# Recurrence of esophageal varices after endoscopic band ligation: single centre experience

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<sup>2</sup> Clinic of Gastroenterology, Nephrourology and Surgery, Medical Faculty, Vilnius University, Vilnius, Lithuania **Background and objective.** Endoscopic band ligation is the main endoscopic treatment for esophageal varices, but the main problem after endoscopic treatment is variceal recurrence. The aim of this study was to evaluate and determine the esophageal varices recurrence rate and the time interval after endoscopic band ligation and to investigate possible risk factors affecting recurrence.

Material and methods. The retrospective analysis of endoscopic band ligation procedures, performed in Vilnius University Hospital Santariskiu Clinics during the period 2006–2010, was made. 133 endoscopic band ligation procedures were included in the study.

**Results.** After endoscopic band ligation, esophageal varices recurred in 45% of cases. The early recurrence of esophageal varices occurred in 46.7% of cases. Extrahepatic portal hypertension and a greater size of varices lead to the statistically significant early recurrence of esophageal varices after endoscopic band ligation.

**Conclusions.** Endoscopic band ligation is associated with a high recurrence rate of esophageal varices and half of these cases were indentified as early variceal recurrence.

Key words: endoscopic band ligation, variceal recurrence, risk factors

# INTRODUCTION

Liver cirrhosis is an end-stage liver disease of any progressing chronic liver disease leading to the loss of the liver function and the formation of portal hypertension. Portal hypertension is a syndrome of elevated pressure in the portal vein system due to the increased resistance of intrahepatic vessels and

Correspondence to: Laura Mašalaitė, Vilnius University Hospital Santariskiu Clinics, Santariskiu St. 2, Vilnius, Lithuania. E-mail: lauramasalaite@yahoo.com a higher portal inflow. A gold standard to measure portal pressure is a hepatic vein pressure gradient, the increase of which to more than 10 mmHg is clinically relevant and related to complications (1). Portal hypertension leads to the formation of porto-systemic collaterals. Esophageal varices are the most common complication of portal hypertension and are present in approximately 50% of patients with cirrhosis (2). Variceal bleeding from ruptured esophageal varices is the main and most severe complication of cirrhosis related to higher morbidity and mortality compared with other causes of bleeding. Variceal bleeding occurs in 30% of patients with cirrhosis and portal hypertension (3) and each episode has a reported mortality risk between 30% and 70% (4).

The treatment of esophageal varices requires a good outcome with no recurrence. Endoscopic band ligation (EBL) is the main endoscopic treatment for esophageal varices. However, the recurrence of varices occurs after EBL and it remains unclear why some patients experience early recurrence and others have later or no recurrence. In this article we present our single centre experience regarding variceal recurrence after EBL. The aim of the present study was the evaluation of the variceal recurrence rate and timing after EBL with the intention to investigate factors affecting recurrence.

#### MATERIAL AND METHODS

We made a retrospective analysis of all EBL procedures, performed in Vilnius University Hospital Santariskiu Clinics from January 2006 to December 2010. All EBL procedures were included, performed in patients with active variceal bleeding, for primary and secondary prophylaxis. EBL was performed using a standard endoscopic band ligation technique using one or multiband ligation devices under general anesthesia. Written, informed consent was obtained from each patient. We did not repeat EBL sessions until complete eradication was achieved, so EBL was repeated only if variceal recurrence developed or in cases of active variceal bleeding. We evaluated the size of esophageal varices (F) according to the criteria of the Japan Society for Portal Hypertension: F1, F2 and F3 (5). The recurrence of esophageal varices was determined by surveillance endoscopic findings as re-increasing in the size of varices when F was reduced after EBL and variceal bleeding between EBL sessions. We defined early recurrence as variceal recurrence within 3 months after the ligation session and late recurrence - later than 3 months. After EBL each patient underwent surveillance endoscopy every 3 months for more than a year period. We also collected demographic, clinical and laboratory data as possible risk factors affecting variceal recurrence. For the statistic analysis we used SPSS 19.0 and p-value of less than 0.05 was considered to be statistically significant.

