The Egyptian Journal of Hospital Medicine (January 2023) Vol. 90, Page 914-919

Prevalence of H. Pylori Infection among Patients with Acute Myocardial Infarction

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ABSTRACT

Background: Infection with Helicobacter pylori (HP) is the most prevalent infection worldwide, particularly in underdeveloped nations. It is reported to be associated with many extra gastrointestinal manifestations.

Objective: The current work aimed to study the prevalence of HP infection among patients with acute myocardial infarction (AMI). **Patients and Methods:** This cross-sectional study included STEMI and NSTEMI patients admitted with AMI in the CCU of the Cardiology Department at Benha University Hospital, Egypt.

Results: The prevalence of HP among the studied patients was 72%. Our study showed that those with positive HP Ig-G had considerably lower triglycerides than those with negative HP Ig-G. No significant differences were observed regarding hemoglobin, TLC, platelets, serum creatinine, blood urea, ALT, AST, total cholesterol, HDL, and LDL. In patients with positive HP Ig-G, significant negative correlations were observed between ejection fraction (EF) and blood urea, serum creatinine, and AST.

Conclusion: In this study, HP is related to the incidence of MI. The elimination of HP prevents the onset of CAD and associated consequences.

Keywords: Prevalence, HP, Infection, Acute Myocardial Infarction.

INTRODUCTION

Currently, the most prevalent risk factors for coronary heart disease (CHD) caused by atherosclerosis are dyslipidemia, diabetes, smoking, and hypertension [1]. For many years, monocytes and macrophages have been recognized as components of atheromatous plaque. The involvement of an active inflammatory process in the etiology of atherosclerosis in coronary circulation is developing rapidly [2].

The Helicobacter pylori (HP) infection is the most prevalent infection worldwide, especially in underdeveloped nations. Other gastrointestinal symptoms have been linked to HP infection, according to **Yokota et al.** [3]. Several signs point to a connection between persistent infections, atherosclerosis, and vascular disease. The prevalence of inflammation as a cardiovascular risk factor and the existence of HP in extra-digestive illnesses motivated researchers to study the role of HP in atherosclerosis progression. There was a significant incidence of active HP infection among patients with myocardial infarction, which may be a modifiable risk factor for upper gastrointestinal bleeding [4].

Given that HP infection has emerged as a potentially modifiable risk factor for UGIB and early termination of DAPT in patients with AMI, we believed that examining the incidence of HP infection in a group of patients with AMI would be of considerable interest. This might be considered a step forward in proving the feasibility of Hp testing as part of the standard therapeutic therapy for AMI patients. This may stimulate the continued use of DAPT, so optimizing its advantages [5].The present work aimed to study the prevalence of HP Infection among patients with acute myocardial infarction.

PATIENTS AND METHODS

This cross-sectional study included STEMI and NSTEMI patients admitted with AMI in the CCU of the Cardiology Department at Benha University Hospital, Egypt.

The study was done after being approved by the institutional ethical committee and informed consent was obtained from all participants included.

The inclusion criteria were patients with age ≥18 years old, who were admitted with acute myocardial infarction (STEMI and NSTEMI).

The exclusion criteria included severe renal failure (creatinine >2 mg/dL), anemia, hepatic failure, neurological or endocrine diseases, previous HP infection treatment, and malignancies.

All participants were subjected to full history taking including [Age, gender, BMI, HTN history, Stroke, Cardiac diseases and cardiac failure, DM, smoking (Patients who had quit smoking for fewer than 10 years were categorized as smokers), CCU admission, and renal insufficiency].

Clinical examination including [Biochemical parameters, thorough Complete blood picture, ESR, CRP, Total cholesterol, LDL, HDL, TG, liver, and kidney function tests, ECG, and ECHO].

Trained research assistants gathered demographic information and the use of medicines having known effects on atherogenesis.

