# Portal Hypertensive Gastropathy and Colopathy

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**KEYWORDS**

• Cirrhosis • Hemorrhage • Bleeding • Pressure • Portal hypertension

**KEY POINTS**

- PHG and PHC can cause acute and/or chronic gastrointestinal bleeding.
- Diagnosis for both is endoscopic.
- The specific management of PHG and PHC depends on the clinical presentation.
- For acute bleeding, hemodynamic stabilization with intravenous (IV) fluids, IV antibiotics, and blood transfusion as needed should be begun immediately. This should be followed by IV pharmacologic therapy to decrease portal pressure, and subsequently by nonselective β-blockers.
- In patients with chronic bleeding, therapy with β-blockers and iron replacement is recommended. The role of TIPS is controversial.
- Patients with refractory bleeding should be managed on an individual basis.

## INTRODUCTION

The most common cause of portal hypertension is liver cirrhosis, which causes so-called intrahepatic or sinusoidal portal hypertension. Other disorders including presinusoidal and postsinusoidal diseases (ie, portal vein thrombosis, schistosomiasis, veno-occlusive disease, cardiac failure) may also cause increased portal pressure. Portal hypertension likely causes hemodynamic and mucosal changes in the entire gastrointestinal (GI) tract. This article focuses on the pathogenesis, diagnosis, and treatment of portal hypertensive gastropathy (PHG) and colopathy (PHC) (**Table 1**).

The cause of PHG and PHC is incompletely understood. However, available data indicate that portal hypertension is a critical component. It has been recognized

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**Table 1**
**Features of portal hypertensive gastropathy and colopathy**

| Features | Portal Hypertensive Gastropathy | Portal Hypertensive Colopathy |
|---|---|---|
| Endoscopic characteristics | Mosaic pattern and red spots | Mosaic pattern and red spots, sometimes, vascular ectasia appearance |
| Pathology | Dilated capillaries and venules, no inflammation | Edema and capillary dilatation, lymphocytes and plasma cells, in lamina propria |
| Treatment | Iron-replacement therapy<br>Transfusions<br>Portal pressure–reducing agents | [a]Iron-replacement therapy<br>Transfusions<br>Portal pressure–reducing agents |
| Salvage treatment | TIPS/shunt surgery<br>APC<br>Liver transplantation | TIPS/shunt surgery<br>APC<br>Liver transplantation |

Current practice is based on case and case series reports.
[a] There are insufficient data for standard recommendations in PHC bleeding.

that mucosal changes in the gastric mucosa of patients with portal hypertension were different pathologically from inflammatory gastritis; this led to the early description "congestive gastropathy."[1] The primary pathologic change was characterized by vascular ectasia. PHG is recognized endoscopically as a mosaic-like pattern called snakeskin mucosa with or without red spots.[2] Additionally, the terms portal hypertensive enteropathy[3,4] and PHC[5,6] were created to describe similar changes in the small bowel and colonic mucosa, respectively. PHC is characterized by erythema of the colonic mucosa, vascular lesions including cherry-red spots, telangiectasias, or angiodysplasia-like lesions.

PHG and PHC are important clinically because they may lead to chronic and/or acute GI bleeding. Both disorders are often confused with other diseases that can present similarly. Careful investigation is essential to accurately delineate the proper diagnostic needs and to start specific treatment.

## PORTAL HYPERTENSIVE GASTROPATHY

### *Epidemiology*

The prevalence of PHG in patients with cirrhosis varies from 20% to 98%.[2,7–12] This variation seems to be caused by several factors, including the study of different populations and variable patient selection, different interpretation of endoscopic lesions, and lack of uniform diagnostic criteria and classification.

Some studies have demonstrated a higher prevalence of PHG in patients with advanced liver disease, esophageal varices, or history of sclerotherapy or ligation for esophageal varices.[7,9,10] In general, the available data suggest that PHG is often associated with more severe portal hypertension.[13] It has also been suggested that the prevalence of PHG increases as esophageal varices are obliterated,[2] although this point is controversial.

### *Clinical Findings*

Most patients with PHG are asymptomatic, but a significant number of patients exhibit symptoms related to chronic GI bleeding and chronic blood loss/iron deficiency anemia. A smaller proportion of patients exhibit evidence of active GI bleeding.