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Gastric Endoscopic Mucosal Resection and Polypectomy among Patients with Liver Cirrhosis and Esophageal Varices in the setting of acute upper gastrointestinal bleeding

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Abstract

Background

Gastric polyps are not infrequently reported among cirrhotic patients. Endoscopic resection of gastric polyps among patients with liver cirrhosis and esophageal varices carries the risk of post-polypectomy bleeding. This may explain why endoscopists are reluctant to its excision.

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The aim is to evaluate the incidence of immediate (intraoperative) and delayed (within 30 days) post-polypectomy bleeding among cirrhotic patients with esophageal varices and portal hypertension and determine its risk factors.

Methods

This study comprised 39 cirrhotic patients with portal hypertension and varices who presented with gastrointestinal bleeding, and they had gastric polyps detected during the endoscopic intervention to control the acute bleeding or during follow-up. All patients were exposed to the entire history, clinical examination, and basic laboratory workup. Esophagogastroduodenoscopy was done to combine bleeding control and polypectomy simultaneously.

Results

Immediate (intraoperative) post-polypectomy bleeding occurred in 38.8% of patients, and no delayed bleeding was reported. Most of the reported bleeding was mild and clinically non-significant, and it stopped spontaneously or endoscopically. Furthermore, no mortality was reported.

The risk of immediate (intraoperative) bleeding significantly increased with advanced age, advanced liver disease, increased portal hypertension with large varices, and decreased platelet count; meanwhile, the sex of patients, size, location, and method of polypectomy did not significantly increase the risk of gastric post-polypectomy bleeding among cirrhotic patients with portal hypertension and esophageal varices.

Conclusions.

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Among patients with cirrhosis and portal hypertension, gastric polypectomy simultaneously done during endoscopic intervention for esophageal varices is considered a safe maneuver.

Keywords: Gastric polypectomy, liver cirrhosis, post polypectomy bleeding, portal hypertension.

Introduction

Advanced liver disease with cirrhosis is complicated by portal hypertension and the formation of portosystemic collaterals and varices at different sites (esophageal, gastric, rectal, and ectopic sites) through the gastrointestinal tract. It may extend beyond the gastrointestinal tract, as in the retroperitoneal area. There is also splenomegaly complicated with thrombocytopenia [1]. Patients with liver cirrhosis (LC) tend to bleed due to a disruption of coagulation factor synthesis in the liver, and this disruption is often accompanied by thrombocytopenia and portal hypertension [2].

One critical study found that portal hypertensive polyposis is familiar in patients with portal hypertension and has benign characteristics [1].

Some studies suggested the management of portal hypertensive polyposis with APC, but the safety of polypectomy or band ligation of polyps has not been extensively investigated [3].

The incidence of gastric polyps increased; however, most polyps are asymptomatic and usually discovered incidentally during routine endoscopic interventions to manage acute GIT bleeding or follow-up [4].

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Previous studies indicated that patients with LC are more prone to bleeding following an invasive procedure such as surgery or liver biopsy [5].

On the other side, the results of the study conducted by Winter et al. suggest that patients with portal hypertension are not at a higher risk of bleeding during polypectomy when compared to patients with healthy livers [6].

In cirrhotic patients, endoscopy is not only used to detect esophageal varices (OV). Still, we can see further gastrointestinal complications of portal hypertension, such as portal hypertensive gastropathy, gastric varices, and portal hypertensive polyps (PHP) [7].

With the increased and frequent use of endoscopy in cirrhotic patients with portal hypertension and varices, the detection of gastric polyps is increasing, as is the need for polyp removal to decrease the risk of recurrent spontaneous bleeding [8].

Gastric polyps are detected in 6% to 8% of all upper endoscopic examinations, with most polyps being classified as fundic gland polyps (77–80%) and hyperplastic polyps (17–19%) [9].

The prevalence of hyperplastic polyps in patients undergoing esophagogastroduodenoscopy (EGD) is estimated to be about 1% in patients with cirrhosis [10].

Gastric hyperplastic polyps have been identified as a cause of transfusion-dependent iron-deficiency anemia or obscure gastrointestinal bleeding. They are more commonly seen in patients with chronic gastritis (e.g., *Helicobacter*-associated) [11]. Although endoscopic resection (ER) has

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been described for large GHP resection, there is no data regarding its clinical impact on gastrointestinal blood loss [12].

