Ulcerative Colitis And Periodontitis – A Cross Sectional Study From A Norwegian Cohort Diagnosed With Ulcerative Colitis For More Than 10 Years

Helena. O. Haugbo (helenaoh@student.odont.uio.no)
Lovisenberg Diakonale Sykehus
Pasquale Klepp
Lovisenberg Diakonale Sykehus
Anders Verket
University of Oslo

Research Article

Keywords: Periodontitis, ulcerative colitis, cross-sectional study, OHIP-14

Posted Date: March 16th, 2022

DOI: https://doi.org/10.21203/rs.3.rs-1439796/v1

Abstract

Background: Inflammatory bowel disease (IBD) includes the major diseases Crohn's disease (CD) and ulcerative colitis (UC). IBD is understood to be an inflammatory systemic disorder that may have an impact on periodontal disease through shared inflammatory mechanisms. The two subtypes of IBD are often described together although they differ with regards to affected sites in the gastrointestinal tract, histology and oral manifestations. The primary aim of this study was to investigate the prevalence of periodontitis in a cohort of UC according to the classification scheme in periodontology. The secondary aim was to assess a potential correlation of periodontal status with UC disease parameters, years with UC diagnosis and oral health related quality of life.

Method: In a cross-sectional study a cohort from a community hospital in Norway with confirmed ulcerative colitis diagnosis for more than 10 years was comprehensively diagnosed according to the new classification of periodontitis. Periodontal parameters, oral health related quality of life (OHRQoL) (OHIP-14), demographic data were collected. Previous UC data including colon activity index (CAI), Mayo score and years of UC diagnosis was used to correlate with the periodontal status.

Results: A total of 50 of 63 invited patients participated. According to the new classification scheme on periodontitis, 74% of the patients presented periodontitis. No correlation was found between periodontitis (stage, grade, BoP or PPD>6mm) and CAI, Mayo score, or years with UC diagnosis. The OHIP-14 scores reported were in line with that of a normal population.

Conclusions: Localized stage III periodontitis grade B was the most prevalent periodontal diagnosis. No correlation between periodontal disease and previous UC disease markers or years with UC diagnosis was found. In this population of subjects with well-treated UC, both the prevalence of periodontitis and OHRQoL were in line with that of the general population.

Background

The two main forms of inflammatory bowel disease (IBD) are Crohn's disease (CD) and ulcerative colitis (UC). They are chronic, relapsing, and remitting diseases of multifactorial etiology which involve the gastrointestinal tract [1]. CD is characterized by transmural inflammation and may occur at any site of the entire gastro-intestinal tract. In UC, the inflammatory process is restricted to the colon and invariably involves the rectum and may extend proximally in a continuous fashion.

In Europe, incidence rates for UC range from 0.9 to 24.0 per 100 000 person-years. The prevalence of UC varies from 2.4 to 294 cases per 100 000 persons. In a Norwegian population cohort study the prevalence of UC was found to be 0.51% [3]. In a recent study from Norway the incidence of UC was found to be 24.7 and 28.4 per 100 000 persons- years in the years 2010 and 2017 [4]

UC is currently not curable, and treatment is aimed at symptomatic relief, reduction of inflammation during exacerbations, maintenance of remission, and increasing quality of life. Surgical treatment is

indicated in patients who fail drug treatment or develop severe complications. Approximately 20% of patients with UC eventually require surgery [5].

The peak age for diagnosis of UC is 30–40 years [6] and the disease occurs more frequently in men: 53.8–60% [4, 6, 7]. The first signs are often melena or hematochezia. Extraintestinal manifestations like arthritis, uveitis, erythema nodusum, aphtous ulcers are common, and many patients report that IBD negatively influences their health-related quality of life (HRQoL) and ability to work [8, 9].

Inflammatory bowel disease (IBD) and periodontitis share some similar immunopathogenic responses in addition to being non-curable and chronic diseases. UC and periodontitis are characterized by a hypersensitivity immune response to commensal gut bacteria and dental plaque bacteria, respectively, which may disrupt local homeostasis in susceptible individuals [10]. Periodontitis is described as the result of an imbalance of the oral microbiota in the dento-gingival area, which results in a host response leading to inflammation and destruction of the periodontium in susceptible individuals. Previous studies have shown greater attachment loss and higher prevalence and severity of periodontitis in adults with IBD than in controls [10]. The risk of having periodontitis with the IBD diagnosis varies across studies with odds ratios ranging from 3.95 to 7.0 [10–12]. It has also been found that one sixth of IBD individuals are also diagnosed with a rheumatoid diagnosis and that there is a 13% increased risk for periodontitis with increased probing depths and attachment loss in individuals with rheumatoid arthritis [13, 14]. According to current knowledge [15] there is a significant association between inflammatory bowel disease and periodontitis, although the overall quality of evidence is considered weak to moderate [16]. Since there are marked differences between CD and UC [2], information on oral health may be lost when combined as IBD hence this study focuses on UC. Increased knowledge of UC pathogenesis and complications outside the intestine could contribute and influence the clinical management of periodontal disease for UC subjects.

