

[ORIGINAL ARTICLE]

Portal Hypertensive Gastropathy in Liver Cirrhosis: Prevalence, Natural History, and Risk Factors

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Abstract:

Objective Portal hypertensive gastropathy (PHG) is a common finding in patients with liver cirrhosis (LC) and may cause both acute and chronic bleeding. A number of risk factors for PHG have been identified. The present study explored the characteristics of Japanese patients with LC who develop PHG.

Methods Clinical findings (age, sex, etiology, the presence of esophageal varices, splenomegaly and severity of LC), laboratory data, and whether or not atrophic gastritis was found on endoscopy were retrospectively reviewed in patients with LC who had undergone esophagogastroduodenoscopy. PHG was endoscopically graded as absent, mild, or severe.

Results Of 262 patients with LC (mean age, 69 years old; 145 men), 158 had no PHG, 41 had mild PHG, and 63 had severe PHG. In a univariate analysis, a younger age, male sex, non-viral etiology, absence of atrophic gastritis, presence of esophageal varices, splenomegaly, severe LC, low platelet count, and low hemoglobin concentration were associated with PHG. A multivariate analysis showed a significant association of PHG with the absence of atrophic gastritis ($p < 0.048$), presence of esophageal varices ($p < 0.001$), non-viral etiology ($p < 0.033$), splenomegaly ($p < 0.048$), and severe LC ($p < 0.005$). There were no cases of massive bleeding from PHG during follow-up.

Conclusion Esophageal varices, splenomegaly, severe liver cirrhosis, the absence of atrophic gastritis, and etiology were found to be risk factors for PHG in Japanese patients.

Key words: portal hypertensive gastropathy, liver cirrhosis, atrophic gastritis, esophageal varices, splenomegaly

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Introduction

Portal hypertensive gastropathy (PHG) is a common endoscopic finding in patients with liver cirrhosis (LC) (1-3). It is a known cause of anemia and acute or insidious gastrointestinal (GI) bleeding as well as esophageal and gastric varices and gastric antral vascular ectasia in these patients (4-6). The reported prevalence of PHG varies from 3.7% to 75% in patients with portal hypertension (3) and from 15.1% to 100% in patients with LC (3). This wide variability likely reflects variability in definitions, classification criteria, interpretation of endoscopic findings, patient

selection, and severity of liver impairment.

McCormack et al. classified PHG with features of fine pink speckling (scarlatina-type rash) and a mosaic pattern (snakeskin appearance) as mild and that with discrete red spots or diffuse hemorrhagic lesions as severe (7) (Fig. 1). This endoscopic classification is important because of the increased risk of gastric hemorrhaging in severe cases of PHG (4, 8, 9).

Histologic evidence of gastritis and various endoscopic findings, including redness, swelling, erosions, atrophy, enlarged folds, and nodularity, have been found in patients with *Helicobacter pylori* (*H. pylori*) infection (10). In some cases, the findings of redness and swelling caused by *H. py-*

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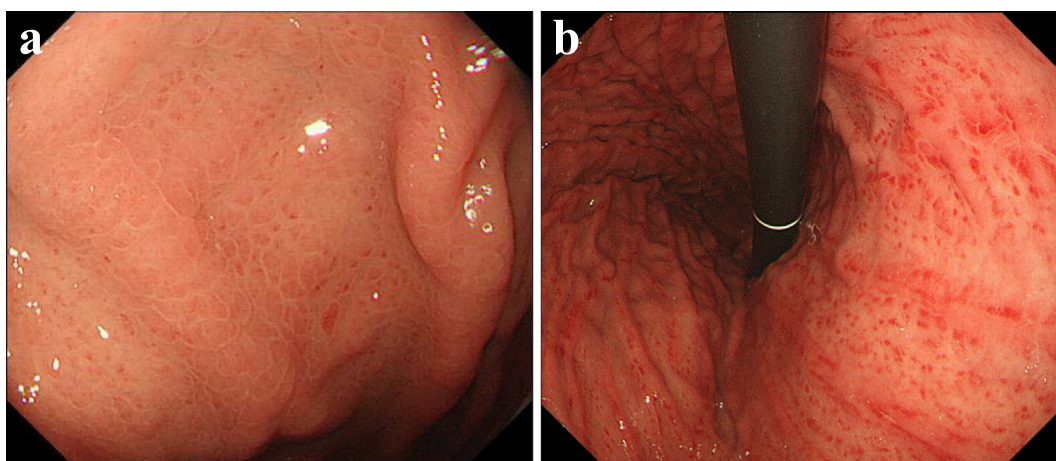


