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Esophageal Motility Changes Following Endoscopic Variceal Band Ligation in Cirrhotic Patients with Esophageal Varices: A Prospective High-Resolution Manometry Study Using Chicago Classification v4.0

Short Running Title: Esophageal Motility Changes After EVBL

Awan Verma[1], Saurabh Singhal[2], Abhishek Yadav[3]

[1]Post-Graduate Department of General Medicine, Subharti Medical College and Chhatrapati Shivaji Subharti Hospital, Swami Vivekanand Subharti University, Meerut, Uttar Pradesh, India
Corresponding Author Email: *awanverma[at]gmail.com*

[2]Post-Graduate Department of General Medicine, Subharti Medical College and Chhatrapati Shivaji Subharti Hospital, Swami Vivekanand Subharti University, Meerut, Uttar Pradesh, India

[3]Department of Gastroenterology, Subharti Medical College, Swami Vivekanand Subharti University, Meerut, Uttar Pradesh, India

Abstract: ***Endoscopic variceal band ligation (EVBL) is the standard endoscopic treatment for esophageal varices in cirrhosis, but its effect on esophageal motility, when assessed using high-resolution manometry (HRM) and interpreted by the Chicago Classification version 4.0 (CCv4.0), remains poorly characterised. We aimed to evaluate esophageal motility changes before and after EVBL in cirrhotic patients with esophageal varices. In this prospective observational study conducted at a tertiary care centre in northern India, 26 consecutive cirrhotic patients with grade 2–3 esophageal varices scheduled for EVBL underwent HRM before the procedure and again 4–6 weeks afterward. Lower esophageal sphincter (LES) and upper esophageal sphincter (UES) pressures, integrated relaxation pressure (IRP), and esophageal body motility parameters were compared using paired t-tests or Wilcoxon signed-rank tests as appropriate. Categorical HRM diagnoses were compared using McNemar's test. Subgroup analysis was performed by Child-Pugh class, and correlation between the number of bands applied and change in IRP (ΔIRP) was assessed using Pearson and Spearman coefficients. Mean LES basal pressure rose from 30.13 ± 8.12 to 39.53 ± 9.53 mmHg (Δ +9.40 mmHg; p<0.0001), median IRP increased from 12.82 ± 4.16 to 21.70 ± 4.84 mmHg (Δ +8.88 mmHg; p<0.0001), and UES basal pressure increased from 82.82 ± 14.51 to 102.00 ± 28.83 mmHg (Δ +19.18 mmHg; p=0.0002). Esophagogastric junction outflow obstruction (EGJOO) on HRM was newly identified in 88.5% of patients post-EVBL, compared with 11.5% pre-EVBL (McNemar p<0.0001), with no patient reverting from EGJOO to a normal pattern. Esophageal body motility parameters (failed, ineffective, and intact swallow percentages; premature contractions; panesophageal pressurisation) did not differ significantly between the two time points. The rise in IRP was statistically significant across all Child-Pugh classes (A, B, and C) but was numerically attenuated in Class C. The number of bands applied did not correlate with the magnitude of ΔIRP (Pearson r=0.04, p=0.86). EVBL is not motility-neutral: it produces a substantial, consistent, and largely focal rise in esophagogastric junction resistance detectable by HRM, meeting manometric criteria for EGJOO in most patients, while esophageal body peristalsis remains preserved. These findings, obtained using contemporary Chicago Classification v4.0 criteria, provide a measurable physiological explanation for post-EVBL dysphagia and chest pain, and support consideration of HRM in symptomatic patients after variceal band ligation.***

Keywords: esophageal variceal band ligation; high-resolution manometry; Chicago Classification; esophagogastric junction outflow obstruction; liver cirrhosis; portal hypertension

