Modern American Journal of Medical and Health Sciences
ISSN (E): 3067-803X
Volume 01, Issue 09, December, 2025
Website: usajournals.org

CLINICAL, BIOCHEMICAL AND FUNCTIONAL ASPECTS OF HARD DENTAL TISSUE ALTERATIONS IN PATIENTS WITH GASTROINTESTINAL DISEASES AND BILIARY DYSFUNCTION: A NARRATIVE REVIEW

Akhtamova Irodakhon Akbarovna
Assistant Department of Dentistry, Alfraganus University.

Rajabov Otabek Asrorovich
Head of the Department of Therapeutic Dentistry,
Bukhara State Medical Institute, DSc, Associate Professor

Abstract

Gastrointestinal diseases and biliary dysfunction are among the most prevalent chronic systemic disorders and are frequently associated with metabolic disturbances that affect multiple organs and tissues, including the oral cavity. In recent years, increasing scientific attention has been directed toward the relationship between gastrointestinal pathology and alterations in hard dental tissues, particularly enamel and dentin. These changes are primarily mediated by disturbances in mineral metabolism, acid–base balance, and salivary protective mechanisms.

This narrative review summarizes and analyzes current scientific evidence regarding clinical, biochemical, and functional changes in hard dental tissues in patients with gastrointestinal diseases and biliary dysfunction. Special emphasis is placed on pathogenetic mechanisms of enamel demineralization, erosive tooth wear, and dentin hypersensitivity, as well as the role of saliva in maintaining dental hard tissue homeostasis. The review highlights the importance of comprehensive and interdisciplinary diagnostic and preventive approaches for managing dental complications in this patient population.

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Keywords: Hard dental tissues, enamel, dentin, gastrointestinal diseases, biliary dysfunction, saliva, demineralization, erosive tooth wear

Introduction

Hard dental tissues, including enamel and dentin, are highly mineralized structures whose integrity depends on systemic metabolic balance and local protective mechanisms within the oral cavity. Enamel, characterized by its high mineral content and limited regenerative capacity, is particularly vulnerable to disturbances in mineral metabolism, acid–base equilibrium, and salivary composition. Consequently, systemic diseases that disrupt these processes may significantly compromise the structural and functional stability of dental hard tissues.

Gastrointestinal diseases (GIDs) and biliary dysfunction represent a major group of chronic conditions associated with long-term metabolic, biochemical, and inflammatory changes. Disorders such as gastritis, peptic ulcer disease, gastroesophageal reflux disease, malabsorption syndromes, and functional biliary disorders are known to alter nutrient absorption, mineral bioavailability, and systemic acid–base balance. These alterations may indirectly or directly affect the oral environment, predisposing patients to enamel demineralization, erosive lesions, and non-carious tooth defects.

Despite growing recognition of the oral–systemic connection, the impact of gastrointestinal and biliary disorders on hard dental tissues remains insufficiently systematized in the literature. Existing studies often focus on isolated clinical manifestations without integrating biochemical and functional parameters. Therefore, a comprehensive review of current evidence is required to clarify the mechanisms underlying dental hard tissue alterations in this patient group and to identify potential diagnostic and preventive strategies.

Physiological Role of the Gastrointestinal Tract in Dental Hard Tissue Mineralization

The gastrointestinal tract plays a fundamental role in maintaining mineral homeostasis essential for the formation and preservation of dental hard tissues.

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Calcium, phosphate, magnesium, and trace elements involved in enamel and dentin mineralization are absorbed primarily in the small intestine, while their bioavailability depends on gastric acidity, intestinal mucosal integrity, and hepatobiliary function[1.3].

Chronic gastrointestinal disorders are frequently associated with impaired absorption of minerals and fat-soluble vitamins, particularly vitamin D, which is critical for calcium–phosphate metabolism. Reduced intestinal absorption leads to systemic mineral deficiency, negatively influencing enamel crystallization and dentin mineral density. As a result, enamel becomes more permeable and susceptible to acid-induced dissolution.

Biliary dysfunction further exacerbates these disturbances by impairing lipid digestion and absorption, contributing to deficiencies in vitamins A, D, E, and K. These deficiencies may compromise antioxidant defense mechanisms and mineral metabolism, indirectly affecting the resistance of dental hard tissues to erosive and demineralizing challenges[2.5].

