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COMPARISON OF TOTAL ANTIOXIDANT SERUM LEVEL IN POSITIVE AND NEGATIVE H. PYLORI GASTRITIS PATIENTS

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Abstract

Gastritis is an inflammatory of stomach that can occur either acutely or gradually as a chronic condition. Incidence of gastritis in Indonesia is high, which is 274,396 cases out of 238,452,952 inhabitants. One of the most common causes of gastritis is *Helicobacter pylori* (*H. pylori*).Gastric mucosa damage can be caused by Reactive Oxygen Species (ROS) which stimulated by *H. pylori*. Cells are protected against the harmful effects of ROS by antioxidant enzymes and other non-enzymatic antioxidants, so that the amount of antioxidant and free radicals in the body can be balanced. There were 40 samples obtained through consecutive sampling in March-July 2018. Gastritis was ensured by endoscopy (Olympus, Tokyo, Japan). *H.pylori* is established through a change in color from yellow to red, magenta, pink, or orange in the examination of Campylobacter Like Organism test (CLO). Total antioxidants level examination using a serum total antioxidant kit with the ELISA method (kit Randox Total Antioxidant, Cat. No. NX 2331, Randox Laboratories Ltd, UK). Data were collected and analyzed using SPSS version 22. There was significant association between level of total antioxidant serum and gastritis due to*H. pylori* infection with p value p = 0.004.

Introduction

Gastritis is an inflammatoryin the stomach tissue that can occur either acutely or gradually as a chronic condition.(1) Incidence of gastritis in Indonesia is high, which is 274,396 cases out of 238,452,952 inhabitants. One of the most common causes of gastritis is Helicobacter pylori (*H. pylori*).(2-5) Gastric mucosa damage can be caused by Reactive Oxygen Species (ROS)which stimulated by *H. pylori*. ROS played important pathogenic factor through fat peroxidation and covalent bonds which results in basal degradation of epithelial membranes, disruption of cell metabolism, and disruption of deoxyribonucleic acid (DNA). Cells are protected against the harmful effects of ROS by antioxidant enzymes and other non-enzymatic antioxidants, so that the amount of antioxidant and free radicals in the body can be balanced.(6) *H. pylori* stimulates the production of ROS and results the expression of inflammatory mediators caused of an imbalance in the process of apoptosis and proliferation of infected tissue. Oxidative stress can also be defined as an increase in oxidants or a decrease in antioxidant capacity, and various oxidants and other antioxidants have an additive effect on oxidative status. (7)

Several studies have been conducted to assess antioxidant levels in gastritis patient. Study by Uwa et al found that total antioxidant capacity was significantly lower in the positive group of *H. pylori* compared to the negative group *H. pylori* (1.36 ± 0.33 and 1.70 ± 0.50; P <0.001).(7) Whereas Basyigit et al showed that total oxidant status, total antioxidant capacity and oxidative stress index value were significantly higher in subjects with positive *H. pylori* compared to the negative (p <0.05) .(6) This aims of this study to determine serum ratio of total antioxidant levels between positive and negative *H. pylori* gastritis patients.

Method

Patient Selected

There were 40 samples obtained through consecutive sampling in March-July 2018. Gastritis was ensured by endoscopy (Olympus, Tokyo, Japan). Mucosa undergoes edema, erythema (spotted, patchy, linear), exudate, bleeding, erosion and histopathology (marked by inflammatory cells in the gastric mucosa) is diagnosed with gastritis. *H.pylori* is established through a change in color from yellow to red, magenta, pink, and orange in the examination of Campylobacter Like Organism test(CLO).Total antioxidants level examination using a serum total antioxidant kit with the ELISA method (kit Randox Total Antioxidant, Cat. No. NX 2331, Randox Laboratories Ltd, UK).

Data analysis

Data analysis of total antioxidant level on *H. pylori* gastritis patients were univariate and bivariate. Univariate analysis to determine the characteristics of *H. pylori* gastritis patients and the prevalence of *H. pylori* gastritis patients. Bivariate analysis to determine the ratio of total serum antioxidant levels between positive and negative *H. pylori* gastritis patients using independent T-test if the data are normally distributed, or Mann Whitney U test if the data are abnormally distributed. All data were analysed by SPSS 22 version. A value of $p< 0.05$ with a 95% confidence interval was considered statistically significant.

Result

The proportion of gastritis was more in male (62.5%) than in female (37.5%). Based on ethnicity, the highest proportion of gastritis sufferers was Batak (57.5%), and the lowest was Acehnese (12.5%). Based on the occupation, the highest was housewives (40%) and civil servants (10%) become the lowest. Based on education, the highest proportion of patients is at high school level (70%) and the lowest proportion at the University level (5%). Mean of body mass index (BMI) was 24.16 ± 3,62 kg / m2 and mean of total antioxidant serum levels obtained in the study was 1.72 ± 0,36 mmol / L (Table 1). There was no relationship between sex, occupational rate, and education with *H. pylori* infection. There were no differences in age and BMI in both groups (Table 2).