Figure 1. Typical endoscopic findings for portal hypertensive gastropathy. A mosaic pattern (snakeskin appearance) (mild, a) and discrete red spots (severe, b) can be seen.

lori are similar to the endoscopic findings for PHG. Furthermore, anti-reflux agents, such as proton pump inhibitors (PPIs), cause specific changes in the gastric mucosa, including a cobble-stone appearance (11), gastric polyps (11), and black spots (12). PPIs are widely used in patients with LC, especially those with esophageal or gastric varices, and may influence the endoscopic findings in the gastric mucosa. Therefore, there may be cases in which it is difficult to distinguish between PHG and *H. pylori*-positive gastritis or PPI-induced gastropathy.

Although there have been many clinicopathologic studies of PHG (13-15), there are no recent reports on the frequency of PHG, the factors involved in its occurrence, or its prognosis in Japan (16). Considerable progress has been made in the treatment of LC, including the use of PPIs and targeted treatment of *H. pylori*, which is involved in inflammation of the gastric mucosa. However, there is a possibility of changes in the endoscopic findings, pathophysiology, and clinical course in patients with PHG. Therefore, to ensure good clinical practice, we investigated the clinical characteristics of PHG detected by experienced endoscopists at our institution.

The present study assessed the prevalence of PHG in patients with LC at Kawasaki Medical School and clarified the factors associated with the development of PHG in Japan.

Materials and Methods

This retrospective cross-sectional study included adult patients (≥ 20 years old) with an established diagnosis of LC who underwent esophagogastroduodenoscopy at Kawasaki Medical University between April 2014 and March 2020. Patients were consecutively enrolled. Laboratory data, clinical images, including endoscopic findings, and details of their social history (use of gastric acid secretion inhibitors and other drugs) were collected from medical records as available.

The study was conducted in accordance with the 1964

Declaration of Helsinki and its later amendments and was approved by the ethics committee of Kawasaki Medical University. All patients consented to undergo the endoscopic procedure. The need for specific informed consent for inclusion in this report was waived in view of the retrospective study design, the anonymity of the data, and the routine nature of all clinical procedures and tests performed.

Selection criteria

The diagnosis of LC was made primarily based on imaging findings. The findings were comprehensively judged based on the bluntness/rounding or irregularity of the liver edge, regenerative nodules in the liver parenchyma, evidence of portal hypertension, such as splenomegaly and a collateral circulation, and changes in the shape of the liver, such as right lobe atrophy (17). Histology reports were also examined as necessary. Patients who had undergone liver transplantation or gastrectomy, those with advanced cancer and tumor thrombus in the portal vein, those with non-cirrhotic portal hypertension, those in whom endoscopic observation was inadequate, and those who have been taking antibiotics or other drugs for that affect *H. pylori* infection or gastric mucosal atrophy were excluded.

Endoscopy

The GI tract was evaluated for PHG, gastric mucosal atrophy, and esophageal varices by three doctors (2 board-certified fellows of the Japan Gastroenterological Endoscopy Society with more than 20 years of endoscopic experience who were familiar with the diagnosis of *H. pylori* infection gastritis, and a board-certified hepatologist of the Japan Society of Hepatology with more than 15 years of endoscopic experience). Each case was compared with clear endoscopic images presented in the literature and performed under consensus (18, 19). With the recent advancement of endoscopic image analysis technology, the diagnosis rate of PHG is expected to improve using image enhancement systems, such as narrow-band imaging (NBI) and blue-laser imaging

