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Dental caries, oral hygiene and *Streptococcus mutans* serotypes in patients with inflammatory bowel disease

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Abstract

Introduction and Objective. Inflammatory bowel disease (IBD), comprising Crohn's disease (CD) and ulcerative colitis (UC), is a chronic inflammatory condition with systemic and extra-intestinal manifestations. Clinical findings in the oral cavity include, among others, a higher caries burden and microbiome dysbiosis linked to the oral-gut axis with *Streptococcus mutans* playing an important role.

Materials and Method. The study included 110 IBD patients in remission (73 CD, 37 UC) and 50 controls. Participants underwent oral cavity examination (DMFT, API) and completed a questionnaire on oral hygiene. Unstimulated saliva from IBD patients was analysed for *S. mutans* detection by PCR and serotyping by multiplex (c, e, f) and singleplex (k) PCR.

Results. DMFT was higher in CD and UC patients (mean=19.08 and 19.51) than controls (mean=15.04; p=0.007), with elevated D (p<0.001) and M (p=0.011) components in CD group and no difference in F component. API was higher in CD group than in controls (mean=67.77 vs 53.03; p=0.020), with significant differences in API ranges for CD vs controls (p<0.001) and UC vs controls (p<0.05). Insufficient oral hygiene was noted in 49% of CD and 43% of UC patients, and average hygiene in 41% of CD and 46% of UC. Oral hygiene habits differed only in toothbrushing frequency, with CD patients brushing less frequently than controls (p=0.017). Higher D component counts and API were associated with increased IBD odds. CD and UC patients were 5.95- and 5.17-fold more likely to have insufficient or average oral hygiene. *S. mutans* was detected in 78% of IBD patients, with serotype k in 31%. In UC group, rural residence was linked to higher DMFT, with no such effect in CD patients.

Conclusions. These findings underscore the importance of targeted oral hygiene education for IBD patients and support further research on the oral microbiome.

Key words

inflammatory bowel disease, dental caries, oral hygiene, DMFT, *Streptococcus mutans*, Crohn`s disease, ulcerative colitis, Approximal Plaque Index

INTRODUCTION

Inflammatory bowel disease (IBD) represents a group of chronic, relapsing inflammatory disorders of the gastrointestinal tract, including Crohn's disease (CD) and ulcerative colitis (UC). CD is characterized by transmural, segmental inflammation that can affect any part of the gastrointestinal tract, whereas UC involves continuous mucosal inflammation limited to the colon and rectum [1]. The exact aetiology of IBD has not been fully elucidated, however, current evidence supports a multifactorial disease model arising from complex interactions among genetic predisposition, immune system dysregulation, microbial factors and environmental triggers [1, 2].

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Globally, IBD affects approximately 230 per 100,000 people, with both CD (84.2 per 100,000) and UC (120.4 per 100,000) contributing substantially to this burden. In Europe, 2.5 to 3 million individuals are estimated to live with IBD with increasing incidence and prevalence, and in Poland recent nationwide data indicate that nearly 97,000 people lived with the disease in 2020, including over 23,000 with CD and 73,000 with UC [3–5].

IBD causes numerous systemic complications such as anaemia, micronutrient deficiencies, osteoporosis, fatigue and impaired quality of life. Extra-intestinal manifestations occur in up to 50% of IBD patients, commonly involving the skin, joints, eyes and oral cavity, which is increasingly recognized as a site affected by systemic inflammation [6–8]. In the oral cavity IBD population presents with mucosal lesions, gingival inflammation, xerostomia, and higher prevalence of dental caries and periodontal disease compared with healthy controls [9, 10].

Dental caries is a highly prevalent, multifactorial, biofilm-

mediated disease involving the demineralization of tooth tissues by acids produced from bacterial fermentation of dietary carbohydrates [11, 12]. Modern cariology emphasizes ecological changes in the oral microbiome rather than attributing disease to single pathogens alone [12]. While *Streptococcus mutans* and *Lactobacillus* spp. are still considered key contributors, caries development is increasingly understood as a consequence of broader dysbiosis within the dental biofilm [2, 13]. In this context, traditional oral hygiene practices – including toothbrushing frequency and duration, flossing and regular dental visits – remain crucial etiopathogenetic factors. Together with dietary habits such as sugar intake, they influence the composition and ecological balance of the dental biofilm, thereby modulating an individual's risk of developing caries [11].

