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RESEARCH ARTICLE

Oral Fluid Characteristics and Mineral Metabolism in Patients with Gastrointestinal Diseases and Biliary Dysfunction

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Abstract: The article examines the characteristics of oral fluid and mineral metabolism in patients with gastrointestinal diseases and biliary dysfunction. Particular attention is given to salivary pH, buffer capacity, calcium and phosphate levels, and the mineralizing potential of oral fluid as key factors determining enamel resistance to demineralization. Chronic gastrointestinal pathology may be associated with impaired digestion, altered absorption of minerals and fat-soluble vitamins, changes in intestinal microbiota, and systemic metabolic disturbances. Biliary dysfunction, including impaired bile secretion and biliary sludge formation, may additionally affect lipid digestion, bile acid metabolism, vitamin D-dependent calcium-phosphate regulation, and the biochemical conditions required for enamel and dentin mineral stability. The article substantiates the need for a comprehensive clinical and biochemical assessment of oral fluid in patients with gastrointestinal and biliary disorders in order to identify early risk factors for enamel demineralization, dental erosion, caries progression, and non-carious lesions [1; 5].

Key words: Oral fluid, saliva, salivary pH, buffer capacity, calcium, phosphates.

INTRODUCTION

Oral fluid is one of the most important biological media involved in maintaining the structural stability of dental hard tissues. It provides mechanical cleansing of the enamel surface, neutralization of acids, regulation of the oral microbiocenosis, and delivery of calcium and phosphate ions necessary for enamel remineralization. The protective function of saliva depends not only on its quantity, but also on its biochemical composition, pH, buffer capacity, mineral saturation, and ability to restore the hydroxyapatite structure after acid exposure [3; 4].

In patients with gastrointestinal diseases, the composition and functional properties of oral fluid may change under the influence of digestive, inflammatory, metabolic, and microbiotic disturbances. Chronic gastritis, gastroduodenitis, inflammatory bowel diseases, reflux-associated conditions, intestinal dysbiosis, and malabsorption may alter the acid-base balance of the oral cavity and reduce the mineralizing potential of saliva. As a result, enamel becomes more vulnerable to demineralization, erosion, hypersensitivity, and progression of carious and non-carious lesions.

Mineral metabolism is directly involved in the preservation of enamel and dentin resistance. Calcium and phosphate ions participate in the restoration of enamel mineral structure, while disturbances in their concentration reduce the effectiveness of remineralization. In chronic gastrointestinal pathology, impaired absorption of minerals and vitamins may negatively affect calcium-phosphate homeostasis. This is especially significant in patients with long-term digestive disorders, dietary restrictions,

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recurrent dyspeptic symptoms, and systemic metabolic imbalance [2; 4].

Biliary dysfunction is an additional factor that may influence oral fluid and mineral metabolism indirectly. Impaired bile secretion changes the physiological entry of bile into the duodenum, disrupts lipid digestion, and may affect the absorption of fat-soluble vitamins, including vitamin D. Since vitamin D is essential for calcium-phosphate regulation, biliary disorders may contribute to reduced mineral availability and decreased enamel resistance to demineralizing factors [5; 6]. According to recent gastroenterological data, biliary sludge is characterized by heterogeneous bile, suspended microparticles, paste-like bile, altered bile acid metabolism, intestinal dysbiosis, and increased lithogenicity of bile, which confirms the systemic metabolic significance of biliary dysfunction.

Clinical studies also indicate that gastrointestinal diseases are associated with more frequent lesions of dental hard tissues. In adults with Crohn's disease and ulcerative colitis, higher rates of dental caries, enamel erosion, wedge-shaped defects, and increased tooth wear were observed compared with control groups. Importantly, these changes were not fully explained by differences in oral hygiene, which suggests the involvement of systemic mechanisms, including altered oral fluid properties, mineral imbalance, chronic inflammation, and reduced adaptive capacity of the organism [1; 2].

The biochemical assessment of oral fluid in patients with gastrointestinal diseases and biliary dysfunction should include determination of salivary pH, buffer capacity, calcium concentration, phosphate concentration, and general mineralizing potential. These parameters make it possible to evaluate the ability of saliva to neutralize acids and support enamel remineralization. Such assessment is especially important in patients with bile secretion disorders, since biliary dysfunction may be associated with impaired digestion, intestinal dysbiosis, altered bile acid circulation, and metabolic changes affecting mineral homeostasis.

Purpose of the research

The aim of the study is to analyze the characteristics of oral fluid and mineral metabolism in patients with gastrointestinal diseases and biliary dysfunction, and to substantiate the diagnostic value of salivary pH, buffer capacity, calcium, phosphates, and mineralizing potential in the early detection of enamel demineralization and dental hard tissue lesions [3; 5].

