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# Vitamin D Deficiency in Patients with Chronic Pancreatitis and its Association with Disease Severity: A Hospital-Based Cross-Sectional Study.

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## Abstract

***Background and Aims:*** Vitamin D deficiency is common but under-evaluated in chronic pancreatitis (CP). We estimated the prevalence of hypovitaminosis D in CP and examined its association with endocrine and exocrine insufficiency and with disease severity graded by endoscopic ultrasound (EUS). ***Methods:*** This hospital-based cross-sectional study enrolled 63 adults with established CP at a tertiary-care gastroenterology unit in southern India between August 2024 and July 2026. Serum 25-hydroxyvitamin D (25OHD) was measured by liquid chromatography–tandem mass spectrometry. Disease severity was graded using the Rosemont EUS classification; exocrine insufficiency was assessed clinically (steatorrhoea and low body mass index) and endocrine insufficiency by the presence of diabetes mellitus. Associations were tested using the chi-square test, with $p<0.05$ considered significant. ***Results:*** Mean serum 25OHD was 32.73 ± 16.57 nmol/L (range 15.0–75.5). Hypovitaminosis D was near-universal (92.1%): 28 patients (44.4%) had deficiency and 30 (47.6%) insufficiency, while only 7.9% had normal levels. Vitamin D status was significantly associated with body mass index category (p=0.019), endocrine insufficiency (p=0.015), exocrine insufficiency (p=0.039), duration of hospital stay (p=0.016) and the presence of complications (p=0.025), but not with age (p=0.117), sex (p=0.266), alcohol intake (p=0.223), grouped EUS features (p=0.252) or mortality (p=0.736). ***Conclusions:*** Hypovitaminosis D is highly prevalent in CP and tracks with functional pancreatic insufficiency and overall disease burden rather than with demographic factors, supporting routine 25OHD screening and structured nutritional surveillance in all patients with CP.

**Keywords:** Chronic pancreatitis; Vitamin D deficiency; 25-hydroxyvitamin D; Exocrine pancreatic insufficiency; Rosemont classification; Endoscopic ultrasound.



## INTRODUCTION

Chronic pancreatitis (CP) is a progressive fibro-inflammatory disorder of the pancreas that culminates in irreversible destruction of the exocrine and endocrine parenchyma, producing maldigestion, pancreatogenic (type 3c) diabetes, chronic pain and a substantial nutritional burden [1]. Because impaired delivery of pancreatic lipase leads to fat maldigestion, patients with CP are particularly susceptible to deficiencies of the fat-soluble vitamins A, D, E and K [2,3]. Among these, vitamin D has attracted increasing attention. Hypovitaminosis D is already widespread in the general population—driven by limited ultraviolet exposure, adiposity, ageing and skin pigmentation [4]—and in CP these population-level determinants are compounded by disease-specific mechanisms, including exocrine pancreatic insufficiency (EPI), systemic inflammation and altered hepatic and renal hydroxylation of vitamin D [5-7].

Beyond its classical skeletal role, vitamin D deficiency in CP has been linked to secondary hyperparathyroidism, low bone mineral density and an increased fracture risk [8-10], and may also modulate pancreatic inflammation and β-cell function [11]. A systematic review confirmed that hypovitaminosis D is common across CP cohorts, although reported estimates vary widely with assay method, latitude, cut-off definitions and the assessment of EPI [12]. However, few studies have simultaneously related vitamin D status to markers of exocrine and endocrine function and to a standardised imaging measure of structural severity such as the Rosemont endoscopic ultrasound (EUS) classification [13,14], particularly in Indian patients, among whom baseline vitamin D insufficiency is already highly prevalent [15].

We therefore conducted a hospital-based cross-sectional study to (i) estimate the prevalence of vitamin D deficiency and insufficiency in adults with CP, and (ii) examine the relationship between vitamin D status and endocrine and exocrine insufficiency and the Rosemont EUS criteria.

## MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of Medical Gastroenterology of a tertiary-care teaching hospital in Bengaluru, southern India, between August 2024 and July 2026. Consecutive adults (≥18 years, either sex) with an established diagnosis of CP—based on characteristic morphology on contrast-enhanced computed tomography or magnetic resonance imaging, EUS (Rosemont criteria) or endoscopic retrograde cholangiopancreatography (Cambridge criteria)—were included. Patients receiving hormone replacement therapy, vitamin D or calcium supplementation, and those with hepatic or cholestatic disease, inflammatory bowel disease or cystic fibrosis that could independently affect vitamin D metabolism, were excluded. Sixty-three consecutive eligible patients were enrolled, and data were complete for all participants. The study was approved by the Institutional Ethics Committee of Ramaiah Medical College (MSRMC/EC/PG-24/08-2024, dated 30 August 2024) and conducted in accordance with the Declaration of Helsinki; written informed consent was obtained from every participant.

### Vitamin D estimation

Serum 25-hydroxyvitamin D (25OHD; the sum of 25-OH-D3 and 25-OH-D2) was measured in all participants by a certified isotope-dilution liquid chromatography–tandem mass spectrometry (LC-MS/MS) method (accuracy and precision within 7.5%; linearity 3.0–300.0 nmol/L). Results were expressed in nmol/L (nmol/L = 2.5 × ng/mL). Vitamin D status was categorised as deficiency (<25 nmol/L; <10 ng/mL), severe insufficiency (25–<50 nmol/L; 10–<20 ng/mL), mild insufficiency (50–<62.5 nmol/L; 20–<25 ng/mL) and normal (≥62.5 nmol/L; ≥25 ng/mL). The cut-off applied for the normal category (≥25 ng/mL) is lower than the 75 nmol/L (30 ng/mL) sufficiency threshold used in some frameworks [4]; this is acknowledged in the limitations.

### Assessment of disease severity and function

Structural disease severity was graded on EUS using the Rosemont classification, which weights major and minor parenchymal and ductal features into the categories "consistent with", "suggestive of", "indeterminate for" or "normal" for CP [13]. Exocrine pancreatic insufficiency was defined clinically by the presence of steatorrhoea and low body mass index (BMI), supported by faecal elastase-1 where available [16]; endocrine insufficiency was defined by the presence of diabetes mellitus.

### Statistical analysis

Data were analysed using SPSS version 26 (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as frequencies and proportions and continuous variables as mean ± standard deviation. Associations between vitamin D status and categorical variables were tested using the chi-square test (with Fisher's exact test or Yates' correction where applicable). A p-value <0.05 was considered statistically significant. The sample size of 63 was derived from an expected vitamin D deficiency prevalence of 37.5% in CP, with 95% confidence and 12% absolute precision.

## RESULTS

### Baseline characteristics

Sixty-three patients with CP were studied (Table 1). The cohort was predominantly male (50/63, 79.4%) and middle-aged, with the 41–50-year group being the largest (41.3%). Alcohol-related CP predominated overall (57.1%) and among men (66.0%), whereas non-alcoholic/idiopathic disease was more common in women. Most patients were overweight (71.4%). Diabetes mellitus was the commonest comorbidity (65.1%), followed by dyslipidaemia (27.0%) and hypertension (22.2%).

**Table 1. Baseline demographic and clinical characteristics of the study population (n = 63)**

| Characteristic | Category | n (%) |
|---|---|---|
| Sex | Male | 50 (79.4) |
| | Female | 13 (20.6) |
| Age group (years) | 18–20 | 1 (1.6) |
| | 21–30 | 4 (6.3) |
| | 31–40 | 16 (25.4) |
| | 41–50 | 26 (41.3) |
| | 51–60 | 16 (25.4) |
| BMI category | Normal weight | 12 (19.0) |
| | Overweight | 45 (71.4) |
| | Obese | 6 (9.5) |
| Aetiology | Alcohol-related | 36 (57.1) |
| | Non-alcoholic/idiopathic | 27 (42.9) |
| Comorbidity | Diabetes mellitus | 41 (65.1) |
| | Dyslipidaemia | 17 (27.0) |
| | Hypertension | 14 (22.2) |

BMI, body mass index. Percentages may not total 100 owing to rounding.