In the present study, the clinical periodontal status in a cohort diagnosed with ulcerative colitis for more than 10 years was assessed. The primary aim of this cross-sectional study was to evaluate the prevalence of periodontitis and periodontal health and oral health related quality of life (OHRQoL) in a Norwegian cohort with established and medically treated UC. The secondary aims were to assess a potential correlation between periodontal severity and the UC severity defined by Mayo scores (MAYO) and clinical activity index (CAI).

Materials And Methods

The present article was written following the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. The study was performed at the dental clinic at Lovisenberg Diaconal Hospital (LDS) in Oslo, Norway, in collaboration with the Faculty of Dentistry at the University of Oslo. The cohort was recruited from a study that was performed to consider DNA markers for cancer in stool samples from patients with UC [22]. Both degree of inflammation according to the Mayo

endoscopic score (Mayo score)[23] and colitis activity index (CAI)[24] as a measure of disease activity, was registered by two dedicated gastroenterologists in the original study.

The CAI is a questionnaire used to measure symptoms of UC. The Mayo score is characterized by four classes of severity ranging from 0 to 3, with 0 indicating inactive disease with normal mucosa; 1 mild disease (erythema and mild friability); 2 moderate disease (marked erythema, friability, absent vascular pattern and erosions); and 3 severe disease (spontaneous bleeding and diffuse ulceration).

Due to general data protection regulations (GDPR), the patients were initially contacted by personnel directly involved in the original multicenter study. The main investigator (HOH) was kept unaware of the identity until they had accepted the invitation for an oral examination. Following agreement patients were invited to an examination at the dental clinic at LDS. A total of 63 patients were invited.

Subjects were considered eligible for the study if they were more than 18 years old and if they have had the UC diagnosis for more than 10 years, regardless of presence or absence of symptoms. Pregnant or lactating women were excluded as well as persons unable to provide an informed consent. The study protocol was approved by the Regional Committee for Medical and Health Research Ethics (REC Project NO. 2010/1093) with updates in 2019.

Patient population

Patients were recruited from a population-based surveillance cohort of UC patients registered in the database of LDS from 1999–2013.

Patients underwent an oral examination from June 2020 - January 2021 following informed consent to participate. A questionnaire was used to record level of education, age of UC debut, self-reported smoking habits and the use of complementary medication.

Background information on oral-health-related behavior was collected, including self-reported medication, comorbidities, oral dryness (hyposalivation) according to the Challacombe Scale [25], and decayed-missing-filled teeth (DMFT). A questionnaire on OHRQoL (Oral Health Impact Profile, OHIP-14) was used [26]. All subjects were asked for information concerning disease activity, comorbidity and medication.

Education

The patients were asked to report their level of education defined by years of schooling i.e., elementary school (7 years), lower secondary school (3 years) and upper secondary school (3 years) in addition to vocational education (2–3 years) or upper secondary education (> 3 years).

Clinical Recordings

All periodontal recordings were done on six sites per tooth excluding third molars. Clinical data included oral hygiene (plaque registered dichotomously) [27] with a manual probe (LM 52851 Perioprobe ErgoNorm, LM instruments, Parainen, Finland), periodontal probing depth (PPD), clinical attachment level

(CAL), bleeding on probing (BoP) (10 s)[28], horizontal furcation defect (degree I-III) with a Nabers probe [29] and mobility (0-III)[30]. Clinical attachment level (CAL) was defined as the probing depth and the distance from the gingival margin to the cementum enamel junction (CEJ).

Radiographic recordings

All patients had a series of fourteen intra-oral periapical radiographs and two vertical bitewings using conventional position holders (Eggensholder)[31] and Digora® photostimulable phosphor plates. Periapical radiographs were taken by a 63-kV intraoral x-ray system with an exposure time of 0.08–0.12 seconds depending on anterior or posterior positioning. Intraoral standardized radiographs were taken with the long-squared cone using parallel technique. Bone loss (BL) was evaluated by measuring the linear distance between the CEJ to the level where a normal width of lamina dura could be detected. These measurements were done with a dedicated dental software (VisiQuick).

The clinical assessment was performed during the same appointment as the radiographic recordings and the questionnaire. Both clinical and radiographic recordings and interpretations were done by the main investigator (HOH).

