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CLINICAL AND BIOCHEMICAL FEATURES OF PATIENTS WITH DIFFERENT SERUM CALCIDIOL (25(OH)D) LEVELS

¹ Aripova Nargiza Nusratovna,

E-mail: aripovan755@gmail.com

ORCID ID: <https://orcid.org/0000-0001-7781-9207>

² Tagaeva Mavjuda Khalmatovna,

E-mail: tagaevamavjuda@gmail.com

ORCID: <https://orcid.org/0000-0002-2363-5215>

³ Mansurov Jaxongir Nozimjon o'g'li,

E-mail ID: jahongirmansurovofficial@gmail.com

ORCID ID: <https://orcid.org/0009-0009-0533-2681>

⁴ Muxitdinjonov Abdulloh Maxmudjon o'g'li,

E-mail: abdullohmukhiddinjonov@gmail.com

ORCID ID: <https://orcid.org/0009-0000-0572-8752>

⁵ Mamatxonov Umarxon Baxodirjon o'g'li,

E-mail: mamatxonovumarxon9550@gmail.com

ORCID ID: <https://orcid.org/0009-0002-3497-5300>

⁶ Abduxamidov Abdurasul Alisher o'g'li

E-mail: monarxgold@gmail.com

ORCID ID: <https://orcid.org/0009-0008-5547-4015>

^{1,2,3,4,5,6} Tashkent State Medical University, Republic of Uzbekistan.

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Abstract

Background. Chronic pancreatitis (CP) is a progressive inflammatory disease of the pancreas characterized by irreversible structural changes, impaired exocrine function, and disturbances in nutrient absorption. Exocrine pancreatic insufficiency contributes to impaired absorption of fats and fat-soluble vitamins, particularly vitamin D. Vitamin D deficiency may aggravate metabolic disturbances, bone mineral disorders, and clinical manifestations of chronic pancreatitis. However, the relationship between serum calcidiol [25-hydroxyvitamin D, 25(OH)D] levels, exocrine pancreatic function, biochemical parameters, and clinical characteristics of CP remains insufficiently studied.

Objective. To evaluate serum calcidiol [25(OH)D] levels in patients with chronic pancreatitis and to determine their relationship with biochemical parameters, calcium metabolism, pancreatic enzyme activity, fecal pancreatic elastase-1 levels, and clinical features of the disease.

Materials and Methods. The study included 94 patients with chronic pancreatitis and 15 apparently healthy individuals as a control group. Serum 25(OH)D concentrations were measured, and patients were classified according to vitamin D status into groups with normal levels, insufficiency, deficiency, and severe deficiency. Biochemical parameters, including serum calcium and α -amylase activity, were assessed. Exocrine pancreatic function was evaluated by measuring fecal pancreatic elastase-1 activity. Clinical manifestations, etiological forms of chronic pancreatitis, comorbid conditions, pancreatic morphological changes by ultrasonography, and bone mineral status by ultrasound densitometry were analyzed. Statistical analysis included comparative and correlation methods.

Results. Vitamin D insufficiency and deficiency were detected in the majority of patients with chronic pancreatitis. Decreased serum 25(OH)D levels were associated with disturbances in calcium metabolism, reduced serum calcium concentrations, and a higher prevalence of osteopenia and osteoporosis. A significant reduction in fecal pancreatic elastase-1 activity was observed in

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patients with lower 25(OH)D levels, indicating an association between vitamin D deficiency and exocrine pancreatic insufficiency. Ultrasonographic examination revealed a higher frequency of pancreatic parenchymal fibrosis, calcification, ductal changes, and structural abnormalities in patients with vitamin D deficiency. The most pronounced morphological changes were observed in patients with mixed-etiology chronic pancreatitis.

Conclusion. Serum calcidiol [25(OH)D] concentration is closely associated with clinical, biochemical, and functional characteristics of chronic pancreatitis. Assessment of vitamin D status may serve as an important diagnostic and prognostic tool for evaluating nutritional status, exocrine pancreatic function, and the risk of complications in patients with chronic pancreatitis. Regular monitoring and correction of vitamin D deficiency may improve the effectiveness of comprehensive management and rehabilitation of these patients.

