Pak Heart J

ASSOCIATION BETWEEN HELICOBACTER PYLORI INFECTION AND ACUTE MYOCARDIAL INFARCTION (AMI)

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Date Received: November 01,2016
Date Revised: November 23,2016
Date Accepted: December 14,2016

Contribution
SFAH concieved the idea, planned the study and critically revised the manuscript. SB helped in data collection and statistical analysis. All authors contributed significantly to the submitted manuscript.

All authors declare no conflict of interest.

This article may be cited as: Bai S, Hashmi SFA. Association between helicobacter pylori infection and acute myocardial infarction (ami). Pak Heart J 2017; 50 (01): 33-8.

ABSTRACT

Objective: To determine the association between helicobacter pylori infection and acute myocardial infarction.

Methodology: A six months case-control study was conducted at Cardiology Department of Liaquat Medical University Hospital, Jamshoro. The study comprised of patients presenting with acute myocardial infarction (cases) admitted with myocardial injury and controls that were volunteer individuals. The relevant laboratory investigations were advised and the data was analyzed using SPSS version 22.0.

Results: Total 218 individual were evaluated. Of them 109 (50%) were controls and 109 (50%) were cases during study period. About 110 (50.45%) participants were males. The mean age of controls was 51.88±.7.90 years and cases 50.93±8.13 years with range of 35-65 (t= -.870, p=.385). H-Pylori status was positive in 75.2% of cases as compared to 41.2% of controls ($\chi 2=5.945$, p=.001). Also higher in males 85.4% than females (t=7.348 and p=.000).

Conclusion: It was concluded that H-Pylori infection was significantly associated with Acute myocardial infarction patients compared to controls. Therefore, monitoring of H-Pylori infection as a possible risk factor of coronary artery disease may be of clinical value.

Key Words: Helicobacter pylori, Acute myocardial infarction.

INTRODUCTION

Helicobacter pylori causes chronic gastritis that results in persistent low grade inflammatory response and induce changes in coagulation system with elevated serum levels of fibrinogen, prothrombin fragments, plasminogen-activating inhibitor-1 (PAI-1), and factor VII.[1] Many other mechanisms like elevated concentrations of tumor necrosis factor, (TNF), interleukin-6 (1L-6), interleukin-8 (IL-8) and lipid profile changes are also responsible for onset of CHD and AMI.[2-4]Helicobacter pylori DNA was isolated by polymerase chain reaction from atherosclerotic plaque and was supposed to directly effect on lesion progression and activation of inflammation which lead to acute coronary syndrome.[5]Some studies have found the role of CagA positive strain of this pathogen in CHD.[6,7] Khodaii et al. confirmed that the patients with AMI had a significantly higher prevalence of helicobacter pylori infection and CagA seropositivity than the control population.[8]

Hence the purpose of this study was to find out the association between H-Pylori infection and AMI as early evaluation and eradication can prevent the patients to acquire coronary heart diseases.

METHODOLOGY

This case control study of six months was conducted at Cardiology department of Liaquat Medical University & Hospital Jamshoro from January to June 2014.The study population included patients of both genders, between the ages of 35 to 65 years consecutively admitted to the CCU, who met the AMI diagnostic criteria. For controls healthy individuals of age ranging from 35 to 65 years, of either gender with no history of heart disease or peptic ulcer disease were selected. The sample size was calculated based on the information available in the literature. Patients with age < 35 years or > 65 years, having angina only, with cardiogenic shock, post PCI, post CABG, known case of significant chronic medical illness (hyper or hypothyroidism, renal disease, or malignancy) or females with pregnancy was excluded.

For controls, patients with age < 35 years or > 70 years, with well known peptic ulcer disease, getting treatment for ulcer disease or eradication therapy for H. pylori in the last 12 months were excluded. Cases were the patients of AMI, selected from CCU and Cardiology ward at Liaquat university hospital Hyderabad. Controls were healthy volunteer individuals with no heart disease were selected from various wards. Cases and controls were matched for age, and gender. Investigations like ECG, anti H. pylori IgG and IgA (ELISA method) were done in all patients, while cardiac enzymes markers of myocardial injury and Necrosis (CK, CK-MB, cardiac troponins I and T were done only in AMI patients. Data of cases and controls was collected on specially designed performa and analyzed. Privacy and confidentiality of all patients was maintained. No new intervention was done except for standard protocols of the ward.

