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【 EDITORIAL 】

Discrimination between Portal Hypertensive Gastropathy and *Helicobacter pylori*-related Gastritis

Yoshihiro Furuichi [1,2], Yohei Koyama [1,2], Masakazu Abe [1], Yuu Yoshimasu [1], Hirohito Takeuchi [1] and Takao Itoi [1]

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We read with great interest the original article written by Nishino et al. Their retrospective study was performed with univariate and multivariate logistic regression analyses, demonstrating that the risk factors of portal hypertensive gastropathy (PHG) were the existence of esophageal varices, splenomegaly, severe liver cirrhosis (LC), the etiology, and the absence of atrophic gastritis (1). In Japan, there have been no reports clarifying the frequency of PHG, onset factors, prognosis, or relationship with *Helicobacter pylori* infection.

PHG is a mucosal lesion that presents with redness, edema, and bleeding in the upper corpus of the stomach, similar to *H. pylori*-related gastritis, and is often observed in LC patients with portal hypertension. In their manuscript, non-viral (alcoholic) LC, liver dysfunction of Child-Pugh class >B, and splenomegaly with a spleen index of >19.5 cm^2 were concretely shown to be independent predictors of PHG. The most interesting point in their report was the finding of a negative correlation between the appearance of redness in PHG and the existence of atrophic gastritis.

PHG is endoscopically classified based on five findings according to the McCormick classification (2). Findings of fine pink speckling, superficial reddening, and a snakeskin (mosaic) pattern are considered mild PHG. Cherry red spots and diffuse hemorrhaging are considered severe PHG. Edema of the gastric mucosa and dilatation of the vessels with a significant increase in the gastric mucosal blood flow are sometimes observed (3, 4), and the incidence is reported to be around 50% to 90% in LC patients with portal hypertension (5).

The endoscopic findings of redness on the body of stomach with persistent *H. pylori* infection are known to resemble those of PHG mentioned above. Strictly speaking, however, it is possible to distinguish between PHG and *H. pylori* gastritis. We previously reported that the redness (due to vasodilation) was weak in *H. pylori* gastritis and that the white border line at the gastric area (due to edema) were also weak compared with the snakeskin pattern of PHG, resulting in reduced contrast on the mosaic appearance of *H. pylori* gastritis (Figure) (6). In other words, by observing the red and white contrast of the mosaic appearance, it is possible to distinguish these entities to some extent. In cases with mild redness, identifying whether the portal hypertension potentially exist or not is important. If gastritis patients with mild redness have both portal hypertension and *H. pylori* infection, it may be difficult to discriminate these entities. However, severe redness, such as cherry red spots and diffuse hemorrhaging, are findings specific to PHG. Therefore, even if patients have *H. pylori* infection or a history thereof, these finding strongly support the diagnosis of PHG.

The relationship between PHG and *H. pylori* infection is controversial. Balan et al. reported that the serum pepsinogen I concentrations, the ratio of polymeric to degraded gastric mucus, and the rate of gastric emptying were not significantly different between PHG patients with and without *H. pylori* infection. They also concluded that *H. pylori* infection was unlikely to be involved in the pathogenesis of PHG (7). McCormick et al. reported that *H. pylori* infection was identified in 26% patients with PHG and was not related to the severity of the endoscopic appearances (8). Hayashi et al. reported that the appearance rate of dot redness and mottled redness, categorized as fine pink speckling or superficial reddening, increased from 36% to 78% by *H. pylori* infection in the 162 patients with PHG (9). They also stated that a snakeskin (mosaic) appearance (unlike fine pink speckling and superficial reddening) was unchanged follow-

[1]Department of Gastroenterology and Hepatology, Tokyo Medical University, Japan and [2]Department of Gastroenterology, Niiza Shiki Central General Hospital, Japan
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Correspondence to Dr. Yoshihiro Furuichi, furuichi@tokyo-med.ac.jp

