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Association of *Helicobacter pylori* and left ventricular ejection fraction in patients with acute myocardial infarction

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ABSTRACT

Background: *Helicobacter pylori* (*H. pylori*) causes continuous inflammation of the stomach. It is suspected that *H. pylori* infection is associated with an inflammatory response in acute myocardial infarction (AMI). This research was aimed at analyzing the association of *Helicobacter pylori* infection and the degree of reduction in left ventricular ejection fraction (LVEF) in patients with AMI, the number of coronary heart disease (CHD) risk factors with *H. pylori* on AMI incident, and association of type of AMI and *H. pylori* on AMI incident.

Method: This is a prospective cohort study with observational-analytic method in AMI patients at ICCU of Prof. Dr. R. D. Kandou General Hospital Manado in 2015-2016. Data retrieval was done by filling out questionnaires, examination of blood samples, serology test to examine the presence of *H. pylori*, and echocardiography examination in patients with AMI.

Results: Statistical analysis showed no significant association between *H. pylori* with the degree of reduction in LVEF in AMI patients (p=0.713), and the number of CHD risk factors (p=0.087). There was a significant association between H. pylori and types of IMA (p=0.017). There were more ST-Elevation Myocardial Infarction (STEMI) patients infected with *H. pylori*.

Conclusion: There is no significant association between *H. pylori* infection and the degree of reduction in LVEF in AMI patients, and also the number of CHD risk factors. However, there is a significant association between AMI type and *H. pylori* on AMI, to wit there are more STEMI patients with *H. pylori* infections.

Keywords: acute myocardial infarction, *Helicobacter pylori,* left ventricular ejection fraction

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INTRODUCTION

AMI is a disease characterized by partial necrosis of the heart muscle, which is one of the most frequent causes of death in the United States and other developed countries. Coronary atherosclerosis is often an underlying factor in the occurrence of myocardial infarction. More recently, a possible association between infectious agents with atherosclerosis has been predicted.[1,2]

According to worldwide data on coronary heart disease in 2001, it was estimated that 11.8 % of all deaths (5.7 million) in low-income countries and 17.3 % of all deaths (1.36 million) in high-income countries were caused by AMI. Approximately there were 865,000 Americans suffer from acute myocardial infarction. *Riset Kesehatan Dasar* (Riskesdas) 2007 data showed that the prevalence of cardiovascular disease in Indonesia ranged from 2.6% in Lampung up to 12.6% in Aceh. The proportion of deaths from the disease reached 4.6%.[3,4] Indonesia's National Health Survey, conducted by the Ministry of Health of the Republic of Indonesia in 2012 showed that cerebral-heart disease was the leading cause of death in Indonesia. A 13-year cohort study in three districts in Jakarta showed that coronary artery disease was the leading cause of death in Jakarta, the capital city of Indonesia.[5]

Helicobacter pylori is a spiral-shaped bacteria, Gram-negative rods with lophotrichous flagella. *H. pylori* infect only humans and is transmitted through the fecal-oral route. Pathogenicity factors include cell motility in order to find the target cells, adhesion to the surface epithelial cells of the stomach, urease will release ammonia from urea to facilitate the survival of the bacterial cells in a highly acidic environment, subsequently, a vacuolating cytotoxin (VacA) of bacterial cells will destroy epithelial cells.[xxx]

Once the pathogen has infected abdominal tissues, acute gastritis will occur. It is a journey that may or may not involve any obvious symptoms.[6] Gastric mucosa is very well protected from bacterial infection, however *H. pylori* has an excellent adaptability to the ecological environment of the stomach, with a unique series of steps to get into the mucosa, swims and performs spatial orientation in the mucosa, attaches to the gastric epithelial cells, evades the immune response, and eventually becomes colonized and give rise to persitent transmission.[7,8] Upon entering the gastrointestinal tract,

this bacterium should avoid bactericidal activity contained in the lumen of the stomach contents, then into the mucous layer. Urease production and motility have very important roles in the initial steps of this infection. Urease hydrolyzes urea into carbon dioxide and ammonia so that *H. pylori* can survive in an acidic environment. Motility is very important in the colonization of bacteria, and flagellum of *H. pylori* is very well adapted to the stomach.[7-10]

