LETTER TO THE EDITOR

ORAL MANIFESTATIONS IN INFLAMMATORY BOWEL DISEASES: WHAT DENTISTS SHOULD KNOW

MANIFESTAÇÕES ORAIS NAS DOENÇAS INFLAMATÓRIAS INTESTINAIS: O QUE O CIRURGIÃO-DENTISTA DEVE SABER

Danielle Nobre Lopes[1], Noemia Pereira de Oliveira[1], Rafaela Elvira Rozza-de-Menezes[1]

DEAR EDITOR,

Inflammatory bowel diseases (IBDs) are chronic diseases that affect the gastrointestinal system from the mouth to the anal region, mainly consisting of Crohn's disease (CD) and ulcerative colitis (UC) (1). They show alternating periods of remission and activity; their common symptoms include diarrhea, abdominal pain, fever, nausea, vomiting, and weight loss (2).

UC predominantly manifests itself as bloody diarrhea in the colon (between the cecum and the rectum) (3). It causes higher morbidity and mortality than CD, which shows a more heterogeneous and systemic clinical picture, including malaise, anorexia, and fever, and can affect any segment of the digestive tract (4). CD causes transmural inflammation that spans from the mucosa to the serosa. It mainly occurs in the ileum and colon (5). Complications such as intestinal obstructions, perianal fistulas, and abscesses occur more often in CD than in UC, resulting in a greater need for surgical intervention (3,6).

The incidence of IBDs has increased in industrialized countries together with changes in eating habits and lifestyle (7). They occur more prevalently in Western countries, especially in northern Europe and North America (7). They show no predilection for sex, and the onset of their symptoms usually occurs from age 15 to 25 years in CD and from age 25 to 35 years in UC (8). The scarce epidemiological data in Brazil stems from the lack of compulsory notification (9). In a study carried out in São Paulo (2012-2015), Gasparini *et al*. (10) observed a 13.3/100,000 annual incidence (6.14 for CD and 7.16 for UC) and a 52.6/100,000 prevalence. Most patients were women (59.7%) with a mean age of onset of 42.7±16.2 years for CD and 47.9±16.6 years for UC.

The multifactorial etiopathogenesis of IBDs involves immune responses, genetic predisposition, environmental factors, and gut microbiota (11). A central hypothesis proposes an anomalous interaction between the microbiota and local immune responses that cause dysbiosis and activate dysfunctional immune cells, such as macrophages and autoreactive T lymphocytes (12).

In addition to intestinal involvement, IBDs, especially CD, show extraintestinal manifestations that may involve the musculoskeletal system, eyes, skin, oral cavity, and biliary tract, significantly contributing to morbidity and increasing mortality, which varies from 6 to 47% (13,14).

Oral manifestations of inflammatory bowel diseases

The first cases of oral manifestations of IBDs were described in 1969 in two individuals with CD (15). Since then, varying methodologies have found their heterogeneous prevalence (16). This variability also results from the difficulty in distinguishing whether the lesions result from the course of the disease, adverse effects of treatment drugs, or from nutritional deficiencies due to intestinal malabsorption (16).

Oral lesions occur more often in CD than in UC, although some studies have found no significant differences (16-18). They can emerge with intestinal manifestations or precede them in up to 60% of cases (16). In about 30% of them, they persist even during remission, especially in children (16,18). Their histological classification as specific and non-specific follows the presence of granulomas of epithelioid macrophages, lymphocytes,

[1] Department of Pathology, Medical School, Universidade Federal Fluminense, Niterói, RJ, Brazil

How to cite this article: Lopes DN, Oliveira NP, Rozza-de-Menezes. Oral manifestations in inflammatory bowel diseases: what dentists should know. Nav Dent J. 2025;52(2):1-4.

Received: 08/13/2025
Accepted: 09/10/2025

DOI: https://doi.org/10.22491/1983-7550-52-2-C1

and multinucleated giant cells. These poorly defined granulomas have no central caseous necrosis and only occur in CD (17,19).

Specific lesions (less common than non-specific ones) include lip edema, deep linear ulcerations, tissue growth similar to fibrous hyperplasia, linear edema, granulomatous mucosa (cobblestoning), and granular and hyperplastic mucogingivitis with or without ulcerations (1,20). Cobblestoning mainly occurs in the posterior mucosa, showing multiple normochromic and corrugated papules that are grouped into plaques and interfering with speech and swallowing (16). Linear ulcerations show hyperplastic, firm, or firm-elastic borders, mainly occurring in the lower vestibule and the retromolar trigone (16). Lip edema occurs often in CD and may involve one or both lips. It usually causes no pain, firmly resists palpation, and may be associated with vertical fissures (21,22).

