BMC Oral Health

SYSTEMATIC REVIEW

Open Access

A systematic review and network meta-analysis of the association between periodontitis and inflammatory bowel diseases

Ariyan Ayati[1†], Sajad Khodabandelu[2†], Sara Khaleghi[2], Anita Nourmohammadi[3], Farnaz Jafari[4], Mina Ahmadianghalehsorkh[5], Zahra Vatani[6], Hanieh Sadat Bashiri[7], Mahta Ahmadi[8], Mohammadsadegh Jafari[9], Hossein Soltaninejad[10*] and Mohammad Rahmanian[11*]

Abstract

Objectives Several earlier studies have shown that IBD (including its two subtypes, ulcerative colitis (UC) and Crohn's disease (CD)) increases the risk of periodontal disease. This study aimed to evaluate the relevance among periodontitis and IBD subcategories.

Methods This study was conducted based on PRISMA guidelines. The Web of Science, PubMed, Google Scholar, and Scopus databases were searched up to February 2024 using pertinent keywords. Case series, review articles, and animal studies were excluded. The risk of bias in this research was evaluated through the Joanna Briggs Institute (JBI) criteria. The meta-analysis was conducted using R statistical software.

Results A total of 9134 patients within 13 studies after the screening process were evaluated. Our study has shown that periodontitis is significantly more prevalent among IBD patients (UC and CD). According to prior meta-analyses, PD morbidity was found to be significantly high among CD patients (OR: 4.30; 95% CI: 3.72–4.98; I2 = 0%). Similarly, UC elevated PD risk (OR: 4.55; 95% CI: 3.76–5.50; I2 = 0%). The risk of periodontitis was not significantly different between CD and UC patients (OR: 0.96; 95% CI: 0.65–1.43; I2 = 34%).

Conclusions UC and CD patients were more likely to develop periodontitis, with low heterogeneity between studies, while the prevalence of periodontitis among UC and CD patients was not meaningfully different.

Clinical relevance The higher risk of periodontitis in patients with IBD indicates the necessity of screening for periodontitis. Considering the various oral manifestations and poor quality of life associated with IBD, it is important to be aware of the symptoms of periodontitis.

Keywords Inflammatory bowel disease, Periodontitis, Crohn's disease, Ulcerative colitis, Systematic review

†Ariyan Ayati and Sajad Khodabandelu contributed equally to this work and are co-first authors.

*Correspondence:
Hossein Soltaninejad
Hossein.soltani@modares.ac.ir
Mohammad Rahmanian
mmdrahmanian@gmail.com
Full list of author information is available at the end of the article

© The Author(s) 2025. **Open Access** This article is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License, which permits any non-commercial use, sharing, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if you modified the licensed material. You do not have permission under this licence to share adapted material derived from this article or parts of it. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by-nc-nd/4.0/.

Introduction

Inflammatory bowel disease (IBD) represents a persistent inflammatory disorder that predominantly affects the gastrointestinal tract. IBD is a worldwide public health issue that is receiving more scholarly attention due to its rising incidence, mortality, and life years with disabilities [1–3]. It comprises two principal categories: Crohn's disease (CD) and ulcerative colitis (UC) [4]; they can be distinguished by their histopathology and clinical appearance (symptoms and site) [5]. Concisely, any part of the gastrointestinal system can be affected by CD, while UC mostly induces pathologies in the rectum and colon [6]. In addition to intestinal inflammation and associated difficulties, numerous patients may experience extraintestinal diseases affecting other areas, such as chronic kidney disease [7], celiac disease [8], acute coronary syndrome [9], and Rosacea [10, 11]. Despite the unknown etiology of IBD, it is widely acknowledged that integrating multiple genetic and environmental variables modifies the immune system's reactions to microorganisms [12].

Periodontitis is one of the most common illnesses among humans, with a prevalence of 50% to 90% in developing countries and varying from 4 to 76% in developed countries [13]. In this inflammatory disease, the alveolar bone and periodontal ligament undergo progressive destruction, which is indicated by recession and increased probing depth, or both, and at an advanced level, it contributes to gingival infections, tooth loss, and resorption of the alveolar bone [14, 15]. Periodontitis is a multifactorial disorder. The tooth surface biofilm is the primary cause of periodontitis. Numerous variables can either exacerbate or improve disease manifestations and progression, including the host response, calculus, plaque, environmental variables, systemic health, lifestyle behaviors, and several social factors [16, 17].

Several prior original and meta-analytic studies have established a positive correlation between periodontitis and IBD, indicating that IBD is associated with a greater risk of developing periodontitis [18–23]. In contrast, a cohort study directed by Buchbender et al. in 2021 demonstrated no evidence of clinical impairment to the periodontal tissue in IBD cases. A further analysis reported that the periodontal data exhibited no obvious gap between the IBD patients and the control group [24].

Our research sought to perform a systematic review and meta-analysis to evaluate the relationship between periodontitis and inflammatory bowel disease (IBD) statistically (consisting of UC and CD). We established this goal to conduct precision research and explore contradictions. Moreover, we included recent studies that had been absent in former reviews.

Materials and methods

This systematic review is reported using PRISMA standards [25]. Following the standards, the protocol for the greater review was registered and documented in PROSPERO: CRD42024571071. A librarian developed a search strategy for inflammatory bowel disease and periodontitis.

Literature search

All searches included PubMed, Google Scholar, Scopus, and Web of Science, conducted in February 2024. The methodology employed an extensive array of terms related to periodontitis and inflammatory bowel disease for a complete search strategy. In the course of a systematic search, the subsequent search term was employed: ("IBD" OR "inflammatory bowel disease" OR "Crohn's disease*" OR "Crohn's Disease*" OR "Ulcerative Colitis*") AND (Periodontitis OR Pericementitis) and their MeSH terms. A more database-specific search strategy is available within the supplementary material (See Table S1).

Study selection and data collection

After the methodical inquiry was executed, the citations were incorporated into a bibliographic management application (EndNote X9, Clarivate Analytics, Philadelphia, PA, USA) and evaluated for redundancies, which were eliminated both automatically and through manual verification. Following the exclusion of duplicate studies, the selection was executed autonomously by two evaluators, initially by article title and abstract. Subsequently, the complete text of manuscripts satisfying our exclusion parameters was examined. All phases of the title interface by data generalization were finalized by two autonomous team members, and discordances were resolved through discussion.

We included studies in which periodontal conditions of cases with known IBD diagnoses and their control group in the absence of IBD were studied. Those with a background of a systematic disease or pharmacologic intervention use that may have an impact on periodontal tissues were excluded.

The following are the inclusion criteria:

1- Studies on human subjects;
2- Cross-sectional studies, case–control studies, and cohort studies examining patients with identified IBD and no IBD;
3- Studies that defined periodontitis as a predominant observation;
4- English-full text studies;
5- Studies that reported original data.

We excluded case series, reports, animal studies, review articles, in vitro studies, posters, abstracts, letters, and editorials from the selection. A more detailed PECOS strategy for inclusion/exclusion is explained in Table 1.

Based on the criteria for inclusion and exclusion, two independent researchers completed each phase of the title screen through data abstraction. The following data were extracted: the first author's surname, the date of publication, the study's design, duration of follow-up, the number of samples collected, subtype of IBD, demographic information such as the average age of study participants, exposure evaluation, study quality score, modifications, and measured risk estimates (HR, OR, or RR).

Risk of bias

We evaluated methods of randomization, treatment allocation, and blinding. Joanna Briggs Institute (JBI) criteria [26] were approached to evaluate internal validity. Two reviewers were involved in the quality assessment, and the disagreement between them was reconciled by a third reviewer.

Statistical analysis

The odds ratio for the difference in PD treatment in IBD patients was calculated using a mixed treatment comparison random-effects frequentist network meta-analysis. We used the Netmeta package of the software program R statistical version 4.4.1 (R Foundation for Statistical Computing, Vienna, Austria) for our analysis. Pairwise treatment effects were estimated using a random-effects network meta-analysis and reported as odds ratios (ORs) featuring a 95% confidence interval (CI). In the case in which the 95% confidence interval excludes 1, it signifies a statistically significant variation in the odds. Funnel plots were generated to estimate the risk of publication bias.

