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Original Research Article

Esophagogastric Junction Outflow Obstruction After Corrosive Esophageal Injury

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Conflict of interest: Nil

Abstract

Background: This study evaluated symptomatic patients after corrosive esophageal injury who had normal follow-up endoscopy, with a focus on patients with esophagogastric junction outflow obstruction (EGJOO) identified on high-resolution manometry.

Aim: To characterize the clinical and high-resolution manometry profile of esophagogastric junction outflow obstruction among symptomatic adult patients after corrosive esophageal injury with normal six-week follow-up esophagogastroduodenoscopy.

Methods: This subgroup analysis was derived from a cross-sectional observational cohort of 100 adult patients with a history of corrosive ingestion. All patients underwent initial endoscopy within three days of ingestion and repeat endoscopy at six weeks. Patients with persistent esophageal symptoms despite normal six-week endoscopy underwent high-resolution esophageal manometry. EGJOO was identified from high-resolution manometry findings. Clinical variables, nature of corrosive agent, initial Zargar grade, contraction vigour, contraction pattern, intrabolus pressure pattern, basal lower esophageal sphincter pressure, integrated relaxation pressure, distal latency, and distal contractile integral were analyzed.

Results: Esophagogastric junction outflow obstruction was identified in nine of 100 patients. All nine patients had dysphagia. Acid ingestion was documented in five patients and alkali ingestion in four. Initial Zargar grade was 2A in six patients and 3A in three. All nine patients showed normal contraction vigour and intact contraction pattern. Esophagogastric junction intrabolus pressurization (EGJ pressurization) was present in four patients. Mean basal lower esophageal sphincter pressure was 33.06 ± 8.49 mmHg and mean integrated relaxation pressure was 26.07 ± 3.56 mmHg.

Conclusion: Esophagogastric junction outflow obstruction constituted a distinct post-corrosive physiology characterized by dysphagia, preserved contraction pattern, elevated basal lower esophageal sphincter pressure, elevated integrated relaxation pressure, and selective esophagogastric junction pressurization. Functional testing should be considered when post-corrosive dysphagia persists despite normal follow-up endoscopy.

Keywords: Corrosive Ingestion; Dysphagia; Esophagogastric Junction Outflow Obstruction; High-Resolution Manometry; Integrated Relaxation Pressure; Lower Esophageal Sphincter; Post-Corrosive Injury.

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Introduction

Corrosive ingestion remains an important cause of acute upper gastrointestinal injury and chronic esophageal morbidity. The degree of injury depends on the corrosive agent, concentration, quantity ingested, contact time, and depth of tissue involvement [1,3,4]. Early endoscopy has a central role in documenting mucosal injury, prognosticating complications, and applying the Zargar classification (table 1) system, which remains widely used for grading corrosive burns [1,2,5]. Adult corrosive ingestion differs from accidental pediatric ingestion because it is often intentional, involves larger volumes, and is associated with more severe injury and long-term sequelae [3,4]. The conventional long-term focus after corrosive ingestion is stricture formation. However, symptoms after corrosive injury are not always explained by a residual luminal narrowing. Corrosive burns can injure the muscularis propria and the intramural neural plexuses, leading to

impaired peristalsis, altered lower esophageal sphincter function, and abnormal esophagogastric junction physiology [6-9]. Dantas and Mamede reported impaired esophageal motility in patients with previous caustic injury, and subsequent work using manometry in caustic strictures also demonstrated that dysphagia may persist even after a mechanically adequate lumen is restored [6,10].

High-resolution manometry provides spatially detailed pressure topography of esophageal body contraction and esophagogastric junction relaxation. It has become the standard physiological test for classifying esophageal motility disorders and is recommended in the evaluation of non-obstructive dysphagia after structural disease has been excluded [11-17]. The Chicago Classification version 4.0 refined diagnostic criteria for manometric disorders and emphasized that esophagogastric junction outflow obstruction should be interpreted in the context of symptoms and supportive evidence rather than as an isolated numerical abnormality [11,12].