H. pylori causes continuous inflammation of the stomach causing discomfort in the chest. It is suspected that there is a corrupting influence of *H. pylori* and its products such as cytokines and cytotoxins in the coronary endothelium, causing a denudation and erosion of plaques of coronary arteries due to toxins released by the bacteria. That's why it has been suspected that *H. pylori* infection is associated with an inflammatory response in acute myocardial infarction (AMI), but this is still under debate.[6,11] This study aimed to determine (i) the association of *H. pylori* and the degree of reduction in LVEF in AMI patients, (ii) the association of the number of CHD risk factors and *H. pylori* and the relation to the severity of AMI incident, and (iii) the association of AMI types and *H. pylori* to the incidence of AMI.

METHOD

A twelve-year-old boy with one-week history of pain on This research is a prospective cohort study with observational-analytic methods to determine the association between *H. pylori* and LVEF in AMI patients. The research was conducted at ICCU of Prof. Dr. R. D. Kandou General Hospital Manado in 2015-2016. The population of these patients are patients with acute myocardial infarction. Affordable population in this study was patients with acute myocardial infarction in ICCU of Prof. Dr. R. D. Kandou General Hospital Manado. Samples in this study were patients with acute myocardial infarction listed as patients at ICCU who did echocardiography. Sampling was conducted purposively.

There were three variables in this research: (1) independent variable (*H. pylori*), (2) dependent variable (LVEF), and (3) confounding variables (age, sex, genetics, history of hypertension, history of diabetes mellitus, dyslipidemia, history of smoking, menopause, and infarct location). The study group was patients with myocardial infarction. Data on the presence of *H. pylori* in patients were (1) primary data based on blood samples directly and (2) secondary data based laboratory results in the patient's medical record. Data retrieval was done by filling out questionnaires and examination of blood samples, then serology test to determine the *H. pylori* antibody-antigen and echocardiography in patients with AMI. Data were analyzed using SPSS version 23 using cross tabulation analysis test to find out the correlation between independent variables and the dependent variables, as well as descriptive analysis to see the distribution of samples.

RESULTS

The number of samples in this study were 120 patients with AMI, consisted of 40 patients infected with *H. pylori* and 80 patients not infected with *H. pylori*. AMI patients who received treatment at the hospital ICCU were 120 people, consisting of 11 people (9.17%) with age <40 and 109 people (90.83%) with age ≥ 40. Meanwhile, of 40 AMI patients with *H. Pylori*, all aged ≥ 40 years (100%). A total of 15 people (12.5%) were female and 105 people (87.5%) were male. The AMI patients with *H. pylori* infection were 49 people and all of them (100 %) were male. Of 120 patients, 65 people (54.17%) did not have history of AMI ran in their family, and 55 people (45.83%) with AMI ran in their family. Of 40 AMI patients with *H. pylori* infection, 25 of them (62.5%) did not have family history with AMI and the other 15 (37.5%) had family history with AMI.

On the measurement of blood pressure, 57 people (47.5%) had systolic blood pressure of <120 mmHg (normal), 32 people (26.66%) with systolic blood pressure between 120-139 mmHg (prehypertension), 8 people (6.66 %) with systolic blood pressure between 140-159 mmHg (stage 1 hypertension), and 23 people (19,16 %) with systolic blood pressure of ≥160 mmHg (stage 2 hypertension). %). Meanwhile, 57 (47.5%) had normal diastolic blood pressure (< 80 mmHg), 28 (23.33%) had prehypertension (diastolic blood pressure between 80-89 mmHg), 28 (23.33%) had stage 1 hypertension (diastolic blood pressure between 90-99 mmHg), and 7 (5.83%) had stage 2 hypertension (diastolic blood pressure of ≥100 mmHg).