The data was analyzed through SPSS version 22. The main independent variable of the study were H-Pylori seropositivity between cases and controls and H-Pylori negative cases or controls were compared using the Chi-squared test. Stratification was done to control effect modifier like age, sex of both groups. P value of 0.05 was taken as significant.

RESULTS

This study comprised of 218 (100) individuals, from which 109 (50%) apparently healthy individuals were labeled as controls and 109 (50%) were patients of AMI labeled as cases. Total 110 (50.45%) participants were male in both cases and controls. Total 124 (56.88%) participants were residents of urban areas. The mean ages of controls and cases were 51.88±.7.90 years and 50.93±8.13 years with ranges of 35-65 in both cases and controls. Age was further divided into three groups, group 1: 35 to 45 years of age of 42 individuals having 20 (18.3) controls. Group II: 46-55 years of 113 individuals having 58 (53.2) controls, group III: 56 to 65 years of 63 having 31 controls. Table 1 illustrates personal profile of the study population. About 82 (75.2%) as compared to 45 (41.2%) controls were positive for H. pylori. The difference between the two groups was significant ($\chi 2=5.945$, p=.001) with higher distribution of H. pylori among cases. Distribution of H-Pylori among the study population is presented in Table 2.

Table 3 shows the relationship between H. pylori and gender in cases. Number of male individuals infected with H. pylori (47) (85.4) was higher (t=7.348 and p=.000).

Table 4 shows gives the relationship between H. pylori and the cardiac enzymes. Although the activities of CK, CKMB enzymes and Troponin were higher in H-Pylori positive than negative cases, the differences between the two groups were not significant (p>0.05).

Helicobacter Pylori Infection and Lipid Profile is shown in table 5. The mean level of triglyceride in positive cases was significantly higher than that in negative cases (189.38±104.37vs 255.52±16.91 mg/dl, p=.000). The mean levels of cholesterol and LDL-C were also higher in positive than in negative cases (271.22±68.41 and 220.60±44.57mg/dl, vs255.52±16.91 and 117.35±14.33 mg/dl)(p=0.00). On the other hand the mean level of HDL-C was significantly lower in positive compared to negative cases (37.12±12.75 vs51.37±11.04 mg/dl, p=.000).

Table 6 showed no significant difference in body mass index between positive and negative cases (t=1.046, p=0.300)

Table1: Demographic variables of the Study Population (n=218)

Demographic variables		Cases n=109 n (%)	Controls n=109 n (%)	Test	Value	Difference	p-value
Mean age (years)		50.93±8.13	51.88±.7.90				
Age groups (years)	35-45	22 (20.1)	20 (18.3)	t	-.870	-.94	.678
	46-55	55 (50.4)	58 (53.2)				
	56-65	32 (29.3)	31 (28.4)				
Sex	Male	55 (50.4)	55 (50.4)	2	.018	1	0.892
	Female	54 (49.5)	54 (49.5)				
Resident	Urban	66 (60.5%)	58 (53.2%)	2	4.18	1	0.042
	Rural	43 (39.5%)	51 (46.7%)				

Table 2: Distribution of Helicobacter Pylori Among the Study Population (n=218)

Helicobacter pylori	Controls (n=109) n (%)	Cases (n=109) n (%)	x^2	p-value
Positive	45 (41.2)	82 (75.2)	5.945	0.001
Negative	64 (58.7)	27 (24.7)		

Table 3: Gender based Distribution of Helicobacter pylori Infection of Study Population (n=218)

Helicobacter pylori status	Cases		t	p-value
	Male n=55 n (%)	Female n=54 n (%)		
Positive	47 (85.4)	35 (64.8)	7.348	0.000
Negative	8 (14.5)	19 (34.5)		

Table 4:Association of Helicobacter Pylori Infection and Cardiac Enzymes in Study Population (n=218)

Cardiac enzymes (IU/L)	Helicobacter status	n	M ean±SD	t	p-value
CK	Positive	82	302.63±210.06	1.144	0.255
	Negative	27	256.19±16.20		
CKMB	Positive	82	46.50±23.81	0.467	0.642
	Negative	27	43.70±35.11		
Trop I	Positive	82	24.82±13.19	1.903	0.060
	Negative	27	20.36±9.57		

Table 5: Association of Helicobacter Pylori Infection to Lipid Profile in Study Population (n=218)