Additionally, in this study, with the help of the NMAoutlier library in R, a forward search (FS) algorithm was used to find studies exhibiting extreme outcomes in the Network meta-analysis (NMA) model.

Results

A detailed process of study selection is demonstrated in Fig. 1. A total of 1162 studies were found through searching in databases. After duplicate article removal, six hundred and thirty-three articles entered the title and abstract screening phase; the process led to having seventy-two studies left. From these, fifty-nine studies were excluded due to various reasons. For example, ten studies were in non-English languages, seventeen studies were abstracts or poster presentations, and despite having relevant topics, ten studies did not assess periodontal parameters numerically suitable and were therefore excluded. Finally, our study encompassed thirteen studies: nine case–control [20, 21, 23, 27–32], two cross-sectional [2, 33], and two cohort studies [14, 34].

Included Study Characteristics

Among the included studies, 13 investigated the odds ratio (OR) of PD prevalence in IBD patients (CD, UC, or both) compared to healthy people controls. Among these, three studies compared three groups against the normal group, five studies compared two groups against the normal group, and the rest only compared two groups. The total number of pairwise comparisons

Table 1 PECOS Strategy

	Inclusion criteria	Exclusion criteria
People (P)	Human subjects diagnosed with IBD (including its subgroups UC and CD)	- Individuals with a history of chronic/systemic diseases affecting the periodontal structure - Individuals receiving pharmacological interventions affecting the periodontal structure (e.g., bisphosphonates)
Exposure (E)	Diagnosis of periodontitis through clinical, radiographic, or self-reported assessments (Several clinical parameters can be assessed to help diagnosis of PD, including bleeding on probing (BOP), plaque index (PI), probing pocket depth (PPD), clinical attachment loss (CAL), and Community Periodontal Index of Treatment Needs (CPITN))	Studies without precise diagnostic methods for PD detection
Comparison (C)	Healthy controls without a history of IBD or similar gastrointestinal diseases	Single-arm studies (without a control group)
Outcomes (O)	Prevalence of PD; Clinical periodontal parameters assessed in the study (BOP, PPD, CAL, etc.)	Studies not reporting the prevalence of PD or related measurements
Study Design (S)	Observational studies	Case series, reports, animal studies, review articles, in vitro studies, posters, abstracts, letters, and editorials

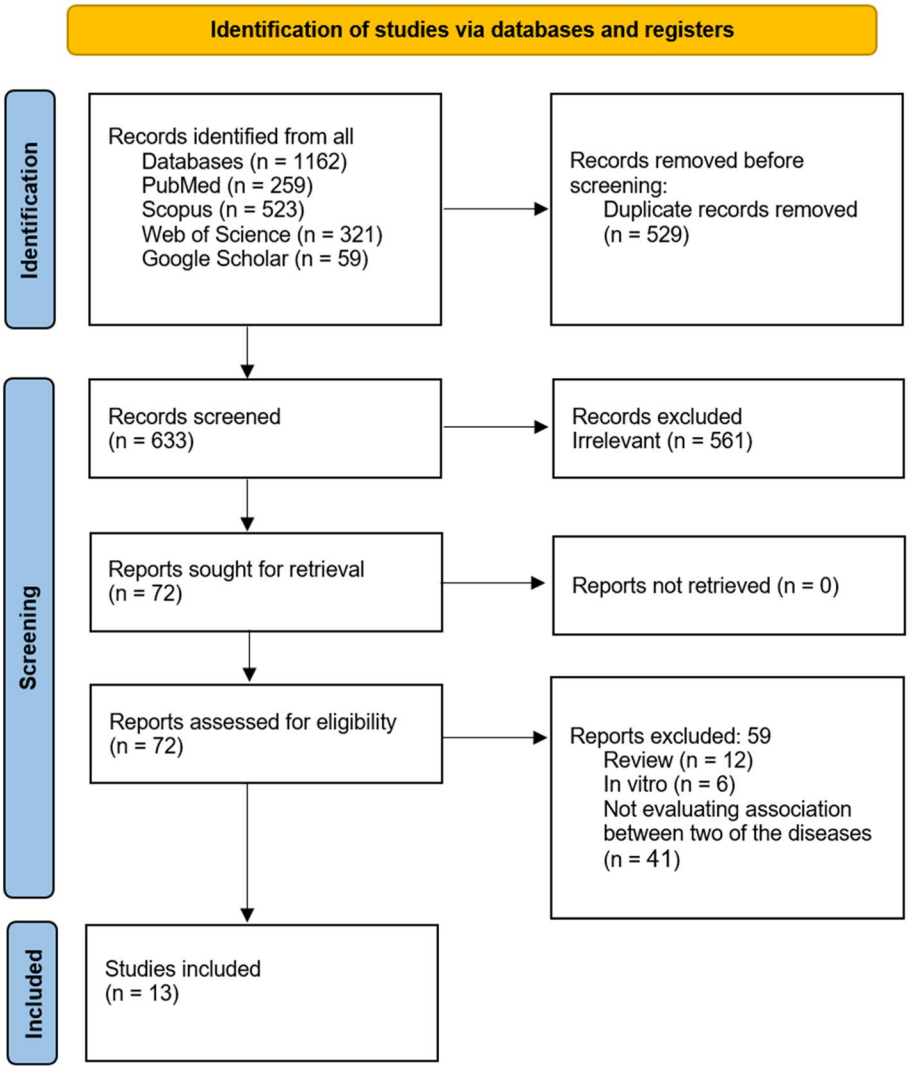

Fig. 1 PRISMA flowchart for identification of eligible studies

in the network was equal to 29. Also, the total number of people present in this network was equal to 51,044 people.

Before starting the meta-analysis network analysis, we used the FS test to investigate the studies with outliers from the network. Table 2 depicts the summary of indicators related to the FS test. According to this test, pairwise comparisons 1, 23, 11, 10, 13, 14, and 28 were excluded from the network due to an unreasonable increase in heterogeneity. Figure 2 shows the funnel plot before and after removing studies from the network, respectively. After removing the studies identified as outliers, the heterogeneity of the network meta-analysis random effects model decreased from $I^2 = 91\%$ (89.7–94%) to $I2 = 0\%$ (0–48%). After this, the characteristics of 13 of the encompassed studies were gathered (see Table 3).

Quality assessment of the included studies

According to JBI critical appraisal checklists for experimental studies, the result was an average quality score of 81% for case–control studies (see Table S2), 100% for cross-sectional studies (see Table S3), and 68% for cohort studies (see Table S4). These scores highlighted the proper quality of the included studies [26].

Pairwise meta-analysis

Figure 3 shows the results of OR effect size for studies to compare the prevalence of PD in one of the subtypes of IBD. According to these results and the estimated value of direct pooled OR for comparing CD versus control, it shows that the chance of PD in CD patients is 4.3 times higher compared to the control (95% CI: 3.72–4.96). This value is equal to 4.55 (95% CI: 3.76–5.5) and 1.86 (95% CI: 1.63–2.13) for comparing UC against control and