Among 40 AMI patients with *H. pylori* infection, 29 of them (72.5 %) had normal systolic blood pressure (< 120 mmHg), 11 (27.5%) had stage 2 hypertension (systolic blood pressure of ≥ 160 mmHg). Meanwhile 22 of them (55%) had normal diastolic blood pressure (<80 mmHg), 9 (22,5%) had prehypertension (diastolic blood pressure between 80-89 mmHg), and 9 (22,5%) had stage 1 hypertension (diastolic blood pressure between 90-99 mmHg). Of 120 AMI patients, 71 (59.17%) did not have diabetes mellitus (DM) history and 49 (40.83%) had DM history. Of 40 AMI patients with *H. pylori* infection, 31 (77.5%) did not have DM history and 9 (22.5%) had DM history.

Table 1 **Demographic Characteristics of Patients. Variables are shown in mean ± standard deviations, NS: not shown, AMI: Acute Myocardial Infarction, DM: Diabetes mellitus, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, TGL: Triglyseride, LVEF: Left Ventricular Ejection Fraction**

Risk Factors	AMI (*n=120*)	AMI *H. pylori* (+) (*n=40*)	Min	Max
Age	52.4±8.95	56.3±7.99	38	78
Sex (M/F)	105/15	40/0		
Family history (-/+)	65/55	25/15		
Blood pressure				
DSystolic	126.3±27.33	124.5±32.60	90	190
Diastolic	80.8±13.84	77.9±11.52	60	98
History of DM (-/+)	71/49	31/9		
Lipid				
Total cholesterol	185.5±51.07	186.7±56.16	70	270
HDL	37.4±11.74	39.8±12.11	20	59
LDL	116.6±45.54	125.8±39.46	47	205
TGL	166.13±53.90	139.0±24.35	90	199
Smoking history (-/+)	19/101	9/31		
Menopause (No/Yes)	109/11	40/0		
Helicobacter Pylori (-/+)	80/40	40/0		
LVEF	49.90±19.07	47.1±18.83	15	80
Number of Risk Factors	5.18±1,24	5.35±1.17	3	7
Infarction location				
Anterior	68	19		
Inferior	18	9		
InteroAn	22	0		
InteroPo	6	6		
Posterior	6	6		

Table 2 **Association between *H. pylori* infection and LVEF in AMI patients. Analyzed using cross tabulation**

		H. pylori			
		Negative	Positive	Total	*p*-value
The number of CHD risk factor	<5	17 (21.25%)	15 (37.5%)	32 (26.67%)	0.087
	5	29 (36.25%)	15 (37.5%)	44 (36.67%)	
	≥6	34 (42.5%)	10 (25%)	44 (36.67%)	
Total		80 (100%)	40 (100%)	120 (100%)	

Table 3 **Association between the number of CHD risk factor and *H. pylori* infection with AMI incident. Analyzed using cross tabulation**

		H. pylori infection			
		Negative	Positive	Total	*p*-value
AMI type	STEMI	36 (45%)	28 (70%)	64 (53.33%)	0.017
	NSTEMI	44 (55%)	12 (30%)	56 (46.67%)	
Total		80 (100%)	40 (100%)	120 (100%)	

On the measurement of cholesterol level, 70 (58.33%) had total cholesterol level of < 200 mg/dL (normal), 31 (25.83%) had 200-239 mg/dL (borderline high), and 19 (15.83%) had ≥240 mg/dL. While of 40 AMI patients with *H. pylori* infection, 22 (55%) of them had normal cholesterol level (< 200 mg/dL), 9 (22.5%) with borderline high cholesterol level (200-239 mg/dL), and 9 (22.5%) had high cholesterol level (≥240 mg/dL). As many as 71 (59.17%) had low level of high density lipoprotein (HDL) (< 40 mg/dL, a major risk factor for heart disease) and 49 (40.83%) had moderate level of HDL (40-59 mg/dL, the higher, the better). Among 40 AMI patients with *H. pylori* infection, 21 (52.5%) had low level of HDL (<40 mg/dL) and 19 (47.5%) had moderate level of HDL (40-59 mg/dL).

