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ORAL MUCOSAL CHANGES IN COELIAC DISEASE, DIABETES MELLITUS, AND INFLAMMATORY BOWEL DISEASES – A SYSTEMATIC REVIEW

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ABSTRACT

Background: The oral cavity is sometimes referred to as a "diagnostic window" of systemic health, as even subtle systemic disturbances can manifest as changes in the oral mucosa. Coeliac disease, diabetes mellitus, and inflammatory bowel diseases (IBD) are chronic conditions that frequently present with oral mucosal lesions, which may precede or accompany the classical symptoms of these diseases.

Aim: To systematically review and compare the oral mucosal changes reported in coeliac disease, diabetes mellitus, and IBD. The most common clinical manifestations are characterized, potential pathophysiological mechanisms are discussed, and the relevance of oral findings for early recognition and monitoring of these systemic diseases is evaluated.

Materials and Methods: A systematic literature search was conducted in PubMed and Google Scholar using combinations of English keywords (including "oral mucosa," "oral manifestations," "coeliac disease," "diabetes mellitus," "inflammatory bowel disease," "Crohn's disease," "ulcerative colitis," "aphthous ulcers," "xerostomia," "oral microbiome," and "oral–gut axis"). A total of 37 relevant peer-reviewed articles (clinical studies and reviews) were identified and analyzed.

Results: The most frequently described oral manifestations in these conditions include recurrent aphthous stomatitis, inflammatory and atrophic changes of the tongue (such as geographic tongue and atrophic glossitis), angular cheilitis, xerostomia, and recurrent opportunistic infections (especially oral candidosis). In coeliac disease, oral lesions (aphthae, glossitis, cheilitis) and dental enamel defects often occur, sometimes even years before diagnosis, and tend to improve after the introduction of a strict gluten-free diet. Diabetes is associated with salivary gland dysfunction leading to dry mouth, elevated salivary glucose, and immune dysfunction – factors that contribute to candidosis, poor wound healing, burning mouth sensations, and an increased incidence of oral ulcers. IBD (Crohn's disease and ulcerative colitis) can produce a broad spectrum of oral changes: Crohn's disease in particular is characterized by specific granulomatous lesions (such as persistent lip swelling, mucosal "cobblestoning," and deep linear ulcers) as well as nonspecific lesions (recurrent aphthae, pyostomatitis vegetans, glossitis, cheilitis). Oral manifestations of IBD are more common in Crohn's disease and in pediatric patients and can precede intestinal symptoms or correlate with intestinal disease activity.

Conclusions: Oral mucosal changes represent an important extraintestinal component of coeliac disease, diabetes, and IBD. They can serve as early warning signs of these disorders or indicators of disease activity and control. Recognition of characteristic oral lesions by dentists and physicians is crucial, as it can expedite diagnosis and prompt timely management (e.g. initiation of a gluten-free diet in coeliac disease or improved glycemic control in diabetes). Regular oral examinations should be an integral part of the care of patients with these conditions. Interdisciplinary collaboration – especially between dentists, gastroenterologists, and diabetologists – is essential for early detection, comprehensive monitoring, and improved patient outcomes.

KEYWORDS

Coeliac Disease, Diabetes Mellitus, Inflammatory Bowel Disease, Oral Mucosa, Oral Manifestations

CITATION

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Introduction

The oral cavity constitutes the first segment of the gastrointestinal tract and, at the same time, one of the most accessible parts of the body for clinical examination, where early manifestations of numerous systemic diseases may appear. Owing to continuous exposure to microbiological, mechanical, and chemical factors, as well as unique morphological and immunological properties, the oral mucosa functions as a dynamic protective barrier, helping maintain local and systemic homeostasis. Its rich vascularization, abundant innervation, high metabolic activity of epithelial cells, and the presence of a well-developed mucosa-associated lymphoid tissue (MALT) mean that even subtle systemic disturbances can manifest as clinical changes in the oral cavity. For this reason, the oral mucosa is sometimes referred to as a "diagnostic window," allowing detection of pathological processes before full expression of the organ-specific symptoms typical of the underlying disease [9-10,22-23,27,29]. The literature emphasizes that oral manifestations can precede the classic symptoms of systemic diseases, occur during phases of low disease activity, and sometimes constitute the only clinically detectable sign of an evolving disorder. These changes encompass a wide spectrum of clinical presentations, such as recurrent erosions and ulcers, erythema and edema of the mucosa, atrophic changes of the tongue, angular cheilitis, xerostomia, recurrent opportunistic infections (especially candidiasis), as well as pain and burning of the mucosa in the absence of obvious macroscopic lesions. In practice, particular importance is attached to chronic, recurrent, or treatment-resistant mucosal lesions – their presence should prompt a thorough differential diagnosis that considers hematologic, metabolic, immunologic, and gastroenterologic causes [9-10,22-23,27,29].

Among systemic diseases in which oral changes are reported especially often and can have significant clinical relevance are coeliac disease, diabetes mellitus, and inflammatory bowel diseases (IBD). These conditions are chronic, with complex pathogenesis and a substantial involvement of immunological, metabolic, and microbiological mechanisms that can influence the state and function of the oral mucosa and salivary glands [1-7,11-14,17-18,27-29]

Coeliac disease

Coeliac disease as a systemic disorder

Coeliac disease is a chronic, immune-mediated inflammatory disease of the small intestine triggered by gluten in genetically predisposed individuals, most commonly carriers of the HLA-DQ2 and/or HLA-DQ8 haplotypes. The pathogenesis of the disease involves an abnormal immune response to gluten peptides (gliadin), leading to villous atrophy, crypt hyperplasia, and chronic inflammation of the small-intestinal mucosa. The current understanding of coeliac disease, however, extends beyond the classic view of it as a condition limited to the gastrointestinal tract – increasing emphasis is placed on its systemic nature and heterogeneous clinical picture with numerous extraintestinal manifestations [11-14,28-31]. Besides intestinal symptoms (e.g. chronic diarrhea, abdominal pain, bloating, weight loss), an important group consists of symptoms resulting from deficiencies (e.g. iron-deficiency anemia, bone demineralization disorders) as well as neurological manifestations, fertility disorders, and changes in the oral cavity. Importantly, oral and dental lesions can precede the diagnosis of coeliac disease by several or even a dozen years; in atypical or paucisymptomatic cases, they are often the main diagnostic clue, giving them special clinical importance [11-14,28-31

