appearance of PI includes hyperplasia, ulceration, bleeding, suppuration, swelling, and pain, while radiological exams reveal bone loss around the affected implant [4].

The presence of oral cancer, particularly around dental implants, is rare [5]. However, a differential diagnosis should be considered in cases of persistent lesions that do not respond to conventional treatments and show rapid progression [6–8]. Early diagnosis of oral malignancy is directly associated with treatment success rates. It is crucial to perform a differential diagnosis, especially in patients with recognized risk factors such as tobacco use, alcohol consumption, and a history of cancer [9–11]. While both PIM and PI represent inflammatory responses around implants, PI is generally more severe and shares closer clinical and radiographic similarities with peri-implant oral malignancies (PIOM), potentially masking the malignant lesions in their early stages [12].

Moreover, an increasing number of reports describe patients developing PIOM despite the absence of traditional risk factors, with PI-like clinical features being the most commonly observed findings [13–18]. These cases suggest a potential role of chronic inflammation around dental implants in malignant transformation [19] and highlights the need for improved clinical awareness and diagnostic accuracy [4,6,8].

This systematic review aimed to assess the potential association between peri-implant inflammation and oral malignancies. Specifically, this review was designed to answer two key questions: (1) Could peri-implant inflammation serve as a risk factor for oral malignancies? and (2) What is the incidence of misdiagnosis of PIOM as PI in patients with dental implants?

2. Materials and methods

This systematic review and meta-analysis were conducted and reported according to the PRISMA guidelines [20,21]. The review protocol was registered with PROSPERO: International prospective register of systematic reviews (CRD42024622566)

2.1. Eligibility criteria and information sources

The complete list of inclusion and exclusion criteria for this systematic review, along with the sources of information used to identify relevant records, are detailed in Table 1.

2.2. Search strategy and selection process

The search terms were identified based on the PICO (population, intervention comparison outcome) criterion in database searches. The search terms were medical subject headings (MeSH) in the “all fields” category and were combined using appropriate Boolean operators (“OR” / “AND”) to structure the search strategy. The complete list of search terms and the implemented strategy are described in Table 1.

Four electronic databases were searched: Medline (PubMed), Embase, the Cochrane Central Register of Controlled Trials (CENTRAL) and Web of Science. An initial electronic search was performed by the first reviewer (T.F.), and the search strategy was repeated by a second reviewer (K.S.) to confirm the number of discovered articles.

The results from each database were imported into Rayyan (https://www.rayyan.ai), a web-based collaboration software platform that streamlines the production of systematic reviews. The first reviewer (T.F.) analyzed and removed duplicate records. Both reviewers (T.F. and K.S.) independently performed a thorough title and abstract screening. Discrepancies in this initial screening were resolved by consensus, and the shortlisted studies were selected for a full-text analysis. Subsequently, the selected articles with the full text available were read by both reviewers and the final articles were chosen for the data extraction based on mutual agreement.

Table 1
Focus question, criteria for inclusion, sources of information, search terms, search strategy, search filters, and search dates.

Focus questions	(1) Is peri-implant inflammation a potential risk factor for peri-implant oral malignancies (PIOM)? (2) In patients with dental implants, what is the incidence of misdiagnosis of PIOM as peri-implantitis (PI)?	
Criteria	Inclusion criteria	• Clinical studies reporting the risk or occurrence of oral cancer in the patients with dental implants. • Clinical studies include randomized controlled trials, cross-sectional studies, case-control studies, cohort studies, retrospective studies, longitudinal studies, or case reports/series. • The risk of oral cancer is defined as the occurrence of oral cancer in proximity to dental implants. • Oral cancer includes peri-implant oral malignancies, squamous cell carcinoma (SCC), verrucous carcinoma, mucoepidermoid carcinoma, adenoid cystic carcinoma, basal cell carcinoma, adenocarcinoma, lymphomas, melanoma.
	Exclusion criteria	• Reviews • Congress proceedings • Animal studies • Invitro- and proof-of-concept experiments.
Information sources	Electronic databases	Medline (PubMed), Embase, CENTRAL, Web of Science
	Journals	All peer reviewed journals available online in databases: Medline (PubMed), Embase, CENTRAL, Web of Science
	Others, including gray literature sources	Popular online internet search engines (e.g. Google, Yahoo, Bing), Online internet research community websites (https://www.researchgate.net/), reference crosschecks, personal communications, hand-searches.
Search Terms (*PICO*)	Population:	Adult patients MeSH Terms: Humans; Adult
	Intervention or exposure:	#1: dental implants OR peri-implant inflammation OR peri-implant mucositis OR peri-implantitis
	Comparison:	Patients with dental implants without a history of peri-implant mucositis or peri-implantitis
	Outcome:	#2: mouth neoplasms OR oral cancer OR carcinoma OR squamous cell carcinoma OR verrucous carcinoma OR mucoepidermoid carcinoma OR adenoid cystic carcinoma OR basal cell carcinoma OR adenocarcinoma OR lymphomas OR melanoma OR peri-implant oral malignancy) AND (risk OR risk factors OR association OR relation OR diagnosis OR prevalence OR incidence OR odds ratio OR relative risk OR hazard ratio OR risk ratio OR incidence rate ratio OR hazard rate ratio OR risk difference OR number needed to harm OR percentage OR proportion
Filters	Language	Not applied

(*continued on next page*)

Table 1 (*continued*)

	Species	Humans [MeSH]
	Ages	Not applied
	Journal categories	Not applied
Search Builder	Search combination	(#1 AND #2)
Search query as performed in the electronic databases	PubMed (Medline), Embase, CENTRAL, Web of Science	(dental implants OR peri-implant inflammation OR peri-implant mucositis OR peri-implantitis) AND ((mouth neoplasms OR oral cancer OR carcinoma OR squamous cell carcinoma OR verrucous carcinoma OR mucoepidermoid carcinoma OR adenoid cystic carcinoma OR basal cell carcinoma OR adenocarcinoma OR lymphomas OR melanoma OR peri-implant oral malignancy) AND (risk OR risk factors OR association OR relation OR diagnosis OR prevalence OR incidence OR odds ratio OR relative risk OR hazard ratio OR risk ratio OR incidence rate ratio OR hazard rate ratio OR risk difference OR number needed to harm OR percentage OR proportion)
Search dates	Publications until December 2024	A final confirmatory online search was performed on 10 December 2024. No further online searches were conducted after this date

Further searches were conducted to identify additional studies from gray literature sources, including internet search engines (e.g., Google), academic research community websites (e.g., ResearchGate), reference cross-checks, personal communication with authors, and hand-searches of relevant conference materials or non-indexed publications. The final update for all the searches was performed on December 10, 2024.

2.3. Data collection process and data items

Information relating to type of article, demographic aspects including sex, age, location (maxilla or mandible) and association factors (smoking and/or alcohol consumption, the presence or previous history of oral potentially malignant disorder or previous cancer, history of periodontitis, as well as prosthesis type) were extracted from the identified records. The clinical presentation (exophytic lesions, ulcerations, white and red lesions) and bone loss was also collected.

Additionally, data regarding the diagnostic process such as, time until diagnosis after implant placement, previous PI diagnosis, initial clinical diagnosis, final diagnosis (including misdiagnosis) were extracted. The data extraction was performed by two reviewers (T.F. and K.S.), who were reciprocally blinded to each other's data extraction.

