Gastric Mucosa Mucous Layer Thickness in Liver Cirrhosis with Portal Hypertensive Gastropathy Compare to Functional Dyspepsia

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ABSTRACT

Background: This study aimed to investigate gastric mucosa mucous layer thickness in portal hypertensive gastropathy (PHG) compare to normal mucosa in functional dyspepsia and its correlation with several variables such as child class, severity of esophageal varices and gastropathy.

Materials and Methods: Biopsy specimens were taken from the antrum and corpus from both group of patients with PHG and functional dyspepsia. The specimen was given cryometric for frozen section. Tissue were sliced by sagital section 11 µm, placed in object glass, fixed and stained to evaluate mucous thickness and giemsa stained to observe Helicobacter pylori. Measurement of mucous thickness was done upward muscularis mucosa started from upper epithelial layer from foveale tip until outer mucous layer on 15 points which were marked randomly and calculate the mean value by micrometer (µm).

Results: Mean value of antral mucous thickness in PHG was 13.30 ± 6.5 µm, while in the functional dyspepsia it was 25.59 ± 5.66 µm. Statistical analysis for both kinds of mucous thickness was $p<0.001$. Mean corpus mucous thickness in PHG was 10.6 ± 6.81 µm, while mucous thickness in dyspepsia was 32.54 ± 6.51 µm. Statistical analysis revealed $p<0.001$. This result showed significant difference of mucous thickness of antrum and corpus statistically between PHG and dyspepsia as control group.

Conclusion: The study had proven the presence of decreased gastric mucosa mucous layer thickness in corpus and antrum in PHG. Thus, therapeutic approach to increase mucous thickness must be considered in patients with PHG.

Keywords: *Gastric mucosa, PHG, liver cirrhosis*

INTRODUCTION

Liver cirrhosis has been a major health problem in all over the world caused by alcoholism in western countries and viral hepatitis B and C in the Asian and African countries. In India, about 56% of liver cirrhosis caused by viral hepatitis B, 11% by viral hepatitis C and 14% by co-infection of viral hepatitis B and C.[1] According to Vital and Health Statistic 1998 in the US liver cirrhosis had been the 10th leading cause of death. It had mortality rate of 10,368 to 24,025 people per year with mean mortality rate 9.4 per 100,000 population.[2] In Indonesia, it was reported that 38% to 52% of hospitalized patients had liver cirrhosis.[3] Most frequent complications of liver cirrhosis are portal hypertension and liver carcinoma. One of the most severe complications is upper gastrointestinal bleeding due to rupture of varices.[4] About 60% of varices were found in decompensate liver cirrhosis, while 30% found in compensated ones.[5] More than 30% patients with varices have high risk of spontaneous bleeding. Mortality rate in initial bleeding is very high reaching more than 30%. Recurrent bleeding will occur ±70% after initial bleeding. Mortality rate per year after variceal bleeding ranging from 32% to 80%.[6] The mortality rate depends on Child class and almost 90% Child C patients will die in 12 months.[7]

Source of bleeding may originate from gastric mucosa lesion and is assumed due to portal hypertension.[7-9] Issue on this matter had been developing and incidence of PHG had been reported to worsen after variceal eradication by sclerotheraphy with higher risk of bleeding.[10-12] In 1985, Mc Cormack et al[13] had mentioned that morphologic study described vascular dilatation in mucosa and submucosa without inflammation. Thus, the gastric lesion was more likely due to congestion than gastritis which was also supported by Pique.[10]

Gastric mucosal lesion in patients with portal hypertension had been identified on agreement in the Baveno II Consensus in Italy (1996) with the term *portal hypertensive gastropathy.*[14] Diagnosis of portal hypertensive gastropathy (PHG) is based on combination of endoscopic and histopathologic findings indicates changes in gastric mucosal conditions associated with the presence of dilatation and vascular ectasia of mucosal and submucosal microvascular structure without significant evidence of inflammation.[10,13,15] Endoscopic description of gastric mucosa in PHG according to OMED may be classified into mild and severe grade. If we find scarlatina rash, snake skin appearance or mosaic pattern appearance indicate mild grade, while cherry red spot and black brown spot of diffuse mucous bleeding are sign of more severe grade.[10,16,17]

Toyonaga et al[18] reported the incidence of PHG was varied ranging from 4% to 98% with mean prevalence of 53% cases of portal hypertension due to liver cirrhosis. The prevalence of PHF had increased in accordance with severity of liver disease. This was supported by several studies conducted by Iwao,[19] D'Amico et al,[20] and Nasution[21] found that Severe PHG had impaired liver metabolism. In contrast, Vigneri et al[22] and Ohta et al[23] reported no correlation between endoscopic findings, severity of liver disease (Child Pugh score)[24] and esophageal varices grade (Beppu score).