A comprehensive study of oral fluid in this group of patients has practical importance for both dentistry and gastroenterology. It allows identification of patients with increased risk of enamel demineralization, dental erosion, caries progression, and non-carious lesions. Integration of dental examination with biochemical assessment of saliva and gastroenterological history may improve early diagnosis, personalize preventive measures, and support interdisciplinary management of patients with chronic gastrointestinal and biliary pathology [4; 6].

RESULTS

Oral fluid plays a central role in maintaining the mineral stability of enamel and dentin. Its protective function depends on several measurable characteristics: salivary pH, buffer capacity, calcium and phosphate content, and the ability to support enamel remineralization after acid exposure. In patients with gastrointestinal diseases and biliary dysfunction, these parameters may change due to chronic digestive disorders, reflux symptoms, impaired absorption of minerals and vitamins, intestinal dysbiosis, and disturbances of bile secretion.

When the pH of oral fluid shifts toward acidic values, enamel becomes more vulnerable to mineral loss. If the buffering capacity of saliva is reduced, acids remain active on the tooth surface for a longer time. This condition is especially important in patients with gastroesophageal reflux, chronic dyspepsia, gastritis, gastroduodenitis, inflammatory bowel diseases, and biliary disorders. In such patients, enamel demineralization may begin with subtle

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clinical signs: chalky spots, loss of gloss, hypersensitivity, and early erosive changes.

Table 1.

Main oral fluid indicators relevant to enamel stability

Indicator	What it shows	Clinical meaning
Salivary pH	Acidity of oral fluid	Low pH increases enamel demineralization
Buffer capacity	Ability to neutralize acids	Low buffering prolongs acid damage
Calcium level	Mineral supply for enamel	Low calcium weakens remineralization
Phosphate level	Mineral support for hydroxyapatite	Low phosphate slows enamel repair
Salivary flow	Natural cleansing of teeth	Reduced flow increases plaque and acid retention

This table shows the most practical indicators for assessing oral fluid in patients with gastrointestinal and biliary pathology. Salivary pH and buffer capacity are important for evaluating acid resistance, while calcium and phosphate levels reflect the ability of saliva to restore the mineral structure of enamel. Salivary flow is also clinically relevant because reduced flow decreases natural oral cleansing and increases the duration of acid contact with enamel.

Biliary dysfunction may influence oral fluid and mineral metabolism indirectly. Disorders of bile secretion affect lipid digestion and may reduce the absorption of fat-soluble vitamins, including vitamin D. Since vitamin D is involved in calcium-phosphate metabolism, chronic biliary dysfunction can create unfavorable conditions for enamel remineralization. Recent data on biliary sludge also show that bile disorders are associated with altered bile composition, intestinal dysbiosis, impaired bile acid circulation, and metabolic disturbances, which confirms the systemic importance of biliary pathology.

From a clinical point of view, patients with gastrointestinal diseases and biliary dysfunction should be examined not only for caries, but also for early signs of enamel instability. The most important signs are chalky enamel spots, enamel opacity, hypersensitivity, erosive defects, wedge-shaped lesions, and increased tooth wear. These findings may indicate that enamel is losing mineral resistance and that saliva is no longer providing sufficient protection.

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Table 2.

Clinical signs suggesting impaired enamel mineralization

Clinical sign	What the dentist observes	Possible interpretation
Chalky spots	White opaque areas	Early enamel demineralization
Loss of gloss	Matte enamel surface	Initial surface mineral loss
Hypersensitivity	Pain from cold, sour, or sweet stimuli	Enamel thinning or exposed dentin
Enamel erosion	Smooth shallow defects	Acid-related enamel dissolution
Increased wear	Loss of enamel on chewing surfaces	Reduced resistance of hard tissues

The table presents simple clinical signs that can be detected during a routine dental examination. Chalky spots and loss of gloss are early signs of mineral imbalance. Hypersensitivity may indicate weakening of the enamel-dentin complex. Erosions are especially important in patients with reflux or chronic digestive disorders because they reflect chemical dissolution of enamel. Increased tooth wear may become more pronounced when enamel has reduced mineral resistance.

Clinical data confirm that patients with chronic gastrointestinal diseases may have more frequent hard dental tissue lesions than control groups. In adults with Crohn's disease and ulcerative colitis, enamel erosion was detected in 21.3% and 20.6% of cases respectively, while in the control group it was found in 10.4% of cases. These findings support the need to assess enamel changes together with gastrointestinal history and oral fluid characteristics.

