GASTROINTESTINAL DISORDERS AND ORAL HEALTH: SURGICAL CONSIDERATIONS, ORAL MANIFESTATIONS, AND DENTAL MANAGEMENT. A NARRATIVE REVIEW

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ABSTRACT

Gastrointestinal disorders may affect oral health through inflammatory, immune-mediated, microbial, metabolic, nutritional, and treatment-related mechanisms. This narrative review examines the relationship between gastrointestinal disease and oral manifestations, with particular emphasis on clinically relevant implications for dental and oral surgical management. Inflammatory bowel disease may be associated with recurrent aphthous lesions, mucosal changes, periodontal involvement, and apical periodontitis, whereas gastroesophageal reflux disease is primarily linked to dental erosion. Celiac disease may present with enamel defects, aphthous stomatitis, and other manifestations related to malabsorption, while chronic liver disease may influence periodontal health, mucosal integrity, bleeding risk, and postoperative healing. Bariatric surgery and liver transplantation introduce additional considerations related to nutritional deficiencies, reflux, immune status, and infection susceptibility. Emerging evidence also supports a role for oral-gut microbial interactions involving *Helicobacter pylori*, *Fusobacterium nucleatum*, and periodontal pathogens. Recognition of these associations may facilitate earlier identification of systemic disease and improve treatment planning. Individualized dental care and interdisciplinary collaboration are particularly important when invasive procedures are considered in patients with gastrointestinal or hepatobiliary disorders.

Keywords: gastrointestinal disorders, oral manifestations, oral-gut axis, dental management, oral surgery, microbiome.

1. INTRODUCTION

Gastrointestinal disorders comprise a heterogeneous group of inflammatory, immune-mediated, infectious, metabolic, hepatobiliary, and functional conditions whose clinical consequences may extend well beyond the digestive tract. Among these, inflammatory bowel disease has received particular attention because oral lesions may accompany intestinal activity, precede gastrointestinal symptoms, or persist independently of bowel manifestations. Crohn's disease and ulcerative colitis have been associated with recurrent aphthous ulceration, mucosal swelling, cobblestoning, mucosal tags, pyostomatitis vegetans, periodontal involvement, and other inflammatory changes that may provide clinically relevant clues to systemic disease [1–4]. Beyond mucosal lesions, the relationship between inflammatory bowel disease and periodontal or periapical pathology suggests that chronic oral inflammation and intestinal inflammation may share overlapping immunological and microbial pathways [5,6]. Increasing evidence therefore supports a bidirectional oral–gut axis in which dysbiosis, epithelial barrier dysfunction, and altered host immune responses may contribute to disease expression at both sites [7].

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Other gastrointestinal conditions produce distinct oral phenotypes. Gastroesophageal reflux disease is strongly associated with intrinsic dental erosion, particularly on surfaces repeatedly exposed to gastric acid, making oral examination potentially useful in recognizing otherwise underestimated reflux-related damage [8,9]. In celiac disease, recurrent aphthous stomatitis, developmental enamel defects, glossitis, and other oral findings may reflect immune dysregulation, malabsorption, and nutritional deficiencies, with some abnormalities being especially relevant in pediatric populations [10–12]. Chronic liver disease also has important oral implications, including xerostomia, mucosal changes, periodontal disease, altered healing, and increased bleeding susceptibility, while hepatitis C infection may further influence oral inflammatory and immune-mediated disorders [13,14].

The relationship between gastrointestinal disease and oral health becomes particularly relevant in surgically treated patients. Bariatric procedures may alter nutritional status, salivary composition, reflux patterns, and dietary behavior, thereby influencing dental erosion, caries risk, and periodontal health [15,16].

Oral microbial reservoirs may also have gastrointestinal significance. Periodontal treatment has been associated with improved eradication of gastric *Helicobacter pylori*, while recent studies have explored the possible bidirectional relationship between oral colonization and gastric infection [17,18]. Similarly, oral microbial dysbiosis has been investigated in upper gastrointestinal disorders and colorectal carcinogenesis, with particular interest in organisms such as *Fusobacterium nucleatum* and other periodontal pathogens [19–22].

In cirrhosis, thrombocytopenia and coagulation abnormalities may complicate invasive dental procedures, although appropriate patient selection and local hemostatic measures can permit minor oral surgery with acceptable safety [23,24]. The principal biological and clinical pathways linking gastrointestinal disorders with oral health are summarized in Figure 1, which provides a conceptual framework for the mechanisms and clinical implications discussed throughout this review.