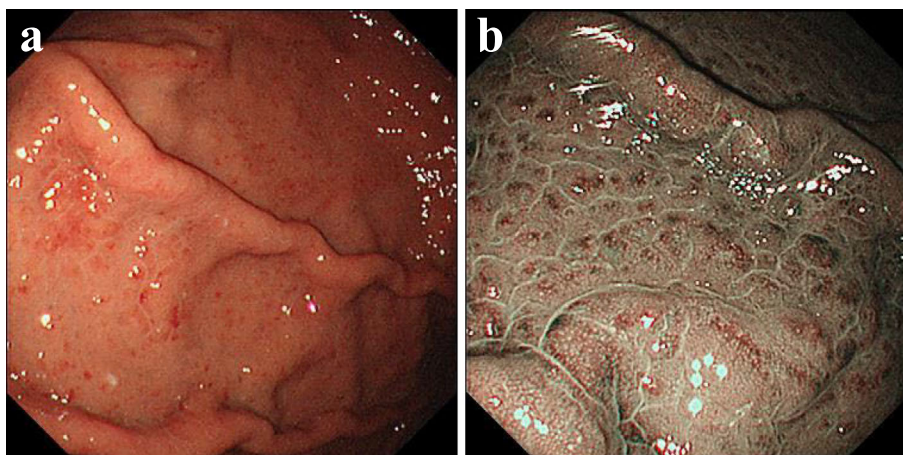


Figure 2. Narrow-band imaging (NBI) image of portal hypertensive gastropathy. (a) White-light imaging image, b) NBI image; the NBI image shows vasodilatation and the boundaries of the gastric area more clearly.

(BLI) (18, 19) (Fig. 2), but in this study, they were used only in some cases, so this determination was made using white-light imaging (WLI). Findings suggestive of PHG were graded as per the McCormack criteria (7). Gastric mucosal atrophy was categorized as open or closed based on the Kimura-Takemoto classification (20). Since the diagnosis of *H. pylori* infection using the serum antibody assay and urea breath test was not made in many cases, we assessed the rate of *H. pylori* infection based on the endoscopic findings of the Kyoto classification of gastritis. Our previous study of the Kyoto classification of gastritis assessed *H. pylori* infection, with a sensitivity of 98.5% and a specificity of 100% (21). In cases with esophageal varices, the presence, location, form, presence of a red color sign, history of treatment, and course of treatment for treated patients were reviewed according to the general rules for studies of portal hypertension (22).

Assessments of splenomegaly

Splenomegaly was assessed by ultrasound imaging. According to the definition of the Japanese Society of Sonographers, splenomegaly was defined in cases with a Spleen Index (product of the diameter from the hilum to the anterior margin of the spleen and the diameter perpendicular) $>20 \text{ cm}^2$.

Stratifying severity of liver disease

Data were collected for the complete blood count, renal and liver function tests, abdominal ultrasonography, abdominal computed tomography, prothrombin time, and international normalized ratio. The severity of liver disease was assessed by the Child-Pugh class (23, 24) and the model for end-stage liver disease (MELD) score (25). The patients were stratified into two groups according to the Child-Pugh class: A (5-6) and B/C (7-15).

Statistical analyses

Continuous variables are reported as the mean and stan-

dard error of the mean or as the median [interquartile range (IQR)]. Categorical variables are summarized as the number and percentage. Continuous variables were compared using logistic regression analyses or the chi-squared test as applicable. Univariate and multivariate logistic regression analyses were performed to assess the risk factors for PHG. All statistical analyses were performed using the JMP 14 software program (SAS Institute, Cary, USA). A 2-sided p value of ≤ 0.05 was considered statistically significant.

Results

The clinical characteristics and results of univariate analyses are summarized in Tables 1 and 2. Thirty-one of the 293 patients screened were excluded, leaving data for 262 patients with cirrhosis available for inclusion in the study (Fig. 3).

Patient demographics

One hundred and four (39.7%) of the 262 patients with LC had PHG (mild, $n=41$; severe, $n=63$), and 158 (60.3%) had no PHG.