2.4. Summary measures and synthesis of results

Cohen's unweighted kappa (κ) was used to assess inter-investigator reliability [22]. A meta-analysis of proportions was conducted on the included retrospective studies that reported more than five PIOM cases. The proportion was calculated by dividing the number of initial PI diagnosis cases by the total number of PIOM cases. The inverse variance (IV) method was applied to estimate the pooled patient-level proportion with corresponding 95 % confidence intervals (*CIs*). The meta-analysis was performed using MetaAnalysisOnline.com, a web-based tool built on R utilizing the meta, metafor, and RTSA packages [23]. The I-squared (I^2) statistic was used to assess heterogeneity among studies. A random-effects model was applied to account for the expected clinical and methodological variability across the included retrospective studies, such as differences in populations, follow-up durations, and diagnostic protocols [24].

2.5. Publication biases and additional analyses

A descriptive analysis was performed on all included studies to report their country, implant location, clinical appearance of the lesion, factors, initial and final diagnosis, and time until final diagnosis. Time-to-incidence analysis was performed on pooled patient-level data from included case reports and case series, using IBM SPSS Statistics version 29.0.2.0 (IBM Corp., Armonk, NY, USA), employing Kaplan–Meier survival estimates and log-rank tests to assess differences across subgroups. Publication bias was assessed using Egger's statistical test and funnel plots when the meta-analysis included more than ten studies. In cases of fewer than ten, the publication bias assessment was not conducted [25].

3. Results

3.1. Study selection and study characteristics

The search strategy retrieved 3288 records: Medline (PubMed) (n = 775), CENTRAL (n = 80), Embase (n = 1477), Web of Science (n = 956). After excluding 842 duplicate records, 2446 records were screened. Following a comprehensive evaluation of titles and abstracts, 2344 articles were excluded. Inter-reviewer agreement during this title and abstract screening phase was moderate, with a κ value of 0.5. Subsequently, a full-text review was conducted on 102 studies, of which 49 were excluded based on: full text not available (n = 3), wrong population (n = 26), wrong publication type (n = 5), reviews (n = 9), wrong outcome (n = 4) and wrong study design (n = 2). Agreement between reviewers during full-text screening was substantial, with a κ value of 0.8. A cross-check of the reference of the included articles was performed, and 4 additional records were identified and included. Finally, 57 studies remained for data extraction and final analysis in the review. The final analysis included 51 case reports and case series [1,4,5,12,13,15–19,26–66] involving 68 patients, along with 6 retrospective studies [7,9,10,14,67,68] that each reported more than five cases of PIOM, comprising of 93 patients. The study selection process is summarized in a PRISMA flow diagram (Fig. 1). Details of the excluded studies, along with the reasons for their exclusion, are provided in **Supplementary material 1**.

3.2. Synthesis of results: descriptive analysis of the included studies

All included studies are reported descriptively in Tables 2–4.

3.2.1. Demographic characteristics

A total of 68 cases of PIOM were identified from 51 published case reports and case series spanning from 1983 to 2024. The mean age was 67.57 ± 10.06 years, ranging from 38 to 90 years. In the retrospective studies, which included 93 patients, the mean age was similar, varying between 60.43 to 73.4 years. In the case reports, the distribution between sexes was relatively balanced, with 31 males (45.59 %) and 37 females (54.41 %). However, in the retrospective studies, women were more affected, with 53 cases compared to 40 cases in men. In the total cases (161 patients) females were more affected than males (55.9 % vs. 44.1 %).

In terms of the geographical distribution of published case reports and case series, Japan and the United States reported the highest number of cases, with 10 cases each. Israel and Spain followed, each contributing 8 cases, while the United Kingdom reported 7 cases. Other countries with notable case contributions included Korea (6 cases), The Netherlands (5 cases), and Italy (3 cases). Several countries, including Belgium, China, France, India, Portugal, Sweden, and Switzerland, reported one case each (**Supplementary material 2**).

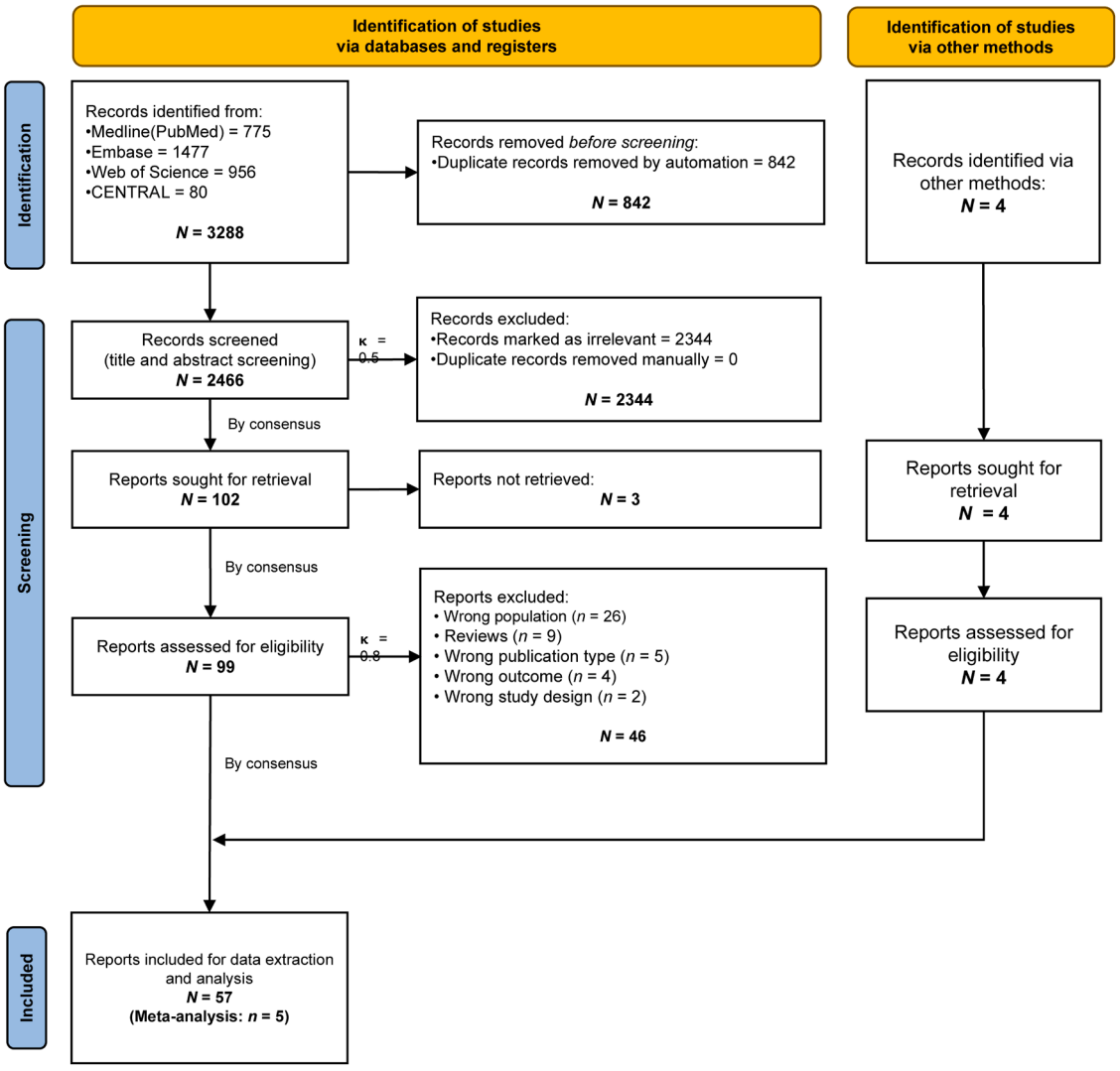

Fig. 1. PRISMA flow diagram summarizing the study selection process, including records identified, duplicates removed, records screened, and studies included in the meta-analysis. *N* or *n*, number; κ, Cohen's Kappa value.