Table 2 The progression of the study entered the FS algorithm iterations

Iter	IDs	Entered Studies; Author (Pairwise Comparisons)	Q^{tot}	Q^{het}	Q^{inc}	I^2
1	[19,26,29,22,25,24]	[Schmidt.J et.al.J et.al (CD versus Control|UC and UC versus Control); Zervou.F et.al (CD versus Control); Piras.V et.al (CD + UC versus Control); Chieh Yu.H et.al (CD versus Control)]	0.88	0.88	0	0
2	20	Zervou.F et.al (UC versus Control)	0.89	0.89	0	0
3	3	Segura-Sampedro.J et.al (CD and UC versus Control)	0.93	0.93	0	0
4	16	Baima.G et.al (CD and UC versus Control)	1.15	1.15	0	0
5	4	Habashneh.R.A et.al (CD versus Control)	1.48	1.48	0	0
6	17	Zhang.L et.al (CD versus Control)	1.5	1.49	0	0
7	21	Zervou.F et.al (CD versus UC)	2.08	2.07	0.01	0
8	5	Habashneh.R.A et.al (UC versus Control)	2.84	2.72	0.12	0
9	7	Brito.F et.al (UC versus Control)	3.46	3.02	0.45	0
10	2	Koutsochristou.V et.al (CD and UC versus Control)	5.32	4.87	0.45	0
11	9	Bertl.K et.al (CD versus UC)	5.75	5.73	0.02	0
12	15	Baima.G et.al (CD versus UC)	7.9	7.79	0.11	0
13	18	Zhang.L et.al (UC versus Control)	8.79	8.36	0.42	0
14	8	Brito.F et.al (CD versus UC)	11.25	11.12	0.14	0
15	27	Schmidt.J et.al (CD and UC versus Control)	13.81	13.67	0.14	0
16	12	Bertl.K et.al (CD and UC versus Control)	13.89	13.75	0.14	0
17	6	Brito.F et.al (CD versus Control)	17.89	17.58	0.31	0
18	28	Poyato-Borrego.M et.al (CD and UC versus Control)	24.45	24.14	0.03	0.016
19	14	Baima.G et.al (UC versus Control)	31.93	31.92	0.32	0.0374
20	13	Baima.G et.al (CD versus Control)	46.27	45.96	0.02	0.0777
21	10	Bertl.K et.al (CD versus Control)	81.91	77.76	0.01	0.1452
22	11	Bertl.K et.al (UC versus Control)	119.01	118.71	0.1	0.1921
23	23	Chieh Yu.H et.al (UC versus Control)	154.54	150.3	0.41	0.2223
24	1	Chen Chi.Y et.al (CD versus Control)	330.61	324.44	0.05	0.3611

Abbreviations: Q^{tot} generalized Cochran's *Q* Q^{Het} *Q* statistic within designs, Q^{inc} *Q* statistic, I^2 I^2 statistics

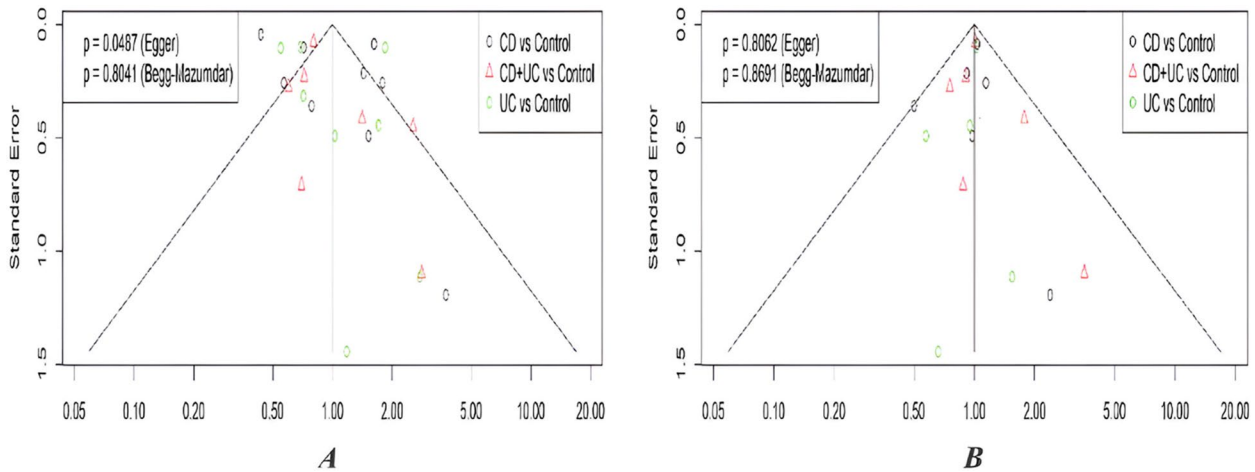

Fig. 2 Funnel plot for base NMA with all studies (**A**) and after removing outliers detected by FS (**B**)

CD + UC against control, respectively. However, comparing the odds ratios of PD prevalence in the two groups of CD and UC did not differ statistically between the two groups (pooled OR = 0.96).

The degree of heterogeneity in the comparison subgroup of CD against UC was estimated to be 34%. In other subgroups, the degree of heterogeneity was 0.

