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Low serum vitamin D levels as a risk factor for peptic ulcer development in Iraqi patients

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ABSTRACT

The pathophysiology of peptic ulcer (PU) involves a multifactorial interplay between gastric acid secretion, mucosal barrier integrity, *Helicobacter pylori* (*H. pylori*) infection, and the modulatory effects of various nutrients on the ulcer onset and severity. This cross-sectional study aimed at investigating whether PU pathophysiology and severity are associated with serum vitamin D levels. A total of 110 individuals aged 20–65 years were enrolled. Blood samples were collected between July and October 2024 from patients attending the Morgan Teaching Hospital and the Imam Sadiq Hospital in the Babil Province, Iraq. Following upper gastrointestinal endoscopy, participants were stratified into two groups: those without PU (N=40) and those diagnosed with PU (N=70). Each participant provided 5 mL of venous blood for vitamin D quantification and biochemical profiling. Among the PU patients, females were more prevalent (55.7%). Body mass index (BMI) was lower in the PU group. Vitamin D deficiency was significantly more frequent in PU patients (75.7%) compared to controls (32.5%), with mean serum vitamin D levels markedly reduced (14.61 ± 6.74 ng/mL *vs.* 26.22 ± 9.00 ng/mL). *H. pylori* infection was more common in PU patients (58.6%). Additionally, PU patients exhibited elevated lipid profile parameters. No statistically significant sex-based differences were observed within either group. In conclusion, serum vitamin D levels appear to be a strong predictor of PU severity, suggesting a potential role for vitamin D status in PU pathogenesis and clinical management.

1. Introduction

Peptic ulcers (PUs), defined as open sores occurring in the stomach or duodenum, continue to pose a substantial global health burden. Their

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pathogenesis involves a multifaceted interaction among gastric acid secretion, mucosal barrier integrity, and *Helicobacter pylori* (*H. pylori*) infection. Recent studies have also highlighted the potential influence of various nutrients and biomarkers on the development and severity of PUs[1].

Vitamin D, traditionally recognized for its role in calcium homeostasis and bone metabolism, is increasingly acknowledged for its immunomodulatory functions. It is synthesized in the skin upon exposure to ultraviolet radiation and subsequently converted into its active form, calcitriol, *via* hepatic and renal hydroxylation. Calcitriol exerts its biological effects through the vitamin D receptor, which is expressed in multiple tissues, including the gastrointestinal tract. Emerging evidence suggests that vitamin D deficiency may elevate the risk of gastrointestinal disorders such as inflammatory bowel disease and PUs. One proposed mechanism by which vitamin D may influence the PU pathogenesis is through the modulation of the immune response[2]. Vitamin D regulates the activity of immune cells including macrophages and T lymphocytes, which are integral to inflammation control and mucosal repair. This is particularly relevant in the context of *H. pylori* infection; a primary etiological factor in PU disease. Studies have demonstrated that vitamin D can inhibit *H. pylori* growth and attenuate its virulence.

On the other end, vitamin D deficiency has been associated with increased mucosal inflammation, delayed ulcer healing, and heightened risk of complications. It may contribute to PU development and exacerbation by impairing immune defences and compromising mucosal integrity[3]. The present study aimed at evaluating the serum vitamin D levels as a predictive marker associated with increased risk and severity of PUs.

2. Methodology

This cross-sectional study included 110 patients (both male and female), aged 20–65 years, diagnosed with PUs. Samples were collected between July and October 2024 from the Morgan Teaching Hospital and the Imam Sadiq Hospital in the Babil Province of Iraq. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. Verbal and analytical consent was obtained from all participants prior to sample collection. The study protocol, participant information sheet, and consent form were reviewed and approved by the local ethics committee under document number 2541, dated 11 May 2023.

Each participant underwent upper gastrointestinal endoscopy for diagnosis and evaluation of PUs. During the procedure, an endoscope was inserted orally in order to visualize the stomach and the duodenum. Venous blood samples (5 mL) were collected post-endoscopy for biochemical screening. Blood was placed in gel tubes, centrifuged at 3,000 rpm for 5 min in order to separate the serum, which was then aliquoted into Eppendorf tubes and stored at -20°C until the vitamin D quantification by enzyme-linked immunosorbent assay (ELISA), using a kit supplied by Bioassay Technology Laboratory Company.

