Endoscopic Sclerotherapy

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UPPER gastro intestinal bleeding due to gastro-esophageal varices is a common medico-surgical problem. Most of the available reports show that oesophageal varices are the most important causes of upper gastro intestinal bleeding in the third world. Patients may present as medical emergency due to massive haemorrhage or may seek help in the interval period of bleeding from varices. A few patients may have varices but without any history of bleeding.

Oesophageal varices are a consequence of portal hypertension. Disease processes that damage or altar the flow of blood through the liver or its major vessels are responsible for development of portal hypertension. The causes of portal hypertension are therefore broadly categorised into extra hepatic and intrahepatic. The common extrahepatic causes are portal vein thrombosis, portal vein anomaly and splenic vein thrombosis. The commonest intrahepatic cause of portal hypertension is cirrhosis. Non-cirrhotic portal fibrosis is increasingly being recognised in tropical countries as a cause of portal hypertension. Hepatic venous outflow obstruction (Budd-Chiari Syndrome) is responsible for portal hypertension in the remaining cases.

The major life threatening complication that accompanies portal hypertension is haemorrhage. As portal pressure rises, the rectal veins, abdominal wall veins and oesophago gastric veins attempt to lower portal pressure and become dilated and distended. Veins of the stomach and oesophagus are the most susceptible to rupture.

Oesophageal varices will develop in approximately 50% of patients with cirrhosis and more than one-third of these will bleed. Conventional medical and surgical treatments for those who bleed are not always satisfactory. Endoscopic sclerotherapy is a recently revived and modified technique, which provides an important therapeutic option for the management of variceal haemorrhage.

Endoscopic sclerotherapy was introduced into medicine in 1939 by two Swedish otolaryngologists, Craford and Freckner. In this technique a sclerosing solution is injected into or around oesophageal varices via a long, flexible needle that passes through an endoscope. The procedures used vary widely in the type of endoscopes, injectors, sclerosants and adjuncts employed, in the site of injection, the amount of contrast, and the timing of the injection.

The various sclerosants used are given below:

Sclerosing agents commonly used

(1) Fatty acid derivatives:

(a) Sodium Morrhuate

(b) Ethanolamine oleate

(2) Synthetic products:

(a) Sodium Tetradecyl Sulphate

(b) Polydocanol (Hydroxy-Polyethoxydodecan)

(3) Other agents:

(a) Alcohol

(b) Phenol

Mechanism of action: Thrombosis occurs within 24 hours of injection of the sclerosant, along with tissue necrosis. This is followed by ulceration in a few days. Submucosal fibrosis develops in a month's time.

Injection sclerotherapy is given in one treatment session with several injections. For chronic varices, injection is done every 1-4 weeks until all veins in the lower oesophagus are obliterated. Rebleeding is most frequent before the varices have been completely obliterated. However, new vessels undoubtedly develop, and the patient has to be followed up endoscopically every six months.

Indications for Sclerotherapy

(1) Control of acute variceal haemorrhage

The patients with acute variceal bleeding have an increased risk of rebleeding within the first two weeks of original bleeding. So the aim of treatment is to stop the active bleed and to prevent further recurrence. Bleeding stops spontaneously in one half to two-thirds of patients with cirrhosis. Resuscitation is the first step in the management of patients with acute bleeding. Emergency sclerotherapy is found to be effective in achieving haemostasis in 72-100% of cases. However, it is always advisable to resort to simpler methods initially to control bleeding before resorting to sclerotherapy. These include nor-adrenaline gastric lavage, vasopressin infusion and Sengstaken-Blakemore tube introduction. Once initial haemostasis is achieved, patients are subjected to sclerotherapy. This will prevent rebleeding.

(2) Bleeding free interval sclerotherapy

This is done to prevent recurrent variceal haemorrhage. Repeated sclerotherapy lead to obliteration of varices, reduce the re-bleeding rate, and improve survival of patients.

(3) Prophylactic sclerotherapy

Variceal bleed is a common cause of death in patients with cirrhosis. It is therefore necessary to identify those patients who are at high risk for bleeding from varices. It is found that approximately 50% of cirrhotics who bled, did so within the first

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year of diagnosis. So it is argued that if the patients with high risk can be identified and subjected to sclerotherapy, bleeding could be prevented and mortality reduced. But the problem is that there are no definite predictive features available to denote which patient will bleed. There is no correlation between portal pressure or size of the varices and the chance of bleeding. Moreover, there is a subset of patients with varices, who may not bleed at all. Considering all these facts, prophylatic sclerotherapy is now-a-days not recommended.

Technique

In our medical college, sclerosant is injected paravariceally and intravariceally. This ensures adequate occlusion of the varices. The procedure has to be explained since co-operation of the patient is very important for the proper introduction of the endoscope. Patient is positioned in the left lateral recumbent after giving 5-10 mg of Inj. Diazepam and 50 mg Inj. Hyoscine intravenously. After 5-10 minutes a mouth gag is placed in the mouth. Through the mouth gag the fibroptic endoscope is introduced till it reaches the lower end of oesophagus. Through the endoscope the sclerosant is injected into and around the varices till they are blanched and are engorged by the sclerosant. The dose is 2 ml per varix upto 15-30 ml per session. Then the endoscope and mouth gag are removed. Following this the patient has to be observed for early detection of complications.

Complications of injection sclerotherapy

The complications associated with endoscopic sclerotherapy can be severe and life threatening. Following sclerotherapy complications can occur in 10-15% cases. The common complications related to the procedure are oesophageal ulcerations, necrosis, perforations, mediastinitis, pleural effusion and oesophageal stenosis. Oesophageal ulcers are often found 5-7 days after injection and can occur in 30-80% of patients. Most of them heal with the passage of time. Most oesophageal strictures respond to dilatation.

Complications of injection sclerotherapy

(1) Oesophageal:
   (a) Ulceration
   (b) Haemorrhage
   (c) Perforation
   (d) Stricture
   (e) Intramural haematoma

(2) Pulmonary:
   (a) Aspiration pneumonitis
   (b) Pleural effusion
   (c) Mediastinitis
   (d) Emphysema
   (e) Transient pulmonary infiltrates
   (f) Acute respiratory distress like syndrome
   (g) Broncho oesophageal fistula

(3) Miscellaneous:
   (a) Bacteremia
   (b) Pyrexia
   (c) Chest pain
   (d) Portal vein thrombosis
   (e) Spinal cord paralysis
   (f) Development of other intestinal varices.

Conclusion

Variceal haemorrhage is a catastrophic complication for some patients with portal hypertensisn. Endoscopic sclerotherapy has emerged as a valuable alternative therapeutic option; however its precise indications are yet to be defined. Studies have indicated that this method can stop active bleeding in 72 to 93% patients. It appears to be superior to balloon temponade and vasopressin therapy either alone or in combination; and it is equally effective as emergency porto-caval shunts and stapling transection in the immediate cessation of bleeding. It has a definite role in prevention of recurrent variceal bleeding. The role of prophylactic sclerotherapy is controversial.

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