Oral mucosal and tongue changes in coeliac disease

A wide spectrum of oral mucosal changes has been described in patients with coeliac disease. The most frequently observed are recurrent aphthous stomatitis, inflammatory and atrophic changes of the tongue (including geographic tongue and atrophic glossitis), angular cheilitis, and subjective symptoms such as burning, pain, and a feeling of dryness in the mouth [11-14,28-31]. The pathogenesis of these changes is multifactorial – it includes both consequences of malabsorption syndrome and the chronic autoimmune inflammatory process, disturbances in mucosal immune responses, as well as alterations in the gut–oral axis (oral microbiome) [11-14,28-29,35].

Recurrent aphthous stomatitis (RAS) is one of the most frequently reported oral manifestations in coeliac patients. In some patients, aphthae occur many years before the diagnosis and are the only extraintestinal symptom of the disease [11-14,28-29]. These ulcers are usually minor or mixed aphthae, located on the mobile mucosal areas of the oral cavity (lips, cheeks, ventral tongue) and have a recurrent course [11-14,28-29]. Comparative studies have shown a significantly higher frequency of aphthae in patients with coeliac disease compared to the general population – in both children and adults [11-13,28-31]. Moreover, in some patients a

marked decrease in aphthae recurrence was observed after the introduction of a strict gluten-free diet, confirming their association with the activity of the underlying disease [11-13,28-30].

An important group of manifestations involves changes of the tongue, including geographic tongue (lingua geographica) and atrophic glossitis. Geographic tongue is characterized by focal atrophy of filiform papillae, leading to irregular, erythematous patches with a lighter peripheral rim. Atrophic glossitis presents as a smooth tongue with an intense red, shiny appearance, often accompanied by pain or burning [11-13,28,30-31]. These changes are primarily associated with iron, folate, and vitamin B_12 deficiencies resulting from malabsorption, as well as chronic inflammation and immunologic disturbances [11-13,28,30]. Clinical studies clearly indicate a significantly higher incidence of both geographic tongue and papillary atrophy in coeliac patients compared to healthy individuals [11-13,28-29].

Angular cheilitis (angular stomatitis) in coeliac disease usually presents as recurrent, painful fissures and erosions at the corners of the mouth, often with crusting or secondary Candida superinfection. This lesion is linked to iron and B-vitamin deficiencies, as well as reduced local mucosal immunity [11-13,28,30]. In some patients, angular cheilitis may be one of the first signs of nutritional deficiencies resulting from villous atrophy of the intestines [11-13,28].

It is noteworthy that in coeliac disease there is often a concurrent occurrence of oral mucosal lesions and dental/enamel abnormalities. Enamel defects (enamel hypoplasia, hypomineralization) and delayed tooth eruption can coexist with recurrent aphthae, geographic tongue, and angular cheilitis, creating a characteristic clinical picture – particularly evident in pediatric patients [11-13,28-30]. Although enamel defects do not directly involve the oral mucosa, their co-occurrence with other mucosal changes significantly increases the likelihood of coeliac disease and should prompt further diagnostic evaluation [11-13,28-30].

Involvement of the salivary glands in coeliac disease

Salivary gland involvement is an important, though less frequently discussed, aspect of the extraintestinal manifestation of coeliac disease. In contrast to changes resulting from malabsorption syndrome, salivary gland involvement in coeliac disease may reflect direct immunological mechanisms leading to local inflammatory changes and impaired protective functions of the oral mucosa [31]. Histopathological studies have shown that some coeliac patients exhibit focal lymphocytic sialadenitis (focal lymphocytic inflammation of minor salivary glands). These changes can coexist with a subjective feeling of dry mouth even with preserved saliva secretion from the major salivary glands, indicating that the dominant problem is not always generalized hyposalivation [31]. The discrepancy between severe dryness symptoms (sicca) and normal sialometry results may be due not only to impaired local mucosal lubrication but also to qualitative alterations of saliva (protein composition, viscosity, buffering capacity) and modifications of the local oral microenvironment in the course of chronic inflammation [31].

From a differential diagnostic standpoint, it is important to distinguish salivary gland involvement in coeliac disease from Sjögren's syndrome, in which generalized dysfunction of salivary and lacrimal glands predominates along with measurable hyposalivation. In coeliac disease, dryness symptoms are more often localized and may not correlate with a significant reduction in salivary output, which can lead to underestimation of the problem in routine clinical practice [31]. It should be remembered that coeliac disease frequently co-occurs with other autoimmune diseases, which can complicate the clinical picture and, in justified cases, necessitate further diagnostic work-up [11-14,28-29,31].

The clinical consequences of salivary gland involvement include not only reduced patient quality of life but also an increased susceptibility to oral mucosal inflammation and infections (including candidosis), especially in the presence of coexisting deficiencies and atrophic changes of the tongue [11-13,28,30-31]. Therefore, in patients with suspected or confirmed coeliac disease, it is advisable to include targeted history-taking about dry mouth symptoms, burning sensations, and difficulty eating dry foods, and in selected cases, to evaluate salivary gland function [31].

Significance of oral changes in the diagnosis and monitoring of coeliac disease

Given the frequently asymptomatic or atypical course of coeliac disease, the presence of characteristic oral changes has important diagnostic significance. Recurrent aphthae with an atypical or treatment-refractory course, geographic or atrophic tongue, angular cheilitis, and coexisting enamel defects and delayed tooth eruption – especially if accompanied by anemia or other signs of malabsorption – should prompt physicians and dentists to include coeliac disease in the differential diagnosis [11-14,28-31]. Available data indicate that in some cases it is the dentist who first identifies oral abnormalities and initiates further serological and

gastroenterological diagnostics [11-14,28-31]. Early diagnosis and prompt implementation of a gluten-free diet are crucial for preventing long-term complications of chronic villous atrophy [11-14,28-29].