Various studies and recent meta-analyses have demonstrated significantly elevated decayed, missing, and filled teeth (DMFT) index values in IBD patients compared with controls [10, 14–16]. A potential link between dental caries and IBD may be provided by alterations in the oral microbiome. Studies of salivary and plaque microbiota in IBD populations describe significant shifts toward pro-inflammatory or potentially cariogenic bacterial communities compared to healthy individuals [17]. Evidence further suggests that the oral microbiota may contribute to intestinal inflammation in IBD through the translocation of oral microorganisms to the gut, disruption of intestinal barriers, and modulation of immune responses. This concept, known as the oral–gut axis, may involve bidirectional interactions between oral and gut microbiota, influencing both local and systemic inflammatory responses [2, 18].

Traditionally recognized as a major cariogenic pathogen, *S. mutans* was also reported at altered levels in the oral microbiota of IBD patients, reflecting disease-associated changes in the composition of oral bacterial communities [12, 18, 19]. Within this species, 4 main serotypes (c, e, f, and k) were identified, differing in cell wall polysaccharides and virulence traits, which may influence their pathogenic potential in both oral and extra-oral contexts [20]. The possible relevance of *S. mutans* serotype heterogeneity in IBD remains an open question in current research.

The present study aimed to assess caries status using the DMFT index and its components as well as oral hygiene and related habits in patients with IBD compared with non-IBD individuals. In addition, the study investigated the prevalence and serotype distribution of *S. mutans* within the IBD group, to explore potential microbiological factors contributing to cariogenic and pro-inflammatory oral profiles.

MATERIALS AND METHOD

Participants. The study included 110 patients aged 18–72 years with IBD, comprising 73 diagnosed with CD and 37 with UC. Patients were hospitalized at the Department of Gastroenterology with IBD Unit, St. Jadwiga Queen Hospital No. 2 in Rzeszów, Poland. All IBD patients were in remission of the disease. The control group consisted of 50 individuals, matched for gender, age and place of residence, recruited from patients attending the Chair and Department of Conservative Dentistry with Endodontics of the Medical University of Lublin, with no diagnosis or symptoms of gastrointestinal disease. The IBD and control groups did not differ in socio-demographic characteristics, therefore, these factors were not considered to influence the examined parameters. Participants` characteristics are presented in Table 1.

All participants, from both the study and control groups, underwent a clinical oral examination and completed a questionnaire-based survey. In addition, unstimulated whole saliva samples were collected from patients with IBD.

Clinical examination. Clinical examination included dental assessment with DMFT calculation and evaluation of oral hygiene status. Dental caries were examined under artificial lighting using a plane mouth mirror and DMFT was determined according to WHO criteria [21].

Oral hygiene status was evaluated using the Approximal Plaque Index (API). Plaque presence was assessed by gently placing a dental probe through the approximal spaces. The first and third quadrants were assessed from the oral aspect, and the second and fourth quadrants from the buccal aspect. Presence of plaque at each site was recorded as a positive result. API was calculated as the percentage of positive sites and categorized as: < 25% – optimal hygiene; 25–39% – rather good; 40–69% – average; 70–100% – poor oral hygiene [22].

Questionnaire-based survey. The study-specific questionnaire included socio-demographic data (age, sex, place of residence) and oral hygiene habits, such as toothbrushing frequency and duration, use of additional oral hygiene products and frequency of dental check-up visits.

Saliva collection and analysis. Unstimulated whole saliva samples were collected from all 110 IBD patients. DNA was extracted using the QIAamp DNA Mini Kit (Qiagen). Quantification and identification of *S. mutans* were performed using specific primers in Real-Time PCR (LightCycler 96, Roche) [23]. Multiplex PCR was conducted to detect serotypes c, e, and f using 3 sets of primers (SC-F/SC-R, SE-F/SE-R, SF-F/SF-R) with the following cycling parameters: 96 °C for 2 min, followed by 25 cycles of 96 °C for 15 s, 61 °C for 30 s, and 72 °C for 1 min [23]. Serotype k was detected using singleplex PCR with the following cycling conditions: 95 °C for 4 min, followed by 30 cycles of 95 °C for 30 s, 60 °C for 30 s, 72 °C for 30 s, with a final extension at 72 °C for 7 min [24].

Ethical considerations. The study protocol was approved by the Bioethics Committee of the Medical University of Lublin (Approval No. KE-0254/68/2015) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Statistical analysis. The obtained results were subjected to statistical analysis. Quantitative variables were presented as mean, standard deviation, median, interquartile range, minimum and maximum values, while qualitative variables – as counts and percentages. Associations between qualitative variables were assessed using the Chi-square test. Normality of distribution was evaluated with the Shapiro-Wilk test. Comparisons of DMFT and API between two groups were performed using the Mann-Whitney U test and among 3 groups using the Kruskal-Wallis test. Bonferroni correction was applied for multiple comparisons. Statistical analyses were performed using logistic regression models to explore the associations between oral health parameters and IBD.