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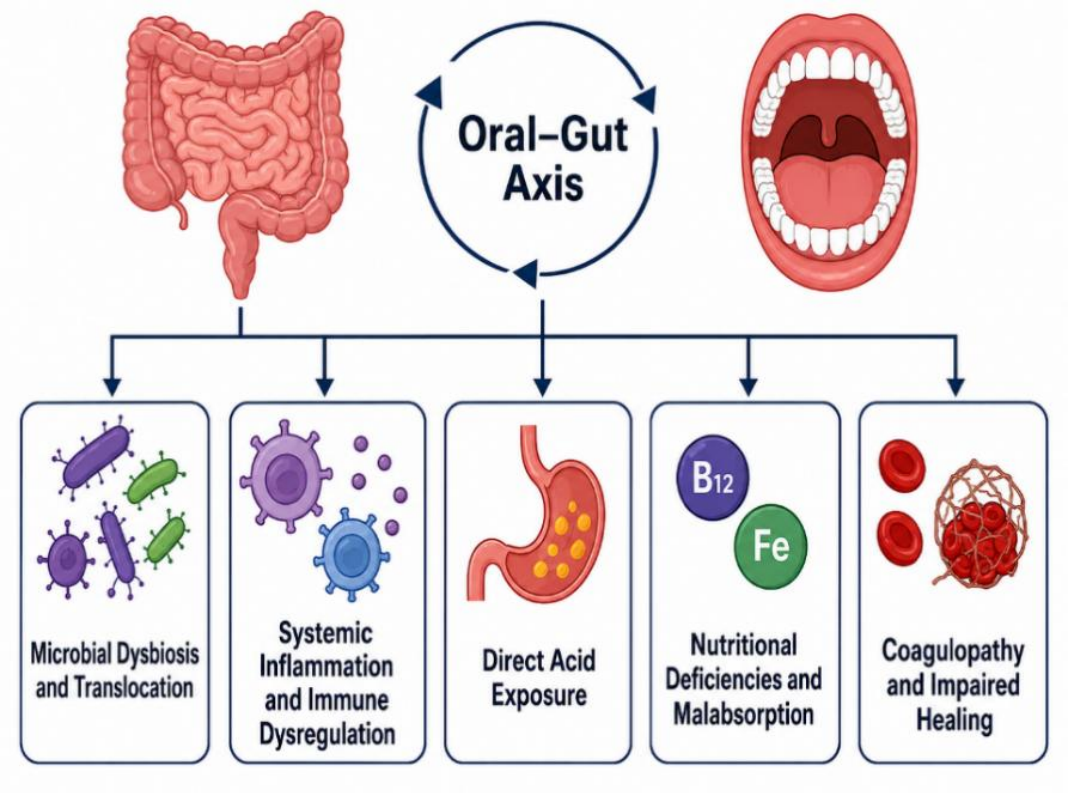


Figure 1. Schematic representation of the principal pathways linking gastrointestinal disorders and oral health. The figure integrates microbial dysbiosis and translocation, systemic inflammation and immune dysregulation, direct acid exposure, nutritional deficiencies and malabsorption, and coagulation abnormalities with impaired healing.

Accordingly, this narrative review examines gastrointestinal disorders from an integrated surgical and oral-health perspective, focusing on pathophysiological links, characteristic oral manifestations, and practical considerations for dental and oral surgical management.

2. PATHOPHYSIOLOGICAL LINKS BETWEEN GASTROINTESTINAL DISORDERS AND ORAL HEALTH

The relationship between gastrointestinal disorders and oral disease extends beyond the coexistence of lesions at two anatomically distinct sites. The oral cavity and gastrointestinal tract form a continuous biological system exposed to shared microbial, immunological, metabolic, and inflammatory influences. Current evidence increasingly supports the concept of an oral–gut axis, in which changes in oral microbial ecology may influence intestinal homeostasis, while systemic and intestinal inflammation can modify periodontal tissues, oral mucosa, salivary function, and dental structures [5,7,19].

Inflammatory bowel disease provides one of the clearest examples of this interaction. Crohn's disease and ulcerative colitis are characterized by chronic immune dysregulation and impaired mucosal barrier function, mechanisms that may also affect oral tissues [1–4]. Patients with inflammatory bowel disease appear to have a higher burden of periodontal disease, suggesting that persistent systemic inflammation and altered immune responses may create conditions favorable to periodontal destruction [5].