STEMI was defined according to the 2017 ESC STEMI guidelines [6], and NSTEMI was defined following the 2020 ESC guidelines [7]. The evaluation of a MI is comprised of three components: clinical

Received: 21/07/2022
Accepted: 23/09/2022

symptoms, ECG abnormalities, and cardiac biomarkers.

ECG: The 12-lead ECG at rest is the most crucial diagnostic tool for ACS. The specimen was taken within 10 minutes of the patient's admission to the emergency department [8]. Electrocardiogram findings indicate persistent coronary artery obstruction (in the absence of left ventricular hypertrophy and bundle branch block) [9].

Biomarker Detection of MI: In succession, the cTn values at 0 hours, 3 hours, and 6 hours were evaluated. The cTn concentration is used as a reference point to calculate the ascending/descending trend. If the cTn baseline value is sufficiently elevated, a minimum difference of 20% between tests indicates myocardial ischemia. Additionally, the MB isoform of creatine kinase was studied.

Coronary Angiography: The femoral artery was utilized to perform coronary angiography. Two seasoned cardiologists who were uninformed of the patients' enrollment status assessed all angiograms. Patients were categorized as having coronary artery disease or not based on angiographic data.

Non-CAD patients were categorized as having negligible atherosclerosis (luminal diameter narrowing of less than 50 percent) or normal atherosclerosis (without luminal diameter narrowing).

Angiographic scoring: Angiograms were evaluated using three criteria: At least fifty percent lumen stenosis in the number of vessels. According to the ACC/AHA 16-segment model of the coronary tree, the angiographic severity score is the number of coronary artery segments with at least 50% stenosis.

Blood samples: The blood samples for CBC were collected and placed in tubes containing tri-potassium ethylenediaminetetraacetate (K3-EDTA) which was used as an anticoagulant. CBC was analyzed using the CELL-DYN Ruby Hematology Analyzer (CELL-DYN 3700, Abbott, Canada). After 12 hours of fasting overnight, Serum was collected in serum gel tubes for all biochemical parameters. The serum was separated from the blood samples and stored at 80 C until the analysis. Biochemical parameters were measured on a Cobas c 711 analyzers (Roche Diagnostics®).

Serum HP IgG antibody was measured using ELISA kit: 201-12-5758 an HP antibody ELISA Kit. To detect HP Ab, the ELISA kit is based on the double-antibody sandwich approach. Not be utilized for medical diagnosis; solely for research purposes.

Test principle: The kit uses a double-antibody sandwich enzyme-linked immunosorbent assay (ELISA) to assay the HP antibody level in samples. HP Ab was added to HP Ab micro ELISA well, incubated; then washed. HRP-tagged HP antibody was added. Following additional incubation and washing, the unbound enzyme was removed. Due to the addition of Chromogen Solutions A and B, the color of the liquid changed from red to blue, and subsequently to yellow due to the acid. The presence of HP antibodies in the sample was evaluated by comparing the observed absorbency to the critical value using a microplate reader with a 450 nm wavelength to detect absorbency (OD value).

Assay procedure: Several unfilled wells remained (blank wells do not contain sample and HRP-conjugate reagent; all other steps are identical). 50 ul of standard dilution was applied to both the control and sample wells during standard well testing. Before incubation at 37 °C, the final sample dilution was 5-fold, and the plate was gently shaken for 30 minutes. Each well was filled with 30 times-diluted washing solutions, oscillated for 30 seconds, and then drained using absorbent paper. The affected region was dried with a towel. This instruction was repeated five times. Except for the blank well, each well received 50 ul of HRP-conjugated reagent. Shaking was utilized to mix the components, which were then incubated at 37 °C for 30 minutes. Each well was filled with 30 times-diluted washing solutions, oscillated for 30 seconds, and then drained using absorbent paper. Five times, followed by drying with a towel. Each well received 50 liters of solution A and 50 liters of solution B. A 10-minute incubation at 37°C with moderate mixing was performed. Each well received 50 ul of Stop Solution to cease the reaction (the blue color changed to yellow color Immediately). Following 15 minutes of Stop Solution injection, the optical density (OD) of the blank well was measured to be 450 nm.