Two separate analytical approaches were applied, addressing different outcome variables. In the first approach logistic regression models were fitted to identify risk factors associated with IBD. From these models, odds ratios (OR) and 95% Confidence Intervals (CI) were derived, corresponding p-values were obtained using Wald's test. Goodness of fit was checked using Hosmer and Lemeshow's test and statistical significance was set at p<0.05 (two-tailed). In the second approach, logistic regression was used to estimate the likelihood of average or insufficient oral hygiene (API ≥ 40%) in relation to selected parameters. Variables that reached statistical significance in univariate analyses were subsequently included in a multivariate logistic regression model.

A p-value < 0.05 was considered statistically significant. Analyses were performed using Statistica 9.1 and PQStat 1.8.2.

RESULTS

The study population comprised 65 men (47 with CD and 18 with UC) and 45 women (26 with CD and 19 with UC) (Tab. 1).

Analysis of the DMFT index revealed a statistically significant difference between the compared groups (p = 0.007). DMFT was significantly higher in patients with CD (mean = 19.08, median = 19) compared with the control group (mean = 15.04, median = 15), as well as in UC patients (mean = 19.51, median =18) compared to controls. No statistically significant difference in DMFT was observed between CD and UC patients (Tab. 2).

DMFT components analysis demonstrated that for the D component a significant difference (p < 0.001) was found between the CD group (mean = 4.66, median = 4) and controls (mean = 2.10, median = 2). For the M component, a significant difference (p = 0.011) was also noted only between the CD group (mean = 5.49, median = 3) and the control group (mean = 3.22, median = 2). In contrast, no statistically significant differences were found for the F component among the 3 groups (p = 0.233) (Tab. 2).

API was significantly higher in the CD group (mean = 67.77, median = 68.8) than in controls (mean = 53.03, median = 50.0; p = 0.020). No other significant API level differences were observed among the remaining groups (Tab. 2).

When API values were analysed by numerical ranges, statistically significant differences between groups were identified (p = 0.0004). Pairwise comparisons revealed significant differences between CD patients and controls (p < 0.001) and between UC patients and controls (p < 0.05), with no difference between CD and UC patients (p > 0.05) (Tab. 3). Assessment of oral hygiene status based on API ranges showed that 40.0% of controls had optimal or relatively good hygiene. In contrast, in the CD group, 49.32% of patients exhibited insufficient hygiene and 41.10% had an average level. In the UC group, insufficient and average hygiene were observed in 43.24% and 45.95% of patients, respectively (Tab. 3).

Regarding oral hygiene habits a statistically significant difference was observed only for toothbrushing frequency (p = 0.017), with CD patients brushing teeth less frequently than controls (Tab. 3).

Analysis of the studied parameters in the CD and UC groups by place of residence (urban vs. rural) showed statistically significant differences only in the UC group. Rural residents in this group had a higher DMFT index (p = 0.018), a lower D component (p = 0.049), and a higher M component (p = 0.02) than urban residents. No residence–related differences were found in the CD group (Tab. 4).

Logistic regression analyses examined associations between oral health parameters and IBD. In the first set of models, higher numbers of decayed teeth (D) and increasing API values were significantly associated with higher odds of IBD (D: OR = 1.33, 95% CI 1.14–1.55; p < 0.0001; API: OR = 1.014, 95% CI 1.0–1.03; p = 0.046) (Tab. 5). In the second set, with oral hygiene status (average or insufficient, API ≥ 40%)

Table 1. Baseline characteristics of the participants

Parameter			CD	UC	Controls	p value
Sex	Male	N	47	18	22	$Chi^2 = 5.606$ p = 0.061
		%	64.38%	48.65%	44.00%	
	Female	N	26	19	28	
		%	35.62%	51.35%	56.00%	
Age	M ± SD		33.9±12.2	33.8±9.1	33.2±12.3	H = 0.782 p = 0.677
	Me [Q1–Q3]		32 [25–40]	33 [28–36]	29 [23–44]	
	Min – Max		18–72	20–58	19–59	
Disease duration (in years)	M ± SD		5.34±4.71	4.16±4.14	–	Z = 1.281 p = 0.200
	Me [Q1–Q3]		5 [1–8]	3 [0.5–7]	–	
			0–17	0–13	–	
Place of residence	Rural	N	32	13	19	$Chi^2 = 0.896$ p = 0.639
		%	43.84%	35.14%	38.00%	
	Urban	N	41	24	31	
		%	56.16%	64.86%	62.00%	
Cigarette smoking	No	N	38	25	33	$Chi^2 = 3.553$ p = 0.169
		%	52.05%	67.57%	66.00%	
	Yes	N	35	12	17	
		%	47.95%	32.43%	34.00%	