Oral changes may also reflect the degree of disease control and adherence to a gluten-free diet. In some patients, a resolution or decreased frequency of aphthae and an improvement in the condition of the tongue and mouth corners are observed after eliminating gluten from the diet. Conversely, persistence or recurrence of these lesions may suggest insufficient disease control, unintentional gluten intake, the presence of additional nutritional deficiencies, or coexisting diseases. Therefore, oral examination should be an integral part of both the diagnostic process and long-term monitoring of patients with coeliac disease [11-13,28-30].

Diabetes mellitus

Diabetes mellitus as a systemic disorder

Diabetes mellitus is a chronic metabolic disease characterized by persistent hyperglycemia resulting from impaired insulin secretion, impaired insulin action, or a combination of both mechanisms [1-3,26,34]. Regardless of the type of diabetes (1 or 2), long-term hyperglycemia leads to the development of numerous organ complications, including microangiopathy, macroangiopathy, neuropathy, and immune dysfunction. These changes result in increased susceptibility to infections, delayed wound healing, and an abnormal inflammatory response [1-3,26,34]. Diabetes is now regarded as a systemic disease in which metabolic disturbances affect the functioning of many tissues and organs, including structures of the oral cavity. In the course of diabetes, numerous changes involving the oral mucosa, periodontium, and salivary glands are observed – these may represent both direct manifestations of the disease and secondary consequences of chronic hyperglycemia and its treatment [1-3,24-26,33-34]. In particular, uncontrolled diabetes leads to quantitative and qualitative disturbances in saliva secretion, which promote oral dryness, microbiome destabilization, and an increased risk of fungal infections and chronic mucosal inflammation [1-3,24-26,33-34].

The bidirectional relationship between diabetes and periodontal disease has also been emphasized in the literature. Chronic periodontitis can worsen insulin resistance and hinder glycemic control, whereas uncontrolled diabetes promotes the development of periodontitis and a more severe course of that disease [1-3,25-26,34]. Although the main focus of this review is on oral mucosal changes, recognizing this interrelationship is important for an overall interpretation of the oral clinical picture in diabetic patients. Inflammatory processes in the periodontal tissues and mucosa often coexist and exacerbate one another [1-3,25-26,34].

Pathogenetic mechanisms of oral mucosal changes in diabetes

The pathogenesis of oral mucosal changes in diabetic patients is multifactorial and results from the chronic impact of hyperglycemia and its accompanying metabolic, vascular, immunological, and neuronal disturbances. Key mechanisms include microangiopathy, immune cell dysfunction, neuropathy (including autonomic neuropathy), increased oxidative stress, and the deposition of advanced glycation end products (AGEs) in tissues [1-3,26,34].

Chronic hyperglycemia promotes the formation of AGEs, which accumulate in vascular basement membranes and the extracellular matrix. AGEs impair collagen structure and function, increase its susceptibility to degradation, and exacerbate oxidative stress. Furthermore, by interacting with RAGE receptors, they activate numerous pro-inflammatory pathways, leading to enhanced cytokine production and maintenance of chronic inflammation [1-3,26,34]. In the oral mucosa, this results in impaired epithelial regeneration, increased vulnerability to injury, a tendency toward chronic inflammation, and delayed healing of erosions and ulcers – particularly in patients with poorly controlled diabetes [1-3,25-26,33-34].

Diabetic microangiopathy also plays an important role. Thickening of the basement membrane, narrowing of small vessel lumens, and impaired perfusion lead to chronic tissue hypoxia and limited delivery of nutrients. This perpetuates inflammation, accelerates degenerative processes, and significantly impairs the reparative capacity of the oral mucosa [1-3,26,34].

An additional factor in pathogenesis is immune dysfunction. Diabetes is associated with impaired neutrophil chemotaxis, adhesion, and phagocytosis, as well as macrophage dysfunction, resulting in reduced ability to eliminate microorganisms and a predisposition to opportunistic infections [1-3,25-26,33]. Particularly important is the increased glucose concentration in saliva and gingival crevicular fluid, which creates a conducive environment for the growth of Candida yeasts, especially in the presence of xerostomia and poor metabolic control [1-3,25-26,33].

Diabetic neuropathy (including autonomic neuropathy) affects both salivary gland function and sensory perception in the oral cavity. Damage to autonomic innervation leads to dysregulation of salivary secretion, while damage to sensory fibers may underlie pain, paresthesia, and burning sensations in the mucosa – symptoms characteristic of conditions like BMS [1-3,22,25-26,33-34].

Salivary gland dysfunction in diabetes

Salivary gland dysfunction is one of the key manifestations of diabetes and has a critical impact on the state of the oral mucosa. Diabetic patients exhibit both quantitative changes (reduced unstimulated and stimulated salivary flow) and qualitative changes (altered composition and protective properties of saliva). Importantly, the subjective feeling of dry mouth does not always correlate with objectively reduced salivary output [1-3,24-26,33-34].

The principal underlying factors in these disturbances are salivary gland microangiopathy and autonomic neuropathy, which lead to impaired regulation of salivary secretion [1-3,25-26,34]. Dehydration, electrolyte imbalances, and polypharmacy also contribute, including antihypertensives, diuretics, and centrally-acting medications that can exacerbate dryness [1-3,25-26,34]. Qualitative changes in saliva in people with diabetes include elevated glucose levels, reduced buffering capacity, and altered microbiological composition, which encourage fungal infections and chronic mucosal inflammation [1-3,25-26,33-34].

The clinical consequences of salivary gland dysfunction in diabetes include diminished quality of life (difficulties with chewing, swallowing, and speaking), chronic halitosis, increased caries risk, and greater susceptibility to mucosal infections. Persistent xerostomia may also intensify pain symptoms, predispose to mechanical injury, and impair healing of post-procedural wounds [1-3,25-26,33-34].

