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Prevalence of dental caries and periodontal disease in patients with inflammatory bowel disease: a case–control study

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Abstract

Aim: Previous reports suggest a higher incidence of dental caries in patients with inflammatory bowel disease (IBD) and similarities in the immunopathogenesis of IBD and periodontitis. This study assessed the prevalence of periodontal disease and caries in patients with IBD.

Methods: In the present case–control study, 62 patients seeking treatment of IBD and 59 matched healthy controls of a dental practice were clinically examined. Oral soft-tissue alterations, the decayed, missing and filled tooth surface (DMF-S) index, dentine caries, plaque index (PI), bleeding on probing (BOP), probing pocket depth (PPD) and clinical attachment loss (CAL) were evaluated in each patient and in the controls.

Results: Patients with IBD showed a significantly higher number of oral manifestations compared with controls. The DMF-S index showed no significant differences, but there was a significantly higher number of subjects with dentine caries in patients with IBD. The mean PPD in patients with IBD was 2.08 *versus* 2.23 mm in controls ($p = 0.014$). Compared with controls, patients with IBD had more sites with CAL of at least 4 mm (81% *versus* 64% in controls, $p = 0.07$) and 5 mm (63% *versus* 46%, $p = 0.07$), respectively.

Conclusions: The results of this case–control study demonstrate a higher frequency of dentine caries in patients with IBD but the periodontal findings showed no distinct differences between cases and controls.

Key words: case–control study; clinical attachment level; dentine caries; inflammatory bowel disease; periodontal disease

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Inflammatory bowel disease (IBD) is a complex civilization disease and has a prevalence of up to 0.5% in the Northern European community (Probert et al. 1993). The disease preferentially manifests in the second and third decade of live. The severe chronic symptoms have a significant impact on the quality of life. Genetic influences (Hampe et al. 2001, Hugot et al. 2001, Stoll et al. 2004) in combination with environmental factors (Russel et al. 1998, Hampe et al. 2003) are discussed as pathogenic factors. Considerable overlap exists between the two forms of IBD, Crohn's disease (CD) and ulcerative colitis (UC), with regard to pathophysiology and some clinical manifestations. CD (regional enteritis or regional ileitis) is an idiopathic IBD that can affect any part of the alimentary canal, from the mouth to the rectum. It is characterized by a granulomatous chronic inflammatory involvement of the gastrointestinal tract, particularly the terminal ileum. Oral manifestations of CD were first described by Dudeney (1969). Oral soft-tissue lesions associated with CD can precede or occur concomitantly with the intestinal symptoms. Cobblestone architecture of the mucosa, aphthous-like ulcerations, non-specific swellings of the mucosa and lips and lymphadenopathy have been observed (Dudeney 1969, Carr 1974, Snyder & Cawson 1976, Talbot et al. 1984, Scheper & Brand 2002, Harty et al. 2005). The lesions may occur at any time during the disease process and may occur in different forms in the same patient. Less is known about oral manifestations in patients with UC. Pyostomatitis has been described as the main oral complication (Hansen et al. 1983, Chan et al. 1991).

There is only little systematic information about the oral status of patients with IBD. In a few studies with smaller samples, it was reported that CD patients have a higher prevalence of caries than healthy control subjects (Sundh & Hulten 1982, Rooney 1984, Bevenius 1988, Schütz et al. 2003). The higher risk of dental caries has been suggested to be a result of nutritional deficiencies and changes in salivary and microbiologic conditions in the oral cavity (Sundh & Emilson 1989). Most reports on the oral features of CD deal with soft-tissue lesions rather than periodontal findings. A possible association between IBD and periodontal disease was assumed, in particular that IBD patients are more susceptible to periodontal destruction than the general population (Lamster et al. 1978, 1982, van Dyke et al. 1986, Engel et al. 1988, Flemmig et al. 1991, Sigusch 2004).

Patients with IBD were clinically examined and compared against a control population of a dental practice. The aim of this case–control study was to assess the prevalence of periodontal disease and caries in patients with IBD.

Material and Methods

Patient selection

Sixty-two patients treated as in- or outpatients for IBD at the Department of General Internal Medicine, University of Kiel, consented to participate in the study. The diagnosis of either CD or UC was confirmed by previously established clinical, radiological and endoscopic criteria (type of lesions, distribution; Truelove & Pena 1976, Lennard-Jones 1989), and histological findings also had to be confirmative or compatible with this diagnosis.