More recently, an association between inflammatory bowel disease and apical periodontitis has also been reported, indicating that the impact of intestinal inflammation may

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extend to periapical tissues and not only to the oral mucosa and periodontium [6].

Microbial dysbiosis represents a major mechanistic link. Periodontitis is associated with disruption of the normal oral microbial community and increased abundance of potentially pathogenic microorganisms. Repeated swallowing of oral bacteria and their products provides a plausible route by which oral dysbiosis may influence the gastrointestinal environment. Conversely, intestinal inflammation may alter systemic immune signaling and modify the host response to periodontal microorganisms. These interactions involve epithelial barrier dysfunction, microbial translocation, altered innate and adaptive immunity, and increased production of inflammatory mediators, creating a potentially self-sustaining cycle between periodontal and intestinal inflammation [7].

The oral–gastric relationship is also relevant in Helicobacter pylori infection. The organism has been detected within oral niches, particularly dental plaque and periodontal pockets, raising the possibility that the oral cavity may act as an extra-gastric reservoir. The observation that periodontal treatment may improve gastric H. pylori eradication supports the clinical relevance of oral microbial control in selected patients [17]. Nevertheless, oral colonization and gastric reinfection remain complex processes, and current evidence does not support considering the oral cavity as the sole source of recurrent infection [18].

A further mechanism involves direct chemical exposure. In gastroesophageal reflux disease, repeated contact between dental surfaces and gastric contents lowers intraoral pH and promotes demineralization of enamel and dentin. When acid exposure exceeds the protective capacity of saliva, progressive erosive tooth wear may develop. The pattern and severity of damage are influenced by reflux frequency, duration, salivary clearance, buffering capacity, dietary factors, and the presence of nocturnal reflux [8,9]. Thus, unlike immune-mediated oral manifestations, dental erosion in reflux disease primarily results from a direct physicochemical mechanism.

Malabsorption and nutritional deficiencies represent another important pathway linking gastrointestinal disease to oral health. Celiac disease can impair the absorption of iron, folate, vitamin B12, calcium, vitamin D, and other nutrients required for normal epithelial turnover and dental development. These disturbances may contribute to recurrent aphthous stomatitis, glossal changes, delayed eruption, and developmental enamel defects, particularly when the disease is active during odontogenesis [10–12]. Similar mechanisms may occur following bariatric surgery, where reduced intake, altered gastrointestinal anatomy, changes in digestion, and micronutrient deficiencies can affect both mineralized and soft oral tissues [15,16].

Chronic liver disease introduces additional systemic mechanisms. Impaired hepatic synthesis of coagulation factors, thrombocytopenia associated with portal hypertension, altered immune function, changes in drug metabolism, and nutritional abnormalities may collectively influence gingival bleeding, periodontal status, mucosal integrity, infection susceptibility, and tissue healing [13,14]. These changes become particularly relevant when invasive dental treatment or oral surgery is required.

The oral microbiome has also emerged as a potential contributor to gastrointestinal carcinogenesis. Oral microorganisms can reach the gastrointestinal tract through continuous salivary swallowing, and dysbiotic communities associated with periodontal disease have been investigated in relation to colorectal cancer [19,20].

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Particular attention has focused on Fusobacterium nucleatum, a common oral anaerobe that has been repeatedly identified in colorectal tumor tissue and associated with mechanisms involving inflammation, immune modulation, epithelial adhesion, and tumor microenvironment alterations [20,21]. Periodontal pathogens may therefore represent more than epidemiological markers, although causality and the magnitude of their contribution to colorectal carcinogenesis remain incompletely established [20,21].

Taken together, gastrointestinal–oral interactions arise through several overlapping mechanisms: systemic inflammation, immune dysregulation, microbial translocation, dysbiosis, direct acid exposure, nutritional deficiencies, coagulation abnormalities, and treatment-related effects. Their relative importance varies according to the underlying gastrointestinal disorder, but collectively they provide the biological basis for the diverse oral manifestations encountered in these patients and explain why oral findings may occasionally provide clinically useful information regarding gastrointestinal disease activity or systemic complications.

The principal mechanisms underlying gastrointestinal–oral interactions are summarized in Table 1, highlighting the convergence of inflammatory, microbial, metabolic, nutritional, and physicochemical pathways involved in the development of oral alterations.

