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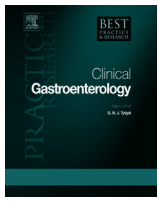

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Variceal and other portal hypertension related bleeding

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A B S T R A C T

Variceal bleeding is one of the commonest and most severe complications of liver cirrhosis. Even with the current best medical care, mortality from variceal bleeding is still around 20%. When cirrhosis is diagnosed, varices are present in about 30–40% of compensated patients and in 60% of those who present with ascites. Once varices have been diagnosed, the overall incidence of variceal bleeding is in the order of 25% at two years. Variceal size is the most useful predictor for variceal bleeding, other predictors are severity of liver dysfunction (Child–Pugh classification) and the presence of red wale marks on the variceal wall. The current consensus is that every cirrhotic patient should be endoscopically screened for varices at the time of diagnosis to detect those requiring prophylactic treatment. Non-selective beta-adrenergic blockers (NSBB) and endoscopic band ligation (EBL) have been shown effective in the prevention of first variceal bleeding. The current recommendation for treating acute variceal bleeding is to start vasoactive drug therapy early (ideally during the transferral or to arrival to hospital, even if active bleeding is only suspected) and performing EBL. Once bleeding is controlled, combination therapy with NSBB + EBL should be used to prevent rebleeding. In patients at high risk of treatment failure despite of using this approach, an early

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covered-TIPS within 72 h (ideally 24 h) should be considered. Data on management of gastric variceal bleeding is limited. No clear recommendation for primary prophylaxis can be done. In acute cardiofundal variceal bleeding, vasoactive agents together with cyanoacrylate (CA) injection seem to be the treatment of choice. Further CA injections and/or NSBB may be used to prevent rebleeding. TIPS or Balloon-occluded retrograde transvenous obliteration when TIPS is contraindicated may be used as a rescue therapy.

Introduction

Variceal bleeding is one of the commonest and most severe complications of liver cirrhosis. Even with the current best medical care, mortality from variceal bleeding is still around 20%. Moreover, variceal bleeding often leads to deterioration in liver function, and it is a common trigger for other complications of cirrhosis, such as bacterial infections or hepatorenal syndrome.

Natural history of varices in cirrhosis

When cirrhosis is diagnosed, varices are present in about 30–40% of compensated patients and in 60% of those who present with ascites [1]. The annual incidence of new varices in those cirrhotic patients who present without is about 5–10% [2]. A hepatic venous pressure gradient (HVPG) over 10 mmHg is the strongest predictor for the development of varices [3].

Once developed, the reported rate of progression from small to large varices is highly variable, ranging from 5% to 30% per year [2]. The factor that has been most consistently associated with variceal progression is baseline Child–Pugh or its worsening during follow-up [2,4]. Other factors were alcoholic etiology of cirrhosis and the presence of red wale markings [2]. It has been shown that when HVPG decreases below 12 mmHg (either "spontaneous" or caused by drug therapy or TIPS) esophageal varices decrease in size [5]. Thus, an increased HVPG plays a key role both in development and progression of the varices.

Once varices have been diagnosed, the overall incidence of variceal bleeding is in the order of 25% at two years [6]. Variceal size is the most useful predictor for variceal bleeding [7], other predictors are severity of liver dysfunction (Child–Pugh classification) and the presence of red wale marks on the variceal wall [8]. These risk indicators have been combined in the NIEC index which allows to classify patients in different groups with predicted one-year bleeding risk ranging from 6% to 76% [8]. The risk of bleeding is very low (between 1 and 2%) in patients without varices at the first examination, and increases to about 5% per year in those with small varices and to 15% per year if medium or large varices are present at diagnosis [1].

Prevention of first bleeding from esophageal varices

Screening for esophageal varices

The current consensus is that every cirrhotic patient should be endoscopically screened for varices at the time of diagnosis [9] to detect those requiring prophylactic treatment. In patients without varices on initial endoscopy, a second (follow-up) evaluation should be performed after 2–3 years [10]. In patients with small varices, if beta-blockers are not initiated, a follow-up endoscopy should be performed every 1–2 years to check for a possible increase in size, based on an expected 10%–15% per year rate of progression of small to large varices. This time must be shortened in case of clinical decompensation. No follow-up endoscopy is needed once beta-blockers are started.

Doppler-US [11] or transient elastography measurements by fibroscan [12–14] have been proposed as possible surrogate markers of the presence of esophageal varices but none has proved to be accurate enough to safely avoid endoscopy.