Periodontitis case definition

The definition of periodontitis according to the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions was applied [32]. Periodontitis was defined as the distance between the CEJ and the AC exceeding 2mm at ≥ 2 non-adjacent teeth on radiographic recordings. Alternatively, periodontitis was diagnosed by a clinical assessment with one of two criteria: either (1) the interdental CAL detectable at ≥ 2 non-adjacent teeth, or (2) buccal or oral CAL of ≥ 3 mm with pocketing > 3 mm is detectable at ≥ 2 teeth. Clinical attachment loss (CAL) was defined as clinical attachment level exceeding 2 mm. In cases of < 10% BoP, < 4 mm PPD a periodontitis case was regarded as stable. In cases where BoP exceeded 10% with presence of PPD ≥ 4mm a case was defined as unstable.

Statistics

All statistical analysis was performed using SigmaPlot version 14.0 (Systat Software GmbH). A Spearman correlation test was performed to analyze correlation between periodontitis and the parameters of endoscopic inflammation (Mayo score), clinical inflammation (CAI) and years with UC diagnosis. The demographic data are presented as descriptive statistics. Recordings of OHRQoL are shown in Fig. 2 at the group level.

Results

The demographic data and oral disease characteristics such as periodontitis, DMFT, hyposalivation and mucosal lesions are presented in Table 1.

Table 1
Patient demographics and disease characteristics

	N (%)	Mean (±)	Range
Women	21 (42)		
Age		51.9 (12.61)	36–81
Level of education, years		15,66	12–20
Age at first diagnosis		26.52 (10.43)	12–56
Disease duration		24.76(9.25)	13–54
Symptoms of UC at time of oral examination	11 (22)		
Daily toothbrushing	50 (100)		
Using interdental brushes daily	8 (16)		
Regular dentist /hygienist	39 (78)		
≥ 2 dental visits per year	14 (28)		
smoking			
-never	27 (54)		
-current	4 (8)		
-previous	19(38)		
DMFT		11.42	1–25
Challacomb scale		0.34	0–10
Genetic susceptibility to periodontitis (other family members with periodontitis)	11 (22)		
FMPI		38,84 (22.34)	
FMBI		53.94 (34.32)	
PPD > 6mm	35 (0.4)		

The study sample consisted of 50 individuals (29 males), aged between 36 and 81 years (mean 51.9 years) Eight of the individuals reported on general health problems (e.g., high blood pressure, diabetes, rheumatic problems and cardiac problems) and were on different types of medication in addition to UC medication.

All but 11 subject regularly visited a dentist or dental hygienist (78%), and all except 3 subjects reported that they never had received any form of “gum treatment”. The number of remaining teeth varied between 18–32 (mean 27,2). Three subjects presented 1 implant each, and 2 subjects had 2 implants each, 7 in total. None of the participants had lost teeth due to periodontitis.

Four individuals were current smokers (3–15 cig/day) and 19 were former smokers. Nine individuals reported daily use of moistened snuff.

Of the patients, 58% were treated with mesalazin, a type of 5- aminosalicylic acid (5- ASA). Other combinations of 5-ASA medication were also reported. None of the patients were currently treated with steroids or immunosuppressant drugs. Data shown in Table 2.

Table 2
Medication for Ulcerative colitis used in this population

Type of medication	Number of patients N, (%)
No medication	9 (18)
Mesalazin(Asacol,Mesavant,Mesasal, Pentasa)	29 (58)
Asacol + Pentasa	1 (2)
Colazid	2 (4)
Others	5 (10)
Asacol + additional	3 (6)
Pentasa + additional	1 (2)
Complementary medicine (cannabis)	3
Vitamin supplementation (D + B,D or B)	17

Mucosal oral lesions were absent except for 4 individuals displaying leukoplakia. Average DMFT score was 11.42 with a range of 25 to 1. Mean hyposalivation according to the challacombe scale was 0.34. A total of 39 participants scored 0.

The distribution of periodontitis stages and grades is shown in Table 3 and Fig. 1. Patients not receiving UC medication (n = 9) were represented in all stages of periodontitis (I, II,III) as well as among the non-periodontitis cases. Periodontal disease status (stage/grade/PPD > = 6mm/BoP) did not correlate to

previous assessments of inflammation in the colon and symptoms (Mayo, CAI) or with number of years with UC disease. FMPI correlated significantly with BOP as shown in Table 4.