3.2.2. Clinical features

Regarding lesion location, most case reports identified lesions in the mandible (82.35 %), while 16.18 % were in the maxilla, and a single case (1.47 %) involved both the maxilla and mandible. The most common lesion appearance was exophytic (28.36 %), followed by multifaceted (23.88 %) and ulcerative lesions (22.39 %). Other presentations included non-specific lesions (14.93 %), erythroplakia or leukoplakia (5.97 %), and fistulas with purulence (4.48 %). Bone loss was observed in 93.88 % of cases that reported this information, while only 6.12 % showed no bone loss. In retrospective studies, the mandible remained the predominant location (66.67 %), and ulcerative lesions were the most frequently reported clinical presentation (43.01 %), followed by exophytic lesions (29.03 %).

3.2.3. Factors

Regarding the 68 patients included in the case reports and case series, among the 40 patients with available smoking history, 47.50 % had a history of smoking or tobacco use, while 52.50 % were non-smokers. Similarly, 32 patients (43.75 %) had a history of alcohol consumption, whereas 56.25 % did not. A history of previous cancer was recorded in 53.85 % of cases, while 46.15 % had no such history. Only two reports explicitly mentioned a history of periodontitis, one case had a documented history of periodontitis, while the other reported no prior periodontitis. Among 57 cases with reported implant-supported prosthesis type, overdentures represented the highest proportion of PIOM cases (43.86 %), followed by single crowns (22.81 %), fixed partial dental prostheses (21.05 %), and fixed complete dental prostheses (12.28 %). Other suspected risk factors were identified in 25 patients, with recurrence being the most frequent (40 %), followed by oral lichen planus (OLP) (28 %), metastasis (20 %), trauma (8 %), and ill-fitting dental implant (4 %). Notably, seven cases [5,13,15–18,26] were reported in which patients developed PIOM despite having no history of smoking, alcohol consumption, prior cancer, or other recognized risk factors. In the retrospective studies, smoking was the most reported factor, in 33 patients (35.48 %), followed by alcohol use (22.58 %). A history of previous cancer was present in 34 cases, with oral cancer accounting for 27 cases and other types of cancer for 7 cases. Other factors, such as oral potentially malignant disorders (OPMD), were documented in 24 cases, while OLP was identified in 13 cases.

3.2.4. Diagnosis

An initial misdiagnosis was common among the included case reports, with 42.11 % of cases initially diagnosed as PI. Meanwhile, 43.86 % were correctly identified as squamous cell carcinoma (SCC), 10.53 % received other diagnoses, and 3.51 % were misdiagnosed as PIM. Following histopathologic analysis, SCC was confirmed as the most

Table 2
Descriptive table of included PIOM case reports and case series.

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
1	1	Ardakani et al., 2016	M	78	Iran	Max (ant.)	Multifaceted (exophytic lesions, pigmented lesions)	N	–	–	N	–	Fixed full arch implant supported denture	–	–	Melanoma	8 y
2	2	Bhandari et al., 2016	F	71	India	Max (#26)	Erythroplakia	Y	N	N	N	–	Single crown	–	Peri-implant mucositis	SCC (well-differentiated)	1.5 y
3	3	Bhatavadekar et al., 2012	M	54	USA	Max (#16)	Exophytic lesions	Y	N	N	N	–	Single crown	–	SCC	SCC (low-grade)	<1 y
4	4	Block et al., 2001	M	72	USA	Mand (ant.)	Multifaceted (exophytic lesions, fistulas and purulence)	Y	Y (prior smoking)	–	Y (left posterior mandible)	–	Bar retained overdenture	Recurrence	SCC	SCC	–
5	5	Chainani-Wu et al., 2016	F	60	USA	Mand (#37)	Fistulas and purulence	Y	N	N	Y (tongue)	–	Single crown	–	Peri-implantitis	SCC (well-differentiated)	4 y
6	6	Chimenos-Küstner et al., 2008	F	62	Portugal	Mand (#31, 41)	Exophytic lesions	–	Y (prior smoking)	Y	N	–	Not delivered	–	SCC	SCC (moderately well-differentiated)	few weeks
7	7	Clapp et al., 1996	F	65	USA	Mand	–	–	N	N	–	–	–	–	–	SCC	3 y
	8		M	79	USA	Mand (right post.)	Ulcerative lesions	–	N	Y (occasionally)	–	–	–	–	–	SCC	4 y
	9		F	90	USA	Mand	Ulcerative lesions	–	Y (prior smoking)	Y (1 drink per day)	Y (buccal mucosa)	–	-	–	–	SCC	7 y
8	10	Coopman et al., 2019	F	76	Belgium	Mand (#43)	Multifaceted (exophytic lesions, erythroplakia, fistulas and purulence)	N	N	N	N	–	Bar retained overdenture	–	Peri-implant mucositis	SCC (well-differentiated, invasive)	8 y
9	11	Czerninski et al., 2006	F	52	Israel	Mand (left post.)	Exophytic lesions	Y	Y	N	N	–	Splinting implant	OLP	Peri-implantitis	SCC (well-differentiated, superficially invasive)	3 y
	12		M	80	Israel	Mand (right post.)	Multifaceted (ulcerative lesions, exophytic lesions)	Y	N	N	Y (left buccal mucosa, carcinoma of the colon with liver metastasis)	–	Bar-retained overdenture	Recurrence	SCC	SCC (moderately-differentiated, invasive)	5 y
10	13	De Ceulaer et al., 2010	F	77	The Netherland	Mand	Non-specific (peri-implantitis-like lesions)	–	–	–	Y (anterior floor of mouth)	–	Overdenture	Recurrence	Peri-implantitis	SCC (well-differentiated)	3 y 6 m

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Table 2 (*continued*)

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
	14		M	71	The Netherland	Mand (right post.)	Non-specific (inflammatory lesion)	–	–	–	Y (right floor of mouth)	–	Overdenture	–	SCC	SCC	7 m
	15		F	62	The Netherland	Mand (left post.)	Non-specific (peri-implantitis-like lesions)	–	–	–	Y (anterior floor of mouth)	–	-	–	Peri-implantitis	SCC (moderately-differentiated)	6 m
11	16	Dib et al., 2007	F	67	Brazil	Max, Mand	Ulcerative lesions	Y	–	–	Y (breast with metastases to BBLL)	–	Not delivered	Metastasis	Metastatic carcinoma from the breast	Metastatic carcinoma from the breast	1 y
12	17	Eguia del Valle et al., 2008	M	76	Spain	Mand (#45, 46)	Exophytic lesions	Y	N	N	N	–	Splinting implant	–	Peri-implantitis	SCC (well-differentiated)	5 y
13	18	Favia et al., 2015	F	66	Italy	Mand (ant.)	Multifaceted (endophytic lesions, fistulas and purulence: necrotic bone exposure)	Y	–	–	Y (breast)	–	–	Metastasis	MRONJ	Metastatic-breast cancer	5 y
14	19	Friedman & Vernon 1983	M	65	USA	Mand	Multifaceted (ulcerative lesions, exophytic lesions, erythroplakia)	Y	Y	–	–	–	Bar-retained overdenture (staple implant)	–	–	SCC	–
15	20	Gallego et al., 2008	F	81	Spain	Mand (right post.)	Exophytic lesions	–	N	N	Y (left posterior mandible)	–	Overdenture	Recurrence	SCC	SCC	3 y
16	21	Gallego et al., 2009	F	70	Spain	Mand (left post.)	Ulcerative lesions	–	N	N	N	–	Bar-retained overdenture	Trauma from cantilever	SCC	SCC (moderately well-differentiated)	10 y
17	22	Granados et al., 2020	M	83	Spain	Mand	Ulcerative lesions	Y	–	–	Y (gum)	–	Overdenture	–	Not specified but treated with implantoplasty	SCC (well-differentiated invasive)	8 y
	23		M	60	Spain	Mand (#37)	Exophytic lesions	Y	–	–	–	–	–	–	SCC	SCC (well-differentiated)	–
	24		F	54	Spain	Mand (right post.)	Non-specific (gum lesion)	Y	–	–	–	–	Overdenture	–	SCC	SCC	–
	25		M	64	Spain	Mand	Exophytic lesions	Y	–	–	–	–	Overdenture	–	SCC	SCC (poorly-differentiated)	2 y
18	26	Gulati et al., 2009	F	62	UK	Mand (ant. and left post.)	Non-specific (peri-implantitis-like lesions)	–	Y	–	Y (left posterior mandible)	–	Bar-retained overdenture	Recurrence	Peri-implantitis	SCC	8 y

