Current Science International
Volume: 12 | Issue: 02| April - June| 2023

EISSN:2706-7920 ISSN: 2077-4435
DOI: 10.36632/csi/2023.12.2.21
Journal homepage: www.curresweb.com
Pages: 223-231

Efficacy and Safety of Endoscopic Band Ligation in the Treatment of Actively Bleeding Portal Hypertensive Gastropathy in Cirrhosis

Amina H. Elsherra, Amal A. Selim, Ahmed F. Selim and Mamdouh A. Gabr

Department of Internal Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt

Received: 03 May 2023 **Accepted:** 01 June 2023 **Published:** 15 June 2023

ABSTRACT

Background: Portal hypertensive gastropathy (PHG) is a common complication of cirrhosis, which is often underestimated in clinical practice. PHG most often lead to chronic GI blood loss manifested as transfusion dependant anaemia, however, it may also presents with acute and even massive GI bleeding.At present, there is no clear recommendations regarding the best treatment of acute bleeding from PHG, and effective management of this condition remains a clinical challenge. Herein, we report our experience in the control of acute bleeding from PHG by targeted endoscopic band ligation (EBL) **Methods:** In the period from January the 1st 2021 to January the 1st 2022, a total of 30 cirrhotic patients with actively bleeding PHG were selected and identified from 800 cirrhotic patients who underwent emergency UGI endoscopy for acute UGI bleeding in our endoscopy center during this period. They were treated by targeted EBL that was applied only for the bleeding PHG lesions. All treated patients were placed on NSBBs (propranolol) in incremental doses, proton pump inhibitors and antibiotics with close monitoring. A follow-up endoscopy was performed for all patients one week after the initial hemostatic procedure. **Results:** Initial hemostasis was achieved in all treated patients (100%) while recurrent bleeding was observed only in 3(10%) patients. No serious complication was observed in any of the treated patients. **Conclusion:** Based on the results of the present study, one may say that targeted EBL is an effective, simple, cheap and safe hemostatic modality for the control of acute bleeding from PHG that should be among the first line options in the endoscopic treatment of this elusive and difficult to treat bleeding PHG.

Keywords: portal hypertensive gastropathy, endoscopic band ligation,cirrhosis.

1. Introduction

Portal hypertensive gastropathy (PHG*)* occurs as a complication of cirrhotic and non-cirrhotic portal hypertension. PHG is a vascular disorder characterized by ectasia of mucosal capillaries and submucosal venules without inflammation (Zhou *et al*., 2002). PHG is clinically important because it may cause acute (and even) massive and /or chronic and insidious blood loss (Bazerbachi *et al.*, 2022).

Acute bleeding from PHG is manifested by hematemesis and/or melena, while chronic and insidious blood loss is manifested by iron deficiency anemia (IDA), however acute bleeding occurs less commonly than chronic bleeding (Rockey and Update, 2019). The diagnosis of acute bleeding from PHG is established when active bleeding from PHG lesions or when removable clots overlying these lesions is observed (Gabr and Gabr, 2014).

At present, there is no clarity in recommendations for emergency treatment of acute bleeding from PHG, and effective management of this condition remains a clinical challenge.

Since portal hypertension is the main pathogenic factor in the development of PHG, reducing portal pressure is therefore the mainstay approach to treat acute bleeding from PHG (Bazerbachi *et al.*, 2022). A number of pharmacotherapies have been used to reduce portal pressure including nonselective beta-blockers, vasoactive drugs with varying success rates (Zhou *et al*., 2002).

Portal decompression by shunt procedures (surgical or TIPS) has been tried as a rescue therapy

Corresponding Author: Amina H. Elsherra, Department of Internal Medicine, Faculty of Medicine, Tanta University, Tanta, Egypt.

when bleeding is refractory to medical therapy, however both have not been extensively analyzed as a treatment for acute PHG bleeding. Moreover, both are invasive and associated with substantial morbidity and mortality (Orloff *et al.,* 1995 & Zhang *et al.,* 2015).

Endoscopic therapy for acute bleeding PHG has been little studied including argon plasma coagulation (APC), hemospray and cryotherapy. Meanwhile, little data exists on the efficacy of these endoscopic therapies in acute PHG bleeding.

Herein, we report our experience in the control of actively bleeding PHG by endoscopic band ligation (EBL) in a group of cirrhotic patients in our endoscopy center in Tanta University Hospital (TUH).

2. Patients and Methods

In the period from January the 1st 2021 to January the 1st 2022, a total of 30 cirrhotic patients with actively bleeding PHG were selected and identified from 800 cirrhotic patients who underwent emergency UGI endoscopy for acute UGI bleeding in our endoscopy center in TUH during this peroid.

