

Helicobacter Pylori Infection may be a New Risk Factor in Developing Acute Myocardial Infarction

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ABSTRACT

Objective: The purpose of present study was to find out the relationship between helicobacter pylori infection and acute myocardial infarction

Study Design: Experimental / Case control study

Place and Duration of Study: This study was carried out the Biochemistry Department, Dow University of Health Sciences, Karachi from 11.02.2013 to 15.12.2013.

Materials and Methods: Serum samples of age and gender matched 80 each of Cardiac and non-cardiac patients with H. Pylori were investigated for the levels of IgG and IgA by kit methods.

RESULTS: Patients who were H.pyloric AMI compared to non-h.pyloric normal had relationship with increased IgG in Acute myocardial infarction patient with H.pyloric infection as compare to control.

Conclusion: The results of present study suggest that H. pylori is a new risk factor for the development of atherosclerosis and enhance the risk factor. Therefore proper treatment and diagnosis can be helpful for cardiac patients.

Key words: H. pylori, Atherosclerosis, Myocardial infarction, IgG, IgA.

INTRODUCTION

Coronary artery disease is known to be the most leading cause of death in industrialized societies¹. A cardiovascular risk factor is due to the inflammatory response to irritation, lipid peroxidation and infection². H. pylori cause chronic gastritis, a persistent low grade inflammatory response increase fibrinogen, a coagulation factor which is a predictor of ischemic heart disease³. Cardiac risk factors can cause impairment of endothelial vasodilator function of coronary resistance arteries⁴⁻⁶.

Most of the patient infected by H.pylori never experience symptoms and complications⁷. 10 – 20% patients with symptoms are risk of developing peptic ulcer while 1-2% is at risk of acquiring gastric carcinoma⁸. H. pylori is present in mucus on the inner lining of epithelium and inside cells⁹. For the first time Mendel et al suggested the relationship between coronary heart disease with H.pylori¹⁰.

MATERIALS AND METHODS

In this study 88 persons with no history of cardiac disease and 87 myocardial infarction patients having age between 35 to 70years old were compared in two separate groups. The case group admitted in CCU ward was selected by convenient non probability sampling method. Patients were all diagnosed MI with confirmed clinical symptoms, ECG changes high cardiac enzymes, while the control had no history of any cardiovascular disease, ECG changes or any positive physical or

clinical examination. Gastrointestinal drugs users excluded in both groups.

Five ml blood sample was drawn from every MI patient and controls which were involved in the study by venupuncture using a 05 ml sterilized disposable syringe. After clotting, serum was separated by centrifugation and stored at -20 °C until analyzed. Levels of parameters were measured by ELIZA methods using monobind kit..Data were analyzed using Chi-square (X²) and T- test in SPSS version 16 software.

RESULTS

Table No.1: Comparison of relative frequency of Positive IgG and IgA in both groups.

	Negative N (%)	Positive N(%)	Total N(%)	p
IgG				
Case	38(43%)	50 (56.81%)	87 (100%)	0.003
Control	60 (68%)	38 (31.8%)	88 (100%)	
IgA				
Case	50 (58.8%)	38 (43%)	87 (100%)	0.45
Control	45 (51.1%)	43 (48.8%)	88 (100%)	

There were all males cases and control, the mean ages in the control and case groups 55.3 ± 11.8 and 57.8 ±

13.7 years respectively ($p = 0.1$). there were also no significant differences were found between case and control groups in diabetes, hypertension and obesity. 50 patients (56.81%) from case group were IgG positive against *H. pylori* and 38 (31.8%) persons in control group were IgG positive against *H. pylori*. The statistically difference was significant ($p = 0.002$), while there were no significant difference between IgA levels of two groups ($p = 0.44$) (Table 1). 38 (43%) cases and 43 (48%) controls had positive IgA against *H. pylori*.

Table No. 2: Relative frequency of positive IgG and IgA in case group.

	IgG	Negative N (%)	Positive N (%)	Total N(%)
IgA				
Positive		10 (26.3%)	28 (73.6%)	38 (100%)
Negative		28 (66.6%)	14 (33.3%)	42 (100%)
Total		38	42	

$\chi^2 = 6.125$ $df = 1$ $p = 0.01$ $OR = 3.5$ $CI: 95\%$
1.3 – 9.5

DISCUSSION

There is a significant difference in our study, in which there is IgG levels of *H. pylori* was shown between cases and control (0.002).

According to some researchers, *H. Pylori* infection is seen in lower socioeconomic society peoples¹⁰ and their frequency is higher in older age people^{11,12}. In our study there is a significant difference between control group and cases with IgA and IgG. The antibodies against *H. pylori* compared with the cardiovascular risk factors were shown significantly diversity.

