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Frequency of dyslipidemia and associated factors among patients with *Helicobacter pylori* infection attending an academic referral center in northern Iran

Farzane Rouhibakhsh[1], Navid Pourzardasht[2], Fatemeh Kheradkish[3], Paridokht Karimian[1] *

[1] Razi Clinical Research Development Unit, Razi Hospital, Guilan University of Medical Sciences, Rasht, Iran
[2] Department of Biochemistry and Biophysics, School of Medicine, Guilan University of Medical Sciences, Rasht, Iran
[3] Shahid Beheshti Hospital, Guilan University of Medical Sciences, Bandar Anzali, Iran

Corresponding author:
Paridokht Karimian
Razi Clinical Research Development Unit, Razi Hospital, Guilan University of Medical Sciences, Rasht, Iran
E-mail: Paridokhtkarimian@yahoo.com

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Abstract

Helicobacter pylori infection is one of the most common chronic bacterial infections worldwide and, beyond mucosal gastroduodenal pathology, has been linked to systemic extra-gastrointestinal manifestations including altered serum lipid homeostasis and accelerated atherogenesis. Dyslipidemia is a major modifiable risk factor for ischemic heart and cerebrovascular disease. To determine the frequency of dyslipidemia and analyze its association with demographic, clinical and lifestyle variables among patients with H. pylori infection attending the Razi Educational-Medical Center in Rasht, Northern Iran. This cross-sectional analytical study was conducted on 200 patients with confirmed H. pylori infection (established by histopathology of endoscopic gastric biopsies) during 2021-2022. Demographic data, anthropometric parameters body mass index (BMI), lifestyle variables (smoking), chronic comorbidities and fasting serum lipid profile (total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)) were extracted from clinical records and the laboratory server. Statistical analysis was performed with SPSS version 26 using the Chi-square test, independent t-test and Fisher's exact test at a significance level of $P<0.05$. Of the 200 patients studied, 137 (68.5%) had dyslipidemia and 63 (31.5%) did not. The mean age was 59.72 ± 16.84 years; 87 patients (43.5%) were male and 113 (56.5%) were female. Mean TC, TG, HDL-C and LDL-C were 185.08 ± 28.41, 136.06 ± 46.11, 48.79 ± 8.41 and 118.15 ± 24.36 mg/dL, respectively. Applying the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) criteria, elevated LDL-C was present in 118 patients (95% CI: 61.9%-74.6%). Age, sex, occupation, BMI, smoking and comorbidities were not significantly associated with dyslipidemia ($P>0.05$), whereas a positive family history of dyslipidemia showed a significant association ($P=0.017$). Dyslipidemia was highly prevalent (68.5%) among patients with H. pylori infection in this Northern Iranian referral center, supporting a possible association between the infection and altered lipid metabolism. Classical demographic factors were not independently associated, highlighting the possible direct pro-atherogenic role of chronic H. pylori infection.

Keywords: Helicobacter pylori, Dyslipidemia, Hyperlipidemias, Cholesterol, Cardiovascular diseases, Northern Iran

1. Introduction

Helicobacter pylori infection (originally named Campylobacter pyloridis), discovered by Marshall and Warren in the early 1980s, is one of the most common chronic bacterial infections in the world (1, 2). H. pylori is a Gram-negative, microaerophilic, motile bacterium of approximately 3.5 x 0.5 microns with a spiral shape; it is not part of the normal flora and causes chronic, diffuse inflammation of the gastric mucosa. Although it grows slowly on blood-containing and Skirrow agar media, it penetrates into the mucus by means of its 2-7 sheathed, unipolar flagella (3). Under adverse conditions, the bacterium converts to a coccoid form to preserve its viability in the environment (4). This pathogen is catalase-, oxidase- and urease-positive, and urease constitutes more than 5% of its total protein (5). The *ureI gene* also maintains bacterial homeostasis in the acidic environment by regulating the pH-dependent urea channel (6). Given the role of this bacterium in chronic gastritis, peptic ulcer disease and malignancies, the World Health Organization (WHO) classified it as a definite class I carcinogen in 1994 (1).