The exact mechanism and pathogenesis of PHG remains unclear, but portal hypertension might be caused by vascular resistance and increased pressure in portal system.[10,18,19,25] How the blood flows in the mucosa of PHG is still controversial. Several researchers found association between size of varices with hepatic portal venous pressure gradient and the incidence of PHG.[23] However, others found decreased gastric mucosal blood flow (congestion).[9,10,14,26,27] A study had reported that PHG was occurred caused by increased portal pressure and decreased hepatic blood flow.[19] Another one reported and claimed that increased portal vein pressure was the only etiologic cause of PHG. There were still controversies on gastric mucosal blood flow. One study had reported it to be increased and another had reported the other way around.[19,28,29] Imanishi et al[30] conducted an experimental study in mice had found that hemodynamic changes due to PHG may cause thinning of gastric mucosa mucous layer.

Gastric acid secretion activity decreased because gastric mucosal barrier is damaged, thus it causes local hemodynamic changes resulting active and passive congestion and hyperemic gastric mucosa.[18] Humoral factors have role in PHG by decreasing mucosa metabolic function, decreased response to pentagastrin, decreased mucosa glycoprotein, decreased prostaglandin E2 level and increased nitrite oxide synthesis. All these will make the luminal gastric acid decreased and cause reduced response of defensive factors to intraluminal stimulation of inciting factors such as H^+ ionic back-diffusion, bile acid and NSAID.[18] All may cause electrical potential changes in the mucosa and increased fragility of gastric mucosa to injury.[8,9,14,18,19]

This study aimed to evaluate the changes in gastric

mucosa mucous thickness with pathophysiology of PHG by comparing the differences of mucous thickness of corpus and antrum between patients with PHG and functional dyspepsia as control group. Aside from that, this study also evaluate the relationship between several factors like severity of liver cirrhosis, esophageal varices grade, gastropathy, endoscopic findings and Helicobacter pylori infection with mucous layer thickness in PHG. This study will evaluate the gastric mucosa mucous thickness in PHG and try to identify the various factors involved. Thus, it could be used in basic clinical research on improving mucous layer as treatment target in PHG. Further research on the benefit of using cytoprotective drugs in PHG hopefully will follow this study.

MATERIALS AND METHODS

Methods of mucous checked measurement should be described. Study design was a cross sectional study comparing two group of patients. One group of patients with PHG and another one with functional dyspepsia as control group.

Population and Sample

Sample subjects were recruited purposively from patients with liver cirrhosis in gastroenterohepatology ward and policlinics in our hospital. Liver cirrhotic patients with PHG who met the inclusion criteria were included and patients with functional dyspepsia as control group.

Research Criteria

Inclusion criteria:

Liver cirrhotic patients with PHG, fully alert, age between 13-65 years old, not in condition of active bleeding and agreed to participate in this study

Exclusion criteria:

Hemostasis disorder, had underwent intervention procedure such as STE/ligation/portosystemic shunt, taking cytoprotective drugs more than one month, history of hematemesis melena due to erosive gastritis, taking drugs which decrease portal hypertension, chronic kidney disease and other severe co-morbid conditions

Control group:

Dyspepsia patients according to Talley's criteria, age between 13-65 years old, no hemostasis disorder and normal or almost normal endoscopic appearance

RESULT

Between September 1999 and January 2001, 84 cases were recruited consist of 40 cases of liver cirrhosis and 44 cases of dyspepsia. From 40 cases of liver cirrhosis, we exclude two cases without PHG, two cases of hepatoma and four cases of non representative biopsy. We exclude 12 cases of dyspepsia with positive Helicobacter pylori infection, two erosive gastritis, two cases of bile reflux and four cases of non-representative biopsy. Samples were taken 64 cases with each group consist of 32 cases.