Oral mucosal changes in diabetes

Patients with diabetes exhibit a broad spectrum of oral changes involving both the mucosa and related structures. The most commonly described are xerostomia, candidosis, angular cheilitis, atrophic glossitis, painful and burning sensations of the mucosa, and changes in tongue appearance (fissured tongue, coated tongue, or atrophic tongue) [1-3,24-26,32-34].

Xerostomia is among the most commonly reported complaints in diabetic patients and significantly affects their quality of life. It can result from decreased salivary secretion, altered saliva composition, as well as from dehydration and fluid–electrolyte imbalances [1-3,24-26,33-34]. The pathogenesis of xerostomia in diabetes is strongly linked to salivary gland microangiopathy, autonomic neuropathy, and medications used for co-morbid conditions [1-3,25-26,34].

Oral candidosis occurs significantly more often in people with diabetes than in the general population. Predisposing factors include hyperglycemia, elevated salivary glucose levels, immune dysfunction, and xerostomia [1-3,25-26,33]. Clinically, candidosis may present in pseudomembranous, erythematous, or chronic hyperplastic forms, and it often involves the corners of the mouth (angular cheilitis). Numerous studies have demonstrated a correlation between glycemic control and frequency of candidal infections – the worse the diabetes control, the more frequent and difficult-to-treat the Candida infections [1-3,25-26,33].

Angular cheilitis in diabetic patients is often chronic and recurrent. Its pathogenesis is associated with recurrent Candida infections, secondary bacterial infections, xerostomia, mechanical factors (e.g. excessive salivation or malocclusion leading to maceration of the lip corners), and vitamin deficiencies – which in diabetes may be exacerbated by metabolic disturbances [1-3,25,33].

Tongue changes – including papillary atrophy with a smooth, erythematous dorsal tongue, fissured tongue, and coated tongue – are common, though nonspecific, manifestations of diabetes. They can result from iron and B-vitamin deficiencies, microcirculatory disturbances, chronic inflammation, xerostomia, and alterations in the oral microbiome [1-3,22-26,33-34].

Burning mouth syndrome (BMS) is a significant clinical problem in diabetic patients. It is thought to be related to diabetic neuropathy, vascular disturbances, xerostomia, and coexisting candidosis [1-3,22,25-26,33-34]. BMS symptoms (burning sensations of the tongue, palate, lips, often without visible changes) significantly reduce quality of life and may persist despite an unremarkable oral examination.

Diabetic patients also more frequently exhibit erosions and ulcerations of the oral mucosa, as well as delayed healing of traumatic or post-surgical lesions. The mechanisms include microangiopathy, immune dysfunction, abnormal collagen synthesis, and AGE deposition in tissues, which impair regenerative processes [1-3,25-26,33-34].

Differences in the oral presentation of type 1 versus type 2 diabetes

While the overall spectrum of oral manifestations is similar in both major types of diabetes, their severity and specific clinical presentation may differ depending on age at onset, disease duration, and the presence of co-morbidities. Type 1 diabetes often features abrupt glycemic swings, greater susceptibility to infections, and markedly delayed wound healing [1-3,25-26,34]. In type 2 diabetes, by contrast, factors such as obesity, hypertension, dyslipidemia, and polypharmacy play a significant role – these contribute to pronounced xerostomia and chronic inflammatory changes of the oral mucosa [24-26,33-34].

Significance of oral changes in the diagnosis and monitoring of diabetes

Oral mucosal changes may serve as important indicators of undiagnosed or poorly controlled diabetes. Population studies have shown that among patients presenting to dental clinics with complaints such as xerostomia, recurrent candidosis, chronic ulcers, angular cheilitis, burning mouth, or tongue changes – a significant percentage have undiagnosed diabetes or prediabetes [2,24-26,32-34]. These findings indicate that the dental office can function as an initial site for identifying carbohydrate metabolism disorders, and that dentists can initiate further internal medicine or diabetologic diagnostics [2,24-26,33-34].

Moreover, the severity of oral changes often correlates with the degree of metabolic control. Improved glycemic control may lead to a reduced frequency of candidal infections, relief of xerostomia, and reduction of pain symptoms (including BMS) [1-3,25-26,33-34]. This underscores the importance of interdisciplinary collaboration between dentists and diabetologists, and of treating the oral cavity as an integral part of patient assessment in diabetes [1-3,25-26,34]. Regular dental check-ups for diabetic patients enable not only the management of local symptoms, but also provide indirect evaluation of the effectiveness of diabetes treatment and an opportunity for patient education on maintaining oral health.

Inflammatory bowel diseases (IBD)

Characterization of IBD as systemic diseases

Inflammatory bowel diseases (IBD) primarily comprise Crohn's disease (CD) and ulcerative colitis (UC). CD can involve any segment of the gastrointestinal tract, and its histopathology typically features discontinuous (segmental) and transmural inflammation, which favors the development of fistulas, abscesses, and strictures [4-7,17,19]. UC is generally limited to the colon and rectum, with continuous, diffuse mucosal and submucosal involvement, manifesting with symptoms such as abdominal pain, urgency, and bloody diarrhea [4,7,17,19,27]. A common element in the pathogenesis of both conditions is chronic, relapsing activation of immune responses to microbial stimuli in the context of a compromised intestinal barrier – a process modulated by genetic and environmental factors [4-7,17,19,27].

IBD are systemic diseases in which the clinical picture extends beyond the gastrointestinal tract. Extraintestinal manifestations may involve, for example, the musculoskeletal system (arthritis), skin (erythema nodosum, pyoderma gangrenosum), eyes (uveitis), biliary tract (primary sclerosing cholangitis), and the oral cavity [5-8,16-19,36-37]. In clinical practice it is noted that extraintestinal symptoms often correlate with intestinal disease activity, but they can also precede gastrointestinal symptoms or persist independently of them [5-7,16-19,36-37]. Oral changes should thus be considered not as an isolated dental problem, but as a potential component of the systemic immune dysregulation and mucosal barrier dysfunction characteristic of IBD [5-7,16-19,36-37].