#### RESULTS

In a 5 year period 183 EBL procedures (Figure) were performed for 118 patients: 52% males (n = 61) and 48% females (n = 57); average age 51 ± 16 years.



**Figure.** Number of EBL procedures per year in Vilnius University Hospital Santariskiu Clinics

We included 133 EBL procedures in the final analysis. The following patients were excluded from the retrospective analysis: 6 patients died, 4 were transplanted within 1 month, 6 patients underwent a vascular shunting operation, in 34 patients surveillance endoscopy after ligation was not performed and we had no further information about them. Esophageal varices developed as the complication of portal hypertension: in most cases (87.3%) esophageal varices developed due to intrahepatic portal hypertension and in other cases (12.7%) they were due to extrahepatic portal hypertension. The main indication for EBL was secondary prophylaxis (65%, n = 119), followed by primary prophylaxis (24%, n = 41) and controlling active variceal bleeding (12.6%, n = 23). After EBL, esophageal varices recurred in 45% (n = 60) cases. The early recurrence of esophageal varices occurred in 46.7% cases (n = 28) and late recurrence in 53.3% (n = 32). In the recurrence group, 73.3%of cases (n = 44), the size of varices (F) re-increased after endoscopic ligation (surveillance endoscopy) and 26.7% of cases (n = 16) developed variceal bleeding between ligation sessions. Patients were

divided into groups according to the following pattern of recurrence: recurrence group (n = 60) and no-recurrence group (n = 73). There were no significant differences between these groups according to several clinical characteristics: age, sex, the etiology of portal hypertension, Child-Pugh class, the size of varices (F) and the presence of "red sings" before ligation, indication for endoscopic ligation, endoscopic injection sclerotherapy before ligation (Table 1).

The extrahepatic portal hypertension and a greater size of varices were significantly different (p = 0.013 and p = 0.038 respectively) in early and late recurrence groups (Table 2).

#### DISCUSSION

Esophageal varices are the most common complication of liver cirrhosis and portal hypertension. The reported prevalence of esophageal varices in cirrhotic patients fluctuates within a wide range and is around 60% (ranging from 24 to 80%) (6). The prevalence of varices differs comparing compensated (30%) and decompensated (60–85%) liver cirrhosis (7, 8). Once varices have developed, they increase in size and eventually cause variceal bleeding. The study by the North Italian Endoscopy Club identified the variceal size, the degree of liver failure assessed by the Child-Pugh classification

Clinic characteristics	Reccurence group, n = 60	No-recurrence group, n = 73	p value
Sex:			
Male	n = 29 (48%)	n = 40 (54.8%)	0.46
Female	n = 31 (51.7%)	n = 33 (45.2%)	
Portal hypertension:			
Intrahepatic	n = 55 (91.7%)	n = 62 (84.9%)	0.24
Extrahepatic	n = 5 (8.3%)	n = 11 (15.1%)	
Child-Pugh class:			
А	n = 20 (32.7%)	n = 22 (30.2%)	0.95
В	n = 26 (43.6%)	n = 32 (44.4%)	
С	n = 14 (23.6%)	n = 19 (25.6%)	
Red signs:			
Yes	n = 21 (35%)	n = 24 (39.7%)	0.58
No	n = 39 (65%)	n = 49 (60.3%)	
F before EBL:			
F1	n = 3 (5%)	n = 5 (6.8%)	0.81
F2	n = 32 (53.3%)	n = 41 (56.2%)	
F3	n = 25 (41.7%)	n = 27 (37%)	
Indication:			
Primary prophylaxis	n = 10 (16.7%)	n = 20 (27.4%)	0.33
Secondary prophylaxis	n = 44 (73.3%)	n = 46 (63%)	
Acute bleeding	n = 6 (10%)	n = 7 (9.6%)	
Sclerotherapy before EBL:			
Yes	n = 9 (15%)	n = 11 (15.1%)	
No	n = 51 (85%)	n = 62 (84.9%)	0.99

