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THE DIFFERENCES LEVEL OF ADIPONECTIN BETWEEN POSITIVE AND NEGATIVE HELICOBACTER PYLORI GASTRITIS AND ITS CORRELATION WITH NUTRITIONAL STATUS

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Abstract
Gastritis is an inflammatory process in the gastric mucous and submucous in response to injury, which can be acute or chronic. Helicobacter pylori bacterial infection is the most common cause of chronic gastritis active throughout the world. The incidence of gastritis increases gradually because adiponectin levels decrease, this is because adiponectin protects the stomach from increased gastric acid through the anti-inflammatory effect of adiponectin. The purpose of this study was to determine the correlation between adiponectin and *H. pylori* gastritis and to find the relationship between adiponectin and nutritional status in *H. pylori* gastritis patients.

In this study, there was difference adiponectinlevel between *H. pylori* infection, that hipoadiponectinemia has increased risk *H. pylori* infection. This study also found adiponectin serum was lower in higher body mass index and abdominal circumference.

Introduction
Dyspepsia is a syndrome consisting of pain or discomfort in the epigastrium, nausea, vomiting, bloating, rapid satiety, full stomach feeling, belching, regurgitation and a burning sensation that spreads in the chest. In Indonesia it is estimated that there are 30% of cases in general practice and 60% in specialist practice.[1]

Gastritis is an inflammatory process in the gastric mucosa and submucosa in response to injury, which can be acute or chronic. Helicobacter pylori bacterial infection is the most common cause of chronic gastritis active throughout the world.[3]Helicobacter pylori infection is estimated to occur in 50% of the population in the world where 70-90% of these infections occur in developing countries and only 40-50% in industrialized countries.[4] NSAIDs are the second risk factor for peptic ulcer after *H. pylori* gastritis.[3] About 11% population in US experiences this problem.[4]

H. pylori infection induces activation of proinflammatory cytokines such as IL-1β, IL-6, TNF-α, IL-8 via NF-κB. The inflammatory response that occurs causes Treg to secrete immunosuppressive cytokines, which maintain levels of *H. pylori* in the gastric mucosa.[5] Epidemiological studies show that there is a strong association between *H. pylori* infection and gastritis with the presence of a duodenum or stomach ulcer. WHO has established these germs as class I gastric carcinogens. [6]

Obesity is a risk factor for gastric cancer mainly because obesity increases the incidence of gastroesophageal reflux, which can damage the mucosa around the gastric cardia, resulting in a increase risk of cancer.[7]

Adiponectin has been a research topic for the past few decades.[8]Hypoadiponectinemia is associated with a variety of diseases including diabetes, hypertension, dyslipidemia, metabolic syndrome, atherosclerosis, non alcoholic fatty liver disease (NAFLD), gastritis, gastroesophageal reflux disease (GERD), inflammatory bowel disease (IBD), pancreatitis and malignancy. In previous studies it was found that the anti-inflammatory

properties of adiponectin have a protective effect on gastrointestinal diseases, such as pancreatitis and gallstones.[9]

Adiponectin levels in blood circulation are found below normal in obese patients. Adiponectin concentration in blood circulation is inversely proportional to visceral fat, but not with body mass index and subcutaneous fat. Decrease in visceral fat will increase adiponectin levels in blood circulation.[10]

The incidence of gastritis increases gradually because adiponectinlevels decrease, this is because adiponectinprotects the stomach from increased gastric acid through the anti-inflammatory effect of adiponectin.[11]

The purpose of this study was to determine the relationship between adiponectin and *H. pylori* gastritis and to find the relationship between adiponectin and nutritional status in *H. pylori* gastritis patients.

Method

This study is a descriptive study with a cross sectional approach. The study will be conducted in July 2019 - December 2019 at the Endoscopic Unit of H. Adam Malik Hospital and Permata Bunda Hospital Medan, North Sumatra. The target population of the study was gastritis patients, while the affordable population was gastritis patients who came to H. Adam Malik Hospital and Permata Bunda Hospital Medan. The sample was gastritis patients who met the inclusion and exclusion criteria. The inclusion criteria were patients aged 18-70 years who were diagnosed with gastritis through histopathological examination, were willing to voluntarily participate in the study after being given an explanation. The exclusion criteria were pregnant woman, patient who had received *H. pylori* eradication therapy in the last 6 months or were on antibiotic therapy commonly used in eradication therapy, consumption of Proton Pump Inhibitors, H2 receptor antagonists, NSAIDs, steroids, alcohol during the last 48 hours, suffering from systemic diseases and malignancies, and patients who refused to participate.

Operational definition

Dyspepsia syndrome is based on questionnaires that refer to Rome III criteria: one or more symptoms of feeling full after eating, feeling full quickly, epigastric pain, or burning sensation in the epigastrium. Complaints occur for the last 3 months with symptom onset at least 6 months before the diagnosis is made. The diagnosis of gastritis is confirmed by endoscopic and histopathology examination. H pylori infection was confirmed by examination of Campylobacter like organism (CLO), ^{14}C-Urea Breath Test (^{14}C-UBT), and Immuno histochemistry (IHC) *H. pylori*. Assessment of nutritional status included: body mass index, arm circumference, abdominal circumference, albumin, and haemoglobin.