1. Introduction

Liver cirrhosis is the end stage of chronic hepatic injury, characterised by diffuse fibrosis, nodular regeneration, and distortion of hepatic vascular architecture that raises intrahepatic resistance and produces portal hypertension.[1] Sustained elevation of portal pressure drives the formation of portosystemic collaterals, of which esophageal varices are the most clinically important, since their rupture causes life-threatening haemorrhage and remains a leading cause of morbidity and mortality in cirrhotic patients.[2],[3]

Endoscopic variceal band ligation (EVBL) is the established endoscopic modality for both primary and secondary prophylaxis of variceal bleeding. The technique mechanically strangulates variceal columns with elastic bands, inducing thrombosis and eventual fibrotic obliteration. Although EVBL is directed at vascular pathology, the procedure inevitably captures adjacent mucosa and submucosa, producing localised ischaemic ulceration that heals with granulation tissue and fibrosis over several weeks. Because esophageal varices and the bands used to eradicate them are concentrated in the distal esophagus and at the esophagogastric junction (EGJ), this healing response has the potential to alter the mechanical and functional properties of the lower esophageal sphincter (LES).

Earlier studies using conventional water-perfused manometry generally suggested that EVBL was motility-neutral or even beneficial, reporting normalisation of peristaltic amplitude and no clinically significant derangement of esophageal function. However, conventional manometry has limited spatial resolution and may fail to detect focal, clinically relevant abnormalities at the EGJ. The introduction of high-resolution manometry (HRM), interpreted using the Chicago Classification, has substantially improved the sensitivity and reproducibility of esophageal motility assessment, and the most recent iteration, Chicago Classification version 4.0 (CCv4.0), has refined the criteria for esophagogastric junction outflow obstruction (EGJOO) to reduce overdiagnosis.

To date, few prospective studies have used HRM with CCv4.0 criteria to characterise motility changes after EVBL, and none, to our knowledge, has done so in an Indian cohort. Given the high burden of cirrhosis-related variceal disease in South Asia and the wide use of EVBL in routine practice, we undertook this prospective study to comprehensively evaluate the impact of EVBL on esophageal motility using paired pre- and post-procedural HRM, and to explore associations with liver disease severity and the number of bands applied.

The aim of this study was to comprehensively evaluate the impact of endoscopic variceal band ligation on esophageal motility by comparing pre- and post-procedural high-resolution manometry parameters in patients with cirrhosis and esophageal varices. The specific objectives were: (1) to evaluate the effect of EVBL on LES and UES pressure parameters;

(2) to determine the prevalence and pattern of HRM-defined motility disorders, including EGJOO, following EVBL; (3) to assess the correlation between motility changes and clinical/laboratory variables including Child-Pugh class, variceal grade, and number of bands applied; and (4) to compare esophageal body motility indices before and after EVBL.

2. Materials and Methods

This was a prospective, observational, single-centre study conducted in the Department of General Medicine and Gastroenterology, Chhatrapati Shivaji Subharti Hospital, Subharti Medical College, Meerut, Uttar Pradesh, India. The study protocol was approved by the Institutional Review Board of Swami Vivekanand Subharti University, and the study was conducted in accordance with the ethical principles of the Declaration of Helsinki and ICMR guidelines. Written informed consent was obtained from all participants.

Consecutive patients aged 18 years or older with liver cirrhosis and esophageal varices (grade 2, 3, or 4) scheduled to undergo EVBL were recruited. Patients were excluded if they had an esophageal stricture or recent esophageal surgery; prior endoscopic intervention for varices (sclerotherapy or EVBL); severe haemodynamic instability precluding HRM; a known primary esophageal motility disorder (e.g., achalasia, diffuse esophageal spasm) diagnosed before EVBL; or significant comorbid conditions independently affecting esophageal motility (e.g., advanced systemic sclerosis).

The sample size was calculated based on data from a previous study reporting a paired change in lower esophageal sphincter pressure after endoscopic variceal ligation (mean difference ≈3.4 mmHg, pooled SD ≈6.2 mmHg). Using these estimates with 95% confidence and 80% power, a minimum of 26 patients was calculated as adequate to detect a clinically meaningful change in esophageal motility parameters.