Seropositivity HP: Seropositivity HP was detected based on serum titers of higher than 30AU/ml.

Sample size

The sample size was calculated using Epi info software version 7.2.2.16 based on a previous study done by **Sung *et al.*** [10] which reported HP prevalence of 20% in patients with acute myocardial infarction. The total sample size calculated was 96 patients as a minimum number. The margin of error and confidence level was adjusted at 8.0% and 95%, respectively.

Ethical consent:

Approval of the study was obtained from Benha University Academic and Ethical Committee. After explaining our research objectives, written informed consent was obtained from all study participants. This study was conducted in compliance with the code of ethics of the world medical association (Declaration of Helsinki) for human subjects.

Statistical analysis:

SPSS 28 is used for information management and statistical analysis (IBM, Armonk, New York, United States). The normality of quantitative data was

determined with the use of the Kolmogorov–Smirnov test, the Shapiro–Wilk test, and direct data visualization tools. The quantitative data were presented as means, standard deviations, medians, and ranges based on the assumption of normality.

We used percentages and quantitative values to summarize category information. For normally and non-normally distributed quantitative variables, the independent t-test or Mann-Whitney U test was used to compare quantitative data across study groups. The Chi-square test was employed for comparing categorical data. Patients with positive IgG exhibited Pearson's or Spearman's correlations between EF and other parameters. Every statistical test produced two outcomes. P-values below 0.05 were deemed statistically significant.

RESULTS

The mean age of the studied patients was 58±12 years. Approximately two-thirds were males (66%). The mean hemoglobin was 13±2.2 g/dl. The median total leucocyte count was 7.8, ranging from 3.6 – 26. The mean platelet count was 218.1 ±72.5. The median serum creatinine was 1.25, ranging from 0.6 – 3.3.

The median blood urea was 44.1, ranging from 17-184. The median ALT and AST were 43.5 and 42.9, respectively. Regarding lipid profile, the mean total cholesterol was 180 ±60 mg/dl. The median triglycerides were 147, ranging from 44-428. The median HDL and LDL were 42 and 93, respectively. Regarding ECG, about half of the patients had NSTEMI (46%), while the other half had STEMI (54%). The mean EF was 51 ±10% **(Table 1).**

Table (1): Demographic characteristics, laboratory and cardiac findings of the studied patients

Demographics	N=100
Age (years)	58 ±12
Gender	
Males	66 (66%)
Females	34 (34%)
Laboratory findings	
Hemoglobin (g/dl)	13 ±2.2
Total leucocyte count	7.8 ± 1.5
Platelets (mcL)	218.1 ±51.4
Serum creatinine (mg/dL)	1.25 ±0.28
Blood urea (mg/dL)	44.1 ± 10.34
ALT (U/L)	34.5 ± 8.31
AST (U/L)	42.9 ± 10.32
Total cholesterol (mg/dl)	180 ±41.36
Triglycerides (mg/dl)	147 ± 33.64
HDL (mg/dl)	42 ± 10.42
LDL (mg/dl)	93 ± 22.81
Cardiac findings	
ECG	
NSTEMI	46 (46)
STEMI	54 (54)
Ejection fraction (EF) (%)	51 ±10

The prevalence of HP among the studied patients was 72%. **Table 2.**

Table (2): Prevalence of HP in the studied patients

HP IgG	N=100 (%)
Positive	72 (72%)
Negative	28 (28%)

There were non-significant differences between those with positive and negative HP Ig-G regarding age (P = 0.453) and gender (P = 0.487). **Figure 1**

