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Original Research Article

Efficacy of Endoscopic Band Ligation in Esophageal Varices Due to Portal Hypertension

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Conflict of interest: Nil

Abstract:

Introduction: Portal hypertension remains the commonest cause of Upper gastrointestinalbleed in India and carries a high risk of rebleed and mortality. Endoscopic variceal band ligation (EVBL) is the cornerstone for the control of acute variceal bleeding.

Aims: To assess the therapeutic efficacy of esophageal variceal band ligation in patientswith esophageal varices due to portal hypertension.

Materials and Methods: This study was conducted as a prospective observational studyconducted at Geetanjali Medical College and Hospital, Udaipur, Rajasthan from January 2019 to June 2020 after institutional ethics committee approval. The study population comprised of patients >18 years of age who presented with portal hypertension and esophageal varices undergoing EVBL, Patients with diagnosis of portal hypertension on imaging or incidentally detected on routine/emergency endoscopy for haematemesis.

Patients requiring intensive care and Patients with terminal illness were excluded.

Results: In our study on 33 patients with esophageal varices due to portal hypertension, the most common etiology was cirrhosis of liver (93%). In 51% patients, cirrhosis was due to alcoholic liver disease, 24% had chronic Hepatitis B infection and 18% due to Non-alcoholic fatty liver disease (NAFLD). 3% had Non Cirrrhotic Portal Vein Fibrosis (NCPF) and 3% had Extrahepatic Portal Vein Obstruction (EHPVO). Out of 33 patients, 4 had complete esophageal variceal obliteration in 2 sessions and 29 had complete esophageal variceal obliteration in 3 sessions. Thus, 2 or 3 sessions were required for complete obliteration of all esophageal varices.

Conclusion: complete obliteration of esophageal varices by repeated EVBL will save lives,which is inexpensive, easily available method with minimal complications and side effects. Our study concluded that 2 or 3 sessions of EVBL can obliterate all the varices of any grade or severity in 8 to 12 weeks, at 4 weekly interval endoscopies.

Keywords: Endoscopic Band ligation, Esophageal Varices, Portal hypertension.

Introduction

Portal hypertension is defined as the elevation of the hepatic venous pressure to a gradient more than 5mmHg. [1] The hepatic vein pressure gradient (HVPG) is a surrogate means to measure pressure in the portal veins. [2]

Portal vein pressure normally ranges from 7 to 12 mm Hg at rest and in fasting conditions. Direct portal pressure measurement, however, is invasive and requires direct cannulation of portalor umbilical veins. [3] Normal HVPG (hepatic vein wedge pressure - free hepatic vein pressure) is around 3-5 mmHg.Elevation in vascular resistance leads to shunting of blood to collaterals causing the development of congested sub epithelial and sub mucosal venous plexus within distal esophagus and proximal stomach; termed as varices [4] Further elevation of portal venous pressure leads to rupture of esophageal varices and bleeding, which is a common complication of portal hypertension and presents as upper gastrointestinal bleed (UGIB). Gastrointestinal varices are abnormally dilated submucosal veins in the digestive tract due to portal hypertension and can potentially cause life-threatening bleeding. Prevalence of varices increases with the severity of liver disease. [5]

The development of portal hypertension in cirrhosis is a multifactorial process with changes in both the portal and systemic circulation.

Material and Methods

This study was a hospital based prospective obser-

vational study conducted at Geetanjali Medical College and Hospital, Udaipur, Rajasthan over a period from January 2019 to June 2020. The study population comprised of patients >18 years of age who presented with portal hypertension and esophageal varices. Inclusion criteria included adult patients with esophageal varices due to portal hypertension undergoing EVBL, Patients with diagnosis of portal hypertension on imaging or incidentally detected on routine/emergency endoscopy for hematemesis, Patients who provided written informed consent. Patients requiring intensive care and Patients with terminal illness were excluded.

Sample Size Calculation

At 90% confidence level and assuming the prevalence as 25.8% and with a relative error of15%, the sample size was estimated was 33 using the formula:

$$n = \frac{(Z\alpha + Z(1-\beta))^2 \times P(1-P)}{E^2}$$

Where Prevalence (P) = 25.8%

Zα be the level of confidence at 90% = 1.96. Z1-β be the Power of study at 80%. = 0.8413 Allowable error (E) of 15%.

Thus, sample size (n) required is 33.

