Journal of Medicine in Scientific Research

Volume 5 | Issue 3 Article 28

Subject Area:

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Recommended Citation

A. Eid, Ashraf M.; Shahin, El-Saied A.; Goda, Feisal; and Fouad, Mariam A. (2022) "Esophageal ulcer after band ligation of esophageal varices," *Journal of Medicine in Scientific Research*: Vol. 5: Iss. 3, Article 28.
DOI: https://doi.org/10.4103/jmisr.jmisr_50_22

Original Article

Esophageal ulcer after band ligation of esophageal varices

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Abstract

Introduction

Band ligation plays an important role in the management of esophageal varices (EV), but banding ligation carries a risk of band slippage, postbanding esophageal ulcer formation, and postbanding bleeding.

Aim

The predisposing factors and frequency of esophageal ulcer after banding ligation of EV were assessed.

Patients and methods

A total of 130 cirrhotic patients with EV were subjected to therapeutic or prophylactic banding ligation, and then the patients were observed for 14 days for detection of incidence of esophageal ulcer bleeding after postbanding ligation. This was a prospective study done to compare the cirrhotic patients without endoscopic variceal ligation (EVL)-induced esophageal ulcer as a complication (nonbleeder group, n==118) with bleeding patients after EVL owing to postbanding ulcer (bleeder group, n = 12). Full present and past medical history taking for all patients, physical examination, laboratory investigation, ultrasonography of abdomen and/or triphasic computed tomography abdomen, upper endoscopy, and EVL, were done. Re-endoscopy was done if rebleeding happens after EVL.

Results

After EVL, the incidence of esophageal ulcer bleeding in cirrhotic patients was 9.2%, and the mortality within the 2-week follow-up was 16.7%. The risk factors of postbanding ulcer bleeding were esophageal reflux, increased aspartate transaminase to platelet ratio index score, and focal hepatic lesion.

Conclusion

Bleeding of esophageal ulcer after EVL is not a rare complication of EVL, which is most commonly detected within 14 days after EVL. Reflux esophagitis, increase in aspartate transaminase to platelet ratio index score, and presence of focal hepatic lesion are predisposing factors for postbanding esophageal ulcer bleeding.

Keywords: Banding ligation and esophageal ulcer, esophageal varices, liver cirrhosis

Introduction

In spite of the great new advancement in many therapeutic modalities of bleeding esophageal varices (EV) in cirrhotic patients, early EV rebleeding still happens more often, which may reach up to 30–40% of patients in some cases. The more the increase in frequency of rebleeding rate of EV, the more the increase in mortality for patients with liver cirrhosis [1]. Traditional banding ligation of EV has been recognized as the best therapeutic modality than injection sclerotherapy for the prevention of rebleeding of EV, mortality, and complications [2]. Complications may occur in more than 40% of cirrhotic patients, and the rate of death is 1–2%. Complications of endoscopic variceal ligation (EVL) include esophageal ulceration, substernal pain, stricture, perforation, and even death [3]. Presently, EVL-induced dysmotility of motor function may be transient in some patients or persistent in others [4]. The prevalence of esophageal ulcer bleeding after EVL is reported

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Access this article online

Quick Response Code:

Website: www.jmsr.eg.net

DOI: 10.4103/jmisr.jmisr_50_22

Submitted: 11-May-2022 **Revised:** 10-Jun-2022 **Accepted:** 26-Jun-2022 **Published:** 23-Nov-2022

How to cite this article: Shahin ESA, A. Eid AM, Fouad MA, Goda F. Esophageal ulcer after band ligation of esophageal varices. J Med Sci Res 2022;5:375-9.

to be 3.6–15% [5]. This bleeding is fatal in some cases [6]. Clot formation starts to develop in the strangulated vessels on the day after EVL [7]. After banding of ~3–7 days, esophageal ulcerations develop after the rubber bands slip of, and the ulcer restores within 14–21 days [8]. If early slipping of rubber bands occurs, before the EV was occluded with mature thrombus, rebleeding from esophageal ulceration can occur. There are few reports on the risk factors, and this serious complication is well proven [9]. Increase in aspartate transaminase to platelet ratio index (APRI) score, increase in prothrombin time, digestive bleeding after EV, and reflux esophagitis have been suggested of being the risk factors for bleeding of esophageal ulcer after banding ligation. Cyanoacrylate injection, EVL, and transjugular intrahepatic portosystemic shunt are considered as traditional treatment methods [10], and new treatment modalities include hemospray [11] and esophageal stents [12].