Table 3 Summary of the included studies

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Vanessa [30] [30]	Italy	Case–control	CD and UC—MC	AP- Periapical radiography (PAI)	3 years	220	%55.45 patients; %51.81 control	NR	■ The prevalence of Apical Periodontitis (AP): IBD patients (64%) > controls (59%) ■ According to the gender-stratified analysis: the difference was not significant among the male groups, but the number of teeth with AP was significantly higher in female patients with IBDs than in the controls ■ AP was more common among patients with IBDs + biological medications	Smoking, diabetes, age	8/10 (%80)
J Schmidt.J et.al et al. 2018 [2]	Germany	Cross-sectional	CD and UC—NR	MTSP- Scaled periodontal probe (PPD, CAL)	during their regular subsequent appointment at 1 year and 4 months	118	%60 CD; %55.17 UC; %57.62 control	49.6 (11.9) CD 50.0 (12.4) UC 51.3 (12.0) Control	■ More severe periodontitis and higher concentrations of active-matrix metalloproteinase-8 (aMMP-8): IBD > control ■ Only in CD: ↑ aMMP-8 was associated with the severity of periodontal disease ■ The role of periodontal pathogenic bacteria in the interrelation between IBD and periodontitis is unclear	Smoking, gender, IBD medication	8/8 (%100)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Manuel Poyato-Borrego et al. 2019 [31]	Spain	Case–control	CD and UC—MC	AP- Periapical radiography (PAI)	1 year	162	%42.6 patients; %42.6 control	43.1 (14.0) IBD 43.1 (13.8) Control	■ Teeth with radiolucent periapical lesions (RPLs): patients with IBD > controls ■ The number of teeth and the number of root-filled teeth are significantly associated with periapical radiolucencies (the number of teeth and the number of RFT: IBD patients = controls) ■ Higher prevalence of AP in IBD > controls	Smoking, number of teeth, root-filled teeth	7/10 (%70)
Giacomo Baima.G et.al et al. 2022 [20]	Italy	Case–control	CD and UC – ECCO	MTSP- Scaled periodontal probe (PPD, CAL)	1 year	360	%47.9 CD; %35.0 UC; %43.3 IBD; %42.8 control	47.9 (13.6) CD 49.3 (17.8) UC 47.8 (14.3) Control	■ The prevalence of Periodontitis in IBD (no differences between CD and UC) < control periodontitis was more significantly associated in the middle age categories (36–50 and 51–65 years) ■ Tailored interdisciplinary preventive and therapeutic programs involving the gum–gut axis ■ Longer disease duration and IBD-associated surgery: negatively associated with periodontitis	Age, smoking, IBD duration, IBD-associated surgery	8/10 (%80)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Kristina Bertl.K et.al et al. 2023 [21]	Denmark	Case–control	CD and UC – HBI and SCCAI	SP- PESS	6 months	5786	%74.5 patient; %79.1 control	48.0 (14.8) IBD 48.9 (13.3) Control	■ IBD: associated with impaired patients' oral-health-related QoL (quality of life) [bilateral relationship] ■ Two- and three-times higher prevalence of a poor oral-health-related QoL in UC and UC ■ Fewer teeth + problems with oral lesions: CD patients > UC patients ■ Prevalence of problems in various daily-life activities: CD patients > UC patients ■ IBD activity and severity + depression + a stressful daily life experience (confounders): ↓ IBD-specific health-related QoL/a longer time since diagnosis: ↑ IBD-specific health-related QoL	Smoking, systemic diseases, family status, daily-life experience, BMI, age, gender, education, income	10/10 (%100)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Fernanda [27] [27]	Brazil	Case–control	CD and UC – CDAI and TWI	PD – Scaled periodontal probe (PPD, CAL)	1 year	253	%68.7 CD; %58.7 UC; %67.6 control	39.5 (10.5) CD 45.0 (9.3) UC 42.1 (7.8) Control	■ Decayed, missing, and filled teeth (DMFT) and prevalence of periodontitis: UC and CD > controls ■ DMFT index: UC > CD ■ Periodontitis: more common among smoking patients with UC prevalence of periodontitis: UC and CD > controls prevalence of periodontitis: smokers with UC > smokers without UC in both smokers and non-smokers a tendency of more CAL and more sites with CAL ≥ 3 mm ■ Among non-smokers: • Sites with plaque and deeper PPD: CD patients < controls • DMFT score and deeper pockets: CD patients > controls • After adjustment for race, gender, age, and plaque: UC and CD > controls	Smoking, age, gender, race, plaque, systemic diseases, medication use	7/10 (%70)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type-Diagnostic method	PD type-Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Ying-Chen [34] [34]	Taiwan	Cohort study	CD- ICD9CM and biopsy	PD- ICD9CM	2 years	33,285	%53.7	Stratified	■ Significant difference in risk between genders or across ages was not present ■ Steroids, aspirin, Plavix, and Vicodin have a protective effect ■ Increased hazard ratio for subsequent periodontitis among CD patients compared to subjects without IBD ■ Significantly worse Decayed/Missing/Filled Teeth index in IBD patients	Socioeconomic status, urbanicity, medical comorbidities, pharmaceutical prescriptions, age, gender	6/11 (%54.5)
Hui-Chieh [14] [14]	Taiwan	Cohort study	CD and UC – ICD9CM	CP- ICD9CM	3.00 years in the IBD group; 3.15 years in the non-IBD group	135	%37 IBD patients; %50 No IBD; %47.4 overall	38.0 (10.8) IBD 36.3 (13.6) Control	■ Risk of having periodontitis: IBD patients (CD > UC) > controls ■ CD: showed a significantly higher risk for developing periodontitis ■ UC: had borderline significance for higher risk of periodontitis ■ Male predominant in IBD patients ☑ ■ Higher risk for developing periodontitis: female IBD patients > non-IBD group ■ IBD group with middle-class to high-class economic status: higher risk for developing periodontitis ■ Rapid socioeconomic development and exposure to environmental risk factors in childhood → association between IBD and periodontitis	Age, sex, urbanization level, socioeconomic status	9/11 (%81.8)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
[32] [32]	Greece	Case–control	CD and UC- CDAI and TWI	PD- Periapical radiography	a long period (> 5 years)	77	not mentioned	40 (16) IBD 43 (12) Control	■ Three or more oral lesions: IBD patients > controls ■ The incidence of gingivitis, periodontitis, and gingival bleeding: significant differences between patients with CD and controls ☑ ■ The same parameters between patients with UC and controls: no significant differences ☒ ■ Other lesions such as leukoplakia, perioral erythema, buccal space, abscess, perioral erythema, and erythema migrants, fissured and geographic tongue and aphthous-like ulcer: no significant differences between patients and controls is observed ■ Lymphadenopathy and salivary gland involvement in IBD patients: ☑ ■ Multiple oral manifestations: CD > UC ■ Oral manifestations in patients with IBD (especially in CD) are a frequent and underestimated event that needs further clinical validation	Age, sex, smoking habit, duration of disease	6/10 (%60)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type- Diagnostic method	PD type- Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
Limin [33] [33]	China	Cross-sectional	CD and UC—ECCO	PD- Scaled periodontal probe (PPD, CAL)	almost one year	54	%35.8 CD; %39.5 UC; %43.4 control	26 (16.3) Control 29 (9.63) CD 39 (9.62) UC	■ The decayed, missing, and filled surfaces indices and percentages of sites with probing pocket depth ≥ 5 mm and clinical attachment loss ≥ 4 mm: CD and UC > Controls ■ Differences in dental caries and periodontal disease between the CD and UC patients: ⊠ ■ Values for DMFT, DMFS, DT, DS, and MT: IBD patients (UC and CD) > controls	Age, sex, education level, smoking, daily frequency of tooth-brushing, and dietary habits	8/8 (%100)
Juan J [23] [23]	Spain	Case–control	CD and UC—MC	AP- Periapical radiography (PAI)	4 years	56	%71.4 IBD; %71.4 control	59.1 (10.9) IBD 58.6 (11.9) Control	■ Prevalence of RFT and percentage of RFT with periapical lesions: UC and CD > controls ■ A similar mean number of teeth between the Control group and study group was observed	Age, sex, number of teeth, periapical status, smoking, diabetes,, cardiovascular disease	9/10 (%90)
Vassiliki [29] [29]	Turkey	Case–control	CD and UC – PC	PD- Probe and CPITN (PPD)	6_12 months	110	%50 CD; %63.2 UC; %54.5 IBD	12.32 (3.41) IBD 12.21 (3.96) Control	■ This study deals with children and adolescents with IBD ■ DMF-T and the mean value of GI-S (the simplified gingival index): IBD patient > controls ■ Mean values of PCR: no difference between groups ■ Patients with IBD on immunomodulators: severe periodontal disease + ↑ periodontal treatment ■ Healthy periodontium is not present in IBD patients	Age, sex, oral hygiene habits, smoking, systemic conditions, medications, dietary habits	8/10 (%80)

Table 3 (continued)

Author (Year) [Reference]	Country	Type of Study	IBD type-Diagnostic method	PD type-Diagnostic method	Follow-up Duration	Population, Sex (Female%)		Mean Age (SD)	Outcomes	Confounding Factors	Quality Score
R A Habashneh et al. 2011 [28]	Jordan	Case–control	CD and UC—ECCO	PD- Scaled periodontal probe (PPD, CAL)	7 months	260	%44.1 CD; %39.6 UC; %38.0 control; %40.0 overall	Stratified	■ Prevalence and severity and extent of periodontitis: IBD patients > controls ■ No significant difference in the prevalence of periodontitis between the three groups but much higher among patients with CD and UC compared with subjects without IBD in the age groups < 36 and 36–45 years old only ■ The average plaque index and average gingival index: UC and CD > controls ■ Difference in average plaque index and average gingival index is not observed ■ The average gingival recession: UC > CD > Controls	Age, sex, education level, occupation, smoking habits, oral hygiene behaviors, number of missing teeth	10/10 (%100)

Symbols: ↑: Increase, ↓: Decrease; Abbreviations: MC: Montreal Criteria for IBD, ECCO: European Crohn´s and Colitis Organization criteria, HBI: Harvey-Bradshaw index, SCCAI: Simple Clinical Colitis Activity Index, CDAI: Clinical Disease Activity Index, TWI: work time impairment, PC: Porto Criteria, AP: Apical Periodontitis, SP: Severe Periodontitis, MTSPD: Mild to severe periodontitis, PPD: Pocket probe depth, CAL: Clinical attachment level, PAI: The periapical index, PESS: Periodontal Screening Score, BOP: Bleeding on probing, ICD9CM: ICD-9-CM diagnosis code, CPITN: Community Periodontal Index of Treatment Needs screening method

