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Correlation between Digestive Disorders in Chronic Pancreatitis with Exocrine Insufficiency and Clinical Indicators of Chronic Periodontitis

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Abstract

This study investigates the correlation between digestive disorders in chronic pancreatitis (CP) with exocrine insufficiency and clinical indicators of chronic periodontitis to elucidate their interaction mechanisms. The study included a main group (24 patients) receiving adapted interdisciplinary therapy and a comparison group (22 patients) undergoing standard treatment. Clinical periodontitis indices (OHI-S, SBI, PI, PMA, periodontal pocket depth), coprological parameters, and fecal pH were evaluated. Weak to moderate correlations ($r = 0,21–0,57$, $p < 0,05$) were identified between oral hygiene, gingival bleeding, pocket depth, and digestive disorders, with a strong correlation between fecal mucus and leukocytes ($r = 0,78$). The findings confirm that malabsorption and dysbiosis in CP exacerbate periodontal pathology via systemic inflammation, highlighting the need for an interdisciplinary approach to diagnosis and treatment.

Keywords: Chronic Pancreatitis, Chronic Periodontitis, Digestive Disorders, Exocrine Insufficiency, Malabsorption, Dysbiosis, Clinical Indicators, Correlation, Systemic Inflammation

Introduction

Periodontal diseases, particularly gingivitis and periodontitis, are among the most prevalent oral conditions globally. Periodontitis ranks as the sixth most common disease worldwide and can lead to severe outcomes, including tooth loss, alveolar bone destruction, and masticatory dysfunction. These complications indirectly but significantly impact nutrition and overall health, resulting in substantial socioeconomic consequences and increased healthcare costs [1,2,6,11,13,18,22].

Chronic pancreatitis (CP) of moderate severity with exocrine pancreatic insufficiency is characterized by pronounced digestive disorders, reflected in coprological findings. These alterations stem from reduced secretion of pancreatic enzymes essential for digesting fats, proteins, and carbohydrates, coupled with impaired gastrointestinal motility. The primary cause of exocrine insufficiency in CP is secondary dysfunction due to asynchronous delivery of pancreatic juice, bile, and gastric contents into the

duodenum. This disrupts fat emulsification and the breakdown of proteins and carbohydrates, exacerbating nutrient deficiencies [3,5,9,12,21,23].

Exocrine pancreatic insufficiency in CP is marked by impaired enzyme secretion, leading to the following clinical manifestations:

Stool abnormalities: frequent defecation (3–6 times daily) with increased fecal volume (polyfecalia) is typical. Stool is liquid or mushy, grayish, and greasy due to steatorrhea. Severe fat malabsorption results in poorly flushing, foul-smelling stool caused by fermentation of undigested substances.

Malabsorption: inadequate enzymatic digestion leads to absorption of large molecules, causing allergic reactions (e.g., pruritus, rashes, or eczematous changes) in 25–35% of patients.

Dyspepsia: nausea, vomiting, and early satiety arise from impaired gastrointestinal motility, including duodenogastric reflux. These symptoms may be transient (during exacerbations) or persistent with severe dysfunction.
Steatorrhea, resulting from pancreatic lipase deficiency, significantly reduces absorption of fat-soluble vitamins (A, D, E, K), with the following consequences:

Vitamin A: deficiency (<0,3 mg/L in 30–40% of patients) manifests as impaired twilight vision, dry skin, and mucosal dryness. Vitamin D: Reduced 25(OH)D levels (<20 ng/mL in 50–60% of patients) increase osteoporosis and muscle weakness risks. Vitamin E: Deficiency (<5 µg/mL in 20–25% of patients) is linked to neurological disorders, including peripheral neuropathy. Vitamin K: Deficiency (in 15–20% of patients) elevates bleeding risk, evidenced by prolonged prothrombin time (>13 seconds).

These changes result from impaired fat emulsification due to asynchronous bile and pancreatic juice delivery, reducing vitamin bioavailability. Protease deficiency impairs protein digestion, leading to:

Creatorrhea: undigested muscle fibers in stool, observed in 60–70% of patients.

Hypoalbuminemia: serum albumin levels drop to 30–35 g/L (normal: 35–50 g/L) in 40% of patients, causing edema and immune dysfunction.

Protein-energy malnutrition: weight loss (5–10 kg over 6–12 months) occurs in 70% of patients, worsened by reduced caloric intake and appetite.

Amylase deficiency causes incomplete carbohydrate digestion, resulting in:
Amylorrhea: Starch residues in stool, detected in 20–30% of patients.

