

Simplified Fibre Endoscopic Sclerotherapy for Oesophageal Varices

P.M. SMITH, MD, FRCP, *Consultant Physician*

D. BRIAN JONES, MB, MRCP, *Medical Registrar*

J.D.R. ROSE, MB, MRCP, *Senior Medical Registrar*

Department of Gastroenterology, Llandough Hospital, Penarth, South Glamorgan

Portacaval shunts for bleeding oesophageal varices may be followed by encephalopathy and neuropsychiatric syndromes[1], even in those with good liver function[2]. Furthermore, controlled trials suggest that survival is not prolonged by shunting[3,4].

Alternatives to shunting have therefore been sought. The Sengstaken tube and intravenous vasopressin produce only temporary arrest of variceal bleeding. Direct surgical attack on the varices, either by a transthoracic approach or through the abdomen using a stapling gun[5], may be followed by further bleeding[6]. Transhepatic catheterisation of the portal vein and embolisation of the collaterals supplying the oesophagus has been no more successful[7]. Other procedures, including the meso-caval and the distal (Warren) shunt, are not convincingly superior to the conventional end-to-side anastomosis.

In contrast, injection sclerotherapy has been shown to be effective, both in controlling acute variceal haemorrhage[8] and in prolonging survival[9,10]. We have treated bleeding varices by injection sclerotherapy through a fibrescope for the last three years, and present our results here.

Patients and Methods

Thirty-eight patients, aged 17–77 years, have been treated by injection sclerotherapy for oesophageal varices. There were 23 males and 15 females. Seventeen had alcoholic cirrhosis, eight primary biliary cirrhosis, five cryptogenic cirrhosis, two chronic active hepatitis, two congenital hepatic cirrhosis, one sarcoidosis, one secondary biliary cirrhosis, one portal vein thrombosis and one non-cirrhotic portal hypertension associated with vinyl chloride monomer exposure. The severity of their liver disease was graded according to a modification of Child's classification at the time of admission[6]. Acutely bleeding patients were dealt with by blood transfusion and initial passage of a Sengstaken tube, with sclerotherapy the next day when haemorrhage had ceased.

After spraying the throat with lignocaine, intravenous diazepam sufficient to produce dysarthria or ptosis pre-

ceded passage of an end-viewing endoscope (Olympus GIFD 3), with the patient in the left lateral position. No outer sheath was used[11]. Orientation was facilitated by passage of a thin plastic tube down the biopsy channel and a few drops of water were injected. The direction of fall of the drops was taken to be 6 o'clock, and the position of varices was noted on a clock-face system at 30, 35 and 40 cm. Photographs were taken for reference purposes. Using a fine injection needle (Olympus NM3), 2–3 ml of sodium tetradecyl sulphate was injected into each varix just above the gastro-oesophageal junction and the patient was returned to the ward. Sodium tetradecyl sulphate was preferred to ethanoleamine as it is slightly less viscous and preliminary warming facilitated injection; the degree of tissue necrosis produced by the two agents is similar[12]. If variceal bleeding was precipitated, a rare event, a Sengstaken tube was passed for four hours. Further injections were performed, initially weekly until the risk of bleeding had receded, but thereafter at four-weekly intervals on a day admission basis until all varices had disappeared.

Results

Thirty-eight patients received between two and 13 injections of sclerosant (Table 1). In 30, the varices disappeared after a mean of 6.2 treatments, large varices requiring more sclerotherapy than small ones. The remaining eight patients died before therapy could be completed, two from hepatic malignancy, five from hepatic failure and one only from oesophageal haemorrhage; four of these fatalities were initially assessed as Child's grade C, two B and two A. During treatment, four patients bled severely, and they underwent simplified oesophageal transection[5]. Two died, one three weeks later from a further massive variceal bleed and one from hepatic failure; another received a total of 10 sclerosant injections, four postoperatively, and his varices eventually disappeared. The fourth was found at operation to have a longitudinal ulcer involving the full thickness of the oesophageal wall, following an injection of sclerosant four days earlier; the necrotic tissue was excised and the varices ligated. Twelve other patients had smaller, easily controlled bleeds before therapy was completed.

Table 1. Outcome of treatment.