At baseline, demographic data, medical history, anthropometric measurements, and laboratory investigations (liver function tests, complete blood count, coagulation profile) were recorded. Upper gastrointestinal endoscopy was performed to assess variceal grade, location, and presence of hiatus hernia, and the Child-Pugh score and class were calculated for each patient.

High-resolution esophageal manometry was performed using a solid-state catheter with closely spaced circumferential sensors. Baseline (pre-EVBL) HRM was performed before the banding procedure using a standard protocol of 10 wet swallows of 5 mL water in the supine position. Parameters recorded included LES basal and residual pressure/IRP, UES basal and residual pressures, and the percentage of failed, weak, ineffective, premature, fragmented, and intact swallows. EVBL was performed as per standard clinical practice using a multiband ligator device, with the number of bands determined by the endoscopist based on variceal size and distribution. Post-EVBL HRM was performed 4–6 weeks after the banding procedure, using the same protocol and equipment, to allow the acute inflammatory phase to subside before assessing motility change.

The primary outcome was the change in LES residual pressure/IRP before and after EVBL. Secondary outcomes included changes in LES basal pressure, UES parameters, esophageal body motility indices, and the proportion of patients developing HRM-defined EGJOO after the procedure, classified according to Chicago Classification v4.0 criteria.

Data were analysed using Python (SciPy v1.11). Continuous variables were tested for normality using the Shapiro-Wilk test. Paired comparisons employed the paired t-test for normally distributed data and the Wilcoxon signed-rank test for non- normally distributed data. The shift in categorical HRM diagnosis (normal versus EGJOO) was evaluated using McNemar's test with continuity correction. Correlations between continuous variables were assessed using Pearson's or Spearman's coefficients, as appropriate. Subgroup analyses by Child-Pugh class employed paired t-tests within each stratum. Statistical significance was set at a two-tailed $p<0.05$.

3. Results

A total of 26 patients completed both pre- and post-EVBL HRM assessments and formed the analytic dataset.
The mean age of the study population was 49.5 ± 14.7 years (range 28–78 years), with the majority (69.3%) aged between

30 and 60 years. Males predominated markedly, comprising 24 of 26 patients (92.3%). Grade 3 varices were present in 18 patients (69.2%) and grade 2 varices in 8 (30.8%); no patient had grade 4 varices. The mean number of bands applied per session was 3.85 ± 1.05 (range 2–6). By Child-Pugh classification, 7 patients (26.9%) were Class A, 12 (46.2%) Class B, and 7 (26.9%) Class C. Biochemical parameters were consistent with moderately advanced chronic liver disease, including hyperbilirubinaemia (mean total bilirubin 4.41 ± 4.70 mg/dL), elevated aminotransferases (AST 119.2 ± 111.4 IU/L; ALT 68.9 ± 71.1 IU/L), hypoalbuminaemia (2.53 ± 0.47 g/dL), prolonged INR (2.02 ± 1.07), and thrombocytopenia (127.2 ± 40.0 ×10³/μL). Full demographic and clinical characteristics are shown in Table 1.

Table 1: Demographic and baseline clinical characteristics (n=26)

Parameter	Value
Age (years), mean ± SD	49.5 ± 14.7
Age range	28–78 years
Sex (Male / Female)	24 / 2 (92.3% / 7.7%)
Variceal grade: Grade 2 / Grade 3	8 (30.8%) / 18 (69.2%)
Number of bands, mean ± SD	3.85 ± 1.05 (range 2–6)
Child-Pugh Class A / B / C	7 (26.9%) / 12 (46.2%) / 7 (26.9%)
Child-Pugh score, mean ± SD	8.1 ± 1.9
Total bilirubin (mg/dL), mean ± SD	4.41 ± 4.70
AST (IU/L), mean ± SD	119.2 ± 111.4
ALT (IU/L), mean ± SD	68.9 ± 71.1
Albumin (g/dL), mean ± SD	2.53 ± 0.47
INR, mean ± SD	2.02 ± 1.07
Platelet count (×10³/μL), mean ± SD	127.2 ± 40.0