Statistical analyses were performed using IBM SPSS version 28. Nominal data were reported as frequencies and percentages, with comparisons made using the chi-square test. Numerical data were expressed as mean ± standard deviation (SD), and group comparisons were conducted using independent *t*-tests, analysis of variance (ANOVA), and *post hoc* tests. A p-value <0.05 was considered statistically significant.

3. Results and Discussion

Table 1 presents the demographic and clinicopathological characteristics of participants with and without a PU. No significant difference was observed in age distribution between PU and non-PU groups (p=0.970). Females comprised a higher proportion of PU patients (55.7%) than males (44.3%), although this difference was not statistically significant (p=0.113). Body mass index (BMI) was significantly lower in PU patients (p<0.001). Notably, 72.9% of the PU patients were underweight or of normal weight, compared to 30.0% in the non-PU group, whereas overweight and obesity were more prevalent among individuals without PU (70.0%) than those with PU

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Table 1. Demographic and clinicopathological characteristics of all studied participants with and without peptic ulcer (PU). Other abbreviations used: HDL, high-density lipoprotein; LDL, low-density lipoprotein; SD, standard deviation; VLDL, very-low-density lipoprotein.

Demographic and clinical characteristics		Groups		
		PU	Without PU	*p*-value (chi-square)
Age	mean ± SD (in years)	40.54 ± 16.19	37.3 ± 11.81	0.270
	≤ 40 years (in N (%))	44 (62.9%)	25 (62.5%)	0.970 (0.001)
	> 40 years (in N (%))	26 (37.1%)	15 (37.5%)	
Sex	male (in N (%))	31 (44.3%)	24 (60.0%)	0.113 (2.51)
	female (in N (%))	39 (55.7%)	16 (40.0%)	
Body max index (BMI)	mean ± SD (in kg/m^2)	21.84 ± 3.39	26.61 ± 3.86	<0.001
	<25 kg/m^2 (in N (%))	51 (72.9%)	12 (30.0%)	<0.001 (19.11)
	>25 kg/m^2 (in N (%))	19 (27.1%)	28 (70.0%)	
Vitamin D status	<20 ng/mL (in N (%))	53 (75.7%)	13 (32.5%)	<0.001 (19.81)
	≥20 ng/mL (in N (%))	17 (24.3%)	27 (67.5%)	
H. pylori	Ve+ (in N (%))	41 (58.6%)	10 (25.0%)	<0.001 (11.54)
	Ve- (in N (%))	29 (41.4%)	30 (75.0%)	
Type of PU	none (in N (%))	0 (0%)	40 (100%)	0.137 (3.97)
	duodenal ulcer (in N (%))	18 (25.7%)	0 (0%)	
	gastric ulcer (in N (%))	31 (44.3%)	0 (0%)	
	duodenal and gastric (in N (%))	21 (30.0%)	0 (0%)	
Cholesterol levels	mean ± SD (in mg/dL)	179.84 ± 44.74	157.96 ± 33.59	0.008
Triglyceride levels	mean ± SD (in mg/dL)	184.87 ± 70.63	147.71 ± 52.6	0.005
LDL-cholesterol levels	mean ± SD (in mg/dL)	110.32 ± 39.08	91.78 ± 31.2	0.012
VLDL-cholesterol levels	mean ± SD (in mg/dL)	36.97 ± 14.13	29.54 ± 10.54	0.004
HDL-cholesterol levels	mean ± SD (in mg/dL)	32.55 ± 6.92	36.64 ± 10.96	0.018
Vitamin D levels among studied categories				
Vitamin D levels	mean ± SD (in ng/mL)	14.61 ± 6.74	26.22 ± 9.00	<0.001
Age	≤40 years	17.56 ± 6.34	27.39 ± 10.38	<0.001
	>40 years	9.61 ± 3.83	24.29 ± 5.89	
***p*-value**		<0.001	0.177	
Sex	male	15.51 ± 7.2	26.3 ± 9.09	<0.001
	female	13.89 ± 6.34	26.11 ± 9.16	
***p*-value**		0.381	0.938	
H. pylori	Ve+	12.65 ± 5.65	24.37 ± 12.5	<0.001
	Ve-	17.37 ± 7.26	26.84 ± 7.67	
***p*-value**		0.010	0.366	
Vitamin D status	<20 ng/mL	11.65 ± 4.6	16.77 ± 1.54	<0.001
	≥20 ng/mL	23.8 ± 2.84	30.78 ± 7.36	
***p*-value**		<0.001	<0.001	