Lipid profile	Helicobacter status	n	Mean±SD	t	P – value
Cholesterol	Positive	82	271.22±68.41	- 9.020	.000
	Negative	27	255.52±16.91		
Triglyceride	Positive	82	189.38±104.37	- 14.903	.000
	Negative	27	111.5±71.57		
HDL-C	Positive	82	37.12±12.75	- 5.195	.000
	Negative	27	51.37±11.04		
LDL-C	Positive	82	220.60±44.57	- 12.426	.000
	Negative	27	117.35±14.33		

Table 6: Association of Helicobacter Pylori to Body Mass Index in Study Population (n=218)

Parameter	Helicobacter Status	n	mean±SD	t	p-value
BMI (kg/m^2)	Positive	82	30.77±6.40	-1.902	0.000
	Negative	27	29.81±.91		

DISCUSSIONS

Helicobacter pylori causes chronic gastritis that results in persistent low grade inflammatory response and shows that Helicobacter pylori infection induces changes in coagulation system with elevated serum levels of fibrinogen, prothrombin fragments, plasminogen-activating inhibitor-1 (PM-1), and factor VII. Helicobacter pylori DNA was isolated by polymerase chain reaction from atherosclerotic plaque and was supposed to direct effect on lesion progression and activation of inflammation, which lead to acute coronary syndrome.

However our hypothesis that H-pylori infection as a cause of acute myocardial infarction is too old but still a debatable issue, because some clinical and epidemiological studies had shown a possible association, and on the other hand, some studies did not show any association between H-Pylori and CAD.[9,10]

This study comprised of 218 individual. Total 110 (50.45%) participants were male in both cases and controls. Prashant PJ, et al reported 70% males.[11] Shrikhande SN, et al reported 186(70.19) males in their study.[12] Simarly Rogha M, et al reporting mean age ± SD) 62.4 ± 9.5 with p=0.12.[13] Ramzil, AM reported 31 males in their study.[14]

In present study H-Pylori was positive in (75.2%) cases as compared to controls 45 (41.2%) with significant difference (χ^2=5.945, p=.001).Ramzi AM reported 46 (74.2%) positive cases as compared to 26 (41.9%) controls. (χ^2=13.248, p=0.000).[14] Rogha M et al also reported H-Pylori in 30 subjects with CHD and 16 participants without CHD (p = 0.06).[13] Shrikhande SN et al also showed similar results. The study detected an OR of 2.50 (95% CI: 1.71-3.65) suggesting that AMI patients with IgG positive for this infection is 2.5 times more likely to suffer from AMI. Vafaeimanesh J et al suggested that higher rate of H-Pylori infection was observed in 76.3% of patients with Multi Vessel Disease and 70% of patients with Single Vessel Disease had HP infection while it was 50% in normal patients ($p < 0.05$).[15]

In present study the number of males infected with H. pylori (47) (85.4) was higher than that of females, (t=7.348 and p=.000).Ramzi AM reported in his study that males infected with H. pylori 25 (80.6%) was higher than that of females 21 (67.7%), (χ^2=1.384 & p=0.192).[14]

In current study the activity of CK, CKMB enzymes and Troponin was raised in H. pylori positive individuals.Ramzi AM showed, CK and CKMB enzymes were raised in H. pylori subjects.[14] In this study the mean level of triglyceride in H-Pylori positive cases was higher (189.38±104.37vs 255.52± 16.91 mg/dl, p=.000).

In our study the mean levels of cholesterol and LDL-C were also higher in positive than in negative cases (271.22±68.41 and 220.60±44.57mg/dl, vs 255.52± 16.91 and 117.35± 14.33 mg/dl), (p=.000 and .000, respectively).Ramzi AM showed that the mean levels of cholesterol and LDL-C were also higher in positive (215.5±42.0 and 136.7±43.1 mg/dl, vs 189.7±35.8 and 116.9±35.7 mg/dl).[14] While in present study the mean level of HDL-C was lower in positive subjects as compared to negative subjects (37.12±12.75 vs 51.37± 11.04 mg/dl, p=.000).Ramzi AM reporting in his study that the mean level of HDL-C was significantly lower in positive compared to negative cases (31.7±8.0 vs 39.5±12.3 mg/dl, p=0.005).[14]

In this study there was no significant difference in body mass index among negative and positive cases, the findings was also observed by Ramzi A M.[14]

CONCLUSION

The existence of H-Pylori was significantly higher in cases than in healthy controls. More over serum triglycerides was significantly increased whereas HDL level was significantly lower in H-Pylori positive patient.

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