

# Different Factors Correlated to Early Rebleeding in Cirrhotic Patients Treated by Variceal Band ligation versus Endoscopic Sclerotherapy

Kamal Amer, Amal Jouda, Sameh M. Abdel Monem

Tropical Medicine Department, Faculty of Medicine, Zagazig University, Egypt

**Corresponding Author**  
Kamal Amer

**Mobile:**  
+201226204876

**E mail:**  
dr\_kamalamer@yahoo.com

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**Background and study aim :** Endoscopic treatment has become the principal first-line intervention in patients with bleeding oesophageal varices, both during the acute event and for long-term therapy to prevent recurrent bleeding. Several clinical considerations affect the prognosis in individual patients including the severity portal hypertension, the location of the bleeding varices, residual hepatic function, the presence of associated systemic disease, and others. Early rebleeding has been shown to be a strong predictor of mortality and recurrent variceal bleeding substantially increases the risk of complications which further contribute to mortality. This study aimed to evaluate early rebleeding after different methods of endoscopic intervention and investigate the different parameters of the patient that can be correlated to it.

**Patients and methods:** Hundred and four cirrhotic patients with first attack of variceal bleeding were included in this study. They were randomly allocated to two groups, group I: 52 patients who were managed by endoscopic variceal sclerotherapy and group II: 52 patients who were managed by endoscopic variceal band ligation to control their attack. The patients were followed up for six weeks and all their clinical, laboratory, endoscopic parameters were monitored. The rate of mortality and

early rebleeding was measured and correlated to these different patients' parameters

**Results:** There was no significant difference between the two groups as regards rate of early rebleeding (15.4% in group I vs 9.6% in group II  $P=0.374$ ). The rate of early rebleeding was significantly correlated to Child's score ( $r=+0.136$   $P=0.014$ ), PT ( $r=+0.35$   $P<0.001$ ), INR ( $r=+0.419$   $P<0.001$ ), grade of OV ( $r=+0.233$   $P=0.001$ ), risky signs ( $r=+0.179$   $P=0.001$ ), units of blood received ( $r=+0.387$   $P<0.001$ ), amount of ethanolamine oleate ( $r=+0.329$   $P=0.017$ ) and number of rubber bands used ( $r=+0.245$   $P=0.039$ ). Mortality rates showed also no significant difference during the six weeks of follow up (19.2% in group I vs 21% in group II  $P=0.647$ ), as well as mortality rates in rebleeding cases (37.5% in group I vs 40% in group II  $P=0.925$ ).

**Conclusion:** The factors that are strongly correlated to rate of early rebleeding after endoscopic management of OV are severely decompensated liver disease, larger OV size and presence of risky signs, use of more blood units during resuscitation, use of large amount of ethanolamine oleate during sclerotherapy and use of more rubber band during banding. Sclerotherapy and band ligation are comparable to each other in most outcomes especially early rebleeding and mortality.

## INTRODUCTION

Portal hypertension commonly complicates liver cirrhosis and the development of oesophageal varices is one of the major complications of portal hypertension [1]. The prevalence of oesophageal varices at diagnosis ranges from 0-10% in patients with compensated cirrhosis, to 60% to 80% in patients with decompensated cirrhosis and the reported mortality from variceal

bleeding ranges from 17% to 57% [2]. The progression from small to large varices occurs in 10% to 20% of cases annually [3].

Endoscopic treatment has become the principal first-line intervention in patients with bleeding oesophageal varices, both during the acute event and for long-term therapy to prevent recurrent bleeding [4].

After control of the index bleed, there is a 40% chance of rebleeding with a similar mortality. The risk of rebleeding is greatest during the first few days after initial variceal hemorrhage [5]. Survival after variceal bleeding depends largely on the rapidity and efficacy of initial primary hemostasis and the presence and severity of underlying liver disease and hepatic functional reserve [6].