Before EVBL, mean LES basal pressure was 30.13 ± 8.12 mmHg and median IRP was 12.82 ± 4.16 mmHg; 23 of 26 patients (88.5%) had a normal HRM study and 3 (11.5%) met criteria for EGJOO (Table 2). After EVBL, LES basal pressure rose to 39.53 ± 9.53 mmHg and IRP rose to 21.70 ± 4.84 mmHg, with the proportion meeting EGJOO criteria increasing to 23 of 26 patients (88.5%), while only 3 (11.5%) retained a normal pattern (Table 3).

Table 2: Pre-EVBL HRM parameters (n=26)

Parameter	Mean ± SD	Range
LES basal pressure (mmHg)	30.13 ± 8.12	13.4–48.5
UES basal pressure (mmHg)	82.82 ± 14.51	60.2–122.2
UES residual pressure (mmHg)	21.85 ± 5.04	12.0–33.6
Failed swallows (%)	8.85 ± 12.38	0–50
Ineffective swallows (%)	8.81 ± 12.60	0–50
Intact swallows (%)	91.19 ± 12.60	50–100
Premature contractions (%)	23.65 ± 23.76	0–92
Panesophageal pressurisation (%)	7.19 ± 10.64	0–40
Normal study, n (%)	23 (88.5%)	—
EGJOO, n (%)	3 (11.5%)	—

Table 3: Post-EVBL HRM parameters (n=26)

Parameter	Mean ± SD	Range
LES basal pressure (mmHg)	39.53 ± 9.53	19.4–62.2
Median IRP (mmHg)	21.70 ± 4.84	11.0–30.8
UES basal pressure (mmHg)	102.00 ± 28.83	61.8–224.6
UES residual pressure (mmHg)	35.70 ± 23.43	15.3–112.0
Failed swallows (%)	7.92 ± 14.06	0–67
Ineffective swallows (%)	7.04 ± 13.80	0–67
Intact swallows (%)	91.15 ± 15.37	25–100
Premature contractions (%)	20.81 ± 19.07	0–58
Panesophageal pressurisation (%)	6.69 ± 12.26	0–58
Normal study, n (%)	3 (11.5%)	—
EGJOO, n (%)	23 (88.5%)	—

EVBL produced a statistically significant increase in LES basal pressure (Δ +9.40 mmHg; 95% CI 6.24–12.56; p<0.0001), median IRP (Δ +8.88 mmHg; 95% CI 6.99–10.76; p<0.0001), and UES basal pressure (Δ +19.18 mmHg; 95% CI 10.55–27.81; p=0.0002). UES residual pressure also increased significantly (Δ +13.85 mmHg; p=0.0052), an effect driven partly by a small number of outliers. In contrast, esophageal body motility parameters- failed, ineffective, and intact swallow percentages, premature contractions, and panesophageal pressurization- did not differ significantly between pre- and post-EVBL studies (all p>0.05), indicating that peristaltic function was preserved despite the marked EGJ changes (Table 4).

Table 4: Paired statistical comparison of HRM parameters (n=26)