The oral–gut axis, dysbiosis, and pathogenesis of oral changes in IBD

In recent years, the concept of the "oral–gut axis" has developed significantly, describing functional and immunological connections between the oral and intestinal microbiota and mucosal immunity [20-21,37]. The oral cavity is the first segment of the gastrointestinal tract and is inhabited by a complex, highly organized microbiota forming biofilms on the mucosa and tooth surfaces, while in constant contact with the local mucosal immune system [20-21,37]. From an immunological perspective, both the oral cavity and the gut are components of the MALT system – activation of immune responses in one segment can influence responses in another through shared cytokine pathways, migration of effector cells, and changes in mucosal tolerance regulation [20-21,37].

A key phenomenon in the oral–gut axis is "oralization" of the gut microbiota – the ectopic presence of microorganisms typical of the oral cavity within the intestine (both in the lumen and attached to the mucosal surface) under conditions that favor their survival and colonization [20-21,35,37]. Under normal conditions, microbes from the mouth are largely eliminated by the gastrointestinal barrier, peristalsis, and immune

mechanisms. In IBD, however – in the setting of epithelial barrier damage, an altered gut microenvironment (e.g. different oxygen and nutrient availability), immune dysregulation, and chronic inflammation – conditions may arise that facilitate the survival and colonization of oral-origin bacteria in the intestine [20-21,37]. From the perspective of IBD pathogenesis, this is significant because microorganisms tolerated in the oral niche may act as potent immunological stimuli in the gut, triggering an inflammatory response in an environment of disrupted mucosal tolerance to the microbiota [4-7,20-21,37].

Dysbiosis in IBD is also noteworthy in that it is neither static nor necessarily purely primary. In many pathogenic models, dysbiosis is part of a vicious cycle: chronic inflammation promotes changes in the composition and function of the microbiota, while the altered microbiota perpetuates and intensifies the inflammatory response [35,37]. The oral–gut axis broadens this model by indicating that the oral cavity can serve as an additional reservoir of microorganisms and immune signals modulating the course of intestinal disease, while conversely intestinal inflammation can secondarily influence the oral microenvironment (e.g. saliva composition, susceptibility to opportunistic infections, severity of local inflammatory changes) [20-21,36-37].

Moreover, changes in the oral cavity and salivary glands have also been noted in other gastrointestinal disorders, such as irritable bowel syndrome (IBS) and microscopic colitis, suggesting that oral–gut connections may extend beyond classic IBD [15,20-21].

Oral manifestations in IBD – clinical presentation and frequency

In clinical practice and literature, a division of oral lesions in IBD is often proposed into specific lesions (highly suggestive of CD, related to the granulomatous process) and nonspecific lesions, which may accompany both CD and UC but also occur in other chronic diseases and deficiency states [5-7,17-19,36-37]. This classification is useful for organization but, importantly, for diagnosis: specific lesions – especially when confirmed by appropriate histopathology – can greatly facilitate and expedite the diagnosis of IBD in a patient without a previously established intestinal disease [5-7,16-19,36-37].

Specific lesions, particularly characteristic of CD (falling under the spectrum of so-called orofacial Crohn's disease), include: chronic, usually painless swelling of the lips (macrocheilia), often with radiating fissures; deep, linear ulcerations of the vestibular and buccal mucosa; a "cobblestone" appearance of the mucosa – irregularly thickened, folded mucosa with a pebbled texture and fissures; mucogingivitis – diffuse, granulomatous gingival inflammation with swelling, redness, and a tendency to bleed; as well as tag-like or polypoid mucosal lesions [5-7,17,19,36-37]. Histologically, these lesions often show non-caseating granulomas, similar to those found in the intestines in Crohn's disease, confirming their association with the disease process [5-7,17,19]. Clinically, it is important that orofacial manifestations can precede full-blown intestinal symptoms by months or even years. In such cases, the oral cavity becomes the "first point of contact" with the disease, and the role of the dentist extends beyond symptomatic treatment to initiating etiological diagnostics [5-7,16-19,36-37].

Nonspecific lesions most often include recurrent aphthous stomatitis (usually minor aphthae, less often major or multiple), angular cheilitis, nonspecific mucosal erosions and ulcerations, erythematous and atrophic tongue changes, as well as pain and burning sensations of the mucosa [5-7,17-19,36-37]. A particular entity among the nonspecific lesions is pyostomatitis vegetans – a rare but clinically very important manifestation strongly associated with IBD (especially ulcerative colitis). It presents with multiple tiny pustules on an erythematous mucosal background, which can coalesce into the characteristic "snail track" appearance (mucosa covered with mucopurulent crusts resembling a snail's trail). Although most often described in UC, pyostomatitis vegetans can also occur in CD [5-7,17-19,36-37]. In contrast to granulomatous lesions, pyostomatitis vegetans usually does not show granulomas on histology. Its importance lies mainly in the fact that the presence of such lesions in a patient should prompt an active search for IBD, even if intestinal disease has not yet been diagnosed [5-7,17-19,36-37].

The reported prevalence of oral lesions in IBD varies – cross-sectional studies range from a few percent to several tens of percent, and in many reports the rate is higher in CD than in UC [5-7,16-19,36-37]. The pediatric population deserves special emphasis, as oral manifestations are described more frequently in children and can be a significant part of the disease presentation – even when intestinal symptoms are mild or atypical [8,16,36]. In this group, systematic oral examination is important not only for managing local symptoms but also for reducing diagnostic delay in IBD, which can translate into better prognosis and lower risk of complications [16,36].

Relationship of oral lesions to disease activity, nutritional status, and immune factors

The relationship between intestinal inflammation activity and oral lesions in IBD is complex. Some studies have found that oral symptoms (especially aphthae, erythematous-edematous changes, lip swelling, and pyostomatitis vegetans) appear more often during IBD flares and may correlate with higher intestinal disease activity [5-7,17-19,36-37]. At the same time, there are reports of oral lesions persisting despite intestinal remission or recurring independently of it. This suggests that in some patients orofacial manifestations can also be modulated by local factors (e.g. periodontal status, oral microbiome composition), ongoing immune dysregulation, and environmental factors [5-7,17-19,36-37]. Consequently, oral lesions should not be interpreted solely as a simple marker of current intestinal activity, but rather as a phenotype of extraintestinal manifestations with multifactorial determination [5-7,16-19,36-37].