Table 1. Distribution of Subject Characteristic Based on Demographic and Habitual Factor

	Dyspepsia N= 32	PHG N = 32	N	Test
Demographic Factor				
Age				
Mean ± SD	36.22 ± 11.67	49.06 ± 11.9		
Gender				
Male	13 (40.63%)	21 (65.63%)	34	p = 0.540
Female	19 (59.37%)	11 (34.37%)	30	
Habitual Factor				
Smoking				
Non smoker	24 (75.00%)	18 (56.25%)	42	p = 0.114
Smoker	8 (25.00%)	14 (43.75%)	22	
Drink alcohol/coffee				
Not drink/person	17 (53.13%)	19 (59.38%)	36	p = 0.014
Drink coffee/person	6 (18.75%)	12 (37.50%)	8	
Drink alcohol/person	9 (28.12%)	1 (3.12%)	10	

PHG = Portal hypertensive gastropathy, N = number of patient

Subject Characteristic

Mean age in dyspepsia group was 36.22 ± 11.67 years ranging from 16 to 60 years old, while the mean age in PHG group was 49.06 ± 11.90 years ranging from 21 to 63 years old. Distribution was not significant different between male and female patients (34 vs. 30 patients). The number of male in dyspepsia group was 13 from 34 patients (40.63%), less than female patients which were 19 from 30 patients (59.37%). On the other hand, male patients were found more in PHG group which were 21 from 34 (65.63%). Female patients were only 11 from 30 patients (32.37%) and p=0.540 was not statistically significant. The number of dyspepsia patient who were non smoker were 24 from 42 patients (75.00%), more than smoker who were only 8 from 22 patients (25.00%). Almost the same with those in PHG group who were found 18 from 42 patients (56.25%) more than smoker about 14 from 22 patients (43.75%), with p=0.114 and it was not statistically significant.

From the table above, it was shown that patients who did not drink alcohol or coffee in dyspepsia group were 17 from 36 patients (53.13%) and 19 from 36 patients in PHG group (59.38%). It was found that patients who drink coffee in dyspepsia group were 18.75% while in PHG we found it 37.50%. Alcoholism was found 28.12% in dyspepsia group and 3.12% in PHG group, p=0.014 is statistically significant.

In this study, mild PHG were found 23 from 32 patients (71.87%) and it was more than in severe PHG group which were only found 9 patients (28.13%) according to endoscopic appearance. There was no esophageal varices grade I found. The number of esophageal varices grade III was found 15 from 32 patients (46.87%). It was more frequent compare to grade II which had 12 patients (37.5%) and grade IV were 5 patients (15.63%).

Child B were found 18 from 32 patients (56.25%), more frequently than child C only 3 patients (9.37%) or child A which was only 11 patients (34.37%). Helicobacter pylori infection was merely found in 5 from 32 patients (15.63%), less frequent than non infected in 27 patients (84.37%)

From the endoscopic appearance, it showed that both scarlatina and mosaic pattern were found 18 from 32 patients (56.25%), compare to mosaic pattern only in 5 patients (15.63%) and CRS pattern in 9 patients (28.12%).

Table 2. Distribution of Subject Characteristics Based on Independent Variables in PHG

	Mild PHG (patient)	Severe PHG (patient)	Total
Grade of varices			
Grade II	10	2	12 (37.50%)
Grade III	11	4	15 (46.88%)
Grade IV	2	3	5 (15.62%)
Child class			
Child A	8	3	11 (34.37%)
Child B	14	4	18 (56.25%)
Child C	1	2	3 (9.38%)
Helicobacter pylori			
Negative	20	7	27 (84,37%)
Positive	3	2	5 (15.63%)
Gastroscopic appearance			
Mosaic pattern	5	0	5 (15.63%)
Scarlattina-mosaic	18	0	18 (56.25%)
Scar-mosaic-CRS	0	9	9 (28.12%)