Keywords: Chronic pancreatitis; vitamin D; calcidiol; 25-hydroxyvitamin D; exocrine pancreatic insufficiency; pancreatic elastase-1; calcium metabolism; osteopenia; osteoporosis.

Introduction

Relevance of the Study. Chronic pancreatitis (CP) remains one of the most significant and challenging problems in modern gastroenterology due to its high prevalence, progressive and recurrent course, and substantial medical and socioeconomic burden. The disease is characterized by progressive inflammatory and fibrotic changes in the pancreatic parenchyma, leading to irreversible impairment of both exocrine and endocrine pancreatic functions. As the disease progresses, exocrine pancreatic insufficiency (EPI) develops, resulting in impaired digestion and absorption of nutrients, particularly fats and fat-soluble vitamins (A, D, E, and K). Consequently, patients develop nutritional

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deficiencies, metabolic disturbances, compromised immune function, and a significant deterioration in quality of life [5, 6, 7].

In recent years, increasing attention has been directed toward the role of vitamin D metabolism in the pathogenesis of chronic pancreatitis. Beyond its classical function in maintaining calcium-phosphorus homeostasis, vitamin D acts as a pleiotropic hormone involved in the regulation of immune responses, inflammatory processes, cellular proliferation and differentiation, as well as antifibrotic mechanisms. Calcidiol (25-hydroxyvitamin D; 25(OH)D), the principal circulating metabolite of vitamin D, is considered the most reliable biomarker for assessing vitamin D status. Reduced serum 25(OH)D concentrations are associated not only with disturbances in calcium-phosphorus metabolism, secondary hyperparathyroidism, decreased bone mineral density, osteopenia, and osteoporosis, but also with unfavorable outcomes in several inflammatory and metabolic disorders [1, 15].

According to published studies, vitamin D deficiency is highly prevalent among patients with chronic pancreatitis, with reported rates ranging from 60% to 80%. Vitamin D deficiency adversely affects not only skeletal health but is also associated with muscle weakness, chronic fatigue, depressive symptoms, immune dysfunction, increased susceptibility to infections, and a higher risk of cardiovascular diseases [7, 8]. Furthermore, impaired intestinal absorption of dietary fats resulting from exocrine pancreatic insufficiency is considered one of the principal pathogenetic mechanisms underlying vitamin D deficiency in these patients. These findings emphasize the importance of routine assessment of vitamin D status and evaluation of its clinical significance in patients with chronic pancreatitis [6, 11].

Fecal pancreatic elastase-1 is currently recognized as one of the most reliable noninvasive biomarkers for assessing exocrine pancreatic function. Decreased fecal elastase-1 levels objectively indicate exocrine pancreatic insufficiency and reflect reduced pancreatic enzyme secretion, which contributes to impaired

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absorption of fat-soluble vitamins, particularly vitamin D [6, 12]. Therefore, investigating the relationship between serum 25(OH)D concentrations and fecal elastase-1 levels, as well as other coprological parameters, may provide valuable information regarding the severity of exocrine pancreatic dysfunction and the clinical course of chronic pancreatitis.

Moreover, evaluation of the association between serum calcidiol concentrations and biochemical markers of mineral metabolism, including calcium, phosphorus, alkaline phosphatase, parathyroid hormone, and related laboratory parameters, may facilitate early detection of metabolic bone disorders. Comprehensive assessment of these indicators may improve the understanding of the clinical consequences of exocrine pancreatic insufficiency, enable earlier diagnosis of skeletal complications, and contribute to the optimization of preventive and therapeutic strategies in patients with chronic pancreatitis.

Vitamin D deficiency is also highly prevalent among the population of Uzbekistan. Its development is influenced by multiple factors, including dietary habits, seasonal variations in sunlight exposure, impaired intestinal fat absorption, hepatobiliary diseases, and chronic gastrointestinal disorders [5, 6]. However, despite the growing body of international evidence, the relationship between serum 25(OH)D levels and biochemical parameters, calcium-phosphorus metabolism, fecal pancreatic elastase-1 activity, coprological findings, and the clinical and morphological characteristics of chronic pancreatitis has not been comprehensively investigated in the Uzbek population. In this context, determining serum calcidiol (25-hydroxyvitamin D; 25(OH)D) concentrations in patients with chronic pancreatitis and comprehensively evaluating their correlations with clinical, biochemical, and coprological parameters are of considerable scientific and practical importance. Such investigations may contribute to a better understanding of the pathogenetic mechanisms of chronic pancreatitis, facilitate the early identification of exocrine pancreatic insufficiency and mineral metabolism disorders, improve risk

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stratification for disease-related complications, and enhance the effectiveness of comprehensive treatment, rehabilitation, and long-term clinical management of patients with chronic pancreatitis.