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Table 2 (*continued*)

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
19	27	Hee Oh et al., 2018	M	75	Korea	Mand (#36)	Multifaceted (exophytic lesions, erythroplakia)	Y	–	–	N	–	Not delivered	–	Peri-implantitis	Solitary plasmacytoma	2 m
	28		M	43	Korea	Mand (right post.)	Ulcerative lesions	Y	–	–	–	–	Single crown	–	SCC	SCC (well-differentiated)	1 y
20	29	Ito et al., 2017	M	62	Japan	Max (#21)	Ulcerative lesions	–	Y (prior smoking)	Y	N	Y	Single crown	–	Peri-implantitis	SCC	6 y
21	30	Jang et al., 2020	F	56	Korea	Mand (#35, 36, 37)	Fistulas and purulence	Y	–	–	Y (thyroid)	–	Splinting crown	–	Peri-implantitis	SCC (well-differentiated)	4 y
22	31	Jin et al., 2016	M	55	Korea	Mand (#47)	Fistulas and purulence	Y	–	–	Y (diffuse large B-cell lymphoma)	–	Splinting crown	Metastasis	Peri-implantitis	B-cell lymphoma (diffuse, large)	–
23	32	Kataria et al., 2024	M	74	USA	Mand	Ulcerative lesions	Y	Y (prior smoking)	–	–	–	Overdenture	trauma from overdenture	Traumatic ulcer	SCC	9 m
24	33	Kwok et al., 2008	M	62	UK	Mand (right post.)	Ulcerative lesions	–	Y	Y	N	–	Overdenture	–	SCC	SCC	3 m
	34		M	71	UK	Mand	Non-specific (inflammatory lesion)	–	Y (prior smoking)	Y	N	–	Overdenture	–	SCC	SCC	6 y
	35		F	67	UK	Mand	Exophytic lesions	–	Y (prior smoking)	Y	Y (tongue and breast)	–	Overdenture	–	SCC	SCC	1 y
25	36	Lubamba et al., 2024	M	59	China	Max (#16)	Multifaceted (exophytic lesions, pigmented lesions)	Y	–	–	–	–	Single crown	Ill-fitting implant	Pigmented oral diseases	melanoma	3 y
26	37	Malthiéry et al., 2019	M	73	France	Mand (right post.)	Exophytic lesions	Y	N	–	N	–	Bar-retained overdenture	–	Peri-implantitis	SCC (keratinizing differentiated)	–
27	38	Marini et al., 2013	F	51	Italy	Mand (#44)	Exophytic lesions	Y	N	N	N	–	Bridge	–	Peri-implantitis	SCC (moderately-differentiated)	4 y
28	39	McGuff et al., 2008	F	38	USA	Max (#16)	Non-specific (pain and swelling)	N	–	–	N	N	Single crown	–	Peri-implantitis	Chondroblastic osteosarcoma (low-grade)	11 m
29	40	Meijer et al., 2010	F	65	The Netherland	Mand (ant.)	Exophytic lesions	–	–	–	Y (right anterior floor of mouth)	–	Bar-retained overdenture	Recurrence	SCC	SCC	4 y
30	41	Moshref et al., 2011	F	67	Iran	Mand (right post.)	Exophytic lesions	Y	N	N	N	–	Overdenture	–	Not specified but treated with antibiotics	SCC (moderately-differentiated)	1 y

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Table 2 (*continued*)

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
31	42	Moxley et al., 1997	F	74	USA	Mand (ant.)	Exophytic lesions	Y	N	N	Y (verrucous carcinoma at left posterior mandible)	–	Overdenture (staple implant)	Recurrence	SCC	SCC (moderately well differentiated)	10 y
32	43	Murai et al., 2022	M	78	Japan	Mand (#35)	Ulcerative lesions	–	Y	Y	N	–	Single crown	–	Peri-implantitis	SCC (carcinoma cuniculatum, rare SCC subtype)	–
33	44	Naaj et al., 2007	F	70	Israel	Mand (ant.)	Exophytic lesions	Y	N	–	Y (thyroid and Breast)	–	Splinting implant	–	–	SCC	12 y
	45		M	72	Israel	Mand (#43)	Ulcerative lesions	Y	Y	–	N	–	Overdenture	OLP	–	SCC	15 y
34	46	Nariai et al., 2016	F	58	Japan	Mand (right post.)	Exophytic lesions	Y	Y	Y	Y (right lower gingiva)	–	Overdenture	Recurrence	SCC	SCC	3 y
35	47	Nestares et al., 2018	F	85	Spain	Mand (#34, 45, 46)	Erythroplakia and Leukoplakia	–	Y	–	–	–	Single crown	–	–	SCC (poorly-differentiated)	–
36	48	Noguchi et al., 2017	F	65	Japan	Mand (#46)	Multifaceted (exophytic lesions, ulcerative lesions)	Y	N	Y (socially)	Y (breast)	–	Splinting implant	–	Peri-implantitis	Oral intra-epithelial neoplasia/ carcinoma in situ (OIN/CIS)	7 y
37	49	Noguchi et al., 2019	F	78	Japan	Mand (left post.)	Erythroplakia and Leukoplakia	Y	N	Y (socially)	N	–	Fixed full arch implant supported denture	OLP	Peri-implantitis	SCC	–
38	50	Ogane et al., 2023	F	72	Japan	Mand (#46, 47)	Exophytic lesions	Y	N	N	–	–	Single crown	–	Peri-implantitis	Primary ameloblastic carcinoma	37 y
39	51	Osawa et al., 2024	M	79	Japan	Mand (#37)	Exophytic lesions	Y	Y	Y	Y (lung cancer)	–	Splinting implant	–	Peri-implantitis	SCC (well-differentiated)	12 y
	52		M	62	Japan	Mand (right post.)	Ulcerative lesions	Y	Y	Y	–	–	Splinting implant	–	Peri-implantitis	SCC (well-differentiated)	–
	53		M	68	Japan	Max (left post.)	Ulcerative lesions	Y	N	Y	N	–	Single crown	OLP	SCC	SCC (well-differentiated)	–
40	54	Park et al., 2022	M	57	Korea	Max (#26)	Ulcerative lesions	Y	Y	N	N	–	Single crown	–	SCC	SCC (moderately well-differentiated, verrucous)	17 y
41	55	Pfammatter et al., 2012	F	55	Switzerland	Mand (ant.)	Multifaceted (exophytic lesions,	Y	–	–	Y (pancreatic carcinoma and non-small cell lung cancer)	–	Long span bridge	metastasis	Peri-implantitis	Pulmonary cancer metastasis	–