On the measurement of low density lipoprotein (LDL) cholesterol, of 120 AMI patients, 48 people (40%) of them had normal LDL level (< 100 mg/dL), 22 (18.33%) had a near optimal/above optimal LDL level (100-129 mg/dL), 22 (18.33%) had a borderline high LDL level (130-159 mg/dL), 22 (18.33%) had a high LDL level (160-189 mg/dL), and 6 (5%) had a very high LDL level (≥190 mg/dL). Of 40 AMI patients with *H. pylori* infection, as many as 9 people (22.5 %) had a normal LDL level (< 100 mg/dL), 17 (42,5%) had a near optimal/above optimal LDL level (100-129 mg/dL), 8 (20%) had a borderline high LDL level (130-159 mg/dL), and 6 (15%) had a very high LDL level (≥190 mg/dL).

On the measurement of triglyceride level, 68 (56,66%) people had normal level (< 150 mg/dL), 29 (24.16%) had a borderline high level (150-199 mg/dL), and 23 (19.16%) had a high level (200-499 mg/dL). Among 40 AMI patients with *H. pylori* infection, as many as 31 people (77.5%), had a normal triglyceride level (< 150 mg/dL) and 9 (22.5%) had a borderline high triglyceride level (150-199 mg/dL).

In this research, 11 patients had already on the stage of menopause. Among 40 AMI patients with *H. pylori* infection, none of them was already menopause since all of them were male. As many as 19 (18.46%) people did not have smoking history and 101 (81.54%) had smoking history. Among 40 AMI patients with *H. pylori* infection, as many as 9 people (28%) did not have smoking history and 31 (72%) had smoking history. Based on the location of infarction, 68 (53.85%) people had an infarction in the anterior part, 18 (15.38%) in the inferior part, 6 (6.15%) in the posterior part 22 (18.46%) in the inferoanterior part, and 6 (6.15%) in the inferoposterior part. Among 40 AMI patients with *H. pylori* infection, 19 (47,5%) of them had an infarction in the anterior part, 9 (22.5%) in the inferior part, 6 (15%) in the posterior part, and 6 (15%) in the inferoposterior part.

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Table 4 **Association between AMI types and *H. pylori* infection with AMI.Analyzed using cross tabulation**

		LVEF				
		≤30%	31-54%	≥55%	Total	*p*-value
H. pylori	Negative	16 (61.54%)	24 (64.86%)	40 (70.18%)	80 (66.67%)	0.713
	Positive	10 (38.46%)	13 (35.14%)	17 (29.82%)	40 (33.33%)	
Total		26 (100%)	37 (100%)	57 (100%)	120 (100%)	

Table 5 **Association between CHD risk factors and *H. pylori* in AMI incidence analyzed using cross tabulation. Variables are shown in mean ± standard deviations. RF: Risk Factors, DM : Diabetes Mellitus, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein**

Risk factors	*H. pylori*		*p*-value
	Positive (*n*=40)	Negative (*n*=80)	
Age	56.3±7.99	50.5±8.83	0.001
Sex (M/F)	40/0	65/15	0.003
Family history (-/+)	25/15	40/40	0.195
Blood pressure			
Syctolic	124.5±32.60	127.2±24.46	0.321
Diastolic	77.9±11.52	82.3±14.71	0.048
DM history (-/+)	31/9	40/40	0.004
Lipid			
Total cholesterol	186.7±56.16	184.9±48.68	0.426
HDL	39.8±12.11	36.2±11.44	0.058
LDL	125.8±39.46	111.9±47.85	0.047
TGL	139.0±24.35	179.7±59.36	0.0001
Smoking history (-/+)	9/31	10/70	0.157
LVEF	47.1±18.83	51.3±19.14	0.126
Number of RF	4.83±1.17	5.35±1.24	0.014
Menopause (-/+)	40/0	69/11	0.009*
IMA (NSTEMI/STEMI)	12/28	44/36	0.010

Based on LVEF, as many as 26 (21.66%) people had LVEF of ≤30%, 37 (30.83%) had LVEF between 31-54%, and 57 (47.5%) had LVEF of ≥55%. Meanwhile, of 40 AMI patients with *H. pylori* infection, 10 (25%) people had LVEF of ≤30%, 13 (32.5%) had LVEF between 31-54%, and 17 (42.5%) had LVEF of ≥55%.