Etiology

The cause of cirrhosis was chronic viral hepatitis B or C in 145 patients (55.3%), alcohol in 50 (19.1%), and other (e. g. nonalcoholic steatohepatitis, primary biliary cholangitis) in 67 (25.6%). The prevalence of PHG in patients with alcohol-induced LC was significantly higher than that of viral LC [62% (31/50) vs. 32.4% (47/145), $p<0.001$] and other etiologies [62% (31/50) vs. 38.8% (26/67), $p<0.014$], but there was no significant difference between the rates of viral LC and other etiologies [32.4% (47/145) vs. 38.8% (26/67), $p<0.363$].

Endoscopy

According to the Kimura-Takemoto classification, 165 cases had closed-type gastric mucosal atrophy, and 97 cases

Table 1. Clinical Characteristics of Patients in This Study.

Patients (n)		262
Age (years)		69.0±11.2
Sex (n, %)	Male	145 (55.3)
	Female	117 (44.7)
Etiology (n, %)	Viral	145 (55.3)
	Non-viral	117 (44.7)
PHG (n, %)		104 (39.7)
Atrophy (Kimura-Takemoto classification) (n, %)	Closed type	165 (63.0)
	Open type	97 (37.0)
	C1/C2/C3	128/28/9
	O1/O2/O3	48/24/25
Esophageal varices (n, %)		133 (50.8)
Distribution	esophageal only	100 (38.2)
	esophagogastric	33 (12.6)
	gastric only	6 (2.3)
Esophageal varices	Li/Lm/Ls	23/76/34
	F1/F2/F3	80/46/7
	RC0/RC1/RC2/RC3	96/22/10/5
Treatment	ALL	43
	EVL	21
	EIS	17
	BRTO	5
Time from treatment (years)		3.19±3.85
GAVE (n, %)		30 (11.5)
Splenomegaly (n, %)		139 (54.1)
Child-Pugh class	A	180 (68.7)
	B	62 (23.7)
	C	20 (7.6)
MELD score		8 [7-11]
PLT		110 [78-149]
Hb		12.5 [10.6-14.1]
AST		37 [26-57]
ALT		24 [18-43]
GGT		45 [26-90]
Bil		1.0 [0.7-1.4]
Alb		3.8 [3.3-4.2]
ChE		208 [140-275]
T-Cho		163 [133-194]
Cre		0.77 [0.63-0.96]
NH ₃		41 [29-65]
Gastric acid secretion inhibitor (n, %)		145 (55.3)

Continuous variables are reported as mean±standard error, or median [interquartile range].

Li: location inferior, Lm: location midium, Ls: location superior, F: form, RC: red color sign, EVL: Endoscopic variceal ligation, EIS: Endoscopic injection sclerotherapy, BRTO: balloon-occluded transfemoral obliteration, MELD: Model for End-Stage Liver Disease, PLT: Platelets 10⁹/L, Hb: Hemoglobin g/dL, AST: Aspartate aminotransferase IU/L, ALT: Alanine aminotransferase IU/L, GGT: Gamma-glutamyl transferase IU/L, Bil: Bilirubin mg/dL, Alb: Albumin g/dL, ChE: Cholinesterase IU/L, T-Cho: Total cholesterol mg/dL, Cre: Creatinine mg/dL

had the open type. The prevalence of the closed type was significantly higher in patients with PHG than in those without PHG [73.1% (76/104) vs. 56.3% (89/158), $p<0.006$]. The rate of *H. pylori* infection was 43.5% in the PHG group and 55.1% in the non-PHG group ($p<0.118$).

One hundred and thirty-three patients (50.8%) had esophageal varices; the prevalence of esophageal varices was

significantly higher in patients with PHG than in those without PHG [75.0% (78/104) vs. 34.8% (55/158), $p<0.001$]. Furthermore, the prevalence of PHG was slightly higher in patients with concomitant gastric and esophageal varices than in those with esophageal varices only [69.7% (23/33) vs. 55% (55/100)].

Table 2. Comparison of Clinicopathological Characteristics between Patients with and without Portal Hypertensive Gastropathy.