Table 1. Clinical characteristics between the variceal recurrence and no-recurrence groups

Table 2. Significant differences between the early recurrence and late recurrence groups

	Early recurrence group, n = 28	Late recurrence group, $n = 32$	p value
Portal hypertension:			
Intrahepatic	n = 23 (82.1%)	n = 32 (100%)	0.013
Extrahepatic	n = 5 (17.9%)	n = 0 (0%)	
F grade before EVL:			
F1	n = 2 (7.1%)	n = 1 (3.1%)	0.038
F2	n = 9 (35.7%)	n = 22 (68.8%)	
F3	n = 15 (57.2%)	n = 9 (28.1%)	

and endoscopic "red signs" as the major independent risk factors of the first variceal bleeding episode (3). Hepatic venous pressure gradient is useful in clinical practice selecting cirrhotic patients at the highest risk of variceal bleeding because the mean hepatic venous pressure gradient in patients with large (F2-F3) varices was significantly higher than that in patients with small (F1) varices  $(17.8 \pm 4.8 \text{ mmHg vs } 14.6 \pm 4.8 \text{ mmHg},$ p = 0.007) (9). The rate of variceal size enlargement from small to large is also not well defined, with reported prevalence ranging between 8 and 31% per year (8, 10). Variceal bleeding occurs in 30% of patients with cirrhosis and portal hypertension (3, 11) and each episode has a mortality risk between 30% and 70% (12). Despite the reduction of mortality due to variceal bleeding from 42% in 1981 (13) to 15–20% at present (14, 15), the recurrence rate of esophageal varices remains high. A six-week rebleeding rate is approximately 17% and the mortality rate of an episode of esophageal variceal bleeding is 20% (14). Because of high rates of variceal recurrence, rebleeding and related mortality all cirrhotic patients should be screened for esophageal varices at different intervals according to the presence and size of the varices (16). Once large varices have developed, the patients should be treated to prevent bleeding.

After controlling the acute variceal bleeding episode, the main problem remains as follows: rebleeding and recurrence of varices after endoscopic treatment. The treatment of esophageal varices requires a good outcome with no recurrence what is very important in the long-term management of patients with portal hypertension. According to Baveno V consensus (16), there are a few treatment modalities and the choice of them depends on local endoscopic possibilities, experience and patients decision. EBL became the preferred technique of the endoscopic treatment of esophageal varices when it was proved in several randomized trials to be as effective as endoscopic sclerotherapy but with fewer serious adverse events (17). EBL is a method of the mechanical obliteration of esophageal varices and it was first introduced in 1986 by Van Stiegmann (18). At present, EBL is the main endoscopic treatment option not only for controlling acute bleeding from esophageal varices but also to prevent rebleeding during the long-term management (Table 3).

There is no clear agreement how frequently ligation sessions should be repeated during the initial course of eradication and the intervals vary from 1 to 4 weeks (19, 20). EBL sessions are usually repeated at 2–4 week intervals until the complete obliteration of all esophageal varices has been achieved with the first surveillance endoscopy performed 1–3 months after obliteration and then every 6–12 months to check for variceal recurrence (2). Complete obliteration can be achieved in about 90% of patients after 2–4 EBL sessions (21).

However, varices after EBL have a higher tendency of recurrence and variceal recurrence is higher after EBL compared with injection sclerotherapy (22). This can be explained by the fact that EBL is a local mechanical method of variceal obliteration and does not decrease portal pressure. That is why the EBL effect is of limited duration and has no effect on other complications of portal hypertension (23). Also, this observation can be explained by the fact that EBL only achieves the eradication of the varices in the mucosal and submucosal layers, leaving untouched the perforating veins, which join the submucosal vascular channels to collateral veins.