Early rebleeding has been shown to be a strong predictor of mortality and recurrent variceal bleeding substantially increases the risk of complications which further contribute to mortality [6]. Rapid and sustained control of variceal bleeding remains the principal imperative of endoscopic intervention [7]. Several important clinical considerations influence the prognosis in individual patients. These include the natural history of the disease causing the portal hypertension, the location of the bleeding varices, residual hepatic function, the presence of associated systemic disease, continuing drug or alcohol abuse, patency of major splanchnic veins and the response to each specific treatment [8].

Until now, there has been no general consensus on the risk factors and measures to prevent early rebleeding in cirrhotic patients in Egypt. Variceal Band ligation and Endoscopic Sclerotherapy can be effective methods to manage variceal bleeding and may be prevent it primarily and secondarily. However, early recurrent bleeding as a vital complication after variceal band ligation and endoscopic sclerotherapy has not been studied fully.

#### **Aim of the work:**

The aim of the present study was to evaluate the different factors that can affect the rate of early rebleeding of early rebleeding after different endoscopic treatments of variceal bleeding which help better management of variceal bleeding.

## **PATIENTS AND METHODS**

This prospective randomized study was conducted in the Intensive Care Unit (ICU), Inpatient and Endoscopy Units of Tropical Medicine Department, Faculty of Medicine Zagazig University, during the period from October 2012 to October 2014. It included 104 patients with first attack of hematemesis and melena diagnosed as bleeding esophageal varices by upper

endoscopy. The Sample size was calculated using Epi info version 6.04.

They were divided into 2 groups (age, sex and severity of liver disease matched):

- **Group I:** included 52 patients who were treated by endoscopic sclerotherapy.
- **Group II:** included 52 patients were treated by endoscopic band ligation.

#### **Inclusion criteria:**

- 1- Presence of liver cirrhosis, the diagnosis of cirrhosis was based on clinical, biochemical and ultrasonographic findings with Child-Pugh grading.
- 2- First attack of upper GIT bleeding, which was proven by upper GIT endoscopy as bleeding esophageal varices.

#### **Exclusion criteria:**

- 1- Patients <18 and >60 years old
- 2- Patients who refuse participation in this study.
- 3- Hepatic patients with other causes of upper GIT bleeding than esophageal varices.
- 4- Patients with bleeding gastric varices.
- 5- Patients with recurrent attacks of bleeding oesophageal varices.
- 6- Patients with intra or extrahepatic malignancy.
- 7- Patients who had uncontrolled bleeding for 24 h after endoscopic treatment.

#### **All patients were subjected to the following:**

1. Thorough medical history taking including:
2. Thorough clinical examination including:
3. The following laboratory investigations:
  - Complete blood picture (haemoglobin level, red blood cell count, white blood cell count and platelet count)
  - Biochemical liver tests on including: Total and direct serum bilirubin in mg/dl, Total serum protein and serum albumin in gm/dl, Serum Aspartate amino Transferase (AST) and serum Alanine amino Transferase (ALT) (IU/L), Prothrombin time in seconds and international randomization ratio (INR).
  - Kidney function tests including blood urea and serum creatinine.
  - Serum Bilharzial antigen: using ELISA/ soluble egg antigen (SEA)
  - AST platelet ratio index (APRI): APRI = (AST/upper limit normal) x 100/platelet count. Score <0.5 excludes fibrosis. Score >2 suggests fibrosis.[9]

- **Abdominal Ultrasonography :** All the patients were examined using esaotemylab device. They were examined according to the standard maneuvers. Color Doppler ultrasound: All measurements were done by a single radiologist using color Doppler sonography with subjects in the supine or left lateral position. A Power Vision SSA-380A system (Esaotemy lab device) with (3 to 5 MHz) convex and sector pulsed probes. Sonographic examinations were carried out 8 hours after the last meal. In

our study, we measured two parameters by Doppler ultrasound:

- Portal vein velocity (cm/sec) PVV was measured directly using color Doppler ultrasound.
  - Hepatic artery resistive index (RI) = (peak systolic velocity - end diastolic velocity) / peak systolic velocity [10].
4. Child-Pugh classification for all patients into: A,B, and C class according the severity of cirrhosis [11] :

Measure	1 point	2 points	3 points
Total bilirubin, (mg/dl)	<2	2-3	>3
Serum albumin, g/dl	>3.5	2.8-3.5	<2.8
Pt (seconds prolonged)	0-4	4-6	>6
Ascites	None	Mild	Moderate to Severe
Hepatic encephalopathy	None	Grade I-II (or suppressed with medication)	Grade III-IV (or refractory)

Points	Class
5-6	A
7-9	B
10-15	C

## 5. Upper gastrointestinal endoscopy :

### Before endoscopy:

- Patients admitted to the Intensive Care Unit (ICU) for the first attack of variceal bleeding. Initial resuscitation following the classic Airway, Breathing, Circulation scheme was done followed by nasogastric lavage to remove particulate matter, fresh blood, and clots from the stomach to facilitate endoscopy.
- The patients were given the following medications along with volume replacement with plasma expanders: vitamin K 10mg/day IM, pantoprazole 40 mg/12 h IV, Somatostatin analogue (sandostatin ) initial bolus 500 µg iv followed by 250 µg/hour for 24 hours and prophylactic antibiotic (cefotaxime sodium 1 gm IV /12h)

**At time of endoscopy:** Endoscopy was done by a single experienced endoscopist using end flexible videoendoscope (PENTAX VIDEO unit of endoscopy). The patients were positioned on their left lateral position, with head supported on a small firm pillow to remain in a comfortable neutral position and a bite guard in their mouth. Medazolam I.V. was used as sedation. Patients

meeting the inclusion criteria were randomized alternatively to undergo Endoscopic Injection Sclerotherapy (EIS) or Endoscopic variceal Band Ligation (EBL).

- EIS was performed using a 25-gauge disposable injection needle for intravariceal and paravariceal injection. The sclerosant used was ethanolamineolyte.
- EVL was performed with PENTAX EG endoscope by the same experienced endoscopist using endoscopic ligating devices: an over tube or multi-band ligators.

### Esophageal varices were graded into 4 grades as follows: [12]

- **Grade I:** small straight cords of varices continued to lower 1/3of the esophagus.
- **Grade II:** moderate sized clubbed varices with well-defined areas of normal mucosa between them, forming several distinct vertical cords and confined to lower third of esophagus.
- **Grade III:** gross varices extending into the proximal half of the esophagus, which is so large and tortuous, that normal mucosa may

not be visible in between unless the esophagus is fully distended with air.

- **Grade IV:** varices are like those of grade III but with dilated capillaries on top or in between varices, (varix over varix).

**Portal hypertensive gastropathy was classified as follows: [13]**

- **PHG grade I:** mild reddening and congestive mucosa, no mosaic like pattern.
- **PHG grade II:** Severe redness and a fine reticular pattern separating the areas of raised edematous mucosa (mosaic like pattern) or fine speckling.
- **PHG grade III:** Point bleeding + grade II.

#### **After endoscopy:**

The patients used non selective beta blocker carvedilol for prevention of recurrent variceal bleeding, starting with 12.5 mg orally single daily dose as recommended by Banares et al. [14].

The patients were evaluated according to the presence or absence of the following symptoms: epigastric pain, heart burn, retrosternal chest pain, dysphagia, dyspepsia, and odynophagia upon discharge and during the follow up visits every two weeks.

#### **Follow up:**

The follow up of the patients was done every 2 weeks for 6 weeks as regards the following:

- 1- The patients' general condition like development or improvement of ascites, lower limb edema, jaundice, and hepatic encephalopathy (HE).
- 2- Development of infections e.g diarrhea, chest infection, and abdominal pain and tenderness as indicators for SBP.
- 3- Laboratory tests; CBC, total and direct bilirubin, serum albumen and serum creatinine.
- 4- Upper GIT endoscopy with commenting on the variceal condition as previous, PHG, bleeding and development of sclerosant or post banding ulcer.
- 5- Rebleeding.