Esophagogastric junction outflow obstruction is clinically important in post-corrosive disease because it differs mechanistically from hypocontractile disorders. In ineffective esophageal motility or absent contractility, dysphagia is driven mainly by weak or absent propulsion. In esophagogastric junction outflow obstruction, the esophageal body may generate preserved contractions, but impaired relaxation or opening at the esophagogastric junction produces outflow resistance, elevated integrated relaxation pressure, and sometimes intrabolus pressurization [11,15,16]. Caustic injury has also been associated with achalasia-like physiology in older literature, reinforcing the biological plausibility of a post-injury outflow phenotype [18]. The present manuscript was designed as a focused subgroup analysis of esophagogastric junction outflow obstruction from a parent cohort of symptomatic post-corrosive patients who underwent high-resolution manometry after normal follow-up endoscopy. The analysis was intentionally restricted to esophagogastric junction outflow physiology to avoid overlap with broader phenotype-distribution and symptom-correlation manuscripts from the same cohort.

Materials and Methods

Study design and setting: This was a focused subgroup analysis of a cross-sectional observational study conducted in the Department of Medical Gastroenterology, Gandhi Medical College Hospital, Secunderabad. The parent study evaluated high-resolution manometry findings in 100 adult patients after corrosive ingestion.

Study Population: The parent cohort included adult patients aged more than 18 years with a history of corrosive ingestion, persistent esophageal symptoms, and normal follow-up esophagogastroduodenoscopy at six weeks. The present analysis included only patients whose manometric diagnosis was esophagogastric junction outflow obstruction (EGJOO).

Eligibility Criteria: Patients were eligible for the parent study if they were aged more than 18 years, had a history of corrosive ingestion, had persistent esophageal symptoms, and had normal esophagogastroduodenoscopy at six weeks.

Pregnant women, clinically unstable patients, patients with esophageal strictures, patients with previous foregut surgery, and patients unwilling to provide consent were excluded.

Clinical and endoscopic assessment: Detailed clinical history, nature of corrosive agent, comorbidity status, and predominant symptoms were recorded. Initial esophagogastroduodenoscopy was performed within three days of corrosive ingestion to grade acute injury according to the Zargar classification, as shown in table 1 [1,2,5].

Repeat esophagogastroduodenoscopy was performed at six weeks. Endoscopic assessment was performed with a diagnostic upper gastrointestinal video endoscope system (Olympus CV-150 series, Olympus Corporation, Tokyo, Japan) with GIF-Q150.

Table 1: Zargar Endoscopic Classification of Corrosive Upper Gastrointestinal Injury

Zargar grade	Endoscopic finding
Grade 0	Normal mucosa
Grade 1	Edema and erythema of mucosa
Grade 2A	Hemorrhage, erosions, blisters, exudates, superficial ulcerations
Grade 2B	Deep discrete ulcerations or circumferential ulcerations
Grade 3A	Focal areas of necrosis with deep gray or brownish-black ulcers
Grade 3B	Extensive necrosis
Grade 4	Perforation

High-resolution manometry protocol and equipment: High-resolution esophageal manometry was performed using a 16-channel water-perfused manometry system (KangarooJeff, Royal Melbourne Hospital, Australia). The recordings were analyzed using Trace software, and manometric diagnoses were assigned according to Chicago Classification version 4.0.

A transnasal catheter with multiple circumferential pressure sensors was positioned to record pressures across the pharynx, esophageal body, lower esophageal sphincter, and proximal stomach. Recorded variables included basal lower esophageal sphincter pressure, integrated relaxation pressure, distal latency, distal contractile integral, contraction vigour, contraction pattern, and intrabolus pressure pattern.

Diagnostic criteria for EGJOO: Manometric interpretation was based on the Chicago Classification version 4.0 framework and related high-resolution manometry methodology [11,12,14-17].

EGJOO was defined as impaired esophagogastric junction relaxation with preserved esophageal body contraction pattern in a symptomatic patient, after exclusion of visible stricture on six-week follow-up endoscopy.

Because Chicago Classification version 4.0 recommends clinical correlation and supportive testing for conclusive EGJOO, this manuscript uses the term as a high-resolution manometry subgroup phenotype within a post-corrosive symptomatic cohort [11-13].

Software and statistical analysis: Data were entered into Microsoft Excel (Microsoft Corporation, Redmond, WA, USA) and analyzed using IBM SPSS Statistics (IBM Corp., Armonk, NY, USA). Categorical variables were expressed as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD). Associations between categorical variables were analyzed using the Chi-square test or Fisher's exact test, as appropriate. Comparisons of continuous variables were performed using the independent-samples t-test or one-way analysis of variance (ANOVA), as appropriate. A two-sided p-value <0.05 was considered statistically significant.

Ethical considerations: The study was approved by the Institutional Ethics Committee of Gandhi Medical College Hospital, Secunderabad. Written informed consent was obtained from all participants.