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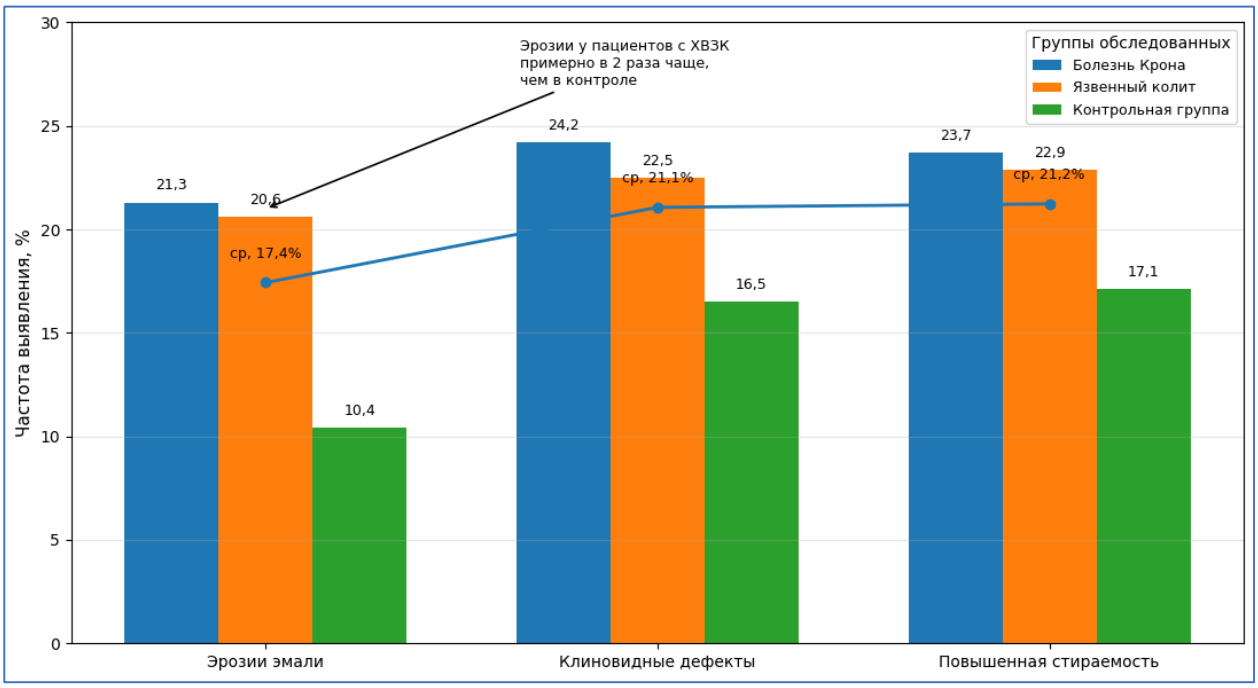
Figure 1.

Comparative frequency of non-carious dental hard tissue lesions in patients with chronic intestinal pathology, %



The figure demonstrates that non-carious dental hard tissue lesions are more frequent in patients with chronic intestinal pathology than in the control group. Enamel erosion was detected in 21.3% of patients with Crohn's disease and 20.6% of patients with ulcerative colitis, compared with 10.4% in the control group. Wedge-shaped defects and increased tooth wear also showed higher values in both clinical groups. This pattern indicates that chronic gastrointestinal pathology may be associated not only with caries progression, but also with reduced structural resistance of enamel and dentin. The greatest difference is observed for enamel erosion, which supports the diagnostic relevance of acid exposure, altered oral fluid properties, and impaired mineral balance in patients with gastrointestinal and biliary disorders.

The diagnostic approach in such patients should include three components. The first is clinical dental examination with registration of enamel erosion, chalky spots, hypersensitivity, wedge-shaped defects, tooth wear, and carious lesions. The second is biochemical evaluation of oral fluid, including pH, buffer capacity, calcium, and phosphate levels. The third is medical history taking, with attention to reflux, dyspepsia, chronic intestinal disease, gallbladder dysfunction, biliary sludge, intolerance to fatty foods, and signs of impaired bile secretion.

This approach is important because early enamel changes may remain clinically underestimated. At the initial stage, patients often complain only of mild sensitivity or discomfort after acidic food. However, if salivary pH is low, buffering is weak, and calcium-phosphate balance is disturbed, these early changes may progress to visible erosion, enamel thinning, dentin exposure, and increased risk of caries.

Patients with gastrointestinal diseases and biliary dysfunction should therefore be considered a group with increased risk of enamel demineralization and non-carious lesions. Preventive management should include control of acid exposure, remineralizing therapy, correction of oral hygiene, dietary recommendations, evaluation of salivary

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parameters, and cooperation between dentists and gastroenterologists.

