Treatment of Bleeding Gastroesophageal Varices: A Report of Forty-four Cases

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Summary

Bleeding gastroesophageal varices is associated with a high morbidity and mortality. Forty-four cases of bleeding gastroesophageal varices were treated at the Department of Surgery, Universiti Kebangsaan Malaysia, General Hospital, Kuala Lumpur over four and a half years. Thirty-two of them had liver cirrhosis. Hepatitis B infection was noted in 13 and alcoholic abuse was present in 14 patients. Five patients had associated hepatoma. Thirty-four percent had gastric fundal varices and a third of these bled from them. A total of 179 endoscopic injection sclerotherapy sessions were performed averaging 4 per person. Rebleeding rate was 4% and mortality was high (50%) in these cases. It was concluded that injection sclerotherapy is a safe and effective means of controlling bleeding oesophageal varices. Operative surgery was employed in those who rebled after injection and would be considered in those in Child's A.

Key words: Portal hypertension, oesophageal varices, gastric varices, injection sclerotherapy, portasystemic shunt

Introduction

Gastroesophageal varices result from portal hypertension, which is mainly due to liver cirrhosis. Treatment of acute variceal bleeding may pose a problem to clinicians in terms of choice and timing of specific therapy. In addition, these patients also had varying degrees of liver insufficiency. The mortality is high, especially in those associated with hepatic decompensation.

Material and Methods

A retrospective analysis of 44 cases of bleeding gastroesophageal varices was undertaken, with respect to etiological factors, modes of treatment and their outcome, at the University Department of Surgery, General Hospital, Kuala Lumpur, over four and a half years, from April 1986 to October 1990. The diagnoses of gastroesophageal varices were made by upper gastrointestinal endoscopy.

All patients except 2 had endoscopic injection sclerotherapy performed for bleeding varices. fibreoptic oesophagoscopy and intravariceal injections were employed using light sedation with topical anaesthesia. Sodium tetradecyl (1% or 3%) was used as the sclerosant. The total volume injected per session ranged from 6 to 15 millilitres.

Results

The patients' ages ranged from 11 to 77 years with the mean at 49.6. The male to female ratio was 2:1. The racial distribution was as shown in Table I.

Table I
Racial distribution

	Gastroesophageal varices		Gastric varices	
	n	(%)	n	(%)
Malays	19	(43.2%)	4	(21%)
Chinese	12	(27.3%)	8	(67%)
Indians	10	(22.7%)	2	(67%)
Others	3	(6.8%)	1	(33%)
Total	44	(100%)	15	

Fourteen patients imbibed alcohol regularly while 24 of them never did. It was noted that 70% of the Indian patients did so while 90% of the Malay patients did not. Thirteen patients tested positive for hepatitis B surface antigen while 19 of them were negative. The virology status of the remaining 12 patients could not be ascertained. Two patients fell into Child's classification[1] A, 21 into B and 9 into C. In 2 of the patients, their liver status could not be established.

Thirty-two patients had liver cirrhosis based on clinical criteria while 10 of them were not cirrhotic. The liver status of the remaining 2 patients could not be ascertained. Five of the cirrhotic patients also had hepatoma. Only 2 of these were histologically proven while the others had very suggestive clinical, radiological and biochemical features. Two of the patients with hepatoma tested positive for hepatitis B surface antigen while the remaining 3 tested negative. Of the patients without cirrhosis, 1 had carcinoma of the pancreas and 2 had portal vein thrombosis. The remaining 7 did not have sufficient clinical information available.

Seven cases of malignancies were recorded: hepatoma in 5 and 1 each for rectal carcinoma and carcinoma of the pancreas. About 20% of the cases had associated peptic diseases of the stomach or duodenum.

Table I also shows the racial distribution of patients with gastric fundal varices. Thirty-four percent (n=15) of the patients had gastric fundal varices. These were present initially (documented at endoscopy or at operation) or were developed subsequently. One third (n=5) of them had bled before. The proportion of Chinese patients with gastric fundal varices is significantly higher than that of the other races (p=0.031).