(27.1%) (p<0.001).

Vitamin D deficiency, defined as serum levels <20 ng/mL, was significantly more prevalent in PU patients (75.7%) than in controls (32.5%) (p<0.001).

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Mean serum vitamin D levels were markedly lower in PU patients (14.61 ± 6.74 ng/mL) compared to non-PU individuals (26.22 ± 9.00 ng/mL) (p<0.001). *H. pylori* infection was significantly more common among PU patients (58.6%) than in controls (25.0%) (p<0.001). Among PU patients, 44.3% had gastric ulcers, 25.7% had duodenal ulcers, and 30.0% had both types, though these differences were not statistically significant (p=0.137).

The undertaken lipid profile analysis revealed significantly elevated levels of total cholesterol (p=0.008), triglycerides (p=0.005), low-density lipoprotein cholesterol (LDL-cholesterol; p=0.012), and very-low-density lipoprotein cholesterol (VLDL-cholesterol; p=0.004) in PU patients. High-density lipoprotein cholesterol (HDL-cholesterol) levels were significantly lower in PU patients compared to controls (p=0.018).

Vitamin D levels were significantly reduced in PU patients aged ≤40 years (9.61 ± 3.83 ng/mL) compared to those aged >40 years (17.56 ± 6.34 ng/mL) (p<0.001). No significant age-related differences were observed in the non-PU group (27.39 ± 10.38 *vs.* 24.29 ± 5.89 ng/mL). Sex-based comparisons revealed no significant differences in vitamin D levels within either group: PU males (15.51 ± 7.20 ng/mL) *vs.* PU females (13.89 ± 6.34 ng/mL), and non-PU males (26.30 ± 9.09 ng/mL) *vs.* non-PU females (26.11 ± 9.16 ng/mL).

Among the PU patients, those positive for *H. pylori* had significantly lower vitamin D levels (12.65 ± 5.65 ng/mL) compared to *H. pylori*-negative individuals (17.37 ± 7.26 ng/mL; p=0.010). No significant difference in vitamin D levels was observed between *H. pylori*-positive and -negative individuals in the non-PU group (24.37 ± 12.50 *vs.* 26.84 ± 7.67 ng/mL).

These findings reinforce the association between vitamin D deficiency and PU, particularly in the context of *H. pylori* infection. Vitamin D deficiency may impair immune responses, increase susceptibility to *H. pylori*, and influence treatment outcomes. Prior studies have corroborated this relationship, suggesting that adequate vitamin D levels may confer protection against *H. pylori* infection[4,5].

Age-stratified analyses have revealed that vitamin D deficiency was more pronounced in PU patients under 40 years of age, which is consistent with previous findings[6]. Although recent trends suggest an increasing prevalence of PUs among women, this study has found no significant sex-based differences in *H. pylori* infection rates, thereby contrasting with reports indicating higher infection rates among men[7]. Additionally, the PU patients in this study exhibited elevated cholesterol levels, in disagreement with findings that suggest reduced cholesterol levels in PU patients[8].

4. Conclusion

PU patients – particularly those who are younger, vitamin D-deficient, and *H. pylori*-positive – appear to be at increased risk of disease severity. Age, triglycerides, VLDL-cholesterol, and BMI were inversely associated with vitamin D levels. These findings support the role of vitamin D as a predictive biomarker for PU severity.

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Conflicts of interest

None exist.

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