Table 1 . Baseline and clinical characteristics of subjects

Socio-demographics	N=40
Gender	
Male	25 (62.5%)
Female	15 (37.5%)
Ethnicity	
Batak	23 (57.5%)
Jawa	12 (30.0%)
Aceh	5 (12.5%)
Occupation	
Private employees	12 (30.0%)
Housewives	16 (40.0%)
Enterpriser	8 (20%)
Civil Servants	4 (10%)
Education	
Elementary	3 (7.5%)
Middle school	7 (17.5%)
High School	28 (&0%)
University	2 (5.0%)
Gastritis	
H. pylori (+)	20 (50%)
H. pylori (-)	20 (50%)
BMI * (kg/m2)	24,16 ± 3,62
ATS ** (mmol/L)	1,72 ± 0,36

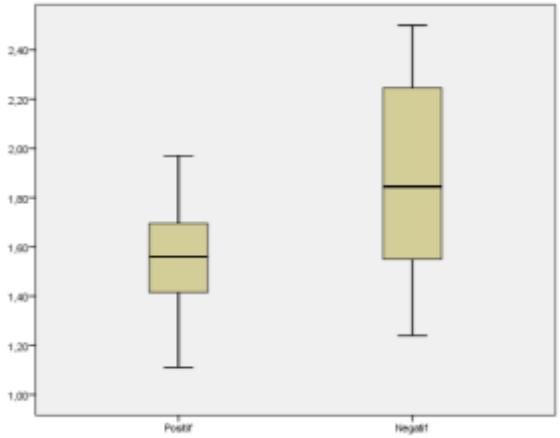
*BMI : Body mass index
** ATS : antioxidant total serum

Table 2 : Correlation between characteristics of subjects and H. pylori Infection

Variabel	***Helicobacter pylori***		**P**
	Positive	**Negative**	
Gender			
Male	12 (48)	13 (52)	0,744
Female	8 (53,3)	7 (46,7)	
Age (years)	48,9 ± 14,91	47,25 ± 13,3	0,714
Ethnicity			
Batak	11 (47,8)	12 (52,2)	0,749

Jawa	7 (58,3)	5 (41,7)	
Aceh	2 (40)	3 (60)	
Occupation, n (%)			
Housewives	8 (50)	8 (50)	0,841
Civil Servants	2 (50)	2 (50)	
Private employees	7 (58,3)	5 (41,7)	
Enterpriser	3 (37,5)	5 (62,5)	
Education, n (%)			
Elementary+Junior High School	4 (40)	6 (60)	0,465
Senior high school + University	16 (53,3)	14 (46,7)	
Body Mass Index kg/m^2	24,65 ± 3,84	23,66 ± 3,42	0,395

There was a significant difference in ATS levels between positive and negative *H. pylori* gastritis (p = 0.004). ATS levels were significantly lower in positive *H. pylori* gastritis (1.56 + 0.23 mmol / L) than negative (1.88 + 0.4 mmol / L).(Picture 1 and Table 3)



Helicobacter pylori

Picture 1 : Boxplot Diagram of Antioxidant total serum levels between positive and negative H. pylori

Table 3. Differences of ATS Levels between positive and negative H. pylori Gastritis Patients

Variabel	***Helicobacter pylori***		**P**
	Positif	**Negatif**	
TAS, mmol/L	1,56 ± 0,23	1,88 ± 0,4	0,004*

Discussion

In this study, it was showed that majority of gastritis patients were male (62.5%). This result is similar to the research conducted by Chen, *et al* in China with 2,051 people (51.7%) of whom were men.(8) Other study by Dairi, et al in Indonesia also showed similar results that 24 people (60%) patients with gastritis are men.(9) However, study by Mahmoud et al in Egypt showed the inversely result that 255 people (63.8%) gastritis sufferers are women.(10) This difference may be due to the influencing by various other factors, such as racial, demographic, and other comorbid factors such as Gastroesophageal Reflux Disease disorders.(11) Some factors in the work are known to affect the virulence of *H. pylori* and also the immune response to infection. In this study, housewives was obtained the highest percentage. A study by Tajalli et al found things be related to the findings of this study, where gastritis is more common among women who live in a crowded environment. This is because these women are more likely to have close contact with children, which is said to often transmit *H.*