Patients and Methods

The study was done in the Internal Medicine and Tropical Medicine Department of Shebin El-kom Teaching Hospital Menoufia, Egypt, from December 2021 to April 2022. Ethical committee approved number HSH00036. The study included 130 patients who had liver cirrhosis and were subjected to EVL for the treatment of EV, as prophylactic (primary or secondary) or therapeutic. Patients with history of previous injection sclerotherapy were excluded.

Early postbanding ulcer bleeding after EVL was considered as endoscopically conformed active bleeding (spurting or oozing) happening 24 h to 14 days after the procedure from unhealed ulcer, which was formed as a result of early slippage of rubber bands [13].

Patients with active variceal hemorrhage were admitted to ICU, and initial resuscitation was done. Blood transfusion replacement was done early to maintain an average blood pressure of patients of around 100 mmHg in systole [14]. Medical treatment was given in the form of intravenous proton pump inhibitor (PPI, 40 mg) every 12 h [15]. Vitamin K 10 mg/day intramuscular was given at the time of admission and continued for 3–5 days [16]. Somatostatin analog (sandostatin) was infused as an initial bolus of 50 µg intravenous followed by 50 µg/h for 2–5 days along with prophylactic antibiotic (ceftriaxone 1 g intravenous/24) [17].

Upper forward-viewing endoscopy (PENTAX EPM 3500 videoscope) was performed under conscious sedation with 5 mg midazolam given to all patients [18]. EVL (using Medical Endoscopy 6 shooter Saeed multiband ligator-Cook) was done to banding the protruding varix with an elastic rubber ring [13].

After EVL, the patients were prescribed PPI for 2 weeks, as well as broad-spectrum antibiotics and nonselective beta blocker. The patients were allowed to eat 12 h after prophylactic EVL.

All patients were followed for 14 days with re-endoscopy for patients who rebleed after EVL to confirm that postbanding ulcer was the cause of bleeding.

In the final analysis, the patients were classify into two groups as follows: bleeder group (group 1) comprised 12 hemorrhagic patients with bleeding after EVL owing to past-banding ligation ulcer and without other upper gastrointestinal bleeding source, and nonbleeder group (group 2) comprised 118 patients who did not bleed after EVL.

All patients received the following: full medical history taking, including age, sex, history of attack of hematemesis or melena, blood transfusion, cause of liver disease, history of previous endoscopy, or injection sclerotherapy, and complete physical examination, with focus on pulse, blood pressure, body temperature, and signs of liver cirrhosis and portal hypertension. Laboratory tests included complete blood count; liver function tests such as serum bilirubin (total and direct), total serum protein and albumin, SGOT, SGPT, prothrombin time, international normalized ratio, and blood urea; serum creatinine; and APRI [19]. Modified Child–Turcotte–Pugh (CTP) score [20] and MELD score (model for end stage of liver disease) were estimated [21]. Abdominal ultrasonography and/or triphasic computed tomography abdomen were performed.

Statistical analysis

We analyzed the data using SPSS statistical package. Data were determined as mean ± SD for quantitative variable and number and percentage for qualitative one. Fisher test, χ^2 test, *t* test, and paired *t* test were used. *P* value less than 0.05 was consider statistically significant.

Results

Table 1 shows that there was a nonstatistically significant difference in both studied groups regarding CTP classification (A, B, and C) and MELD score ($P > 0.05$).

Table 2 shows that there was a statistically significant difference in ARPI score between bleeder (group 1) versus nonbleeder (group 2) ($P < 0.05$) and no statistical difference in other laboratory investigations between both groups ($P > 0.05$).