The purpose of the study was explained to the study subjects in the local language with the help of information sheet. All enrolled patients underwent clinical examination and routine laboratory tests. Following tests were done: Complete Blood Count (CBC), Liver Function Tests (LFT), Viral Serology: done by HEPACARD immunoassay based on antigen capture method for Hepatitis B Antigen detection, and 4th Generation HCV (Hepatitis C Virus) Tri-dot RapidVisual Test for detection of antibodies to Hepatitis C, Imaging Studies: Ultrasonographyof abdomen or CT scan of Abdomen was done wherever needed.

Upper GI endoscopy (UGIE) was performed using an Olympus CV 150 video endoscope by expert consultants in Gastroenterology and was assisted by the investigator. The endoscopy was conducted under local anesthesia and/or Intravenous sedation as per patient preference and consent on empty stomach. The patients were kept in left lateral position and the endoscope was inserted into the esophagus through the mouth with protection gag. All the patients were evaluated by UGIE for esophageal varices to know their severity, location, number and extent. Varices were classified as follows: [6].

Grade	Endoscopic findings
0	Absence of esophageal varices
I	Microvessels that sketch varicose strings located in the esophagogastric transition or in the distal esophagus
II	One or two fine-caliber varices (smaller than 3 mm diameter) located in the distal esophagus
III	Medium caliber varices (between 3 or 6 mm diameter) or more than varices up to 3 mm that may reach up to a third medium third of the esophagus.
IV	Thick caliber varices, larger than 6 mm diameter, in any part of the esophagus.

Figure 1:

EVBL consists of placement of elastic bands on a varix after it is sucked into a clear plastic cylinder attached to the tip of the endoscope. [7] After the diagnostic endoscopy was performed and the culprit varix identified, the tip of the endoscope was pointed toward it and continuous suction was applied so it could fill the cap (Figure 1). [8] Once inside the cap, a ‘‘red out’’ sign appeared and at this point the band was fired. Multi band ligator-Barrel (6 bands)was used at our center. The application of bands was started at the gastroesophageal junction and then progressed upwards in a helical fashion to avoid circumferential placement of bands at the same level. The application of bands progressed for approximately 6-8 cm. After banding was done over esophageal varices, their sloughing followed, after which, there was formation of shallow esophageal ulcers at ligated sites and reduction in the diameter of esophageal varices. (Figure 2).

Patients were advised to start with liquids after 2 hours of the procedure for the first 12 hours and then take soft foods gradually.

Post each EVBL session, patients were monitored for adverse effects like: Retrosternal pain, Dysphagia, Hematemesis, Fever. All the patients underwent repeat UGIE every 4th week [ix] and repeated EVBL was done till complete obliteration of esophageal varices achieved.

Our study was completed when all the esophageal varices were obliterated in all the patients. Any adverse effect or complication were noted and managed.

The data was entered in MS EXCEL software version 17 and analyzed using Statistical Packages for Social Sciences (SPSS). The data was assessed in proportions, mean and frequency tables. The categorical data was analyzed using Chi-square test, paired/unpaired t-test and Mc-Nemar's test. P value less than 0.05 was considered statistically significant.

Observation and Results

Out of total 33 patients, who had esophageal varices in whom EVBL was done. Age ranged from 31-80 years. 13 (39.4%) patients were of age group 41-50 years, 9 (27.3%) of 51-60 years, 4 (12.1%) of 31-40 years and 61-70 years each, 3 (9.1%) of 71-80 years. Mean age was 52.96 years. 27 (81.8%) patients were male and 6 (18.2%) were females of esophageal varices in whom EVBL was done. 29 (87.9%) patients presented with hematemesis, 17 (51.5%) with melena, 13 (39.4%) with pain in abdomen, and 5 (15.15%) with jaundice.

14 (42.4%) patients had a history of alcohol consumption, 9(27.3%) were known cases of esophageal varices, 9 (27.3%) had history of acid peptic disease, 5 (15.15%) had history of jaundice. 15 (45.45%) patients presented with pallor, 8 (24.24%) with anasarca, 6 (18.18%) with jaundice, 2 (6.06%) with pallor and jaundice, 2 (6.06%) with pallor, anasarca and jaundice. 15 (45.45%) patients had splenomegaly, 10 (30.3%) had hepatomegaly and splenomegaly, 7 (21.21%) had hepatomegaly, splenomegaly and ascites, and 1 (3.03%) patient had caput medusae and splenomegaly. Mean Hb (haemoglobin) in males was 8.71 gm/dL, 4 (14.81%) patients had Hb <7gm/dL, 22 (81.48%) had Hb 7-13 gm/dL, 1 (3.70%) had Hb ≥13 gm/dL. Mean Hb in females was 8.43 gm/dL, all the 6 female (100%) patients had Hb ranging from 7-12 gm/dL. Mean TLC was 7.63/mm^3, all the 33 (100%) patients had TLC ranging from 4000-11000/mm^3. Mean platelet count was 3.19 lakhs/mm^3. 12 (36.3%) patients had platelet count<1.5 lakhs/mm^3, 21 (63.6%) patients had platelet count ranging from 1.5-4.5 lakhs/mm^3. None had thrombocytosis.