Inclusion criteria:

Only cirrhotic patients with endoscopic evidence of actively bleeding PHG lesions were included in the study. They were selected and identified from all cirrhotics who underwent emergency UGI endoscopy for acute UGI bleeding.

Exclusion criteria:

1- Cirrhotic with hepatic encephalopathy.
2- Cirrhotic patients without endoscopic evidence of actively bleeding PHG (ooze or removable clots) or bleeding from other lesions.
3- Patients with bleeding PHG previously treated by another mode of endoscopic therapy.

This study is in agreement with the ethical guidelines of the Declaration of Helsinki and it follows the ethical standards of Tanta faculty of medicine approval code (34316/12/20) all patients were aware of the steps, and goal of the study and they were included after obtaining written informed consent from them.

All the study group were subjected to the following:

- **Full history taking.**
- **Complete physical examination** searching for stigmata of chronic liver disease (CLD).
- **Laboratory investigations**:
    a. Urine analysis.
    b. Complete blood count (CBC).
    c. Prothrombin time and activity (INR).
    d. Blood urea and serum creatinine.
    e. Liver biochemical tests:
        - Bilirubin (direct &indirect).
        - ALT&AST.
        - Serum albumin.
        -
- **Abdominal US scan**:
    - Liver status.
    - Portal vein diameter.
    - Splenic size.
    - Ascites.

The severity of liver disease was determined by CTP score based on clinical ,biochemical and ultrasonographic findings

- **Emergency UGI Endoscopy**:

After initial assessment and resuscitation in the emergency unit, an informed consent was obtained and emergency UGI endoscopy was performed searching for the source of bleeding.
PHG lesions were considered the source of bleeding if they are actively bleeding, covered with densely adherent clot or it is the only lesion detected in the presence of fresh or altered blood in the upper GI tract.
After identification of the bleeding PHG lesions, EBL was applied only for these bleeding lesions. All treated patients were placed on NSBBs (propranolol) in incremental doses and proton pump inhibitors.
A follow-up endoscopy was done one week after EBL to confirm the efficacy of the procedure as well as to detect any complications related to the procedure.

The patients were instructed to report and notify any further bleeding episodes in the follow-up period (6 months).

Statistical analysis of the data:

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp) Qualitative data were described using numbers and percentages. The Kolmogorov-Smirnov test was used to verify the normality of distribution. Quantitative data were described using range (minimum and maximum),mean and standard deviation.

3. Results

The demographic and base_line characteristics of our study group with actively bleeding PHG lesions are reported in table (1).

Table 1: Demographic and clinical characteristics of the studied groups: (N=30)

Demographic data	**Cases (n = 30)**	
	No.	**%**
Gender		
Male	20	66.7
Female	10	33.3
Age (Years)		
Min.–Max	40 – 81	
Mean ± SD.	57.72+ 9.13	
Residence		
Rural	25	83.3
Urban	5	16.7
Clinical presentation		
Melena	22	73.0
Hematemsis & Melena	8	27.0
Hemodynamic status		
Stable	27	90.0
Unstable	3	10.0
Previous endoscopic therapy		
EIT	18	60.0
EBL	4	13.3
No	8	26.6
Splenomegaly		
Yes	25	83.3
No	5	16.6
Ascites		
Yes	16	53.3
No	14	46.7

EIT endoscopic injection therapy, EBL endoscopic band ligation, SD, standared deviation

All our patients with actively bleeding PHG were cirrhotics, predominantly males ,mostly from rural areas with relatively older age (57.72±9.13 y).

The main clinical presentation was melena (73%), while (27%) of them presented with both hematemesis and melena and 3 patients (10%) were hemodynamically unstable at presentation indicating massive bleeding.

Additionally, (73.3%) of them underwent previous endoscopic therapy for esophageal varices while in 8 patients (27%) this was the index bleeding.

Splenomegaly was clinically detected in the vast majority of our patients 25(83.8%) indicating portal hypertension, while clinically detectable ascites was present in (53.33%) of our patients indicating advanced cirrhosis.

The laboratory findings in our treated patients with actively bleeding PHG are represented in table (2).