		PHG n=104	No PHG n=158	p value Univariate analysis
Age (years)		67.2±11.8	70.2±10.6	0.03
Sex (n, %)	Male	67 (64.4)	78 (49.4)	0.016
	Female	37 (35.6)	80 (50.6)	
Etiology (n, %)	Viral	47 (45.2)	98 (62.0)	0.007
	Non-viral	57 (54.8)	60 (38.0)	
Atrophy (Kimura-Takemoto classification) (n, %)	Closed type	76 (73.1)	89 (56.3)	0.006
	Open type	28 (26.9)	69 (43.7)	
	C1/C2/C3	57/17/2	71/11/7	0.013
	O1/O2/O3	17/5/6	31/19/19	
Esophageal varices (n, %)		78 (75.0)	55 (34.8)	<0.001
Distribution	esophageal only	55 (52.9)	45 (28.5)	<0.001
	esophagogastric	23 (22.1)	10 (6.3)	<0.001
	gastric only	1 (1.0)	5 (3.2)	0.216
Esophageal varices	Li/Lm/Ls	12/44/22	11/32/12	0.627
	F1/F2/F3	41/35/2	39/11/5	0.005
	RC0/RC1/RC2/RC3	51/17/6/4	45/5/4/1	0.131
Treatment (n, %)	ALL	34 (32.7)	9 (5.7)	<0.001
	EVL	15 (14.4)	6 (3.8)	0.433
	EIS	15 (14.4)	2 (1.3)	
	BRTO	4 (3.8)	1 (0.6)	
Time from treatment (years)		2.69±2.86	5.08±6.25	0.119
GAVE (n, %)		13 (12.5)	17 (10.8)	0.666
Splenomegaly (n, %)		73 (70.2)	66 (41.8)	<0.001
Child-Pugh class	A	52 (50.0)	128 (81.0)	<0.001
	B	35 (33.7)	27 (17.1)	
	C	17 (16.3)	3 (1.9)	
MELD score		9 [8-14]	8 [6-10]	<0.001
PLT		93 [62-139]	117 [86-156]	0.001
Hb		11.5 [9.7-13.2]	13.3 [11.3-14.7]	<0.001
AST		40 [27-62]	36 [26-53]	0.116
ALT		23 [18-43]	25 [17-40]	0.806
GGT		56 [31-96]	40 [24-77]	0.043
Bil		1.2 [0.8-1.7]	0.9 [0.7-1.3]	0.005
Alb		3.5 [2.9-3.9]	4.0 [3.5-4.3]	<0.001
ChE		160 [103-225]	237 [176-298]	<0.001
T-Chol		150 [125-179]	172 [138-201]	0.001
Cre		0.82 [0.64-1.0]	0.75 [0.62-0.95]	0.267
NH ₃		49.5 [34.8-78.3]	35.5 [25.3-48.8]	<0.001
Gastric acid secretion inhibitor (n, %)		68 (65.4)	77 (48.7)	0.008

Continuous variables are reported as mean±standard error, or median [interquartile range].

Li: location inferior, Lm: location midium, Ls: location superior, L: location, F: form, RC: red color sign, EVL: Endoscopic variceal ligation, EIS: Endoscopic injection sclerotherapy, BRTO: balloon-occluded transfemoral obliteration, MELD: Model for End-Stage Liver Disease, PLT: Platelets 10⁹/L, Hb: Hemoglobin g/dL, AST: Aspartate aminotransferase IU/L, ALT: Alanine aminotransferase IU/L, GGT: Gamma-glutamyl transferase IU/L, Bil: Bilirubin mg/dL, Alb: Albumin g/dL, ChE: Cholinesterase IU/L, T-Chol: Total cholesterol mg/dL, Cre: Creatinine mg/dL

Severity of liver disease

In terms of the severity of liver disease, 180 patients (68.7%) were in Child-Pugh class A, 62 were in Child-Pugh class B, and 20 were in Child-Pugh class C. The prevalence of PHG significantly increased as the Child-Pugh class became more severe [A vs. B: 28.9% (52/180) vs. 56.5% (35/

62), p<0.001, A vs. C: 28.9% (52/180) vs. 85.0% (17/20), p<0.001, B vs. C: 56.5% (35/62) vs. 85.0% (17/20), p<0.029].