Objective

To comprehensively evaluate serum calcidiol (25-hydroxyvitamin D; 25(OH)D) concentrations in patients with chronic pancreatitis, to determine their relationship with biochemical parameters reflecting calcium metabolism and pancreatic enzyme activity, as well as with indicators of exocrine pancreatic function, particularly fecal pancreatic elastase-1 activity, and to assess the correlations between vitamin D status, clinical manifestations, nutritional status, and the functional severity of chronic pancreatitis in order to clarify the role of vitamin D deficiency in the pathogenesis and clinical course of the disease.

Materials and Methods

The study included 94 patients diagnosed with chronic pancreatitis. The control group consisted of 15 apparently healthy individuals matched for age and sex. The diagnosis of chronic pancreatitis was established in all patients according to current clinical guidelines based on clinical presentation, laboratory findings, and imaging examinations.

Serum calcidiol (25-hydroxyvitamin D; 25(OH)D) concentrations were measured in all participants. According to vitamin D status, patients were stratified into four groups: those with normal serum 25(OH)D levels ($n = 22$), vitamin D insufficiency ($n = 17$), vitamin D deficiency ($n = 39$), and severe vitamin D deficiency ($n = 16$).

In addition to assessing vitamin D status, serum calcium concentration and α -amylase activity were determined as key biochemical parameters. Exocrine pancreatic function was evaluated by measuring fecal pancreatic elastase-1

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activity. Coprological examinations were also performed to assess the degree of digestive impairment.

A comprehensive clinical assessment was conducted for all patients, including evaluation of clinical symptoms, disease duration, etiological forms (biliary, idiopathic, and mixed), concomitant diseases, and the clinical characteristics of chronic pancreatitis. Morphological changes in the pancreas were assessed using abdominal ultrasonography, while bone mineral status was evaluated by quantitative ultrasound densitometry.

Statistical analysis was performed using contemporary biostatistical methods. Depending on the distribution of the data, both parametric and non-parametric statistical tests were applied. Differences between groups were considered statistically significant at $p < 0.05$. Correlation analysis was performed to evaluate the relationships between serum calcidiol (25-hydroxyvitamin D; 25(OH)D) concentrations and clinical, biochemical, coprological, and instrumental parameters.

Results and Discussion

In patients with chronic pancreatitis (CP), exocrine pancreatic insufficiency results in impaired intestinal absorption of dietary fats and fat-soluble vitamins, leading to nutritional deficiencies and metabolic disturbances. The risk of vitamin D deficiency in patients with CP is estimated to be approximately 60%, which in turn increases the risk of osteoporosis, muscle weakness, depression, and cardiovascular diseases [7].

As reported previously, vitamin D deficiency is also prevalent among the population of Uzbekistan. This condition may be associated with impaired intestinal absorption of fats and fat-soluble vitamins, gluten-sensitive enteropathy, chronic pancreatitis, or liver cirrhosis [5, 6]. Furthermore, increased catabolism of vitamin D or reduced synthesis of vitamin D and its metabolites may also contribute to its deficiency. Therefore, the present study aimed to

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determine serum vitamin D levels in patients with chronic pancreatitis and to evaluate their relationship with the clinical course of the disease.

The results demonstrated that serum 25-hydroxyvitamin D [25(OH)D] levels were within the normal range in 22 (23.4%) patients (32.57 ± 1.32 ng/mL), indicated vitamin D insufficiency in 17 (18.1%) patients (23.84 ± 0.83 ng/mL), vitamin D deficiency in 39 (41.5%) patients (15.55 ± 0.61 ng/mL), and severe vitamin D deficiency in 16 (17.0%) patients (7.00 ± 0.75 ng/mL) (see Table 3.2). In the healthy control group, the mean serum 25(OH)D concentration was 25.90 ± 1.53 ng/mL.

