

Long-Term Outcomes of Combined Endoscopic Ligation and Partial Splenic Artery Embolization for Gastroesophageal Variceal Bleeding in Patients with Cirrhosis: A Comparative Cohort Study

Umarov Zafarbek¹, Allanazarov Allanazar², Sabirova Sevara³

¹²³ Department of Surgical Diseases and Transplantology, Urganch State Medical Institute, Urganch, Uzbekistan

doctor_uzz186@mail.ru

Abstract: Background: Gastroesophageal variceal bleeding is a major complication of cirrhosis and clinically significant portal hypertension and is associated with recurrent hemorrhage, hepatic decompensation, and death. Endoscopic ligation provides direct local treatment of esophageal varices but does not independently correct the portal hypertensive hemodynamic environment responsible for recurrent variceal formation and bleeding. Partial splenic artery embolization may reduce splenic arterial inflow and influence portal venous hemodynamics. This study evaluated the long-term outcomes of combined endoscopic ligation and partial splenic artery embolization compared with endoscopic ligation alone. Methods: A comparative clinical cohort comprising 171 patients with cirrhosis complicated by gastroesophageal variceal bleeding was evaluated. Patients underwent endoscopic ligation alone or combined endoscopic ligation and partial splenic artery embolization. The primary outcome was recurrent gastroesophageal variceal bleeding during long-term follow-up. Cumulative rebleeding rates at 1, 3, and 5 years were compared. Absolute risk reduction, relative risk, relative risk reduction, and approximate number needed to treat were calculated from the reported cumulative event rates. Results: Combined treatment was associated with consistently lower cumulative rebleeding. At 1 year, rebleeding occurred in 29.3% after endoscopic ligation alone and 8.5% after combined treatment (absolute risk reduction 20.8 percentage points; relative risk 0.29). At 3 years, rates were 41.4% and 18.6%, respectively (absolute risk reduction 22.8 percentage points; relative risk 0.45). At 5 years, rates were 50.0% and 25.4%, respectively (absolute risk reduction 24.6 percentage points; relative risk 0.51). The approximate number needed to treat ranged from 4 to 5 patients. Conclusions: Combined endoscopic ligation and partial splenic artery embolization was associated with lower long-term cumulative rebleeding than endoscopic ligation alone. The complementary strategy of direct variceal treatment combined with modification of splenic inflow may provide more durable control of recurrent gastroesophageal variceal bleeding. Prospective studies with complete patient-level data and formal time-to-event analyses are warranted.

Keywords: cirrhosis; portal hypertension; gastroesophageal variceal bleeding; endoscopic ligation; partial splenic artery embolization; rebleeding; endovascular treatment.

Introduction

Cirrhosis is a leading cause of chronic liver disease morbidity and mortality worldwide, and its major complications, including portal hypertension and variceal hemorrhage, account for a substantial share of liver-related hospitalizations and deaths [1]. Portal hypertension is a major pathophysiological consequence of advanced chronic liver disease and cirrhosis. Increased intrahepatic vascular resistance together with increased splanchnic blood flow promotes the formation of portosystemic collaterals, including esophageal and gastric varices. Variceal hemorrhage remains one of the most dangerous

manifestations of portal hypertension and may precipitate recurrent bleeding, hospitalization, hepatic decompensation, and death [2–7].

Portal hypertension is not solely a structural consequence of fibrosis. Progressive architectural distortion, nodular regeneration, endothelial dysfunction, and dynamic changes in the hepatic and splanchnic circulation contribute to increased portal pressure [2–3,7–10]. The resulting collateral circulation can enlarge over time, while increased wall tension and variceal vulnerability increase the probability of rupture [4–6].

Modern management of acute and recurrent variceal bleeding is multidisciplinary. Pharmacological therapy, endoscopic treatment, non-selective beta-blockade [11–12], radiological interventions, and portal decompressive procedures are used according to clinical presentation and risk [2–6,13–14]. Endoscopic variceal ligation is a cornerstone of treatment because it directly eradicates or reduces high-risk esophageal varices [4–6,15]. Nevertheless, recurrent bleeding remains clinically important because local variceal eradication does not necessarily normalize the portal hypertensive circulation.

In selected high-risk patients, transjugular intrahepatic portosystemic shunt can provide effective portal decompression and reduce treatment failure and recurrent bleeding [2–3,16]. However, TIPS is not appropriate for every patient because treatment decisions depend on hepatic functional reserve, anatomy, comorbidity, technical feasibility, and the potential for complications, including hepatic encephalopathy.

Partial splenic artery embolization is an endovascular technique intended to reduce splenic arterial perfusion. By decreasing splenic inflow, PSAE may reduce the splenic venous contribution to the portal system and thereby influence portal hemodynamics [17–18]. Published experience has also suggested potential benefits in selected patients with portal hypertension and hypersplenism, although procedural complications and patient selection require careful consideration.