Table 3
Allocation of periodontal stage and grade in number (N) and percentage (%) of the total population assessed radiographically and clinically

Stage	Total	Grade A	Grade B	Grade C	Localized N (%)	Generalized N (%)
No periodontitis	13 (26)					
Stage I	9(18)	1 (2)	8(16)		0	9 (18)
Stage II	11(22)		10 (20)	1(2)	5 (10)	6 (12)
Stage III	17 (34)		16 (32)	1(2)	16 (32)	1 (2)
Stage IV	0				0	0

Table 4
Correlation analysis of previous intestinal inflammation (Mayo score and CAI) and periodontitis

	Periodontitis (stage 0,1,2,3)	Periodontitis (grade A,B,C)	PPD ≥ 6 (n)	BOP (n_b)	OHIP-14
	r, P-value	r, P-value	r, P-value	r, P-value	r, P-value
CAI (0,1,2,3..8)	-0.163	-0.159	-0.114	-0.0427	-0,179
	0.273	0.286	0.444	0.775	0.227
MAYO (0,1,2,3)	-0,289	-0.0363	0.0993	-0.0564	0.0169
	0.144	0.818	0.530	0.722	0.914
FMPI	0.248	-0.044	-0.147	0.0535	-0,196
	0.0822	0.760	0.308	0.000072	0.172

n = number of PPD ≥ 6mm, n_b= number of sites with bleeding on probing

The observed results from the OHIP-14 questionnaire shows highest total score (33/200) on Question 3 (Q3, functional limitation due to physical pain from teeth) and Question 4 (Q4, functional limitation because of discomfort while eating). The distribution of OHIP-14 is shown in Fig. 2 and represents the total score for the group.

Discussion

In this study, a periodontal examination was conducted in a population of UC-subjects and periodontitis defined according to the new classification of periodontal disease. Furthermore, periodontal status was

correlated to previous data on disease severity and symptoms of UC (Mayo Score and CAI), and years with UC diagnosis. The majority of subjects presented with periodontitis, of which 34% stage III, 22% stage II and 18% stage I. 26% of the population were diagnosed as non-periodontitis cases. Two patients were diagnosed as periodontally healthy and 11 patients with gingivitis. No correlations were found between previous scores of UC disease severity and symptoms (MAYO and CAI), years with UC diagnosis and the severity of the periodontal disease (stage, grade, BoP, PPD > 6mm).

A recently published article stated there is an emergent need to perform studies on the periodontal disease according to the World Workshop on the Classification scheme for periodontal and peri-implant diseases and conditions 2017 in subjects with IBD [34],[35].

The findings of the present study is in agreement with a recent study on the prevalence of periodontitis in Norway by Stødle et al. [33]. In their sample population of nearly 5000 individuals from Trøndelag county, the prevalence of periodontitis was reported to be 2.3%, 15%, 40% and 12% for stages IV, III, II, and I, respectively, and only 28% were not diagnosed with periodontitis. The present study recorded no case of stage IV and a higher prevalence of stage III. This difference might be explained by the smaller sample size of the present study but also by the diagnostic means. In contrast to Stødle et al., the present study included a more comprehensive examination including clinical assessments of furcation defects and CAL. This most likely led to the diagnosis of more stage III localized cases as compared to the large-population study with a higher percentage of stage II. When summarizing all periodontitis cases regardless of stage the outcome in both populations was similar (74% vs 72.4%). This study shows a higher prevalence of periodontitis compared to other recent studies from Norway [36, 37] and this could be explained by different demographics, inter-observational differences between the examiners and different thresholds for defining periodontitis.

Periodontitis showed no correlation with Mayo and CAI scores (Table 4). This could be explained by the time lapse between the periodontal examination and the historical records of Mayo and CAI. Another explanation is the fact that most patients were medically well-maintained and that the majority (78%) of the patients reported no symptoms of UC at the time of periodontal examination. As pointed out by Vavrica et al., there are very few studies analyzing the effect of IBD medication or disease activity on the periodontal status [10]. This study could not confirm any such association. This may be because the association was too weak to be detected given the small population with limited symptoms.

Based on studies reporting on the symptom-relieving effect of tobacco use among UC patients [39], information on smoking habits and the use of smokeless tobacco was analyzed to see if this was of significance [40]. A recent study by Kang et al. [41] reported that periodontitis and smoking increase the risk of UC. Since there were few patients reporting on tobacco use in the current study, no conclusions could be drawn. Interestingly, there were three patients (6%) smoking cannabis as self-medication. This finding is supported by a newly published article which states that approximately 10–20% patients with IBD are active cannabis users [40].

The OHIP-14 data showed limited negative impact on QoL in this population. This is partly in contrast to studies reporting on the negative influence of UC on health-related quality of life [42]. A recent study by Goldinova et al. [43], reported a near-significant correlation of the OHIP-14 scores with a simple clinical colitis activity index (SCCAI) and IBD questionnaire (IBDQ-9). In a national cross-sectional Norwegian study from 2011 using the OHIP-14 [44] the proportion of individuals who reported problems ranged from 11–56%, with pain as the most frequently reported item. The most frequently reported problem was physical pain (56%), followed by psychological discomfort (39%) and psychological disability (30%). The most frequently experienced problems were physical pain, such as aching in the mouth (Q3), and discomfort eating food(Q4), which is in agreement with our study and other Scandinavian studies [45, 46]. To our knowledge this is the first study to report on oral-health-related quality of life, periodontitis and UC combined.