#### **Statistical analysis:**

Data were checked, entered and analyzed using SPSS version 19 EPI-INFO 6 and for data processing and statistic. Numerical data were expressed as mean and standard deviation and the comparison between numerical data is done with simple t test for normally distributed data and with Mann Whitney U test when data

distribution is skewed. We used number and percentage to express categorical data and chi-square test to compare them. The correlation between numerical data was done by Spearman's correlation coefficient. The correlation between numerical and categorical data used Spearman's rank correlation.

## **RESULTS**

Comparison between the two studied groups as regards age, gender distribution, incidence of diabetes, hypertension and bilharziasis revealed no significant differences as shown in table (1). Table (1) shows also that there were no significant differences as regards the cause of cirrhosis and the previous use of primary prophylaxis.

Table (2) shows that there were no significant differences between the studied groups as regards the liver and spleen size as detected by sonography. There were also no significant differences between the two studied groups as regards portal vein diameter and velocity as well as hepatic artery resistive index measured by coloured doppler, as shown in table (2). Table (2) also shows that there were no significant differences between the two groups as regards all laboratory parameters.

Comparison between the studied groups as regards the endoscopic examination revealed no significant differences between the two groups as regards grade of OV, number of cords, grade of PHG and incidence of duodenopathy at the beginning of the study as shown in table (3).

Table (4) compares the studied groups as regards the incidences of the common post-endoscopy symptoms encountered by the patients and shows that there were no significant differences as regards any of these symptoms.

Table (5) compares the studied groups as regards rate and causes of rebleeding and mortality rate and shows that there were no significant differences between them. Correlation between the rate of rebleeding and study parameters revealed that the rate of rebleeding has significant positive correlation with Child's score, PT, INR, grade of OV, presence of risky signs, number of units of blood transfused during resuscitation, amount of sclerosing agent and number of rubber bands used as shown in table (6).

**Table (1):** Demographic data, cause of cirrhosis and comorbidity

		Group I No=52	Group II No=52	Test value	P	Sig.
Age		50.1±12.1	49.7±10.6	t= 0.15	0.877	NS
Gender	Male	36(69.2%)	33(63.5%)	<b>X<sup>2</sup>=0.388</b>	0.534	NS
	Female	16(30.8%)	19(36.5%)			
Diabetes		7(13.5%)	8(15.4%)	0.078	0.78	NS
Hypertension		4(7.7%)	6(11.5%)	0.443	0.506	NS
Cause of cirrhosis	HBV	5(9.6%)	6(11.5%)	0.102	0.750	NS
	HCV	45(86.5%)	45(86.5%)	0.000	1.000	NS
	others	2(3.8%)	1(1.9%)	0.343	0.558	NS
Positive bilharzial Ag		7(13.5%)	10(19.2%)	0.633	0.426	NS
Primary prophylaxis		9(17.9%)	10(19.2%)	0.064	0.800	NS