The combination of EL and PSAE therefore has a clear mechanistic rationale. EL acts directly on the variceal source of bleeding, whereas PSAE may influence a hemodynamic component of portal venous inflow. The present study examined whether this combined strategy was associated with improved long-term control of recurrent gastroesophageal variceal bleeding compared with EL alone. The primary objective was long-term cumulative rebleeding; secondary clinical observations concerned liver function stability and progression toward decompensated cirrhosis.

Materials and Methods

1. Study design and population

This comparative clinical cohort study evaluated patients with cirrhosis complicated by gastroesophageal variceal bleeding. According to the available clinical documentation, the total study cohort included 171 patients. Patients underwent one of two therapeutic strategies: endoscopic ligation alone or combined endoscopic ligation and partial splenic artery embolization.

The currently available source documentation did not provide the exact study period, complete allocation of all 171 patients between treatment strategies, or a full patient-level baseline dataset. These variables were therefore not reconstructed, estimated, or artificially generated. The present manuscript reports and analyzes only information supported by the available study materials.

2. Clinical assessment

Patients were clinically evaluated for cirrhosis, portal hypertension, and gastroesophageal variceal bleeding. Endoscopic examination was used to identify gastroesophageal varices and to provide endoscopic treatment. For patients undergoing endovascular treatment, relevant vascular anatomy was assessed to support planning of partial splenic artery embolization.

3. Endoscopic ligation

Endoscopic ligation was used as the direct treatment modality for gastroesophageal varices. The objective of the procedure was mechanical ligation of the variceal vessels to control the bleeding source and reduce the risk of recurrent hemorrhage. EL was the reference treatment strategy for the comparative analysis.

4. Partial splenic artery embolization

In the combined-treatment strategy, PSAE was performed as an endovascular adjunct to EL. The therapeutic rationale was reduction of splenic arterial inflow with a potential downstream effect on

splenic venous contribution to the portal circulation. The splenic artery and its relationship to the celiac and portal circulations were relevant to procedural planning.

5. Outcomes and follow-up

The primary outcome was recurrent gastroesophageal variceal bleeding during long-term follow-up. Cumulative rebleeding was reported at 1, 3, and 5 years. Secondary clinical observations in the source documentation included liver function stability and progression toward decompensated cirrhosis; numerical values for these secondary outcomes were not available.

6. Statistical analysis

Because complete patient-level counts at each follow-up point were not available, descriptive comparative measures were calculated from the reported cumulative event percentages. Absolute risk reduction (ARR) was calculated as risk in the EL group minus risk in the EL+PSAE group. Relative risk (RR) was calculated as risk in the EL+PSAE group divided by risk in the EL group. Relative risk reduction (RRR) was calculated as $1 - \text{RR}$. Approximate number needed to treat (NNT) was calculated as $1/\text{ARR}$, with ARR expressed as a proportion.

Exact confidence intervals, P values, hazard ratios, Kaplan–Meier curves, and log-rank testing were not calculated because the exact number of patients at risk and event counts at each follow-up time point were not available. All calculated comparative measures should therefore be interpreted as descriptive estimates derived from the reported cumulative percentages.

Results

1. Study cohort

The available clinical documentation reported a total cohort of 171 patients with cirrhosis and gastroesophageal variceal bleeding. The source material also contained a figure label identifying EL (n=58) and EL+PSAE (n=59). Because these figures do not account for the complete cohort of 171 patients, missing denominators were not inferred. Comparative calculations were based exclusively on the reported cumulative rebleeding percentages.

2. One-year outcome

At 1-year, cumulative gastroesophageal variceal rebleeding was reported in 29.3% of patients treated with EL alone and 8.5% of patients treated with combined EL+PSAE. The corresponding ARR was 20.8 percentage points. The RR was 0.29, indicating that the reported cumulative risk in the combined-treatment group was approximately 29% of that observed with EL alone. The RRR was 71.0%, and the approximate NNT was 4.8, corresponding to approximately 5 patients.

3. Three-year outcome

At 3 years, cumulative rebleeding was 41.4% in the EL-only group and 18.6% in the combined-treatment group. The ARR was 22.8 percentage points, the RR was 0.45, and the RRR was 55.1%. The approximate NNT was 4.4, corresponding to approximately 4 patients.

4. Five-year outcome

At 5 years, cumulative rebleeding reached 50.0% among patients treated with EL alone compared with 25.4% among patients treated with EL+PSAE. The ARR was 24.6 percentage points. The RR was 0.51, the RRR was 49.2%, and the approximate NNT was 4.1, corresponding to approximately 4 patients.