Mean serum bilirubin 1.99 mg/dL, 12 (36.36%) patients had bilirubin 0.2 - 1 mg/dL and, 21(63%) had bilirubin from 1 - 4.35 mg/dL. Mean direct bilirubin was 1.22 mg/dL, 1 (3.1%) patient had bilirubin <0.25 mg/dL, while 32 (96.9%) patients had bilirubin from 0.25 - 4.03 mg/dL. Mean indirect bilirubin was 0.77 mg/dL, 4 (12.12%) patients had bilirubin <0.2 mg/dL. 16 (48.48%) had bilirubin from 0.25 - 0.75 mg/dL, while 13 (39.39%) had bilirubin from 0.75 - 3.33 mg/dL. Mean total protein was 6.18 gm/dL, 20 (60.6%) had protein <6.6 gm/dL, 13 (39.3%) had protein from 6.6 - 8.3 gm/dL, while none had protein > 8.3 gm/dL. Mean serum albumin was 2.69 gm/dL. 29 (87.8%) had <3.5gm/dL, 4(12.12%) had between 3.5 - 5 gm/dL.

None had >5 gm/dL. Mean serum globulin was 3.49 gm/dL, 3 (9%) had <2.3 gm/dL, 17 (51%) had between 2.3-3.5 gm/dL, while 13 (39.3%) had between 3.5- 5.77 gm/dL. Mean PT (prothrombin time) was 21.64 seconds, 7 (21%) had PT from 11 - 16 seconds, 26 (78.7%) had PT from 16- 71 seconds. Control PT was 13.5 seconds. Mean INR (international normalized ratio) of the patients was 1.80, 2 (6%) had INR of 1.1, while 31 (93.9%) had INR in range of 1.1-5.5. Mean AST (Aspartate Transaminase) of the patients was 89.1 IU/L. 12 (36.3%) had AST from 10-45 IU/L, 13 (39.3%) had AST from 46-135,8 (24.2%) had AST from 135- 450IU/L, while none had AST > 450 IU/L. Mean ALT (Alanine Transaminase) of patients was 38.6 IU/L, 27 (81%) had ALT from 10-45 IU/L, 6 (18%) had ALT from 46-135 IU/L, while none had ALT from 135-450 or > 450 IU/L. 2 (6%) patients had AST: ALT <1.0, 31 (93.93%) had >1.0. AST was upto 7 times higher than ALT. Mean ALP (Alkaline Phosphatase) was 107.3 IU/L, 1 (3%) patient had ALP <40 IU/L, 24 (72.7%) had normal ALP from 40-130 IU/L, while 8 (24.2%) had ALP from 130-205 IU/L. 31 patients were having cirrhosis of liver, in whom 17 (51%) patients were due to chronic alcoholism, 8 (24.2%) due to chronic Hepatitis B infection, and 6(18.18%) due to NAFLD. 1 (3%) patient had NCPF, and 1 (3%) had EHPVO as a cause of portal hypertension in patients with esophageal varices (Table 1). 9 (27.27%) patients had Grade I, 14 (42.4%) had Grade II, and 10 (30.3%) had Grade III esophageal varices. At 4th week of follow up, 14 (42.42%) had Grade I, 13 (39.3%) had Grade II, and 6 (18.18%) had Grade III esophageal varices. No complete esophageal variceal obliteration was achieved by the end of 4th week and all patients required repeat

2). At 8th week of follow up, 17 (51.5%) patients had

Grade I, 12 (36.3%) had Grade II,and none had Grade III esophageal varices, thus further severity of grading was reduced. 4 patients achieved all variceal obliteration. 29 patients required further EVBL. At 12th week of follow up, remaining 29 patients achieved obliteration of all esophagealvarices. Thus, 2 or 3 sessions are needed for complete obliteration of all esophageal varices. 11(33.33%) patients had retrosternal pain, and 3 (9.09%) had fever, post EVBL (Table 3).