Analysis of serum 25(OH)D concentrations according to the etiology of chronic pancreatitis revealed a tendency toward lower values in patients with the biliary form (20.72 ± 1.07 ng/mL) compared with the healthy control group. In contrast, patients with idiopathic and mixed forms of CP exhibited significantly lower serum 25(OH)D concentrations, which were 1.63-fold ($P < 0.01$) and 2.18-fold ($P < 0.001$) lower than those of the control group, reaching 15.87 ± 3.39 ng/mL and 12.32 ± 1.28 ng/mL, respectively.

Further analysis showed that among patients with biliary chronic pancreatitis, normal serum 25(OH)D levels were observed in 19.4%, vitamin D insufficiency in 25.0%, vitamin D deficiency in 44.4%, and severe vitamin D deficiency in 11.2% of cases. In patients with idiopathic CP, 75% had vitamin D deficiency, whereas the remaining 25% had severe vitamin D deficiency. Among patients with the mixed form of CP, vitamin D deficiency and severe vitamin D deficiency were each identified in 50% of cases. These findings indicate that the most pronounced disturbances in vitamin D status were observed in patients with the mixed etiology of chronic pancreatitis.

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Table 1.1. Serum 25-Hydroxyvitamin D [25(OH)D] and Calcium Concentrations, and Fecal Pancreatic Elastase Activity in Patients with Chronic Pancreatitis (Mean $\pm$ SEM).

Groups	25-OH-D, ng/ml	Calcium, mmol/L	Fecal elastase-1, μ g/g
Apparently healthy individuals	25,90 $\pm$ 1,53	1,98 $\pm$ 0,03	208,27 $\pm$ 10,83
Patients with chronic pancreatitis according to serum 25-hydroxyvitamin D [25(OH)D] levels:			
Normal vitamin D status, n = 22	32,57 $\pm$ 1,32*	2,12 $\pm$ 0,08	254,45 $\pm$ 25,01 *
Vitamin D insufficiency, n = 17	23,84 $\pm$ 0,83	1,95 $\pm$ 0,07	166,37 $\pm$ 19,01 *
Vitamin D deficiency, n = 39	15,55 $\pm$ 0,61 *	1,75 $\pm$ 0,05 *	143,50 $\pm$ 9,39
Severe vitamin D deficiency, n = 16	7,00 $\pm$ 0,75 *	1,51 $\pm$ 0,07 *	116,37 $\pm$ 11,73 *
Patients with chronic pancreatitis according to etiological factors:			
Biliary form, n=73	20,72 $\pm$ 1,07	1,85 $\pm$ 0,03	176,69 $\pm$ 7,12
Idiopathic form, n=8	15,87 $\pm$ 3,39	1,67 $\pm$ 0,16	169,75 $\pm$ 25,88
Mixed form, n=13	12,32 $\pm$ 1,28	1,60 $\pm$ 0,03	125,00 $\pm$ 5,44

Note: Statistically significant differences compared with the apparently healthy control group ($p < 0.05$).

It should be noted that serum calcium concentrations in patients with chronic pancreatitis changed in parallel with serum 25-hydroxyvitamin D [25(OH)D] levels. Patients with normal 25(OH)D concentrations demonstrated a tendency toward higher serum calcium levels compared with the apparently healthy control group, whereas those with vitamin D insufficiency showed a tendency toward lower calcium concentrations. In patients with vitamin D deficiency and severe vitamin D deficiency, serum calcium levels were significantly reduced by 1.13-fold ($P < 0.05$) and 1.31-fold ($P < 0.05$), respectively. Analysis according to the etiology of chronic pancreatitis revealed a tendency toward lower serum calcium

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levels in patients with the biliary form (1.85 ± 0.03 mmol/L) compared with healthy controls, while significantly lower values were observed in the idiopathic and mixed forms, decreasing by 1.19-fold ($P < 0.05$) and 1.24-fold ($P < 0.05$) to 1.67 ± 0.16 mmol/L and 1.60 ± 0.03 mmol/L, respectively. These findings indicate that disturbances in calcium metabolism are most pronounced in patients with the mixed form of chronic pancreatitis.