Patient No.	Age	Child	No. of treatments	Outcome	Complications	Follow-up	
						Duration (months)	Progress
1	64	C	4	death from hepatic failure	Oesophageal ulcer		
2	49	B	4	death from angiosarcoma			
3	37	A	5 and transection	death from bleeding varices			
4	77	B	7	death from hepatoma	Oesophageal ulcer		
5	63	C	5	death from hepatic failure			
6	61	A	4	varices obliterated	Oesophageal stricture	4	death from hepatoma
7	56	A	10	varices obliterated		22	1 rebleed
8	51	A	10	varices obliterated		22	1 rebleed
9	59	A	6	varices obliterated	Oesophageal ulcer	26	well
10	70	C	3	varices obliterated		13	death from septicaemia
11	32	A	10 and transection	varices obliterated	Oesophageal stricture	18	1 recurrent varix
12	63	B	4	varices obliterated		25	1 rebleed
13	65	A	4	varices obliterated	Oesophageal stricture	23	1 recurrent varix
14	53	B	4	varices obliterated	Oesophageal stricture	24	1 recurrent varix
15	59	C	8	varices obliterated	Oesophageal stricture	20	well
16	46	B	7	varices obliterated	Oesophageal stricture	20	1 recurrent varix
17	34	A	3 and transection	varices obliterated	Oesophageal ulcer	15	well
18	54	C	5	varices obliterated	Oesophageal ulcer	15	1 rebleed
19	49	A	3	varices obliterated	Oesophageal stricture	15	well
20	56	B	3	varices obliterated	Oesophageal ulcer	13	well
21	41	C	6	varices obliterated	Oesophageal stricture	12	1 rebleed
22	71	C	7	varices obliterated	Oesophageal ulcer	3	death from bronchopneumonia
23	69	B	2	varices obliterated	Oesophageal stricture	11	well
24	69	A	9	varices obliterated	Oesophageal ulcer	10	well
25	61	A	3	varices obliterated		9	well
26	35	A	13	varices obliterated		3	
27	60	A	9	varices obliterated	Oesophageal ulcer	7	
28	48	C	1	Death from hepatic failure			
29	52	A	5	Death from hepatic failure			
30	29	A	6	varices obliterated		2	
31	50	A	8	varices obliterated		3	
32	49	B	6			2	death from gastric bleeding
33	66	C	6 and transection	death from hepatic failure			
34	52	A	6	varices obliterated	Oesophageal stricture	4	
35	17	A	3	varices obliterated		1	
36	65	B	10	varices obliterated	Oesophageal stricture	1	
37	43	A	5	varices obliterated		1	
38	48	C	8	varices obliterated	Oesophageal stricture	1	

Follow-up of the 30 successfully treated patients has averaged 11 months (range 1—26 months). Nine have developed small oesophageal varices necessitating a further single injection, but only four of these have bled, in no case severely. One patient has died of a hepatoma, another of septicaemia associated with alcoholic cirrhosis, and one of broncho-pneumonia. A fourth suffered a fatal haematemesis from gastric varices only two months after the completion of sclerotherapy. Twelve patients developed an oesophageal stricture after 3 to 10 injections, coinciding with the disappearance of varices; another had a post-transection stricture. In four the stricture resolved spontaneously, but the remaining eight required one or two dilatations by the Eder-Puestow technique[13]. Nine

patients were noted at endoscopy to have a linear oesophageal ulcer at the site of a previous injection of sclerosant. These ulcers had healed by the next endoscopy four weeks later. Many patients complained of substernal pain for up to 48 hours after treatment. This was relieved by alkalis. There were no instances of mediastinitis or oesophageal perforation, but two patients developed pyrexias lasting up to five days after sclerotherapy.

Discussion

We have found that the injection of sclerosant into oesophageal varices, using an end-viewing fibroscope, is successful in obliterating varices. Only one of 38 patients

died of bleeding oesophageal varices, and none have developed mediastinitis or oesophageal perforation. The technique is simple and has allowed us to discard the portacaval shunt. Unlike the other methods described, we have been able to avoid the rigid oesophagoscope[8,14] and to dispense with general anaesthesia, and an outer sleeve[11]. The acutely bleeding varix is difficult to inject, and we prefer to rely on a Sengstaken tube for initial haemostasis. If further profuse bleeding renders sclerotherapy impracticable, oesophageal transection with a stapling gun[5] provides a period of several months during which sclerotherapy can be used: we have not found that transection alone leads to permanent cure.