Association between *H. pylori* infection and LVEF in AMI patients

This research showed that there were 16 (61.54%) AMI patients with negative *H. pylori* infection had LVEF of < 30%, 24 (64.86%) had LVEF between 31-54%, and 40 (70.18%) had LVEF of ≥55%. There were 10 (38.46%) AMI patients with positive *H. pylori* infection had LVEF of < 30%, 13 (35.14%) had LVEF between 31-54%, and 17 (29.82%) had LVEF of ≥55%. The Value p=0.713 in this research infers that there was no significant association between the infection of *H. pylori* and the degree of reduction in LVEF in AMI patients (Table 2).

Association between the number of CHD risk factor and *H. pylori* infection with AMI incident

Table 3 shows that there were 17 (21.25%) AMI patients with <5 risk factor did not have *H. pylori* infection, 15 (37.5%) with <5 risk factor had *H. pylori* infection, 29 (36.25%) with 5 risk factor did not have *H. pylori* infection, 15 (37.5%) with 5 risk factor had *H.*

Association between AMI types and *H. pylori* infection with AMI

Table 4 shows that there are 36 (45%) AMI patients with STEMI were not infected by *H. pylori*, 28 (70%) were infected, 44 (55%) AMI patients with Non ST-Elevation Myocardial Infarction (NSTEMI) were not infected by *H. pylori*, 12 (30%) were infected. The value of p=0.017 indicates that there are significant association of AMI types and *H. pylori* infection with AMI.

Association between CHD risk factors and *H. pylori* in AMI incidence

Table 5 shows that risk factors that have significant association with *H. pylori* in AMI incident are age, sex, DM history, diastolic blood pressure, triglyseride level, LDL level, the number of ejection fraction, menopause, and AMI (p=<0.05)

DISCUSSION

AMI is a condition that occurs when the circulation to the heart becomes blocked and resulting in necrosis. AMI prevalence increases with age and other risk factors: hyperlipidemia, diabetes mellitus, smoking, hypertension, male, and family history. All those risk factors contribute to the formation of atherosclerosis.[12-14] This research used 120 AMI patients as sample. The data were obtained from Prof. Dr. R. D. Kandou General Hospital Manado, North Sulawesi. Among 120 cases, there were 40 (33.3%) of them infected by *H. pylori*. This shows that AMI patients with *H. pylori* infection were fewer than those AMI patients with no *H. pylori* infection. This is supported by research conducted by Dario et al. that there were 29% samples of AMI patients infected by *H. pylori* in 100 samples, and 25% in 93 samples as control.[15]

All of 40 samples with *H. pylori* infection were older than 40 years old. This is in accordance with

the theory stating that seropositive of *H. pylori* increased with age. However, the transmission and process of infection is still under debate. Because human is the only host, there is a possibility in developed countries that *H. pylori* infects from siblings or parents through the gastro-oral route. While in developing countries, fecal-oral may occur.[16] All 40 AMI patients who are infected by *H. pylori* are male. This is similar to research done by Sreenivasan, *that H. pylori* infection is more dominant in males than females. The differences in the genders may be due to the different lifestyle and habits of a community of men and women, especially in smoking and alcohol drinking, that may activate *H. pylori* infection. A recent study showed that *H. pylori* used 70kDa Lf receptor to obtain iron substance directly from stomach. Because the smoke from the tobacco leaf has 84 ug/g of iron, then smoking is one of the strongest risk factors. While in women, loss of menstrual blood can be considered as a factor to lower iron content, so the prevalence of *H. pylori* is lower.[17]

This research found that 15 (37.5%) of the samples with *H. pylori* infection had family history of AMI, while 25 (62.5%) had no family history of AMI. This shows that *H. pylori* infection is fewer in samples with AMI history in family than without AMI history. This may happen because there are studies indicated that there were no significant association between *H. pylori* and AMI history in family.[18]

Based on data in this research, of 40 samples with *H. pylori* infection, as many as 31 (77.5%) patients did not have DM history and 9 (22.5) did. This shows that patients with *H. pylori* infection that did not have DM history are more susceptible than those who did. This result does not correspond to the research conducted by Abdulbari et al. in Qatar that the prevalence samples with DM and *H. pylori* infection was 63.3%.[19]