Bone mineral status was also evaluated by quantitative ultrasound densitometry. The findings were consistent with the alterations observed in serum 25(OH)D and calcium concentrations, demonstrating the development of osteopenia and osteoporosis in patients with chronic pancreatitis. In the apparently healthy control group, ultrasound densitometry of the long bones revealed signs of osteopenia in 26.7% of individuals. Among patients with chronic pancreatitis, osteopenia was detected in 50 (53.2%) patients, whereas osteoporosis was diagnosed in 18 (19.1%) patients. In patients with normal serum 25(OH)D levels, osteopenia and osteoporosis were each observed in 9.1% of cases. Among patients with vitamin D insufficiency, these proportions were 35.3% and 23.5%, respectively. In the vitamin D deficiency group, osteopenia was detected in 25.6% and osteoporosis in 66.7% of patients, whereas among those with severe vitamin D deficiency, osteopenia and osteoporosis were identified in 25% and 75% of patients, respectively.

Analysis of ultrasound densitometry findings according to the etiology of chronic pancreatitis demonstrated osteopenia in 46.6% and osteoporosis in 19.2% of patients with biliary chronic pancreatitis. In the idiopathic form, osteopenia and osteoporosis were observed in 25% and 50% of patients, respectively. Notably, all patients (100%) with the mixed form of chronic pancreatitis exhibited osteopenia.

Measurement of serum α -amylase activity demonstrated values near the upper limit of the normal reference range, with a mean level of 121.17 ± 5.39 U/L, whereas the corresponding value in the apparently healthy control group was

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54.33 ± 3.84 U/L. When analyzed according to serum 25(OH)D status, α -amylase activity was 118.36 ± 9.82 U/L in patients with normal vitamin D levels, 104.57 ± 6.35 U/L in those with vitamin D insufficiency, 125.44 ± 9.45 U/L in patients with vitamin D deficiency, and 124.93 ± 14.38 U/L in those with severe vitamin D deficiency. These findings suggest that serum α -amylase activity is not directly associated with serum 25(OH)D concentrations.

It is well established that plasma α -amylase consists of pancreatic (P-type) and salivary (S-type) isoenzymes, with approximately 40% of total circulating α -amylase originating from the pancreas and 60% from the salivary glands. An increase exceeding twofold above the upper limit of normal is generally indicative of pancreatic injury. However, α -amylase activity usually declines within 2–3 days after the onset of acute pancreatic injury and subsequently returns to normal levels. Consequently, serum α -amylase has limited value as a diagnostic marker of chronic pancreatic dysfunction. The results of the present study are consistent with these observations.

It is well known that chronic pancreatitis is characterized by progressive impairment of exocrine pancreatic function, and fecal pancreatic elastase-1 is considered one of the most reliable laboratory markers for its assessment. In the present study, patients with normal serum 25(OH)D levels demonstrated fecal pancreatic elastase-1 activity that was 1.22-fold higher than that of the healthy control group ($P < 0.05$). In contrast, patients with vitamin D insufficiency, vitamin D deficiency, and severe vitamin D deficiency exhibited significantly lower fecal elastase-1 activity, decreasing by 1.25-fold ($P < 0.05$), 1.45-fold ($P < 0.01$), and 1.79-fold ($P < 0.001$), respectively. These findings suggest that vitamin D deficiency in patients with chronic pancreatitis may be associated with impaired exocrine pancreatic function. Analysis according to disease etiology further demonstrated significant reductions in fecal elastase-1 activity in the biliary, idiopathic, and mixed forms of chronic pancreatitis by 1.18-fold ($P < 0.05$), 1.23-fold ($P < 0.05$), and 1.67-fold ($P < 0.01$), respectively. The most

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pronounced impairment of exocrine pancreatic function was observed in patients with the mixed form of chronic pancreatitis.