A total of 179 injection sclerotherapy episodes were performed. Each patient had an average of 4 injection sessions. The maximum number of injection sessions per person was 15. The complications included rebleeding (n=8), ulceration (n=5) and stricture (n=1). Of those who rebled, 3 had successful reinjection sclerotherapy, 2 had emergency operations, 2 developed encephalopathy and the remaining 1 aspirated. Only 1 of the 2 cases who had emergency operations survived, together with the 3 who had successful reinjection sclerotherapy (Tables II and III).

Table II
Complications of injection

Rebleeding	8	(4.5%)
Ulceration	5	(2.8%)
Stricture	1	(0.6%)

Table III
Outcome of rebleeding

Successful reinjection	3	
Emergency operation	2	(one died)
Encephalopathy	2	(both died)
Aspiration	1	(died)

Ulcerations secondary to injection sclerotherapy healed with time. The 1 case of stricture was the result of injection sclerotherapy followed by stapled oesophageal transection with devascularisation of the lower oesophagus. However, the patient was symptom-free after only 1 dilatation.

Table IV
Operations

Oesophageal transection	4
Shunt	1
Underunning of varix	2
Distal pancreatectomy	1
Splenectomy	4

Eleven patients were operated upon. Three of them were for problems not related to oesophageal varices. Table IV shows the types of operations performed on the remaining 8 patients. Stapled oesophageal transection was combined with devascularisation of the lower oesophagus and upper stomach. Subsequently, the patient had endoscopic surveillance and had reinjection sclerotherapy if varices were noted to have recurred. The shunt performed was a lienorenal shunt for portal vein thrombosis. Underunning of varix was performed for bleeding gastric fundal varix as an emergency procedure. Both patients remain well to this day. Splenectomy was performed as part of another operation.

There were a total of 9 deaths. Three were due to unrelated causes (i.e., 2 from hepatoma and 1 from carcinoma sigmoid). Four deaths were directly related to rebleeding. The other 2 died of aspiration and encephalopathy respectively.

Discussion

Gastroesophageal varices tend to bleed from the lower end of the oesophagus probably because of the unique venous anatomy of that area. The veins in the lower oesophagus (2 to 5 cm from the

oesophagogastric junction) were located in the lamina propria subepithelially. These veins increase in number and in the total area covered in cases of portal hypertension[2] making them more likely to bleed. Other factors like high oesophageal pressures in that area and gastric reflux have been implicated but the exact causes have not been proven[3].

The incidence of bleeding in cirrhotic patients has been reported to be 28.6% and almost all of those who bled did so within 2 years of the diagnosis[4]. Two-thirds survived the first bleed but less than 10% were still alive at the end of the 6 year study[4]. In patients with hepatic decompensation, the cause for their bleeding upper GIT was usually bleeding oesophageal varices. They also tended to bleed more severely and had a higher rate of mortality[5].

Gastric varices were noted to be not problematic in at least 1 study[6]. However in our series, one-third of our patients had gastric fundal varices and a third of these bled. Operative underunning of the bleeding point was required for 2 of these patients.

Oesophageal varices that did not bleed did not need treatment[4]. Prophylactic shunt surgery[7,8] had been abandoned because of associated high mortality and morbidity. Treatment of acute variceal bleeding entailed initial resuscitation, early endoscopic diagnosis, arrest of bleeding point and appropriate support for underlying liver insufficiency. Lowering of portal hypertension could be achieved by medical or operative means. Arrest of the bleeding source could be nonoperative, operative or a combination of these.

Nonoperative treatment[9] includes medical therapy, balloon tamponade, injection sclerotherapy, electrocautery, laser photocoagulation and percutaneous transhepatic obliteration of varices. Operative treatment could take the form of direct attack on the varices or a portasystemic shunting to reduce the portal pressure.

Direct attack on the bleeding varices is performed via oesophageal transection and devascularisation of oesophagogastric area. This was proposed by Sugiura and Futagawa[10] in 1973 and modified by others[11,12,13]. Good results with regards to haemostasis were encountered. Rebleeding ranged from none to 15% in the follow-up period of up to 13 years. Mortality ranged from 12% to 56%. Higher mortality was encountered in those undergoing emergency operation and/or those with severe liver disease (Child's C).