**Table (2):** Baseline sonographic, Doppler data and Child's score and laboratory data

		Group I No=52	Group II No=52	Test value	P	Sig.
Liver size	enlarged	2(3.8%)	2(3.8%)	0.877#	0.645	NS
	Average	14(26.9%)	10(19.2%)			
	shrunk	36(69.2%)	40(67.9%)			
Spleen size	average	3(5.8%)	5(9.6%)	0.542#	0.462	NS
	enlarged	49(94.2%)	47(90.4%)			
Portal vein diameter (cm) Mean ± SD		1.58 ± 0.21	1.53 ± 0.21	1.188•	0.238	NS
Portal vv velocity (cm/sec) Mean ± SD		13.27 ± 3.84	13.24 ± 4.11	0.299*	0.765	NS
Hepatic aa resistive index Mean ± SD		0.78 ± 0.07	0.77 ± 0.06	0.104*	0.917	NS
Child's grade	A	0.77 ± 0.06	10(19.2%)	<b>X<sup>2</sup>= 2.049</b>	0.359	NS
	B	9(17.3%)	15(28.8%)			
	C	30(57.7%)	27(51.9%)			
Hemoglobin (g/dl)		8.87 ± 1.53	9.05 ± 1.52	0.576•	0.566	NS
WBC's (cellx10 <sup>3</sup> /ml)		6.20 ± 3.86	6.30 ± 3.86	0.137*	0.891	NS
Platelet(x10 <sup>3</sup> )/ml		85.09 ± 33.87	92.34 ± 44.34	0.582*	0.560	NS
Albumin (g/dl)		2.54 ± 0.67	2.48 ± 0.66	0.368*	0.713	NS
Bilirubin (mg/dl)		2.34 ± 1.45	2.19 ± 1.51	0.973*	0.330	NS
GOT (IU/ml).		58.94 ± 33.13	61.17 ± 35.29	0.228*	0.820	NS
GPT (IU/ml)		50.03 ± 31.51	52.63 ± 38.46	0.085*	0.933	NS
PT (sec)		16.83 ± 3.42	16.72 ± 3.42	0.137*	0.891	NS
INR		1.49 ± 0.35	1.46 ± 0.32	0.251*	0.802	NS
APRI score		1.89 ± 1.08	1.83 ± 1.09	0.319*	0.750	NS

# Chi-square •independent t test \*Mann-Whitney U test, NS non significant

**Table (3):** Endoscopic findings in both groups at the beginning of the study and after two weeks

		Group I No.=52		Group II No.=52		X2	P	Sig.
		No.	%	No.	%			
Risky signs	Absent	11	21.2	8	15.4	0.58	0.446	NS
	Present	41	78.8	44	84.6			
No. of Oesophageal varices cords	2	18	34.6	17	32.75	0.57	0.449	NS
	3	27	51.9	27	51.9	0.73	0.394	NS
	4	7	13.4	8	15.4	0.06	0.811	NS
Oesophageal varices(OV) grade	OV I	0	0	0	0	0.04	0.847	NS
	OV II	16	30.7	15	28.8			
	OV III	24	46.2	26	50			
	OV IV	8	15.4	9	17.3			
Amount of EO (cc) Mean $\pm$ SD		10.2 $\pm$ 4.3						
Number of rubber bands Mean $\pm$ SD				5.1 $\pm$ 0.9				
PHG grade	PHGI	4	7.69	3	5.76	0.29	0.593	NS
	PHGII	23	44.2	22	42.3	0.04	0.833	NS
	PHGIII	23	44.2	25	48.1	0.16	0.689	NS
Duodenopathy		16	30.7	18	34.6	0.24	0.628	NS

NS non significant

**Table (4):** Post endoscopy symptoms

	Group I No.=50		Group II No.=50		X2	P	Sig.
	No.	%	No.	%			
Dysphagia	35	67.3	32	61.5	0.17	0.68	NS
Epigastric pain	47	90.3	40	76.9	1.78	0.182	NS
Heart burn	40	76.9	38	73.1	0.23	0.629	NS
Odynophagia	29	55.7	33	63.4	0.68	0.409	NS
Retrosternal pain	47	90.3	41	78.8	1.82	0.186	NS
Dyspepsia	45	86.5	39	75	1.96	0.161	NS