Epidemiologically, more than half of the world population is infected with this bacterium, although its geographical prevalence is heterogeneous (7-11). The prevalence is higher in developing countries and in lower socioeconomic strata (12, 13). The global burden of this infection in 2015 was estimated at more than 4.4 billion people, with the highest rates recorded in Nigeria, Portugal, Estonia, Kazakhstan and Pakistan and the lowest in Switzerland (12, 13). The structured review by Mezmale et al. also indicated a high prevalence of infection in regions including Russia, Jordan, Iran, China, Canada and Latin America (9). In Turkey, Uyanikoglu et al. reported a prevalence of H. pylori above 70% in dyspeptic patients (14), a finding confirmed by Ozen et al. in children and by Ozaydin et al. in national screenings (above 75-80%) (12, 15, 16). Soylu et al. likewise observed high infection rates in gastric biopsies of men with dyspepsia (17). Similar patterns have been reported in Nepal (4). In Iran, the prevalence has been reported between 88.4% and 93% in those aged 35-55 years, and between 30.6% and 47.5% in younger age groups in Ardabil, Yazd and Tehran (18).

Bacterial transmission occurs mainly through person-to-person and environmental routes (19). Person-to-person transmission predominates in developed countries, whereas contaminated water and food play the prominent role in developing countries (20, 21). Consumption of raw vegetables irrigated with contaminated water and swimming in untreated water increase the probability of infection (4, 11). Oral-oral, fecal-oral and gastro-oral routes are the principal pathways of interpersonal transmission, and isolation of the organism from dental plaque and saliva demonstrates the ease of intrafamilial transmission (19, 22-25). Diagnosis is performed by invasive methods requiring endoscopy and biopsy (rapid urease test, histology, culture, and polymerase chain reaction (PCR)) and non-invasive methods (serology, stool antigen and urea breath test); combining these methods improves diagnostic accuracy (26-28).

The pathogenesis of the bacterium relies on ammonia production by urease, which has direct cytotoxic effects on the epithelium (29-31). Protease and phospholipase enzymes also destroy the mucus barrier and facilitate acid penetration (4). The vacuolating cytotoxin A (VacA) toxin induces the formation of pores in the mitochondrial membrane and stimulates apoptosis of host cells (32). The cytotoxin-associated gene A (CagA) oncoprotein, injected through the type IV secretion system, disrupts cell signaling, autophagy and the cytoskeleton and promotes dysplasia (33-35). In addition, the lipopolysaccharide (LPS) antigen diverts humoral immunity by mimicking Lewis blood group antigens, and the blood group antigen-binding adhesin (BabA) intensifies tissue destruction by binding to epithelial cells (4, 36).

The persistent presence of the organism induces dysbiosis and chronic systemic inflammation (37-39). Interaction of bacterial components with macrophages induces the release of interleukin-1β (IL-1β) and interleukin-8 (IL-8) and the production of reactive oxygen species (ROS) (4, 40). CagA-positive strains induce lipid peroxidation through stimulation of IL-8 secretion and neutrophil recruitment, activation of CD11b/CD18 and oxidative stress (41-43). Standard therapy consists of a combination of proton pump inhibitors and antibiotics (clarithromycin, amoxicillin or metronidazole), but drug resistance has created a major therapeutic challenge (44-46).

Chronic H. pylori inflammation plays a key role in the development of dyslipidemia (reduction of high-density lipoprotein cholesterol (HDL-C) and elevation of low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and total cholesterol (TC)) (47). Alongside traditional risk factors such as age, genetics, hypertension, diabetes, obesity, physical inactivity and smoking (48), which lead to angina, myocardial infarction, cerebrovascular accidents, coronary artery disease and

vascular insufficiency (49), the role of chronic bacterial infections in atherogenesis has gained increasing importance. Dyslipidemia is often asymptomatic and therefore diagnosed late (50). H. pylori accelerates the atherosclerotic process by inducing low-grade systemic inflammation, endothelial dysfunction, hyperhomocysteinemia and oxidative stress (51, 52). Furthermore, CagA-positive strains facilitate the atherogenic phenotype through autoimmune responses to heat-shock proteins, platelet aggregation, structural vascular changes and alteration of the lipid profile (53). Given the association between H. pylori-related dyslipidemia and cardiovascular events, the present study was undertaken to determine the frequency of dyslipidemia in patients with H. pylori infection attending Razi Hospital in Rasht, Iran.