Results

Frequency and clinical profile of esophagogastric junction outflow obstruction: Esophagogastric junction outflow obstruction was identified in 9 of 100 patients in the parent symptomatic post-corrosive cohort. All nine patients with EGJOO obstruction had dysphagia as the predominant symptom. Acid ingestion was documented in five patients and alkali ingestion in four patients. Initial endoscopic injury grade was Zargar 2A in six patients and Zargar 3A in three patients. All patients in the EGJOO subgroup had normal contraction vigour and intact contraction pattern. The detailed clinical and manometric profile is shown in Table 2.

Table 2: Clinical and manometric profile of the esophagogastric junction outflow obstruction subgroup

Variable	n/N	Percentage
EGJOO cases in parent cohort	9/100	9.0
Predominant symptom: Dysphagia	9/9	100.0
Acid ingestion	5/9	55.6
Alkali ingestion	4/9	44.4
Comorbidities among study subjects	0/9	0.0
Initial Zargar grade 2A among EGJOO cases	6/9	66.7
Initial Zargar grade 3A among EGJOO cases	3/9	33.3
Normal contraction vigour	9/9	100.0
Intact contraction pattern	9/9	100.0
EGJ Pressurization	4/9	44.4

Values are presented as n/N and percentage. EGJOO: Esophagogastric junction outflow obstruction.

Quantitative manometric profile: EGJOO subgroup showed a distinct obstructive physiology. Basal lower esophageal sphincter pressure (LES pressure) and integrated relaxation pressure (IRP) were markedly higher in patients with EGJOO than in the remaining cohort. Distal latency (DL) was preserved and not statistically different from the non-EGJOO obstruction group, supporting that the dominant abnormality was outflow physiology rather than premature contraction. The exploratory esophagogastric junction outflow obstruction versus non-esophagogastric junction outflow obstruction comparison is shown in Table 3.

Table 3: Exploratory comparison of EGJOO and non-EGJOO patients using selected high-resolution manometry metrics

Variable	EGJOO (n=9)	Non-EGJOO (n=91)	Statistical test and effect size
Basal LES pressure (mmHg)	33.06 ± 8.49	14.19 ± 5.66	t=6.52; df=8.72; p<0.001; Hedges g=3.15
IRP (mmHg)	26.07 ± 3.56	8.08 ± 1.88	t=14.95; df=8.44; p<0.001; Hedges g=8.64
DL (sec)	5.14 ± 0.25	5.00 ± 0.45	t=1.42; df=13.75; p=0.179; Hedges g=0.31
DCI (mmHg·s·cm)	2872.22 ± 1035.35	1033.30 ± 2172.92	t=4.45; df=16.21; p<0.001; Hedges g=0.87

Continuous values are presented as mean ± standard deviation. Comparisons were performed using Welch t test. Degrees of freedom are Welch-Satterthwaite degrees of freedom. Hedges g is reported as the effect size.

LES: Lower Esophageal Pressure, IRP: Integrated relaxation pressure, DL: Distal latency, DCI: Distal contractile integral, mmHg, millimeters of mercury; mmHg·s·cm, millimeter of mercury-seconds-centimeter.

Intrabolus pressure pattern: EGJ pressurization was seen in four of nine patients with EGJOO and in none of the 91 non-esophagogastric junction outflow obstruction patients. Thus, in this cohort, EGJ pressurization was highly specific for the EGJOO subgroup, although it was not present in every esophagogastric junction outflow obstruction case. The intrabolus pressure comparison is shown in Table 4.

Table 4: Intrabolus pressure pattern in esophagogastric junction outflow obstruction and non-esophagogastric junction outflow obstruction groups

Intrabolus pressure pattern	EGJOO (n=9)	Non-EGJOO (n=91)	Statistical test and effect size
EGJ pressurization	4 (44.4)	0 (0.0)	Fisher exact test p<0.001; odds ratio not finite because the non-EGJOO pressurization cell was zero; Pearson χ^2=42.13, df=1, φ=0.65
No EGJ pressurization	5 (55.6)	91 (100.0)	Included in same 2×2 Fisher exact comparison above

Categorical values are presented as n (%). Statistical comparison was performed using Fisher exact test for the 2×2 table. No table cell is left blank; the second row states that the row belongs to the same exact-test comparison. Phi (φ) is reported as an effect size from Pearson chi-square. EGJ: Esophagogastric junction

Comparison with other high-resolution manometry diagnostic categories: Across diagnostic categories, EGJOO showed the highest basal LES pressure and the highest IRP. Hypercontractile esophagus showed the highest distal contractile integral, whereas distal esophageal spasm (DES) had the shortest distal latency. These findings support EGJOO as a distinct post-corrosive physiological subgroup rather than a nonspecific dysmotility label. The diagnostic-category comparison is shown in Table 5.