The data from this population could not confirm a correlation between periodontitis severity and Mayo score, CAI or years with UC diagnosis.

The use of Mayo score and CAI obtained at a single time point some years before the periodontal examination can be questioned.

This study has other noteworthy limitations with the small number of participants being the most obvious. On the other hand, the recruitment of a high number of participants with long-term UC diagnosis may be challenging. Although the study coincided with the first and second waves of the COVID-19 pandemic in Norway, the majority of invitees (50/63) were willing to participate in the study (78%), which reflects a highly motivated and compliant cohort. The study was conducted in a community hospital in which patients may present with a less aggressive UC than seen in advanced units, as pointed out in the study by Klepp et al [22]. The population examined was a selection of subjects remaining throughout the original study and furthermore agreeing to a periodontal examination years later. It is therefore possible that those with more severe UC did not take part. In general, the population examined were successfully treated as suggested by the OHRQoL-data and patient-reported data reported herein.

The strength of the study is the periodontal diagnostic means according to the new classification and consideration of patients' OHRQoL with a long duration of UC. In agreement with Lorenzo-Pouso et al. [34], more evidence of a potential link between periodontitis and UC is needed.

Conclusion

Within the limitations of this study, periodontitis among patients with UC in the examined population was in line with that reported in a general population. No correlation between periodontitis or periodontal inflammatory indexes and UC disease indexes or years with UC diagnosis was observed. Our findings require confirmation in further studies with a larger population of UC patients.

Abbrevations

AC: Alveolar bone crest

ASA: aminosalicylic acid

B.O.P: Bleeding on probing

BD: Bone defect

BL: Bone loss

CD: Crohns Disease

CRC: Colorectal cancer

CAI: Colitis activity index

CAL: clinical attachment loss

CAM: Complementary and alternative medicine

FMPI: Full mouth plaque score

FMBS: Full mouth bleeding score

DMFT: Decayed missing filled teeth

HRQoL: Health related quality of life

IBD: Inflammatory bowl disease

LDS: Lovisenberg Diaconal Hospital

OHIP: Oral health related impact

OHRQoL: Oral health related quality of life

PPD: Periodontal probing depth

UC: Ulcerative colitis

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Regional Committee for Medical and Health Research Ethics (REC Project NO. 2010/1093) and all methods are in accordance with the Declaration of Helsinki. All participants signed an informed consent on participation and for publication of the results.

Availability of data and materials

The datasets used and/or analyzed and questionnaires during the current study are available from the corresponding author on request.

Competing interests

The authors declare that they have no competing interests.

Funding

Not applicable.

Authors' contributions

HOH and AV designed the study. HOH collected the data. HOH and AV analyzed, and interpreted the data. HOH and AV drafted the manuscript. HOH, AV and PK review the manuscript. HOH, AV and PK revised the manuscript. All authors have read and approved the final version of the manuscript.

Acknowledgment

We thank: Anita Tollisen, Unger-Vetlesen Institute, Department of Internal Medicine, Lovisenberg Hospital, Lovisenberggt.17, 0456 Oslo,Norway.

Author information

Helena O.Haugbo[1,3*] (HOH), Pasquale Klepp[2] (PK),Anders Verket [3] (AV)

[1]Master of dentistry, DDS, specialist candidate in Periodontology, University of Oslo. hohaugbo@student.odont.uio.no; ho.haugbo@lds.no

[2]PhD, MD, Lovisenberg Diakonale Sykehus, Oslo. pasklepp@gmail.com

[3]PhD, DDS, Department of Periodontology, University of Oslo, Norway. anderver@odont.uio.no

References

1. Levine JS, Burakoff R. Inflammatory Bowel Disease: Medical Considerations. In: Greenberger NJ, Blumberg RS, Burakoff R, editors. CURRENT Diagnosis & Treatment: Gastroenterology, Hepatology, & Endoscopy,. 3e ed. New York, NY: McGraw-Hill Education; 2016.
2. Bouma G, Strober W. The immunological and genetic basis of inflammatory bowel disease. Nat Rev Immunol. 2003;3(7):521-33.
3. Bengtson MB, Solberg C, Aamodt G, Sauar J, Jahnsen J, Moum B, et al. Familial aggregation in Crohn's disease and ulcerative colitis in a Norwegian population-based cohort followed for ten years.

J Crohns Colitis. 2009;3(2):92-9.