NS non significant

**Table (5):** Rates of rebleeding and mortality

		Group I No=52		Group II No=52		X2	P	Sig.
		No	%	No	%			
Rebleeding	2 weeks	5	9.5	3	5.76	2.17	0.14	NS
	4 weeks	2	4.25	1	2.04	2.05	0.81	NS
	6 weeks	1	2.22	1	2.22	0	1	NS
	No	44	84.6%	47	90.4%	0.791	0.374	NS
	Yes	8	15.4%	5	9.6%			
Cause	Ulcers	5	62.5%	3	60%	0.008	0.928	NS
	PHG	2	25%	1	20%	0.043	0.835	NS
	OV	1	12.5%	0	0%	0.677	0.411	NS
	GV	0	0%	1	20%	1.733	0.188	NS
Mortality	Survival	41	79%	40	79.8%	0.210	0.647	NS
	Death	11	21%	10	19.2%			
Mortality after rebleeding	Survival	5	62.5%	3	60%	0.008	0.928	NS
	Death	3	37.5%	2	40%			

NS non significant

**Table (6):** Correlation between rebleeding and selected study parameters

	Group I (N=52)		Group II (N=52)		All studied cases (N=104)	
	r	P (Sig.)	r	p(Sig.)	r	P (Sig.)
Age	+0.016	0.910 (NS)	+0.148	0.296 (NS)	+0.048	0.625 (NS)
Sex (Male, Female)	+0.062	0.661 (NS)	+0.023	0.869 (NS)	+0.038	0.698 (NS)
Diabetes (No, Yes)	+0.144	0.308 (NS)	+0.042	0.769 (NS)	+0.093	0.347 (NS)
Hypertension (No, Yes)	+0.277	0.347 (NS)	+0.118	0.406 (NS)	+0.074	0.456 (NS)
Bilharzial antigen	+0.434	0.326 (NS)	+0.376	0.287 (NS)	+0.334	0.187 (NS)
Primary prophylaxis	-0.393	0.295 (NS)	-0.243	0.182 (NS)	-0.274	0.187 (NS)
Ascites(Absent, Mild,...)	+0.192	0.173 (NS)	+0.126	0.374 (NS)	+0.163	0.099 (NS)
Child classification (A,B, C)	+0.145	0.078 (NS)	+0.123	0.093 (NS)	+0.136	0.014 (S)
PT	+0.436	0.001 (HS)	+0.250	0.044 (S)	+0.350	<0.001 (HS)
INR	+0.526	<0.001 (HS)	+0.283	0.042 (S)	+0.419	<0.001 (HS)
OV grade(2 , 3 , 4)	+0.259	0.008 (HS)	+0.207	0.024 (S)	+0.233	0.001 (HS)
Risk signs	+0.221	0.007 (HS)	+0.139	0.045 (S)	+0.179	0.001 (HS)
Units of blood (0-5)	+0.377	0.006 (HS)	+0.393	0.004 (HS)	+0.387	<0.001 (HS)
Hepatic a. RI	+0.126	0.372 (NS)	+0.055	0.701 (NS)	+0.096	0.332 (NS)
PV velocity	-0.293	0.095 (NS)	-0.243	0.082 (NS)	-0.264	0.067 (NS)
PV diameter	+0.090	0.527 (NS)	+0.083	0.557 (NS)	+0.075	0.448 (NS)
Amount of EO injected	+0.329	0.017 (S)	---	---	---	---
Number of rubber bands	---	---	+0.245	0.039 (S)	---	---
APRI score	+0.634	0.526 (NS)	+0.326	0.744 (NS)	+0.389	0.697 (NS)

## DISCUSSION

In our study, 104 patients with first attack of variceal bleeding were randomized in two groups; group I (52 patients were treated by endoscopic sclerotherapy, their mean age was 50.1 year, 36 male and 16 female) and group II (52 patients were treated by endoscopic band ligation), their mean age was 49.7 year, 33male

and 19 female. There was no significant difference between both groups regarding age and sex. There was no statistically significant difference between the studied groups regarding the cause of chronic liver disease, the majority of patients in both groups have chronic HCV infection, and this is mostly because HCV is the leading cause of chronic liver diseases in Egypt [15].