The portal hypertensive polyposis mainly had the histologic characteristics of hyperplastic polyps, with hyperplasia and markedly proliferating, ectatic capillaries in the lamina propria.

PHP accounts for over 80% of diagnosed polyps and is the most frequently discovered gastric polyps. Adenomas are rare; duodenal polyps are in 8% of patients, with fewer hyperplastic polyps than in the stomach [1]. Macroscopically by endoscopy, PHP cannot be distinguished from normal, hyperplastic polyps but frequently presents with small ulcerations and may have a risk of repeated bleeding or anemia, which might need repeated blood transfusions [13].

Even histologically, there are similarities between hyperplastic and PHP. There still needs to be clear diagnostic criteria for PHP. However, typical features of PHP reportedly include foveolar hyperplasia of the epithelium and proliferating ectatic capillaries in the lamina propria; this indicates their portal hypertensive nature and distinguishes them from inflammatory polyps [14]. Most studies did not investigate the risks, such as post-polypectomy bleeding or other complications, and the benefits of endoscopic resection of portal hypertensive polyps or other polyp removal in patients with portal hypertension who presented with bleeding.

Endoscopists are often hesitant to perform endoscopic polypectomy in patients with liver cirrhosis and esophageal varices because of the potential risk of post-polypectomy bleeding. However, the risk of bleeding following polypectomy in patients with liver cirrhosis has not been

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extensively evaluated. And little is known about the feasibility, safety, and outcome of gastric polypectomy in hepatic patients with portal hypertension.

The current study aims to determine the incidence of immediate (intraoperative) and delayed (within 30 days) post-polypectomy bleeding among cirrhotic patients with esophageal varices and portal hypertension and the associated risk factors.

Methodology

This study was done in the internal medicine department of Zagazig University Hospital and the GIT department of Suez Canal University Hospital in Ismailia, Egypt

Starting from February 2021, after approval of the proposal from the IRB at Zagazig University with IRB No. (ZU-IRB #6857) and consent assignment from participants, we performed a prospective study of patients with liver cirrhosis and esophageal varices who presented with acute GIT bleeding or, during follow-up endoscopy, underwent intervention to control GIT bleeding and polypectomy simultaneously at the same session. We reported the incidence of immediate (intraoperative) and delayed (within 30 days) follow-up post-procedure in these patients. Also, we studied the associated risk factors of polypectomy-related bleeding. Immediate (intraoperative) post-polypectomy bleeding is defined as developing bleeding from a polypectomy site during the endoscopic procedure. Delayed post-polypectomy bleeding is defined as the occurrence of GIT bleeding within 30 days of the endoscopic procedure [15].

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The study included thirty-nine patients with LC with portal hypertension and varices presenting with GIT bleeding that had upper GI polyps during intervention for control bleeding or follow-up eradication of OV; in some cases, the polyps showed signs of bleeding or had a risk of bleeding.

All patients with LC and OV that had upper GI polyps in age > 18 years of both sexes presented with GIT bleeding that had upper GI polyps during intervention for control bleeding or follow-up for variceal eradication were included in this study. With informed consent from the patients included in the study, we excluded patients who refused to participate and those who were vitally unstable.

Tools and instruments:

The patient's laboratory workup, CBC, liver function tests, Creatinine, viral hepatitis markers (HCV Ab and HBs Ag), and blood glucose concentration were assessed by biochemical laboratory methods, abdominal ultrasound to determine liver status, esophagogastroduodenoscopy for upper GI to control variceal bleeding, and polypectomy in patients in whom polyps were discovered.

Operational design:

The entire history, considering age, sex, the etiology of liver disease, GIT symptoms, and medications.

Laboratory findings such as CBC, serum albumin, total bilirubin, prothrombin time, platelet counts, viral markers (HCV Ab and HBs Ag), and Histopathological examination of GI polyps that were removed by polypectomy or biopsy from the polyp Child-Pugh (CP) scores, number,

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size, and location of the polyps, underlying diseases (diabetes mellitus, hypertension, dyslipidemia, coronary heart disease, cerebrovascular disease, and chronic kidney disease), and whether the patient was taking concomitant antiplatelet and anticoagulant medications. Patients who received transfusions of fresh frozen plasma or platelet concentrates for coagulopathy before the procedures were included.