(*continued on next page*)

Table 2 (*continued*)

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
							ulcerative lesions)										
42	56	Poggio et al., 2007	F	75	Italy	Mand (ant.)	Multifaceted (exophytic lesions, ulcerative lesions)	Y	–	–	Y (plasmacytoma at spine)	–	Bar-retained overdenture	Recurrence	Plasmacytoma	Plasmacytoma	3 y
43	57	Raiser et al., 2016	M	72	Israel	Max (#23)	Multifaceted (exophytic lesions, ulcerative lesions)	Y	–	–	–	–	Fixed full arch implant supported denture	–	Peri-implantitis	B-cell lymphoma	–
	58		F	55	Israel	Mand (left post.)	Multifaceted (exophytic lesions, leukoplakia)	Y	–	–	–	–	Fixed full arch implant supported denture	OLP	SCC	SCC (well-differentiated)	–
	59		F	70	Israel	Mand (right post.)	Multifaceted (exophytic lesions, erythroplakia)	Y	–	–	–	–	Single crown	OLP	SCC	SCC	–
44	60	Schache et al., 2008	M	77	UK	Mand (left post.)	Exophytic lesions	Y	–	–	N	–	Splinting implant	–	SCC	SCC (moderately-differentiated)	5 y
45	61	Seo et al., 2019	M	73	Korea	Mand (#42, 43, 44)	Leukoplakia	Y	N	N	–	–	Splinting implant	Recurrence	Peri-implantitis	SCC	10 y
46	62	Shaw et al., 2004	M	67	UK	Mand (left post.)	Exophytic lesions	Y	–	–	Y (left buccal mucosa, labial sulcus)	–	Fixed full arch implant supported denture	–	Peri-implantitis	SCC	5 y
	63		F	69	UK	Mand (right post.)	Multifaceted (exophytic lesions, leukoplakia)	–	–	–	Y (right buccal mucosa)	–	–	–	SCC	SCC	7 y
47	64	Shnaiderman-Shapiro et al., 2015	F	75	Israel	Mand (#36)	Non-specific (poor healing)	Y	–	–	N	–	–	–	Failing dental implant	Osteogenic sarcoma (high-grade)	5 y
48	65	Silva et al., 2024	F	52	Brazil	Max	Ulcerative lesions	Y	Y	N	Y (verrucous carcinoma oral)	–	Fixed full arch implant supported denture	OLP	SCC	SCC	6 y
49	66	Tanneby et al., 2021	F	81	Sweden	Mand (left post.)	Non-specific (normal)	Y	–	–	Y (left tongue)	–	Bar-retained overdenture	–	Peri-implantitis	SCC	15 y
50	67	Verhoeven et al., 2007	F	67	The Netherland	Mand (#33)	Non-specific (inflammatory lesion)	Y	–	–	Y (lung cancer)	–	Bar-retained overdenture	Metastatic carcinoma	Not specified but treated with instrumentation	SCC (poorly-differentiated,	9 m

(*continued on next page*)

Table 2 (*continued*)

Study no.	Case no.	Author / Year	Sex	Age	Country	Implant location	Clinical appearance	Bone loss (Yes or No)	Smoking or tobacco use (Yes or No)	Alcohol use (Yes or No)	History of previous cancer (Yes or No)	History of periodontitis (Yes or No)	Implant-supported prosthesis type	Other suspected risk factors	Initial diagnosis	Final diagnosis	Time until final diagnosis after implant placement (m: months, y: years)
														from the lung		metastatic lung cancer)	
51	68	Watanabe et al., 2022	M	74	Japan	Max (palate)	Multifaceted (ulcerative lesions, induration and fixation)	–	N	N	N	–	Fixed full arch implant supported denture	–	SCC	SCC (well-differentiated)	20 y

M, male; F, female; Max, maxilla; Mand, mandible; ant., anterior; post., posterior; Y, yes; N, no; -, data not available or not reported; OLP, oral lichen planus; MRONJ, medication-related osteonecrosis of the jaw; SCC, squamous cell carcinoma; y, years; m, months.

Table 3
Summary of pooled data from case reports and case series.

Data	*n*	%
Demographic data		
Age (mean ± SD)	67.57 ± 10.06 years	
Age (range)	38 to 90 years	
Male	31	45.59
Female	37	54.41
Clinical features		
Location		
Max	11	16.18
Mand	56	82.35
Max & Mand	1	1.47
Appearance		
Not reported	1	
Exophytic lesions	19	28.36
Multifaceted	16	23.88
Ulcerative lesions	15	22.39
Non-specific	10	14.93
Erythroplakia or leukoplakia	4	5.97
Fistulas and purulence	3	4.48
Bone loss		
Not reported	19	
Yes	46	93.88
No	3	6.12
Factors		
History of smoking or tobacco use		
Not reported	28	
Yes	19	47.50
No	21	52.50
History of alcohol use		
Not reported	36	
Yes	14	43.75
No	18	56.25
History of previous cancer		
Not reported	16	
Yes	28	53.85
No	24	46.15
History of periodontitis		
Not reported	66	
Yes	1	50.00
No	1	50.00
Implant-supported prosthesis type		
Not reported	8	
Not delivered	3	
Overdenture	25	43.86
Single crown	13	22.81
Fixed partial dental prosthesis	12	21.05
Fixed complete dental prosthesis	7	12.28
Other suspected risk factors		
Not reported	43	
Recurrence	10	40.00
OLP	7	28.00
Metastasis	5	20.00
Trauma	2	8.00
Ill-fitting dental implant	1	4.00
Diagnosis		
Initial diagnosis		
Not reported	11	
SCC	25	43.86
Peri-implantitis	24	42.11
Others	6	10.53
Peri-implant mucositis	2	3.51
Final diagnosis		
SCC	55	80.88
Metastasis	3	4.41
Melanoma	2	2.94
B-cell lymphoma	2	2.94
Plasmacytoma	2	2.94
Others	4	5.88

n, patient number; Max, maxilla; Mand, mandible; OLP, oral lichen planus; SCC, squamous cell carcinoma.

Table 4
Descriptive table of included retrospective studies reporting more than five PIOM cases.