Once the varices have been obliterated, do they recur? Our experience, which agrees with a recently reported series[15], suggests that they do; nine recurrences were observed in 18 patients followed-up for 9 months or more. Our practice is to re-endoscope patients at three-monthly intervals and to inject any new varices.

While every attempt is made to inject sclerosant directly into the varix, some almost certainly penetrates sub-mucosally, producing oesophageal ulceration. Healing fibrosis is presumably responsible for the high incidence of oesophageal strictures, which have fortunately proved easy to manage. Visual fluoroscopic control has shown that sclerosant injected intravenously rapidly escapes from the sub-mucosal varices to the peri-oesophageal veins[16]. Variceal obliteration has resulted despite the fact that we have not used an over-tube[11] or a rigid endoscope to compress the varices. Short-term compression may have little advantage, as endothelial damage after injection of a detergent sclerosant such as sodium tetradecyl sulphate can follow after only one second's contact, adherent thrombus developing during the next 12 hours[17]. The detergent can also be diluted without significant loss of its potency in reducing surface tension[18].

Since the direction of the blood flow in the varix is upwards, we have found that treatment confined to the lower 5 cm of the gullet is sufficient to eliminate oesophageal bleeding. Only one patient has bled from gastric varices, but a patient in a preliminary study underwent sclerosis of a gastric varix, which resulted in necrosis of the adjacent stomach wall. The problem of varices devel-

oping in the stomach after successful oesophageal sclerotherapy remains to be solved.

Other workers have suggested that sclerotherapy will control bleeding, allowing an elective portacaval shunt to be performed later[8,14]. We believe that the simple procedure of sclerotherapy will ablate varices, removing the need for a subsequent shunt and avoiding the risk of encephalopathy.

Acknowledgement

We would like to thank Mr M. H. Wheeler for performing the oesophageal transections.

References

1. Read, A. E., Sherlock, S., Laidlaw, J. and Walker, J. G. (1967) *Quarterly Journal of Medicine*, **36**, 135.
2. Voorhees, A. B. Jr., Chaitman, E., Schneider, S., Nicholson, J. R., Kornfeld, D. S. and Price, J. B. Jr. (1973) *Archives of Surgery*, **107**, 659.
3. Conn, H. O. (1974) *Gastroenterology*, **67**, 1065.
4. Rueff, B., Prandi, D., Degos, F., Sicot, J., Degos, J. D., Sicot, C., Maillaird, J. N., Vauvert, R. and Benhamou, J. P. (1976) *Lancet*, **1**, 655.
5. Johnston, G. W. (1977) *Annals of the Royal College of Surgeons of England*, **59**, 404.
6. Pugh, R. N. H., Murray-Lyon, I. M., Dawson, J. L., Pietroni, M. C. and Williams, R. (1973) *British Journal of Surgery*, **60**, 646.
7. Smith-Laing, G., Scott, J., Long, R. G., Dick, R. and Sherlock, S. (1981) *Gastroenterology*, **80**, 1031.
8. Johnston, G. W. and Rodgers, H. W. (1973) *British Journal of Surgery*, **60**, 797.
9. Terblanche, J., Northover, J. M. A., Bornman, P., Kahn, D., Silber, W., Barbezat, G. O., Sellars, S., Campbell, J. A. H. and Saunders, S. J. (1979) *Surgery, Gynecology and Obstetrics*, **148**, 323.
10. MacDougall, B. R. D., Westaby, D. and Williams, R. (1981) *Gut*, **22**, 886.
11. Williams, K. G. D. and Dawson, J. L. (1979) *British Medical Journal*, **2**, 766.
12. Blenkinsopp, W. K. (1968) *British Journal of Experimental Pathology*, **49**, 197.
13. Price, J. D., Stanciu, C. and Bennett, J. R. (1974) *Lancet*, **1**, 1141.
14. Paquet, K. J. and Oberhammer, E. (1978) *Endoscopy*, **10**, 7.
15. Clark, A. W., MacDougall, B. R. D., Westaby, D., Mitchell, K. J., Silk, D. B. A., Strunin, L., Dawson, J. L. and Williams, R. (1980) *Lancet*, **2**, 552.
16. Barsoum, M. S., Khatter, N. Y. and Risk-Allah, M. A. (1978) *British Journal of Surgery*, **65**, 588.
17. Schneider, W. and Fischer, H. (1974) *Phlebologie*, **27**, 411.
18. Imhoff, E. and Stenmer, R. (1969) *Phlebologie*, **22**, 143.