2. Methods

2.1. Study design and setting

This was an observational, cross-sectional analytical study designed to evaluate the prevalence and relational structure of dyslipidemia among patients with H. pylori infection. The study population comprised all patients with H. pylori infection who presented to the endoscopy and pathology units of the Razi Educational-Medical Center in Rasht, Guilan Province, northern Iran, during the years 2021 (1400) and 2022 (1401) for evaluation of gastrointestinal complaints.

2.2. Ethical considerations

The study protocol was approved by the Ethics Committee in Biomedical Research of Guilan University of Medical Sciences under the official code **IR.GUMS.REC.1402.395**. The study was conducted in accordance with the ethical standards of the Declaration of Helsinki (1964) and its later amendments. Patient information was handled confidentially; data were collected anonymously and de-identified, no personal names or identifiers were entered into the study checklists, and the data were used exclusively for the scientific purposes of this study.

2.3. Study population and eligibility criteria

Inclusion criteria: (1) patients attending the Razi Educational-Medical Center of Rasht during 2021-2022; (2) definitive evidence of active H. pylori infection based on standard histopathological examination (Giemsa or hematoxylin-eosin staining) of endoscopic gastric mucosal biopsies; and (3) availability of complete fasting lipid profile data in the patient record.

Exclusion criteria: (1) missing or unrecorded lipid parameters, anthropometric measurements or key clinical variables in the hospital archiving system; (2) a known history of severe familial (genetic) dyslipidemia or use of hypolipidemic agents (statins, fibrates, ezetimibe or niacin) prior to testing; and (3) end-stage liver disease (advanced cirrhosis), acute or advanced chronic renal failure, severe untreated thyroid disorders (overt clinical hypothyroidism) and long-term high-dose systemic corticosteroid therapy.

2.4. Sample size and sampling strategy

Sampling was performed as a census of all eligible patients presenting to Razi Hospital of Rasht during the defined time interval. In total, the clinical and biochemical records of 200 patients (n=200) with definitively confirmed infection who fulfilled the eligibility criteria were entered into the final analysis.

2.5. Data collection and laboratory measurements

Data on age (years), sex (male/female), occupational group (self-employed, homemaker, employee, farmer, worker and unemployed), geographic place of residence (counties and areas of Guilan Province and outside the province), cigarette smoking (active smoker vs. non-smoker), history of chronic underlying diseases (diabetes, hypertension, heart disease) and body mass index (BMI, weight in kilograms divided by the square of height in meters) were extracted from clinical records. H. pylori infection was diagnosed based on the microscopic report of a pathologist on antrum and corpus biopsies obtained during upper gastrointestinal endoscopy. Serum biochemical assays were performed on venous blood samples obtained after a minimum 10-12 hour overnight fast in the central hospital laboratory using an automated analyzer (Hitachi 912, Roche, Germany) with standard enzymatic methods. Variables included TC, TG, HDL-C and LDL-C, all expressed in mg/dL. Low-density lipoprotein cholesterol was calculated by the Friedewald formula (LDL-C = TC - HDL-C - TG/5).

2.6. Operational definition of dyslipidemia

Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) guidelines as the presence of one or more of the following: total cholesterol ≥200 mg/dL (hypercholesterolemia), triglycerides ≥150 mg/dL (hypertriglyceridemia), LDL-C ≥130 mg/dL (elevated LDL), HDL-C <40 mg/dL in men or <50 mg/dL in women (low HDL). Patients meeting any of these cut-

off values were classified as having dyslipidemia.

2.7. Statistical analysis

The extracted data were coded and entered into SPSS software version 26 (IBM Corp., Armonk, NY, USA). Normality of the distribution of continuous variables was assessed with the Kolmogorov-Smirnov test. Quantitative data were described using mean, standard deviation (SD), median, interquartile range (IQR) and range (minimum and maximum). Qualitative data were reported as absolute frequencies (counts) and relative frequencies (percentages). Between-group comparisons of quantitative variables were performed using the independent samples t-test, and associations between categorical demographic and background variables and dyslipidemia status were examined using the chi-square test; where more than 20% of the cells of a contingency table had an expected frequency below 5, Fisher's exact test was applied. The type I error rate (α) was set at 0.05, and all tests were two-tailed.