pylori infection.Most gastritis sufferers were found in the Batak tribe (57.5%), followed by Javanese (20%) and Acehnese (12.5%). Our previous study also showed similar results where gastritis sufferers were most often found in the Batak tribe (57.5%) then Javanese (30%) and Acehnese (12.5%). There were 20 cases (50%) gastritis due to *H. pylori* infection and 20 other cases (50%) were non *H. pylori* gastritis. Epidemiological data suggest that the spread of H. pylori infection depends largely on geography, socio-economic status, hygiene and the environment in which it lives.(12)In this study there were differences in the mean levels of total antioxidant status between gastritis due to *H. pylori* and non *H. pylori* gastritis. This is supported by a study by Aslan *et al* who found a significantly lower association between total levels of antioxidant in the*H. pylori*gastritis than non *H. pylori* patients ($p < 0.001$), with a significantly higher total ROS ($p < 0.1$).(13) Navvabi *et al* in Iran also found similar result when assessing the relationship between *H. pylori* infection and a decrease in total antioxidant capacity in gastric epithelial cells ($p < 0.1$).(14) Another study conducted by Sadreddini *et al*in Iran also showed a decrease in the value of ferric reducing antioxidant power (FRAP), a method in assessing antioxidant levels in the body, which was significant in the group of patients infected with *H. pylori* compared to those who did not ($p = 0.028$).Inflammatory cells will stimulate the production of ROS in In infected tissue. *H. pylori* activates NADPH oxidase which produces ROS. ROS mediates the activation of the enzyme mitogen-activated protein kinase (MAPKs) and transcription factors that are sensitive to the redox, NF-κB and activator protein-1 (AP-1) processes, which induce expression of inflammatory mediators such as interleukin [IL] -8, inducible nitric oxide synthase [iNOS], and COX-2 in gastric epithelial cells. ROS induces lipid peroxidation and tissue damage. In addition, ROS interferes with the immune system, which stimulates the response and release of type 1 (Th1) and interferon (IFN) -γ helper T cells into the network.

Conclusion

There is an association between serum level of antioxidant and gastritis due to *H.pylori* infection. There was lower total antioxidant serum levels in the group of gastritis patients due to *H. pylori* infection than non *H. pylori* gastritis patients.

References

1. Elseweidy MM. *Helicobacter pylori* infection and its relevant to chronic gastritis.*Altern Integr Med*.2017; 6(1): 231.
2. Yulida E, Oktaviyanti IK, Rosida L. Gambaran derajat infiltrasi sel radang dan infeksi *Helicobacter pylori* pada biopsi lambung pasien gastritis. *Berkala Kedokteran*. 2013; (1):47-58.
3. Toljamo K. Gastric erosions – clinical significance and pathology. A long-term follow-up study. *Acta Univ Oul*; 2012: 1-80.
4. Rugge M, Pennelli G, Pilozzi E, Fassan M, Ingravallo G, Russo VM, et al. Gastritis: the histology report. *Digestive and Liver Disease*. 2011; 43S:S373–84.
5. Salama NR, Hartung ML, Muller A. Life in the human stomach: persistence strategies of the bacterial pathogen *Helicobacter pylori. Nature Reviews. Microbiology*. 2013; 11(6):385-399.
6. Liqing L, Kong L, Song H. The therapeutic effect of zerumbone on chronic gastritis via antioxidant mechanisms. *Experimental and Therapeutic Medicine*. 2017; 14:2505-2510.
7. Uwa LM. The Anti-aging Efficacy of Antioxidants. *Curr Trends Biomedical Eng & Biosci*. 2017; 7(4): 555716
8. Chen S, Ying L, Kong M, Zhang Y, Li Y. The Prevalence of *Helicobacter pylori*Infection Decreases with Older Age in Atrophic Gastritis. *Gastroenterology Research and Practice*. 2013;2013:1-7.
9. Dairi L, Siregar G, Sungkar T. The Comparison of Serum Malondialdehyde Level Between *H. pylori* Positive and *H. pylori* Negative Gastritis Patients. *The Indonesian Journal of Gastroenterology, Hepatology and Digestive Endoscopy.* 2018; 19(1): 1-4.
10. Mahmoud S, Gasmi F, Solan Y, Al-Harbi F, Al-Harbi S, Hummadi T, et.al. Prevalence and Predictors of Gastritis among Patients Attending Health Care Facilities in Jazan, KSA. *International Journal of Preventive and Public Health Sciences*. 2016; 2(1): 1-8.
11. Roland B, Lee D, Salem A, Ibrahim S, Mori Y, Meltzer S. Prevalence and Associated Risk Factors of *Helicobacter pylori* Negative Gastritis. *Journal of Gastrointestinal & Digestive System*. 2016;06(01).

12. Binh T, Tuan V, Dung H, Tung P, Tri T, Thuan N et al. Molecular Epidemiology of *Helicobacter pylori* Infection in a Minor Ethnic Group of Vietnam: A Multiethnic, Population-Based Study. *International Journal of Molecular Sciences*. 2018;19(3):708.
13. Aslan, M., Nazligul, Y., Horoz, M., Bolukbas, C., Bolukbas, F., Aksoy, N., Celik, H. and Erel, O. Serum prolidase activity and oxidative status in *Helicobacter pylori* infection. *Clinical Biochemistry*, 2007; 40(1-2):37-40.
14. Azita, N., Mohammad, H., Nazila, N., Sara, K. and Yousef, R. Effect of *Helicobacter pylori* infection on oxidative stresses in patients with chronic gastritis. *African Journal of Microbiology Research*, 2013;7(50):5632-5636.
15. Talley NJ, Vakil N. Guidelines for the Management of Dyspepsia. *Am J Gastroenterol*. 2005;100:2324–2337.