Table 5: Selected high-resolution manometry metrics across diagnostic categories

HRM diagnosis	Basal LES pressure (mmHg)	IRP (mmHg)	DL(sec)	DCI(mmHg·s·cm)
Absent contractility	17.40 ± 4.69	9.14 ± 1.47	5.07 ± 0.31	82.86 ± 7.56
DES	14.84 ± 4.22	7.81 ± 1.25	3.77 ± 0.30	784.29 ± 133.52
EGJOO	33.06 ± 8.49	26.07 ± 3.56	5.14 ± 0.25	2872.22 ± 1035.35
Hypercontractile esophagus	17.47 ± 2.85	10.98 ± 1.05	5.20 ± 0.28	9083.33 ± 360.09
IEM	13.74 ± 6.07	7.47 ± 1.15	5.13 ± 0.24	257.50 ± 61.57
Normal study	13.21 ± 5.71	8.24 ± 2.45	5.06 ± 0.30	819.63 ± 167.78
One-way ANOVA result	F(5,94)=17.66; p<0.001; η^2=0.48	F(5,94)=149.73; p<0.001; η^2=0.89	F(5,94)=32.65; p<0.001; η^2=0.63	F(5,94)=829.10; p<0.001; η^2=0.98

Continuous values are presented as mean ± standard deviation. Overall comparisons were performed using one-way analysis of variance. F statistics are reported with between-group and within-group degrees of freedom as F(df1,df2). Eta-squared (η^2) is reported as the effect size. DES: Distal Esophageal Spasm, EGJOO: Esophagogastric junction outflow obstruction, IEM: Ineffective Esophageal Motility,mmHg, millimeters of mercury; sec, seconds; mmHg·s·cm, millimeter of mercury-seconds-centimeter.

Discussion

This focused subgroup analysis identifies esophagogastric junction outflow obstruction as a distinct physiological pattern after corrosive esophageal injury. Although it accounted for only nine percent of the symptomatic post-corrosive cohort, its profile was consistent and clinically meaningful: all patients had dysphagia, contraction vigour was preserved, contraction pattern was intact, basal lower esophageal sphincter pressure and integrated relaxation pressure were elevated, and esophagogastric junction pressurization was present in a subset. These findings support the concept that post-corrosive dysphagia can reflect functional obstruction at the esophagogastric junction even when follow-up endoscopy does not reveal a visible stricture.

The finding is biologically plausible. Corrosive injury can progress beyond the mucosa and submucosa to affect the muscularis propria and neural elements of the esophageal wall [4,6-9]. Previous conventional manometry and high-resolution manometry studies have shown that caustic injury can lead to severe esophageal motor abnormalities, including aperistalsis, segmental dysmotility, defective lower esophageal sphincter function, and persistent dysphagia despite restoration of luminal patency [6-10]. The present analysis adds a more specific observation: a subset of post-corrosive patients can preserve esophageal body contraction but develop manometric evidence of impaired esophagogastric junction relaxation.

The most striking quantitative finding was the elevation of IRP in the EGJOO subgroup. IRP is a central metric in Chicago Classification-based evaluation of esophagogastric junction relaxation [11,12,14]. In this study, the mean IRP in the EGJOO group was more than three times that of the non-EGJOO group. The very large effect size supports that this was not a marginal numerical difference but a defining feature of the subgroup. Basal LES pressure was also markedly higher, indicating a high-pressure junctional phenotype.

The preserved DL in the EGJOO subgroup helps separate it from DES. Chicago Classification interpretation emphasizes that premature contractions and outflow obstruction represent different physiological processes [11,12,16]. In the present analysis, DL in EGJOO was not significantly different from non-EGJOO cases, while DES had the shortest DL across diagnostic groups. This supports the interpretation that the dominant abnormality in EGJOO was impaired junctional relaxation rather than spastic timing.