Fifty-nine healthy patients of a dental practice in Kiel were compared with the study patients. Patients with IBD (cases) and the control group were recruited from the same population (Schleswig Holstein). The control group and patients with IBD were matched referring to age, sex and smoking status (number of years smoked and cigarettes/packs per day). As epidemiologic studies have identified age (Lavstedt et al. 1986), smoking history (Bolin et al. 1986, Lavstedt et al. 1986, Calsina et al. 2002, Tonetti & Claffey 2005) and gender (Papapanou et al. 1989, Reichert et al. 2002) as the main established risk factors for periodontitis as co-variates (Loos et al. 2005), patients with IBD and controls were matched for these criteria. In addition to these factors, poorly controlled diabetes has been described as another established risk factor which has been an exclusion criterion in this study.

For each patient with IBD, a control with the same gender and of the same age ($\pm$ 3 years) was selected. Smoking habits were matched according to the number of smoked cigarettes per day (cig/day): <10, 10–20 and >20 cig/day. For three of the 62 patients with IBD, it was not possible to find controls who matched the selected criteria. All cases and controls were unrelated Caucasians of Northern European ancestry. The experiments were undertaken with the understanding and written consent of each study participant. The study protocol was approved by the Ethics Committee of the University of Kiel.

Clinical examination

Patients of the study group and the control group were interviewed with respect to their general medical status, medical treatment and their dietary habits using a standardized questionnaire (including e.g. dietary incompatibilities, any nutritional changes as the beginning of the disease, frequency of meals).

Among the IBD patients were 46 patients with CD and 16 with UC. Disease activity of IBD was assessed using the CD activity index, (CDAI; Best et al. 1976). Twenty-two patients of 62 (35%) were active (CDAI >150) during the clinical examination (18 with CD, four with UC).

Patients received standard medical treatment including corticosteroids ($n = 20$), immunosupressants ($n = 24$; e.g. azathioprine, methotrexate), aminosalicylate ($n = 39$), anti-TNF ($n = 13$) and antibiotics ($n = 12$) as mono- or combination therapy. None of the patients of the control group received any of the above-mentioned medical treatments.

In patients with IBD, 34 individuals were non-smokers [55%; controls: 29 (49%)], three quit smoking at least a year ago [former smokers, 5%; controls: 6 (10%)] and 25 were smokers [40%; controls: 24 (41%)]. Smokers with IBD (24 with CD, one with UC) smoked an average ($\pm$ SEM) of 0.90 ± 0.38 packs per day for 18.32 ± 8.18 years. Smokers of the control group smoked an average of 0.88 ± 0.50 packs per day for 17.20 ± 9.22 years.

Intra-oral examination

The intra-oral examinations were conducted using a dental chair (with the light attached to the unit) and included the assessment of mucosa lesions (the number of findings for each patient was recorded), decayed, missing and filled tooth surface (DMF-S) index, dentine caries, plaque index (PI), and periodontal measurements [bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment loss (CAL)].

Dental caries assessment (DMF-S index, dentine caries)

Dental caries (including primary and secondary carious lesions) was diagnosed on the basis of a clinical inspection. The DMF-S index (expressed per subject) was determined according to the criteria of the World Health Organization (WHO; Oral health surveys: basic methods, 1997). All permanent teeth were examined (except wisdom teeth) using cotton rolls, plane mouth mirrors and a PCP11 periodontal probe (Hu-Friedy®, Chicago, IL, USA). The assessment of caries was carried out only by visual examination according to the criteria of the WHO. Probing of lesions was deliberately avoided unless plain visual criteria were not sufficient; then, the PCP11 periodontal probe was used to examine the tooth surface carefully.

Unerupted teeth, persistent primary teeth and teeth extracted for orthodontic reasons were excluded from the DMF-S indices. The proportion of decayed, missing or filled tooth surfaces as percentage of all tooth surfaces present in the mouth was calculated. Furthermore, the prevalence of dentine caries (including only primary carious lesions) in the IBD group and patients of the control population was assessed using a dental explorer.

