Techniques in Gastrointestinal Endoscopy 19 (2017) 79–83

Contents lists available at ScienceDirect

Techniques in Gastrointestinal Endoscopy

journal homepage: www.techgiendoscopy.com/locate/tgie

Nonendoscopic management of acute esophageal variceal bleeding

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ARTICLE INFO

Article history:
Received 10 October 2016
Accepted 12 March 2017

Keywords:
Portal hypertension
Acute esophageal variceal bleeding
Terlipressin
Balloon tamponade
Transjugular intrahepatic portosystemic shunt
Self-expanding metal stents

ABSTRACT

Acute esophageal variceal bleeding is a life-threatening complication of portal hypertension in patients with liver cirrhosis. Its management has improved over the past several years, leading to a significant reduction in rebleeding episodes and in bleeding-related deaths. Although endoscopic therapy is an integral part in the management of the acute variceal bleeder, pharmacologic and radiologic therapies are important interventions, in addition to optimal supportive care. Herein, we highlight the non-endoscopic management of acute esophageal variceal bleeding.

1. Introduction

Acute esophageal variceal bleeding (AVB) is a frequent and severe complication among patients with liver cirrhosis and portal hypertension. Up to 40% of patients with cirrhosis will have an episode of AVB during the course of their disease. Advances in the general and specific management of the AVB episode have led to a reduction in rates of rebleeding and death. The aim of this review is to highlight the nonendoscopic management of AVB (Figure).

2. General management

2.1. Resuscitation

The immediate goals of management are to achieve hemodynamic stability and prevent bleeding-related complications (bacterial infections, hepatic decompensation, and renal failure). Initial resuscitation should follow the airway, breathing protection, and circulation stability principles to maintain the aerobic metabolism and provide appropriate delivery of oxygen to tissues (which depends on oxygen saturation, cardiac output, and hemoglobin concentration).

Airway should be immediately secured, especially in patients with severe hepatic encephalopathy (HE), to prevent aspiration of gastric contents and blood [1], a risk further exacerbated by endoscopic procedures [2]. Therefore, endotracheal intubation is mandatory before endoscopy if there is concern about airway safety. At least 2 large-bore intravenous catheters (14-18 G) should be in place to enable rapid administration of volume expanders, as appropriate, during initial resuscitation. A central line for central venous pressure and oxygen saturation monitoring is advisable [3]. Although evidence regarding its benefit of survival is lacking, the use of a nasogastric tube may be useful to monitor either active hemorrhage or recurrence [4]. It also allows removing blood from the stomach and improving its visualization during endoscopy.

2.2. Volume replacement

Volume restitution should be cautiously administered to maintain adequate tissue oxygenation. Blood volume replacement should be initiated as soon as possible with plasma expanders to maintain systolic blood pressure of approximately 100 mm Hg. A certain degree of hypovolemia and hypotension may help stop the bleeding by promoting the activation of endogenous vasoactive agents leading to splanchnic vasoconstriction and, therefore, a reduction in portal blood flow and portal pressure. On the

Conflict of interest: Prof J.C.G.P. received speaker fees from Gore and research Grants from Novartis (CPLATFORM2017) and Exalenz (CSPH-EX0414).

Financial support: Supported in part by grants from Ministerio de Educacion y Ciencia (Grant number SAF-2013-44723-R), and from Instituto de Salud Carlos III, Spain (Grant no. PI14/00182). CIBERehd is funded by Instituto de Salud Carlos III, Spain. Gilberto Silva-Junior is funded by CAPES Foundation, Ministry of Education of Brazil, Brazil (Process no. BEX 5960/13-4).

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http://dx.doi.org/10.1016/j.tgie.2017.03.001
0049-0172/

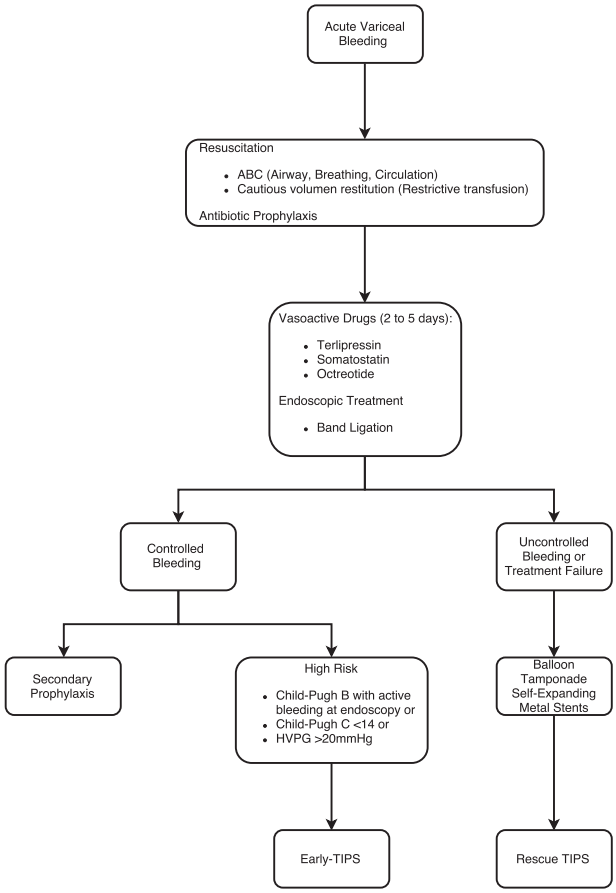

Fig. Suggested approach to the management of acute esophageal variceal bleeding.