Mucous Thickness

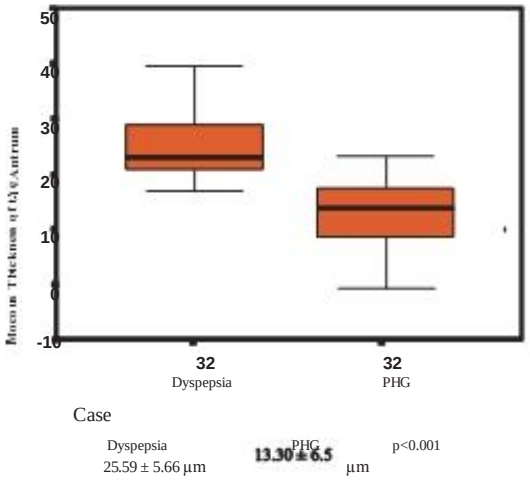

Figure 1. Box Plot Diagram of Mucous Thickness of the Antrum

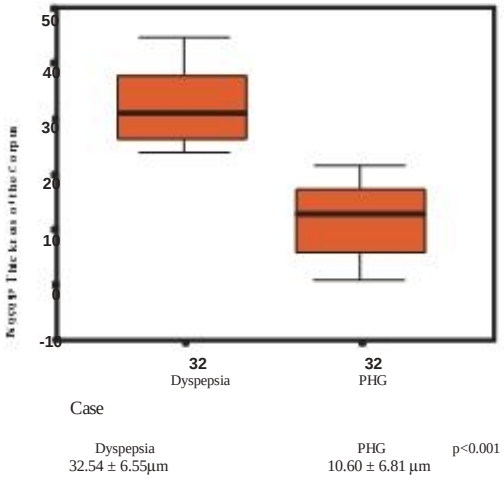

Figure 2. Box Plot Diagram of Mucous Thickness of the Corpus

Mean antral mucous thickness in PHG was 13.30 ± 6.5 µm, while in dyspepsia was 25.59 ± 5.66 µm with p<0.001. The mean mucous thickness in corpus in PHG was 10.6 ± 6.81 µm, while in dyspepsia was 32.54 ± 6.51 µm with p<0.001. This result showed statistically significant difference of mucous thickness in antrum and corpus between PHG and dyspepsia.

Correlation between age and mucous thickness in patients with PHG and dyspepsia

This study had divided the patients with dyspepsia and PHG into several age groups and analyzed by anova method as shown in this table below.

Table 3. Mucous Thickness in PHG Based on Age Group

Age Group (years old)	N	Mean (µm)	SD (µm)
< 20	0	0	0
21-30	5	8.7060	8.7122
31-40	0	0	0
41-50	10	9.8090	6.7611
51-60	13	11.8862	6.3541
> 61	4	10.8625	8.0266

N = number of patient; SD = standard of deviation

There were significant difference of mucous thickness based on age group in patients with PHG but it showed that mucous layer did not decrease in older group. Bivariate correlation parametric by Pearson method revealed correlation coefficient in antrum r = +0.23 with p=0.20, while in the corpus r = +0.15 with p=0.41 which suggests no significant correlation between older age and decreased mucous thickness.

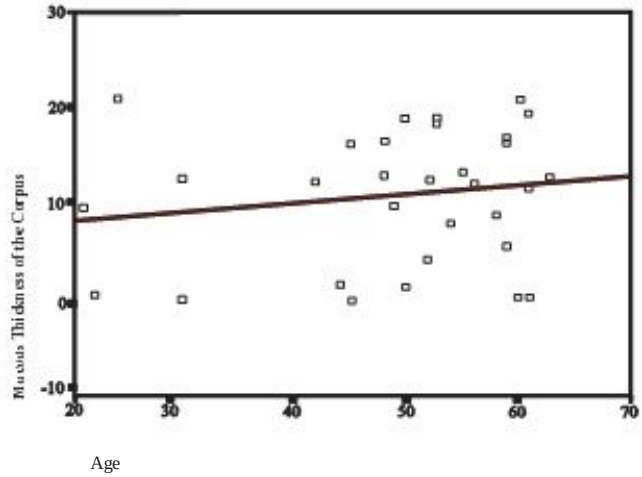

Figure 3. Linear-Regression Correlation between Age and Mucous Thickness in PHG

Table 4. Mucous Thickness In Dyspepsia Based on Age Group

Age Group (years old)	N	Mean (µm)	SD (µm)
< 20	4	31	8.3774
21-30	8	25.3625	7.0057
31-40	6	26.8667	4.4428
41-50	10	24.3600	3.9540
51-60	4	21.8500	1.1446