Plaque index

The evaluation of the presence or absence of marginal plaque (O'Leary et al. 1972) was performed at four sites per tooth (disto-buccal, mid-buccal, mesio-buccal and mid-lingual or mid-palatinal) at three teeth (mid-incisor, canine and first molar) in ipsilateral quadrants (upper right and lower right

or upper left and lower left quadrant alternating from one patient to the other) using a dental probe. Results were expressed as percentage of sites with plaque.

Periodontal examination

All periodontal measurements (PPD, BOP, CAL) were assessed at four sites of all teeth in two quadrants (upper right and lower right or upper left and lower left quadrant alternating from one patient to the other): disto-buccal, mid-buccal, mesio-buccal and mid-lingual or mid-palatinal. For each individual, the mean of the individual measurements was calculated and regarded as the representative value for that subject. All measurements were performed with a periodontal probe (PCP 11; Hu-Friedy®) and the readings were recorded to the nearest 1 mm. The PPD was measured from the gingival margin to the base of the probeable pocket. The presence or absence of BOP (as percentage of positive bleeding sites) was simultaneously recorded for each subject. The CAL represented the distance between the cementoenamel junction (CEJ) and the base of the probeable pocket. Only fully erupted teeth with a clearly detectable CEJ were included. In case a restoration extended apically to the CEJ or an abrasion was present at the tooth cervix, the position of the CEJ was estimated by extrapolating the position of the CEJ from the adjacent teeth. If the location of the CEJ could not be determined, the sites were not recorded. Results were recorded as percentage of patients with IBD or controls with at least one site of all sites measured with CAL $\geqslant 4$ and $\geqslant 5$ mm, respectively. Measurements were taken by the same examiner throughout the study for patients with IBD and controls of the dental practice.

Statistical analysis

Statistical analyses were performed with the SPSS software Program (version 11.0). The prevalence of dentine caries and attachment loss were described in contingency tables and analysed with Fisher's exact test and odds ratios (OR) [including 95% confidence intervals (CI)]. Quantitative measurements (number of existing teeth, DMF-S index, plaque and BOP) were not normally distributed (Kolmogorov–Smirnov test). Therefore, these data were described by median and quartile values (inter-quartile ranges) in tables. Differences between cases (patients with IBD) and controls were evaluated by the Mann–Whitney *U*-test. All statistical tests were carried out at a 5% level of significance. Stepwise logistic regression analysis was performed in order to identify relevant variables that allowed to distinguish between individuals with IBD or healthy controls.

Results

Clinical examination

The group with IBD included 38 females (controls: 35) and 24 males (controls: 24). The mean age was 38.4 ± 10.3 years for patients with IBD and 38.2 ± 10.0 years in the control group.

Dietary habits were different between patients and controls. Because of the limitations imposed by the systemic illness, almost half of all patients with IBD ($27/62 = 44\%$) reported that they had been eating more frequently but smaller amounts of food as the beginning of their disease. There was a high frequency of highly fermentable carbohydrate snack food consumption between meals. Because of an existing intolerance, 47 of 62 patients (76%) had to avoid certain groups of food. In contrast, only seven people ($7/59 = 12\%$) of the control population mentioned dietary incompatibilities. Food that was avoided included e.g. fresh fruit or fruit juices (cases: 13/47, controls: 4/7), milk products (cases: 18/47, controls: 1/7), spices (cases: 12/47, controls: 1/7), some vegetables like onions or beans (cases: 15/47, controls: 0), food rich in fat (cases: 12/47, controls: 3/7) and sugar/sweets (cases: 6/47, controls: 0).

Intra-oral examination

The examination of the oral mucosa revealed significant differences between patients with IBD and controls (Mann–Whitney *U*-test: $p < 0.001$). In 29 patients with IBD, 56 oral lesions were found (0.9 ± 1.264) compared with 11 patients with 14 oral lesions in the control group (0.24 ± 0.536). Thirteen patients had one oral lesion (controls: 8), in 10 patients two lesions were found (controls: 3) and three to six oral lesions were diagnosed in six patients with IBD (controls: none). The most common oral findings were focal areas of mucobuccal hyperplasia or oedema ($n = 15$), swellings of the gingiva ($n = 17$), ulcera ($n = 5$), aphthae ($n = 6$), candidiasis ($n = 5$), lichen planus ($n = 3$), leucoplakia ($n = 2$) and labial rhagades ($n = 3$). Eleven patients with oral lesions presented with IBD disease activity [CDAI > 150 (Best et al. 1976); $11/29 = 38\%$, nine with CD, two with UC] and 18 patients with oral lesions showed no disease activity at the time of examination ($18/29 = 62\%$).