contrary, it is also important to avoid prolonged hypotension to prevent infections, renal failure and deterioration of liver function, situations associated with an increased risk of rebleeding, and death [3]. Overtransfusion should be avoided as it might induce a rebound in portal pressure and aggravate bleeding, or increase the risk of early rebleeding as well as pulmonary congestion [5,6]. Transfusion of packed red blood cells must follow a restrictive strategy. Indeed, a blood transfusion strategy aimed at maintaining hemoglobin to a target level of 7-8 g/dL has been shown to improve survival in patients with Child-Pugh class A and B cirrhosis having gastrointestinal bleeding. Therefore, packed red blood cells are administered when the hemoglobin drops less than 7 g/dL [7]. Exceptions to this rule are patients with massive exsanguinating hemorrhage and patients with ischemic cardiovascular disease [8].

2.3. Coagulation status

The prothrombin time and international normalized ratio are not reliable indicators of the coagulation status in patients with cirrhosis. Furthermore, recent studies suggest a hypercoagulable state in patients with cirrhosis owing to an imbalance between procoagulant and anticoagulant factors [9]. This may explain why the use of recombinant activated factor VII (NovoSeven), which aims to improve coagulation abnormalities [10], has not been shown to be useful in the management of acute variceal bleeding in 2 randomized controlled trials (RCTs) [11,12]. Although a meta-analysis of these 2 trials showed a small benefit in the subgroup of high-risk patients (Child-Pugh score ≥ 8 points and active bleeding at endoscopy), the use of recombinant activated factor VIIa should only be considered on the basis of the individual patient characteristics [13]. Indeed, the Baveno VI consensus states that recommendations regarding management of coagulopathy and thrombocytopenia cannot be made based on currently available data [8].

2.4. Infection

Infection is a strong prognostic indicator in AVB [14], and a potential contributor to perpetuating bleeding or precipitating renal failure and liver dysfunction. In the setting of AVB, approximately 20% of patients are infected on the day of hospital admission and, in the absence of antibiotic prophylaxis, up to 50% of patients develop an infection during their hospitalization [15]. The most commonly reported infection processes are bacteremia (19%-56%), spontaneous bacterial peritonitis (19%-37%), urinary tract infections (12%-34%), and pneumonia (12%-19%) [15-18].

The use of prophylactic antibiotics in the setting of AVB has been shown to reduce both the risk of rebleeding and mortality [15,17,19]. Therefore, antibiotic prophylaxis should be administered in all patients with AVB upon admission and continued for 5-7 days [20]. The antibiotic of choice should be based on individual patient risk characteristics and local antimicrobial susceptibility patterns. Quinolones are frequently used owing to its ease of administration and low cost [21]. In high-risk patients with ≥ 2 of the following factors: ascites, jaundice, serum bilirubin > 3 mg/dL, malnutrition, or HE, intravenous ceftriaxone (1 g/d for 7 days) has shown to be superior to oral norfloxacin in preventing infections [22]. Quinolone resistance was the major cause of quinolone failure in these patients. Therefore, intravenous ceftriaxone should be considered the antibiotic of choice in patient populations with severely decompensated cirrhosis, high prevalence of quinolone resistance, or prior use of a quinolone as prophylaxis [20]. The risks of bacterial infection and mortality are very low in patients with Child-Pugh class A cirrhosis. Although it has been proposed recently that the use of antibiotics in this subgroup of patients may be avoided, additional evidence-based data from prospective studies are needed [23].

2.5. Hepatic encephalopathy

Societal guideline recommendations [24] regarding the management of HE in the setting of AVB include the following: (1) initiating care for altered consciousness; (2) searching for and treating altered mental status; (3) identifying and correcting precipitating factors; and (4) starting empiric HE treatment. Lactulose is used as first-line therapy for episodic HE precipitated by AVB, with 25 mL of the agent given every 12 hours followed by dose titration to achieve and maintain at least 2-3 soft bowel movements per day. Careful titration is important as excessive lactulose can lead to dehydration, hypernatremia, acute kidney injury, aspiration, and even precipitation of HE. As per the guidelines, once the symptoms of HE resolve and the precipitating event (ie, AVB) is brought under control, continued prophylactic lactulose may not be needed [24].

3. Hemostatic therapy

Hemostatic therapy in AVB is essential to achieve control of the acute bleeding episode, but also to prevent early rebleeding. First-line therapies for AVB include vasoactive drugs, started as soon as possible even before diagnostic endoscopy, and endoscopic therapy, preferably band ligation, once the diagnosis of AVB is confirmed by upper endoscopy. If initial endoscopic therapy fails, balloon tamponade or covered self-expanding metal stents can be used to control bleeding as a temporary “bridge” to definitive