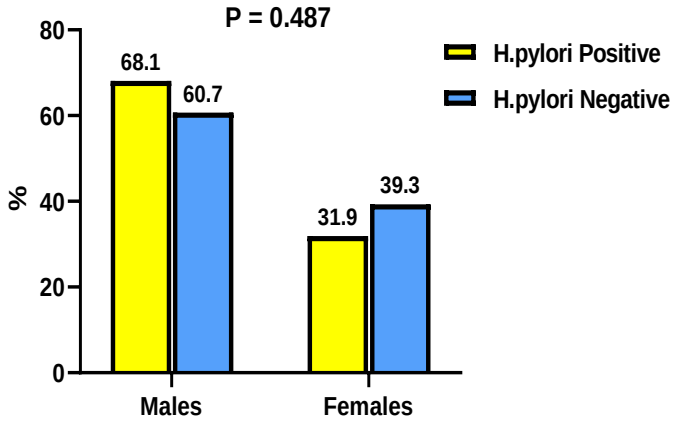

Figure (1): Gender distribution according to HP status.

Those with positive HP Ig-G had significantly lower triglycerides (median = 133 vs. 172 mg/dl, P = 0.028) than those with negative HP Ig-G. There were no significant differences observed regarding hemoglobin, TLC, platelets, serum creatinine, blood urea, ALT, AST, total cholesterol, HDL, and LDL (**Table 3).**

Table (3): Laboratory findings according to HP status

	HP IgG		P-value
	Positive (n = 72)	Negative (n = 28)	
Hemoglobin (g/dL)	13 ±2.2	12.9 ±2	0.783
TLC	7.65 ± 1.51	8.68 ± 2.2	0.618
Platelets (mcL)	224.1 ±55.8	202.7 ±48.5	0.187
Serum creatinine (mg/dL)	1.25 ± 0.29	1.2 ± 2.13	0.537
Blood urea (mg/dl)	43.1 ± 10.62	45.4 ± 11.2	0.529
ALT (U/L)	34 ± 8.71	35.3 ± 8.61	0.664
AST (U/L)	43.1 ± 10.31	40 ± 9.72	0.985
Total cholesterol (mg/dl)	180 ±43.21	181 ±44.33	0.920
Triglycerides (mg/dl)	133 ± 32.21	172 ± 42.21	0.028 *
HDL (mg/dl)	41 ± 9.72	46 ± 11.61	0.454
LDL (mg/dl)	90 ± 21.34	102 ± 24.32	0.224

* Significant; Data were presented as mean ±SD or median (min-max)

Cardiac findings according to HP status: There were insignificant differences between those with positive and negative HP Ig-G regarding ECG findings(P = 0.401) and ejection fraction (P = 0.236) **(Table 4).**

Table (4): Cardiac findings according to HP status

	HP IgG		P-value
	Positive (n = 72)	Negative (n = 28)	
ECG			
NSTEMI	35 (0)	11 (0)	0.401
STEMI	37 (0)	17 (0)	
EF	50 ±11	53 ±10	0.236

In patients with positive HP Ig-G, statically significant negative correlations were found between EF and blood urea (r = -0.263, P = 0.026), serum creatinine (r = -0.407, P < 0.001), and AST (r = -0.311, P = 0.008). There are no significant correlations with age, hemoglobin, TLC, platelets, ALT, total cholesterol, triglycerides, HDL (P = 0.882), and LDL **(Table 5).**

Table (5): Correlation between EF and other parameters

	Ejection fraction	
	r	P-value
Age (years)	-0.133	0.264
Hemoglobin (g/dL)	-0.036	0.765
TLC	-0.109	0.362
Platelets (mcL)	-0.122	0.307
Blood urea (mg/dl)	-.263	0.026*
Serum creatinine (mg/dL)	-.407	<.001*
ALT (U/L)	-0.219	0.065
AST (U/L)	-.311	0.008*
Total cholesterol (mg/dl)	-0.031	0.796
S. triglycerides (mg/dl)	0.105	0.378
HDL (mg/dl)	-0.018	0.882
LDL (mg/dl)	-0.148	0.214

* Significant; r: Correlation coefficient

DISCUSSION

Chronic infection, such as HP infection, is crucial to the etiology of coronary heart disease. Several studies indicate that HP infection increases the risk of coronary heart disease. However, several investigations have shown that HP infection is not directly connected to CHD [11, 12].