M – mean; SD – standard deviation; Min – minimum; Max – maximum; Me – median; Z – Mann-Whitney test; H – Kruskal-Wallis test; p – p-value

Table 2. DMFT index, its components (D, M, F) and Approximal Plaque Index (API) values in study groups and controls

Parameter	Group	M	SD	Min	Q1	Me	Q3	Max	p value
DMFT	CD	19.08	5.12	8	16	19	22	31	H = 10.062 p = 0.007 CD > Controls*, UC > Controls*
	UC	19.51	6.64	6	15	18	25	32	
	Controls	15.04	7.74	0	9	15	22	29	
D	CD	4.66	3.53	0	2	4	7	16	H = 18.785 p < 0.001 CD > Controls***
	UC	3.43	2.95	0	1	4	5	12	
	Controls	2.10	2.20	0	0	2	4	8	
M	CD	5.49	5.96	0	2	3	6	25	H = 9.114 p = 0.011 CD > Controls*
	UC	5.35	6.78	0	2	4	5	31	
	Controls	3.22	4.39	0	0	2	4	19	
F	CD	8.93	5.53	0	5	8	13	21	H = 2.910 p = 0.233
	UC	10.73	5.67	1	8	11	13	23	
	Controls	9.72	5.15	0	7	10	14	19	
API	CD	67.77	23.22	16.0	53.6	68.8	89.3	100.0	H = 7.793 p = 0.020 CD > Controls*
	UC	63.75	24.95	0.0	42.9	61.9	84.4	100.0	
	Controls	53.03	32.74	0.0	26.9	50.0	89.3	100.0	

DMFT – decayed, missing, filled teeth index; API – Approximal Plaque Index; CD – Crohn Disease; UC – Ulcerative Colitis; IBD – Inflammatory Bowel Disease; M – mean; SD – standard deviation; Min – minimum; Q1 – lower quartile; Me – median; Q3 – upper quartile; Max – maximum; Z – Mann-Whitney test; H – Kruskal-Wallis test.
p – p-value; * p < 0.050; ** p < 0.010; *** p < 0.001

Table 3. Approximal Plaque Index (API) values and oral hygiene habits in study groups and controls

Parameter			CD	UC	Controls	p value
Oral hygiene state						
API	Optimal / relatively good	N	7	4	20	Chi^2 = 20.238 p = 0.0004 CD-Controls*** UC-Controls*
		%	9.59%	10.81%	40.00%	
	Average	N	30	17	15	
		%	41.10%	45.95%	30.00%	
	Insufficient	N	36	16	15	
		%	49.32%	43.24%	30.00%	
Oral hygiene habits						
Dental check-ups	Every 6 months	N	36	16	23	Chi^2 = 3.748 p = 0.441
		%	49.32%	43.24%	46.00%	
	Every year	N	19	13	20	
		%	26.03%	35.14%	40.00%	
	Less frequently	N	18	8	7	
		%	24.66%	21.62%	14.00%	
Toothbrushing frequency	3x / day	N	10	5	12	Chi^2 = 12.119 p = 0.017 CD-Controls*
		%	13.70%	13.51%	24.00%	
	2x / day	N	39	25	34	
		%	53.42%	67.57%	68.00%	
	1x / day	N	24	7	4	
		%	32.88%	18.92%	8.00%	
Toothbrushing duration	≥ 3 min	N	18	12	12	Chi^2 = 2.016 p = 0.733
		%	24.66%	32.43%	24.00%	
	2 min	N	45	21	34	
		%	61.64%	56.76%	68.00%	
	≤ 1 min	N	10	4	4	
		%	13.70%	10.81%	8.00%	
Additional oral hygiene aids	No	N	22	7	7	Chi^2 = 4.785 p = 0.091
		%	30.14%	18.92%	14.00%	
	Yes	N	51	30	43	
		%	69.86%	81.08%	86.00%	

p – p-value; * p < 0.050; ** p < 0.010; *** p < 0.001

Table 4. DMFT index, its components (D,M,F) and Approximal Plaque Index (API) in study groups and controls by place of residence