Table 1: Etiology of Portal Hypertension in patients with esophageal varices

Etiology		Number of Patients	Percent(%)	Total
Cirrhosis	Alcoholic	17	51	31
	Chronic Hepatitis B	8	24.2	
	NAFLD	6	18.18	
Non- Cirrhosis	NCPF	1	3	1
Extra Hepatic	EHPVO	1	3	1

Table 2: Endoscopic variceal grading and EVBL performed on admission and follow up endoscopy every 4th week till completeobliteration achieved

	On Presentation		4th Week		8th Week		12th Week	
	No. of Patients	Percent (%)	No. of Patients	Percent (%)	No. of Patients	Percent (%)	No. of Patients	Percent (%)
Variceal Grading								
Grade I	9	27.27	14	42.4	17	51.5	-	-
Grade II	14	42.42	13	39.3	12	36.3	-	-
Grade III	10	30.3	6	18.18	-	-	-	-
EVBL performed	33	100	33	100	29	87.87	-	-
Variceal obliteration achieved	-	-	-	-	4	12.12	29	87.7

Table 3: Complications occurring during and post EVBL sessions.

Complications	No. of patients	Percent (%)
Retrosternal pain	11	33.33
Fever	3	9.09
Hematemesis (due to varices)	0	0
Hematemesis (due to ulcer formation at site of band ligation)	0	0
Stricture	0	0

Figure Legends

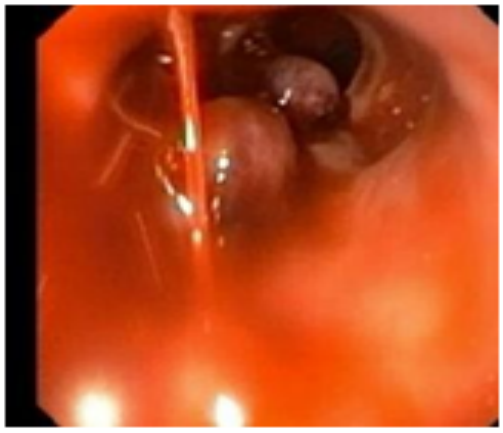

Figure 1: Shows active bleeding Varices

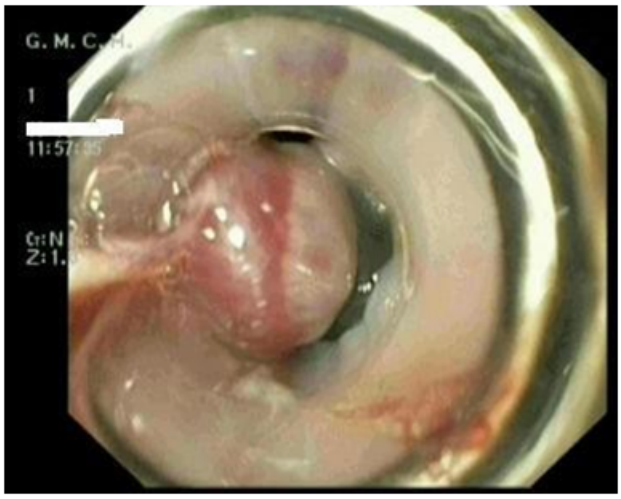

Figure 2: Shows Post band ligation

Discussion

Our present study was hospital based prospective observational, conducted at Geetanjali Medical College and Hospital, Udaipur, (Rajasthan) India, enrolling a total of 33 patients of portal hypertension with esophageal varices in whom EVBL was done repeatedly till completeobliteration of varices was achieved. The study was aimed to analyze the efficacy of the EVBL in obliteration of esophageal varices. In our study of 33 patients of esophageal varices, the age of patients ranged from 31 to 80 years with mean age of 52.96 years. Maximum number of patients were in the age group 41-50 years (13 patients). In a study by Inokuchi et al [10], the ageof patients ranged from 16 to 72 years with a mean age of 48.4 years. 33 patients of esophagealvarices were studied, in which 27 (81.8%) were males and 6 (18.2%) were females. Similar to our study, Sakthivel, H. et. al. [11], also observed in 60 patients with esophageal varices where 44 (73.3%) were males and 16 (26.7%) were females.