Table 3 shows abdominal ultrasound findings. There was a significant difference between hepatic focal lesion of both groups (<0.05) and a nonsignificant difference among both groups regarding ascites, splenic span, and portal vein diameter ($P > 0.05$).

Table 1: Child-Turcotte-Pugh classification and MELD score of studied groups

Variables	Group 1 (*n*=12) (9.2%) [*n* (%)]	Group 2 (*n*=118) (90.8%) [*n* (%)]	*P*
CTP classification			
A	3 (25)	33 (28)	>0.05
B	3 (25)	32 (27)	
C	6 (50)	53 (45)	
MELD score	15±4	15±5	>0.05

CTP, Child-Turcotte-Pugh.

Table 4 shows that there was a nonsignificant difference regarding indication of EVL (therapeutic, primary, and secondary prophylaxis) among both groups ($P > 0.05$).

Table 5 shows that regarding endoscopic findings of both groups, there was a significant difference regarding reflux esophagitis between group 1 and group 2 (<0.05) and a nonsignificant difference regarding grading of EV, risky signs, and number of ligation bands.

Table 6 shows the postbanding management of both groups. There was a nonsignificant difference regarding blood transfusion, β-blocker, and antibiotics ($P > 0.05$).

Table 7 shows that among patients of group 1 (bleeder group), the postbanding ulcer bleeding occurred within 4–14 days, with a mean of 9.1 ± 3.6 after EVL, and the mortality rate was 8.3%. (Figs. 1–5).

Discussion

Esophageal ulcer bleeding after EVL occurs in 3.6–15% of cases [5]. Although esophageal ulcer bleeding risk factor after EVL have not been well known, and the guidelines for therapy of this potentially fetal complication are not well identified [13]. In our study, the frequency of bleeding from postbanding ulcer following EVL in cirrhotic patients was found to be 9.2% in contrast to 7.9 by Soha *et al*. [22]. However, Shendy *et al*. [23] estimated that the rate of early rebleeding after EVL was 11%. On the contrary, this result is higher than previously published rates by Petrasch *et al*. [24].

Our study failed to find a statistically significant relation between CTP classes or MELD score and occurrence of postbanding ulcer bleeding, similar to a previous study by Soha *et al*. [22]. This may relate to the postendemic phase of bilharziasis in Egypt, which causes more vascular decompensation than cellular decompensation. So, it is represented by more increase in portal hypertension than decreased in synthetic functions, which affect Child and MELD score. In another study, deterioration of liver condition (CTP-Class C and increase MELD score) was identified as a predictive factor of rebleeding in cirrhotic patients [23]. Decrease coagulation ability and increased vascular fragility.

A large extension of submucosal EV and its fragility might explain the importance of bleeding from esophageal ulcer after EVL without effective local thrombosis.

Table 2: Baseline laboratory data of studied groups

Variables	Bleeder group 1 (*n*=12)	Nonbleeder group 2 (*n*=118)	*P*
HB	9.2±1.7	9.8±1.8	>0.05
WBCs	8.1±3.1	7.8±3.2	>0.05
Platelets	123±66	129±50	>0.05
Total protein	5.6±1	5.9±0.98	>0.05
Albumin	2.55±0.6	2.53±0.5	>0.05
INR	1.7±0.5	1.6±0.6	>0.05
ALT	41±34	42±31	>0.05
AST	59±40	53±34	>0.05
Total bilirubin	2.8±2.7	2.9±2.8	>0.05
Direct bilirubin	1.7±1.3	1.8±1.4	>0.05
Serum creatinine	1.1±0.5	1.2±0.6	>0.05
APRI score	1.3	0.95	<0.05

ALT, alanine transaminase; APRI, aspartate transaminase to platelet ratio index; AST, aspartate transaminase; HB, hemoglobin; INR, international normalized ratio; WBC, white blood cell.

Table 3: Abdominal ultrasound of studied groups

Variables	Bleeder group 1 (*n*=12) [*n* (%)]	Nonbleeder group 2 (*n*=118) [*n* (%)]	*P*
Focal lesion	5 (42)	20 (17)	<0.05
No ascites	2 (17)	24 (20)	<0.05
Mild ascites	3 (25)	31 (26)	>0.05
Moderate ascites	3 (25)	25 (21)	>0.05
Tense ascites	4 (33)	38 (32)	>0.05
Splenic span (cm)	16.7±1.2	16.3±1.1	>0.05
PVD (mm)	15.6±3	15.1±2	>0.05

PVD, portal vein diameter.