Since disorders of lipid metabolism are commonly associated with obesity, body mass index (BMI) was also evaluated. The mean BMI in the apparently healthy control group was $26.82 \pm 1.03 \text{ kg/m}^2$. Patients with chronic pancreatitis demonstrated a statistically significant increase in BMI; however, these changes were not associated with serum 25(OH)D status or disease etiology. Specifically, the mean BMI values were 31.70 ± 0.75 , 31.48 ± 0.91 , 32.84 ± 0.62 , and $31.39 \pm 1.11 \text{ kg/m}^2$ in patients with normal vitamin D levels, vitamin D insufficiency, vitamin D deficiency, and severe vitamin D deficiency, respectively. According to disease etiology, BMI was 32.06 ± 0.46 , 31.42 ± 1.00 , and $32.01 \pm 1.35 \text{ kg/m}^2$ in patients with biliary, idiopathic, and mixed chronic pancreatitis, respectively. Previous studies have demonstrated that obesity is one of the major risk factors for vitamin D deficiency, as vitamin D is sequestered within adipose tissue, thereby reducing its bioavailability in the circulation.

In addition, we analyzed the relationship between the etiological factors of chronic pancreatitis and serum 25(OH)D concentrations but did not identify any significant association (see Table 1.2). Across all study groups, the principal factors associated with disease recurrence were alcohol consumption, cigarette smoking, and psychological stress.

Table 1.2. Major Factors Associated with Recurrence of Chronic Pancreatitis, %

Triggering Factors	Frequency of Vitamin D3 Deficiency, %			
	Normal vitamin D3 status	Vitamin D3 insufficiency	Vitamin D3 deficiency	Severe vitamin D3 deficiency
Alcohol consumption	45,0	76,0	31,0	31,0
Cigarette smoking	27,3	64,7	23,0	19,0
Smokeless tobacco use (naswar/nasal tobacco)	9,0	12,0	13,0	13,0
Psychological stress	81,1	100,0	100,0	100,0

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In the subsequent stage of the study, we analyzed comorbid conditions associated with chronic pancreatitis. The performed analysis demonstrated that hepatobiliary system disorders may be considered among the comorbidities most closely associated with serum 25-hydroxyvitamin D [25(OH)D] status (see Table 1.3). Specifically, chronic acalculous and chronic calculous cholecystitis were observed in 18.2% and 13.3% of patients with normal vitamin D status, respectively, whereas their prevalence increased to 30–47% among patients with vitamin D deficiency. Similar patterns were observed in patients with chronic liver diseases and those with post-cholecystectomy conditions, where the frequency of these comorbidities was also higher in the presence of vitamin D deficiency.

Table 1.3. Frequency of Comorbidities in Patients with Chronic Pancreatitis According to Serum 25-Hydroxyvitamin D [25(OH)D] Levels

Comorbidities	Frequency of Vitamin D3 Deficiency, %			
	Normal vitamin D3 status	Vitamin D3 insufficiency	Vitamin D3 deficiency	Severe vitamin D3 deficiency
Chronic acalculous cholecystitis	18,2	29,0	41,0	37,5
Chronic calculous cholecystitis	13,3	45,0	47,0	31,0
Post-cholecystectomy state	0,0	31,23	13,0	31,3
Chronic viral hepatitis	18,2	0,0	5,0	0,0
Reactive hepatitis	27,3	12,0	13,0	31,3
Steatohepatitis	18,2	64,7	33,3	13,0
Chronic gastritis	36,4	29,0	23,0	6,0
Peptic ulcer disease	9,0	12,0	0,0	0,0
Duodenal ulcer disease	9,0	12,0	0,0	0,0
Irritable bowel syndrome	0,0	0,0	5,0	0,0
Gastroesophageal reflux disease (GERD)	18,2	0,0	5,0	0,0
Enterocolitis	18,2	0,0	25,6	6,0
Asthenoneurotic syndrome	18,2	0,0	15,0	0,0
Ischemic heart disease	63,3	24,0	41,0	31,0
Hypertension	36,4	12,0	13,0	13,3

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However, no similar association was observed among gastrointestinal disorders. Comparable patterns were also identified in patients with cardiovascular diseases. Thus, among comorbid conditions, a potential relationship may exist between hepatobiliary system disorders and vitamin D [25(OH)D] deficiency.

In addition, we compared the clinical manifestations of chronic pancreatitis according to serum 25(OH)D concentrations (see Table 1.4). The analysis demonstrated that symptoms such as loose stool consistency, epigastric pain, girdle-like abdominal pain, postprandial pain, and pain in the Mayo–Robson area were common among the majority of patients with chronic pancreatitis; however, their occurrence was not associated with serum 25(OH)D levels.