Parameter	Group	N	M	SD	Min	Q1	Me	Q3	Max	p value
					CD					
DMFT	Rural	32	19.59	5.42	8	16	18	25	28	Z = 0.702 p = 0.483
	Urban	41	18.68	4.90	9	14	19	22	31	
D	Rural	32	4.66	2.66	0	2.5	4	7	10	Z = 0.682 p = 0.495
	Urban	41	4.66	4.11	0	1	4	6	16	
M	Rural	32	6.41	7.12	0	1.5	3.5	7.5	25	Z = 0.654 p = 0.513
	Urban	41	4.78	4.83	0	2	3	6	20	
F	Rural	32	8.53	5.81	0	4	8.5	13	21	Z = -0.596 p = 0.551
	Urban	41	9.24	5.35	0	5	8	13	20	
API	Rural	32	67.27	24.48	17.86	49.78	64.30	91.52	100.0	Z = -0.184 p = 0.854
	Urban	41	68.17	22.48	16.00	53.60	71.42	85.70	100.0	
					UC					
DMFT	Rural	13	22.92	7.47	7	18	26	28	32	U = 82.0 p = 0.018
	Urban	24	17.67	5.47	6	14.5	17	21.5	27	
D	Rural	13	2.46	3.38	0	0	1	3	12	U = 94.0 p = 0.049
	Urban	24	3.96	2.61	0	2	4	5.5	10	
M	Rural	13	9.92	9.66	0	4	5	15	31	U = 61.5 p = 0.002
	Urban	24	2.88	2.21	0	1	3	4.5	8	
F	Rural	13	10.54	7.61	1	5	9	17	23	U = 144.0 p = 0.718
	Urban	24	10.83	4.49	1	9.5	11	12.5	19	
API	Rural	13	60.18	27.34	0.00	42.86	61.90	83.33	100.0	U = 144.0 p = 0.718
	Urban	24	65.68	23.95	32.14	42.86	62.50	88.62	100.0	
					Controls					
DMFT	Rural	19	15.58	9.08	4	6	16	24	29	U = 283.0 p = 0.828
	Urban	31	14.71	6.92	0	10	15	20	26	
D	Rural	19	2.37	2.41	0	0	2	4	8	U = 267.5 p = 0.593
	Urban	31	1.94	2.08	0	0	2	4	6	
M	Rural	19	5.21	5.49	0	0	4	10	19	U = 179.5 p = 0.021
	Urban	31	2.00	3.04	0	0	0	4	11	
F	Rural	19	8.00	5.60	1	4	6	12	19	U = 205.0 p = 0.075
	Urban	31	10.77	4.63	0	8	10	14	19	
API	Rural	19	57.73	33.97	3.60	23.80	60.71	100.0	100.0	U = 262.0 p = 0.526
	Urban	31	50.15	32.19	0.00	28.57	46.43	71.43	100.0	

M – mean; SD – standard deviation; Min – minimum; Q1 – lower quartile; Me – median; Q3 – upper quartile; Max – maximum; Z, U – Mann-Whitney test; p – p-value.

Table 5. Logistic regression analyses

Parameter	Univariate model			Multivariate model		
	p	OR	95% CI OR	p	OR	95% CI OR
Factors associated with IBD (IBD vs. controls)						
DMFT	0.014	1.07	(1.01–1.13)	–	–	–
D	0.0001	1.37	(1.17–1.60)	<0.0001	1.33	(1.14–1.55)
M	0.103	1.06	(0.99–1.14)	–	–	–
F	0.208	0.96	(0.9–1.02)	–	–	–
API	0.031	1.02	(1.01–1.03)	0.046	1.014	(1.0–1.03)
Factors associated with average or insufficient oral hygiene (API ≥40%)						
CD [vs. controls]	<0.001	6.286	2.400–16.463	0.001	5.948	2.165–16.343
UC [vs. controls]	0.005	5.500	1.687–17.933	0.007	5.165	1.553–17.177
IBD [vs. controls]	<0.001	6.000	2.586–13.919	–	–	–

DMFT – Decayed, Missing, Filled Teeth index; API – Approximal Plaque Index; CD – Crohn's disease; UC – ulcerative colitis; IBD – inflammatory bowel disease; OR – odds ratio; CI – confidence interval.
Note: ORs represent odds ratios for each model. For the first part, ORs correspond to the change in odds of IBD per unit increase in each clinical parameter. For the second part, ORs correspond to the change in odds of average or poor oral hygiene (API ≥ 40%) relative to controls. Intercepts are not reported as they do not represent clinically interpretable effects.

as the outcome, CD patients were 5.95 times more likely (OR = 5.95, 95% CI 2.17–16.34; p = 0.001) and UC patients 5.17 times more likely (OR = 5.17, 95% CI 1.55–17.18; p = 0.007) to exhibit average or insufficient oral hygiene (Tab. 5).

