

Severe bleeding following endoscopic variceal ligation : should EVL be avoided in Child C patients ?

H. Van Vlierberghe, M. De Vos, M. Hautekeete (†), A. Elewaut

Department of Gastroenterology and Hepatology, University Hospital, Gent.

Abstract

In the last decade there has been an evolution in the treatment of bleeding oesophageal varices. Endoscopic variceal ligation (EVL) is one of those new techniques that not only has shown to be more effective than sclerotherapy, but also causes less side effects, resulting in less episodes of rebleeding and improving survival.

We describe severe bleeding in 3 patients after EVL, occurring between 5 and 10 days after the initial ligation. Two Child C patients could not be resuscitated and died shortly after this event. Severely impaired clotting function as a result of the liver disease and the greater size of the ulcers induced by EVL may contribute to this dramatic complication.

Severe bleeding due to postligation ulceration may lead to death, which occurred in 2 of our Child C patients. Since more and more endoscopists are using EVL in the treatment of oesophageal variceal bleeding, they should be aware of the possible complications caused by this rather new technique. (*Acta gastroenterol. belg.*, 1999, 62, 175-177).

Key words : liver cirrhosis, portal hypertension, varices, ligation, complications.

Introduction

Endoscopic variceal ligation (EVL) is one of the treatment modalities used in the management of bleeding oesophageal varices. Since its introduction by Stiegmann *et al.* (1), several trials have been published, not only comparing its efficacy with endoscopic variceal sclerotherapy (EVS) but also comparing the combination of both techniques (2-8). Actually, EVL has become the treatment of choice in many endoscopy units.

In addition to fewer complications, ligation is also significantly superior to sclerotherapy in terms of the need for fewer treatment sessions to eradicate varices (5, 6), has a lower rebleeding rate (5), and improves survival (8). Since the introduction of multiband devices (Speed band R, Six Shooter R), serious complications due to the use of an overtube have completely disappeared. So most physicians treating patients with bleeding oesophageal varices are using EVL.

However several case reports describing serious complications of the EVL technique, not related to the use of an overtube (9, 10), have been published.

We report on the history of a major complication occurring in three patients out of a series of 14 patients treated with EVL.

Patients and methods

Patients

Fourteen patients with liver cirrhosis and portal hypertension were treated with EVL for bleeding oesophageal varices between October 1995 and August 1996. The patient characteristics are presented in table 1.

Table 1. — Patient characteristics

male/female	9/5
age	median : 64 range : 37 - 85
Child A/B/C	6/5/3
Child-Pugh points	median : 7 range : 5 - 13
PTT %	median : 60 range : 12 - 96
Albumin (mg/dl)	median : 3.4 range : 2.5 - 4.1
Bilirubin (mg/dl)	median : 1.6 range : 0.6 - 10
Platelets	133 ± 71
Fibrinogen (mg/dl)	median : 367 range : 149 - 782
Origin of liver disease	Alcohol : 11 Viral : 1 Primary biliary cirrhosis : 1 Cryptogenic ; 1

Methods

Three patients were ligated with the Speed Band device R (Microvasive), the other patients were treated with the Six Shooter R (Wilson Cook). All patients presented with hematemesis and/or melena. After hemodynamic stabilisation, a diagnostic upper GI endoscopy confirmed the presence of acute bleeding varices or showed stigmata of recent variceal bleeding (clot, nipple) and ruled out other causes of GI bleeding. When the variceal cause of bleeding was confirmed, the patients received Somatostatin (UCB) intravenously

Correspondence : H. Van Vlierberghe, M.D., Department of Gastroenterology and Hepatology, Universitair Ziekenhuis, 1K12 IE, De Pintelaan 185, 9000 Gent, Belgium.

(250 µg as a bolus and thereafter at a continuous dosis of 6 mg/24 hours) and EVL was performed. The varices were ligated by two physicians (HVV, MDV) and this at a level of 1-5 cm above the gastro-oesophageal junction. A maximum amount of 6 bands was put. At the end of the procedure, no active bleeding was seen. Somatostatin was continued for 5 days.

Case report

Eleven patients had no serious events apart from non-complicated ulcerations at the place of the ligation. These ulcerations did not cause bleeding; when seen on the control endoscopy, which was performed 5 days after EVL, omeprazole 20 mg o.i.d. was started.

A major complication occurred in 3 patients; their history is reported here.

A 67 year old man with Child A alcoholic liver cirrhosis had a massive episode of haematemesis with hemodynamic instability, seven days after ligation with 4 bands. The control endoscopy on day 5 showed 4 large ulcerations, one of them had an adherent clot. At day 7, an upper GI endoscopy showed large bleeding ulcerations at the place of the ligations. Local treatment could not stop the bleeding. At that moment the PTT was 56% and the platelet count was 100,00/µl.

A Linton tube was placed and a TIPSS procedure was performed. The bleeding stopped and there was no rebleeding during a 6-months follow up period.