Table 1.4. Association Between Clinical Complaints of Patients with Chronic Pancreatitis and Serum 25-Hydroxyvitamin D [25(OH)D] Levels

Clinical Manifestations	Vitamin D3 Status Categories			
	Normal vitamin D3 status	Vitamin D3 insufficiency	Vitamin D3 deficiency	Severe vitamin D3 deficiency
Loose stools	55,0	35,3	33,0	44,0
Epigastric pain	81,8	88,2	92,0	87,5
Girdle-like abdominal pain	55,0	75,0	67,0	38,0
Pain during meals	72,7	76,0	100,0	81,3
Pain in the Mayo–Robson area	72,7	76,0	90,0	62,5
General weakness	81,8	100,0	100,0	100,0
Constipation	45,0	53,0	44,0	62,5
Muscle pain	27,3	58,8	56,0	87,5
Irritability	81,1	100,0	100,0	100,0

However, the following clinical manifestations were detected in all patients with vitamin D insufficiency and deficiency: general weakness was observed in 100% of patients, constipation in 53–63%, muscle pain in 56–88%, and irritability in

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100% of cases. The frequency of these symptoms was significantly higher compared with patients who had normal serum 25(OH)D levels.

Ultrasonographic examination of the pancreas demonstrated that a progressive decrease in serum 25(OH)D concentrations was accompanied by an increased frequency of diffuse enhancement of pancreatic parenchymal echogenicity, preservation of the pancreatic contour, marked variability in the amplitude and duration of echo signals, and increased occurrence of pancreatic tissue calcification (see Table 1.5).

Specifically, diffuse increased echogenicity of the pancreatic parenchyma with preserved contours was detected in 40% of apparently healthy individuals and in 70–80% of patients with chronic pancreatitis; however, no statistically significant association with serum 25(OH)D levels was established.

The “stone bridge” ultrasound pattern was not observed in healthy individuals. Among patients with chronic pancreatitis and normal serum 25(OH)D levels, this sign was detected in 45% of cases. In contrast, among patients with vitamin D insufficiency, deficiency, and severe deficiency, this finding was observed in 100.0%, 64.1%, and 62.5% of cases, respectively. Compared with patients with normal vitamin D status, the frequency of this ultrasound sign was significantly higher by 2.22-fold ($P < 0.001$), 1.42-fold ($P < 0.05$), and 1.40-fold ($P < 0.05$), respectively.

Medium- and high-density echo signals with uneven distribution against a normal background were detected in 26.7% of apparently healthy individuals. Among patients with chronic pancreatitis and normal serum 25(OH)D levels, this ultrasound feature was observed twice as frequently (54%, $P < 0.01$). Its prevalence increased further in patients with vitamin D insufficiency, deficiency, and severe deficiency, reaching 82.2%, 74.6%, and 87.5%, respectively.

Heterogeneous distribution of echo signals with alternating dense and cystic areas was observed in 45% of patients with chronic pancreatitis who had normal

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vitamin D levels. This finding was detected in 60–63% of patients with vitamin D insufficiency and deficiency.

Marked variability in the amplitude and duration of echo signals was observed in 20% of apparently healthy individuals, whereas among patients with chronic pancreatitis and normal serum 25(OH)D levels, this feature was detected in 54.5% of cases. The frequency of these ultrasound changes increased progressively among patients with vitamin D insufficiency, deficiency, and severe deficiency, reaching 76.5%, 77.0%, and 93.8%, respectively, in patients with deficiency and severe deficiency.

Pancreatic enlargement was observed in all patient groups; however, its prevalence was highest among patients with vitamin D deficiency and severe deficiency, reaching 80–90%. Pancreatic tissue calcification was detected in 31.8% of patients with normal serum 25(OH)D levels, whereas in groups with vitamin D deficiency and severe deficiency, it was observed in 48.7% and 50.0% of patients, respectively.