	Author/ Year	Study design	Country	Year period	Years	Cases (*n*)	Number of initial PI diagnosis	Number of PIOM cases	Percentage (%) of PIOM misdiagnosed as PI	Sex		Age (Mean ± SD or range in years)	Location			Factors (*n*)	Clinical appearance (*n*)	Bone loss (*n*)	Time until diagnosis (Mean ± SD or range in months)	Final diagnosis (*n*)
										M	F		Max	Mand	Other					
1	Brabyn et al., 2018	Retrospective case series	Spain	2008–2017	9	6	–	6	–	2	4	66.8 ± 9.9	1	5	–	Oral CA (4), OPMD (1), Smoking (3), Drinking (1)	–	–	57.9 ± 34.2 m	SCC (6)
2	Kaplan et al., 2017	Retrospective case series	Israel	2000–2016	16	7	6	7	85.71	2	5	69 (44 - 89)	2	5	–	Oral CA (2), OLP (2), Smoking (1)	Exophytic (6), Ulcer (1)	3	–	SCC (5), Basal Cell Carcinoma (1), Metastasis (1)
3	Galvis et al., 2021	Retrospective case series	Brazil	2010–2019	9	13	4	13	30.77	3	10	73.4 (59 - 90)	3	10	–	Oral CA (4), OPMD (2), Smoking (3)	Exophytic (2), Ulcer (6), White and red (5)	5	37.8 m (1- 96)	SCC (13)
4	Moergel et al., 2014	Retrospective case series	Germany	1995–2011	4	15	1	15	6.67	7	8	66.1 ± 12.5	1	14	–	Oral CA (9), OLP(3), OPMD (11), Smoking (5), Drinking (6), Poor OH (2)	Exophytic (10), Ulcer (4), BOP (1)	13	53.4 ± 39 m	SCC (15)
5	Ramos et al., 2020	Retrospective case series	Brazil	2009–2020	11	31	5	31	16.13	13	18	68.8 ± 12.12	6	14	11	Oral CA (5), Other CA (6), OLP(4), OPMD (10), Smoking (18), Drinking (12), Poor OH (1)	Ulcer (27), Plaque (4)	–	56.4 m	SCC (31)
6	Seo et al., 2024	Retrospective case series	Korea	2006–2014	8	21	21	21	100.00	13	8	60.43 ± 9.35	7	14	–	Oral CA (3), Other CA (1), OLP (2), Smoking (3), Drinking (2), HPV (12), Poor OH (5)	Exophytic (9), Exophytic + ulcer (10), Ulcer (2)	13	49.13 ± 33.63 m	SCC (20), Melanoma (1)

BOP, blending on probing; CA, carcinoma; F, female; *n*, patient number; M, male; Mand, mandible; Max, maxilla; OH, oral hygiene; OLP, oral lichen planus; OPMD, oral potential malignant disorders; PI, peri-implantitis; PIOM, peri-implant oral malignancy; SCC, squamous cell carcinoma.

prevalent final diagnosis (80.88 %), followed by metastasis (4.41 %), melanoma (2.94 %), B-cell lymphoma (2.94 %), plasmacytoma (2.94 %), and other malignancies (5.88 %). In retrospective studies, SCC was also the most prevalent final diagnosis, occurring in 90 patients (96.77 %). The remaining three patients were diagnosed with melanoma ($n = 1$), basal cell carcinoma (BCC) ($n = 1$), and metastasis ($n = 1$).

3.2.5. Time to incidence of PIOM after implant placement

The Kaplan–Meier analysis of pooled patient-level data from case reports and case series, analyzing 51 PIOM cases, demonstrated the overall median time from implant placement to final PIOM diagnosis was 5 years (95 % CI: 4.06 to 5.94), with a mean time of 6.28 years (95 % CI: 4.55 to 8.01) (Fig. 2a). The cumulative incidence curves stratified by sex showed no significant difference in time to PIOM diagnosis between males and females. The median incidence time for males was 5 years (95 % CI: 3.89 to 6.11), compared to 4 years for females (95 % CI: 2.66 to 5.34). The log-rank test revealed no statistically significant difference between the two groups ($\chi^2 = 0.144$, $p = 0.704$) (Fig. 2b). Implant location (maxilla, mandible, or both) did not significantly influence the time to PIOM diagnosis ($\chi^2 = 3.720$, $p = 0.156$). The median incidence times were 6 years (95 % CI: 1.97 to 10.03) and 5 years (95 % CI: 4.16 to 5.84) for maxillary and mandibular implants, respectively (Fig. 2c).

No significant difference was observed in time to PIOM diagnosis based on smoking history ($\chi^2 = 0.386$, $p = 0.535$). The median incidence times were 5 years (non-smokers) and 6 years (smokers) (Fig. 2d). Alcohol consumption did not significantly affect PIOM incidence ($\chi^2 = 1.192$, $p = 0.275$). Non-drinkers had a mean incidence time of 8.68 years (95 % CI: 4.41 to 12.95) versus 5.14 years (95 % CI: 2.80 to 7.48) for drinkers (Fig. 2e). The analysis showed no statistically significant association between previous cancer history and time to PIOM diagnosis ($\chi^2 = 0.471$, $p = 0.492$). Patients without prior cancer had a mean incidence time of 6.44 years (95 % CI: 3.75 to 9.12), while those with a history of cancer had a mean incidence time of 5.74 years (95 % CI: 4.26 to 7.23). The median incidence time was 5 years for both groups (Fig. 2f).

The median time from implant placement to PIOM diagnosis was 4.00 years (95 % CI: 1.01 to 6.99) for overdentures, 3.00 years (95 % CI: 0.00 to 6.47) for single crowns, 5.00 years (95 % CI: 3.54 to 6.46) for fixed partial dental prostheses, and 6.00 years (95 % CI: 3.06 to 8.94) for fixed complete dental prostheses. Despite observed variations across implant-supported prosthesis types, the log-rank test indicated no statistically significant difference in time to PIOM diagnosis among groups ($\chi^2 = 1.932$, $p = 0.582$) (Fig. 2 g). Detailed statistical results, including survival tables, mean and median survival times with confidence intervals, and log-rank test outcomes for all subgroup analyses, are provided in the **Supplementary material 3**.

3.3. Synthesis of results: meta-analysis

A total of five retrospective studies [9,10,14,67,68] were eligible for the meta-analysis, comprising 87 cases of PIOM from a total of 93 involved patients (Fig. 3). The pooled proportion of cases initially diagnosed as PI before the subsequent confirmation of PIOM was 0.50 (95 % CI: 0.07 to 1.00). The wide prediction interval (0.00 to1.00) reflects substantial variability in effect sizes across the included studies. Heterogeneity among the included studies was significant, with an I^2 value of 95 %, suggesting considerable variability ($Tau^2 = 0.2596$; $Chi^2 = 74.51$, df = 4, $p < 0.01$).

3.4. Publication biases

Funnel plot assessments were not conducted, as the meta-analysis included fewer than 10 studies.

4. Discussion

This systematic review and meta-analysis were designed to investigate the potential association between PI and PIOM, specifically assessing whether PI could serve as a risk factor for PIOM. However, the current evidence remains insufficient to conclusively address this objective, as the available studies are limited to retrospective analyses, case reports, and case series. Despite these limitations, patient-level pooled data from these studies were comprehensively extracted and analyzed to provide meaningful insights based on the best available evidence.