Portasystemic shunts had been established to be effective treatment for bleeding oesophageal varices[8]. Various shunts[14] had been performed, i.e., portacaval, lienorenal, distal splenorenal and mesocaval with its various modifications. Overall operative mortality ranged from 12% to 42%, shunt failure ranged from 5% to 50% and the incidence of encephalopathy was 34%[15,31]. A 5 year survival rate of 60% had been found in those who received a shunt procedure as compared to 10% in those who did not[17] but no significant difference was encountered in another study[18].

Rebleeding after portacaval shunt was 7% as compared to 65% in nonshunted patients[16]. Two-thirds of this rebleeding occurred within 12 months of the surgery. Emergency portacaval shunting[19,20] resulted in greater long-term survival compared to one performed electively. Major side-effects of portacaval shunt were hepatic encephalopathy and accelerated hepatic failure. To prevent these, hepatic arterialisation[21] or a selective distal splenorenal shunt[22,23] could be performed. Selective shunt was contraindicated in the presence of massive ascites. Child's criteria[1] staged the degree of hepatic dysfunction and was used in the selection of patients for surgery. Operative mortality was 6% for Child's A, 24% for B and 50% for C[24].

Endoscopic injection sclerotherapy was initially practised in 1936[25] but renewed interest only came about in 1973[26] when the results of 15 years of sclerotherapy were reviewed. The bleeding varices were controlled in 93% of admissions. The total admission mortality was 18% and mortality per injection was

under 12%. A randomised controlled study[6] comparing injection sclerotherapy with medical treatment and balloon tamponade revealed that the results of treatment of the former were unequivocally superior than the latter in terms of initial control of bleeding and in-hospital mortality. It has been found[30] that it takes an average of about 4 injection sessions to achieve obliteration of varices irrespective of whether the intervals between the injections were 1 week or 3 weeks. Mucosal ulcerations were encountered more frequently (80%) in those with weekly injections as compared to those (30%) on 3-weekly injections. However, this was not associated with a greater frequency of post-injection pain, dysphagia or long-term stricture formation. Bleeding after the start of the injection sessions occurred in about 40% of the patients. Most (70%) of these cases of bleeding occurred in the first 3 weeks. Furthermore, the differences encountered in the overall frequency of rebleeding in relation to size of the varices and the Child's stage of the patients were found not to be statistically significant. Terblanche et al[6] did not encounter recurrent bleeding from oesophageal varices in their patients once the varices had been eradicated.

Rigid oesophagoscopes were used in the past and general anaesthesia was required for the procedures. A recent study[27] using fibreoptic oesophagoscope and sedation for injection sclerotherapy achieved hemostasis in 91% of cases, with a complication rate of 16% and an overall mortality of 7%. Complications included chest infection (5.8%), post-injection bleeding (5.8%), oesophageal perforation (1.2%), oesophageal ulcer (2.2%) and others (1%). They were found to be higher in those who were in Child's C, alcoholic, had acute injection sclerotherapy and where rigid oesophagoscopes were used. Rigid oesophagoscope does not appear to confer any advantage over that done with fibreoptic oesophagoscope. However, the higher complication rate with its use may contribute to the overall mortality.

Post-injection bleeding occurred in 12% of cases after acute variceal bleeding, compared to only 2% of cases when performed more electively[27]. In our study, rebleeding was found in 4% of all our patients. These included post-injection bleeding as well as failure of injection sclerotherapy, i.e., bleeding after 24 hours of injection. We found that rebleeding after injection was associated with high mortality.

Preliminary studies with a somatostatin analogue (octreotide) had shown it to be effective in the acute treatment of bleeding oesophageal varices. Control of bleeding was achieved in more than 90% of cases[28,29].

Management of bleeding oesophageal varices involves a multi-disciplinary approach. Repeated endoscopic sclerotherapy is effective in achieving hemostasis with low morbidity and mortality. Oesophageal transection and devascularisation of oesophagogastric area may be used for those who bleed after injection sclerotherapy. Portasystemic shunting may be considered for those in Child's A in centres where this skill is available.

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