N = number of patients; SD = standard of deviation

Table 5. Correlation between Mucous Thickness of the Antrum and Corpus in Patients with Dyspepsia

		Mucous Thickness Antrum	Mucous Thickness Corpus
Age	Pearson correlation	-0.338	-0.373
	Significance (2-tailed)	0.058	0.36
	Number of patients	32	32

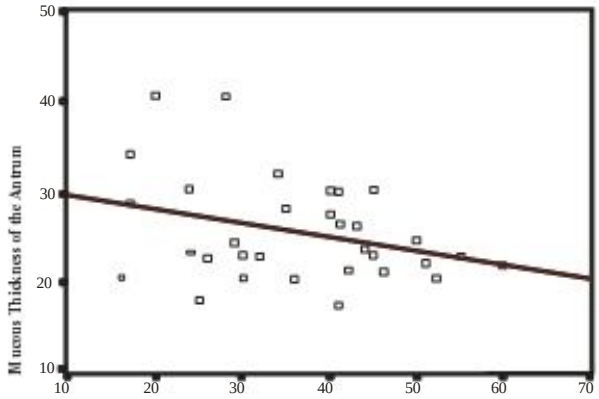

Figure 4. Linear- Regression Correlation between Age and Mucous Thickness in dyspepsia

Based on age group, it showed that there was difference of mucous thickness on each age group in dyspepsia patients. Decreased mucous layer was seen in older age group. By Pearson method we found correlation coefficient of antral mucous thickness in dyspepsia was r = -0.338 with p=0.058, while in the corpus r = -0.373 with p=0.036 indicated not very strong correlation between older age and decreased mucous layer.

The influence of mucous thickness in patients with PHG was correlated with other variables eg. esophageal varices, child class and endoscopic appearance shown in table below.

Table 6. Correlation between Mucous Thickness and other Variables: Esophageal Varices, Child Class and Gastropathy

Variable	N	Mucous Thickness in Corpus	
		x ± SD (µm)	Test
Child Class			
Child A	11	10.36 ± 6.0	p=0.071
Child B	18	12.12 ± 7.0	
Child C	3	2.48 ± 1.6	
Grade of esophageal varices			
Grade II	12	10.16 ± 6.81	p=0.587
Grade III	15	11.78 ± 6.67	
Grade IV	5	8.19 ± 7.97	
Gastropathy			
Mild gastropathy	23	11.38 ± 6.97	p=0.312
Severe gastropathy	9	8.63 ± 6.33	

Note: N= number of patients, x= mean, SD=standard of deviation

Mean mucous thickness in esophageal varices grade II was 10.16 ± 6.81 μm, grade III 11.78 ± 6.67 μm and grade IV 8.19 ± 7.97 μm. It showed that grade IV EV had thinner mucous than grade I and II. Statistical test by anova method found p=0.587. This means mucous thickness did not correlate significantly with variceal grade.

In this study, mucous in child A was 10.36 ± 6.0 μm much thinner than child B which was 12.12 ± 7.0 μm. The mucous was extremely thin in child C with mean 2.48 ± 1.6 μm compare to child A and B. This data showed the decreased mucous thickness in more severe child class. However, by anova method mucous thickness and child class was not statistically significant with p=0.071.

Mean mucous layer in severe PHG was 8.63 ± 6.63 μm, thinner than mean mucous layer in mild PHG which was 11.38 ± 6.97 μm. Result of t-test was p=0.312 which suggests mucous thickness didn't have significant correlation statistically with gastropathy.

Table 7. Correlation between Mucous Thickness and Helicobacter pylori Infection

Helicobacter pylori	Mean (μm)	Significance
Negative	10.07 ± 6.36	p=0.305
Positive	13.53 ± 8.32	

In this study, it revealed that mean mucous layer in Helicobacter pylori infected patients with PHG was 10.07 ± 6.36 mm thicker than non infected ones 13.53 ± 8.32 mm. It was not significant statistically with p=0.305.