Dental caries assessment (DMF-S index, dentine caries), PI

There were no significant differences in the number of existing teeth in patients with IBD and the controls (Table 1). Table 1 also represents the results for the DMF-S index and the PI. For the DMF-S index, there were no significant differences between cases and controls but compared with the controls, patients with IBD showed significantly more plaque ($p = 0.032$). The prevalence of dentine caries in patients with IBD and the control population is shown in Table 2. There was a significantly higher prevalence of dentine caries in patients with IBD ($p = 0.033$) compared with controls with an OR for dentine caries of 2.37 (95% CI: 1.0–5.8).

Periodontal examination (PPD, BOP, CAL)

The results of the PPD and BOP measurements are represented in Table 1. Values for PPD were significantly higher in controls ($p = 0.014$) when compared with patients with IBD. Values for BOP were similar for cases and controls. Results for the prevalence of CAL $\geqslant 4$ and $\geqslant 5$ mm are represented in Table 2. The comparison showed that 81% of the patients with IBD had at least one site with CAL $\geqslant 4$ mm with an OR of 2.29 (controls: 64%). Sixty-three percent of the patients with IBD had at least one site with CAL $\geqslant 5$ mm with an OR of 2.0 (controls: 46%). Even though the respective values for CAL in IBD patients were distinctly higher than those for the controls, these differences failed to reach statistical significance ($p = 0.07$).

Regression analysis

A multiple logistic stepwise regression model for IBD as the dependent variable regarding the number of existing teeth, DMF-S index, dentine caries, plaque, BOP, PPD and CAL $\geqslant 4$ and $\geqslant 5$ mm

as independent variables confirmed that IBD could be predicted from some relevant variables. Table 3 summarizes significant factors: PPD (risk ratio 0.245), PI (risk ratio 1.027), dentine caries (risk ratio 2.82) and CAL $\geqslant$5 mm (risk ratio 2.47). In this model, we assumed a linear effect of PPD and PI on IBD. Seventy percent of the cases (43) and 70% of the controls (41; Table 3) could correctly be predicted either as cases or controls using the logistic regression model.

Table 1. Number of existing teeth and DMF-S index, plaque index (as percentage of sites with plaque), bleeding on probing (as percentage of positive bleeding sites) and probing pocket depth (in mm) in patients with IBD and a control population (mean values with standard deviation)

	n	Mean ± SD	Min	Max	Percentiles			*p*-value*
					25	50 (median)	75	
Number of existing teeth								
Patients with IBD	62	24.8 ± 5.0	2	28	24	27	28	0.148
Controls	59	26.1 ± 2.8	17	28	26	27	28	
DMF-S index								
Patients with IBD	62	54.1 ± 31.6	1	128	32.8	46.0	66.3	0.212
Controls	59	46.5 ± 26.5	3	106	27.0	44.0	61.0	
Plaque index								
Patients with IBD	62	42.3 ± 28.7	0	100	16.7	33.3	62.5	0.032
Controls	59	29.9 ± 21.4	0	100	16.7	25.0	41.7	
Bleeding on probing								
Patients with IBD	62	23.4 ± 20.1	0	100	8.3	16.7	30.8	0.958
Controls	59	20.8 ± 13.5	0	55	12.5	16.7	29.2	
Probing pocket depth								
Patients with IBD	62	2.22 ± 0.57	1.5	4.9	1.82	2.08	2.34	0.014
Controls	59	2.29 ± 0.33	1.5	3.3	2.09	2.23	2.48	

*Mann—Whitney *U*-test.
min, minimum values; max, maximum values; median values including 25/75 percentiles.
DMF-S, decayed, missing and filled tooth surface; IBD, inflammatory bowel disease.

Table 2. Prevalence of dentine caries (including only primary carious lesions) in patients with IBD and a control population

	Pairwise comparison			
	n (%)	significance (*p*)	odds ratio	95% CI
Dentine caries				
Patients with IBD	25 (40%)	0.033	2.37	1.00–5.80
Controls	13 (22%)			
CAL $\geqslant$4 mm				
Patients with IBD	50 (81%)	0.07	2.29	0.94–5.79
Controls	38 (64%)			
CAL $\geqslant$5 mm				
Patients with IBD	39 (63%)	0.07	2.00	0.91–4.43
Controls	27 (46%)			

Percentage of patients with IBD and a control population with at least one site of all sites measured with clinical attachment loss (CAL) $\geqslant$4 and $\geqslant$5 mm, respectively. For comparison, Fisher's exact test and odds ratios (OR) were used (95% confidence interval CI).
IBD, inflammatory bowel disease.