Regarding the current work, the mean hemoglobin of our cases was 13 ±2.2 g/dl. The median total leucocyte count was 7.8, ranging from 3.6 – 26. The mean platelet count was 218.1 ±72.5, while **Júnior *et al.*** [13] revealed in their study that, 466 patients (mean age 64.2 ± 12.8 years, 61.6% male) and AMI with STEMI (70%). The mean hemoglobin was 13.0 ± 2, the median leukocyte count was 10.5 ranging from 8.4 to 12.8, and the mean Platelet count was 231, ranging from 195.7 to 278, which agreed with our results.

In addition, the median serum creatinine in our study was 1.25, ranging from 0.6 – 1.9. The median blood urea was 44.1, ranging from 17-184. The median ALT and AST were 43.5 and 42.9, respectively, these results differed from the results by **Baars *et al.*** [14]**,** who found that serum creatinine in AMI patients with stenosis diameter more than or equal to 50% was 1.36± 0.04, The median ALT and AST were 59.23±1.33 and 121.42± 1.72, respectively. These different results may be contributed to the different number of participants as well as different ages and comorbidities with myocardial infarction.

Regarding lipid profile, the mean total cholesterol was 180 ±60 mg/dL. The median triglycerides was 147, ranging from 44 to 428 mg/dL. The median HDL and LDL were 42 and 93, respectively which agreed with **Kumar *et al.*** [15] who reported that patients with AMI had Mean ± SD total cholesterol of 207.5 ± 30.5, mean triglycerides were 153.8 ± 10.2, mean LDL-C was 149.0 ± 41.2, mean HDL-C was 46.6 ± 9.9. Regarding ECG, about half of the patients had NSTEMI (46%), while the other half had STEMI (54%). The mean EF was 51 ±10% which agreed with **Júnior *et al.*** [13] who detected STEMI in 70% of their study cases with AMI

In the current work, the prevalence of HP among the studied patients was 72%. As **Rahmani *et al.*** [16] investigated the association between HP and MI, their findings were comparable to those of previous research. The risk ratio for MI was 1.73 times greater in those with HP than in those without MI, according to the research. However, this quantity was found to be 2.1 times bigger when low-quality publications were omitted, which is mostly consistent with our research findings.

The OD of MI in patients with HP in Asian nations was 1.75 times larger than in the control group, indicating that Iranians are more likely to be exposed to HP than Asians [17]. In addition, many studies have shown a link between HP and MI, with a greater incidence of infection in case groups compared to control groups [18, 19].

Although the aforementioned research indicated a substantial association between HP and MI, a few of them contradicted this association by indicating a low incidence of HP. **Nakic *et al.*** [20] detected incidence rates of 29% vs. 26% for HP in the case and control groups, however, **Tsai *et al.*** [21] reported incidence rates of 69% vs. 72%, showing no statistically significant difference. However, a recent meta-analysis indicated a 15% increase in CAD risk.

There are two probable reasons for the association between HP and the risk of myocardial infarction. **Reszka *et al.*** [22] identified HP deoxyribonucleic acid in ischemic heart disease patients' aortic tissue and atherosclerotic plaques. This research shows that microorganisms have a direct role in the pathophysiology of ischemic heart disease and,

subsequently, at the beginning of MI.

Second, viral infection may decrease HDL cholesterol and increase triglyceride levels. Blood levels of coagulation and inflammatory markers such as fibrinogen, prothrombin fragments, tumor necrosis factor, and interleukin 6 and 8 may be high. These factors may modify the association between hypertension and ischemia [16].