Data analysis

The data will be analyzed with univariate analysis. The data will be analyzed with independent T test if the data is normally distributed, and if the data is not normally distributed the Mann Whitney test will be used. The independent T test is a comparative test where the variable is nominal data and ratio. The desired deviation (α) is 0.05. The correlation between nutritional status and adiponectin levels will be analyzed. If the data are normally distributed Pearson correlation will be used, if not normally distributed Spearman correlation will be used.

Results

From 60 patients enrolled in the study, 34 (56,7%) were male and 26 (36%) were female. The mean age 46,5 and the most subjects were Bataknese. In this study 29 patients were HP + (48,3%) and 31 patients HP – (51,7%), and the mean BMI in HP + and HP – groups was 24,22±4 and 22,53±3, respectively, and it was not statiscally significant (p=0,103).

The mean abdominal circumference (AC) in HP + and HP – groups was 86,6±8,7 cm and 82,5±7,4 cm, respectively, and it was not statistically significant (p=0,057). In HP + and HP – groups, the mean Haemoglobin was 12,5 and 14,6, and it was statistically significant (p=0.009). The mean albumin was 4,6±0,6 and 4,5±0,6 in HP + and HP – groups, and not statistically significant (p=0.53).

Tabel 4.1 Characteristics Of Subjects

Variable	n=60	%
Age, (years)[b]	46,5 (24-66)	
AC(cm)[a]	84,5±8,3	
BMI(kg/m^2)[a]	23,35±3,98	
Sex		
Male	34	56,7
Female	26	43,3
Ethnic		
Bataknese	37	61,7
Javanese	18	30,0
Acehnese	5	8,3
Occupation		
Enterpreneur	27	45,0
Housewife	14	23,3
Employee	16	26,7
Civil servants	3	5,0
Education		
Elementary school	3	5,0
Middle school	14	23,3
High school	32	53,3
University	11	18,3
H pylori		
Positif	29	48,3
Negatif	31	51,7

Table 4.2 Comparison Clinical And Biochemical Finding Based H. pylori Infection

Variable	H pylori (+)	H pylori (-)	P
Ages (years)[b]	48,9±9,0	45,1±10,7	0,405
Sex			0,768
Male	17	17	
Female	12	14	
BMI (kg/m^2)[a]	24,22±4,52	22,53±3,26	0,103
AC(cm)[a]	86,6±8,7	82,5±7,4	0,057
Haemoglobin (g/dL)[b]	12,5 (10,4-16,0)	14,6 (10,4-16,0)	0,009*
Albumin (g/dL)[b]	4,6±0,6	4,5±0,6	0,530
Adiponectin[b]	6,24 (2,46-7,78)	6,96 (5,40-7,89)	0,007*

In HP + and HP – groups, the mean serum adiponectinwere 6,24 and 6,96, it was statistically significant (p=0.007). There was negative correlation between adiponectinand BMI, it was statistically significant (p=0.001). This study found negative correlation between adiponectinand abdominal circumference, and it was statistically significant (p<0.001).

Negative correlation was found in this study, between adiponectinand albumin, it was not statistically significant (p=0.808). Positive correlation was found between adiponectinand haemoglobin, but not statistically significant (p=0.849).

Table 4.3 Correlation BetweenAdiponectin and Nutritional Status

Variable	Adiponectin (r)	p
Haemoglobin	0,025	0,849
Albumin	-0,032	0,808
BMI	-0,407	0,001*
AC	-0,474	<0,001*

Discussion

In this study, there was decrease adiponectinlevel in positive *H. pylori* group compare to negative group. Same as previous study by Kishida et al 2014, in addition to gastritis lower adiponectinalso found in the other inflammatory condition as pancreatitis, IBD, NAFLD and obesity. The mechanism how hipoadiponectinemia found in *H. pylori* infection is still remain unknown. The mechanism that can be occur is because adiponectinprotects the stomach from increased gastric acid through the anti-inflammatory effect of adiponectin.

In Vafaeimanesh, et al(2014) obtained levels adiponectinserum no different statistically in the *H. pylori* positive than group *H. pylori* negative in people with diabetes mellitus. Research is equal to Zhang, et al(2016) that the increase in infection *H. pylori* because of a decrease in adiponectinserum caused by polymorphism genes promoter adiponectinin patients NAFLD.The same thing found in Ando,et al (2012) , that there are increased levels of adiponectinserum after conducted eradication *H. pylori* in Japanese.

Conclusion

In this study, there was difference adiponectinlevel between *H. pylori* infection, that hipoadiponectinemia has inreased risk *H. pylori* infection. This study also found adiponectinserum was lower in higher BMI and abdominal circumference.

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