Generally, patients taking antiplatelet or anticoagulant agents were advised to hold the medication as follows: aspirin for seven days, clopidogrel for five days, heparin for 6 hours, low molecular weight heparin for 12 hours, warfarin for 3 to 5 days, and a new oral anticoagulant for 1 to 2 days before the procedures. Liver cirrhosis severity was classified into Child-Pugh classes A to C based on each patient's CP score (CP-A, 5–6 points; CP-B, 7–9 points; CP-C, 10–15 points).

General and Local examination: with special consideration of vital signs, oxygen saturation, and manifestations of liver cell failure.

Steps of performance and techniques: either snare polypectomy or endoscopic mucosal resection (including injection-assisted, cap-assisted, and ligation-assisted plans) according to the medical situation. In patients with small solitary polyps, either biopsy samples were obtained or polypectomy was performed so that the polyp was examined microscopically for histologic characterization, and most polyps were discarded after EMR by cap- or BL-assisted techniques.

Statistical analysis:

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The organization, tabulation, presentation, and data analysis were performed using SPSS IBM Chicago, version 23. Qualitative data were divided into categories and presented as frequency numbers and percentages, with the chi-square test used to determine the relationship between groups. Quantitative data were presented as mean \pm SD, and the relationship between groups was done using an independent student t-test. The level of significance adopted was $p > 0.05$.

Results

Table (1) Baseline data of the studied patients:

Patients	N=39	%
Age (year)	56.18 \pm 5.89	47 – 70
Male sex	19	48.7%
Presenting symptoms:		
Melena	15	38.5%
Hematemesis	19	48.7%
Follow up	5	12.8%
CPS:		
A	20	51.3%
B	15	38.5%
C	4	10.3%
OV:		
Grade I	21	53.8%
Grade II	12	30.8%
Grade III-IV	6	15.4%
Ascites:		
Absent	24	61.5%

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Mild	10	25.6%
Moderate	5	12.8%
Comorbidities:		
DM	8	20.5%
Hypertension	12	30.8%
Both	7	17.9%
Absent	12	30.8%
U/S:		
Cirrhotic liver	39	100%
Splenomegaly	10	25.6%

Table 1 shows the essential characteristics of the study populations. This study included thirty-nine patients ranging in age from 47 to 70 years with a mean age of 56.18 years; 48.7% were males; 87.2% presented with GIT bleeding; and 12.8% presented for follow-up. About 49% were children with child- Pugh B and C; all the patients had OV and portal hypertensive gastropathy; about 46% had OV \geq grade II; % had ascites; and 69.2% had associated comorbidities.

Table (2): baseline characters of polyps.

Polyp characters	N=39	%
Site of polyp		
Proximal	19	48.7%
Distal	13	33.3%
Mixed	7	17.9%
Shape of polyps		
Sessile	21	53.8%

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Pedunculated	18	46.2%
Polyp size ≥ 10 mm	10	25.6%
Polypectomy method:		
EMR	17	56.4%
Snare	15	43.6%

Most detected polyps are small (10 mm), 48.7% were present in the proximal part of the stomach, 53.8% were sessile, and most were removed with EMR (cap-assisted technique) during a single EMR session. Most of the detected polyps were either inflammatory or hyperplastic polyps.

Table (3): Incidence of post-polypectomy bleeding (early and late)

Bleeding	Yes	%
Early bleeding:		
No	27	69.2%
Mild	12	30.8%
Delayed bleeding		
No	0	0%

Early bleeding occurred in twelve patients (30.8%) of the studied patients, and this bleeding was mild and easily controlled endoscopically; there was no need for a blood transfusion or extra admission. Follow-up patients for 30 days following polypectomy showed no delayed attacks of bleeding.

Table (4) baseline laboratory results of studied patients.