Parameter	Pre (Mean ± SD)	Post (Mean ± SD)	Δ Mean	p-value	Significance
LES basal pressure	30.13 ± 8.12	39.53 ± 9.53	9.4	<0.0001	Significant
Median IRP	12.82 ± 4.16	21.70 ± 4.84	8.88	<0.0001	Highly significant
UES basal pressure	82.82 ± 14.51	102.00 ± 28.83	19.18	0.0002	Significant
UES residual pressure	21.85 ± 5.04	35.70 ± 23.43	13.85	0.0052	Significant
Failed swallows (%)	8.85 ± 12.38	7.92 ± 14.06	-0.92	0.79	Not significant
Ineffective swallows (%)	8.81 ± 12.60	7.04 ± 13.80	-1.77	0.64	Not significant
Intact swallows (%)	91.19 ± 12.60	91.15 ± 15.37	-0.04	0.99	Not significant
Premature contractions (%)	23.65 ± 23.76	20.81 ± 19.07	-2.85	0.64	Not significant
Panesophageal pressurisation (%)	7.19 ± 10.64	6.69 ± 12.26	-0.5	0.88	Not significant
UES residual pressure	21.85 ± 5.04	35.70 ± 23.43	13.85	0.0052	Significant
Failed swallows (%)	8.85 ± 12.38	7.92 ± 14.06	-0.92	0.79	Not significant
Ineffective swallows (%)	8.81 ± 12.60	7.04 ± 13.80	-1.77	0.64	Not significant
Intact swallows (%)	91.19 ± 12.60	91.15 ± 15.37	-0.04	0.99	Not significant
Premature contractions (%)	23.65 ± 23.76	20.81 ± 19.07	-2.85	0.64	Not significant
Panesophageal pressurisation (%)	7.19 ± 10.64	6.69 ± 12.26	-0.5	0.88	Not significant

Of the 26 patients, 20 shifted from a normal HRM impression pre-EVBL to EGJOO post-EVBL, 3 remained EGJOO at both time points, and 3 remained normal at both time points; no patient reverted from EGJOO to normal (Table 5). McNemar's test with continuity correction confirmed that this shift was highly significant (p<0.0001), and its strictly unidirectional nature- occurring only from normal toward EGJOO- supports a causal link between band ligation and the

development of an EGJOO pattern rather than random temporal variability.

Table 5: HRM impression before and after EVBL (n=26)

HRM impression	Pre-EVBL, n (%)	Post-EVBL, n (%)
Normal	23 (88.5%)	3 (11.5%)
EGJ outflow obstruction (EGJOO)	3 (11.5%)	23 (88.5%)

The rise in IRP after EVBL was statistically significant in all three Child-Pugh strata: Class A (13.03 ± 6.28 to 22.14 ± 5.66 mmHg; Δ +9.11 mmHg; p=0.010), Class B (12.84 ± 4.08 to 23.18 ± 4.13 mmHg; Δ +10.33 mmHg; p<0.001), and Class C (12.59 ± 1.38 to 18.73 ± 4.39 mmHg; Δ +6.14 mmHg; p=0.008) (Table 6). Baseline IRP was similar across classes, but the magnitude of post-procedural rise was numerically greatest in Class B and smallest in Class C, suggesting that the effect of EVBL on EGJ resistance is robust across the spectrum of liver disease severity, though somewhat attenuated in the most advanced disease.

Table 6: IRP changes by Child-Pugh class

Child Pugh Class	a	Pre- EVBL IRP (Mean ± SD)	Post- EVBL IRP (Mean ± SD)	p-Value
Class A	7	13.03 ± 6.28	22.14 ± 5.66	0.010
Class B	12	12.84 ± 4.08	23.18 ± 4.13	<0.001
Class C	7	12.59 ± 1.38	18.73 ± 4.39	0.008

No significant correlation was found between the number of bands applied and the magnitude of IRP change (Pearson r=0.04, p=0.86; Spearman ρ=−0.01, p=0.98). Linear regression demonstrated a negligible, non-significant slope (ΔIRP = 0.18 × number of bands + 8.20; R^2=0.001), indicating that within the range of bands used in this cohort (2–6 per session), the extent of post-procedural IRP elevation was largely independent of the absolute number of bands deployed, and likely reflects other factors such as banding location relative to the EGJ, tissue captured per band, and individual healing response.