Impact of Gastroesophageal and Duodenogastric Reflux on Enamel Integrity

One of the most significant mechanisms linking gastrointestinal pathology to dental hard tissue damage is the exposure of teeth to acidic gastric contents. Gastroesophageal reflux disease (GERD) and duodenogastric reflux result in the repeated contact of enamel surfaces with hydrochloric acid and bile acids, leading to chemical dissolution of the mineralized enamel layer[3.4].

Erosive tooth wear associated with reflux is characterized by smooth, shallow lesions predominantly affecting the palatal surfaces of maxillary anterior teeth and occlusal surfaces of posterior teeth. The severity of erosive damage correlates with the frequency and duration of acid exposure, as well as with salivary buffering capacity and individual enamel resistance.

Importantly, reflux-related enamel erosion may occur in the absence of dental caries, emphasizing the non-bacterial nature of these lesions. Early stages are often asymptomatic, delaying diagnosis and increasing the risk of progressive enamel loss and dentin exposure.

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Saliva plays a central role in preserving the structural integrity of hard dental tissues by providing buffering capacity, mineral ions for remineralization, and protective proteins that modulate oral homeostasis. In patients with gastrointestinal diseases and biliary dysfunction, both quantitative and qualitative salivary parameters are frequently altered, creating a microenvironment that favors demineralization and erosive tooth wear[7.8].

Numerous studies have demonstrated that gastrointestinal pathology is associated with a reduction in salivary flow rate, particularly in patients with chronic gastritis, peptic ulcer disease, and functional biliary disorders. Hyposalivation prolongs the contact time between acids and enamel surfaces, reducing the clearance of erosive agents and increasing the risk of mineral loss. Additionally, decreased salivary flow compromises the delivery of calcium and phosphate ions required for enamel remineralization[5.6].

Biochemically, patients with gastrointestinal and biliary disorders often exhibit a decrease in salivary pH and buffering capacity. This shift toward an acidic oral environment lowers the critical pH threshold for enamel dissolution, rendering dental hard tissues more susceptible to acid attacks of both endogenous and exogenous origin. Reduced concentrations of ionized calcium and phosphate in saliva further limit the reparative potential of enamel, especially in areas exposed to repeated acid challenges.

Alterations in the organic composition of saliva also contribute to dental hard tissue vulnerability. Decreased levels of salivary proteins, enzymes, and antimicrobial peptides impair the protective barrier of the acquired pellicle, facilitating direct acid contact with the enamel surface. In this context, saliva becomes not only a marker of systemic disturbances but also a mediator of dental hard tissue pathology.

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Table 1. Reported salivary changes in patients with gastrointestinal diseases and biliary dysfunction

Salivary parameter	Direction of change	Clinical relevance
Salivary flow rate	Decreased	Prolonged acid contact with enamel
pH level	Decreased	Enhanced enamel demineralization
Buffering capacity	Reduced	Weakened acid neutralization
Calcium and phosphate	Reduced	Impaired remineralization potential

Clinically, hard dental tissue alterations in patients with gastrointestinal and biliary disorders present as a spectrum of non-carious lesions. The most frequently reported manifestations include initial enamel demineralization, erosive tooth wear, dentin hypersensitivity, and pathological tooth wear. These changes often exhibit a symmetrical distribution and involve multiple teeth, reflecting the systemic nature of the underlying etiological factors.

Erosive lesions related to gastrointestinal disorders typically affect smooth enamel surfaces and are characterized by a loss of surface luster, shallow concavities, and rounded enamel margins. In advanced cases, enamel thinning leads to dentin exposure, resulting in pronounced hypersensitivity and increased susceptibility to mechanical wear. Unlike carious lesions, erosive defects progress without bacterial involvement, emphasizing the chemical nature of tissue destruction[2.4].

Dentin hypersensitivity is a common complaint among patients with gastrointestinal pathology and is frequently associated with erosive enamel loss and dentin exposure. The condition significantly affects quality of life, as patients experience pain in response to thermal, chemical, or tactile stimuli. From a clinical perspective, hypersensitivity may serve as an early indicator of underlying systemic disorders affecting dental hard tissues.