Laboratory	Mean \pm SD	Range
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Hemoglobin (g/dl)	10.21 ± 1.1	8.1 – 12.4
Platelet count	124.0 ± 56.7	50 – 263
INR	1.24 ± 0.23	0.9 – 1.9
S albumin (g/dl)	3.61 ± 0.57	2.1 – 4.6
Total bilirubin (mg/dl)	1.47 ± 0.44	1.1 – 2.8
AST (U/L)	34.99 ± 13.56	12 – 85
ALT (U/L)	35.87 ± 16.66	13.9 – 104
S creatinine (mg/dl)	1.01 ± 0.18	0.67 – 1.4

Table (5) Relation between the incidence of early bleeding and the studied parameters

Parameter	Early bleeding		χ^2	P value	COR (95% CI)
	present N=12 (%)	Absent N= 27 (%)			
Male sex	7 (58.3%)	13 (48.1%)	0.345	0.557	1.51(0.38 – 5.96)
Age (≥ 65 years)	7 (58.3%)	4 (14.8%)	Fisher	0.017*	8.1(1.7 – 38.44)
Child-pugh:					
A	5 (41.7%)	16 (59.3%)	6.794	0.009*	1
B	3 (25%)	10 (37%)			0.96 (0.19 – 4.92)
C	4 (33.3%)	1 (3.7%)			12.8(1.15 – 142.6)
OV:					
I	3 (25%)	18 (66.7%)			1
II	4 (33.3%)	7 (25.9%)	7.921	0.005*	3.43(0.61 – 19.4)
III	5 (41.7%)	2 (7.4%)			15(1.94 – 115.97)
Ascites:					
Absent	6 (50%)	18 (66.7%)	3.427	0.06	1
Mild	2 (16.7%)	8 (29.6%)			0.75(0.12 – 4.56)
Moderate	4 (33.3%)	1 (3.7%)			12 (1.11 – 129.42)
Polyp size ≥ 10 mm	5 (41.7%)	5 (18.5%)	2.335	0.127	3.14(0.7 – 14.13)
Platelet ≤ 50	5 (41.7%)	2 (7.4%)	Fisher	0.02*	15(1.94 – 115.96)
Site (lower):					
Proximal	4 (33.3%)	15 (55.6%)			

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Middle	4 (33.3%)	9 (33.3%)	2.795	0.095	1
Distal	4 (33.3%)	3 (11.1%)			1.67(0.33 – 8.37)
					5 (0.78 – 32.1)
Type					
Sessile	6 (50%)	15 (55.6%)	0.103	0.748	
Pedunculated	6 (50%)	12 (44.4%)			1.25(0.32 – 4.88)
Operation:					
EMR	5 (41.7%)	12 (44.4%)	0.026	0.872	
Snare polypectomy	7 (58.3%)	15 (55.6%)			1.12(0.28 – 4.43)
Comorbidities:					
DM	4 (33.3%)	5 (18.5%)			4 (0.54 – 29.81)
Hypertension	4 (33.3%)	4 (14.8%)	MC	0.487	5 (0.64 – 39.06)
Both	2 (16.7%)	5 (18.9%)			2 (0.21 – 18.69)
Absent	2 (16.7%)	10 (37%)			1 (reference)

Table (6) Relationship between the incidence of early bleeding and the studied parameters

Laboratory results	Early bleeding		T	P value
	Present	Absent		
	N=12 (%)	N= 27 (%)		
Hemoglobin (gm/dl)	9.44 ± 1.13	10.55 ± 0.92	3.326	0.003*
INR	1.3 ± 0.29	1.21 ± 0.19	-0.954	0.555
Albumin (gm/dl)	3.61 ± 0.75	3.61 ± 0.48	0.037	0.971
Bilirubin (mg/dl)	1.51 ± 0.4	1.46 ± 0.47	-0.315	0.754
Creatinine (mg/dl)	1.03 ± 0.25	1.01 ± 0.15	-0.259	0.799
AST (U/L)	28.5(18.75 – 43)	33(31 – 42)	-1.147	0.251
ALT (U/L)	24(19 – 34.75)	35(29 – 45)	-2.315	A0.021*

‡ data are expressed as median (interquartile range) and compared using Mann Whitney test *p<0.05 is a statistically significant t-independent sample t-test χ^2 Chi-square test. MC Monte Carlo test.

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Tables 5 and 6 show the relationship between the incidence of early bleeding and the studied parameters.

Advanced child score, size of the present OV, thrombocytopenia, anemia, and low ALT level. Child C, OV grade III, age ≥ 65 years, had a platelet count of 50 000, significantly increasing the risk of bleeding by 12.8, 15, 8.1, and 15 folds, respectively.

Table (7) Multivariate analysis of factors significantly associated with early bleeding among the studied patients:

	β	p	AOR	95% C.I.	
				Lower	Upper
Platelet	-0.053	0.0003*	0.948	0.916	0.982
ALT (U/L)	-0.086	0.08	0.918	0.834	1.01

AOR adjusted odds ratio CI confidence interval.