HDL levels are significantly lowers in *H. pylori* seropositive cases according to En-zhi-jin¹³. Chronic infections which may be viral and bacterial may play a role in aggravation of atherosclerosis¹⁴. Like infections, hypertension, diabetes and smoking are also major risk factors which cause cardiovascular events¹⁵.

Release of inflammatory cytokines during chronic infections which causes endothelial dysfunction and blockage of small vessels that leads to decrease blood flow^{16,17,18,19}. *H. pylori* infection is considered as a new risk factors in developing atherosclerosis due to increased risk in chronic inflammation. This disease may increase risk in the patients with low birth weight²⁰.

CONCLUSION

From this study, it may be concluded that *H. pylori* infection can a considered as a new risk factors in developing chronic inflammation in developing

atherosclerosis. Therefore proper treatment and diagnosis can decrease the hazards of atherosclerosis.

REFERENCES

- Goode GK, Miller JP, Heagerty AM. Hyperlipidemia, hypertension and coronary heart disease. *lancet* 1995;345:362-264.
- De luis DA, Lahera M, Canton R, et al. Association of helicobacter pylori infection with cardiovascular and cerebrovascular disease in diabetic patients. *Diabetes care* 1998; 21: 1129 – 32.
- Parente F, Bianchi Porro G. The association between helicobacter pylori and ischemic heart disease: facts or fancy? *Helicobacter* 1997;2: 67– 72.
- Treasure CB, Klein L, Vita JA, Manoukian SV, Renwick GH, Selwyn AP, et al. Hypertension And left ventricular hypertrophy.
- Egashira K, Inou T, Hirooka Y, Kai H, Sugimachi M, Suzuki S, et al. Effects of age on endothelial dependent vasodilation of resistance coronary artery by acetylcholine in humans. *Circulation* 1993; 88:77 -81.
- Egashira K, Inou T, Yamada A, Hirooka Y, Marouka Y, Takeshita A. Impaired coronary blood flow response to acetylcholine in patients with coronary risk factors and proximal atherosclerotic lesions. *J Clin Invest* 1993; 91:91 – 37.
- Butzer P, Dahlerp JF, Eriksen JR, Jarbol DE, Rosenstocks, Wildt S. Diagnosis and treatment of *Helicobacter pylori* infection. *Dan Med Bull* 2011; 58(4): C4271.
- Suerbaum S, Michetti P. *Helicobacter Pylori* infection. *N Engl J Med* 2002;347(15):1175 – 86.
- Petersen AM, Krogfelt KA.. *Helicobacter pylori*, an invading microorganism? A review *FEMS. Immunol Med Microbiol* 2003;36(3):117 – 26.
- Saraf – Zadehan N, Amiri M, Maghsoudloo S. *Helicobacter pylori* relation to acute myocardial infarction in an Iranian sample. *Coronary Health Care* 2001;5:202-7.
- Manolakis A, Kapsoritakis NA, Potamianos SP. *Helicobacter* 2007;287.
- D. Nakic, et al. *Helicobacter pylori* infection and myocardial infarction. *Coll Antropol* 2011;3: 781 -785.
- Jia EZ, Zhao FJ, Hao B, et al. *helicobacter pylori* infection is associated with decreased serum levels of high density lipoprotein, but not with the severity of coronary atherosclerosis. *Lipids Health Dis* 2009; 8:59.
- Nocent R, Gentiloni N, Cremonini F, et al. Resolution of syndrome X after eradication of virulent *cag A* Positive *helicobacter pylori*. *South Med J* 2000; 93:1022-3.

15. Kumar V, Abbas AK, Fausto N, Mitchell R. Robbins Basic Pathology, 8th ed. USA: WB Saunders Company 2007.p.70- 77.
16. Su YC, Wang WM, Wang SY, et al. The association between helicobacter pylori infection and functional dyspepsia in patients with irritable bowel syndrome. Am J Gastroenterol 2000;95: 1960 -5.
17. Annuziata P, Figura N, Galli R, Murganinin F, Lenzi C. Association of anti –GMI antibodies but not of anticytomegalovirus, campylobacter jejuni and helicobacter pylori IgG, with a poor outcome in Guillain – Barre syndrome. J Neurol Sci 2003; 213:55-60.
18. Tsai WC, Li YH, Sheu BS, et al. Association of elevation of anti-helicobacter pylori antibody with myocardial ischemic events in coronary artery disease. AM J Cardiol 2001;87:1005–7.
19. Azarkar Z, Jafarnejad M, Gholamreza. The relationship between helicobacter pylori infection and myocardial infarction. Caspian. J Intern Med 2011; 2(2): 222 – 225.
20. Barker DJP, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birthweight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. Br Med J 1991;303: 671 – 5.

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