The reported prevalence of variceal recurrence after EBL varies within a wide range. After complete variceal eradication by EBL, variceal recurrence occurs frequently, with 20–75% of these patients requiring repeated EBL sessions (21). One follow-up study showed that variceal rebleeding occurred in 3.9% of patients after complete variceal eradication achieved by EBL, and after a 22.3 month follow-up period the recurrence of esophageal varices was observed in 11.9% of patients (24). In another follow-up study, the recurrence of esophageal varices

Table 3. Indications for endoscopic band ligation	
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1. Active bleeding	- to stop active variceal bleeding	
2. Primary prophylaxis	<ul> <li>to prevent the first variceal bleeding episode in patients with large and / or high risk varices</li> </ul>	
3. Secondary prophylaxis	- to prevent the recurrence of variceal bleeding in patients who had an initial episode of variceal bleeding	

was observed in 5% after a 10.6 month follow-up period (25). In a prospective study of 102 cases, the recurrence of esophageal varices after EBL was observed in 22% after an average follow-up of 7 months (26). Silvano S in his study observed that once obliteration was achieved, varices reappeared in 28% of patients (27).

Early variceal rebleeding after EBL (24 hours – 14 days after the EBL procedure) is also very important because it is related to high mortality rates (28). Early rebleeding prevalence after EBL is 9–19% with 80.8% rebleedings between day 7 and 13 (29, 30). It can be due to the spontaneous slippage of a rubber band leaving the ulcer or it can be related to early variceal rebleeding. There are some risk factors for early variceal rebleeding after EBL: previous upper variceal bleeding, peptic esophagitis, the number of varices (28, 31), Child-Pugh score (32), the volume of ascites, the number of rubber bands used to ligate, the severity of varices and prolonged prothrombin time (29).

The details and mechanism of variceal recurrence after EBL are still controversial, but they seem to be related to esophageal collateral veins located around the esophagus. It has been known that the gastroesophageal junction is drained by an intrinsic mucosal and submucosal venous plexus that communicates with an extrinsic plexus of veins via perforating veins (32). Collateral veins are found adjacent to or outside the esophageal wall in all patients with esophageal varices but not in any control subjects (34, 35). The variation of variceal recurrence (rates, time interval) may be related to differences in these collateral veins located around the esophageal varices and associated hemodynamics.

Several studies have shown endoscopic ultrasound (EUS) to be useful in predicting variceal recurrence by referring collaterals around the esophagus (36, 37). According to the recent recommendations of the Japanese Society for Portal Hypertension (5), collaterals veins on EUS are classified as perforating veins, periesophageal veins (a group of small vessels adjacent to the muscularis externa of the esophagus or partly invading the muscular wall of the esophagus) and paraesophageal veins (a group of large vessels distal to the muscularis externa of the esophagus).

The role of these feeding vessels as risk factors for variceal recurrence and recurrent variceal he-

morrhage in patients undergoing the endoscopic treatment of variceal hemorrhage has been investigated in two well designed studies (37, 38). Both studies indicated that patients with large paraesophageal varices (>5 mm diameter) were more likely to have variceal recurrence and the subsequent episodes of variceal bleeding. Gin H assessed the endoscopic treatment (sclerotherapy vs EBL) impact on paraesophageal varices and found that the prevalence of paraesophageal varices was higher in the EBL group compared with the sclerotherapy group (86% and 51% respectively). Esophageal varices recurred in 70% of the ligation group and 43% of the sclerotherapy group (38). This study confirmed that patients with large residual paraesophageal veins had a higher variceal recurrence rate.