Multivariate analyses

In the multivariate analyses, gastric mucosal atrophy, etiology of LC, presence of esophageal varices, splenomegaly,

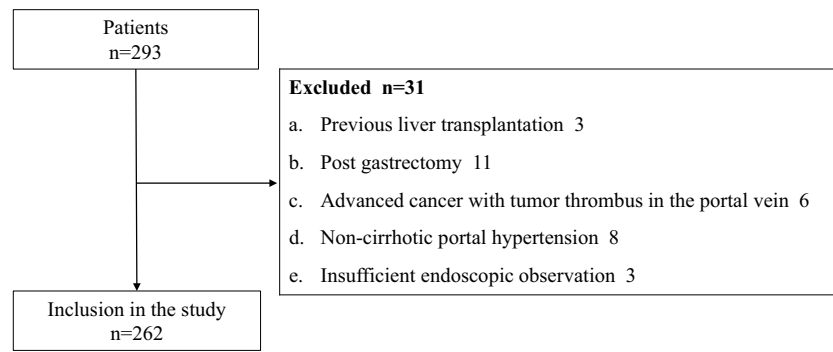


Figure 3. Flowchart of this study.

Table 3. Results of Multivariate Analysis.

	Univariate analysis				Multivariate analysis			
	OR	95% CI		p value	OR	95% CI		p value
Etiology: Non viral	1.981	1.201	- 3.286	0.007	1.883	1.052	- 3.398	0.033
Atrophy: Closed type	2.104	1.241	- 3.631	0.006	1.852	1.005	- 3.469	0.048
Esophageal varices: present	5.618	3.273	- 9.889	<0.001	3.707	1.963	- 7.122	<0.001
Splenomegaly: (+)	3.394	2.004	- 5.859	<0.001	1.870	1.007	- 3.475	0.048
Child-Pugh class: B/C	4.267	2.472	- 7.489	<0.001	2.484	1.315	- 4.727	0.005

OR: odds ratio, CI: confidence interval

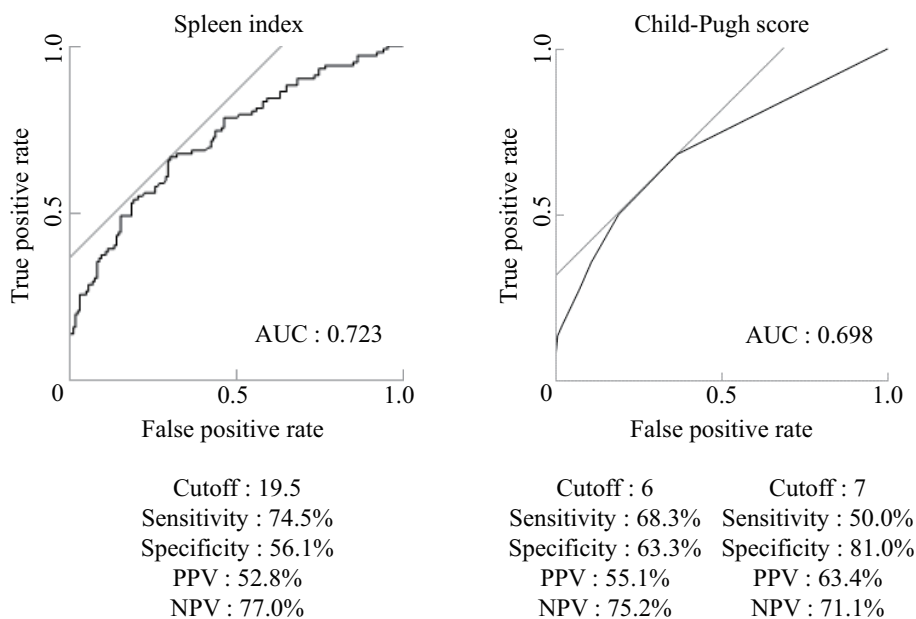


Figure 4. Receiver operating characteristic curve of the Spleen Index and Child-Pugh score.

and severe liver disease remained significant risk factors for PHG (Table 3).

Factors that predict the development of PHG

The factors predicting the development of PHG were examined in terms of splenomegaly (Spleen Index) and the Child-Pugh score. As a result, the cut-off value was 19.5 for the Spleen Index and 6 for the Child-Pugh score (Fig. 4).