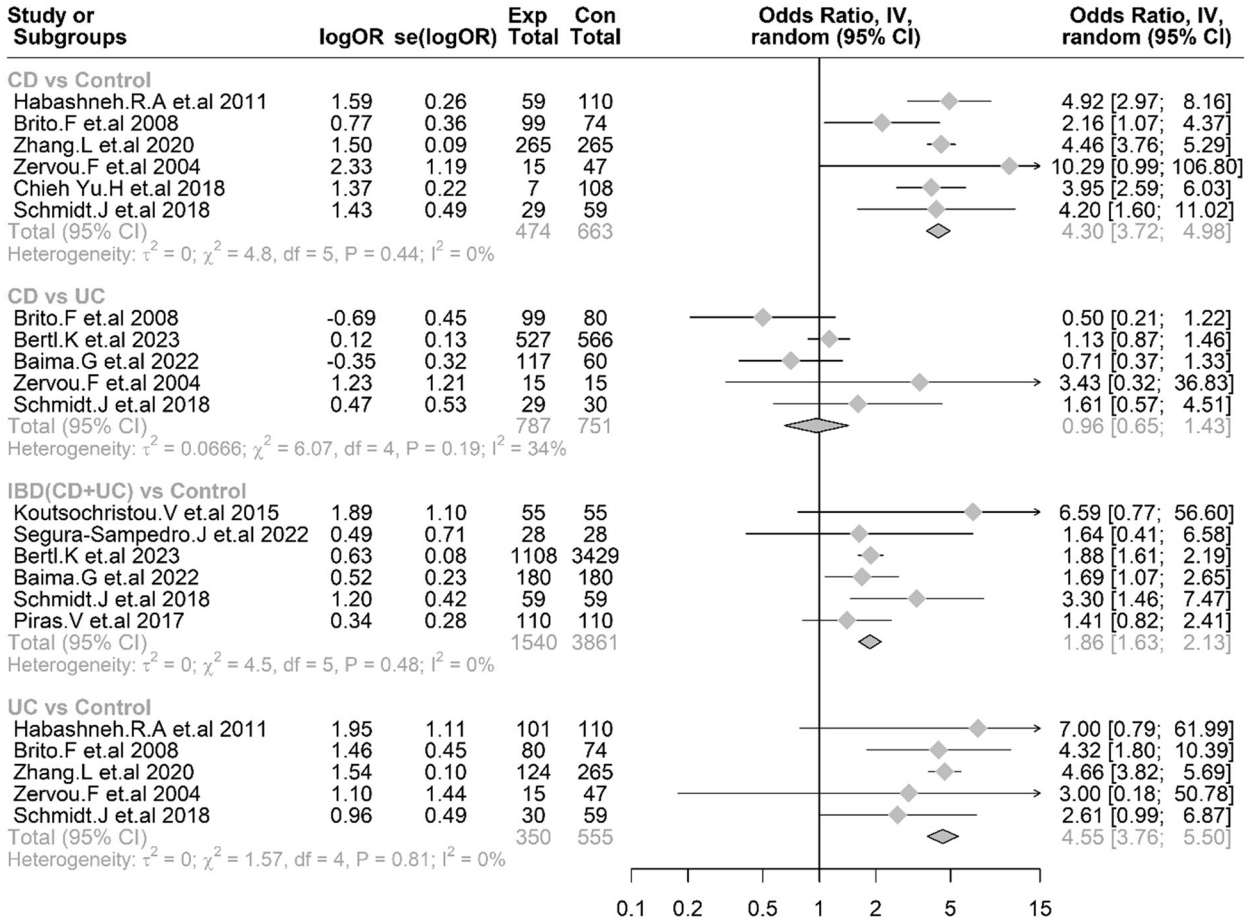

Fig. 3 Forest plot for pairwise comparisons (IBD patient subgroups versus control). CI, confidence interval; P, *P*-Value; df, degrees of freedom; CD, Crohn's disease; UC, ulcerative colitis; χ^2, chi-square test of heterogeneity; I^2, a statistical measure of study heterogeneity

Geometry of network meta-analysis

The geometry of the network meta-analysis illustrates the relationships and comparisons across the included studies, providing a comprehensive view of the evidence. In this study, we constructed a network diagram (Figure S1) to compare the prevalence of periodontitis (PD) across different groups: Crohn's disease (CD), ulcerative colitis (UC), CD + UC, and healthy controls. Each node in the network signifies a distinct group, and the magnitude of the nodes is commensurate with the quantity of constituents within each group. The connections linking the vertices denote the availability of direct comparisons among defined groups, with the thickness of the connections signifying the comparative volume of evidence for each comparison.

The network comprises four major nodes: CD, UC, and controls. Direct comparisons were available for CD versus controls, UC versus controls, CD + UC vs controls, and CD versus UC. The OR for each pairwise comparison was derived based on both direct and indirect evidence, ensuring a robust synthesis of the data.

Network meta-analysis results

The direct effects (green color) and network effect sizes (yellow color) for pairwise comparisons are presented in Supplementary Table 5. Except for the comparison of CD versus UC, the rest of the comparisons show a significant difference in the odds of PD prevalence. It also shows the direct, indirect, and net effects of these comparisons. According to this diagram, for the comparisons of CD against control, UC against control, and CD against UC, the OR is equal to 4.39, 4.40, and 1, respectively (See also Figure S1).

In addition, Fig. 4 shows the network of included studies to compare the PD morbidity rate among IBD patients and healthy individuals.

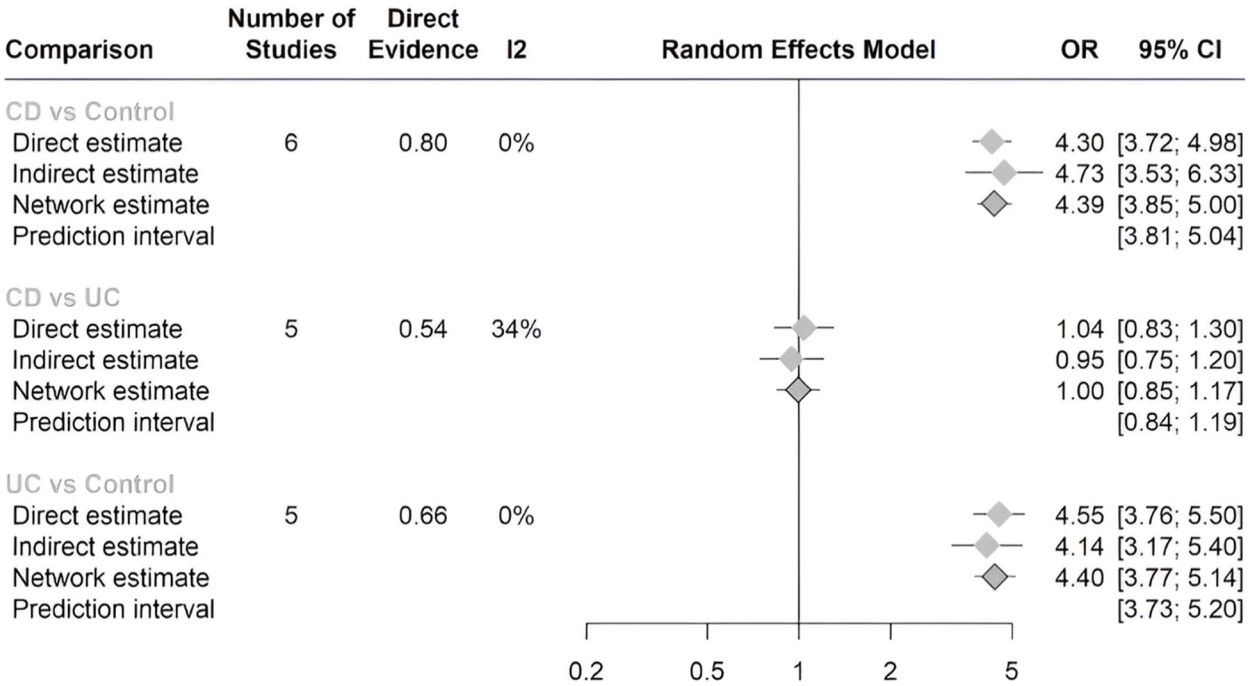

Fig. 4 Forest plot of the direct, indirect, and pooled network effect sizes. CI, confidence interval; OR, odds ratio; CD, Crohn's disease; UC, ulcerative colitis; I^2, a statistical measure of study heterogeneity

Discussion

Periodontitis is known to be a chronic multifactorial inflammatory condition [35]. It is a highly observed oral disease worldwide, and it is deeply intertwined with numerous systemic diseases due to analogous etiological factors [36–38]. Meanwhile, inflammatory bowel disease, encompassing UC and CD, is also a chronic disease with a wide spectrum of genetic and immunologic factors as etiology [39]. The interaction existing between the two aforementioned diseases is yet to be understood thoroughly, but several links have been identified. This systematic review and meta-analysis investigated and summarized the prevalence of periodontitis among IBD (both CD and UC) patients. Records from the total number of 9134 IBD patients and 31,099 controls extracted from the included studies were analyzed. It was demonstrated that periodontitis is significantly more prevalent in the presence of UC and CD. Also, it seemed that there was no significant difference in PD morbidity between UC and CD patients.