### CONCLUSIONS

The recurrence of varices occurs after endoscopic ligation and it remains unclear why some patients develop it and others do not. The time frame of the recurrence of esophageal varices varies markedly among patients. Some factors which affect the recurrence and their variation may be related to differences in the collateral veins surrounding the esophagus, venous structures connected with esophageal varices and associated hemodynamics. A better understanding of endoscopic ultrasound findings in patients with liver cirrhosis would allow us to choose the proper endoscopic and / or medical treatment according to the variceal recurrence risk in order to prevent bleeding from esophageal varices, their recurrence after treatment, to reduce mortality and prolong the time of patients awaiting the liver transplantation. Our data revealed a high esophageal varices recurrence rate after EBL and in half of these cases there was early variceal recurrence. Extrahepatic portal hypertension and a greater size of varices led to a statistically significant early recurrence of esophageal varices after EBL. We are aware that the main disadvantages of our study are that we have not evaluated the use of non-selective beta-adrenoblocators and our ligation technique was not standardized - EBL was repeated only if the recurrence of varices was diagnosed or in case of active variceal bleeding.

The recurrence of varices may become more frequent in the course of time. The reports which

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# References

- 1. Abraldes JG, Villanueva C, Banares R, et al. Hepatic venous pressure gradient and prognosis in patients with acute variceal bleeding treated with pharmacologic and endoscopic therapy. J Hepatol. 2008; 48: 229–36.
- Garcia-Tsao G, Sanyal AJ, Grace ND, Carey WD. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Hepatology. 2007; 46(3): 922–38.
- North Italian Endoscopic Club for the study and treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and large esophageal varices. N Engl J Med. 1988; 319: 983–9.
- Pagliaro L, D'Amico G, Sorensen TIA, Lebrec D, Burroughs AK, Morabito A, et al. Prevention of first bleeding in cirrhosis. A meta-analysis of randomized trials of nonsurgical treatment. Ann Intern Med. 1992; 117: 59–70.
- Tajiri T, Yoshida H, Obara K, Onji M, Kage M, Kitano S, et al. General rules for recording endoscopic findings of esophagogastric varices (2nd edition). Digest Endosc. 2010; 22: 1–9.
- Pascal JP, Calès P, Desmorat H. Natural history of esophageal varices. In: Bosch J, Rodès J, editors. Recent advances in the pathophysiology and treatment of portal hypertension. Serono Symposia Review No. 22; Rome, Italy; 1989. p. 127–42.
- D'Amico G, Luca A. Natural history. Clinical-haemodynamic correlations. Prediction of the risk of bleeding. Bailliere's Clin Gastroenterol. 1997; 11: 243–56.
- Pagliaro L, D'Amico G, Pasta L, Politi F, Vizzini G, Traina M, et al. Portal hypertension in cirrhosis: natural history. In: Bosch J, Groszmann RJ, editors. Portal Hypertension, Pathophysiology and Treatment. Oxford, UK: Blackwell Scientific Publications; 1994. p. 72–92.

- Šilkauskaitė V, Pranculis A, Mitraitė D, Jonaitis L, Petrenkienė V, Kupčinskas L. Hepatic venous pressure gradient measurement in patients with liver cirrhosis: a correlation with disease severity and variceal bleeding. Medicina. 2009; 45(1): 8–13.
- Calès P, Desmorat H, Vinel JP, et al. Incidence of large oesophageal varices in patients with cirrhosis: application to prophylaxis of first bleeding. Gut. 1990; 31: 1298–302.
- Tsokos M, Turk EE. Esophageal variceal hemorrhage presenting as sudden death in outpatients. Arch Pathol Lab Med. 2002; 126: 1197–200.
- Pagliaro L, D'Amico G, Sorensen TIA, Lebrec D, Burroughs AK, Morabito A, et al. Prevention of first bleeding in cirrhosis. A meta-analysis of randomized trials of nonsurgical treatment. Ann Intern Med. 1992; 117: 59–70.
- Graham DY, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology. 1981; 80: 800–9.
- 14. D'Amico G, De Franchis R. Upper digestive bleeding in cirrhosis. Post-therapeutic outcome and prognostic indicators. Hepatology. 2003; 38: 599– 612.
- Stokkeland K, Brandt L, Ekbom A, Hultcrantz R. Improved prognosis for patients hospitalized with esophageal varices in Sweden 1969–2002. Hepatology. 2006; 43(3): 500–5.
- De Franchis R. Revising consensus in portal hypertension: Report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2010; 53: 762–8.
- 17. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey WD. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Am J Gastroenterol. 2007; 102: 2086–102.
- Van Stiegmann G, Cambre T, Sun JH. A new endoscopic elastic band ligating device. Gastrointest Endosc. 1986; 32: 230–3.
- Schepke M, Kleber G, Nurnberg D, Willert J, Koch L, Veltzke-Schlieker W, et al. Ligation versus propranolol for the primary prophylaxis of variceal bleeding in cirrhosis. Hepatology. 2004; 40: 65–72.
- 20. Jutabha R, Jensen DM, Martin P, Savides T, Han SH, Gornbein J. Randomized study comparing banding and propranolol to prevent initial variceal