Table 2: Laboratory findings of the studied group: (N=30)

Variable		**Cases (n = 30)**
Hb(gm/dl)	Min.–Max	6.5 – 12.0
	Mean ± SD.	9.03 + 1.36
	Median	9.0
WBCs (10^3)	Min.–Max	2.66 – 14.50
	Mean ± SD.	6.19 + 3.08
	Median	5.40
Platelets (10^3)	Min.–Max	70.0 –230.0
	Mean ± SD.	128.6 + 67.3
	Median	140.0
Blood Urea	Min.–Max	25.0 – 70.0
	Mean ± SD.	48.35+ 13.25
	Median	50.0
Serum creatinine	Min.–Max	0.9 – 1.7
	Mean ± SD.	1.24 + 0.19
	Median	1.20
Total bilirubin	Min.–Max	1.2 – 2.0
	Mean ± SD.	1.51 + 0.24
	Median	1.50
ALT	Min.–Max	16.0 -68.0
	Mean ± SD.	39.8 + 19.65
	Median	35.0
AST	Min.–Max	13.0 – 72.0
	Mean ± SD.	38.9 + 19.89
	Median	39.0
Serum albumin	Min.–Max	2.29 – 4.5
	Mean ± SD.	3.22 + 0.43
	Median	3.20
PT	Min.–Max	13.5 – 20.0
	Mean ± SD.	16.27 + 1.25
	Median	16.8
INR	Min.–Max	1.1 – 1.7
	Mean ± SD.	1.4 + 0.15
	Median	1.4
Viral markers	**Number**	**Percentage %**
HCV +ve	26	86.66%
HBs +ve	2	6.66%
Viral negative	2	6.66%

ALT: alanine transaminase; AST: aspartate transaminase; INR: international normalized ratio; SD: standared deviation

Almost all our patients with actively bleeding PHG were anemic at presentation (Hb=9.03±1.36 gm/dl), while the vast majority of them have a platelet count less than 100000 which is an indirect marker of portal hypertension.

Moreover, the mean serum albumin was low (3.22±0.43), the bilirubin was high (1.51±0.24),and the INR and prothrombin time were increased indicating advanced cirrhosis.
The US findings in our treated group were represented in table (3).

Table 3: Ultrasonographic findings of the studied group: (N=30)

Ultrasound findings	**Cases (n = 30)**	
	No.	**%**
Liver status		
Cirrhotic	21	70.0
Periportal fibrosis	5	16.7
Mixed	4	13.3
Portal vein diameter		
Min.-Max.	15.0 – 20.0	
Mean ± SD	16.4 + 1.63	
Median	16.0	
Spleen		
Normal	1	3.3
Mild splenomegaly	8	26.7
Moderate splenomegaly	18	60.0
Marked splenomegaly	3	10.0
Splenic size(cm)		
Min.-Max.	12.0 – 20.0	
Mean ± SD	16.3 + 1.83	
Median	17.0	
Ascites		
Yes	25	83.3
No	5	16.7

The liver has cirrhotic echotexture in most of our patients (70%), periportal fibrosis in (16.7%) and mixed pattern in (13.3%).

The portal vein was dilated in all patients (100%), and the spleen was enlarged in almost all of them (96.7%), both indicating the presence of PH in all patients with actively bleeding PHG.
Additionally, Ascites was ultrasonographically detected in the vast majority of them (83.3%) indicating advanced or decompensated cirrhosis.

Table 4: CTP score of the bleeding PHG patients: (N=30)

CTP score	**Cases (n = 30)**	
	No.	**%**
Child A	6	20.0
Child B	17	56.7
Child C	7	23.3
Min – Max	6 - 13	
Mean + SD	6.98 + 1.98	

CTP; Child Turcotte Pugh

The vast majority of our patients (80%) have advanced cirrhosis (child's class B or C) indicating advanced liver disease.

Table 5: Results of emergency upper GI Endoscopy in the bleedings PHG patients (N=30)

Endoscopic findings	Cases (n = 30)	
	No.	%
Bleeding PHG lesions		
Oozing	26	86.7
adherent clots	4	13.3
Non bleeding lesions		
*** Varices**		
a) Esophageal		
Obliterated	19	63.3
Intact	5	16.7
b) Intact fundal varies	3	10.0
c) NO varices	3	10.0

Emergency UGI endoscopy of our patients revealed that, 26 (86.7%) patients have actively bleeding lesions (ooze), and the remaining 4 (13.3%) patients have densely adherent clot over these PHG lesions as a clue of recent bleeding.

Non - bleeding varices (esophageal & gastric) were detected in 27 (90%) patients, while the remaining 3 (10%) patients with bleeding PHG have no varices indicating that bleeding PHG may precede the development of varices in cirrhotic patients .

Mode of endoscopic therapy in the studied group:

All our patients (100%) with bleeding PHG were treated by targeted EBL that was applied only to the bleeding lesions.All treated patients were placed on PPIs and NSBBs in incremental doses.