There was no statistically significant difference between the studied groups regarding post endoscopy symptoms (dysphagia, odynophagia, retrosternal pain, epigastric pain, heart burn and dyspepsia). Oesophageal membrane injuries (erosions or ulcerations) were found in all patients. This agrees with Gimson et al. who found that Complication rates were similar in the two groups [16]. But, this disagrees with Stiegmann et al. who found band ligation to have improved survival and fewer complications [17]. Also, this disagrees with Laine et al. who reported a significant reduction in local complications but no difference in rebleeding or mortality [18]. Moreover, Frequency of treatment induced complications in band ligation were significantly lower as compared with sclerotherapy, mild chest pain and transient fever were significantly more in sclerotherapy as reported by Shafqat et al. [19].

In this study, there was no significant difference between the two studied groups as regards rate of early rebleeding. This agrees with Lo et al. who reported that the rate of early rebleeding following EVL was between 9% and 19%, which is close to results of Xu et al. who stated that the incidence of early rebleeding following EVL was (7.6%). Lo et al. reported 17% rate of rebleeding with band ligation vs. 33% with sclerotherapy, Villanueva et al., (2006), reported 12% incidence rate for re-bleeding for band ligation versus 21% for sclerotherapy [20-23].

Causes of early rebleeding in the sclerotherapy group were: sclerosant ulcer in 5 cases (62.5%), PHG in 2 cases (25%) and OV in one case. This agrees with Sauerbruch et al. who found that early rebleeding following sclerotherapy is caused by sclerosant ulcer in most patients [24]. While, causes of early rebleeding in the band ligation group were: post banding ulcer in 3 cases (60%), PHG in one case (20%) and GV in one case (20%). This result agrees with Vanbiervliet et al. who reported that cases of severe bleeding after EVL were all caused by early slippage of the rubber bands, leaving the unhealed ulcers. Usually, the bands slip spontaneously within the second week after EVL [25].

Mortality among rebleeding cases in the sclerotherapy group was 37.5%, while mortality in the band ligation group was 40%. The mortality rates in the previous literature ranged between 8% and 25%. This lower mortality rates are related to the improvement in the endoscopy

techniques and in the efficacy of vasoactive drugs and prophylactic antibiotics [26-29].

After two weeks of follow up there was no significant difference between both groups as regards clinical, laboratory data and endoscopic findings. Most cases of early rebleeding occur during the first 2 weeks of follow up. Rebleeding was due to development of sclerosant or post banding ulcers (5 cases in the first group and 3 cases in the second group). This agrees with Xu et al. who found that post-EVL bleeding was most likely to occur between the 7<sup>th</sup> and 13<sup>th</sup> day following the procedure [21]. Also, this agrees with Akriyadi et al. who found higher incidence of sclerosant ulcer and rebleeding when endoscopy was repeated earlier, e.g., 70% at 1 week and 30% at two week intervals [30]. Also, Tabibian et al. found that most esophageal ulcers bleeding (28 of 32) occurred within 2 weeks after the latest endoscopic treatment [31]. This can be explained by the more complete healing of the ulcer 2 weeks after endoscopic treatment.

After 4 weeks of follow up there was no significant difference between both groups as regards clinical, laboratory data and endoscopic findings. Rebleeding occurs in 2 cases in group I and one case in group II. The cause of rebleeding in both groups was due to severe portal hypertensive gastropathy (PHG). After 6 weeks of follow up there was no significant difference between both groups as regards clinical, laboratory data and endoscopic findings. Rebleeding occurs in one case in group I (due to bleeding OV) and one case in group II (due to bleeding gastric varix).