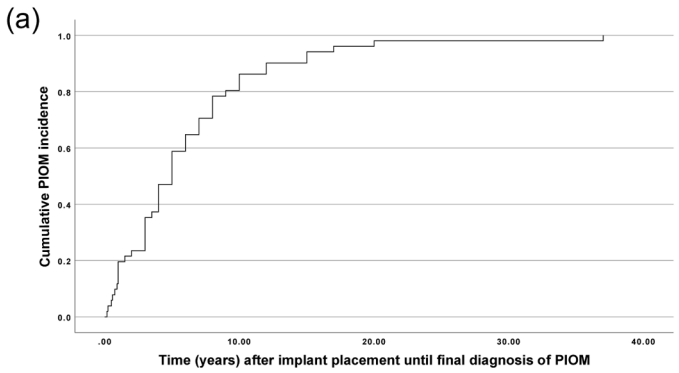
4.1. Principal findings and clinical implications

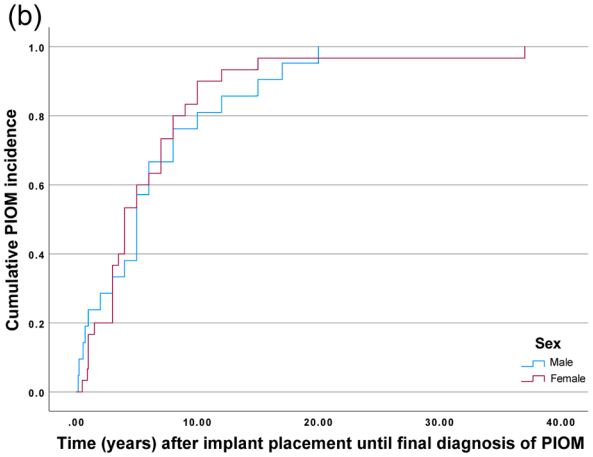
A notable finding of this review was that 50 % of PIOM cases were initially misdiagnosed as PI. This high rate of misdiagnosis underscores the clinical resemblance between benign peri-implant inflammatory conditions and malignancies. The overlap in clinical presentations, such as ulcerations, swelling, and bone loss, complicates early recognition. The predominance of SCC as the final diagnosis (80.88 % from case reports and case series, and 96.77 % from retrospective studies) aligns with previous literature, confirming SCC as the most common oral malignancy [69–71], with the mandible being the most frequently affected site. The Kaplan–Meier analysis showed a median incidence time of 5 years from implant placement to PIOM diagnosis, suggesting that malignancy development may be a late complication. Interestingly, no significant differences in time to incidence were observed across subgroups, including sex, implant location, smoking status, alcohol consumption, previous cancer history, and the prosthesis type. This finding suggests that PIOM may arise even in the absence of traditional risk factors, raising concerns about the role of chronic peri-implant inflammation in carcinogenesis. The absence of these traditional risk factors may indicate a distinct pathophysiological pathway for PIOM development, possibly linked to implant-related factors, such as surface characteristics, corrosion, or mechanical trauma, which warrant further investigation.

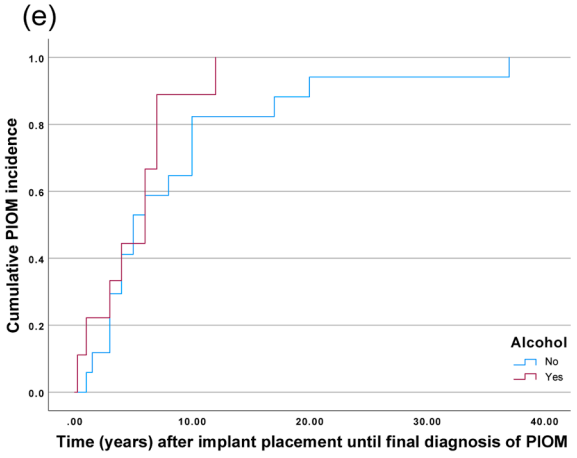
4.2. Potential mechanisms underlying PIOM development

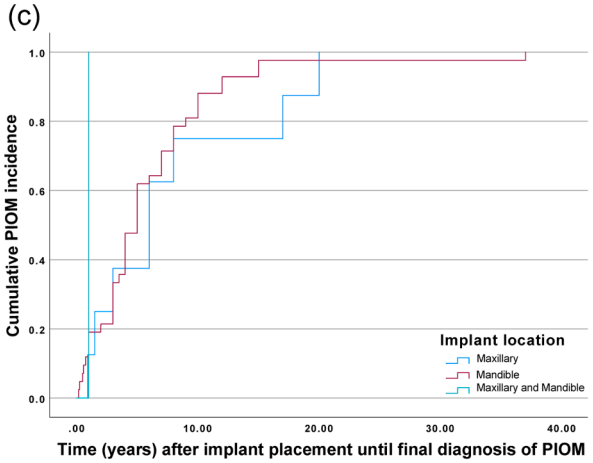
Although the relationship between PIOM and PI could not be definitively established, persistent chronic inflammation due to PI may be a risk factor for carcinogenesis [1,16,19]. The posterior region is the most affected by PIOM and is also the most challenging area for proper hygiene [19,72]. The frequent presence of plaque leads to chronic inflammation and, consequently, increased cell proliferation, which may induce DNA damage. Moreover, inflammation can enhance the expression of genes involved in inflammatory mediator production, cell survival, and proliferation, promoting gene mutations and dysplasia, potentially leading to malignant transformation [73].

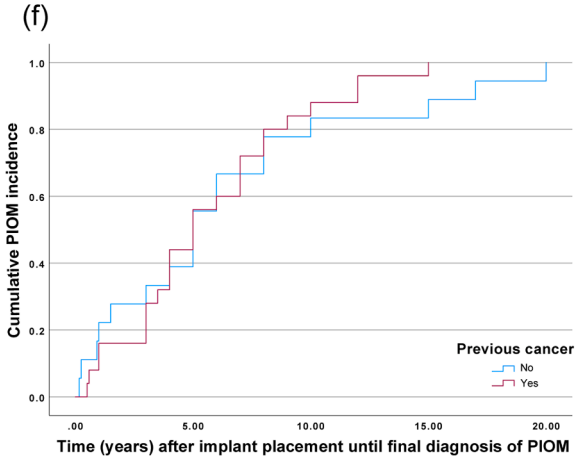
Among the available case reports and case series, implant-supported overdentures were the most frequently reported prosthetic type with PIOM. This finding is somewhat unexpected, given that overdentures are removable and theoretically allow for improved hygiene maintenance compared to fixed restorations [74,75]. One plausible explanation is that overdentures are commonly used in elderly or systemically compromised individuals [76,77], who may have reduced manual dexterity or difficulties adhering to optimal oral hygiene protocols [78]. Furthermore, the design of overdentures often involves extensive mucosal coverage and tissue contact, which can create areas of chronic mechanical irritation or microtrauma to the peri-implant tissues [79]. Another potential contributor is detection bias, overdentures may prompt more frequent clinical evaluations due to their removable nature or the onset of prosthetic complications, increasing the likelihood of lesion detection and subsequent biopsy. Nevertheless, Kaplan–Meier survival analysis revealed no significant differences in time to PIOM diagnosis based on prosthesis type, suggesting that prosthetic design