Table 1.5. Ultrasonographic Findings of the Pancreas in Patients with Chronic Pancreatitis According to Changes in Serum 25-Hydroxyvitamin D [25(OH)D] Levels (%)

Parameters	Apparently healthy control group , n=15	Distribution of Patients with Chronic Pancreatitis According to Serum 25(OH)D Status, n = 94			
		Normal, n=22	insufficiency , n=17	deficiency, n=39	Severe vitamin D3 deficiency, n=16
Diffuse increase in pancreatic parenchymal echogenicity with preserved pancreatic contour	40,0%	72,7%	70,6%	71,8%	81,3%
“Stone bridge” appearance	0,0%	45,0%	100,0%	64,1%	62,5%
Medium- and high-density echo signals with heterogeneous distribution against a normal background	26,7%	54,0%	88,2%	74,4%	87,5%

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Heterogeneous distribution of echo signals with alternating dense and cystic areas	-	45,5%	58,8%	61,5%	62,5%
Marked variability in the amplitude and duration of echo signals	20,0%	54,5%	76,5%	77,0%	93,8%
Pancreatic enlargement	-	77,3%	88,3%	89,7%	81,3%
Pancreatic tissue calcification	-	31,8%	23,5%	48,7%	50,0%
Detection of pancreatic duct calculi	-	18,2%	5,9%	7,7%	37,5%
Presence of pancreatic cysts	-	9,1%	0,0%	5,1%	12,5%
Dilatation of the pancreatic duct (>2.5 mm)	-	0,0%	23,5%	7,7%	12,5%
Increased pancreatic tissue density	33,3%	45,5%	41,2%	49,0%	43,8%

The presence of pancreatic duct calculi was detected in 18.2% of patients with normal serum vitamin D levels, whereas it was observed in 37.5% of patients with severe vitamin D deficiency. The occurrence of pancreatic cysts and pancreatic duct dilatation (>2.5 mm) was also associated with serum 25(OH)D concentrations and demonstrated an increasing trend among patients with vitamin D insufficiency and deficiency. In contrast, increased pancreatic tissue density was not associated with serum 25(OH)D levels. This ultrasound feature was observed in 33.3% of apparently healthy individuals and in 40–49% of patients with chronic pancreatitis.

It should be emphasized that pancreatic enlargement and pancreatic tissue calcification were predominantly characteristic of the mixed form of chronic pancreatitis. In some patients with biliary chronic pancreatitis, enlargement of the pancreatic gland, dilatation of pancreatic ducts, the presence of calculi and cysts, as well as thickening of pancreatic tissue were also observed. Thus, pancreatic ultrasonography demonstrated the presence of various morphological changes, the development of which was directly associated with serum 25(OH)D concentrations. In patients with vitamin D deficiency, ultrasonographic

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examination revealed pancreatic parenchymal fibrosis and calcification, while stool analysis demonstrated features of steatorrhea and impaired absorption of fat-soluble vitamins.

Conclusion. The results of the present study demonstrated that reduced serum calcidiol (25-hydroxyvitamin D; 25(OH)D) levels in patients with chronic pancreatitis are associated with the clinical course of the disease, biochemical alterations, and impaired exocrine pancreatic function. Vitamin D insufficiency and deficiency were detected in the majority of patients with chronic pancreatitis. A decrease in serum 25(OH)D concentrations was accompanied by disturbances in calcium metabolism, hypocalcemia, reduced bone mineral density, osteopenia, and osteoporosis.

A progressive decline in serum calcidiol levels was associated with decreased fecal pancreatic elastase-1 activity, indicating a relationship between vitamin D deficiency and exocrine pancreatic insufficiency. Patients with reduced 25(OH)D concentrations demonstrated a higher frequency of morphological abnormalities on pancreatic ultrasonography, including pancreatic parenchymal fibrosis, calcification, and structural changes of the pancreatic ducts. The most pronounced alterations were observed in patients with the mixed etiological form of chronic pancreatitis.

Therefore, assessment of serum calcidiol (25(OH)D) levels may serve as an important diagnostic and prognostic marker for evaluating nutritional status, exocrine pancreatic function, and the risk of complications in patients with chronic pancreatitis. Regular monitoring of vitamin D status and correction of identified deficiencies may contribute to improving the effectiveness of comprehensive management and rehabilitation strategies in patients with chronic pancreatitis.

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