In our study, it was found that early rebleeding has significant positive correlation with child-Pugh grade. Also this agrees with Yang et al. (2007) who found that the Child-Pugh score for liver function was an independent risk factor of post-EVL rebleeding [32]. This also agrees with Benedeto-Stojanov et al. who stated that patients with the most severe hepatocellular dysfunction (Child's group C) have the shortest period between the first bleeding and rebleeding (mean 20.8 days) [33]. Our results agree with Berreta et al. who proved that Child-Pugh C was an independent risk factor of death from rebleeding [34]. Also, this agrees with Amitrano et al. who concluded that child class C was an independent predictor of recurrent bleeding; mortality was mainly related to the severity of liver failure. This can be explained by the general concept that

patients with hepatic decompensation bleed more severely than those without hepatic decompensation [35,36].

But, this disagrees with Zhao JR et al. who found that child class was not correlated with the risk of rebleeding and mortality based on univariate analyses. This difference because he used another procedure in treating bleeding Oesophageal varices: Percutaneous Trans hepatic variceal embolization (PTVE). During PTVE, the portal vein is catheterized by a percutaneous trans-hepatic approach and the gastric vein feeding the varix is embolized with ethanol, steel coils, or cyanoacrylate glue using multi-detector row computed tomography [27].

Size and extent of esophageal varices seen at index endoscopy were also significantly positively correlated to the rebleeding. This result agrees with Benedeto-Stojanov et al. who found that primary variceal bleeding was present in 50% patients with medium and in 65.38% patients with large varices [33]. There was no bleeding in patients with small varices. Also, our result agrees with Xu et al. who found that the extent and size of varices are independent risk factors for early rebleeding. Varices that extend along the entire esophagus are much more dangerous than varices that are limited to the middle and lower part. Moreover, a greater extent of varices often means that more rubber bands are needed, increasing the possibility of rebleeding [21]. It also agrees with Varghese, et al. who stated that higher grades of varices, presence of cherry-red spots and fundal varices predict variceal bleed in patients with liver cirrhosis [37]. The only exception to this is a study done by Koch et al. who found that 35% of patients with small varices bled, while only 20% of patients with large varices also bled. This difference because of small sample size, most cases were child class A and longer duration of follow up (36 months) [38].

In our study; there was significant positive correlation between rebleeding and presence of risky signs on varices. All early rebleeding cases in both groups had risky signs on varices at index endoscopy. This agrees with the study of the Northern Italian Endoscopic Club (NIEC) has shown that endoscopic finding of "red signs" is related to the variceal bleeding [39]. Also, Benedeto-Stojanov et al. has shown that endoscopic finding of "red signs" is related to the variceal bleeding. The "red signs" were found in 85% of large varices with bleeding [33].

There was positive significant correlation between rebleeding and the amount of EO injected in sclerotherapy group and number of rubber bands used in band ligation group. This agrees with Xu et al. who found that the number of rubber bands was an independent risk factor for re-bleeding after EVL. Therefore, for varices which were in the mild to moderate class, it may not be reasonable to launch many rubber bands. For severe varices, however, it's usually unavoidable to use more bands [21].

## CONCLUSION

Sclerotherapy is associated with higher incidence of rebleeding than band ligation. Most cases of early rebleeding occur during the first 2 weeks of follow up and were due to development of sclerosant or post banding ulcers. Early rebleeding in both groups was correlated to child pugh classification grade (early rebleeding more in child class C>child class B>child class A), elevated coagulation parameters (elevation in PT, INR) among studied groups, grade of oesophageal varices: Most cases of early rebleeding cases had esophageal varices grade IV and presence of risky signs on varices. No significant correlation between rebleeding and ascites, PV diameter and color Doppler studies could be detected. No statistically significant difference between endoscopic sclerotherapy and band ligation regarding post endoscopy complications could be detected. No significant differences between sclerotherapy and band ligation as regards overall mortality or mortality after rebleeding.

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**Peer reviewer:** Amira Suliman, Professor of Tropical Medicine and Hepatogastroenterology, Faculty of Medicine, Zagazig University, Egypt.  
**Editor:** Mohamad Emara, Assistant Professor of Tropical Medicine and Hepatogastroenterology, Faculty of Medicine, Zagazig University, Egypt