Patients with esophageal varices presented with multiple symptoms. Among 33 patients, the commonest presenting symptom was hematemesis in 29 (87.9%) patients, followed by melena in 17 (51.5%), pain abdomen in 13 (39.4%), and jaundice (15.15%). While Maskey et al [12] observed among 90 patients, 76 (84.4%) patients had abdominal distension and jaundice each, 30 (30%) had fever, 27 (30%) had hematemesis, and 32 (35.5%) had melena. Among 33 patients, 14 (42.4%) patients had a history of alcohol consumption.9 (27.3%) were known cases of esophageal varices, 9 (27.3%)had history of acid peptic disease, and 5 (15.2%) had history of jaundice. Liu et al [13] observed in 59 patients that 13 (22%) patients had history of alcoholism, 35 (59.3%) were known cases of esophageal varices, 3 (5.1%) had acid peptic disease, and 8 (13.7%) had historyof jaundice. Alcohol being the most common cause of cirrhosis, esophageal varices are due tothis cirrhosis and patient comes repeatedly due to upper gastrointestinal bleed. In our study 15(45.45%) patients presented with pallor, 8 (24.24%) with anasarca, 6 (18.18%) with jaundice, 2 (6.06%) with pallor and jaundice, 2 (6.06%) with pallor, anasarca and jaundice. More than one feature was present in few patients.

On abdominal examination, 15 (45.45%) patients had splenomegaly, 10 (30.3%) had hepatomegaly and splenomegaly, 7 (21.21%) had hepatomegaly, splenomegaly and ascites, and 1 (3.03%) patient had caput medusae and splenomegaly. Again, more thanone feature waspresent in few patients. Sakthivel, H. et al [xi] observed in 30 patients that 24 (80%) had pallor,8 (26.7%) had jaundice, and 21 (70%) had splenomegaly. Hossain et al [xiv] observed that 95%had anasarca, and 64% had jaundice. In our study on 33 patients, mean Hb in 27 males was 8.71 gm/dL, 14.81% had Hb less than 7gm/dL, 81.48% had Hb in range of 7-13 gm/dL and 3.70% had Hbmore than 13gm/dL. All the males were anemic except one patient, 4 males were severely anemic. In 6 females, mean Hb was 8.43 gm/dL and all had Hb in the range of 7- 12gm/dL. All the females were anemic. Mean TLC was 7.63/mm^3 and mean platelet count was 3.19 lakhs/mm^3. Bedel et al [15] in his study observed the mean Hb 9.44 gm/dL, mean TLC 11.0/mm^3 and mean platelet count 2.53 lakhs/mm^3.

The mean bilirubin in the 33 studied patients was 1.99 mg/dL, 21 (63%) hadraised bilirubinbetween 1-4.35 mg/dL and 12 (36.36%) had 0.2-1 mg/dL. Mean direct bilirubin was 1.22 mg/dL, 32 (96.9%) had between 0.25-4.03 mg/dL, and 1 (3%) had <0.25 mg/dL. Mean indirect bilirubin was 0.77 mg/dL, 16 (48.48%) had between 0.2-0.75 mg/dL, 4 (12.12%) hadbetween 0.75-3.33 mg/dL. Similarly, Lay et al [xvi] observed mean bilirubin of the patients was 3.1 mg/dL with 42 (67%) having raised direct bilirubin > 0.25 mg/dL,

and 19 (30%) having raised indirect bilirubin >0.75 mg/dL. The mean total protein in the 33 studied patients was 6.18 gm/dL, 20 (60.6%) had serum protein <6.6 gm/dL, 13 (39.3%) had between 6.6 - 8.3 gm/dL. Mean serum albumin was 2.69 gm/dL. 29 (87.8%) had <3.5gm/dL, 4(12.12%) had between 3.5 - 5 gm/dL. Mean serum globulin was 3.49 gm/dL, 3 (9%) had <2.3 gm/dL, 17 (51%) had between 2.3-3.5 gm/dL, while 13 (39.3%) had between 3.5- 5.77 gm/dL. Similar to our study, Wang et al [17] on 103 patients observedthe mean serum protein 6.22 gm/dL, mean serum albumin 2.9 gm/dL, and mean serum globulin 3.32 gm/dL.

Mean PT of patients in our study was 21.64 seconds, with a control of 13.5 seconds. 78% of the patients had raised PT between 16 to 71 seconds. Mean INR of the patients was 1.80 and 93% had raised INR between 1.1 to 5.25. Similar to our study, Rocha et al [18] observed in 160 patients that 73% of patients had raised INR >1.5.