3. Results

In this study, 200 patients with H. pylori infection attending Razi Hospital of Rasht during 2021 and 2022 were evaluated. The mean age of the patients was 59.72 ± 16.84 years (range: 14 to 96 years), and 68.5% (n=137) were female. The mean BMI was 25.02 ±3.02 kg/m2, and 75.5% (n=151) were non-smokers. Regarding residence, 49.5% (n=99) lived in urban areas and 50.5% (n=101) in rural areas.

According to **Table 1**, the mean TC of the patients was 185.08 ± 28.41 mg/dL (minimum 128, maximum 249), the mean TG was 136.06 ± 46.11 mg/dL (minimum 65, maximum 403), the mean HDL was 48.79 ± 8.41 mg/dL (minimum 35, maximum 64) and the mean LDL was 118.15 ± 24.36 mg/dL (minimum 59, maximum 159).

Of the 200 patients evaluated, 137 (68.5%) had dyslipidemia and 63 (31.5%) had no dyslipidemia.

In examining the association of demographic variables with the frequency of dyslipidemia **(Table 2)**, dyslipidemia was present in 62.6% (n=62) of patients younger than 61 years and in 74.3% (n=75) of patients aged 61 years or older, although this difference was not statistically significant (P=0.077). The frequency of dyslipidemia was almost identical in women (68.1%) and men (69%) (P=0.901). The associations of dyslipidemia with place of residence (P=0.581), occupation (P=0.736), BMI (P=0.648) and cigarette smoking (P=0.364) were also not significant. However, a significant association was observed between a family history of dyslipidemia and the occurrence of dyslipidemia in patients with H. pylori infection (P=0.017); dyslipidemia was present in 72.7% of patients with a hereditary family history and in 100% of those with a non-hereditary family history.

4. Discussion

Helicobacter pylori infection is recognized as one of the most common, persistent and challenging chronic bacterial infections worldwide, affecting more than half of the global population. Whereas the attention of researchers and clinicians was formerly focused mainly on the local consequences of this microorganism in the upper gastrointestinal tract, such as chronic gastritis, peptic ulcer disease, mucosa-associated lymphoid tissue (MALT) lymphoma and gastric adenocarcinoma, accumulating evidence in recent decades has shown that this pathogen acts beyond the gastric mucosa and induces numerous systemic manifestations. Among the most prominent consequences are its adverse effects on metabolic homeostasis, particularly disturbance of the lipid profile and the development of dyslipidemia. Dyslipidemia, as one of the principal prerequisites in the pathogenesis of atherosclerosis and ischemic heart disease, imposes a heavy economic and healthcare burden on health systems annually.

The findings of the present study showed that among 200 patients with H. pylori infection attending the Razi Educational-Medical Center in Rasht, 68.5% (137 patients) had dyslipidemia. This high prevalence provides clear evidence of a considerable association between colonization by this bacterium in the gastrointestinal tract and the development of unfavorable changes in serum lipid and lipoprotein concentrations. More detailed analysis of the quantitative variables showed that the mean concentrations of TC, TG, HDL-C and LDL-C in the evaluated patients were 185.08, 136.06, 48.79 and 118.15 mg/dL, respectively. This lipid pattern indicates a metabolic shift towards a pro-atherogenic phenotype that may expose patients harboring chronic infection to premature vascular risk.

Several biological mechanisms have been proposed to explain this association. Chronic H. pylori infection provokes a persistent low-grade systemic inflammatory response at the cellular and tissue level. Colonization of gastric epithelial cells and activation of nuclear factor kappa B (NF-κB) lead to local and subsequently systemic secretion of high amounts of pro-inflammatory cytokines, including IL-1β, interleukin-6 (IL-6),

Table 1. Lipid profile parameters of patients with Helicobacter pylori infection (n=200)

Parameter	Mean ± SD (mg/dL)	Median (IQR)	Min-Max	Cutoff (mg/dL)	Abnormal, n (%)	Normal, n (%)
Total cholesterol (TC)	185.08 ± 28.41	184 (162-208)	128-249	≥ 200	47 (23.5)	153 (76.5)
Triglycerides (TG)	136.06 ± 46.11	128 (104-159)	65-403	≥ 150	72 (36.0)	128 (64.0)
HDL-C	48.79 ± 8.41	48 (41-56)	35-64	< 40 (M) / < 50 (F)	104 (52.0)	96 (48.0)
LDL-C	118.15 ± 24.36	116 (98-137)	59-159	≥ 130	34 (17.0)	166 (83.0)

