

Leading article

Complications and limitations of injection sclerotherapy in portal hypertension

The pioneering work of Whipple in 1945¹ began an era of portosystemic shunt surgery for portal hypertension which remained the primary form of treatment for nearly 30 years even though the consequences of portal diversion such as liver atrophy and encephalopathy were soon recognised.² Repeated trials in patients with bleeding varices confirmed that the complications of medical management were related to recurrent bleeding whereas surgical treatment was followed by liver failure and encephalopathy. Refinements of surgical technique eventually culminated in the selective shunt procedures developed by Warren *et al*³ which were designed to decompress the gastro-oesophageal varices while preserving portal blood flow and liver function.

Endoscopic sclerotherapy as an alternative form of treatment for bleeding varices was first reported in 1939⁴ in a young patient with extrahepatic portal vein occlusion and was eventually popularised as an alternative to surgery by the work of Johnston and Rodgers.⁵ A number of controlled trials rapidly established the effectiveness of this treatment and the value of sclerotherapy in the initial management of bleeding varices, particularly in the cirrhotic patient, is now generally recognised.⁶⁻¹⁰ The variety of complications that may be associated with sclerotherapy is less well known, and the role of surgery in the management of recurrent bleeding and other sequelae is not always appreciated.

Uncontrolled oesophageal bleeding

Randomised controlled trials in patients with chronic liver disease have shown that endoscopic sclerotherapy reduces the risk of rebleeding from oesophageal varices^{8, 11, 12} and may prolong survival. Rebleeding during a course of endoscopic sclerotherapy treatment occurs in 23%–55% of patients¹³ and remains a significant cause of death in the cirrhotic patient. The reported figures for failure of sclerotherapy vary widely from 5% to 60%,^{8, 10, 14, 15} but this variation can be partly explained by the lack of a universal definition of 'failure of treatment'. Westaby¹⁶ defined a failure of sclerotherapy as an inability to control an episode of variceal bleeding rather than episodes of recurrent bleeding easily controlled by further injections. Using this definition at King's College Hospital, surgical intervention was necessary in 12 of 252 children (4.4%) and 25 of 540 of adults (4.6%) with bleeding oesophageal varices treated by injection sclerotherapy over a five year period. A similar failure rate for sclerotherapy of 6% was reported by Jenkins and Shields¹⁷ in patients presenting with their first episode of variceal haemorrhage. There was a recurrent bleeding rate of 42% in this series, only 13% of whom were uncontrolled by conservative treatment. The failures usually occurred in patients with poor liver function.

The options available for the surgical treatment of variceal haemorrhage during or after sclerotherapy now include hepatic transplantation as well as devascularisation or portosystemic shunting. Control of bleeding with a Sengstaken-Blakemore tube followed by emergency transplantation has been suggested for patients with poor hepatic reserve,¹⁸ and Iwatsuki *et al*¹⁹ have proposed that transplantation is the best

option for most patients with advanced liver disease and recurrent bleeding. This ideal management, however, cannot be offered to most patients, who will either not be on a transplant programme or who may be too ill for such a radical procedure. Other forms of surgery are, therefore, still necessary for the many patients who suffer uncontrollable or repeated haemorrhage after sclerotherapy. It is now clear that an emergency portosystemic shunt or a devascularisation procedure does not necessarily prejudice the subsequent performance of liver transplantation.²⁰

Emergency portosystemic shunting is still recommended by some centres as the most effective therapy for bleeding²¹⁻²⁴ while others prefer staple gun transection of the oesophagus which is the safer technique for the less experienced surgeon.²⁵ Emergency oesophageal transection which includes devascularisation of the lower oesophagus and the upper two thirds of the stomach is associated with mortality rates of up to 30%,²⁶ which are similar to mortality figures reported after emergency shunting. The King's College Hospital series showed mortality rates of 10% for portosystemic shunt therapy and 40% for emergency devascularisation procedures, but these treatment groups were selected on the basis of portal anatomy and liver function status and were not therefore comparable.