Table 1. Main pathophysiological mechanisms linking gastrointestinal disorders and oral health.

Gastrointestinal condition	**Main pathophysiological mechanism**	**Oral consequence**	**Ref.**
Inflammatory bowel disease	Chronic systemic inflammation, immune dysregulation, epithelial barrier dysfunction, microbial dysbiosis	Periodontitis, apical periodontitis, recurrent mucosal lesions	[1–7]
Gastroesophageal reflux disease	Repeated exposure of dental surfaces to gastric acid, reduced intraoral pH, insufficient salivary buffering	Dental erosion, dentin exposure, hypersensitivity	[8,9]
Celiac disease	Malabsorption, micronutrient deficiencies, immune-mediated mucosal injury	Enamel defects, aphthous stomatitis, glossal changes, delayed eruption	[10–12]
Chronic liver disease	Coagulopathy, thrombocytopenia, impaired immune function, altered metabolism and nutrition	Gingival bleeding, periodontal changes, impaired healing, mucosal alterations	[13,14]
Bariatric surgery	Altered gastrointestinal anatomy, malabsorption, dietary changes, reflux, micronutrient deficiencies	Dental erosion, caries susceptibility, periodontal alterations, salivary changes	[15,16]
Helicobacter pylori infection	Possible oral reservoir, periodontal colonization, bidirectional oral–gastric microbial exchange	Persistence or recurrence of infection; periodontal involvement	[17,18]
Upper gastrointestinal disorders	Oral microbial dysbiosis and microbial migration along the gastrointestinal tract	Changes in oral microbial profiles associated with gastrointestinal disease	[19]
Colorectal cancer	Translocation of oral microorganisms, chronic inflammation, immune modulation and microbial dysbiosis	Association with periodontal pathogens, particularly Fusobacterium nucleatum	[20,21]

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3. ORAL MANIFESTATIONS ASSOCIATED WITH GASTROINTESTINAL DISORDERS

Gastrointestinal disorders can produce a broad spectrum of oral findings, ranging from mucosal lesions and periodontal inflammation to alterations of dental hard tissues, salivary function, and postoperative healing. Some changes are directly related to the underlying disease, whereas others arise from malabsorption, chronic inflammation, altered immunity, reflux, medication, or surgical treatment. Recognition of these patterns is clinically relevant because oral abnormalities may accompany gastrointestinal activity, contribute to morbidity, or occasionally prompt investigation of an undiagnosed systemic disorder [1–4,8–16].

Inflammatory bowel disease is among the gastrointestinal conditions most consistently associated with oral pathology. Crohn's disease may present with recurrent aphthous-like ulceration, diffuse or localized mucosal swelling, cobblestoning, mucosal tags, angular cheilitis, lip enlargement, and, less commonly, granulomatous lesions or oral fistulas. Pyostomatitis vegetans remains an uncommon but highly suggestive manifestation, particularly in patients with ulcerative colitis [1–4]. Periodontal involvement is also relevant, as patients with inflammatory bowel disease have been reported to show an increased burden of periodontitis, while recent evidence suggests a possible association with apical periodontitis [5,6]. These findings support the concept that oral involvement in inflammatory bowel disease extends beyond mucosal lesions and may affect multiple components of the dento-periodontal complex.

In gastroesophageal reflux disease, the dominant oral alteration is intrinsic dental erosion caused by repeated exposure to gastric acid. Lesions frequently affect palatal surfaces of maxillary teeth and occlusal or incisal surfaces, with progressive enamel loss potentially leading to dentin exposure, hypersensitivity, altered occlusion, and restorative complications [8,9]. The severity of erosive wear depends on reflux frequency and duration, salivary buffering, nocturnal exposure, dietary factors, and individual susceptibility. Unlike immune-mediated oral manifestations, the damage associated with reflux is largely physicochemical and may therefore progress despite the absence of visible mucosal inflammation.

Celiac disease is associated with a distinct combination of soft-tissue and developmental dental abnormalities. Recurrent aphthous stomatitis, glossitis, angular cheilitis, delayed eruption, and symmetric enamel defects have been described, especially when malabsorption occurs during odontogenesis [10–12]. Enamel hypoplasia or hypomineralization may involve several homologous teeth and can remain clinically evident long after intestinal symptoms have improved. In children, these findings may be particularly valuable because oral abnormalities can precede or accompany nonspecific gastrointestinal complaints [10–12].