4. Lirhus SS, Høivik ML, Moum B, Anisdahl K, Melberg HO. Incidence and Prevalence of Inflammatory Bowel Disease in Norway and the Impact of Different Case Definitions: A Nationwide Registry Study. Clin Epidemiol. 2021;13:287-94.
5. Sica GS, Biancone L. Surgery for inflammatory bowel disease in the era of laparoscopy. World J Gastroenterol. 2013;19(16):2445-8.
6. Cosnes J, Gower-Rousseau C, Seksik P, Cortot A. Epidemiology and natural history of inflammatory bowel diseases. Gastroenterology. 2011;140(6):1785-94.
7. S A, et a. The global, regional, and national burden of inflammatory bowel disease in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(1):17-30.
8. Hoivik ML, Moum B, Solberg IC, Cvancarova M, Hoie O, Vatn MH, et al. Health-related quality of life in patients with ulcerative colitis after a 10-year disease course: results from the IBSEN study. Inflamm Bowel Dis. 2012;18(8):1540-9.
9. Høivik ML, Moum B, Solberg IC, Henriksen M, Cvancarova M, Bernklev T. Work disability in inflammatory bowel disease patients 10 years after disease onset: results from the IBSEN Study. Gut. 2013;62(3):368-75.
10. 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.
11. Flemmig TF, Shanahan F, Miyasaki KT. Prevalence and severity of periodontal disease in patients with inflammatory bowel disease. J Clin Periodontol. 1991;18(9):690-7.
12. Habashneh RA, Khader YS, Alhumouz MK, 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.
13. Fuggle NR, Smith TO, Kaul A, Sofat N. Hand to Mouth: A Systematic Review and Meta-Analysis of the Association between Rheumatoid Arthritis and Periodontitis. Front Immunol. 2016;7:80.
14. Ossum AM, Palm Ø, Cvancarova M, Solberg IC, Vatn M, Moum B, et al. Peripheral arthritis in patients with long-term inflammatory bowel disease. Results from 20 years of follow-up in the IBSEN study. Scand J Gastroenterol. 2018;53(10-11):1250-6.
15. Albandar JM, Susin C, Hughes FJ. Manifestations of systemic diseases and conditions that affect the periodontal attachment apparatus: Case definitions and diagnostic considerations. J Clin Periodontol. 2018;45(S20):S171-S89.
16. Hirschfeld J, Chapple ILC. Inflammatory bowl disease and periodontitis. In: Rangé H, Bouchard P, editors. Periodontitis and systemic diseases : clinical evidence and biological plausibility. Berlin: Quintessence; 2021. p. 112-31.
17. 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.
18. Burisch J, Munkholm P. The epidemiology of inflammatory bowel disease. Scand J Gastroenterol. 2015;50(8):942-51.
19. Cosnes J, Cattan S, Blain A, Beaugerie L, Carbonnel F, Parc R, et al. Long-term evolution of disease behavior of Crohn's disease. Inflamm Bowel Dis. 2002;8(4):244-50.
20. Pittock S, Drumm B, Fleming P, McDermott M, Imrie C, Flint S, et al. The oral cavity in Crohn's disease. J Pediatr. 2001;138(5):767-71.
21. 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.
22. Klepp P, Tollisen A, Røseth A, Cvancarova Småstuen M, Andersen SN, Vatn M, et al. Real-life chromoendoscopy for dysplasia surveillance in ulcerative colitis. World J Gastroenterol. 2018;24(35):4069-76.
23. Schroeder KW, Tremaine WJ, Ilstrup DM. Coated oral 5-aminosalicylic acid therapy for mildly to moderately active ulcerative colitis. A randomized study. N Engl J Med. 1987;317(26):1625-9.
24. Walmsley RS, Ayres RC, Pounder RE, Allan RN. A simple clinical colitis activity index. Gut. 1998;43(1):29-32.
25. Osailan SM, Pramanik R, Shirlaw P, Proctor GB, Challacombe SJ. Clinical assessment of oral dryness: development of a scoring system related to salivary flow and mucosal wetness. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012;114(5):597-603.
26. Slade GD. Derivation and validation of a short-form oral health impact profile. Community Dent Oral Epidemiol. 1997;25(4):284-90.
27. O'Leary TJ, Drake RB, Naylor JE. The plaque control record. J Periodontol. 1972;43(1):38.
28. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J. 1975;25(4):229-35.
29. Hamp SE, Nyman S, Lindhe J. Periodontal treatment of multirooted teeth. Results after 5 years. J Clin Periodontol. 1975;2(3):126-35.
30. Nyman S, Lindhe J, Lundgren D. The role of occlusion for the stability of fixed bridges in patients with reduced periodontal tissue support. J Clin Periodontol. 1975;2(2):53-66.
31. Eggen S. Standardiserad intraoral röntgenteknik. . Sver Tandlakarforb Tidn. 1969;17:867-72.
32. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. J Periodontol. 2018;89 Suppl 1:S159-s72.
33. Stødle IH, Verket A, Høvik H, Sen A, Koldsland OC. Prevalence of periodontitis based on the 2017 classification in a Norwegian population: The HUNT study. J Clin Periodontol. 2021;48(9):1189-99.
34. 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.