Table 2. Association of demographic and clinical factors with dyslipidemia frequency in patients with *Helicobacter pylori* infection (n=200)

Variable	Dyslipidemia positive, n (%)	Dyslipidemia negative, n (%)	Total, n (%)	P-value
Age				
< 61 years	62 (62.6)	37 (37.4)	99 (49.5)	0.077
≥ 61 years	75 (74.3)	26 (25.7)	101 (50.5)	
Sex				
Male	60 (69.0)	27 (31.0)	87 (43.5)	0.901
Female	77 (68.1)	36 (31.9)	113 (56.5)	
Place of residence				
Urban	66 (66.7)	33 (33.3)	99 (49.5)	0.581
Rural	71 (70.3)	30 (29.7)	101 (50.5)	
Occupation				
Employed / self-employed	55 (68.8)	25 (31.2)	80 (40.0)	0.736
Homemaker	74 (67.9)	35 (32.1)	109 (54.5)	
Other (farmer, worker, unemployed)	8 (72.7)	3 (27.3)	11 (5.5)	
BMI (kg/m2)				
< 25	70 (70.0)	30 (30.0)	100 (50.0)	0.648
≥ 25	67 (67.0)	33 (33.0)	100 (50.0)	
Cigarette smoking				
Non-smoker	106 (70.2)	45 (29.8)	151 (75.5)	0.364
Smoker	31 (63.3)	18 (36.7)	49 (24.5)	
Family history of dyslipidemia				
Positive (hereditary)	110 (72.7)	30 (27.3)	110 (55.0)	0.017*
Negative (non-hereditary)	13 (100.0)	0 (0.0)	13 (6.5)	
Total	137 (68.5)	63 (31.5)	200 (100)	-

*Chi-square test; *statistically significant (P<0.05). Categorical variables were compared with the Chi-square test or Fisher's exact test where expected frequencies were below 5; quantitative variables (age, BMI) were compared with the independent samples t-test.*

interleukin-8 (IL-8) and tumor necrosis factor-alpha (TNF-α). Increased circulating levels of these inflammatory mediators disturb metabolic signaling pathways in key tissues such as the liver and adipose tissue. For example, TNF-alpha, by directly inhibiting lipoprotein lipase (LPL) activity in the adipose vasculature, impairs the hydrolysis of chylomicron- and very-low-density lipoprotein (VLDL)-rich triglycerides and thereby delays the clearance of lipids from the blood. Moreover, these cytokines stimulate lipolysis in peripheral adipose tissue, resulting in increased influx of free fatty acids into the liver, stimulation of de novo fatty acid synthesis, and enhanced secretion of VLDL particles and, consequently, LDL.

In addition, oxidative stress arising from the accumulation of reactive oxygen species (ROS) generated by activated neutrophils and macrophages causes oxidation of LDL particles (ox-LDL). Ox-LDL particles, by binding to scavenger receptors on macrophages, accelerate foam cell formation and the development of atherosclerotic plaques. On the other hand, chronic inflammation reduces hepatic synthesis of apolipoprotein A-I, which, together with impaired reverse cholesterol transport, results in a decrease in serum HDL-C levels. Therefore, our data indicating elevated atherogenic indices in infected patients are fully consistent with this complex pathophysiological network.

In a comparative evaluation with the international literature, the 68.5% prevalence of dyslipidemia in our study corresponds remarkably closely to the findings of Hashim et al., who reported the frequency of lipid abnormalities as 68.39% (53). Similarly, our findings are concordant with extensive studies conducted in Ethiopia; in the study by Abdu et al., the prevalence of dyslipidemia in H. pylori-positive patients was 87.2%, and TC, TG and LDL-C were significantly higher in these patients (51). In the study by Nigatie et al., 71.8% of positive individuals had dyslipidemia (47). Although the figures reported in African studies are slightly higher than our findings from northern Iran, these minor differences may be attributed to factors such as differences in genetic background, nutritional status and variation in virulent bacterial strains, particularly the frequency of strains harboring the cagPAI pathogenicity island and the CagA virulence factor, which induce more intense inflammatory responses.