3.5. Summary of comparative effect estimates

Table 1. Long-term cumulative rebleeding and descriptive comparative measures

Follow-up	EL alone	EL+PSAE	ARR	RR	RRR	Approx. NNT
1 year	29.3%	8.5%	20.8%	0.29	71.0%	5
3 years	41.4%	18.6%	22.8%	0.45	55.1%	4
5 years	50.0%	25.4%	24.6%	0.51	49.2%	4

ARR, absolute risk reduction; RR, relative risk; RRR, relative risk reduction; NNT, number needed to treat.

6. Long-term clinical observations

The available clinical documentation indicated more favorable long-term clinical observations among patients receiving combined EL+PSAE, including better liver function stability and less progression toward decompensated cirrhosis. Because numerical measurements for these outcomes were not available, quantitative comparisons and effect estimates were not performed.

Discussion

The principal finding of this comparative cohort was a consistent association between combined EL+PSAE and lower cumulative gastroesophageal variceal rebleeding throughout long-term follow-up. The absolute difference in cumulative rebleeding was already evident at 1 year and remained present at 3 and 5 years.

At 1 year, the difference between strategies was 20.8 percentage points; at 3 years it was 22.8 percentage points; and at 5 years it was 24.6 percentage points. These estimates suggest that the observed separation between treatment strategies was not confined to the early post-treatment period. The combined strategy remained associated with a lower cumulative burden of recurrent bleeding across all reported follow-up intervals.

The biological rationale for this observation is plausible. EL directly addresses the immediate variceal lesion by mechanical ligation. PSAE, in contrast, is intended to modify splenic inflow and may influence portal venous hemodynamics [17–18]. The two procedures therefore target different components of the disease process: the local manifestation of portal hypertension and a component of the circulatory environment that contributes to portal venous inflow.

These findings should be interpreted within the broader therapeutic landscape. Contemporary management of portal hypertensive bleeding emphasizes risk stratification and integration of pharmacological and endoscopic therapy, with portal decompressive interventions such as TIPS reserved for appropriately selected circumstances [2–6,16]. The potential attraction of EL+PSAE is that it may provide an additional hemodynamic intervention without relying exclusively on a portosystemic shunt.

The calculated relative risks were 0.29, 0.45, and 0.51 at 1, 3, and 5 years, respectively. These are descriptive calculations rather than adjusted estimates. Nevertheless, the consistency of the direction of effect across follow-up intervals supports the clinical relevance of the observed pattern. The calculated NNT of approximately four to five patients also provides an intuitive estimate of potential clinical magnitude, although it must not be interpreted as a definitive causal treatment effect in the absence of complete denominator and event-level data.

The source documentation additionally suggested better liver function stability and less progression toward decompensated cirrhosis in the combined-treatment group. This observation is clinically important because recurrent bleeding may occur in parallel with progressive liver disease. However, Child–Pugh class, MELD score, bilirubin, albumin, coagulation parameters, ascites, encephalopathy, and other standardized indicators were not available as numerical data. Future studies should prospectively collect these measures to determine whether improved bleeding control is accompanied by measurable differences in hepatic decompensation.

The study has important limitations. First, complete patient-level baseline characteristics were not available, preventing assessment of comparability between treatment groups and adjustment for confounding. Second, the total cohort was reported as 171 patients, whereas a figure identified subsets of 58 and 59 patients; because the remaining allocation was not documented, no missing values were invented. Third, exact numbers at risk at each follow-up point were unavailable, preventing confidence interval calculation and formal survival analysis. Finally, procedural details such as embolization extent and standardized technical parameters were not available in the source material.

These limitations reduce the ability to establish causality but do not negate the descriptive value of the observed long-term pattern. A future prospective comparative study should include predefined eligibility criteria, baseline liver disease severity, variceal characteristics, standardized procedural reporting, adverse events, complete follow-up denominators, and Kaplan–Meier analysis. Multivariable or propensity-adjusted analyses would further improve assessment of treatment effects.

Overall, the present findings support continued investigation of combined EL+PSAE as a strategy for selected patients with portal hypertensive gastroesophageal variceal bleeding. The combination is mechanistically complementary and, in the available cohort, was associated with a substantially lower cumulative rebleeding burden.

Conclusion

In this comparative clinical cohort, combined endoscopic ligation and partial splenic artery embolization was associated with substantially lower cumulative gastroesophageal variceal rebleeding

than endoscopic ligation alone. The absolute reduction in recurrent bleeding was 20.8 percentage points at 1 year, 22.8 percentage points at 3 years, and 24.6 percentage points at 5 years. The findings suggest that combining direct endoscopic treatment with modification of splenic inflow may provide more durable long-term control of recurrent bleeding. Prospective studies with complete patient-level data, standardized baseline reporting, and formal time-to-event analysis are needed to confirm these findings.