Natural history

Fifty-three patients in the PHG group were followed up by an endoscopic examination for at least one year. During a mean follow-up period of 3.94 ± 2.2 (range 1-6) years, the PHG worsened in 4 patients, improved in 5, disappeared in 11, and remained unchanged in 33.

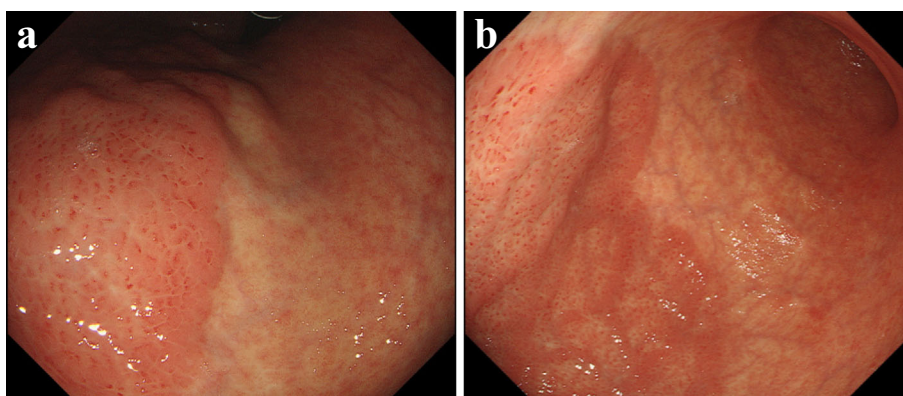


Figure 5. A case of portal hypertensive gastropathy. (a) Endoscopic image of the gastric body observed from the gastric angle. (b) Look-down image of the gastric body. The boundary between the atrophic mucosa, through which blood vessels are clearly visible, and the non-atrophic mucosa is clear. In PHG cases with atrophic gastritis, the findings of PHG may be masked by a reduced gastric mucosal blood flow.

Discussion

In this study, experienced endoscopists found that 39.7% of 262 Japanese patients with LC at our institution had PHG, which was mild in 39.4% of cases and severe in 60.6%. Risk factors for the development of PHG were the absence of atrophic gastritis, presence of esophageal varices, splenomegaly, and severe liver impairment.

This is the first report on the association between atrophic gastritis and PHG. Our multivariate analysis revealed that the prevalence of PHG was significantly higher in patients without atrophic gastritis ($p < 0.048$). Earlier studies indicated that *H. pylori* infection is not associated with the development of PHG (26, 27) and that the prevalence of *H. pylori* is lower in patients with PHG (28). The evaluation of *H. pylori* infection based on the Kyoto classification of gastritis (29) showed that *H. pylori* infection was not involved in the development of PHG in this study. Although there are still conflicting data regarding whether the gastric mucosal blood flow is increased or decreased in patients with PHG (30), a significant increase in gastric mucosal blood flow was suggested in rodent models of PHG (31, 32) and portal hypertension (33). In a study in humans by Takaya et al., the results obtained using the gastric mucosal hemoglobin index suggested that the gastric mucosal blood flow decreased with increasing severity of atrophy (34). Therefore, in PHG cases with atrophic gastritis, the findings of PHG may be masked by reduced gastric mucosal blood flow (Fig. 5).

The multivariate analysis in this study also showed a significant association between PHG and the presence of esophageal varices and splenomegaly ($p < 0.002$). Furthermore, the frequency of PHG was significantly higher in patients with coexisting gastric and esophageal varices. Some studies found no correlation between PHG and the presence of esophageal varices (35, 36), whereas other studies did

find a correlation (37-39). In the New Italian Endoscopic Club study, the prevalence of PHG was significantly higher in patients with esophageal varices [77% (80/104)] than in those without esophageal varices [61% (51/84), $p < 0.007$] (5). Fontana et al. also demonstrated a strong positive association between PHG and the presence of esophageal varices ($p < 0.001$) in a large cohort of patients with chronic hepatitis C virus infection and bridging fibrosis/compensated cirrhosis (37). In a prospective study of 331 patients with LC in South Korea, the severity of PHG was correlated with the portal and splenic vein diameters and the size of the spleen (38). In another study by Anegawa et al., laparoscopic splenectomy had a beneficial effect on severe PHG in 16 of 17 patients (16). These results are consistent with our current finding of a correlation between splenomegaly and PHG. Previous reports on gastric varices have been mixed. Some reports suggest that stasis of blood flow in the gastric mucosa improves when gastric varices appear (40); however, we found no relationship between the prevalence of PHG and gastric varices.