A 65 year old woman with hepatitis C positive liver cirrhosis was admitted with a variceal bleeding. The oesophageal varices were ligated with 5 bands. However she developed a Streptococcus pneumonia complicated with sepsis and liver failure (Child C with a PTT of 12% and a platelet count of 201,000/µl). A control endoscopy was not performed. Ten days after the ligation, she became hemodynamically unstable and presented with recurrent upper GI bleeding. Endoscopy showed large oozing ulcers at the place of the ligation. Local therapy with polidocanol could not stop the bleeding. A Linton tube was placed. The bleeding however continued and after 16 hours the patient died.

A 44 year old male patient with Child C (PTT 25% and platelet count of 44,000/µl) alcoholic liver cirrhosis presented with upper GI bleeding. Five days after the successful initial ligation with 5 bands, haematemesis occurred again. During introduction of the endoscope, he vomited massive amounts of blood and despite intensive resuscitation attempts, he deceased 45 minutes after starting the endoscopy.

Discussion

Over the past 15 years EVS has been the modality of choice to manage acute bleeding of oesophageal varices. This may be ascribed to the high success rates achieved with EVS during acute bleeding (11) and to the de-

creased rates of rebleeding after repeated sessions of EVS (12,13). However the relatively high complication rates associated with EVS (14), has resulted in the development of new techniques such as EVL.

Several studies (3-8) demonstrated that EVL is not only more effective in eradicating oesophageal varices than EVS, but is also associated with a lower rate of complications (3-8).

Especially when comparing EVL with EVS, patients treated with EVL had lower rebleeding rates than those treated with EVS (3-6). Some studies could demonstrate an increase in survival (8).

Initially an overtube was needed to reload the endoscope after each single band that had been fired. Complications of EVL were almost completely attributed to use of the overtube (9). Since the introduction of devices that can fire 5 to 6 bands at one time (Speed Band R and Six Shooter R), the use of the overtube became obsolete and the associated complications disappeared.

EVL is easy to perform and becomes more popular and widely used. Therefore, reports about serious complications are essential.

We report on the results and a major side effect in our first fourteen patients treated with EVL. Three major bleeding episodes occurred between 5 and 10 days after the initial ligation session. One patient with Child A liver cirrhosis could be resuscitated with the use of a Linton tube and the placement of a TIPSS. The other two patients, both with Child C liver cirrhosis died despite intensive resuscitation.

Both had marginal liver function and their coagulation was severely impaired (PTT level in these two patients was below the median value of our total group, cfr table 1). In two of the three patients the site of the bleeding could be identified as a postligation ulcer. Due to the dramatic and sudden event in our third patient the exact cause of the bleeding could not be demonstrated. Since it is our personal impression that EVL bands can loosen as quick as 24-48 hours after placement, the time interval between the ligation and the complication is suggestive for a postligation ulcer. A study comparing the appearance of oesophageal ulcers associated with EVL and EVS showed that ulcers associated with EVS were deeper, whereas ligation induced ulcers had a surface area six times greater than EVS induced ulcers (15).

In Child C patients with severe disturbances in clotting, these large ulcerations are perhaps more at risk for prolonged severe bleeding than smaller EVS induced ulcers. In our patient with Child A cirrhosis the bleeding could be stopped, suggesting that hemostasis, liver function and lowering of the portal pressure due to TIPSS insertion, was sufficient to prevent further bleeding and death in this patient.

A recent report of Sakai *et al.* (10) confirmed the association between Child C status and the development of severe bleeding episodes: in their first 30 patients treated with EVL, early massive rebleeding occurred

only in 4 Child C patients (with two patients dying from this complication). Impaired clotting contributed to these lethal complications. The authors suggested the use of cyanoacrylate glue as an alternative in these patients.

A possible criticism of this and our study is that the severe complications are due to a learning curve effect. The EVL technique seems to be a quite simple technique where the degree of ulceration only depends upon the volume of tissue that can be suctioned to fill the chamber of the ligation device (16). However people are inclined to place the first band under or at the gastro-oesophageal junction. Varices seem more turgid and easily accessible in this region. Moreover the gastro-oesophageal junction is sometimes difficult to locate probably because of the short field of view due to the cap supporting the bands.

In this region (on and under the gastro-oesophageal junction), the risk of developing bleeding due to postligation ulcerations is higher due to a thinner lining.

Our unit has, on the other hand, a long time experience in the use of EVS. EVS is a technique far more difficult to perform with complications not only dependent upon the volume of sclerosing solution, size of injection etc., but also dependent upon the reaction of the patient (gagging, coughing...) (16).

Child C patients are those with the highest mortality after an index bleeding; this is seen whatever the technique used. They have a higher rate of rebleeding, the most often these patients die from liver failure.

Ideally the technique for stopping the index bleeding should be one free of immediate life threatening complications.

Although several trials have shown a lower rebleeding rate in patients treated with EVL compared to EVS, even in Child C patients, physicians should be aware of possible life threatening complications of EVL in Child C patients.