Energy deficit: Limited absorbable carbohydrates, especially in pancreatogenic diabetes (10–15% of cases), reduce glucose availability and increase fatigue.

Intestinal fermentation: Undigested carbohydrates ferment, causing flatulence and foul-smelling stool.

Gastrointestinal disorders, including chronic biliary-dependent pancreatitis, are significant risk factors for chronic periodontitis. Digestive impairments such as malabsorption, dysbiosis, and nutrient deficiencies contribute to systemic inflammation, adversely affecting periodontal tissues [1,3,5,16,19]. The need to elucidate the pathogenic mechanisms linking these conditions to periodontal destruction underscores the importance of developing interdisciplinary therapeutic approaches to enhance treatment efficacy and clinical outcomes [8,10,14,15,17].

The study aimed to investigate the correlation between digestive disorders in chronic biliary-dependent pancreatitis with exocrine insufficiency and clinical indicators of chronic periodontitis to clarify their interaction mechanisms.

Materials and methods

The study was conducted from 2022 to 2025 at the Republican specialized scientific-practical center of therapy and medical rehabilitation in collaboration with the department of propedeutics of therapeutic dentistry, Tashkent state dental institute (TSDI). All participants provided voluntary informed consent. The study analyzed medical data from two groups: 46 patients aged 25–69 years with chronic pancreatitis receiving inpatient treatment and a control group of 20 healthy individuals matched for age, sex, and living conditions.

Special attention was paid to patients' oral complaints, including pain, tongue burning, dryness, and bitter or sour taste. Medical histories recorded symptom frequency, duration, and association with exacerbations of acid-related diseases. Oral examinations followed a standardized protocol.

Periodontal disease diagnosis involved two stages: life history and disease history. Life history interviews identified risk factors, including dietary habits (e.g., vitamin deficiencies or excessive carbohydrates), heredity (family history of periodontal disease), comorbidities (e.g., diabetes, gastrointestinal disorders, immunodeficiency), and environmental conditions. Disease history assessed symptom onset (e.g., gingival bleeding, tooth mobility, pain), progression, external influences, and prior treatments (e.g., conservative, surgical, or physiotherapeutic), including their efficacy.

Data were processed using Microsoft Excel and IBM SPSS Statistics 23. Quantitative variables were expressed as means with standard errors (M±m), with Student's t-test applied to determine significance (P). Qualitative variables were presented as frequencies and percentages. Correlation coefficients were calculated using Kendall's Tau-b and Spearman's rank methods.

Results and Discussion

Among patients with generalized moderate periodontitis and chronic pancreatitis, the mean gingival bleeding score was 2,41±0,10 ($p < 0,05$), corresponding to bleeding severity of grades II–III (blood spots or interdental space filling upon probing).

Periodontal pocket depth averaged 4,20±0,17 ($p < 0,05$), corresponding to 4–6 mm or deeper pockets. Oral hygiene, assessed by the OHI-S index, averaged 2,33±0,09 ($p < 0,05$) across both groups, indicating poor to very poor hygiene.

The O'Leary gingivo-periodontal index revealed acute inflammation, ulceration, and spontaneous gingival bleeding, corresponding to pocket depths of grades II–III. The overall severity of inflammatory-destructive periodontal damage, per the O'Leary index, was 3,47±0,16 ($p < 0,05$).

Coprological Findings. Polyfecalia was observed in most patients with exocrine pancreatic insufficiency, with stool frequency increasing to 3–6 times daily, indicating diarrhea. Stool was mushy or watery due to undigested nutrients and high-water content.

Fatty, "greasy" stool, indicative of steatorrhea, was confirmed by microscopic detection of neutral fats, fatty acids, and soaps (steatorrhea types I–III). Quantitative analysis showed fatty acid excretion in 32,6% (+), 47,8% (++), and 19,6% (+++), and neutral fats in 54,3% (+) and 45,7% (++). Stool was glossy, grayish, and poorly flushing, reflecting significant fat malabsorption.

Digestible plant cells were altered in 87% (+) and 13% (++), while indigestible plant cells were observed in 8,7% (+), 60,9% (++), 21,7% (+++), and 8,7% (++++). Protease deficiency led to creatorrhea, with digestible muscle fibers in 50% (+) and 43,5% (++), and indigestible fibers in 50% (+) and 50% (++). Mucus was absent in 78,3% but present in 4,3% (++).

Carbohydrate digestion impairment caused amylorrhea, with starch in 41,3% (+), 47,8% (++), and 10,9% (+++). Leukocytes ranged from 12–16 per field in 2,2%, 2 in 8,7%, 3 in 8,7%, and were absent in 80,4%. Stool had a foul odor due to protein putrefaction and carbohydrate fermentation in 70–80% of patients. Mean fecal pH in the main group was 8,43±0,35.