hemorrhage in cirrhotics with high-risk esophageal varices. Gastroenterology. 2005; 128: 870–81.

- Garcia-Pagan JC, Bosch J. Endoscopic band ligation in the treatment of portal hypertension. Nat Clin Pract Gastroenterol Hepatol. 2005; 2: 526– 35.
- 22. Hou MC, Lin HC, Lee FY, Chang FY, Lee SD. Recurrence of esophageal varices following endoscopic treatment and its impact on rebleeding: comparison of sclerotherapy and ligation. J Hepatol. 2000; 32: 202–8.
- Pereira-Lima JC, Zanette M, Lopes CV, de Mattos AA. The influence of endoscopic variceal ligation on the portal pressure gradient in cirrhotics. Hepato-Gastroenterology. 2003; 50: 102–6.
- Mei ZC, He L, Chen WQ, Shen W, Shen DM. Evaluation of the effects of dense endoscopic ligation for bleeding esophageal varices [abstract]. Chin J Hepatol. 2005.
- Liu JK, Dong JH, Cai J. Evaluation of the effects of dense endoscopic ligation for bleeding esophageal varices (21 cases report) [abstract]. Chin J Endosc. 2006.
- 26. Bouzaidi S, Ben Hammouda I, Ben Yedder J, Ben Salem M, Jemaa Y, Trabelsi S, et al. Place of endoscopic variceal ligation for the prophylaxis of rebleeding from oesophageal varices. Prospective study about 102 cases. Tunis Med. 2004; 82(6): 531–7.
- Silvano S, Elia C, Alessandria C, Bruno M, Musso A, Saracco G, Rizzetto M, Venon WD. Endoscopic banding for esophageal variceal bleeding: technique and patient outcome. Gastroenterol Hepatol. 2006; 29(6): 323–6.
- Li P, Zhang ST, Yu ZL, Yu YZ, Ji M, Yu L, et al. Analysis of the risk factors in early rebleeding after endoscopic variceal ligation. Chin J Digest Endosc. 2006; 23: 23–6.
- Xu L, Ji F, Xu QW, Zhang MQ. Risk factors for predicting early variceal rebleeding after endoscopic variceal ligation. World J Gastroenterol. 2011; 17(28): 3347–52.
- Lo GH, Chen WC, Chen MH, Lin CP, Lo CC, Hsu PI, et al. Endoscopic ligation vs. nadolol in the prevention of first variceal bleeding in patients with cirrhosis. Gastrointest Endosc. 2004; 59: 333–8.
- 31. Vanbiervliet G, Giudicelli-Bornard S, Piche T, Berthier F, Gelsi E, Filippi J, et al. Predictive factors of

bleeding related to post-banding ulcer following endoscopic variceal ligation in cirrhotic patients: a case-control study. Aliment Pharmacol Ther. 2010; 32: 225–32.