**Vitamin D status**

Serum 25OHD ranged from 15.0 to 75.5 nmol/L (mean 32.73 ± 16.57 nmol/L). Hypovitaminosis D was near-universal, affecting 92.1% of the cohort (Table 2): 28 patients (44.4%) were deficient and 30 (47.6%) insufficient (severe insufficiency 39.7%, mild insufficiency 7.9%), while only 5 patients (7.9%) had normal levels.

**Table 2. Distribution of serum 25-hydroxyvitamin D status (n = 63)**

| Vitamin D status | 25OHD cut-off | n (%) |
|---|---|---|
| Deficiency | <25 nmol/L (<10 ng/mL) | 28 (44.4) |
| Severe insufficiency | 25–<50 nmol/L (10–<20 ng/mL) | 25 (39.7) |
| Mild insufficiency | 50–<62.5 nmol/L (20–<25 ng/mL) | 5 (7.9) |
| Normal | ≥62.5 nmol/L (≥25 ng/mL) | 5 (7.9) |
| Hypovitaminosis D (total) | <62.5 nmol/L | 58 (92.1) |

Mean serum 25OHD 32.73 ± 16.57 nmol/L (range 15.0–75.5). 25OHD, 25-hydroxyvitamin D; nmol/L = 2.5 × ng/mL. Percentages may not total 100 owing to rounding. The cut-off for the normal category (≥62.5 nmol/L / 25 ng/mL) is lower than the 75 nmol/L sufficiency convention; see Limitations.

**Correlates of vitamin D status**

Vitamin D status differed significantly across BMI categories (p=0.019); all obese participants had severe insufficiency. It was also significantly associated with endocrine insufficiency (p=0.015)—none of the patients with endocrine insufficiency had normal 25OHD, compared with 22.7% of those without—and with exocrine insufficiency (p=0.039); among patients with exocrine insufficiency, deficiency predominated (23/45, 51.1%). Poorer vitamin D status was further associated with a hospital stay exceeding five days (p=0.016) and with the presence of complications (p=0.025). In contrast, vitamin D status was not significantly associated with age (p=0.117), sex (p=0.266), alcohol intake (p=0.223), grouped major/minor EUS features (p=0.252) or mortality (p=0.736) (Table 3).

**Table 3. Association of vitamin D status with clinical and functional variables (chi-square test)**

| Variable | $\chi^2$ | df | p-value |
|---|---|---|---|
| BMI category | 15.151 | 6 | 0.019 |
| Endocrine insufficiency (diabetes) | 10.430 | 3 | 0.015 |
| Exocrine insufficiency | 8.379 | 3 | 0.039 |
| Duration of hospital stay (>5 days) | 10.324 | 3 | 0.016 |
| Presence of complications | 9.324 | 3 | 0.025 |
| Age group | 17.959 | 12 | 0.117 |
| Sex | 3.962 | 3 | 0.266 |
| Alcohol intake | 4.387 | 3 | 0.223 |
| EUS features (major/minor)* | — | — | 0.252 |
| Mortality | 1.270 | 3 | 0.736 |

*The EUS analysis compared vitamin D categories across major/minor feature occurrences (126 occurrences in 63 patients) rather than patient-level Rosemont categories; only the p-value was reported. BMI, body mass index; EUS, endoscopic ultrasound; df, degrees of freedom.