Our findings regarding changes in the individual components of the lipid profile also agree with the results of Al-Fawaeir et al. in Jordan (54) and the large-scale study of Shimamoto et al. in Japan (55); both studies confirmed that patients with chronic H. pylori infection have markedly higher TC and LDL-C levels and lower HDL-C levels. Nevertheless, a review of the literature reveals some heterogeneity and contradictory reports. For example, Kim et al. in South Korea (56), while confirming a significant increase in TC and LDL-C, found no statistically significant difference in TG and HDL-C levels between the groups. Heterogeneity is also evident among Iranian studies: whereas Hariri et al. reported a significant increase in all TC, TG and LDL-C parameters (57), which fully corroborates our findings, and Haeri et al. in Qom confirmed an increase in LDL in positive individuals (58), they, unlike our study, found no significant change in TG and HDL levels. These methodological and epidemiological discrepancies across the literature can be attributed to numerous variables, including differences in the laboratory criteria used to define dyslipidemia, differences in the diagnostic techniques for H. pylori (serological antibody assay, immunoglobulin G (IgG) serology, urea breath test or histopathological examination of endoscopic biopsies, each with different sensitivity and specificity), inconsistent control of statin use or lipid-lowering supplements, and marked differences in lifestyle, regional dietary patterns and the physical activity level of participants.

The statistical analysis of demographic variables in the present study showed that factors such as age, sex, BMI, occupation, coexisting underlying diseases and history of cigarette smoking were not significantly associated with the frequency of dyslipidemia. This finding carries an important interpretive weight: the absence of a significant dependence on classical risk indicators such as age and BMI suggests that dyslipidemia in these patients may be an independent biological consequence of the direct inflammatory effects of the bacterium itself, rather than merely a consequence of overweight or age-dependent variables. However, it should be noted that the relative homogeneity of the samples collected in a single-center facility may also have moderated these statistical differences, and the absence of statistical significance should not be interpreted as a lack of clinical importance of these variables at the population level.

5. Limitations

This study has several strengths, including an adequate sample size, a precise histopathological gold standard for confirming H. pylori infection, careful measurement of fasting lipid parameters, and detailed evaluation of multiple demographic and clinical covariates. The main limitations of our study include its cross-sectional design, which precludes definitive causal inference; therefore, it cannot be stated with certainty whether H. pylori is a direct cause of dyslipidemia or merely a marker of co-existence. In addition, the failure to genotype bacterial strains with different invasive capacities, the single-center design, and the lack of data on dietary habits and physical activity are further limitations.

6. Conclusion

The results of this study showed that dyslipidemia is highly prevalent (68.5%) among patients with H. pylori infection. These findings emphasize a possible as-

sociation between H. pylori infection and alterations in lipid metabolism. However, statistical analyses demonstrated that factors such as age, sex, body mass index, underlying diseases, occupation and cigarette smoking had no statistically significant effect on the frequency of dyslipidemia in these patients.

To further examine the association between H. pylori and dyslipidemia, studies in other geographical regions and with different population groups are recommended. Such work could help to clarify the influence of cultural, genetic and environmental factors. Future research may also focus on the biological mechanisms by which H. pylori affects lipid metabolism, including the role of systemic inflammation, oxidative stress and changes in the gut microbiome.

7. Declarations

7.1. Ethical approval and consent to participate

The study protocol was approved by the Ethics Committee of Guilan University of Medical Sciences (code: **IR.GUMS.REC.1402.395**) and was conducted in accordance with the Declaration of Helsinki. As a retrospective record-based study, the requirement for individual informed consent was waived by the Ethics Committee.

7.2. Availability of data and materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

7.3. Conflict of interest

The authors declare that they have no competing interests.

7.4. Financial support

This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors. This research was self-funded.

7.5. Author's contributions

FR, NP, FK, and PK conceptualized and designed the study. FR and FK collected the clinical and laboratory data. NP and PK performed the statistical analyses and data interpretation. FR, NP, and FK drafted the initial manuscript. PK supervised the project and critically revised the manuscript for important intellectual content. All authors have read and approved the final version of the manuscript.

7.6. Artificial intelligence statement

The authors used an artificial intelligence (AI)-assisted language tool solely to improve the readability and language of the manuscript. All scientific content, study design, data collection, data analysis, interpretation of the findings, and final editorial decisions were performed entirely by the authors. The authors have reviewed and approved the final manuscript and accept full responsibility for its accuracy and integrity.

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