35. Papapanou PN, Sanz M, Buduneli N, Dietrich T, Feres M, Fine DH, et al. Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol. 2018;45 Suppl 20:S162-s70.
36. Holde GE, Oscarson N, Trovik TA, Tillberg A, Jönsson B. Periodontitis Prevalence and Severity in Adults: A Cross-Sectional Study in Norwegian Circumpolar Communities. J Periodontol. 2017;88(10):1012-22.
37. Bongo AS, Brustad M, Oscarson N, Jönsson B. Periodontal health in an indigenous Sámi population in Northern Norway: a cross-sectional study. BMC Oral Health. 2020;20(1):104.
38. Goodson JM, Haffajee AD, Socransky SS. The relationship between attachment level loss and alveolar bone loss. J Clin Periodontol. 1984;11(5):348-59.
39. Thomas T, Chandan JS, Li VSW, Lai CY, Tang W, Bhala N, et al. Global smoking trends in inflammatory bowel disease: A systematic review of inception cohorts. PLoS One. 2019;14(9):e0221961.
40. Rozich JJ, Holmer A, Singh S. Effect of Lifestyle Factors on Outcomes in Patients With Inflammatory Bowel Diseases. Official journal of the American College of Gastroenterology | ACG. 2020;115(6):832-40.
41. 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.
42. Huppertz-Hauss G, Høivik ML, Langholz E, Odes S, Småstuen M, Stockbrugger R, et al. Health-related quality of life in inflammatory bowel disease in a European-wide population-based cohort 10 years after diagnosis. Inflamm Bowel Dis. 2015;21(2):337-44.
43. Goldinova A, Tan CX, Bouma G, Duijvestein M, Brand HS, de Boer NK. Oral health and salivary function in ulcerative colitis patients. United European Gastroenterology Journal. 2020;8(9):1067-75.
44. Dahl KE, Wang NJ, Skau I, Ohrn K. Oral health-related quality of life and associated factors in Norwegian adults. Acta Odontol Scand. 2011;69(4):208-14.
45. Einarson S, Gerdin EW, Hugoson A. Oral health impact on quality of life in an adult Swedish population. Acta Odontol Scand. 2009;67(2):85-93.
46. Lahti S, Suominen-Taipale L, Hausen H. Oral health impacts among adults in Finland: competing effects of age, number of teeth, and removable dentures. Eur J Oral Sci. 2008;116(3):260-6

