

# Endoscopic Treatment of Gastric Varices



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## KEYWORDS

- Portal hypertension • Gastric variceal sclerotherapy • Gastric variceal obturation
- Gastric variceal band ligation • Thrombin injection • Combined endoscopic therapy
- EUS-guided therapy

## KEY POINTS

- Gastric variceal (GV) bleeding is the cause of upper gastrointestinal bleeding in 1 of 5 patients with portal hypertension and variceal bleeding.
- GV bleeding is associated with high morbidity and mortality and hence early detection and control of bleeding is important. An algorithmic approach to the management of GV bleeding is desirable.
- Endoscopic GV obturation by glue is the method of choice. Most often a single injection is effective but sometimes repeat sessions every 4 weeks may be needed for complete obturation of varices.

## INTRODUCTION

Gastroesophageal varices are present in approximately 50% of patients with cirrhosis. Their presence correlates with severity of liver disease; although only 40% of patients who are Child A have varices, they are present in 85% of patients who are Child C.<sup>1,2</sup> Gastric varices are present in approximately 20% of patients with portal hypertension (PHT) either in isolation or in combination with esophageal varices (EVs).

Variceal hemorrhage occurs at yearly rate of 5% to 15%, and about 20% of cirrhotic patients with acute variceal bleeding die within 6 weeks.<sup>3–5</sup> Although variceal bleeding ceases spontaneously in 40% to 50% of patients, the incidence of early rebleeding ranges between 30% and 40% within the first 6 weeks, and about 40% of all rebleeding episodes occur within the first 5 days.<sup>4,6</sup> Gastric varices bleed less frequently than EV and are the bleeding source in approximately 10% to 30% of patients with variceal hemorrhage.<sup>7</sup> However, gastric variceal (GV) bleeding tends to be more

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severe with higher mortality. In addition, a high proportion of patients, from 35% to 90%, rebleed after spontaneous hemostasis.

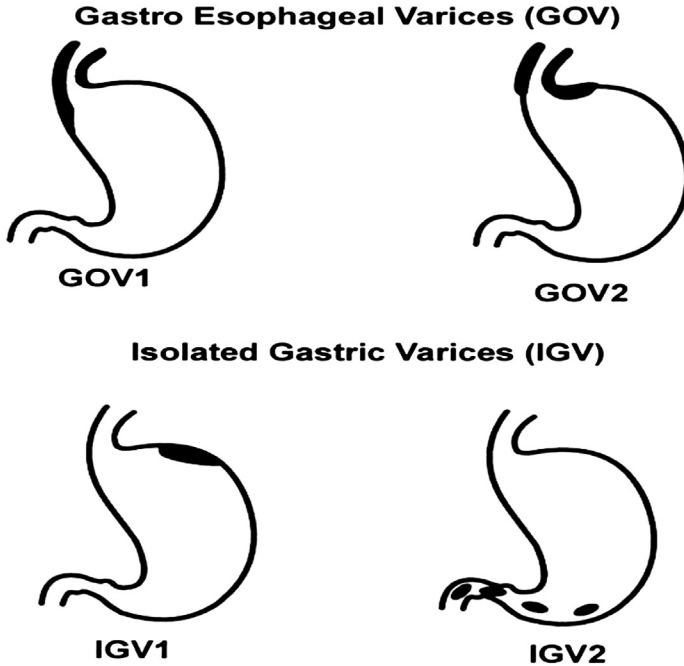
New endoscopic treatment options and interventional radiological procedures have broadened the therapeutic armamentarium for GV. This article provides background information on the classification and pathophysiology of formation of GV to help readers understand the management strategies primarily related to endoscopic approaches.

### CLASSIFICATION OF GASTRIC VARICES

The most widely accepted and used classification system is the Sarin classification (Fig. 1).<sup>7</sup> This system has been recommended for use by the Asian Pacific Association for the Study of the Liver (APASL), American association for the study of liver diseases (AASLD), Baveno guidelines and by the expert panel because it is easy to use, has good correlation with pathophysiology, and guides therapy. It classifies GVs by their location in the stomach and their relationship with EVs. It also helps to propose treatment strategies.

#### *Primary and Secondary GV*

GVs may be considered primary or secondary. Primary GV are present at initial examination or are seen in patients who have never had EV endoscopic variceal sclerotherapy (EVS) or endoscopic variceal band ligation (EVL). Secondary GV are those that develop after endoscopic therapy (either EVS or EVL) for EV.



**Fig. 1.** Gastric varices are categorized into 4 types based on the relationship with EV, as well as by their location in the stomach: gastroesophageal varix (GOV) type 1, GOV type 2, isolated gastric varix (IGV) type 1, and IGV type 2. GOV type 1 is the most common type, accounting for 74% of all gastric varices. However, the incidence of bleeding is highest with IGV type 1 followed by GOV type 2.

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