Discussion

The cariological evaluation revealed that the DMF-S index showed no significant differences between patients with IBD and the control population (patients of a dental practice), whereas in patients with IBD, the prevalence of dentine caries was significantly higher (40% *versus* 22% in the controls).

These results are in accordance with observations in a pilot study including a small sample of 15 patients, suggesting a higher frequency of dental decay in patients with CD compared with healthy control subjects (Bevenius 1988). In two other reports, both the DMF-S index and the risk of baseline caries were increased (Sundh & Hulten 1982, Rooney 1984). The higher risk for dental caries has been attributed to nutritional deficiencies and changes in salivary and microbiologic conditions in the oral cavity. Studies that evaluated the nutritional status of patients with CD found an increased intake of refined carbohydrates relative to normal controls (Martini & Brandes 1976, Kasper & Sommer 1978, Schütz et al. 2003). Especially, patients with an exacerbation of CD eat sugary food because it is more digestible and causes less gastrointestinal symptoms than a fat-containing diet (Järnerot et al. 1983). Sundh and Emilson (1989) examined salivary and microbial conditions and the dental status in 21 patients with CD for 3 years. The caries experience in these patients was higher than that observed in an age- and sex-matched control group. All patients had a normal saliva flow rate and buffer capacity but the number of salivary mutans streptococci and lactobacilli was high both at baseline and after 3 years. These findings and a frequent intake of refined carbohydrates were plausible reasons for the higher caries activity and the rather high incidence of recurrent caries in patients with CD. These results are in contrast with the findings of Halme et al. (1993), who reported that of 53 patients, those with active CD had more ''dental infection foci'' than patients with inactive disease, but the values of DMF and baseline caries did not show significant differences between either activity stages or sexes. Meurman et al. (1994) investigated dental, gingival and salivary aspects during an exacerbation of CD in two groups of patients (with active or inactive disease). Oral examination did not reveal any statistically significant differences in gingival or dental indices between the groups, but patients with active disease tended to have higher scores of gingivitis than patients with inactive disease.

In the present study, 44% (27/62) of the patients with IBD reported that as the beginning of their disease, they had to eat more frequently and take smaller amounts of food to avoid gastrointestinal problems. As plaque scores in the IBD group were significantly higher compared with controls, we assume that this change in eating habits might be the reason for the significantly higher prevalence of dentine caries in the IBD patient group. Additionally, 76% (47/62) of the patients with IBD had to avoid certain groups of food; among them were 18 patients (18/47) who had to avoid milk products. This may be of

Table 3. Multiple stepwise logistic regression analysis with patients with IBD (cases) and controls as dependent variables and probing pocket depth, plaque index, dentine caries and clinical attachment loss (CAL) as selected independent variables

Selected variables (from stepwise logistic regression)	Coefficient	*p*-value	Risk ratio	95% (CI)
Intercept	1.476	–	–	–
Probing pocket depth (mm)	– 1.406	0.012	0.245	0.082–0.730
Plaque index (%)	0.026	0.006	1.027	1.008–1.046
Dentine caries	1.038	0.018	2.822	1.191–6.688
CAL $\geqslant$ 5mm	0.904	0.046	2.469	1.016–5.996

	Predicted (from logistic regression model)	
	controls	cases (IBD)
Controls	41	18
Cases	19	43

IBD, inflammatory bowel disease; CI, confidence intervals.

importance as IBD patients often have osteoporosis, which is considered as a risk factor for periodontitis (Inagaki et al. 2001, Wactawski-Wende 2001, Yoshihara et al. 2004).

In our study, patients with IBD showed a significantly higher number of oral manifestations compared with controls (56 lesions in 29 patients with IBD/14 lesions in 11 controls). Therefore, oral hygiene efforts could have been hampered, which could have contributed to the high caries incidence. Furthermore, several studies have reported a relationship between exacerbations of intestinal disease activity and the incidence of oral lesions (Greenstein et al. 1976, Sundh & Hulten 1982, Halme et al. 1993). Our study revealed that 11 patients with oral lesions presented with IBD disease activity (11/29 = 38%) but 18 patients with oral lesions showed no disease activity at the time of examination (18/29 = 62%). These results are in accordance with studies by Lisciandrano et al. (1996) and Tremaine (1998), who found no correlation between clinical IBD disease activity and frequency of oral lesions.