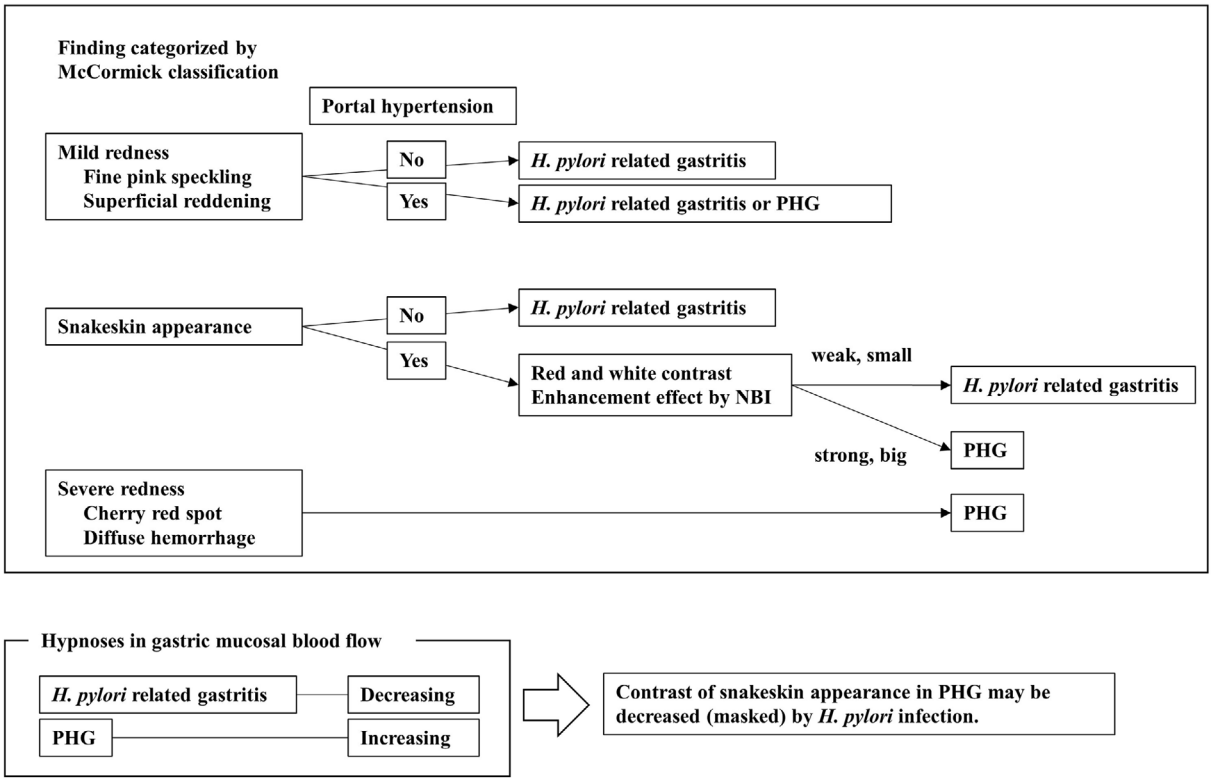

Figure. Endoscopic discrimination between portal hypertensive gastropathy and *Helicobacter pylori*-related gastritis. When mild redness was observed in the gastric mucosa, such as fine pink speckling and superficial reddening according to the McCormick classification, the existence of portal hypertension should be confirmed. If patients have portal hypertension, discrimination may be difficult. When a snakeskin appearance is observed in patients with portal hypertension, the contrast formed by the redness (vasodilation) and a white line at the gastric area (edema) is strong, indicating portal hypertensive gastropathy (PHG). This contrast may be enhanced by narrow-band imaging (NBI), an endoscopic imaging modality. Severe redness, such as cherry red spots or diffuse hemorrhaging, strongly supports the diagnosis of PHG. *H. pylori*: *Helicobacter pylori*

ing *H. pylori* eradication and thus unaffected by the presence of this infection. A snakeskin appearance therefore had seemed to be a finding peculiar to PHG.

However, Nishino et al. showed that the snakeskin appearance of PHG was attenuated in cases with the atrophic findings induced by *H. pylori* infection. They further reported that closed-type atrophic gastritis was a negative predictor of PHG according to a multivariate analysis. This is because the gastric mucosal blood flow increases with PHG but decreases with the progression of atrophic gastritis, and the findings of PHG were masked by atrophic gastritis. They also showed that narrow-band imaging (NBI), an enhanced endoscopic imaging modality, can emphasize the contrast of the snakeskin appearance by increasing the visibility of the reddened areas in PHG. Unfortunately, in their report, the diagnosis of PHG and *H. pylori* was performed by endoscopic findings only. Therefore, a further study including histopathological examinations and a urea breath test or at least *H. pylori* antibody titers is desired.

We propose that future studies elucidate several points. First, the enrolled endoscopic features should be divided into three groups of pure PHG without a history of *H. pylori* infection, PHG with successful *H. pylori* eradication, and PHG and chronic gastritis with persistent *H. pylori* infection. Second, in the group with *H. pylori* infection, the changes in endoscopic findings should be chronologically monitored before and after the eradication of *H. pylori*. Third, in those three groups, the changes in PHG findings should be investigated before and after intervention for portal hypertension, as PHG findings may be influenced by factors related to portal hypertension, such as viral control, drinking amount, esophago-gastric variceal treatment, hypotensive drug, and treatment of hepatocellular carcinoma.

We hope that future studies will help endoscopists who encounter bleeding from the gastric mucosa discriminate PHG and *H. pylori*-related gastritis (even in *H. pylori*-negative cases) and that a diagnosis method using NBI will be established by a multicenter randomized controlled study.

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

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