S. mutans was detected in 78.2% of IBD patients. Among *S. mutans*–positive samples, serotype c was found in 57.5%, e in 9.2%, k in 31.0%, and f in 2.3%. No significant differences were observed between CD and UC patients in serotype prevalence or mean *S. mutans* abundance (Fig. 1).

DISCUSSION

Despite growing interest in potential connections between IBD and oral health, there is still limited systematic information on the oral status of patients with IBD. Most reports on the oral features of IBD focus on soft tissue lesions and periodontal findings rather than dental caries. Therefore, the present study was aimed at evaluation of associations between dental caries and IBD. To assess the

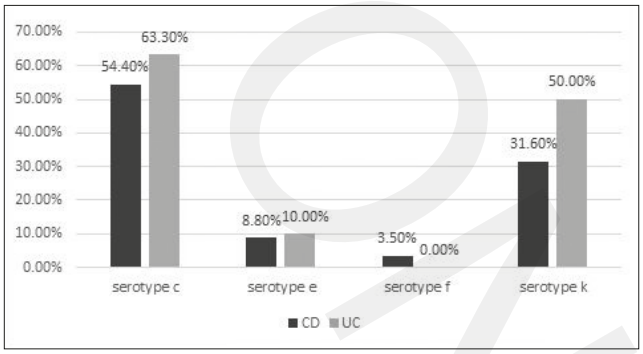

Figure 1. Prevalence of *S. mutans* serotypes c, e, f, and k in CD and UC groups

clinical manifestation of dental caries in this population, the DMFT index was used, providing a clinically relevant measure of cumulative and current caries experience.

In the current study, patients with both CD and UC exhibited significantly higher DMFT values compared with healthy controls, which is consistent with findings from several previous studies and meta-analyses indicating elevated DMFT values in IBD [14–16, 25, 26]. However, results from individual studies remain heterogeneous. Tan et al. [27] reported a significantly higher total DMFT values in patients with IBD than in controls, with a significant increase in CD group but not in UC patients. Similarly, Brito et al. [14], in a study involving only patients with CD, observed higher DMFT values compared with non-IBD individuals. Consistent results were also reported by Koutsochristou et al. [28] in children and adolescents with IBD. Grössner-Schreiber et al. [15] analysed DMF-surface (DMFS) index in IBD patients and found that, although total DMFS scores were higher in the study group compared with controls, the differences were not statistically significant. The authors, however, reported a significantly higher prevalence of dentine caries among IBD patients. Similarly, Rodrigues et al. [29] reported no statistically significant differences in overall DMFT values between patients with CD or UC and controls, although the values were slightly lower in both disease groups. Overall, these findings highlight substantial heterogeneity in caries experience among IBD populations, likely related to differences in study populations, disease characteristics, methodological approaches and other environmental or behavioural factors.

Analysis of the results of the present study revealed no significant difference in total DMFT between CD and UC patients, suggesting that increased caries experience may be a common feature of IBD rather than specific to one subtype of this disease. Similar results were reported by Grössner-Schreiber et al. [15], showing elevated DMFS values in both CD and UC patients compared with healthy controls. Detailed analysis of DMFT components revealed a significantly higher number of active, untreated carious lesions (D component) in CD and UC patients compared with controls, consistent with previous studies reporting elevated caries activity in IBD patients [16].

The potential relevance of untreated caries in the context of systemic disease was highlighted by the results of logistic regression analysis performed in the current study, which showed that higher numbers of decayed teeth (D) were significantly associated with IBD, with a 1.33-fold increase in the odds of the disease. These results align with previous findings: Zhang et al. [16] reported 4.27-fold and 2.21-fold increased odds of caries in CD and UC, respectively, while Grössner-Schreiber et al. [15] observed an association between dentine caries (RR = 2.82) and IBD. Taken together, these data highlight the importance of dental caries in patients with IBD and indicate a potential link between disease-related factors and oral health in this population.