Millikan *et al*²⁷ made an interesting comparison of biochemical and physiological data in 24 patients who had undergone transplantation and a group of patients treated with shunt surgery and sclerotherapy. The authors concluded that patients with good liver function usually progress well after appropriate shunt surgery whereas those with advanced disease will need transplantation. They also proposed that the aetiology of the portal hypertension might influence the type of surgery offered for recurrent bleeding from failed sclerotherapy.

Gastric varices

Gastric varices are not uncommon in cirrhotic patients with portal hypertension.²⁸ Most are a direct continuation of oesophageal varices and may respond to injection sclerotherapy from reflux of sclerosant below the cardia. Sarin *et al*²⁹ found that gastric varices disappeared in 28% of patients with endoscopic obliteration of oesophageal varices while Terblanche *et al*³⁰ reported the disappearance of all gastric varices after successful sclerotherapy. Approximately 5% of patients have a risk of bleeding from gastric varices after successful obliteration of their oesophageal veins.^{31, 32}

Fundal varices originate from the short gastric and epiploic veins in about 12% of patients³³ and occasionally follow an isolated splenic vein thrombosis. They are usually inaccessible to endoscopic injection sclerotherapy, particularly when accompanied by acute bleeding, although Sarin *et al*²⁹ did control haemorrhage in 6 of 8 patients. Gastric varices are particularly difficult to diagnose during active bleeding. Under-running of the bleeding varix with or without devascularisation or portosystemic shunting are possible surgical options.

Ectopic varices

Ectopic varices, defined as varices arising in sites other than the oesophagus or cardia of the stomach, may develop spontaneously following endoscopic obliteration of oesophageal varices or occur as a result of surgery and subsequent adhesion formation. The prevalence of bleeding from this type of varix has been reported in 1.4% to 5% of patients with intrahepatic portal hypertension and in 27% to 40% of patients with extrahepatic portal hypertension.³⁴

Duodenal varices account for one third of all ectopic variceal bleeding and usually occurs in adults.³⁵ However, they are well recognised in children with extrahepatic portal hypertension and almost half of these patients have a history of previous surgery. Successful treatment of duodenal varices has been reported with injection sclerotherapy, but surgical ligation or portosystemic decompression is usually required.

Jejunal and ileal varices account for approximately one third of bleeding ectopic varices.³⁴ These patients usually have a history of previous abdominal or pelvic surgery and collaterals develop via adhesions between the gastrointestinal tract and the abdominal wall. Patients bleed from large submucosal veins within the small bowel and although surgical excision of the affected segment may provide long term relief, recurrent bleeding can occur as further adhesions are formed. A portosystemic shunt is the treatment of choice if liver function is satisfactory.

Bleeding from colonic varices is a rare clinical problem but may account for up to 25% of bleeding ectopic varices, particularly in cirrhotic patients.³⁴ Approximately half of the varices are localised to the distal colon and rectum,³⁶ and 20% of patients have had previous surgical operations.³⁴ The varices may be seen on barium enema or at colonoscopy and aortoportography is diagnostic. The mortality after partial or total colectomy in adult patients with cirrhosis is more than 60%³⁴ and portosystemic shunting or transplantation, depending on the status of the liver disease, is preferable.

Anorectal varices represent collateral formation between the superior rectal vein which drains the submucosa of the lower rectum and upper part of the anal canal and the systemic venous drainage at the anus. In a prospective study of 100 cirrhotic patients, Hosking *et al*³⁷ found anorectal varices in 44% and haemorrhoids in 63% of patients. There was no clear relationship between the severity of portal hypertension and the prevalence of haemorrhoids, which have previously been shown to have no direct communication with the portal vein.³⁸ Anorectal complications of portal hypertension may occur early in life and in a recent study of children 4.7% presenting with bleeding oesophageal varices had symptomatic rectal varices or haemorrhoids.