Chronic liver disease affects oral health through a combination of metabolic, hematologic, immunological, and treatment-related mechanisms. Patients may present with xerostomia, gingival bleeding, mucosal petechiae, periodontal disease, altered taste, glossitis, and delayed healing [13]. Hepatitis C has additionally been associated with lichenoid lesions, lichen planus, salivary gland dysfunction, and other immune-mediated oral disorders [14]. The clinical significance of these manifestations increases when invasive dental procedures are required, because local findings may coexist with thrombocytopenia, impaired coagulation, or altered drug metabolism.

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Oral changes after bariatric surgery are heterogeneous and may reflect dietary modification, reflux, vomiting, salivary changes, and micronutrient deficiencies. Systematic reviews have reported dental erosion, increased caries susceptibility in some cohorts, periodontal changes, dry mouth, altered taste, and other oral complaints following surgery [15,16]. These observations are clinically important because bariatric procedures can improve systemic metabolic status while simultaneously introducing new oral risk factors that require long-term prevention and monitoring.

Microbial interactions add another dimension to gastrointestinal-oral disease. The oral cavity has been proposed as a potential reservoir for Helicobacter pylori, particularly within dental plaque and periodontal pockets, and periodontal therapy may improve gastric eradication rates when used as an adjunct to standard treatment [17,18]. In parallel, oral microbial signatures have been investigated in upper gastrointestinal disease and colorectal cancer [19–21]. Fusobacterium nucleatum has received particular attention because of its oral origin and repeated identification in colorectal tumor tissue, although the presence of a microbial association does not by itself establish direct causality [20,21].

The principal oral manifestations associated with major gastrointestinal conditions are summarized in Table 2. The table emphasizes the clinical pattern of involvement rather than isolated lesions and highlights the main mechanisms that should be considered during dental assessment.

Table 2. Main oral manifestations associated with gastrointestinal disorders.

Gastrointestinal disorder	Main oral manifestations	Underlying mechanisms	Ref.
Inflammatory bowel disease (Crohn's disease, ulcerative colitis)	Recurrent aphthous-like ulcers, mucosal swelling, cobblestoning, mucosal tags, pyostomatitis vegetans, angular cheilitis, periodontal disease, apical periodontitis	Immune dysregulation, systemic inflammation, microbial dysbiosis, epithelial barrier dysfunction	[1–7]
Gastroesophageal reflux disease	Dental erosion, dentin exposure, hypersensitivity, progressive loss of tooth structure	Repeated gastric acid exposure, reduced intraoral pH, insufficient salivary buffering	[8,9]
Celiac disease	Recurrent aphthous stomatitis, enamel defects, glossitis, angular cheilitis, delayed tooth eruption	Malabsorption, micronutrient deficiency, immune-mediated mucosal injury	[10–12]
Chronic liver disease / hepatitis C	Xerostomia, gingival bleeding, mucosal petechiae, periodontal disease, lichenoid lesions, lichen planus, glossitis, delayed healing	Coagulopathy, thrombocytopenia, immune dysfunction, altered metabolism, nutritional deficiency	[13,14]
Post-bariatric surgery	Dental erosion, caries susceptibility, periodontal changes, dry mouth, taste changes, mucosal discomfort	Dietary changes, reflux/vomiting, malabsorption, micronutrient deficiency, altered salivary flow	[15,16]
Helicobacter pylori infection	Periodontal colonization and inflammation; possible oral reservoir	Oral colonization, bacterial persistence, bidirectional oral-gastric microbial exchange	[17,18]
Upper gastrointestinal disorders	Microbiome-associated oral changes, xerostomia, halitosis,	Oral microbial dysbiosis, systemic inflammation,	[19]

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Gastrointestinal disorder	Main oral manifestations	Underlying mechanisms	Ref.
	dysgeusia in selected conditions	medication-related effects	
Colorectal cancer	Association with periodontal disease and oral microbial dysbiosis	Microbial translocation, chronic inflammation, immune modulation, Fusobacterium nucleatum	[20,21]

Overall, the oral manifestations associated with gastrointestinal disorders are heterogeneous and reflect different combinations of inflammatory, immune-mediated, microbial, nutritional, metabolic, and physicochemical mechanisms. Their clinical expression may involve the oral mucosa, periodontal and periapical tissues, salivary function, and dental hard tissues, with patterns that vary according to the underlying disease and its treatment. Recognition of these findings is important not only for symptomatic management, but also because selected oral abnormalities may provide additional clues regarding systemic disease activity, nutritional impairment, or treatment-related complications. Selected findings should therefore prompt a comprehensive medical and dental assessment, particularly when invasive dental procedures are being considered.