The clinical oral picture is also influenced by the patient's nutritional status and IBD-related deficiencies. In Crohn's disease and ulcerative colitis, protein–calorie malnutrition and micronutrient deficiencies are common due to dietary restrictions, chronic diarrhea, bleeding, and malabsorption. Deficiencies of iron, vitamin B_12, or folic acid can contribute to atrophic mucosal changes, atrophic glossitis, angular cheilitis, recurrent erosions and aphthae, as well as a general susceptibility of the mucosa to injury [5-7,17-19,36-37]. Clinically, this means that some lesions labeled "nonspecific" are secondary to anemia or micronutrient deficits – their persistence despite local therapy should prompt an evaluation of the patient's overall condition and basic laboratory parameters, rather than limiting management to dental treatment alone [5-7,17-19,36-37]. Of course, deficiencies do not preclude the simultaneous presence of specific lesions, and multifactoriality can lead to overlapping clinical pictures in the oral cavity of the same patient.

Increasingly, consideration is also given to periodontal status and chronic gingival inflammation as a potential element of the broader oral–gut network. Patients with IBD have been reported to have a higher prevalence of advanced periodontitis and need for dental interventions, which may result from both immunological mechanisms (a tendency toward periodontal inflammation in IBD) and behavioral, dietary factors, as well as the impact of systemic therapy [36-37]. In practice, this means that evaluating an IBD patient requires not only identifying mucosal lesions but also recognizing and managing periodontal inflammation. Neglected gingivitis or periodontitis can modify the local inflammatory milieu and oral microbiota composition and – indirectly – influence the interpretation of salivary biomarkers [36-37].

Salivary gland involvement and xerostomia in IBD

Salivary gland-related symptoms occupy an important place in the oral presentation of IBD: feelings of dryness (sicca), subjective xerostomia, and reported difficulties with swallowing, speaking, or pronounced halitosis [36-37]. The mechanisms behind these phenomena are multifactorial – including systemic inflammatory activity, possible immune disturbances within MALT, the effects of treatment (including medications that promote dryness), and the patient's hydration and nutritional status [7,17-18,36-37]. Immunologic abnormalities in saliva have also been described (e.g. altered levels of select proinflammatory cytokines), which may reflect systemic immune activation [36-37].

It is also worth noting some less frequently discussed but clinically relevant observations indicating that active Crohn's disease can involve granulomatous inflammation of the minor salivary glands, as well as the formation of salivary duct fistulas and recurrent cheek abscesses, which require differential diagnosis and histopathological verification [5-7,17-19]. Although such occurrences are less common than, for instance, aphthae or angular cheilitis, they are significant because they may represent part of the granulomatous lesion spectrum and warrant a different diagnostic and therapeutic approach [5-7,17-19]. In clinical practice, the salivary gland component of IBD is often underappreciated – yet recognizing symptoms of dryness and reduced salivary function is important, as xerostomia increases the risk of opportunistic infections (e.g. candidosis), exacerbates pain, and impedes healing of minor mucosal injuries. All of these can worsen the clinical course of oral lesions in IBD patients [36-37].

Impact of systemic treatment on the oral cavity in IBD

The clinical oral picture in IBD patients is significantly modified by systemic therapies used. Immunosuppressive drugs (e.g. azathioprine, methotrexate), glucocorticoids, and biologic therapies (anti-cytokine antibodies, etc.) – although aimed at controlling intestinal inflammation – can increase susceptibility to opportunistic oral infections (especially fungal infections) and alter the character and dynamics of mucosal inflammatory lesions [7,17-18,36-37]. From a dental perspective, it is critical to differentiate whether observed oral lesions are an extraintestinal manifestation of IBD or a consequence of immunosuppression. This

distinction implies different management – from local treatments and professional oral hygiene measures to potential adjustments of systemic therapy in collaboration with a gastroenterologist [7,17-18,36-37].

Immunosuppression in IBD can clinically manifest as recurrent or chronic oral candidosis (erythematous or pseudomembranous forms, and angular cheilitis), increased pain and burning sensations, as well as a tendency for slower healing of minor mucosal injuries [7,17-18,36-37]. In cases of recurrent fungal or bacterial infections, it is important to consider contributing factors such as xerostomia, poor oral hygiene, advanced periodontitis, or other systemic conditions. Local treatment without simultaneous control of these risk factors may not yield lasting results [36-37].

Drug reactions affecting the oral mucosa during IBD therapy have also been described – for example, erosive lesions, ulcerations or erythema appearing after the use of aminosalicylates or other medications used in IBD. In such cases, establishing a temporal relationship between initiation of a given drug and the onset of lesions is key, as is observing whether lesions resolve or improve upon altering the therapy [7,17-18,36-37].

Regardless of etiology, chronic, atypical ulcerations or lesions that persist despite treatment should prompt more in-depth diagnostics, including – in justified cases – biopsy and histopathological examination of the affected tissues [5-7,17-19,36-37]. This is especially important in patients on long-term immunosuppression, who require a high index of suspicion for potentially premalignant changes or opportunistic infections masquerading as inflammatory lesions [7,17-18,36-37].

From the perspective of dental practice, the above observations necessitate regular follow-ups and an interdisciplinary approach. If oral lesions are suspected to be related to IBD activity or medication side effects, close cooperation with a gastroenterologist is indicated to assess current disease activity and, if necessary, optimize systemic therapy. At the same time, local risk factors (improving oral hygiene, treating periodontal inflammation, stimulating saliva flow, etc.) should always be addressed, since proper local management can prevent lesion exacerbations and improve patient comfort.