Table 4: Indications of endoscopic variceal ligation

Indication of EVL	Group 1 (*n*=12) [*n* (%)]	Group 2 (*n*=118) [*n* (%)]	*P*
Therapeutic	7 (58)	37 (31)	
Primary prophylaxis	0	25 (21)	>0.05
Secondary prophylaxis	5 (42)	56 (47)	

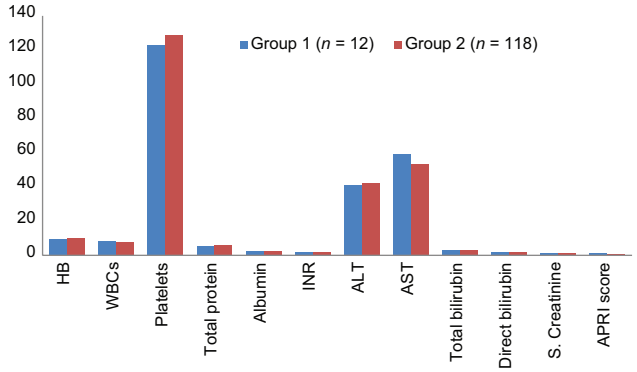

Figure 1: Baseline laboratory data of studied groups.

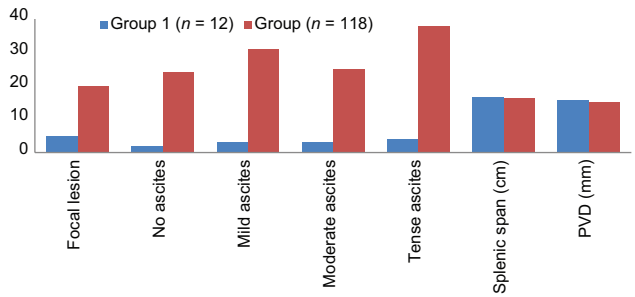

Figure 2: Abdominal ultrasound of studied groups.

Table 5: Endoscopic finding among both studied groups

Endoscopic finding	Bleeder group 1 (*n*=12) [*n* (%)]	Nonbleeder group 2 (*n*=118) [*n* (%)]	*P*
Reflux esophagitis	6 (50)	8 (7)	<0.05
EV grading			
Grade 2	2 (17)	44 (37)	
Grade 3	10 (83)	62 (53)	>0.05
Grade 4	0	12 (10)	
Risky signs	8 (67)	83 (70)	>0.05
Number of ligation bands			
Three bands	3 (25)	30 (25)	>0.05
Four bands	4 (33)	45 (38)	
Five bands	4 (33)	34 (29)	
Six bands	1 (8)	9 (8)	

EV, esophageal varices; EVL, endoscopic variceal ligation.

Table 6: Postbanding management of both studied groups

Variables	Bleeder group 1 (*n*=12) [*n* (%)]	Nonbleeder group 2 (*n*=118) [*n* (%)]	*P*
Blood transfusion	3 (25)	27 (23)	>0.05
PPI	9 (75)	90 (76)	>0.05
B-blocker	6 (50)	55 (47)	>0.05
Antibiotic	7 (58)	70 (59)	>0.05

PPI, proton pump inhibitor.

Table 7: Mortality rate and timing of postbanding ulcer bleeding

Variables	Group 1 (*n*=12)
Time of postbanding ulcer bleeding (days)	9.1±3.6
Range	4-14
Mortality rate after postbanding ulcer bleeding	2 (16.7%)

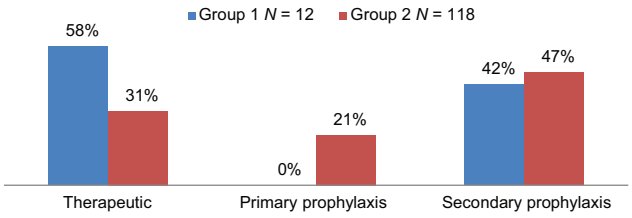

Figure 3: Indications of endoscopic variceal ligation.