4. DENTAL AND ORAL SURGICAL MANAGEMENT IN PATIENTS WITH GASTROINTESTINAL DISORDERS

Dental management of patients with gastrointestinal disease should be guided by the underlying diagnosis, disease activity, current medication, nutritional status, and the anticipated invasiveness of the procedure. Although many routine dental interventions can be performed safely, patients receiving systemic therapy or presenting hepatic dysfunction, malabsorption, or previous major gastrointestinal surgery may require individualized assessment and interdisciplinary coordination.

Patients with inflammatory bowel disease are frequently treated with corticosteroids, immunomodulators, or biological agents. These therapies may influence immune response, susceptibility to infection, and postoperative healing, particularly when invasive oral procedures are planned [22]. The indication for interrupting biological therapy should not be determined routinely by the dentist alone, because unnecessary treatment withdrawal may precipitate gastrointestinal disease exacerbation. Elective oral surgery should preferably be performed when systemic disease is clinically controlled, with careful evaluation of concurrent medication, previous infections, and healing capacity [22].

Patients with chronic liver disease represent a distinct surgical-risk group. Cirrhosis may be associated with thrombocytopenia, reduced hepatic synthesis of coagulation factors, portal hypertension, impaired drug metabolism, and increased vulnerability to infection [13,23]. Before extractions or other procedures involving significant tissue trauma, recent hematological and hepatic parameters should therefore be reviewed according to the clinical severity of liver disease. Nevertheless, available evidence indicates that minor oral surgery can often be performed safely in appropriately selected patients, and postoperative bleeding is generally manageable with local hemostatic measures such as compression, suturing, absorbable hemostatic materials, or antifibrinolytic strategies [23,24]. Drug selection also requires caution because hepatic dysfunction may alter the metabolism and toxicity of commonly prescribed analgesics, antibiotics, and sedatives [13].

Dental evaluation is particularly relevant in candidates for liver transplantation. Potential

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odontogenic infections should be identified and treated before transplantation whenever feasible, thereby reducing the infectious burden before long-term immunosuppression [25]. After transplantation, preventive care, periodontal surveillance, and rapid management of oral infection remain important because immunosuppressive therapy may modify host defense, oral microbiota, mucosal health, and healing responses [25,26].

Patients who have undergone bariatric surgery require a different preventive strategy. Reflux, vomiting, dietary changes, altered salivary function, and micronutrient deficiencies can increase susceptibility to dental erosion and other oral complications [15,16]. Regular dental monitoring, dietary counseling, fluoride-based prevention, management of xerostomia, and correction of relevant nutritional deficiencies should therefore accompany long-term postoperative follow-up.

Overall, safe dental and oral surgical care in gastrointestinal patients depends less on the diagnosis itself than on its systemic consequences. Assessment of bleeding risk, immune status, medication exposure, nutritional deficiencies, and previous surgery allows treatment to be adapted to the individual patient while avoiding unnecessary postponement of essential dental care.

5. CONCLUSIONS

Gastrointestinal disorders may influence oral health through multiple interconnected mechanisms, including chronic inflammation, immune dysregulation, microbial dysbiosis, acid exposure, malabsorption, nutritional deficiencies, and hepatic dysfunction. The resulting clinical spectrum ranges from mucosal and periodontal lesions to dental erosion, developmental enamel defects, salivary disturbances, and impaired tissue healing. Recognition of these findings is clinically relevant because some oral manifestations may accompany gastrointestinal disease activity, reflect systemic complications, or reveal treatment-related effects.

Dental and oral surgical management should therefore be individualized according to the underlying gastrointestinal condition, systemic status, medication, bleeding risk, immune competence, and nutritional profile. Particular attention is required in patients receiving biological or immunosuppressive therapy, those with cirrhosis or liver transplantation, and individuals after bariatric surgery. Close collaboration between dentists, oral surgeons, gastroenterologists, and surgeons may improve prevention, perioperative safety, and long-term oral outcomes, reinforcing the importance of integrating oral assessment into the multidisciplinary care of patients with gastrointestinal disease.

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