Among established elements contributing to dental caries are factors such as frequent sugar intake and inadequate oral hygiene, cariogenic bacteria, enamel quality, salivary flow, fluoride availability and socio-economic status. It is noteworthy that most authors of published studies on this topic assessed dental caries using the DMFT/DMFS index, and also evaluated other selected etiological factors of caries, with the aim of exploring potential associations between this oral condition and the systemic disease that is IBD. Tan et al. [27] reported a higher prevalence of xerostomia in the overall IBD population and in CD patients, while oral hygiene behaviours were largely comparable to those of healthy controls, indicating that daily oral care alone may not explain the observed differences in DMFT. In turn, Szymanska et al. [30] observed higher caries scores in CD patients after resection surgery, accompanied by increased dental plaque, elevated salivary counts of cariogenic bacteria, including *S. mutans*, and more frequent consumption of sweetened beverages. However, no significant differences were observed by these authors between patients and controls with regard to the frequency of dental visits or to oral hygiene habits, including toothbrushing frequency and the use of approximal aids. Grössner-Schreiber et al. [15] found significantly higher plaque indices in IBD patients and linked these findings to changes in eating patterns, including more frequent food intake from the time of diagnosis. In contrast, Zhang et al. [16] reported no significant differences in meal frequency and observed less frequent consumption of sugary foods among IBD patients, although plaque accumulation remained higher and caries risk factors were still elevated. These observations highlight both consistencies and discrepancies in oral health outcomes among different IBD groups.

The present study demonstrated higher API values in both CD and UC patients compared with controls and logistic regression indicated that poorer oral hygiene (API ≥ 40%) was significantly associated with IBD. In line with our findings, Grössner-Schreiber et al. [15] reported a small but significant effect of plaque index on IBD (RR = 1.027), supporting the notion that plaque accumulation may be associated with IBD. However, despite these worse oral health indicators, questionnaire-based assessment in the current study revealed only limited differences in self-reported daily oral hygiene practices. A significantly lower toothbrushing frequency was reported only in CD patients, while toothbrushing duration, use of additional oral hygiene aids, and dental visits did not differ, reflecting the influence of factors such as systemic-oral interactions, treatment, lifestyle, disease severity and self-report limitations. Nevertheless, in one self-reported study, IBD patients frequently experienced severe periodontitis, high rates of tooth loss, and various other oral problems, including discomfort, difficulties with daily oral function, and the need for professional dental care. Many patients reported limited guidance from healthcare providers regarding oral health problems related to IBD, along with high dental treatment costs. These findings underscore the need for enhanced preventive measures, professional support and patient education in IBD beyond daily oral hygiene practices [31].

Singhal et al. [32], investigating oral hygiene and IBD, found that IBD patients practiced more intensive oral hygiene, visited the dentist more frequently, and exhibited more oral complications, such as dental caries, oral ulcers and xerostomia. They suggested these practices might alter the oral microbiota, potentially contributing to gut microbial imbalance and IBD pathogenesis, while oral manifestations may reflect disease-related changes influencing microbial composition. In contrast, Yin et al. [33] reported an inverse association between poor oral hygiene and IBD risk, highlighting the complex, bidirectional link between oral health, microbiota and intestinal inflammation. Taking together both studies appear to suggest that the increased caries experience observed in IBD patients may not be explained solely by deficiencies in routine oral hygiene practices.

Since environmental factors may also contribute to the development of dental caries and IBD, an additional analysis of DMFT, API, oral hygiene status and habits according to place of residence was performed in IBD patients. Statistically significant differences were observed only in the UC group, with higher DMFT index and M component in rural compared with urban residents, whereas no such differences were found in CD patients. To the best of the authors' knowledge, no previous studies have simultaneously evaluated these parameters in IBD populations, limiting the possibility of direct comparison and indicating an area that remains insufficiently investigated.

In the literature there are also studies indicating that increased dental caries experience in IBD patients may be associated with alterations in the oral microbiota rather than with traditional behavioural or dietary factors. Rodrigues et al. [29] reported that in UC patients, DMFT scores were unrelated to sugar intake, oral hygiene practices, disease activity or duration, or pharmacotherapy, despite normal salivary flow and buffering capacity. Elevated levels of *S. mutans* were frequently observed, particularly in patients with active and long-standing disease, indicating that behavioural caries risk factors may not fully account for the high caries prevalence in UC. Consistently, Han et al. [18] demonstrated oral microbiota dysbiosis in IBD patients, marked by increased pathogenic bacteria, including *Streptococcus mutans*, *Porphyromonas gingivalis*, and *Fusobacterium nucleatum*, and reduced beneficial commensals.