As mentioned, it was demonstrated that UC elevated PD, which was in accordance with some former meta-analyses [6, 18, 19, 22, 40–42]. As with UC patients, PD morbidity was also reported to be significantly high among CD patients, like in previous meta-analyses [6, 18, 19, 22, 40]. Both results were associated with low between-study heterogeneity, and their odds ratios were improved compared to the latest meta-analysis on this topic, showing the impact of the latest included studies [40]. In a recent meta-analysis of 2 studies by Wang et al., it was demonstrated that IBD was associated with a higher prevalence of PD (hazard ratio = 1.37), however, the association between its subtypes and PD was not clear [43]. Moreover, in other meta-analyses of 9 and 6 studies by Lorenzo-Pouso et al. and Nijakowski et al. respectively, despite assessing the association of PD with IBD (and its subtypes) and finding it significant, no comparison of PD morbidity within IBD subtypes was performed [6, 42]. Repeatedly, in a meta-analysis of 6 studies by She et al., both UC and CD subgroups were thought to be associated with PD (ORs = 5.37, 3.64), but no comparison was performed between them, as the meta-analysis of 8 studies by Domokos et al. [19, 22]. Finally, Zhang et al. analyzed 6 studies and found that IBD (and both of its subtypes) are associated with PD (ORs for IBD, CD, and UC = 2.1, 1.72, and 2.39) [18]. In our analysis, not only we have included 13 studies and assessed OR within IBD and its subgroups, but also we compared OR for periodontitis among two CD and UC patients via a network analysis to comprehend the interplay more thoroughly.

To explain this connection, Yu et al. performed a bi-directional, two-sample Mendelian randomized study, which examined the connection between IBD (UC and CD) genetic markers and periodontitis [44]. It was shown that this connection was more a kind of correlation rather than a causality; however, another Mendelian randomization study conducted by Wang, Z. et al. showed the opposite: a more causal relationship [45]. This difference was probably due to different datasets and populations included.

Several microbiologic and immunologic similarities can justify the interplay between two diseases. Microbiota alternation can disrupt the normal balance on every mucosal surface. Said, H.S. et al. discovered that there are some alternations associated with IBD in salivary microbiota, including an increase in the genus Prevotella, Leptotrichia, and Saccharibacteria (TM7) and a decrease in the genus Streptococcus. This unsteady state leads to opportunistic bacteria growth, especially in subgingival plaques, and a loss of diversity among the oral microbiome [46]. This loss of diversity is found to be more significant in CD patients by a predominance of genus Wolinella and *Streptococcus mutans* [47, 48], and therefore, a sensible variance may be observed among periodontitis patterns. It is also understood that the oral microbiome can affect gut microbiota and therefore disrupt the gut floor, which may exacerbate intestinal manifestations [49, 50]. A mucosal inflammatory reaction to dysbiosis among oral and gut microbiota is a shared characteristic that reveals another connection between IBD and periodontitis [51]. Arias-Bujanda et al. compared cytokine expression patterns in IBD and periodontitis patients, and higher expressions of MMP-8, IL-17A, and INF-γ were detected [52]. Also, in another similar study conducted by Finamore et al. to elucidate differences between saliva immunologic profiles in CD and UC patients, several clues may be available: for example, UC patients have higher TGF-β[1] and nitric oxide levels [53], and CD patients have elevated levels of salivary TNF-α, IL-1β, and IL-6, resulting in variant oral lesions [54]. After these findings, it was better understood how an anti-TNF pathway can mediate periodontal manifestations [55] as well as IBD [56]. Besides these microbiologic and immunologic interplays between the two diseases, transcriptomic-linked hub genes IGKC and COL4A1 were detected in a bioinformatic analysis by Xiong et al. [57].

Behavioral and demographic risk factors can also influence the comorbidity rate independently or between these two diseases; for example, smoking, poor oral hygiene, socioeconomic status, lack of exercise, and dietary habits are among the most repeated behavioral and lifestyle-related risk factors [14, 20, 28, 33, 58]. To assess the demographic effect, numerous studies agree that an age of more than 50–60 years in IBD patients can be a noteworthy risk factor for developing periodontitis [20, 58, 59]. Such an agreement is not observed regarding sex as a risk factor, while some studies did not recognize sex as a risk factor [28, 33, 58]. Yu et al. demonstrated that female IBD cases are at higher risk of developing periodontitis [14]. Paradoxically, in a study by Kang et al., a significantly higher rate of periodontitis development was observed among male IBD patients [59].

Finally, great population size and proper heterogeneity are major advantages of the present meta-analysis. Due to the larger size of the population compared to previously conducted meta-analyses, our study provides a more reliable result, and as a network meta-analysis, it demonstrates a deeper understanding of the network between IBD subtypes and PD. Despite several previously mentioned advantages, our study has faced numerous limitations. A substantial limitation of our study is the observational design of included studies, as observational studies may be prone to confounding factors and sensible risk of bias, and therefore can induce heterogeneity. Additionally, the most frequent age group evaluated within the included studies was the adult population, however, the study by Koutsochristou et al. evaluated a pediatric population, which could induce heterogeneity among the results [29]. However, we conducted a pairwise meta-analysis after the exclusion of Koutsochristou's study, and the results were the same (See Figure S2). Moreover, the lack of proper geographic distribution may also be a limitation, as most studies were performed in Europe and East Asia, which may affect our findings' generalizability. At last, various guidelines for detecting periodontitis may also be a source of inconsistency within the results.

Conclusion

In summary, it was concluded and confirmed that the abundance of periodontitis is significantly higher among UC and CD patients, and low heterogeneity is observed between studies, while the prevalence of periodontitis between UC and CD patients did not differ meaningfully, and moderate heterogeneity was calculated between studies.

Abbreviations

IBD	Inflammatory Bowel Disease
UC	Ulcerative Colitis
CD	Crohn's Disease
PD	Periodontal Disease / Periodontitis

PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
JBI	Joanna Briggs Institute
OR	Odds Ratio
CI	Confidence Interval
R	Statistical Software R
FS	Forward Search
NMA	Network Meta-Analysis
HR	Hazard Ratio
RR	Relative Risk
ECCO	European Crohn's and Colitis Organization
HBI	Harvey-Bradshaw Index
SCCAI	Simple Clinical Colitis Activity Index
CDAI	Clinical Disease Activity Index
AP	Apical Periodontitis
SP	Severe Periodontitis
PPD	Probing Pocket Depth
CAL	Clinical Attachment Level
PAI	Periapical Index
PESS	Periodontal Screening Score
BOP	Bleeding on Probing
ICD9CM	International Classification of Diseases
9th Edition	Clinical Modification
CPITN	Community Periodontal Index of Treatment Needs
QoL	Quality of Life
MeSH	Medical Subject Headings
DMFT	Decayed, Missing, and Filled Teeth Index
DMFS	Decayed, Missing, and Filled Surfaces Index
aMMP-8	Active Matrix Metalloproteinase-8
BMI	Body Mass Index
TGF-β(1)	Transforming Growth Factor Beta 1
TNF-α	Tumor Necrosis Factor Alpha
IL-1β	Interleukin-1 Beta
IL-6	Interleukin-6
INF-γ	Interferon Gamma
Qt	Generalized Cochran's Q
QHet	Q Statistic within Designs
QInc	Q Statistic for Inconsistency
χ2	Chi-Square Test of Heterogeneity
I2	Statistical Measure of Study Heterogeneity

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12903-025-05830-9.

Supplementary Material 1.

Acknowledgements
None.

Authors' contributions
Conceptualization, M.R.; methodology, M.R.; validation, M.R., S.K., and A.A.; formal analysis, S.K.; investigation, H.B, and M.A.; data curation, M.J., and S.K.; writing—original draft preparation, A.A, A.N., and F.J.; writing—review and editing, A.A., M.A., H.S. and Z.V.; supervision, H.S, and M.R.; project administration, M.R.; All authors have read and agreed to the published version of the manuscript.