In the present study, severe liver disease, as evaluated by the Child-Pugh class, was an independent risk factor for PHG in a multivariate analysis. The prevalence of PHG increased according to the severity of liver disease [A, 28.9% (52/180); B, 56.5% (35/62); C, 85.0% (17/20)]. Some studies have demonstrated a significant association of PHG with the presence or severity of hepatic impairment (37, 39), although others found no such correlation (41). These conflicting data may reflect the fact that this association is not observed in all stages of liver disease. In the present study, Child-Pugh class B and C were combined and compared with Child-Pugh class A. De Lisi et al. reported that the prevalence of PHG was significantly higher in patients with Child-Pugh class B or C liver disease than in those with class A (42). Furthermore, Sarin et al. reported the prevalence of PHG to be 87% in patients with Child-Pugh class C but only 13% in those with Child-Pugh class A (39).

In this study, the Spleen Index and Child-Pugh score were investigated as predictors of the development of PHG. The Spleen Index was 19.5, which is in agreement with the diagnosis of splenomegaly (20). However, a Child-Pugh score of 6 predicted the development of PHG earlier than a Child-Pugh class B/C (score 7-15), and the specificity of a Child-Pugh score of 7 was 81.0%, which was also considered a useful factor.

In the univariate analysis, the prevalence of PHG was significantly higher in patients with alcohol-induced LC than in those with LC with a viral ($p < 0.001$) or other etiology ($p < 0.014$). The reason for the discrepancy in the findings (13) is unclear but may reflect recent developments in the treatment of viral hepatitis or the direct effect of alcohol, which damages the gastric mucosa and may contribute to the development of PHG (43, 44).

The natural history of PHG needs to be clarified because of its propensity to cause anemia and fatal acute GI bleeding (4-6). Reports on the prevalence of acute upper GI bleeding in patients with PHG have been inconsistent. The frequency of acute GI bleeding in patients with PHG was 2.5% in the study by Primignani et al. (5) and 20% in the study by Stewart et al. (8). Furthermore, in a study by McCormack et al., bleeding occurred in 29 (44.6%) of 65 patients (7). This variability in reporting is partly due to inaccuracies in the endoscopic diagnosis of PHG *per se* and in the endoscopic diagnosis of PHG as the cause of bleeding. Major risk factors for bleeding in patients with PHG are an increased duration (4, 5) and severity (4, 8, 9) of PHG. Over 90% of cases of acute bleeding occur in patients with severe PHG, with <10% occurring in those with mild PHG (6, 9, 45). Other risk factors for bleeding in patients with PHG include advanced cirrhosis (37, 39) and prior endoscopic eradication of esophageal varices (9). In the present study, we reviewed the endoscopic findings of 53 patients who were able to be followed up for at least 1 year and found no marked change in PHG in 33 cases (62.3%), exacerbation in 4 cases (7.5%), and improvement or disappearance in 16 cases (30.2%). Surprisingly, there were no episodes of acute bleeding from PHG in this study, although there were four cases of acute bleeding among patients with gastric antral vascular ectasia.

This study has several limitations that should be considered when interpreting its results. First, it had a retrospective design. Second, it was a single-center study involving a relatively small number of patients with LC. Third, not all cases were followed up to evaluate the natural history of PHG. The incidence of LC caused by viral infection, which causes PHG, has been declining in Japan, but that of nonalcoholic steatohepatitis is increasing. Therefore, it is important to investigate the clinical characteristics of PHG as revealed in this study in more recent cases.

Conclusion

The findings of this study suggest that esophageal varices,

splenomegaly, severe liver cirrhosis, absence of atrophic gastritis, and etiology are risk factors for PHG in Japanese patients.

The authors state that they have no Conflict of Interest (COI).

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