References

- [1] E. A. Tsochatzis, J. Bosch, and A. K. Burroughs, "Liver cirrhosis," *Lancet*, vol. 383, no. 9930, pp. 1749–1761, 2014.
- [2] R. de Franchis, J. Bosch, G. Garcia-Tsao, T. Reiberger, C. Ripoll, and Baveno VII Faculty, "Baveno VII—Renewing consensus in portal hypertension," *J. Hepatol.*, vol. 76, no. 4, pp. 959–974, 2022.
- [3] D. E. Kaplan, C. Ripoll, M. Thiele, B. E. Fortune, D. A. Simonetto, G. Garcia-Tsao, and J. Bosch, "AASLD Practice Guidance on risk stratification and management of portal hypertension and varices in cirrhosis," *Hepatology*, 2024.
- [4] G. Garcia-Tsao, J. G. Abraldes, A. Berzigotti, and J. Bosch, "Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management," *Am. J. Gastroenterol.*, vol. 112, no. 3, pp. 488–497, 2017.
- [5] G. Garcia-Tsao and J. Bosch, "Management of varices and variceal hemorrhage in cirrhosis," *N. Engl. J. Med.*, vol. 362, pp. 823–832, 2010.
- [6] D. Tripathi, A. J. Stanley, P. C. Hayes, D. Patch, C. Millson, H. Mehrzad, et al., "UK guidelines on the management of variceal haemorrhage in cirrhotic patients," *Gut*, vol. 64, no. 11, pp. 1680–1704, 2015.
- [7] J. Bosch, R. J. Groszmann, and V. H. Shah, "Evolution in the understanding of the pathophysiological basis of portal hypertension," *J. Hepatol.*, vol. 62, no. 1, pp. S121–S130, 2015.
- [8] J. G. Abraldes, C. Villanueva, R. Bañares, C. Aracil, M. V. Catalina, A. P. Garci, et al., "Hepatic venous pressure gradient and prognosis in patients with acute variceal bleeding treated with pharmacologic and endoscopic therapy," *J. Hepatol.*, vol. 48, no. 2, pp. 229–236, 2008.
- [9] J. Bosch, J. G. Abraldes, A. Berzigotti, and J. C. Garcia-Pagán, "The clinical use of HVPG measurement in chronic liver disease," *Nat. Rev. Gastroenterol. Hepatol.*, vol. 6, no. 10, pp. 573–582, 2009.
- [10] R. J. Groszmann, J. Bosch, N. D. Grace, H. O. Conn, G. Garcia-Tsao, M. Navasa, et al., "Hemodynamic events in a prospective randomized trial of propranolol versus placebo in the prevention of a first variceal hemorrhage," *Gastroenterology*, vol. 99, no. 5, pp. 1401–1407, 1990.
- [11] C. Villanueva, A. Albillos, J. Genescà, J. C. Garcia-Pagán, J. L. Calleja, C. Aracil, et al., "Development of hyperdynamic circulation and response to beta-blockers in portal hypertension," *Hepatology*, vol. 63, no. 1, pp. 197–206, 2016.
- [12] T. Reiberger and M. Mandorfer, "Beta adrenergic blockade and decompensated cirrhosis," *J. Hepatol.*, vol. 66, no. 4, pp. 849–859, 2017.
- [13] M. Sh. Khakimov, U. I. Matkuliev, D. Y. Batirov, and Z. Z. Umarov, "Modern treatment and prevention of bleeding from esophageal and gastric varicose veins with portal hypertension: Review of literature," *Amer. J. Med. Med. Sci.*, vol. 13, no. 5, pp. 762–767, 2023.
- [14] Z. Umarov et al., "Analysis of angiographic examination and endovascular intervention methods in patients with varicose veins of the esophagus and stomach," *Amer. J. Med. Med. Sci.*, vol. 14, no. 10, pp. 2611–2615, 2024.
- [15] I. L. Holster and E. T. Tjwa, "Endoscopic treatment of esophageal varices," *Gastroenterol. Clin. North Am.*, vol. 43, no. 4, pp. 807–818, 2014.
- [16] J. C. García-Pagán, K. Caca, C. Bureau, W. Laleman, B. Appenrodt, A. Luca, et al., "Early use of TIPS in patients with cirrhosis and variceal bleeding," *N. Engl. J. Med.*, vol. 362,

no. 25, pp. 2370–2379, 2010.

- [17] K. G. Koconis, H. Singh, and G. Soares, "Partial splenic embolization in the treatment of patients with portal hypertension: A review of the English language literature," *J. Vasc. Interv. Radiol.*, vol. 18, no. 4, pp. 463–481, 2007.
- [18] V. Pavel, G. Scharf, P. Mester, et al., "Partial splenic embolization as a rescue and emergency treatment for portal hypertension and gastroesophageal variceal hemorrhage," *BMC Gastroenterol.*, 2023