Outcome of targeted EBL of the bleeding PHG lesions is presented in table (6)

Table 6: Outcome of targeted EBL of the bleeding PHG lesions (N=30)

Outcome of the endoscopic therapy	Cases (n = 30)	
	No.	%
Initial Hemostasis	30	100.0
Recurrent bleeding	3	10.0
Complications		
Clean base ulcer	30	100.0
Deep ulcer	0	0.0
Perforation	0	0.0

Initial hemostasis was achieved in all treated patients (100%) and recurrent bleeding was observed in 3 (10%) patients. Regarding complications related to the procedure, a clean base ulcer was observed in all cases (100%), while no serious complications (deep ulcer or perforation) were observed in any of the treated patients (0%).

The three patients with recurrent bleeding managed by conservative treatment.

4. Discussion

In the period from January 2021 to January 2022, a total of 800 cirrhotic patients with acute UGI bleeding underwent emergency UGI endoscopy in our endoscopy center. Out of these patients 30 patients (3.75%) proved to have actively bleeding PHG lesions, who are the subject of the present analysis.

Overview of the demographic and clinical characteristics of our study group revealed that, our patients with actively bleeding PHG are predominantly males (66.7%), from rural areas (83.3%) and

of relatively older age (mean =57.72 ± 9.13 years).However, at present, the association between PHG, age and gender has not reached a consensus. Meanwhile, 3 other studies reported an association between male gender and the occurrence of sever PHG (Hanafy and El Hawary, 2016; Wu *et al.,* 2022; El-Kalla *et al.,* 2018).

The main presenting symptom in our cirrhotic patients with actively bleeding PHG was melena (73%), while hematemesis and melena indicating profuse or massive bleeding was observed in 8 (27%) patients , moreover 3 of them were hemodynamically unstable at presentation (table1). A finding that indicates that PHG may bleed massively unlike what is kept in our minds.

In this regard, another study from USA reported that 21% of their cirrhotic patients with actively bleeding PHG presented with massive bleeding(Zhou *et al*., 2002).Again adding more support to our belief that PHG is an important cause of bleeding in cirrhotic portal hypertensive patients.

Over the years, portal hypertension was considered the initiating factor of both PHG and varices.Many studies have explored the relationship between PHG and the occurrence and grading of varices, but no consensus report has been reached (Wu *et al.,* 2022). Some of these studies found a relationship between variceal grade and the severity of PHG, while others denied this relationship (Wu *et al.,* 2022; Gabr *et al*., 2016).

Interestingly, in the present study, most of our patients with bleeding PHG (73%) underwent previous endoscopic therapy for esophageal varices.A finding that indicates that variceal obliteration by endoscopic therapy may aggravate PHG lesions and increase the risk of bleeding from these lesions.

Meanwhile, 2 other studies also suggested that previous endoscopic therapy for OV increase the severity and the risk of bleeding from PHG (Primignani *et al.,* 2000; Thuluvath and Yoo, 2002). However, these data need further validation in large prospective studies.

The results of laboratory and US studies of our patients with acute bleeding from PHG revealed that manifestations of portal hypertension were present in almost all patients (splenomegaly, dilated PV & thrombocytopenia),this confirm that portal hypertension is the main pathogenic factor for the development of PHG .

Additionally, the vast majority of our patients (80%) have advanced liver disease (child's class B or C), also ascites was detected by US in 25(83.3%) patients indicating that the majority of our patients with acute bleeding from PHG have advanced or decompensated cirrhosis that may be considered a risk factor for acute bleeding from PHG lesions. A finding that was reported by other studies ((Wu *et al.,* 2022; Urrunaga and Rockey, 2014).

Considering all these factors, one can say that portal hypertension is the main pathogenic factor for the development of PHG and the severity of liver disease may be considered a risk factor for acute bleeding from PHG lesions. Similar results were also reported by a Chinese study conducted by Wu *et al.* (2022).

During emergency endoscopy, 26 (86.7%) of our patients have actively bleeding PHG lesions (ooze) and 4 (13.3%) patients have densely adherent clots over these lesions indicating recent hemorrhage.

At present, there is no clear recommendations for emergency treatment of acute bleeding from PHG, and effective management of thiscondition remains a clinical challenge.

Endoscopic therapy for acute bleeding PHG has been little studied .Only one study has evaluated the use of endoscopic therapy with APC in chronic PHG bleeding with good results (Hanafy and El Hawary, 2016). Additionally, the use of hemospray to control acute bleeding from PHG has been reported only in a single case report and few case series (Ishaq *et al.,* 2017; Smith *et al.,* 2014).