Among the non-specific manifestations observed in both diseases, the following stand out: recurrent aphthous stomatitis, angular cheilitis, lichenoid reactions, perioral dermatitis, vegetative pyostomatitis, and periodontal disease (23). Vegetating pyostomatitis, considered the oral counterpart of vegetating pyodermatitis, constitutes a marker of UC activity, which is more common than in CD (1,16,24). Clinically, it presents erythematous, elevated lesions with multiple pustules and superficial erosions, which, when ruptured, cause folds and fissures in a "snail's track" (16,24). The most affected sites refer to the vestibular gingiva, jugal and labial mucosa, hard and soft palates, and the body of the tongue (16).

Individuals with IBDs show an increased periodontal disease prevalence, a risk two-four times higher than in the general population (16). This finding may be related to changes in these patients' oral microbiota and exacerbated inflammatory response (25). In contrast, periodontal disease can influence gut microbiota and compromise epithelial barrier function, contributing to the pathogenesis of IBDs (25). The meta-analysis by Papageorgiou *et al.* (26) has shown a higher risk of severe periodontitis in patients with UC than in those with CD. Patients with UC also have worse oral health indicators, suggesting a unique response to plaque among subgroups (16).

Oral manifestations related to the use of drugs to treat inflammatory bowel diseases

The literature describes oral lesions in patients with IBDs associated with the use of several drugs (1,20). Aminosalicylates such as sulfasalazine and mesalazine may induce adverse reactions in the oral mucosa. Sulfasalazine is related to lichenoid lesions, whereas mesalazine can cause hematological alterations, such as leukopenia, thrombocytopenia, and aplastic anemia, which predispose patients to hemorrhages and oral infections (20,27). In case of lichenoid lesions and taste alterations attributed to these drugs, their replacement is recommended (20).

The prolonged use of corticosteroids (common in severe cases of IBDs) is associated with multiple orofacial adverse effects, resulting from supraphysiologic doses: acne, moon face, petechiae, and ecchymosis due to vascular fragility (28). They also favor opportunistic infections, such as pseudomembranous and atrophic candidiasis.

Thiopurines such as azathioprine also increase the risk of opportunistic infections (candidiasis and herpes simplex virus infections), taste disorders (ageusia, hypogeusia, dysgeusia) and malignant lesions, such as lymphomas, affecting the oral mucosa (20). Methotrexate, a stomatotoxic drug, can induce ulcers and mucositis, the risk of which is proportional to doses, duration of use, advanced age, and drug interactions (29). The absence of folic acid supplementation potentiates these effects by blocking its synthesis by methotrexate (30). As with other immunosuppressive drugs, it also increases patients' predisposition to oral infections (31).

Anti-TNF-α biologic therapies (infliximab, adalimumab) have revolutionized the management of IBDs. However, they have numerous adverse effects, including lichenoid lesions, erythema multiforme, and opportunistic infections (20,30). Erythema multiforme may be associated with herpes simplex virus infections or medication use. On the other hand, Stevens-Johnson syndrome (which is usually triggered by drugs) requires the immediate interruption of treatment and hospitalization, unlike erythema multiforme, which can be controlled on an outpatient basis with systemic and topical corticosteroids (32,33).

The management of oral manifestations depends on the clinical picture as it aims to control pain, heal lesions, and prevent secondary infections. Candidiasis requires topical or systemic antifungal therapy without the need to suspend the underlying medication (20). On the other hand, herpetic ulcers should be treated with acyclovir, and antiviral prophylaxis may be considered in recurrent cases. Pharmacological prescription may be undertaken by the dental surgeon; however, it is essencial that the medical team responsible for the management of IBD

be informed of the therapeutic regimen implemented for the associated oral manifestations (34).

CONCLUSION

Knowledge of the oral alterations of IBDs, which may precede gastrointestinal symptoms, gives dentists a fundamental role in early diagnosis. Moreover, knowledge of the drugs used to treat IBDs and their potential adverse effects on the oral mucosa is essential for appropriate therapeutic approaches. Thus, integrating dental surgeons and gastroenterologists is essential for the effective management of intestinal disease and its oral repercussions.

The authors declare no conflict of interest.

Corresponding author:
Danielle Nobre Lopes.
Hospital Universitário Antônio Pedro, Universidade Federal Fluminense - Av. Marquês do Paraná, 303, 4o andar, sala 18. Centro, Niterói, RJ, Brazil.
ZIP Code: 24033-900.
Email: daninobrelopes@yahoo.com.br.

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