Table 8. Gastroscopic Appearance and Mucous Thickness in PHG

Gastroscopic Appearance	Mucous Thickness	
	Mean (μm)	Test
Mosaic pattern	11.09 ± 6.96	P=0.334
Scarlatina-mosaic	11.68 ± 7.01	
Scar-mosaic-cherry	7.21 ± 5.82	

Mucous thickness in gastroscopic appearance of cherry red spot (CRS) was 7.21 ± 5.82 μm compare to mosaic pattern only 11.09 ± 6.96 μm. The same feature was also seen in CRS pattern compare to scarlatina and mosaic pattern 11.08 ± 7.01 μm. From the statistical analysis by t-test, we got p=0.334. It suggests that CRS pattern had tendency of thinner mucous layer but not significant statistically because p>0.05.

DISCUSSION

This study aimed to identify gastric mucosa mucous thickness indicates decreased gastric mucous production in PHG compare to normal mucosa in functional dyspepsia. Gastric mucous is key factor in providing defensive factors against dangerous intrinsic and extrinsic stimulus. Mucous production has important role in mild irritation which disturb cytoprotective adaptation of gastric mucous in PHG.[13,20,31,32,33] This study aimed to evaluate gastric mucous thickness in PHG and dyspepsia and try to correlate it with several factors.

Age distribution was varied ranging from 16 to 63 years old with mean age 42.64 ± 13.36 years. In dyspepsia group, age distribution was ranging from 16 to 60 years, while in PHG group it was 21 to 63 years. Portal hypertensive gastropathy (PHG) due to liver cirrhosis is known to be chronic disease.[34] This study did not show any correlation between age and mucous thickness with correlation coefficient of antrum r = +0.23 and p=0.20, while at the corpus r = +0.15 and p=0.41. However, in patients with dyspepsia as control group there was weak correlation between age and mucous thickness. The correlation coefficient of mucous thickness of antrum in patients with dyspepsia r = -0.338 and p=0.058, while in corpus r = -0.373 and p=0.036. This suggests that the mucous layer was decreased in older patients.

In this study, smoking habit was not significantly correlated with p=0.114. On the other hand, drinking habit of alcohol and coffee had significant correlation with mucous thickness with p=0.014. These results were not different from studies conducted by Sarfeh et al,[35, 36] Tanoe et al,[37] and Imanishi et al.[30] Decreased mucous defense in portal hypertension includes reduced synthesis of PGE 2 and abnormalities in hydrogen ionic transfer and decreased electronegative charge potential due to increased back diffusion of H^+ and decreased negative potential difference. Alcohol, bile acid or aspirin may cause significant destruction on gastric mucosa in portal hypertension than in normal mucosa.[31]

The mean mucous thickness of antrum in PHG was 13.30 ± 6.5 μm, while in dyspepsia group was 25.59 ± 5.66 μm. The mean mucous thickness of corpus in PHG was 10.6 ± 6.81 μm, while in dyspepsia group was 32.54 ± 6.55 μm. Statistical analysis of mucous thickness on both groups resulted p<0.001. This indicated that there were significant difference of mucous thickness of corpus and antrum between PHG and dyspepsia. This

study indicated the presence of reduced gastric mucosa mucous thickness in PHG.[30,38,39]

Imanishi et al,[30] found that the cause of reduced gastric mucous thickness in PHG might be due to decreased production and excretion of gastric mucous or qualitative changes in mucous layer. Decreased production of mucous may be due to hemodynamic changes e.g. Decreased gastric mucosa blood flow (congestion) causing increased vascular resistance in portal system.[8,23] It may also decrease hepatic blood flow.[21] Thus, oxygen supply and nutritional process of mucous cells are impaired.

Humoral factors are considered to have important role in decreased metabolic function of gastric mucosa, indicated by decreased response to pentagastrin, reduced glycoprotein of mucosa, reduced prostaglandin E2 and increased NO synthesis. All these will make the mucosa more fragile and susceptible to any destructive agents.[8,9,12,14,19,23,29,33,38,40]

To date, the pathophysiology of PHG remains unclear. Portal hypertension due to increased vascular resistance will make varices in the esophagus as collateration of gastric and splenic veins.[5,8,21,23] Changes in gastric mucosa in liver cirrhosis appear as mosaic pattern by endoscopy according to OMED criteria[17] is also a congestive condition as one mechanism of PHG.[12,15,22] The variables considered to be related with pathophysiology of PHG were child class, grade of varices and naturopathy.