Salivary biomarkers in IBD

In the context of growing interest in non-invasive methods of monitoring IBD activity, saliva is being intensely studied as a diagnostic material that can reflect both local inflammatory processes in the oral cavity and the systemic component of chronic inflammation [36-37]. The main goal of research on salivary biomarkers is to evaluate their usefulness as complementary tools alongside standard methods (such as fecal calprotectin, serum inflammatory markers, or endoscopic evaluation) [36-37]. It must be emphasized, however, that salivary biomarkers – promising as they may be – require cautious interpretation, because the levels of many mediators in saliva can be modulated by periodontal status, opportunistic oral infections, local mucosal disease activity, as well as by collection techniques and pre-analytical factors [36-37].

The most extensively analyzed salivary biomarker is calprotectin (S100A8/A9) – a protein well-established in IBD diagnostics as a marker of intestinal inflammation (routinely measured in stool). Attempts to adapt this concept to saliva have yielded inconclusive results. On one hand, elevated salivary calprotectin levels have been reported in IBD patients compared to healthy controls, with suggestions of correlation with disease activity [36-37]. On the other hand, clinical studies have indicated that salivary calprotectin may not correlate significantly with fecal calprotectin or clinical activity indices (e.g. the Harvey–Bradshaw Index in CD or partial Mayo score in UC). This calls into question its utility as a standalone biomarker for monitoring IBD in routine practice [36-37]. Methodologically, this discrepancy is meaningful: saliva, being in direct contact with oral tissues, may reflect local inflammatory processes more than the true activity of intestinal inflammation, and the presence of coexisting oral lesions (e.g. gingivitis, aphthae, erosions) can confound result interpretation [36-37]. Therefore, even proponents of salivary biomarkers stress the need to standardize collection conditions and to account for oral health status and local factors in parallel [36-37].

Apart from calprotectin, salivary proinflammatory cytokines (e.g. TNF-α, IL-1β, IL-6), chemokines, and select acute-phase proteins have also been studied. Some studies have noted elevated levels in IBD patients, suggesting that systemic inflammatory signals penetrate the oral environment [36-37]. However, high variability of results and susceptibility to confounders (especially periodontal status) currently limit the ability to unequivocally recommend these measurements for routine use [36-37]. Pediatric data are particularly pertinent – since less invasive markers than endoscopy are sought – but even in this population salivary biomarkers are to be considered as potential adjuncts and research tools rather than replacements for standard diagnostic pathways [16,36-37].

In light of current data, salivary biomarkers, including calprotectin, remain promising but still experimental tools. Their interpretation must consider oral health status and potential confounders, and their clinical use should not replace proven methods for assessing IBD activity [36-37].

Clinical significance of oral changes in IBD and diagnostic management

Oral lesions in IBD patients have clinical significance on several levels: they can serve as early warning signs of disease, components of active extraintestinal manifestations, consequences of nutritional deficiencies or therapy complications, and as factors worsening quality of life (pain, difficulty eating and speaking, chronic discomfort) [5-7,16-19,36-37]. The highest diagnostic value is attributed to lesions with a granulomatous phenotype typical of Crohn's disease (e.g. lip swelling with fissures, cobblestoning, deep linear ulcerations, mucogingivitis, tag-like lesions). In the appropriate clinical context, these lesions narrow the differential diagnosis and expedite referral of the patient to a gastroenterologist [5-7,16-19,36-37]. Likewise, the presence of pyostomatitis vegetans – although rare – has a high "diagnostic power" for IBD and should prompt comprehensive gastroenterological investigation (particularly for ulcerative colitis), bearing in mind it can also occur in Crohn's disease [5-7,17-19,36-37].

For nonspecific lesions (aphthae, angular cheilitis, nonspecific erosions/ulcers, tongue changes), a holistic approach is key. The mere presence of aphthae does not confirm IBD, but a chronic or recurrent course, resistance to local treatment, coexisting systemic symptoms (anemia, weight loss, low-grade fevers) or gastrointestinal complaints should prompt further diagnostic exploration [5-7,16-19,36-37]. This is especially important in children and adolescents, where IBD can present atypically and oral manifestations appear early and can influence the timing of diagnosis [8,16,36].

Because oral lesions in IBD have a multifactorial etiology, an interdisciplinary approach is essential. Collaboration between dentist and gastroenterologist (and if needed, dermatologist and pathologist) allows proper differentiation of lesions – determining whether they are a direct manifestation of IBD, a result of deficiencies, a side effect of therapy, or an independent oral pathology – and selection of optimal management [5-7,17-19,36-37]. From a dental practice perspective, particular diagnostic vigilance is required for chronic, atypical ulcers, granulomatous changes, persistent soft tissue swellings, and refractory xerostomia. These symptoms may necessitate broader systemic diagnostics, including evaluation for IBD or complications of its treatment [5-7,16-19,36-37]. At the same time, controlling oral inflammation, professional dental cleaning, treating periodontal disease, and managing opportunistic infections remain important for improving patient comfort and reducing symptom burden in the chronic course of IBD [36-37].

To systematize the clinical approach to oral lesions in IBD, a hierarchical strategy is proposed that takes into account both the specificity of lesions for a given disease and probable pathogenetic mechanisms. In practice, a useful classification is: (1) specific lesions with a granulomatous phenotype – most strongly suggestive of Crohn's disease (the so-called orofacial Crohn's disease); (2) lesions strongly associated with IBD but not necessarily granulomatous – the key entity in this group is pyostomatitis vegetans (most often linked to UC, though also reported in CD); (3) nonspecific or secondary lesions, which may result from nutritional deficiencies, chronic inflammation, dysbiosis, opportunistic infections, or medication side effects [5-7,16-19,36-37]. This schema facilitates practical differentiation of lesions with high predictive value for IBD from those common in the general population that may, however, be exacerbated in patients with IBD [5-7,17-19,36-37].