In the control group, fatty acids and neutral fats were present in 100% (+), digestible plant cells in 90% (+) and 10% (++), and indigestible plant cells in 95% (+) and 5% (++). Digestible muscle fibers were in 95% (+) and 5% (++), and indigestible fibers in 75% (+) and 25% (++). Mucus, leukocytes, and starch were absent.

Correlation Analysis: The correlation between OHI-S and SBI was $r = 0,65$ ($p < 0,05$), indicating a moderately strong positive relationship. Worsening oral hygiene (higher OHI-S) was associated with increased gingival bleeding (SBI), reflecting pronounced periodontal inflammation due to poor hygiene.

Correlations between clinical periodontitis indices (OHI-S, SBI, PI, PMA, pocket depth), coprological parameters, and fecal pH ranged from weak to strong ($r = 0,21–0,78$, $p < 0,05$). Poor oral hygiene (OHI-S) showed weak positive correlations with pocket depth ($r = 0,37$), gingival bleeding (SBI, $r = 0,44$), and periodontitis severity (PI, $r = 0,52$), confirming plaque's role in disease progression [8,18,23]. However, a negative correlation with PMA ($r = -0,42$) suggests chronic periodontitis may involve less pronounced gingival inflammation, with plaque playing a secondary role.

Gingival bleeding (SBI) correlated moderately with pocket depth ($r = 0,44$) and PI ($r = 0,57$), confirming the inflammatory nature of the disease [2,17,18], but negatively with PMA ($r = -0,38$), possibly due to localized pathogenesis. Pocket depth correlated with PI ($r = 0,43$) and weakly negatively with PMA ($r = -0,37$), indicating destructive processes dominate over active inflammation in chronic stages.

Fecal pH (8,43±0,35) showed weak positive correlations with OHI-S (r = 0,36), SBI (r = 0,35), and pocket depth (r = 0,39), but negative with PI (r = -0,38), suggesting intestinal dysbiosis from exocrine insufficiency influences systemic inflammation and periodontal pathology.

Coprological analysis confirmed malabsorption: neutral fats correlated with fatty acids (r = 0,51), mucus (r = 0,41), leukocytes (r = 0,55), and starch (r = 0,50), indicating steatorrhea and intestinal inflammation. Indigestible plant fibers showed weak correlations with fats (r = 0,31–0,35), mucus (r = 0,24), leukocytes (r = 0,30), and starch (r = 0,26), reflecting enzymatic insufficiency. Digestible muscle fibers had weak negative correlations with leukocytes (r = -0,21) and starch (r = -0,26), suggesting partial proteolysis preservation. Indigestible muscle fibers correlated with mucus (r = 0,51) and leukocytes (r = 0,38), linking creatorrhea to intestinal inflammation [10,16,19]. A strong correlation between mucus and leukocytes (r = 0,78) highlighted active intestinal inflammation, potentially exacerbating periodontitis via pathogenic flora translocation [3,6,17,19].

Conclusion

Correlation analysis demonstrates a pathogenic link between chronic pancreatitis (CP) and chronic periodontitis. Weak to moderate positive correlations (r = 0,24–0,57) between oral hygiene (OHI-S), gingival bleeding (SBI), periodontitis index (PI), and pocket depth indicate that poor hygiene and gingival inflammation exacerbate periodontal tissue destruction. Systemic inflammation in CP likely aggravates these processes [1,3,6]. Negative correlations with gingival inflammation (PMA, r = -0,36 to -0,42) suggest chronic periodontitis involves predominant tissue destruction over active inflammation.

Coprological data and fecal pH underscore the impact of dysbiosis and malabsorption on periodontal pathology. Moderate correlations (r = 0,50–0,55) between neutral fats, mucus, leukocytes, and starch confirm digestive impairments, including steatorrhea and creatorrhea, linked to enzymatic insufficiency [1,4]. The strong correlation between mucus and leukocytes (r = 0,78) reflects significant intestinal inflammation, likely exacerbating periodontitis via pathogenic flora translocation [11,21,23]. Weak correlations (r = 0,24–0,51) with indigestible fibers, fats, and starch indicate systemic digestive dysfunction, while negative correlations (r = -0,21 to -0,26) with digestible fibers suggest partial digestion may mitigate inflammation.

These findings highlight the need for an interdisciplinary approach, integrating digestive correction (enzyme replacement therapy) and periodontal treatment (improved hygiene, inflammation reduction) for effective management of both conditions.

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