Current trends: Management of esophageal varices

Madam, gastroesophageal varices are a complication of portal hypertension in patients with cirrhosis. These are dilated sub mucosal veins which arise due to increased portal blood flow and increased resistance to its flow. The increased resistance to blood flow is due to structural aberration from fibrosis and endothelial dysfunction. These porto-systemic collaterals are of importance due to excessive bleeding from their rupture.

To assess presence of varices, several modalities are available. In patients with compensated cirrhosis, the gold standard is Esophagogastroduodenoscopy (EGD).[1] Other alternatives are capsule endoscopy and a ratio of platelet count to spleen size. Similarly, different methods are available for risk stratification of patients with varices. Of these methods, Hepatic Venous Pressure Gradient (HVPG) assessment is the best available. Other measures include measurement of liver stiffness, Child Pugh and Model for End-Stage Liver Disease scores.

Three pronged strategy is used for management of patients with varices: prevention of primary bleed (primary prophylaxis), treatment of acute attack and prevention of recurrent bleeds (secondary prophylaxis). The use of beta blockers is discouraged in patients with cirrhosis but no varices, as they do not delay the onset of varices. For primary prophylaxis, non-selective beta blockers can be used in low risk patients along with timely screening for variceal growth. For patients with small varices with high risk of haemorrhage, non selective beta blockers are recommended. In patients with medium to large varices, endoscopic ligation of varices and non-selective beta blockers have been shown to have equal efficacy.[2]

The standard protocol for treatment of acute variceal haemorrhage in low and intermediate risk patients (Child Pugh class A or B; or HVPG <20mm Hg) is trimodal. It consists of a combination of vasoconstrictors, antibiotics and endoscopic therapy. The vasoconstrictors (vasopressin, somatostatin or their analogues) should be administered from time of admission and continued for 2-5 days. Endoscopic therapy consists of variceal ligation and should be performed under 12 hours after admission.[3] Antibiotics of choice in such patients are fluoroquinolones or ceftriaxone. For patients with severe disease, addition of ceftriaxone to treatment plan is recommended. For patients who poorly respond to therapy, Transjugular Intrahepatic Portosystemic Shunt (TIPS) is the salvage treatment.

For patients who are at risk of recurrent haemorrhage, a combination of endoscopic ligation and drug therapy (non-selective beta blockers and nitrates) is more effective than either alone.[4] For those who respond poorly, percutaneous placement of TIPS or a surgical shunt is recommended. Both are equally effective. Although lower HVPG values (better HVPG response to therapies) correlates with lower chance of recurrent bleed, randomized controlled trials are needed to demonstrate that HVPG guided therapy is better than empirical therapy.

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