4. Discussion

This prospective study used HRM interpreted by CCv4.0 criteria to characterise esophageal motility change after EVBL in 26 cirrhotic patients with grade 2–3 varices, and, to our knowledge, is among the first such studies from an Indian cohort. Five principal findings emerged: EVBL produced a substantial and significant rise in LES basal pressure, IRP, and UES basal pressure; this translated into a dramatic categorical shift in HRM diagnosis, with EGJOO newly identified in 88.5% of patients versus a baseline prevalence of 11.5%; esophageal body peristalsis remained largely intact, localising the dysfunction to the EGJ; the rise in IRP was significant across all Child-Pugh strata, though attenuated in Class C; and no correlation was observed between the number of bands applied and the magnitude of IRP change.

Our findings diverge from earlier literature based on conventional water-perfused manometry, which generally reported normalisation or reduction of LES tone after variceal ligation and concluded that EVBL was largely motility-neutral. [12] Conventional manometry has limited spatial resolution and is known to underestimate focal EGJ events; the transition to closely spaced, solid-state HRM catheters and Chicago Classification metrics provides a far more sensitive readout of subtle sphincteric dysfunction, as previously demonstrated for post-sclerotherapy motility assessment.[13] Our results suggest that this enhanced sensitivity reveals previously underappreciated obstructive physiology after band ligation, and that earlier conclusions of motility neutrality warrant re-examination using HRM-based methodology.

The concurrent rise in UES basal pressure, despite the UES being anatomically remote from the banding site, was an unexpected finding. A purely mechanical explanation is implausible given its separate innervation via the vagus and pharyngeal branches; a reflex compensatory response to perceived distal outflow obstruction, mediated through vago-vagal pathways, or generalised sphincter hypertonicity related to post-procedural discomfort, are more plausible explanations, and this observation merits further mechanistic study.

The pathophysiology of post-EVBL EGJ outflow obstruction is likely multifactorial. EVBL deliberately induces ischaemic necrosis of the variceal column and adjacent mucosa/submucosa, followed by ulceration, granulation, and fibrosis, a process that is well documented on follow-up endoscopy.[1] Because varices and the bands used to eradicate them are concentrated in the distal esophagus and cardia, the resulting fibrotic remodelling directly involves the LES, plausibly reducing wall compliance and elevating both basal tone and the pressure required for deglutitive relaxation. A contribution from persistent low-grade inflammation is also plausible, although our decision to perform post-procedural HRM at 4–6 weeks- beyond the acute inflammatory window- argues that the changes observed reflect durable structural and functional remodelling rather than transient oedema. A third potential contributor is disruption of inhibitory (nitrergic) neurotransmission at the EGJ secondary to local ischaemic injury, which could plausibly elevate IRP independent of pure mechanical stiffening.

The near-complete inversion of HRM diagnostic categories- from 88.5% normal pre-EVBL to 88.5% EGJOO post-EVBL- was the most striking categorical finding, and its strictly unidirectional pattern strongly supports causality. However, CCv4.0 deliberately tightened criteria for clinically conclusive EGJOO, requiring elevated IRP in both supine and upright positions, supportive symptoms, and confirmatory testing (timed barium esophagogram or functional lumen imaging probe) before the diagnosis is considered clinically actionable; manometry alone yields a "clinically inconclusive" categorisation in the absence of these features.[1] ,[1] The post-EVBL EGJOO pattern observed here is therefore best understood as a secondary, mechanically induced manometric phenotype rather than a primary motility disorder such as achalasia, and does not by

itself warrant LES-directed therapy such as pneumatic dilatation. Nonetheless, it provides a plausible, measurable physiological substrate for the dysphagia, chest pain, and odynophagia long recognised after EVBL but generally attributed to non-specific post-procedural inflammation.

In contrast to the pronounced EGJ changes, esophageal body motility parameters were essentially unchanged after EVBL. This is biologically coherent: band ligation acts focally on distal mucosa and submucosa rather than on the distributed enteric circuitry that generates peristalsis along the esophageal length, and no patient developed a major peristaltic disorder (e.g., absent contractility or distal esophageal spasm) after the procedure. This pattern — elevated IRP with preserved peristalsis- corresponds precisely to the CCv4.0 category of EGJOO without disordered peristalsis.