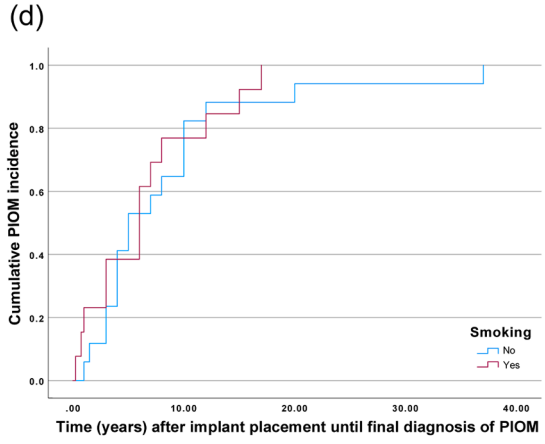












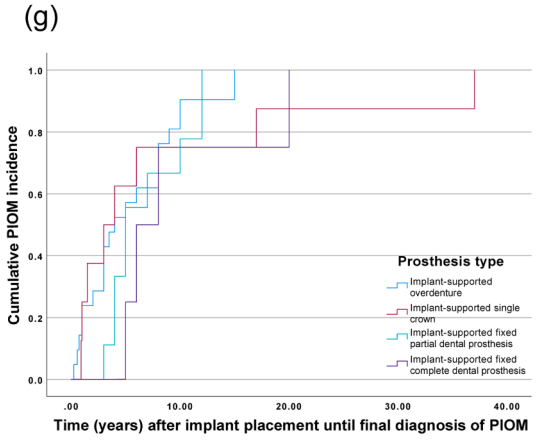


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Fig. 2. Cumulative incidence curves for peri-implant oral malignancy (PIOM) following implant placement, based on pooled data from case reports and case series. The y-axis represents the cumulative incidence of PIOM (1 - survival probability), while the x-axis shows the time (years) from implant placement until the final diagnosis of PIOM. **(a)** Overall cumulative incidence of PIOM over time. **(b)** Cumulative PIOM incidence stratified by sex (male vs. female). **(c)** Cumulative PIOM incidence based on implant location (maxilla, mandible, or both maxilla and mandible). **(d)** Cumulative PIOM incidence according to history of smoking status (yes vs. no). **(e)** Cumulative PIOM incidence by history of alcohol consumption (yes vs. no). **(f)** Cumulative PIOM incidence stratified by history of previous cancer (yes vs. no). **(g)** Cumulative PIOM incidence stratified by implant-supported prosthesis type (overdenture, single crown, fixed partial dental prosthesis, or fixed complete dental prosthesis).

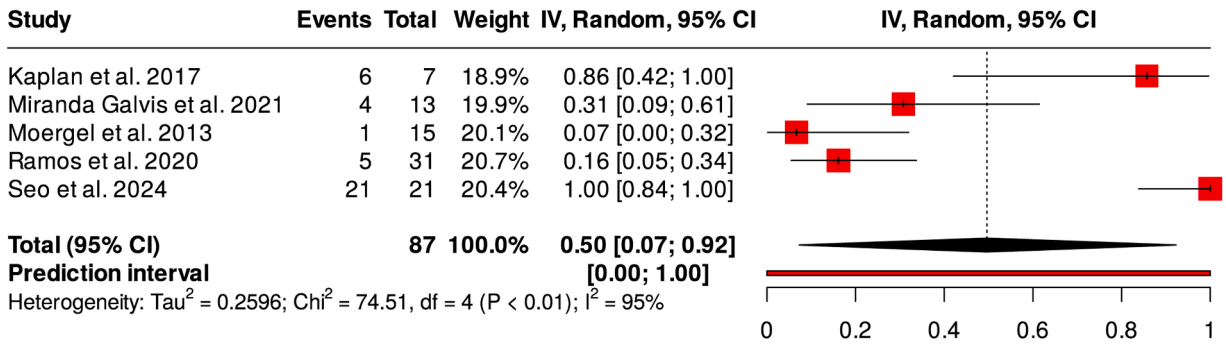

Fig. 3. Forest plot illustrating the pooled proportion of cases initially diagnosed as peri-implantitis (Events) before peri-implant oral malignancies (Total) were later confirmed. CI, confidence interval; IV, inverse variance.

alone may not be a strong independent determinant in the timing of malignant transformation.

In addition to chronic inflammation, other mechanisms have also been proposed to explain the potential relationship between dental implants and carcinogenesis. One of these mechanisms is the role of titanium corrosion products, which promote the release of metal ions. Although titanium has a very low corrosion rate, inflammatory reactions can disrupt its protective oxide layer, leading to the release of metal ions [5,47]. A histopathological study on implant-related lesions found that the released particles further exacerbated the inflammatory response [80]. In addition, these ions may facilitate the migration of malignant cells on the implant surface in contact with both bone and gum tissue [63]. Another theory suggests that the absence of a periodontal ligament may facilitate the invasion of malignant cells in the space between the epithelium and the bone [63], but recent histopathological studies found no evidence of downward growth of malignant lesion along with the implant fixture [81]. Additionally, some researchers propose that undiagnosed malignant or pre-malignant lesions present before implant placement could experience increased cell proliferation following the procedure, potentially leading to malignancy [6].

While various theories have been proposed, none have been conclusively proven, and the role of dental implants in carcinogenesis remains uncertain. The rarity of PIOM further complicates the establishment of a clear causal link [5]. Although the association between chronic inflammation and carcinoma development is well documented [73], the specific contribution of dental implants to this process is still uncertain.

4.3. Clinical implications

Given the high misdiagnosis rate, this review emphasizes the need for a high index of suspicion for malignancies in cases of peri-implantitis that do not respond to conventional treatments. Histopathological examination should be considered for persistent lesions, especially when atypical features such as rapid progression, unusual ulceration patterns, or unexplained bone loss are observed. Clinicians should also be aware of the potential for malignancy even in patients without traditional risk factors, as highlighted by the seven cases [5,13,15–18,26] identified in this review. Routine clinical protocols should include comprehensive risk assessments, and biopsies should not be delayed when malignancy is suspected.

4.4. Limitations and future research

This review has several limitations. Firstly, the predominance of case reports and case series in the available literature introduces potential publication bias, as unusual or severe cases are more likely to be reported. The limited sample sizes and the retrospective nature of included studies may also affect the generalizability of the findings. The high heterogeneity observed in the meta-analysis indicates variability in diagnostic approaches, clinical settings, and reporting standards. Additionally, the lack of longitudinal cohort studies prevents the establishment of a causal relationship between peri-implant inflammation and malignancy. The survival analyses, although insightful, are based on pooled data from heterogeneous case reports, limiting the robustness of temporal inferences.

Future research should focus on well-designed association studies, particularly case-control studies, which are more feasible given the rarity of PIOM and can retrospectively assess the presence or absence of peri-implant disease in affected individuals. While prospective cohort studies would offer stronger evidence for causality, they may be unrealistic due to the low incidence and long latency of PIOM development. Investigations into the molecular and cellular mechanisms underpinning this association are crucial. Studies exploring the role of implant materials, surface properties, and local biomechanical forces may provide insights into modifiable risk factors. Moreover, the development of diagnostic algorithms or risk stratification tools incorporating clinical, radiographic, and biomolecular markers could improve early detection rates. Establishing multicenter registries would also facilitate larger studies and more robust data, allowing for better generalization of findings.

5. Conclusions

Given that the current evidence is predominantly derived from retrospective studies, case reports, and case series, the findings of this review should be interpreted with caution. Although insufficient to establish a definitive causal link, this review synthesized the best available data to provide preliminary insights between PI and PIOM. The results underscore the diagnostic challenges posed by PIOM due to their clinical resemblance to PI. With nearly half of PIOM cases initially misdiagnosed and the absence of significant associations with traditional risk factors, clinicians should maintain a high index of suspicion,

particularly for lesions with atypical presentations or poor treatment responses. Although current evidence suggests a potential link between chronic peri-implant inflammation and malignant transformation, further prospective research is essential to elucidate the underlying mechanisms and develop effective diagnostic protocols.

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CRediT authorship contribution statement

Murali Srinivasan: Writing – review & editing, Writing – original draft, Supervision, Data curation, Conceptualization. **Thalita Fernandes Fleury Curado:** Writing – review & editing, Writing – original draft, Investigation, Conceptualization. **Porawit Kamnoedboon:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Kittipit Srisanoi:** Writing – review & editing, Validation, Investigation, Data curation. **Cláudio Rodrigues Leles:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Piero Papi:** Writing – review & editing, Writing – original draft, Validation, Formal analysis, Data curation. **Umberto Romeo:** Writing – review & editing, Validation, Supervision, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jdent.2025.105773.

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