## DISCUSSION

In this hospital-based cohort of adults with CP, hypovitaminosis D was near-universal, affecting 92.1% of patients, and low vitamin D status was significantly associated with abnormal BMI, endocrine and exocrine insufficiency, longer hospital stay and complications, but not with demographic factors or grouped EUS features. These findings position vitamin D deficiency as a marker of the overall burden of pancreatic dysfunction and nutritional compromise rather than as an isolated laboratory abnormality.

The very high prevalence we observed is consistent with a systematic review reporting a substantial pooled burden of vitamin D deficiency and insufficiency across CP cohorts [12], and with an Indian series in which 25OHD fell with increasing morphological severity [14]. It exceeds the 62.5% deficiency reported by Min et al. in a cohort enriched for EPI

[2] and the lower prevalence in some outpatient series [3], differences plausibly attributable to case mix, disease severity, nutritional intake and assay methodology. The near-universal deficiency argues against viewing vitamin D as a stand-alone parameter: in CP, low 25OHD reflects reduced oral intake, fat maldigestion, loss of pancreatic function and altered body composition operating together [17,18].

The significant association with exocrine insufficiency is biologically coherent, as inadequate lipase delivery impairs micellar absorption of fat-soluble vitamins; a similar relationship was reported by Min et al. [2], although a large multinational cohort did not find a significant association [19], likely reflecting differences in how EPI was defined. The association with endocrine insufficiency, in which no patient with diabetes had normal 25OHD, is in keeping with progressive functional loss and shared nutritional determinants [20]. Longer hospitalisation and complications likely identify patients with more advanced disease and poorer nutritional reserve [18,21], with a substantial burden of under-recognised skeletal and muscle morbidity in this population [10,22]. The lack of association with grouped EUS features may reflect our analysis of feature occurrences rather than patient-level Rosemont categories, and the recognised imperfect concordance between structural and functional impairment [13].

Our findings carry practical implications. Because low vitamin D was common across all demographic subgroups, testing should not be restricted by age, sex or alcohol history. In line with ESPEN guidance, 25OHD measurement is best embedded within a broader nutritional review that includes assessment of weight change, enzyme replacement adequacy, muscle mass and bone health, with targeted repletion and re-testing after 8–12 weeks [4,23]. Normal serum calcium and phosphorus do not exclude vitamin D deficiency, as secondary hyperparathyroidism may maintain calcium within the reference range [9,24].

This study has limitations. The cross-sectional design precludes causal inference, and single-centre recruitment may limit generalisability. The number of women was small, exocrine insufficiency was defined clinically rather than by faecal elastase in all patients, and endocrine insufficiency could not be reliably separated from type 2 diabetes. EUS findings were analysed as feature occurrences rather than patient-level Rosemont strata. Several determinants of vitamin D—season, sun exposure, dietary intake, skin pigmentation, physical activity and parathyroid hormone—were not measured, and no multivariable analysis was performed; the significant associations should therefore be regarded as hypothesis-generating. Finally, the cut-off used for the normal category (≥62.5 nmol/L / 25 ng/mL) is lower than the 75 nmol/L sufficiency threshold applied in several frameworks, which limits direct comparability with cohorts categorised at 75 nmol/L.

## CONCLUSION

Vitamin D deficiency and insufficiency are highly prevalent in chronic pancreatitis and are significantly associated with functional pancreatic insufficiency and greater disease severity rather than with demographic characteristics. These findings support routine measurement of serum 25-hydroxyvitamin D and structured nutritional surveillance—coupled with optimisation of pancreatic enzyme replacement therapy and targeted repletion—as an integral part of the comprehensive management of chronic pancreatitis [23,25]. Larger, multicentre, longitudinal studies using objective functional testing and patient-level Rosemont grading are needed to clarify whether low vitamin D status predicts disease progression or merely reflects greater nutritional and functional impairment.

## DECLARATIONS

Ethics approval and consent: Approved by the Institutional Ethics Committee of Ramaiah Medical College (MSRMC/EC/PG-24/08-2024, dated 30 August 2024); written informed consent was obtained from all participants.

**Conflicts of interest:** None declared.

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