Funding
The authors declare that no funds, grants, or other support were received during the preparation of this manuscript.

Data availability
The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Author details
[1]School of Medicine, Shahid Beheshti University of Medical Sciences, Postal code, Tehran 19839-63113, Iran. [2]Department of Biostatistics and Epidemiology, Student Research Committee, School of Health, Mazandaran University of Medical Sciences, Postal code, Sari 48175-866, Iran. [3]Faculty of Dentistry, Postal Code, Tehran Medical Sciences, Islamic Azad University, Tehran 19468-53314, Iran. [4]Oral and Dental Diseases Research Center, Kerman University of Medical Sciences, Postal code, Kerman 1946853314, Iran. [5]Department of Pediatric Dentistry, Postal Code, Ilam University of Medical Sciences, Ilam 6939177314, Iran. [6]School of Medicine, Ahvaz Jundishapur University of Medical Sciences, Ahvaz 6135715794, Iran. [7]Department of Public Health, School of Public Health and Safety, Shahid Beheshti University of Medical Sciences, Postal code, Tehran 19839-69411, Iran. [8]School of Dentistry, Shiraz University of Medical Sciences, Postal code, Shiraz 71956-15878, Iran. [9]Universal Scientific Education and Research Network (USERN), Tehran, Iran. [10]Department of Stem Cells Technology and Tissue Regeneration, Faculty of Interdisciplinary Science and Technologies, Tarbiat Modares University, Tehran, Iran. [11]Student Research Committee, School of Medicine, Shahid Beheshti University of Medical Sciences, Postal Code, Tehran 19839-63113, Iran.

Received: 23 December 2024 Accepted: 17 March 2025
Published online: 31 March 2025

References

1. Windsor JW, Kaplan GG. Evolving Epidemiology of IBD. Curr Gastroenterol Rep. 2019;21(8):40.
2. Schmidt J, Weigert M, Leuschner C, Hartmann H, Raddatz D, Haak R, et al. Active matrix metalloproteinase-8 and periodontal bacteria-interlink between periodontitis and inflammatory bowel disease? J Periodontol. 2018;89(6):699–707.
3. Wang R, Li Z, Liu S, Zhang D. Global, regional and national burden of inflammatory bowel disease in 204 countries and territories from 1990 to 2019: a systematic analysis based on the Global Burden of Disease Study 2019. BMJ Open. 2023;13(3): e065186.
4. Pithadia AB, Jain S. Treatment of inflammatory bowel disease (IBD). Pharmacol Rep. 2011;63(3):629–42.
5. Panaccione R. Mechanisms of inflammatory bowel disease. Gastroenterol Hepatol (N Y). 2013;9(8):529–32.
6. Lorenzo-Pouso AI, Castelo-Baz P, Rodriguez-Zorrilla S, Pérez-Sayáns M, Vega P. Association between periodontal disease and inflammatory bowel disease: a systematic review and meta-analysis. Acta Odontol Scand. 2021;79(5):344–53.
7. Zadora W, Innocenti T, Verstockt B, Meijers B. Chronic kidney disease in inflammatory bowel disease, a systematic review and meta-analysis. J Crohns Colitis. 2024;18(9):1464–75. https://doi.org/10.1093/ecco-jcc/jjae049.
8. Pinto-Sanchez MI, Seiler CL, Santesso N, Alaedini A, Semrad C, Lee AR, et al. Association Between Inflammatory Bowel Diseases and Celiac Disease: A Systematic Review and Meta-Analysis. Gastroenterology. 2020;159(3):884-903.e31.
9. Zaka A, Mridha N, Subhaharan D, Jones M, Niranjan S, Mohsen W, Ramaswamy PK. Inflammatory bowel disease patients have an increased risk of acute coronary syndrome: a systematic review and meta-analysis. Open Heart. 2023;10(2):e002483. https://doi.org/10.1136/openhrt-2023-002483.
10. Jun YK, Yu D-A, Han YM, Lee SR, Koh S-J, Park H. The Relationship Between Rosacea and Inflammatory Bowel Disease: A Systematic Review and Meta-analysis. Dermatology and Therapy. 2023;13(7):1465–75.
11. Vavricka SR, Manser CN, Hediger S, Vögelin M, Scharl M, Biedermann L, et al. Periodontitis and gingivitis in inflammatory bowel disease: a case-control study. Inflamm Bowel Dis. 2013;19(13):2768–77.
12. Zhang YZ, Li YY. Inflammatory bowel disease: pathogenesis. World J Gastroenterol. 2014;20(1):91–9.