Figures

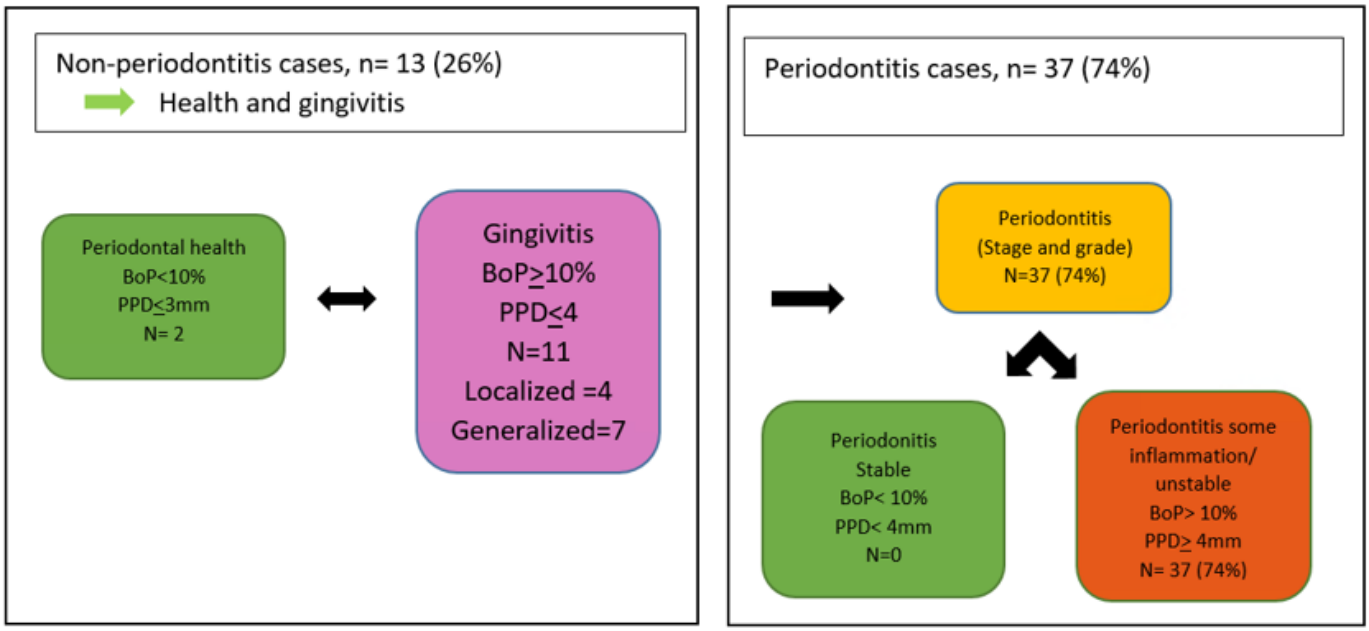

Figure 1

Distribution of participants with periodontal health, gingivitis, and periodontitis. All percentages (%) are calculated from the study population of 50. Definitions of stable periodontitis and periodontitis with some inflammation/unstable are modifications of the 2017 classification.

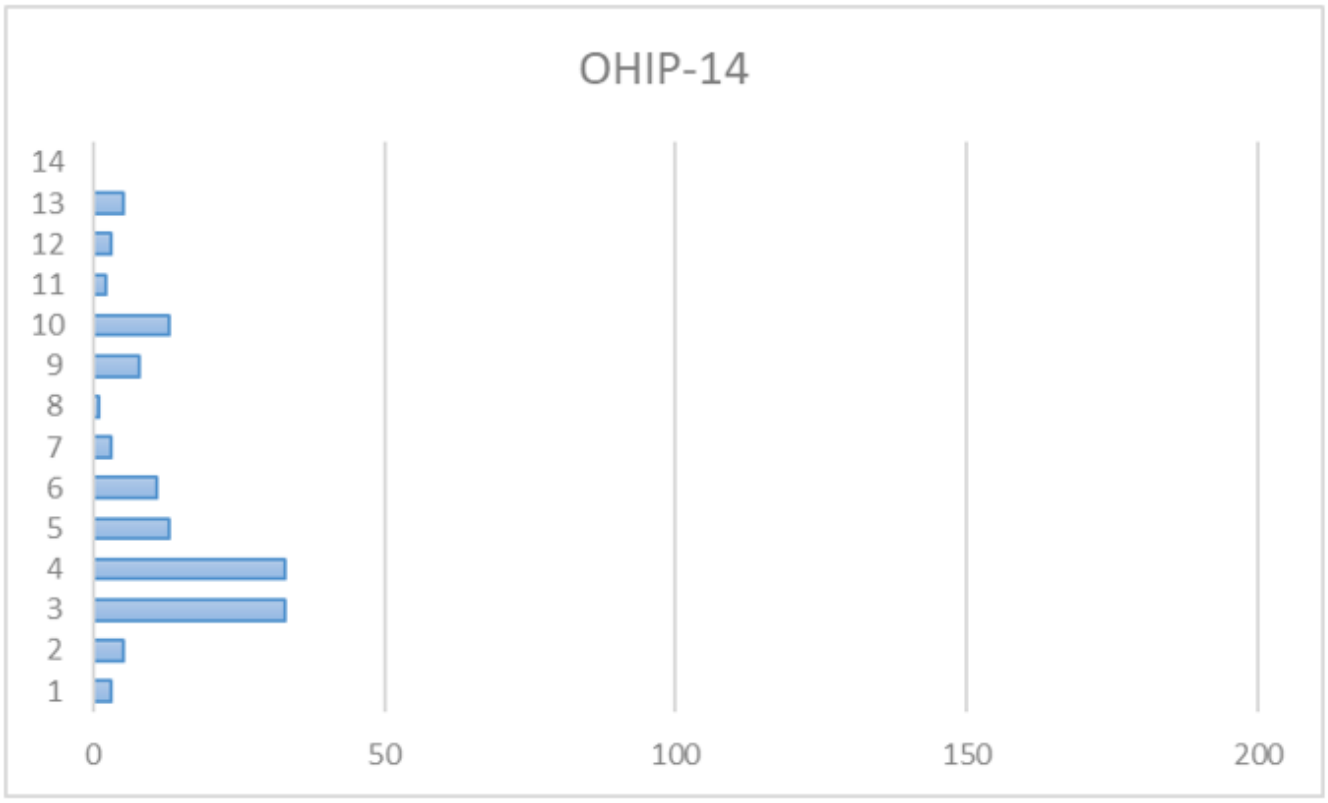

Figure 2

Distribution of OHRQqL all patients as a total