Importantly, the severity of dental hard tissue alterations often correlates with the duration and activity of gastrointestinal disease. Chronic, poorly controlled conditions are associated with more extensive enamel damage and reduced capacity for natural remineralization. This highlights the need for early dental assessment in patients with known gastrointestinal and biliary disorders.

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Table 2. Common clinical manifestations of hard dental tissue alterations in gastrointestinal and biliary disorders

Clinical manifestation	Frequency (reported)	Pathogenetic association
Enamel demineralization	High	Mineral imbalance, low pH
Erosive tooth wear	Very high	Reflux acids, bile acids
Dentin hypersensitivity	Moderate–high	Enamel thinning, dentin exposure
Pathological tooth wear	Moderate	Structural enamel weakness

A comprehensive assessment of hard dental tissue alterations in patients with gastrointestinal diseases and biliary dysfunction requires an integrated diagnostic strategy that combines clinical, biochemical, and functional methods. Given the systemic nature of gastrointestinal pathology, isolated dental examination is often insufficient to identify early changes in enamel and dentin integrity.

Clinical diagnostic approaches primarily involve careful visual inspection of tooth surfaces, focusing on early signs of demineralization, loss of enamel luster, erosive defects, and dentin exposure. The use of standardized clinical indices and hypersensitivity assessment scales allows for objective documentation of lesion severity and distribution. Particular attention should be paid to lesion symmetry and localization, as these features often reflect systemic etiological factors rather than localized mechanical or bacterial influences.

Biochemical assessment of saliva serves as a valuable noninvasive tool for evaluating the oral environment in patients with gastrointestinal and biliary disorders. Measurement of salivary pH, buffering capacity, and mineral ion concentrations provides insight into the remineralization potential of enamel and the degree of acid challenge within the oral cavity. In several studies, salivary biomarkers have been proposed as indicators of both oral and systemic disease activity.

Functional diagnostic methods further enhance the understanding of dental hard tissue vulnerability. Tests assessing enamel remineralization capacity, surface microhardness, and permeability enable early detection of structural changes that may not yet be clinically evident. These methods are particularly useful for risk stratification and preventive planning in patients with chronic gastrointestinal pathology.

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An analysis of contemporary literature reveals that gastrointestinal diseases and biliary dysfunction exert a multifactorial and clinically significant impact on the condition of hard dental tissues. The reviewed studies consistently demonstrate that disturbances in mineral metabolism, salivary composition, and acid–base balance create a biological environment conducive to enamel demineralization and erosive tooth wear.

The chronic nature of many gastrointestinal disorders results in prolonged exposure of dental hard tissues to adverse systemic and local factors. Over time, compensatory mechanisms within the oral cavity become insufficient, leading to progressive loss of enamel integrity and increased susceptibility to dentin involvement. Importantly, the severity of dental alterations appears to be influenced not only by the presence of gastrointestinal disease but also by its duration, activity, and degree of metabolic control.

The literature also highlights the diagnostic value of saliva as a link between systemic disease and oral manifestations. Changes in salivary parameters reflect both gastrointestinal pathology and oral health status, supporting the concept of saliva as a practical biomarker for interdisciplinary assessment. However, variability in study design and diagnostic criteria underscores the need for standardized protocols in future research.

Conclusion

This narrative review demonstrates that gastrointestinal diseases and biliary dysfunction are important systemic risk factors for the development of hard dental tissue alterations. Disruptions in mineral metabolism, changes in salivary quantity and quality, and repeated exposure to endogenous acids collectively compromise enamel and dentin integrity. These processes predispose affected individuals to enamel demineralization, erosive tooth wear, and dentin hypersensitivity, even in the absence of traditional cariogenic factors.

The findings emphasize the necessity of a comprehensive and interdisciplinary approach to the diagnosis and management of dental hard tissue pathology in patients with gastrointestinal disorders. Early identification of salivary and functional changes, combined with close collaboration between dental

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professionals and gastroenterologists, may significantly reduce the progression of non-carious dental lesions and improve overall patient outcomes.

Future research should focus on the development of standardized diagnostic criteria and preventive strategies that address both systemic and local contributors to hard dental tissue degradation. Such approaches may enhance clinical decision-making and support personalized preventive care for patients with gastrointestinal and biliary diseases.

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