13. Jiao J, Jing W, Si Y, Feng X, Tai B, Hu D, et al. The prevalence and severity of periodontal disease in Mainland China: Data from the Fourth National Oral Health Survey (2015–2016). J Clin Periodontol. 2021;48(2):168–79.
14. Yu HC, Chen TP, Chang YC. Inflammatory bowel disease as a risk factor for periodontitis under Taiwanese National Health Insurance Research database. J Dent Sci. 2018;13(3):242–7.
15. Papapanou PN. Periodontal diseases: epidemiology. Ann Periodontol. 1996;1(1):1–36.
16. Mehrotra N, Singh S. Periodontitis. StatPearls. Treasure Island (FL): StatPearls Publishing LLC.; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/NBK541126/. StatPearls Publishing Copyright © 2024, StatPearls Publishing LLC.; 2024.
17. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases Nat Rev Dis Primers. 2017;3:17038.
18. Zhang Y, Qiao D, Chen R, Zhu F, Gong J, Yan F. The Association between Periodontitis and Inflammatory Bowel Disease: A Systematic Review and Meta-analysis. Biomed Res Int. 2021;2021:6692420.
19. Domokos Z, Uhrin E, Szabó B, Czumbel ML, Dembrovszky F, Kerémi B, et al. Patients with inflammatory bowel disease have a higher chance of developing periodontitis: A systematic review and meta-analysis. Front Med (Lausanne). 2022;9:1020126.
20. Baima G, Muwalla M, Testa G, Mazza F, Bebars A, Perotto S, et al. Periodontitis prevalence and severity in inflammatory bowel disease: A case-control study. J Periodontol. 2023;94(3):313–22.
21. Bertl K, Tsakos G, Pandis N, Bogren A, Burisch J, Stavropoulos A. Health-related quality of life aspects of the "Periodontitis prevalence in ulcerative colitis and Crohn's disease" (PPCC) cohort. J Clin Periodontol. 2023;50(12):1601–20.
22. She YY, Kong XB, Ge YP, Liu ZY, Chen JY, Jiang JW, et al. Periodontitis and inflammatory bowel disease: a meta-analysis. BMC Oral Health. 2020;20(1):67.
23. Segura-Sampedro JJ, Jiménez-Giménez C, Jane-Salas E, Cabanillas-Balsera D, Martín-González J, Segura-Egea JJ, López-López J. Periapical and endodontic status of patients with inflammatory bowel disease: Age- and sex-matched case-control study. Int Endod J. 2022;55(7):748–57.
24. Grössner-Schreiber B, Fetter T, Hedderich J, Kocher T, Schreiber S, Jepsen S. Prevalence of dental caries and periodontal disease in patients with inflammatory bowel disease: a case-control study. J Clin Periodontol. 2006;33(7):478–84.
25. Hutton B, Salanti G, Caldwell DM, Chaimani A, Schmid CH, Cameron C, et al. The PRISMA extension statement for reporting of systematic reviews incorporating network meta-analyses of health care interventions: checklist and explanations. Ann Intern Med. 2015;162(11):777–84.
26. Joanna Briggs Institute. The Joanna Briggs institute critical appraisal tools. 2022-08-29. 2017. http://jbi.global/critical-appraisal-tools.
27. Brito F, de Barros FC, Zaltman C, Carvalho AT, Carneiro AJ, Fischer RG, et al. Prevalence of periodontitis and DMFT index in patients with Crohn's disease and ulcerative colitis. J Clin Periodontol. 2008;35(6):555–60.
28. Habashneh R, Khader Y, Alhumouz M, Jadallah K, Ajlouni Y. The association between inflammatory bowel disease and periodontitis among Jordanians: a case–control study. J Periodontal Res. 2012;47(3):293–8.
29. Koutsochristou V, Zellos A, Dimakou K, Panayotou I, Siahanidou S, Roma-Giannikou E, Tsami A. Dental Caries and Periodontal Disease in Children and Adolescents with Inflammatory Bowel Disease: A Case-Control Study. Inflamm Bowel Dis. 2015;21(8):1839–46.
30. Piras V, Usai P, Mezzena S, Susnik M, Ideo F, Schirru E, Cotti E. Prevalence of Apical Periodontitis in Patients with Inflammatory Bowel Diseases: A Retrospective Clinical Study. J Endod. 2017;43(3):389–94.
31. Poyato-Borrego M, Segura-Sampedro JJ, Martín-González J, Torres-Domínguez Y, Velasco-Ortega E, Segura-Egea JJ. High Prevalence of Apical Periodontitis in Patients With Inflammatory Bowel Disease: An Age- and Gender- matched Case-control Study. Inflamm Bowel Dis. 2020;26(2):273–9.
32. Zervou F, Gikas A, Merikas E, Peros G, Sklavaina M, Loukopoulos J, Triantafillidis JK. Oral manifestations of patients with inflammatory bowel disease. Ann Gastroenterol. 2004;17(4):395–401.
33. Zhang L, Gao X, Zhou J, Chen S, Zhang J, Zhang Y, et al. Increased risks of dental caries and periodontal disease in Chinese patients with inflammatory bowel disease. Int Dent J. 2020;70(3):227–36.
34. Chi YC, Chen JL, Wang LH, Chang K, Wu CL, Lin SY, et al. Increased risk of periodontitis among patients with Crohn's disease: a population-based matched-cohort study. Int J Colorectal Dis. 2018;33(10):1437–44.
35. Ray RR. Periodontitis: An Oral Disease with Severe Consequences. Appl Biochem Biotechnol. 2023;195(1):17–32.
36. Arigbede AO, Babatope BO, Bamidele MK. Periodontitis and systemic diseases: A literature review. J Indian Soc Periodontol. 2012;16(4):487–91.
37. Fitzpatrick SG, Katz J. The association between periodontal disease and cancer: a review of the literature. J Dent. 2010;38(2):83–95.
38. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. J Periodontol. 2006;77(8):1289–303.
39. Muzammil MA, Fariha F, Patel T, Sohail R, Kumar M, Khan E, et al. Advancements in Inflammatory Bowel Disease: A Narrative Review of Diagnostics, Management, Epidemiology, Prevalence, Patient Outcomes, Quality of Life, and Clinical Presentation. Cureus. 2023;15(6): e41120.
40. Halboub E, Al-Maswary A, Mashyakhy M, Al-Qadhi G, Al-Maweri SA, Ba-Hattab R, Abdulrab S. The Potential Association Between Inflammatory Bowel Diseases and Apical Periodontitis: A Systematic Review and Meta-Analysis. Eur Endod J. 2024;9(1):8–17.
41. Papageorgiou SN, Hagner M, Nogueira AVB, Franke A, Jäger A, Deschner J. Inflammatory bowel disease and oral health: systematic review and a meta-analysis. J Clin Periodontol. 2017;44(4):382–93.
42. Nijakowski K, Gruszczyński D, Surdacka A. Oral Health Status in Patients with Inflammatory Bowel Diseases: A Systematic Review. Int J Environ Res Public Health. 2021;18(21):11521. https://doi.org/10.3390/ijerph182111521.
43. Wang Q, Chen S, Zhou J, Zhao L. Bidirectional associations between periodontitis and inflammatory bowel disease: A systematic review of longitudinal studies with meta-analysis and trial sequential analysis. J Periodontal Res. 2024;59(6):1083–94.
44. Yu F, Yang Y, Wu D, Chang M, Han C, Wang Q, et al. Deciphering genetic causality between inflammatory bowel disease and periodontitis through bi-directional two-sample Mendelian randomization. Sci Rep. 2023;13(1):18620.
45. Wang H, Wang L, Zeng X, Zhang S, Huang Y, Zhang Q. Inflammatory bowel disease and risk for hemorrhoids: a Mendelian randomization analysis. Sci Rep. 2024;14(1):16677.
46. Said HS, Suda W, Nakagome S, Chinen H, Oshima K, Kim S, et al. Dysbiosis of salivary microbiota in inflammatory bowel disease and its association with oral immunological biomarkers. DNA Res. 2014;21(1):15–25.
47. Brito F, Zaltman C, Carvalho AT, Fischer RG, Persson R, Gustafsson A, Figueredo CM. Subgingival microflora in inflammatory bowel disease patients with untreated periodontitis. Eur J Gastroenterol Hepatol. 2013;25(2):239–45.
48. Docktor MJ, Paster BJ, Abramowicz S, Ingram J, Wang YE, Correll M, et al. Alterations in diversity of the oral microbiome in pediatric inflammatory bowel disease. Inflamm Bowel Dis. 2012;18(5):935–42.
49. Furusho H, Miyauchi M, Hyogo H, Inubushi T, Ao M, Ouhara K, et al. Dental infection of Porphyromonas gingivalis exacerbates high fat diet-induced steatohepatitis in mice. J Gastroenterol. 2013;48(11):1259–70.
50. Atarashi K, Suda W, Luo C, Kawaguchi T, Motoo I, Narushima S, et al. Ectopic colonization of oral bacteria in the intestine drives T(H)1 cell induction and inflammation. Science. 2017;358(6361):359–65.
51. Indriolo A, Greco S, Ravelli P, Fagiuoli S. What can we learn about biofilm/host interactions from the study of inflammatory bowel disease. J Clin Periodontol. 2011;38(Suppl 11):36–43.
52. Arias-Bujanda N, Regueira-Iglesias A, Balsa-Castro C, Nibali L, Donos N, Tomás I. Accuracy of single molecular biomarkers in gingival crevicular fluid for the diagnosis of periodontitis: A systematic review and meta-analysis. J Clin Periodontol. 2019;46(12):1166–82.
53. Rezaie A, Khalaj S, Shabihkhani M, Nikfar S, Zamani MJ, Mohammadirad A, et al. Study on the correlations among disease activity index and salivary transforming growth factor-beta 1 and nitric oxide in ulcerative colitis patients. Ann N Y Acad Sci. 2007;1095:305–14.
54. Finamore A, Peluso I, Cauli O. Salivary Stress/Immunological Markers in Crohn's Disease and Ulcerative Colitis. Int J Mol Sci. 2020;21(22):8562. https://doi.org/10.3390/ijms21228562.
55. Majdi Abunemer R, Saifuddin Shaheen R, Abudullah AR. Correlation of anti-TNF-a biological therapy with periodontal conditions and osteonecrosis in autoimmune patients: A systematic review. The Saudi Dental Journal. 2023;35(7):785–96.
56. Genaro LM, Gomes LEM, Franceschini A, Ceccato HD, de Jesus RN, Lima AP, et al. Anti-TNF therapy and immunogenicity in inflammatory bowel diseases: a translational approach. Am J Transl Res. 2021;13(12):13916–30.

57. Xiong Z, Fang Y, Lu S, Sun Q, Huang J. Identification and Validation of Signature Genes and Potential Therapy Targets of Inflammatory Bowel Disease and Periodontitis. J Inflamm Res. 2023;16:4317–30.
58. Abrol N, Compton SM, Graf D, Parashar P, Heo G, Gibson MP. Inflammatory bowel disease and periodontitis: A retrospective chart analysis. Clin Exp Dent Res. 2022;8(5):1028–34.
59. Kang EA, Chun J, Kim JH, Han K, Soh H, Park S, et al. Periodontitis combined with smoking increases risk of the ulcerative colitis: A national cohort study. World J Gastroenterol. 2020;26(37):5661–72.

Publisher's Note

Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.