Statistical analysis showed that there was no significant association between *H. pylori* infection and the degree of reduction in LVEF in AMI patients (p=0.713). In this research, most of patients with AMI and *H. pylori* infection had LVEF of ≥ 30%. This is in line with research conducted by Dario et al. that there was no significant association between *H. pylori* infection and the severity of AMI incident. Dario et al. used the number of coronary arteries affected by infarction in measuring the severity of AMI incident, while this research used the results of echocardiography in the form of LVEF. Jung Tsai et al. also did not find significant association between *H. pylori* infection and the severity of AMI incident, even though there was higher incident in three affected coronary artery. Further study needs to be conducted in order to gain more information about the role of *H. pylori* in the development of AMI and CHD.[15]

Statistical analysis showed that there was no significant association between the number of CHD risk factors (p=0.087) with *H. pylori* infection on AMI incident. There is no previous research conducted to support this result, however this result occurred allegedly because there is no significant association between *H. pylori* with acute myocardial infarction, so there is a need for reviewing the diagnosis through endoscopic biopsy, which is the gold standard for the diagnosis of *H. pylori* infection. Statistical analysis showed significant association between AMI type and *H. pylori* on AMI incident (p=0.017). This finding supports a case-controlled study showing that there were an association between *H. pylori* infection and AMI type. According to research conducted by McDonagh,[21] there were an association between seropositive on *H. pylori* and AMI type on random male and female samples aged 25-74 in North Glasgow (in the area with high mortality for AMI). Kowalski et al.[22] proved that *H. pylori* significantly weakened the reduction in the lumen of the coronary arteries in patients with AMI after percutaneous coronary angioplasty, probably by reducing the effects of proinflammatory cytokines.

This research showed that the risk factors which are significantly associated with *H. pylori* on AMI incident among others are sex, DM history, diastolic blood pressure, triglyceride level, LDL level, number of ejection fraction, menopause and AMI (p=<0.05). Several mechanisms have been proposed to explain the association between *H. pylori* infection with blood lipids, and several others showed that viral and bacterial infection can change lipid metabolism from infected cells. Other research showed that lipid increased as a result of the body's attempt to fight infection. Other finding showed that LDL had antimicrobial property and was involved directly in deactivating microbial pathogens. This has been confirmed by a research which showed that mice with defective LDL receptor would increase levels of LDL so it can protect mice against infection by gram-negative bacteria such as *H. pylori*.[20]

1. A finding by Rogha et al.[23] showed that *H. pylori* infection was one of possible risk factors for acute myocardial infarction with a history of diabetes mellitus, dyslipidemia, hypertension, status of C-reactive protein and IL-6 levels. The mechanism of the relationship between *H. pylori* infection and acute myocardial infarction are as follows:

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2. The damaging effects of *H. pylori* and its products such as cytokines, cytotoxins in the coronary endothelium.
3. Activation of immune mechanisms by these bacteria which react with the core of monocytes in atherosclerotic vessel wall and cytoplasm of fibroblast cells in atherosclerotic plaque.
4. Induction of *H. pylori* in the release of nitric oxide by the vascular endothelium disrupts fibrinogen levels that cause normal capacity reduction of muscle relaxation and causes vasoconstriction and adverse hemodynamic balance.
5. These infections raised the thromboxane measured as a result of thromboxane B (TXB) resulting in platelet activation.
6. There were denudation and erosion of plaques of coronary arteries due to toxins released by *H. pylori*.

As a result, plaque rupture occured either partial or total which resulted in acute myocardial infarction.

CONCLUSION

In conclusion, this research infers that there was no significant relationship between *H. pylori* infection and the number of CHD risk factors with the degree of reduction in LVEF in AMI patients. However, there was significant association between AMI type and *H. pylori* infection on AMI. This is shown by more STEMI patients compared to NSTEMI patients infected by *H. pylori*. Nevertheless, this study can be used as a consideration so that the doctor can make the *H. pylori* as a risk factor when associated with myocardial infarction.

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