In this study, there were no significant differences in age or gender between positive and negative H.pylori IgG patients. There were no significant differences in ECG fundings and ejection fraction between persons with positive and negative HP IgG antibodies. **Shmuely *et al.*** [23] discovered that individuals with coronary artery disease had a statistically insignificantly greater rate of HP seropositivity than those without CAD. These findings suggest that H. CAD and MI are independently related to the presence of pylori. Anti-CagA seropositivity did not differ significantly between CAD-positive and -negative groups.

In a prior experiment conducted by **Jafarzadeh *et al.*** [24]**,** significantly higher anti-H levels were discovered. Anti-Helicobacter pylori antibodies in the blood of 120 patients with ischemic heart disease relative to healthy controls. Anti-CagA IgG seroprevalence and mean titer did not differ substantially between the patient and control groups. **Pellicano and colleagues**[25]**,** who evaluated 223 consecutive patients with verified acute MI and compared them to matched controls, revealed a similar finding. Anti-CagA antibodies were discovered in 33.8% of infected patients with acute myocardial infarction compared to 26.8% of the control individuals, demonstrating that there is no association between HP infection with CagA-positive strains and ischemic heart disease.

Our study showed that those with positive HP Ig-G had significantly lower triglycerides than those with negative HP Ig-G. No significant differences were observed regarding hemoglobin, TLC, platelets, serum creatinine, blood urea, ALT, AST, total cholesterol, HDL, and LDL. In patients with positive HP Ig-G, significant negative correlations were observed between EF and blood urea, serum creatinine, and AST. No significant correlations were observed with age, hemoglobin, TLC, platelets, ALT, total cholesterol, triglycerides, HDL, and LDL. According to a study of 961 individuals, high blood pressure is not directly related to CAD severity; rather, it is connected to low HDL levels, which play a protective function against atherosclerosis [26].

In contrast, **Haeri *et al.*** [27] discovered that 66.5% of their patients tested positive for HP using serology. According to statistical analysis, HDL and TG levels did not differ substantially between HP-infected and uninfected patients. The levels of LDL and HDL in patients with and without HP infection were not significantly different. Those infected with HP had considerably higher mean fasting blood glucose (FBS) levels than those who were not infected. HP infection in Japanese male patients led to indirect changes in blood lipid profile, including an increase in LDL-cholesterol and a decrease in HDL-cholesterol, according to the findings of Japanese research [28].

Due to differences in the number of patients and risk factors for myocardial infarction between our analysis and that of **Rampengan *et al.*** [29], their findings contradicted ours.

Rampengan *et al.* [29] identified significant associations between AMI and gender, diabetes history, diastolic blood pressure, triglyceride level, LDL level, number of ejection fractions, menopause, and menopause. There have been several hypotheses on the relationship between HP and influenza. Viral and bacterial infections may influence the lipid metabolism of infected cells, as shown by past research. Other studies have shown that the body's attempts to repair illness result in a rise in cholesterol levels. According to subsequent research, LDL exhibits antibacterial properties and is directly engaged in the eradication of dangerous germs. Studies indicate that mice with faulty LDL receptors have higher LDL levels, which protects them from gram-negative bacteria such as HP [30].

This study has some limitations as it included a relatively small sample size which can bias our results. The association of comorbidities of MI should be studied to correlate with H pylori infection. Lacking use of a specific age group made our results different from many studies because of the different prevalence of H pylori infection in different age groups.

CONCLUSION

Our research indicates a connection between HP and myocardial infarction. The association between HP infection and CAD has been questioned in recent years, and there is little scientific evidence to support it. It is essential to conduct well-designed, high-quality, evidence-based, and less biased cross-sectional, cohort, and randomized clinical studies to evaluate and make a decision about this contentious link.

Conflict of interest: There is none to be declared **Sources of funding:** none.
Author contribution: all authors contributed equally to this study.

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