Increased platelets count independently decreased the risk of early bleeding among the studied patients (Table 7).

Discussion

Little information is available regarding the feasibility and safety of gastric EMR in hepatic patients with advanced liver cirrhosis, portal hypertension, and varices. Patients with liver cirrhosis have a high probability and risk of bleeding during interventional procedures such as endoscopic polypectomy due to multiple factors. Our study was performed to evaluate the early and late incidence and risk factors of post-gastric polypectomy bleeding in cirrhotic patients with portal hypertension and esophageal varices.

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This study included thirty-nine cirrhotic patients with portal hypertension and esophageal varices who presented with acute GIT bleeding or for follow-up band ligations of both sexes and different child scores and esophageal varices.

Within the study population, 30.8% of people had IPPB. The bleeding was not severe. Oozing from the polypectomy site could be managed endoscopically without blood transfusions or additional hospitalization. At the same time, the patients were observed and monitored for 30 days following the procedure with no symptoms or signs of DPPB. Despite this high percentage of the population having IPPB, the outcome was good, with no mortality or significant complications.

In a retrospective study conducted by Huang et al. in colonic polypectomy in cirrhotic patients, Immediate bleeding presented in 7.5% of patients, and delayed bleeding occurred in 0.3% of patients; however, all cases of immediate bleeding were mild and controlled endoscopically, and none resulted in significant consequences. This difference might be due to increased portal pressure in the stomach more than in the colon in patients with portal hypertension [16]. This statement matched our results, which showed that most IPPB attacks were mild, self-limited, and easily controlled endoscopically, with no increase in morbidity or mortality.

The study conducted by Badar Hassan found that colonic EMR in cirrhotic patients has an acceptable bleeding risk. Immediate and delayed bleeding rates were 9.2% and 5.8%, respectively [17].

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PPB was shown to occur in 7.5% of CLD patients, and patients in CPS classes B and C had more excellent rates of both IPPB and DPPB, according to SOH et al. [18].

About 14% of cirrhotic individuals had DPPB in a colon polyp, according to a study by Lee et al. [19].

According to a cohort study of elective endoscopy in cirrhotic patients, which revealed a 2% incidence of post-polypectomy bleeding in colon polypectomy, an elective procedure and good pre-procedural preparations of plasma, blood, or platelet transfusion in some cases may account for this difference [20].

According to Kwon et al., the study found no significant difference in the incidence of perforation between the CRF, LC, and control groups. However, immediate bleeding tended to occur more frequently in the CRF + LC group than in the controls. [19]. Patients who bled in this study were statistically older than non-bleeders ($P = 0.017$), and there was no statistical significance about the sex of the patients [21].

In contrast, Kundumadam et al. found that patients who bled after polypectomy were younger than those who did not bleed [20].

About half of the patients in this study had Child-Pugh B and C, and about 46% had large-sized OV. 38.5% of patients had ascites. There is a statistically significant relationship between early post-polypectomy bleeding and CPS, OV grades, and marginally substantial with ascites ($p = 0.06$), and these parameters indicate advanced liver disease, so the

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incidence of PPB was significantly increased with the stage of advanced liver disease.

The results of the study conducted by Lee et al. This showed that the risk of IPPB in the colon was statistically significantly higher in cirrhotic patients with Child-Pugh B and C than in the Child-Pugh A and control no cirrhotic group, which matched our results. These findings cause us to be extremely careful when performing polypectomy on patients with advanced cirrhosis [19].

Another study conducted by Soh et al. to evaluate the risk of post-polypectomy bleeding of colon polyps in cirrhotic patients showed that PPB was statistically higher in patients with Child-Pugh B and C than in patients with Child-Pugh A and chronic hepatitis (p 0.001) [18].

Huang et al. found that PPB in cirrhotic patients increased significantly with advanced liver disease and advanced CPS, where patients with CPS child C had a higher incidence of PPB than patients with child A or B [20]. Kundumadam observed that the risk of bleeding increased with the degree of advanced liver disease and high Child-Pugh, which matched our results [20].

Patients in study groups experienced mild to moderate ascites in about 38.5% of cases. The degree of ascites was statistically higher in patients with a history of IPPB (p = 0.06), which might be explained by the degree of advanced liver disease and CPS.