In conclusion, we have described a major complication in 3 patients of our 14 patients treated with EVL. The patient who belonged to the Child A classification could be resuscitated; the two Child C patients had a fatal outcome. Severely impaired clotting factors could have contributed to the inability to stop the bleeding.

Therefore in patients with Child C liver cirrhosis and especially in those with low levels of coagulation factors, EVL should be used with caution.

References

1. STIEGMANN G.V., CAMBRE T., SUN J.H. A new endoscopic elastic banding device. *Gastrointest Endosc.*, 1986, **32** : 230-233.
2. SAEED Z.A., STIEGMANN G.V., RAMIREZ F.C., REVEILLE R.M., GOFF J.S., HEPPS K.S., COLE R.A. Endoscopic variceal ligation is superior to combined ligation and sclerotherapy for esophageal varices : a multicenter prospective randomized trial. *Hepatology*, 1997, **25** : 71-74.
3. GIN-HO L., KWOK-HUNG L., JING-SHIUNG C., JIA-HUEY H., CHIA-FU C., SAM-MING C., HUNG-TING C. A prospective randomized trial of sclerotherapy versus ligation in the management of bleeding esophageal varices. *Hepatology*, 1995, **22** : 466-471.
4. MING-CHIH H., HAN-CHIEN L., BENJAMIN ING-TIAU K., CHEN-HSIANG C., FA-YAUH L., SHOU-DONG L. Comparison of endoscopic variceal sclerotherapy and ligation for the treatment of esophageal variceal hemorrhage : a prospective randomized trial. *Hepatology*, 1995, **21** : 1517-1522.
5. GIMSON A.E.S., RAMAGE J.K., PANOS M.Z., HAYLLAR K., HARRISON P.M., WILLIAMS R., WESTABY D. Randomised trial of variceal banding ligation versus injection sclerotherapy for bleeding oesophageal varices. *Lancet*, 1993, **342** : 391-394.
6. LAINE L., HUSSEIN M., EL-NEWIHI, MIGOWSKY B., SLOANE R., GARCIA F. Endoscopic ligation compared with sclerotherapy for the treatment of bleeding esophageal varices. *Ann. Intern Med.*, 1993, **117** : 1-7.
7. BARONCINI D., MILANDRI G.L., BORIONI D., PIERMONTESI A., CENNAMO V., BILLI P., DAL MONTE P.P., D'IMPERIO N. A prospective randomized trial of sclerotherapy versus ligation in the elective treatment of bleeding esophageal varices. *Endoscopy*, 1997, **29** : 235-240.
8. STIEGMANN G.V., GOFF J.S., MICHALETZ-ONODY P.A., KORULA J., LIEBERMAN D., SAEED Z.A., REVEILLE R.M., SUN J.H., LOWENSTEIN S.R. Endoscopic sclerotherapy as compared with endoscopic ligation for bleeding esophageal varices. *N. Eng. J. Med.*, 1992, **326** : 1527-1532.
9. JOHNSON P.A., CAMPBELL D.R., ANTONSON C.W., WESTON A.P., SHULER F.N., LOZOFF R.D. Complications associated with endoscopic band ligation of esophageal varices. *Gastrointest. Endosc.*, 1993, **39** : 181-185.
10. SAKAI P., MALUF FILHO F., MELO J.M., ISHIOKA S. Is endoscopic band ligation of esophageal varices contraindicated in Child-Pugh C patients. *Endoscopy*, 1994, **26** : 511-512.
11. WESTABY D., HAYES P.C., GIMSON A.E., POLSON R.J., WILLIAMS R. Controlled clinical trial of injection sclerotherapy for active variceal bleeding. *Hepatology*, 1989, **9** : 274-277.
12. KORULA J., BALART L.A., RADVAN G., ZWEIBAN B.E., LARSON A.W., KAO H.W., YAMADA S. A prospective randomized controlled trial of chronic esophageal variceal sclerotherapy. *Hepatology*, 1985, **5** : 584-589.
13. WESTABY D., MACDOUGALL B.R., WILLIAMS R. Improved survival following injection sclerotherapy for esophageal varices : final analysis of a controlled trial. *Hepatology*, 1985, **5** : 827-830.
14. SCHUMAN B.M., BECKMAN J.W., TEDESCO F.J., GRIFFIN J.W., ASSAD R.T. Complications of endoscopic injection sclerotherapy. *Am. J. Gastroenterol.*, 1987, **82** : 823-830.
15. YOUNG M.F., SANOWSKI R.A., RASCHKE R. Comparison and characterization of ulcerations induced by endoscopic ligation of esophageal varices versus endoscopic sclerotherapy. *Gastrointest Endosc.*, 1993, **98** : 119-122.
16. SAEED Z. A. Endoscopic therapy of bleeding esophageal varices : ligation is still the best. *Gastroenterology*, 1996, **110** : 635-638.