Table 3 showed the relation between mucous thickness based on age group. This study found difference in decreased mucous thickness associated with path physiology of PHG but not statistically significant. Other factors might be the causes of it such as the shunt factor where increased portal vein pressure does not make collateral in gastric and splendid vein but to other collateral veins. Differences in number of cases for each variable may also influence the statistic analysis in this study.

In this study the child A were found 34.37% (11 from 32 patients), child B 56.25% (18 from 32 patients) and child C 9.38 % (3 from 32 patients). Mean difference of mucous thickness was extreme for the child C class where it was found thinner compare to Child A and B (2.48 ± 1.6 µm vs 10.36 ± 6.0 µm and 12.12 ± 7.0 µm) which might be due to chronic process. These data suggests that mucous thickness is reduced more in severe liver cirrhosis than in the mild ones, but not statistically significant. Nurman[7] reported that the incidence of PHG increased in accordance to severity of liver disease. In contrast, Mc Cormack et al[13] had reported that gastropathy was not correlated to severity of liver cirrhosis.

In this study showed that mucous thickness in grade IV varices was thinner than in grade I and grade II (8.19 ± 7.97 µm vs. 10.16 ± 6.81 µm and 11.78 ± 6.67 µm) indicated that reduced mucous production in more severe varices grade. Reduced gastric mucosal blood flow (congestion) caused increased portal pressure and vascular resistance resulting in collateralization to gaster in effort to reduced portal hypertension.[18,23] The occurrence of intra mucosal arterial pressure cause hyperdynamic condition and destroyed mucous cells implicate in decreased mucous thickness.[8,18]

Nurman[7] reported that PHG associated with varices grade. The prevalence is still unknown but it had been reported varied ranging 2% to 100%. Sarin[4] reported 16% to 20%, while Pique 60% to 70%. The difference of these prevalences of gastric varices is due to difficulties in recognizing the vascular structure by endoscopy and could be mistaken as gastric mucosal fold.

The gastric mucosa is objectively related with portal hypertension in PHG.[29] The cherry red spot occurred caused by thinning of gastric wall indicating severe portal hypertension. Facts from morfometric studies by Iwao et al,[29] Mc Cormack et al[13] had supported that dilatation of capillaries in cherry red spot equivalent with subclinical gastrointestinal bleeding.

In this study, the number of mild PHG was 71.87% (23 from 32 patients) and severe PHG 28.12% (9 from 32 patients). There was difference in the mucous thickness both in mild PHG (11.38 ± 6.97 µm) and severe PHG (8.63 ± 6.33 µm) This can be explained by assuming that in severe PHG more severe congestion cause impaired mucous production and more destroyed mucous cells. However, the statistical analysis found no significant correlation of these.

In this study, the Helicobacter pylori infection was only found in 5 patients (15.62%) compare to the control group and dyspepsia group were 71.87% (23 from 32 patients). Simadibrata M[41] found Helicocbacter pylori infection 37.5% and Mc Cormack et al[13] found 26% compare to control group (38%). Mean mucous thickness in Helicobacter pylori infection was higher than the non infected ones (13.53 µm vs. 10.07 µm). The cause of this is still unclear, but the Helicobacter pylori known to live in the mucous layer and not suitable in hypoacidity environment. It seemed that Helicobacter pylori did not have important role in the pathogenesis of PHG. The high prevalence of PHG decreased severity

of PHG supported by D'Amico.[20] Low Helicobacter pylori infection in severe PHG was also supported by Wang JY et al.[33] It was assumed that gastric mucosa in PHG was not suitable for Helicobacter pylori. However, Kitano S et al[31] could not found any correlation between prevalence of Helicobacter pylori and severity of PHG or esophageal varices.

CONCLUSION

Gastric mucous layer of antrum and corpus was significant thinner in patients with PHG compare to control group of functional dyspepsia. There was tendency of decreased mucous thickness in more severe esophageal varices, child class and gastropathy although was not statistically significant

SUGGESTION

This study had proved the presence of decreased gastric mucous layer of antrum and corpus in PHG. It may be used for further research in more basic clinical studies on mucous layer in search for better treatment of PHG and the benefit of using cytoprotective drugs. Due to tendency of decreased gastric mucous thickness in more severe esophageal varices, child class, gastropathy, and gastroscopic appearance; further research need to be conducted with better study design and larger number of samples.

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