- Yang MT, Chen HS, Lee HC, Lin CL. Risk factors and survival of early bleeding after esophageal variceal ligation. Hepato-Gastroenterology. 2007; 54: 1705–9.
- Vianna A, Hayes PC, Moscoso G. Normal venous circulation of the gastroesophageal junction. A route to understanding varices. Gastroenterology. 1987; 93: 876–89.
- 34. Irisawa A, Obara K, Sato Y, Saito A, Takiguchi F, Shishidon H, et al. EUS analysis of collateral veins inside and outside the esophageal wall in portal hypertension. Gastrointest Endosc. 1999; 50: 374–80.
- Caletti G, Brocchi E, Baraldini M, Ferrari A, Gibilaro M, Barbara L. Assessment of portal hypertension by endoscopic ultrasonography. Gastrointest Endosc. 1990; 36: S21–7.
- 36. Dhiman RK, Choudhuri G, Saraswat VA, Agarwal DK, Naik SR. Role of paraoesophageal collaterals and perforating veins on outcome of endoscopic sclerotherapy for oesophageal varices: an endosonographic study. Gut. 1996; 38: 759–64.
- Leung VKS, Sung JJY, Ahuja AT, Tumala IE, Lee YT, Lau JYW, et al. Large paraesophageal varices on endosonography predict recurrence of esophageal varices and rebleeding. Gastroenterology. 1997; 112: 1811–6.
- 38. Lo GH, Lai KH, Cheng JS, Huang RL, Wang SJ, Chiang HT. Prevalence of paraesophageal varices and gastric varices in patients achieving variceal obliteration by banding ligation and by injection sclerotherapy. Gastrointest Endosc. 1999; 49: 428–36.

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# ATSINAUJINANTIS STEMPLĖS VENŲ IŠSIPLĖTIMAS PO ENDOSKOPINIO LIGAVIMO: VIENO CENTRO PATIRTIS

## Santrauka

Darbo tikslas. Pagrindinis išsiplėtusių stemplės venų endoskopinis gydymas yra jų perrišimas guminiais žiedais – endoskopinis ligavimas, tačiau po šio gydymo stemplės venų išsiplėtimas ligoniams atsinaujina. Šios retrospektyvinės analizės tikslas – nustatyti išsiplėtusių stemplės venų (varikozių) atsinaujinimo dažnį ir laiką po endoskopinio ligavimo ir tai sąlygojančius rizikos veiksnius.

Darbo metodika. Atliekant endoskopinių ligavimų (VULSK, 2006–2010 m.) retrospektyvinę analizę, buvo įvertinti demografiniai duomenys, portinės hipertenzijos etiologija, varikozių laipsnis (F), varikozių atsinaujinimas po ligavimo. Statistikos analizei panaudota SPSS 19.0 programa. Skirtumai tarp grupių laikomi statistiškai reikšmingais, kai p < 0,05. **Rezultatai.** Per penkerius metus buvo atlikti 183 ligavimai 118-ai ligonių: 61 vyrui (52 %) ir 57 moterims (48 %); amžiaus vidurkis 51 ± 16 metų; penki iš jų buvo vaikai. Į galutinę analizę dėl varikozių atsinaujinimo įtraukti 133 ligavimai. Po ligavimo varikozės atsinaujino 45 % atvejų: ankstyvo varikozių atsinaujinimo atvejai sudarė 46,7 %, vėlyvo – 53,3 %. Nustatyta, kad esant ekstrahepatinei portinei hipertenzijai ir F3 varikozėms prieš endoskopinį ligavimą, ankstyvas varikozių atsinaujinimas statistiškai reikšmingai buvo dažnesnis nei vėlyvasis (p = 0,013 ir p = 0,038 atitinkamai).

Išvados. Po endoskopinio ligavimo nustatytas gana dažnas varikozių atsinaujinimas (45 %), beveik puse atvejų jis yra ankstyvas (46,7 %). Nėra visiškai aišku, kodėl vieniems ligoniams varikozės atsinaujina greičiau, o kitiems – vėliau. Ekstrahepatinė portinė hipertenzija ir didesnis F laipsnis prieš ligavimą lemia statistiškai reikšmingą ankstyvą varikozių atsinaujinimą.

Raktažodžiai: endoskopinis ligavimas, atsinaujinantis stemplės venų išsiplėtimas, rizikos veiksniai