The rise in IRP was significant across all Child-Pugh classes, indicating that the effect of EVBL on EGJ resistance is robust across the spectrum of liver disease severity, though numerically smaller in Class C, possibly reflecting autonomic neuropathy or reduced baseline smooth-muscle responsiveness in advanced cirrhosis. The absence of a dose–response relationship between band count and ΔIRP suggests that, within the 2–6 band range used clinically, factors such as banding location relative to the EGJ, tissue volume captured per band, and individual fibrotic healing response are likely to be more physiologically important than the raw number of bands placed. This does not diminish the established endoscopic principle of using the minimum number of bands necessary for safe variceal obliteration, which remains appropriate on independent safety grounds.

These findings suggest that patients with persistent or disproportionate dysphagia, chest pain, or odynophagia after EVBL- particularly when endoscopy shows no overt stricture- should be considered for manometric evaluation rather than having their symptoms attributed by default to non-specific post-procedural inflammation. Combined HRM and endoscopic assessment is likely to be more informative than either modality alone, and where clinically significant EGJOO with disabling symptoms is suspected, confirmatory testing (timed barium esophagogram or FLIP) in line with CCv4.0 recommendations would help establish clinical relevance. In cirrhotic patients already at risk of sarcopenia and reduced oral intake, any additional impediment to bolus transit may carry disproportionate nutritional consequences, supporting attention to nutritional status in symptomatic patients after EVBL.

This study has several limitations. First, the sample size, although adequate for the primary manometric comparison based on a priori calculation, was modest and drawn from a single tertiary centre, which may limit generalisability. Second, HRM was performed only in the supine position; upright manometry and provocative testing (rapid drink challenge, timed barium esophagogram, or FLIP), which are recommended by CCv4.0 to confirm clinically relevant EGJOO, were not performed. Third, structured symptom assessment using validated dysphagia or reflux instruments was not incorporated, so we cannot directly correlate the manometric findings with patient-reported symptoms. Fourth, the single post-procedural time point (4–6 weeks) does not allow characterisation of the longer-term trajectory of post-EVBL EGJOO- whether it persists, partially resolves, or worsens with repeated banding sessions. Finally, detailed procedural metadata (precise band location relative to the EGJ, technique of tissue capture) were not systematically recorded, limiting our ability to identify which procedural factors most strongly influence the manometric outcome.

5. Conclusion

In this prospective HRM-based study of 26 cirrhotic patients undergoing EVBL, band ligation produced significant and clinically relevant elevations in LES basal pressure, IRP, and UES basal pressure, with 88.5% of patients newly meeting manometric criteria for EGJOO post-procedure compared with 11.5% at baseline, and no patient reverting from EGJOO to normal. Esophageal body motility remained preserved, confirming that the dysfunction is anatomically focal to the EGJ. The magnitude of IRP elevation was significant across all Child-Pugh classes and did not correlate with the number of bands applied. These findings indicate that EVBL is not motility-neutral and provide a measurable physiological basis for post- EVBL dysphagia, chest pain, and odynophagia. HRM should be considered in symptomatic patients after EVBL alongside conventional endoscopy, and larger multi-centre longitudinal studies incorporating upright manometry, provocative testing, and structured symptom evaluation are needed to define the long-term trajectory of this phenomenon and its relationship to clinical outcomes.

Declarations

The study protocol was approved by the Institutional Review Board of Swami Vivekanand Subharti University, Meerut. Written informed consent was obtained from all participants, and the study was conducted in accordance with the Declaration of Helsinki and ICMR ethical guidelines.

Not applicable.

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

The authors declare no conflict of interest.

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AV: conception and design, data acquisition, analysis, drafting of manuscript. SS: study supervision, guidance, critical revision. AY: gastroenterology/endoscopic expertise, critical revision. All authors read and approved the final manuscript.

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