Alterations in the oral microbiota, including changes in bacterial composition and host immune interactions, may promote the expansion or increased activity of specific species, such as *S. mutans*, thereby contributing to systemic inflammatory responses. Experimental studies have shown that *S. mutans*, a key cariogenic pathogen, can trigger immunological cascades and disseminate beyond the oral cavity using rhamnose-glucose polysaccharides (RGP). The bacterium may enter the bloodstream during dental procedures such as tooth extraction or endodontic treatment, or during routine toothbrushing, evade immune clearance, adhere to vascular and organ tissues and induce inflammation, potentially contributing to systemic conditions including infective endocarditis, cerebral haemorrhage and IBD [20]. These effects are mediated by RGP, collagen-binding proteins and pro-inflammatory cytokines, identifying *S. mutans* as a clinically relevant target. Moreover, oral bacteria, including *S. mutans*, can translocate to the gut and exacerbate intestinal inflammation through systemic and mucosal immune activation. In patients with IBD, oral microbiota alterations – such as increased *Bacteroidetes* and reduced *Proteobacteria* – together with changes in oral immune markers, may further support the expansion or enhanced virulence of *S. mutans*, thereby linking oral dysbiosis to intestinal inflammation [20].

S. mutans is classified into 4 main serotypes (c, e, f, and k) with distinct profiles in cellular adhesion, immune evasion, and systemic pathogenic potential [34, 35]. In healthy populations, serotype c predominates (70–80%), followed by serotype e (~20%), while serotypes f and k typically occur at frequencies below 5%, consistent with findings from various geographical regions [35, 36]. Serotype k, recently identified, carries a defective glucose side-chain in its RGP, conferring resistance to phagocytosis and is characterised by increased systemic invasiveness as evidenced by its frequent isolation from cardiovascular tissues [34].

In the present study, the high proportion of serotype k in the IBD group contrasts with its low prevalence in healthy populations, but aligns with reports of its enhancement in systemic disease contexts [20]. Notably, no significant differences in serotype distribution or mean *S. mutans* load were observed between CD and UC patients, suggesting that the dysbiotic shift toward more virulent serotypes may be a common feature of IBD-associated oral microbiota.

Although studies addressing *S. mutans* serotypes in the IBD are limited, existing evidence suggests that serotypes k and f – particularly those carrying the collagen-binding protein gene (cnm) – may be more frequently detected in IBD patients than in the general population [34]. Experimental models indicate that such virulent *S. mutans* isolates can aggravate colitis, suggesting a potential link between oral microbiota and intestinal inflammation. Mechanistically, cnm-positive strains may facilitate adhesion to host tissues, promote systemic dissemination and trigger pronounced pro-inflammatory responses, potentially contributing to both oral pathology and the exacerbation of mucosal damage in susceptible IBD patients [34]. Such findings suggest that the systemic inflammatory environment in IBD may create a selective niche for *S. mutans* strains with enhanced immune-evasion capabilities. This points towards a potential pathogenic oral-gut axis, where virulent oral isolates could contribute to the maintenance of intestinal dysbiosis.

The results obtained in the present study on selected aspects of dental caries and IBD, along with literature data, indicate an association between these conditions. However, these findings carry some limitations.

Limitations of the study. The study included a moderately sized sample, which may have affected the precision of the estimates. Furthermore, because participants were assessed at a single point in time, causal relationships could not be determined, and associations identified by logistic regression reflected correlations rather than cause-and-effect relationships. The microbiological analysis was limited to *S. mutans*, which did not reflect the full oral microbiome. Additionally, as all patients were in remission, the results in periods of active disease remain unknown. Therefore, further studies are necessary to better understand the underlying mechanisms. Moreover, increased awareness of the mutual impact of dental caries and IBD among dentists and gastroenterologists may support a more comprehensive diagnostic and therapeutic approach, contributing to improved patient quality of life.

CONCLUSIONS

The findings of the present study suggest that IBD is associated with poorer oral health, including higher caries experience and worse oral hygiene, compared with healthy individuals. Increased numbers of decayed teeth and higher plaque levels were linked to greater odds of IBD, with patients with CD and UC being more likely to present with average or insufficient oral hygiene. Rural residence was associated with worse dental status in UC patients, while *S. mutans* was prevalent among IBD patients, showing a relatively high frequency of serotype k. These observations highlight the importance of targeted oral hygiene education for patients with IBD, particularly those with CD and those living in rural areas. These results also point to a potential role of the oral microbiome in disease-related processes and support further research in this field. Promoting clinical awareness of the link between dental caries and IBD among specialists is essential for developing integrated management strategies that can significantly enhance the long-term quality of life for these patients.

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