Endoscopic band ligation (EBL) was first developed in the mid 1980s for the treatment of esophageal variceal bleeding. The use of this therapeutic modality to control acute bleeding from PHG has not been described in current literature except in a single case report and a small case series (Gabr and Gabr, 2014; Philips *et al.*, 2020).

All our patients (100%) were treated by targeted EBL that was applied only for the bleeding PHG lesions.Initial hemostasis was achieved in all patients (100%), and recurrent bleeding was observed only in 3 (10%) patients. The only complication observed is a clean-base ulcer at the site of EBL in all patients (100%), while no serious complications were observed in any of the treated

patients. Similar results were also reported by the only 2 relevant publications [4,16], dealing with the use of EBL in acute PHG bleeding.

Surprisingly, 3 (10%) of our cirrhotic patients with actively bleeding PHG have no varices, indicating that acute bleeding PHG may precede the development of varices in cirrhosis, something that could be considered unique for this study that further raise the importance of acute bleeding PHG as an important cause of bleeding in cirrhotic patients which is often underestimated.

Finally, based on the results of this study, one may say that targeted EBL is an effective, simple, cheap and safe hemostatic modality for the control of acute bleeding from PHG that should be among the first line options in the endoscopic treatment of this elusive and difficult to treat bleeding PHG.

Funding
This paper was not funded.

Declaration of interest

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

References

Bazerbachi, F., M.J. Levy, et al. 2022. Endoscopic Treatment of Portal Hypertension and Its Complications, 76–89.

El-Kalla, F., L. Mansour, A. Kobtan et al., 2018. Blood ammonia level correlates with severity of cirrhotic portal hypertensive gastropathy. Gastroenterol Res Pract. 9067583. doi: 10.1155/2018/9067583

Gabr, M. and M.M. Gabr, 2014. Su1598 Endoscopic Treatment of Actively Bleeding Portal Hypertensive Gastropathy in Cirrhosis. Gastrointest Endosc., 79(5):AB335.

Gabr, M.A., M.A. Tawfik and A.A. El-Sawy, 2016. Non-variceal upper gastrointestinal bleeding in cirrhotic patients in Nile Delta. Indian J Gastroenterol, 2016 Jan;35(1):25-32. doi: 10.1007/s12664-016-0622-7.

Hanafy, A. S. and A. T. El Hawary (2016). Efficacy of argon plasma coagulation in the management of portal hypertensive gastropathy. Endosc Int Open. 2016 Oct; 4(10): E1057–E1062. Published online 2016 Oct 6. doi: 10.1055/s-0042-114979

Ishaq, S., T. Kuwai and K. Siau, 2017. Use of hemostatic powder in bleeding portal hypertensive gastropathy. VideoGIE., 2(9):238–40.

Orloff, J., M.S. Orloff, L. Orloff, et al., 1995. Treatment of Bleeding from Portal Hypertensive Gastropathy by Portacaval Shunt. Hepatology, 21(4):1011-7.

Philips, C.A., S. Kumbar, R. Ahamed, et al., 2020. Managing Acute Portal Hypertensive Gastropathy Bleed During the Time of COVID-19 Pandemic: Novelty or Necessity?. Cureus., 12(5): e8333.

Primignani, M., L. Carpinelli, P. Preatoni, et al., 2000. Natural history of portal hypertensive gastropathy in patients with liver cirrhosis. Gastroenterology, 119(1):181-7.

Rockey DC. An Update, 2019. Portal Hypertensive Gastropathy and Colopathy. Clin Liver Dis., 23(4):643–58

Smith, L.A., A.J. Morris, A.J. Stanley, 2014. The use of hemospray in portal hypertensive bleeding ; a case series. J Hepatol., 60(2):457–60.

Thuluvath, P.J., H.Y. Yoo, 2002. Portal Hypertensive gastropathy. Am J Gastroenterol, 97(12):2973-8.

Urrunaga, N.H. and D.C. Rockey, 2014. Portal hypertensive gastropathy and colopathy. Clin. Liver Dis., 18(2):389-406.

Wu, R., K. Liu, C. Shi, et al., 2022. Risk factors for portal hypertensive gastropathy. BMC Gastroenterol, 22(1):436.

Zhang, H. C.i MD, S. Ashish MD, S.M. Gould. MD, 2015. Refractory Gastrointestinal Bleeding from Portal Hypertensive Gastropathy: 522. American Journal of Gastroenterology 110, S226, October 2015.

Zhou Y., L. Qiao, J. Wu, et al., 2002. Comparison of the efficacy of octreotide, vasopressin, and omeprazole in the control of acute bleeding in patients with portal hypertensive gastropathy: A controlled study. J Gastroenterol Hepatol., 17(9):973-9.