In this context, oral fluid analysis becomes not an additional optional procedure, but an important diagnostic tool. It allows the clinician to identify biochemical conditions that cannot be seen during visual examination, but directly influence enamel resistance. The combination of clinical signs and oral fluid indicators provides a more accurate assessment of dental risk in patients with gastrointestinal and biliary pathology.

DISCUSSION

The analysis of oral fluid characteristics and mineral metabolism in patients with gastrointestinal diseases and biliary dysfunction demonstrates that dental hard-tissue resistance cannot be assessed only through visual examination of enamel and dentin. Saliva is a biologically active diagnostic medium that reflects the local condition of the oral cavity and, at the same time, may indicate systemic metabolic disturbances. In patients with chronic gastrointestinal pathology, changes in salivary pH, buffer capacity, calcium and phosphate levels may create conditions for enamel demineralization, dental erosion, hypersensitivity, and progression of non-carious lesions.

A key mechanism is the disruption of the balance between demineralization and remineralization. Under physiological conditions, oral fluid neutralizes acids and supplies calcium and phosphate ions to the enamel surface. These ions support the restoration of hydroxyapatite crystals after acid exposure. When salivary pH decreases and buffer capacity is weakened, acids remain active for a longer period, accelerating mineral loss from enamel. This mechanism is especially important in patients with gastroesophageal reflux, chronic dyspepsia, gastroduodenal disorders, inflammatory bowel diseases, and biliary dysfunction.

Clinical data support the relevance of this mechanism. In adults with Crohn's disease and ulcerative colitis, a higher frequency of dental hard-tissue lesions was reported compared with the control group. Enamel erosion was observed in 21.3% of patients with Crohn's disease, 20.6% of patients with ulcerative colitis, and 10.4% of individuals in the control group. Wedge-shaped defects and increased tooth wear were also more frequent in patients with chronic intestinal pathology. These findings indicate that chronic gastrointestinal disease may be associated not only with caries, but also with structural weakening of enamel and dentin.

An important diagnostic point is that these changes cannot be explained only by oral hygiene. In the clinical study of patients with chronic inflammatory bowel diseases, no statistically significant differences in oral hygiene status were found between the main and control groups, despite the higher prevalence of carious and non-carious lesions in patients with gastrointestinal pathology. This supports the role of systemic factors: chronic inflammation, altered mineral metabolism, impaired salivary protection, reduced adaptive capacity of the organism, and possible changes in oral fluid composition.

Biliary dysfunction may intensify these processes indirectly. Bile secretion disorders affect lipid digestion, bile acid circulation, intestinal microbiota, and the absorption of fat-soluble vitamins. Vitamin D is especially significant because it participates in calcium-phosphate metabolism and supports the mineral stability of hard tissues. If bile secretion is impaired, the absorption of vitamin D and lipid-soluble components may decrease, which can reduce the systemic background necessary for normal enamel and dentin mineralization.

Recent gastroenterological data on biliary sludge show that biliary dysfunction is associated with changes in the physicochemical properties of bile, altered bile acid metabolism, intestinal dysbiosis, increased lithogenicity of bile, and impaired enterohepatic circulation. These processes do not directly prove dental enamel damage, but they provide a strong pathophysiological basis for considering biliary dysfunction as a systemic risk factor in patients with impaired oral mineral homeostasis.

CONCLUSION

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Oral fluid characteristics and mineral metabolism play a central role in maintaining enamel and dentin stability in patients with gastrointestinal diseases and biliary dysfunction. Salivary pH, buffer capacity, calcium and phosphate levels, and mineralizing potential determine the ability of enamel to resist acid exposure and recover after demineralization.

Chronic gastrointestinal diseases may contribute to dental hard-tissue damage through reflux-related acid exposure, chronic inflammation, altered absorption of nutrients, intestinal dysbiosis, and changes in oral fluid properties. These mechanisms increase the risk of enamel erosion, focal demineralization, hypersensitivity, wedge-shaped defects, increased tooth wear, and caries progression.

Biliary dysfunction may influence oral mineral balance indirectly through impaired bile secretion, altered bile acid metabolism, reduced lipid digestion, intestinal dysbiosis, and possible disturbance of vitamin D-dependent calcium-phosphate metabolism. This creates an unfavorable systemic background for enamel remineralization and dentin resistance.

Comprehensive assessment of patients with gastrointestinal diseases and biliary dysfunction should include clinical dental examination, biochemical analysis of oral fluid, and evaluation of gastroenterological and biliary history. Such an approach allows earlier detection of patients at high risk of enamel demineralization and non-carious lesions.

Preventive management should be interdisciplinary. It should combine remineralizing therapy, acid exposure control, dietary correction, oral hygiene optimization, salivary monitoring, and gastroenterological management of digestive and biliary disorders.

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