The treatment of bleeding anorectal varices includes injection sclerotherapy, banding, cryosurgery, under-running of varices, haemorrhoidectomy, embolisation, and portosystemic shunt.^{39,40} Injection sclerotherapy is usually satisfactory for treating true haemorrhoids, but direct suture or banding are probably the more effective treatments for anorectal varices.^{40,41}

The formation of varices at the mucocutaneous junctions of small or large bowel stomas in patients with portal hypertension can be a source of massive or recurrent haemorrhage. Haemorrhage can be controlled by intraperitoneal closure of a temporary stoma,⁴² but the management of a permanent stoma is more difficult. Resuture of the stoma edge will be followed inevitably by recurrent bleeding and we have found that the therapeutic options are either a portosystemic shunt, if liver function is well preserved, or transplantation.

Spontaneous rupture of intraperitoneal varices is a rare complication in adults. The typical presentation is one of sudden onset of acute abdominal pain, hypotension, and increasing abdominal girth in a patient with cirrhosis. Urgent

surgical treatment by direct suture ligation is the treatment of choice, but the prognosis for most patients is extremely poor.⁴³

Adhesion related varices should be suspected in patients with recurrent melaena with a history of previous surgery and in whom upper intestinal endoscopy is unhelpful. Varices may form along adhesions between the abdominal wall and the gastrointestinal tract, particularly the small bowel.^{44,45} Labelled red blood cells and selective visceral angiography have been recommended to establish the diagnosis.⁴⁶ Localised resection of the affected segment has been performed, but adhesion varices tend to recur and are best treated by portosystemic shunting.

Massive splenic enlargement often accompanied by discomfort or pain in the left side of the abdomen is another uncommon but difficult problem in patients with unrelieved portal hypertension who have undergone variceal sclerosis. Abdominal symptoms or haematological changes of hypersplenism can be severe enough to warrant surgery.

Specific complications associated with endoscopic sclerotherapy

LOCAL COMPLICATIONS

The commonest complaints after injection sclerotherapy are of fever, retrosternal discomfort, and transient dysphagia which resolve within 24–48 hours.^{47,48} The inflammatory reaction underlying this local complication occasionally becomes more widespread and causes mediastinitis, pericarditis, and even cardiac tamponade.⁴⁹ Pleural effusions are found in up to half these patients,⁵⁰ although most are small and without clinical significance. Patients developing these complications have usually had large volume injections of sclerosant and postsclerotherapy chest pain. Other rare local complications of injection sclerotherapy include perforation of the thoracic duct with the development of a massive chylothorax,⁵¹ oesophageal perforation, and broncho-oesophageal fistula from delayed oesophageal necrosis.⁵²

Oesophageal ulceration is common, usually asymptomatic, and more likely to follow large volume or frequent injections of sclerosant.⁵³ Bleeding from these ulcers is often related to portal hypertension and may be controlled with an infusion of somatostatin, but occasionally it is related to arterial damage which will only respond to prompt surgical intervention. Oesophageal strictures are probably caused by a combination of chemical oesophagitis, ulceration, and acid reflux. They were found in 16% of 98 children who had completed a course of sclerotherapy,⁵⁴ all of whom responded to simple dilatation. Three patients continued to complain of intermittent dysphagia suggestive of a motility disturbance and these changes are probably permanent.⁴⁷ There is no clear correlation between the number of injections and the degree of abnormality recorded. Minor falls in the mean resting pressure of the lower oesophageal sphincter and increased gastro-oesophageal reflux have been noted,⁵⁵ but the most striking abnormality has been the incoordinated passage of oesophageal pressure waves.⁴⁷

Severe respiratory complications have also been reported after sclerotherapy including respiratory distress syndrome, pneumonia and atelectasis.