Huang et al. found that mild to moderate ascites predicted the IPPB [22].

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All these results indicate that PPB increases significantly with advanced liver disease, but all showed that bleeding was mild, insignificant, and easily controlled with endoscopic interventions.

Nearly about half of the gastric polyps that were discovered in this study were proximally located in the stomach; 53.8% of polyps were sessile; the majority of polyps were small (<10 mm in size); most polyps are small sized; and they were removed with cap assisted EMR, which is considered a relatively safe technique with good local hemostasis. This resulted in no statistical difference between patients with or without bleeding regarding the polyp site, size, or morphology ($p = 0.095$, 0.127 , and 0.748 , respectively) or the technique of polyp removal ($p = 0.872$).

Huang et al. found no statistical significance between patients with or without bleeding regarding polyp size, site, or morphology. Still, bleeding was common in polyps removed with the cold snare technique [22]. Kundumadam noticed that there was no statistical difference regarding the size of the polyp (10 mm vs. ≤ 10 mm) and the incidence of bleeding [20]. A statistically significant relationship exists between early bleeding and hemoglobin levels and ALT (both were lower in patients who developed bleeding).

According to research by Lee et al., IPPB was not significantly affected by patient laboratory findings such as hemoglobin level, total bilirubin level, albumin level, alanine aminotransferase level, platelet count, and prothrombin time [19].

In this study, the platelet count in the study population ranged between 46 and 263×10^9 (with a mean of $124.0 \pm 56.7 \times 10^9$), and a significant

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association was found between the severity of thrombocytopenia (50×10^9) and the risk of IPPB ($p = 0.02$).

In this study, we noticed that cirrhotic patients with portal hypertension who had IPPB also had significantly lower platelet counts than those who did not ($P = 0.02$), had more portal hypertension and large varices compared to patients without IPPB ($P = 0.005$), and also had relatively significant moderate ascites ($p = 0.06$).

Huang et al. found that the bleeding risk following polypectomy in cirrhotic patients was significantly increased with low platelet counts, increased ascites, and the presence of OV, which matched our results [17]. According to Kundumadam, 29% of patients who bled had a platelet count of $50 \times 10^3 /\text{mm}^3$, compared to 6.3% of those who did not bleed, even though there was no statistically significant difference in platelet counts between patients with or without bleeding after polypectomy (mean 85 vs. 117; $P = 0.17$). ($P = 0.08$) [18]. Also, SOH et al. found that platelet counts less than 50×10^3 were more vulnerable to PPB in cirrhotic patients [18].

In this study, PPB in cirrhotic patients did not significantly increase in patients receiving antiparticles ($p = 0.127$).

No noticeable difference was observed between patients with or without PPB regarding anti-platelets. Kundumadam noticed that antiplatelet and anticoagulant medications were not linked to bleeding, but his study contained only a small number (15 patients) receiving anticoagulants [20].

Increased platelet count and ALT independently decreased the risk of early bleeding among the studied patients.

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All polyps are small and have a small sample size; not all removed polyps are pathologically examined as we used snare or EMR techniques with discarded polyps, and most polyps were removed with cold snare or cap-assisted EMR, which is considered a relatively safe technique, and no ESD technique is used.

Footnotes.

Peer-Reviewers: Emad Hamed (professor of internal medicine), Mohamed Emara (professor of tropical medicine), Samah Soliman (professor of tropical medicine), and Amr Shaban Hanafy Abdallah (professor of internal medicine)., Nevin Fouad (professor of internal medicine).

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Data availability

All the data obtained and analyzed are included in this manuscript.

Conflicts of interest

The authors declare that they have no competing interests.

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Authors' contributions

Bassam Mansour Salama and Amir Abd-Elhameed Ahmed Barakat conceived and supervised the work. Mahmoud Ahmed Sharafeddin and Ahmed Ibrahim Gad planned and conducted the experiments. All authors analyzed the data. All authors wrote the manuscript. All authors read and approved the final manuscript.

Conclusions

Gastric polypectomy during endoscopy in cirrhotic patients with portal hypertension and esophageal varices who are presented with GIT bleeding is considered a safe maneuver if done during an endoscopic session to control bleeding or during a follow-up session. We recommend increasing

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the sample size and using another method of polypectomy, such as ESD or hot snare polypectomy.

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