In the group of lesions specific to CD, particularly important are chronic lip swelling with fissures, a "cobblestone" mucosal appearance, deep linear vestibular ulcers, and mucogingivitis. Finding these should direct differential diagnosis towards granulomatous diseases (primarily CD) and conditions that clinically mimic orofacial Crohn's disease [5-7,17-19,36-37]. The most important conditions to consider in the differential diagnosis include sarcoidosis, tuberculosis, granulomatous cheilitis of other causes (e.g. Melkersson–Rosenthal syndrome), hypersensitivity reactions (e.g. to toothpaste additives), as well as infectious (e.g. granulomatous deep fungal infection) and drug-related lesions [5-7,17-19]. Therefore, in cases of atypical, chronic swellings or ulcers – especially if resistant to local therapy – a biopsy is recommended to look for non-caseating granulomas on histopathology. Depending on the clinical picture and histopathology results, targeted additional tests may be indicated to exclude specific infections or systemic diseases [5-7,17-19,36-37].

Biopsy also plays a crucial role when chronic ulcers or erosions persist, particularly in patients on long-term immunosuppressants who have a higher risk of opportunistic infections, drug reactions, or lesions requiring further investigation. In clinical practice, indications for mucosal biopsy in IBD patients include: (1)

ulcers persisting despite local treatment and elimination of trauma; (2) lesions with atypical morphology (indurated, infiltrated borders, irregular shape, spontaneous bleeding); (3) lesions recurring in the same site multiple times; (4) suspicion of pyostomatitis vegetans or a granulomatous process; (5) situations where it is necessary to distinguish IBD manifestations from treatment complications (e.g. fungal infections, drug-induced ulcers) or from independent mucosal pathology (e.g. neoplasm) [5-7,17-19,36-37]. Such an approach is valuable not only diagnostically but also organizationally – it enables more rapid referral of the patient to a gastroenterologist and initiation of systemic diagnostics in cases where intestinal symptoms are mild or ambiguous [5-7,16-19,36-37].

Conclusions

Oral mucosal changes are an important, though still underutilized, component of the clinical picture of many systemic diseases. An analysis of the literature on coeliac disease, diabetes, and IBD clearly indicates that the oral cavity is not merely a site of local pathologies, but an integral part of systemic immunological, metabolic, and inflammatory processes [9] [10] [22] [23] [27] [29]. Mucosal manifestations can precede the diagnosis of the underlying disease, correlate with its activity, or persist independently of organ symptoms, giving them special diagnostic and monitoring value [5] [6] [7] [11] [12] [13] [14] [16] [17] [18] [19] [36] [37].

In coeliac disease, oral changes include primarily recurrent aphthous stomatitis, atrophic and inflammatory tongue lesions, angular cheilitis, enamel defects, and subjective complaints of oral burning and dryness [11] [12] [13] [14] [28] [29] [30] [31]. Their pathogenesis is multifactorial and encompasses consequences of malabsorption as well as direct immunological mechanisms. Of particular clinical significance is salivary gland involvement in coeliac disease, which can manifest as sicca symptoms despite preserved salivary flow – distinguishing this from Sjögren's syndrome and highlighting the role of qualitative saliva changes and the local mucosal microenvironment [31]. Resolution of some lesions after introducing a gluten-free diet confirms their relationship with disease activity and points to the potential of the oral cavity as an area for monitoring treatment efficacy [11] [12] [13] [28] [29] [30].

In diabetes, the oral clinical picture is a consequence of chronic hyperglycemia, microangiopathy, neuropathy, and disturbances in immunity and saliva composition. The most frequently described changes include xerostomia, candidosis, angular cheilitis, atrophic tongue changes, painful and burning mucosa, as well as delayed wound healing [1] [2] [3] [24] [25] [26] [33] [34]. An essential aspect is the bidirectional relationship between diabetes and oral inflammation (particularly periodontal diseases), which underscores the need for an interdisciplinary therapeutic approach. Clinical data show that characteristic oral changes can be among the first signals of undiagnosed or uncontrolled diabetes, giving the dentist an important role in the early detection of metabolic disorders [2] [24] [25] [26] [32] [33] [34].

Inflammatory bowel diseases are characterized by an especially broad spectrum of oral manifestations – encompassing both specific lesions (especially in Crohn's disease) and nonspecific lesions common to many chronic diseases [5] [6] [7] [17] [18] [19] [36] [37]. The clinical picture can include chronic lip swelling, linear ulcers, a "cobblestone" mucosa, granulomatous gingivitis, as well as recurrent aphthae, angular cheilitis, and atrophic tongue changes. These manifestations are more frequent in Crohn's disease than in ulcerative colitis, with particularly high frequency observed in children. Oral lesions can precede IBD diagnosis, accompany active intestinal disease, or persist independently of it [8] [16] [36]. In recent years, the significance of the oral–gut axis and the role of dysbiosis and mucosal immune disturbances in the pathogenesis of these lesions have been highlighted. This perspective allows the oral cavity to be seen as part of a systemic inflammatory process, rather than merely a site of secondary symptoms [4] [20] [21] [35] [37].

Increasing interest has been directed toward the potential of salivary biomarkers in assessing IBD activity. Salivary calprotectin and select proinflammatory cytokines are being studied as non-invasive indicators of inflammation that could complement classical diagnostic and monitoring methods [18] [36] [37]. At the same time, caution in interpreting results is emphasized due to the influence of local oral inflammatory changes and the lack of full standardization of assay methods. At the present stage, salivary biomarkers should be considered auxiliary tools rather than replacements for standard tests [16] [36] [37].

Examining the oral mucosal changes in coeliac disease, diabetes, and IBD reveals common pathogenetic mechanisms, such as chronic inflammation, immune dysregulation, mucosal barrier dysfunction, and dysbiosis. At the same time, it highlights important differences in clinical presentation that are relevant for differential diagnosis. In clinical practice, a thorough oral examination should be an integral part of evaluating patients with suspected systemic diseases and those already under treatment for such conditions. Collaboration between the dentist and gastroenterologist, diabetologist, or primary care physician can contribute to earlier disease diagnosis, better monitoring of its course, and improved patient quality of life [9] [10] [22] [23] [27] [29] [36] [37].

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