SYSTEMIC COMPLICATIONS OF INJECTION SCLEROTHERAPY

More widespread complications of sclerotherapy have been described and may be commoner than previously suspected.⁵⁶ Portal vein thrombosis may occur. Barsoum *et al*⁵⁷ reported a post mortem finding after sclerotherapy of portal vein thrombus extending into the left gastric vein and the submucosal plexus of the oesophagus. Another post mortem

study of three patients showed portal vein thrombosis with fresh thrombus extending from recently injected varices.⁵⁸ Intimal thickening, medial fibrosis, and microthrombus formation in the splenic vein have also been observed at the time of portosystemic shunt surgery in patients treated previously by injection sclerotherapy.^{56,59} However spontaneous portal vein thrombus may occur in portal hypertension⁶⁰ and the infusion of vasopressin may also play a part.

Case reports of rare complications of sclerotherapy include spinal cord paralysis after injection of sclerosant in the mid-oesophagus,⁶¹ brain abscess,⁶² digital gangrene,⁶³ and acute renal failure.⁶⁴ Mild and transient disseminated intravascular coagulopathy has been described,^{65,66} and transient bacteraemia occurs in 5–8% of patients during upper gastrointestinal endoscopy.⁶⁷ *Staphylococcus aureus* and alpha haemolytic streptococci were the most commonly identified organisms and were usually isolated from the biopsy channel of the endoscope and it seems likely that needle tip contamination is the chief culprit. Antibiotic prophylaxis is imperative for immunosuppressed patients or for those with valvular heart disease or prosthetic graft material. Anaphylactic shock has also been recorded after injection sclerotherapy using ethanolamine oleate.⁶⁸

Summary

Injection sclerotherapy is now the accepted first line treatment for bleeding oesophageal varices, although it is associated with an impressive list of rare complications. The main problem concerns the strategy for uncontrollable or recurrent bleeding. Patients with uncontrolled bleeding may be referred for surgery after considerable blood loss and are then extremely difficult to assess. The effects of blood loss on liver function can lead to an unduly pessimistic assessment of liver status.

An effective choice of emergency surgical procedure may require considerable surgical expertise. Oesophageal transection and devascularisation are satisfactory for many patients with oesophageal varices secondary to cirrhosis and should nearly always control bleeding. Difficulties arise in patients who are grossly obese and in those who have undergone extensive surgery in the upper abdomen. Problems may also be encountered in those treated by repeated sclerotherapy, which may have caused severe inflammatory change and thickening around the lower oesophagus and upper stomach. We believe that an emergency mesocaval shunt using either a vein graft or a synthetic material such as polytetrafluoroethylene is the procedure of choice for this difficult group of very sick patients. The surgical exposure is satisfactory and not unduly prolonged in even the largest patients and the technique does not interfere with any subsequent transplant operation.

There is a greater choice in the management of the patient with less urgent bleeding from recurrent varices after sclerotherapy. Repeat sclerotherapy may be effective for small oesophageal varices while liver transplantation may be indicated in the patient with deteriorating liver function. A selective distal splenorenal shunt should be considered for patients with intact splenic and left renal veins and a mesocaval vein graft for the remainder.

We would therefore suggest that surgery should still be considered for the management of portal hypertension, particularly in the following circumstances:

- (1) Uncontrollable bleeding during the initial course of sclerotherapy;
- (2) Life threatening haemorrhage from recurrent varices;
- (3) Bleeding from ectopic varices not accessible to sclerotherapy;
- (4) Uncontrollable bleeding from oesophageal ulceration secondary to injection sclerotherapy;

(5) Severe, symptomatic hypersplenism;

(6) For patients who live in communities remote from blood transfusion facilities and adequate medical care.

The management of the complications of portal hypertension continues to pose problems. We believe that the best results should come from a combined management approach using injection sclerotherapy as primary treatment and surgery for complications and for haemorrhage from unusual anatomical sites.

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