The results of the periodontal examination revealed similar PPD in patients with IBD (2.08 mm) and the control population (2.23 mm), as the slight difference between IBD patients and controls, even though statistically significant, should not be regarded as clinically significant. Values for BOP were slightly higher in the IBD group, which is in agreement with the higher plaque scores found in this population. Determination of the percentage of subjects with at least one site of all sites measured with CAL $\geqslant$4 and $\geqslant$5 mm, respectively, demonstrated distinctly higher numbers for the IBD group, but failing statistical significance when compared with the control group. Our results showed that in patients with IBD parameters, indicating periodontal disease were not distinctly different from the control group. Thus, susceptibility to periodontal disease did not appear to be elevated in patients with IBD. Overall, these results for periodontal conditions are in correspondence with results of an earlier cross-sectional study (Flemmig et al. 1991) reporting the prevalence and severity of periodontal disease in patients with IBD. The periodontal status of 107 patients was determined and compared with the assessment of Oral Health of United States Adults. IBD patients revealed an 11.9% higher prevalence, but 0.6 mm lower severity of periodontal disease. Similar to our results, the mean probing depth was 2.4 mm in patients with CD and 2.3 mm in patients with UC. The authors concluded that periodontal disease in IBD subjects was more generalized but less severe than in the general population and that the magnitude of the observed differences would suggest no clinical implications for the management of periodontal disease in patients with IBD. Very few reports exist on IBD patients with concomitant periodontitis. Lamster et al. (1978) reported a case of a 28-year-old white male with CD and rapidly progressive periodontitis. They found this patient to have enhanced polymorphonuclear neutrophil (PMN) phagocytosis. Van Dyke et al. (1986) examined 20 patients with IBD, 10 patients presented with periodontal disease and 10 without. They used control groups that were age and sex matched and that were matched to the type and severity of periodontal disease in the IBD group. The results of their study showed that the degree of inflammation of the gingiva of the affected IBD patients was greater than that of age- and sex-matched periodontal disease patients. Further studies of the host response of these patients using a chemotaxis assay (in the presence of 5% patient serum) revealed a serum-mediated defect in neutrophil chemotaxis in all 10 patients with periodontal disease, whereas neutrophil phagocytosis was normal. Unusual microorganisms colonizing the oral cavity of IBD patients were considered to potentially play a role in the pathogenesis of the disease as infectious agents or modifiers of the host response. However, severe periodontitis in IBD patients appeared to be a relatively rare problem. The systemic immunologic and inflammatory alterations of IBD rather seemed to exacerbate or accelerate periodontal breakdown than to be causative. Nevertheless, in our study stepwise logistic regression analysis showed that PPD, PI, dentine caries and CAL $\geqslant$5 mm revealed a significant association with IBD.

In conclusion, our results showed that patients with IBD had a higher prevalence of dentine caries compared with a healthy control group. Altered dietary habits and malabsorption may probably be the main cause for this condition. IBD patients also showed a higher but not significantly different prevalence in CAL, even though they did not present with more clinical signs of gingival inflammation. However, based on the limited number of study participants, definite conclusions should be drawn with caution. This is a pilot study that will give rise to a targeted prospective investigation in a follow-up study.

Emphasis should be focused on the preventive dental care of these patients. Strict oral hygiene should be recommended, and the regular use of fluoride treatment for prevention of dental caries appears to be justified.

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Clinical Relevance

Scientific rationale for the study: Previous investigations have indicated similarities in the immunopathogenesis of IBD and periodontitis; however, there are no data from case–control studies. The aim of this study was to assess the prevalence of periodontal disease and caries in patients with IBD.

Principal findings: Compared with a control population, matched for age, gender and smoking habits, IBD patients did not differ with respect to their periodontal conditions, but presented with a significantly higher number of teeth with dentine caries.

Practical implications: The results of this study suggest that patients with IBD do not require special periodontal treatment but that it might be recommended to intensify preventive dental care for the prevention of dental caries.