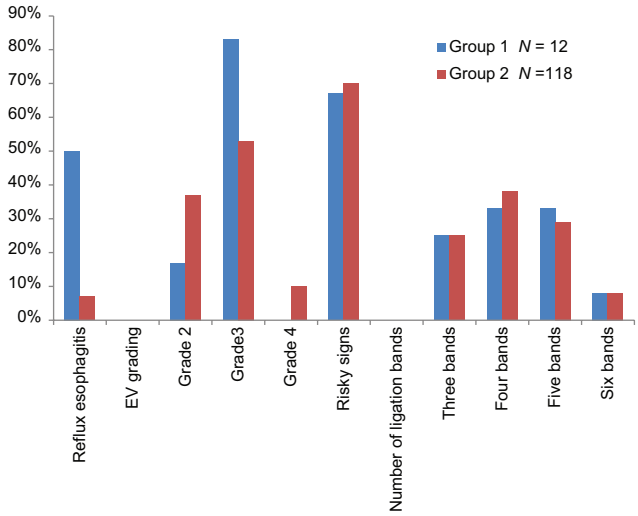

Figure 4: Endoscopic finding among both studied groups.

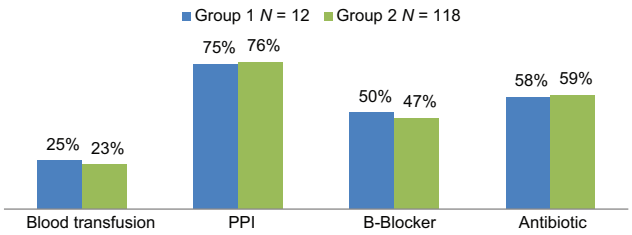

Figure 5: Postbanding management of both studied groups.

In our study, there are significant associations between high APRI score and developed of postbanding ulcer hemorrhage. This is in agreement with Vanbiervliet *et al*. [9]. However, Cho *et al*. [13] found that there is a nonsignificant relation between them. This high APRI score may be attributed to the aspartate transaminase level, which indicates significant cirrhosis, and low platelets, which leads to a defect in hemostasis.

The present study showed a significant difference between presence of hepatic focal lesions and occurrence of postbanding ulcer. This is in agreement with Soha *et al*. [22]. This may be owing to portal vein thrombosis associated with hepatic focal lesions, which causes more increase in portal pressure. This is in contrast to Xu *et al*. [25].

In present study, there was a statistically significant relation between presence of reflux esophagitis by endoscopy and occurrence of postbanding ulcer bleeding, as reported by Soha *et al*. [22] and Sinclair *et al.* [26]. These finding strongly suggest that early slippage of rubber band and postbanding ligation ulcer bleeding may be related to the damage of mucosa caused by the exposure of acid refluxate at the end of esophagus.

In our study, there was a nonsignificant difference between occurrence of postbanding ulcer bleeding and PPI use after EVL, as reported by Vanbiervliet *et al.* [9] and Sinclair *et al.* [26]. In contrast, Kang *et al*. [27] concluded that PPI administration after EVL may have a protective effect against postbanding ulcer bleeding. This could be explained by the improvement of reflux esophagitis, which is a risk factor of esophageal ulcer bleeding after postbanding ligation.

The death rate of the esophageal ulcer bleeding after EVL was 16.7% in the present study, which is an increase than the death rate of a previous report of 10% by Soha *et al*. [22]. Cho *et al*. [13] found that the mortality rate was 27.3%. The patients who died experienced a massive bleeding episode that led to death despite an effective replacement therapy.

Conclusion

Esophageal ulcer bleeding after EVL is not a rare complication and most commonly detected within 2 weeks after EVL. Increase APRI score, reflux esophagitis, and presence of focal hepatic lesion are risk factor for postbanding ulcer bleeding.

Financial support and sponsorship

Nil.

Conflicts of interest

None declared.

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