In our study on 33 patients, mean AST was 89.1 IU/L. 12 (36.3%) had AST between 10-45 IU/L, 13 (39.3%) had AST from 46-135,8 (24.2%) had AST between 135-450 IU/L, while none had AST > 450 IU/L. Mean ALT of patients was 38.6 IU/L, 27 (81%) had ALT between 10-45 IU/L, 6(18%) had ALT between 46-135 IU/L, while none had ALT >135 IU/L. 2 (6%) patientshad AST: ALT ratio <1.0, 31 (93.93%) had ratio >1.0. AST was up to 7 times higher than ALT, which is classically seen in alcoholic cirrhosis of liver.

Lai et al [16] observed in 50 patients the mean AST was 75 IU/L, and 40% had raised up to 3 – 10 times of upper limit of normal. Mean ALT was 64 IU/L and raised up to 2 times the upper limit of normal, and AST:ALT ratio was >1.0.

In our study on 33 patients with esophageal varices due to portal hypertension, the most commonetiology was cirrhosis of liver (93%). In 51% patients, cirrhosis was due to alcoholic liver disease, 24% had chronic Hepatitis B infection and 18% due to NAFLD. 3% had Non Cirrrhotic Portal Vein Fibrosis (NCPF) and 3% had Extrahepatic Portal Vein Obstruction (EHPVO).

Nayak et al [19] reported in 142 cases, the etiology of portal hypertension as follows: 70 (49%)had Cirrhosis of liver due to alcohol (80%) and HBV infection (20%). 22 (15%) had Cirrhosis with HCC, 18 (13%) had NCPF, 22 (15%) had EHPVO, and 10 (7%) had Budd Chiari Syndrome. In our study at the time of presentation the endoscopy revealed, 9 (27.27%) patients had Grade I, 14 (42.4%) had Grade II, and 10 (30.3%) had Grade III esophageal varices. At 4th week of follow up endoscopy, 14 (42.42%) had Grade I, 13(39.3%) had Grade II, and 6 (18.18%) had Grade III esophageal varices and in all EVBL was done. None achieved complete esophageal variceal obliteration by the end of 4th week (2nd endoscopy), although severity of grading was decreased, and all patients required 3rd endoscopy at 8th week to see the esophageal variceal obliteration and further EVBL, although decreased severity of grading was observed.

At 8th week (3rd endoscopy) of follow up, 17 (51.5%) patients had Grade I, 12 (36.3%) had Grade II, and none had Grade III esophageal varices, thus further decrease in severity of grading was observed. 4 patients achieved all esophageal variceal obliteration. 29 patients required EVBL at this time. At 12th week of follow up on 4th endoscopy, remaining 29 patients achieved obliteration of all esophageal varices. Thus, out of 33 patients, 4 had complete esophageal variceal obliteration in 2 sessions and 29 had complete esophageal variceal obliteration in 3 sessions. Thus, 2 or 3 sessions were required for complete obliteration of all esophageal varices.

Further, we observed on 6th month follow up endoscopy on 17 patients (16 patients were lost to follow up), no reappearance of esophageal varices was seen in 12 patients at 6 months, 5 patients where esophageal varices reappeared, underwent EVBL again.

Similar to our observations, Lai et al [16] observed that average number of sessions required were 2.7 $\pm$ 1 at 4 weekly intervals. Pramigani et al [20] observed that 2.5-4.1 sessions were required for complete obliteration of esophageal varices. Similarly, Baron et el [21] observed that median interval between EVL sessions was 5 weeks and patients required 3-4 sessions forcomplete obliteration of esophageal varices. Lo et al [22] observed that average number of sessions required for complete obliteration of esophageal varices was 3.8 $\pm$ 0.4.

In our study, 11 patients (33%) had retrosternal pain and 3 patients (9%) had fever. Sakthivel, H. et al. [11] reported in a study on 30 patients where 3 had retrosternal pain, 2 had fever, and 1 had superficial ulcer.

Conclusion

Portal hypertension is not an uncommon condition, can lead to development of oesophageal varices which can bleed and lead to mortality and morbidity. Thus, complete obliteration of oesophageal varices by repeated EVBL will save lives, which is inexpensive, easily availablemethod with minimal complications and side effects. Our study concluded that 2 or 3 sessions of EVBL can obliterate all the varices of any grade or severity in 8 to 12 weeks, at 4weekly interval endoscopy. However, further follow up is needed at every 6 months to evaluate the recurrence or new development of oesophageal varices in future where again EVBL can be done.

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