Intrabolus pressure further strengthened the mechanistic interpretation. EGJ pressurization was seen only in the EGJOO subgroup. This is consistent with the physiological principle that bolus pressure rises upstream when resistance to emptying occurs at the esophagogastric junction [13,15,16]. However, only four of nine EGJOO cases demonstrated pressurization. Therefore, pressurization appears highly specific when present but insufficiently sensitive to exclude EGJOO when absent. This finding is aligned with Chicago Classification version 4.0, which recommends interpretation of EGJOO using symptoms and supportive evidence rather than relying on a single parameter [11-13].

Compared with the study by Armijo et al., which found substantial motility impairment after previous caustic stricture even when the lumen was open, the present analysis highlights a different but complementary phenotype [10]. Armijo et al. emphasized defective lower esophageal sphincter and absent peristalsis in a small high-resolution manometry series after caustic stenosis. Our EGJOO subgroup, in contrast, had preserved contraction vigour and intact contraction pattern, suggesting that caustic injury can lead to more than one pathway of functional impairment: hypocontractile failure in some patients and outflow resistance with preserved contractions in others.

The results also differ from classic absent-contractility descriptions after caustic injury. Dantas and Mamede demonstrated reduced esophageal body contraction amplitude after caustic injury using conventional manometry [6]. Genc and Mutaf reported motility changes during acute and late periods of caustic esophageal burns [8]. In the present subgroup, the problem was not weak esophageal body contraction. Instead, the high IRP, high LES pressure, and preserved peristaltic pattern suggest a localized esophagogastric junction outflow phenotype. This difference is clinically important because the management implications are not identical. Clinically, these findings suggest that persistent dysphagia after corrosive injury should not be dismissed when endoscopy is normal. High-resolution manometry is recommended for evaluation of obstructive esophageal symptoms when no mechanical cause is found on endoscopy [13,17]. In a post-corrosive patient, identification of EGJOO should prompt confirmation of clinical relevance. Timed barium esophagram with tablet, functional lumen imaging probe (FLIP) assessment,

or careful repeat endoscopic assessment may be useful to evaluate subtle narrowing, reduced compliance, or impaired esophagogastric junction opening [11-13].

Therapeutic decisions in post-corrosive esophagogastric junction outflow obstruction should be individualized. Standard treatments used for idiopathic outflow disorders may not translate directly to the post-corrosive setting because tissue planes may be fibrotic and the risk profile for dilation or myotomy may differ. Conservative management, repeat evaluation, graded dilation, botulinum toxin injection, pneumatic dilation, or myotomy should only be considered after confirming that the manometric pattern is clinically meaningful and anatomically actionable. The present study was not designed to assess treatment response, but it identifies a subgroup that deserves prospective outcome-focused evaluation.

Strengths

- The analysis was derived from a well-defined symptomatic post-corrosive cohort with normal follow-up endoscopy.
- All patients underwent HRM at a standardized post-injury time point.
- The EGJOO subgroup was characterized using both qualitative HRM patterns and quantitative manometric metrics.

Limitations

- This was a single-center subgroup analysis, and the EGJOO subgroup included only nine patients.
- The analysis was derived from a symptomatic cohort with normal six-week follow-up endoscopy; therefore, the findings should not be generalized to asymptomatic patients or patients with established strictures.
- Supportive tests recommended by Chicago Classification version 4.0, such as timed barium esophagram with tablet or functional lumen imaging probe assessment, were not available for confirmation of clinically conclusive EGJOO.
- Long-term follow-up and treatment-response data were not available, so the natural history of post-corrosive esophagogastric junction outflow obstruction could not be determined.

Conclusion

Esophagogastric junction outflow obstruction was identified in nine percent of symptomatic adult patients after corrosive esophageal injury despite normal six-week follow-up endoscopy. This subgroup was characterized by dysphagia, preserved contraction vigour, intact contraction pattern, elevated basal lower esophageal sphincter pressure, markedly elevated integrated relaxation pressure, and selective esophagogastric junction intrabolus pressurization. These findings support high-resolution manometry as an important functional test in post-corrosive patients with persistent dysphagia when endoscopy does not demonstrate stricture. Esophagogastric junction outflow obstruction should be recognized as a distinct post-corrosive physiological phenotype that may require confirmatory evaluation and individualized management.

Declarations

Ethics approval: The study was approved by the Institutional Ethics Committee of Gandhi Medical College Hospital, Secunderabad.

Consent: Written informed consent was obtained from all participants.

Conflicts of interest: The authors declare no conflicts of interest.

Funding: No external funding was received for this study.

Data availability: The data supporting the findings of this study are available from the corresponding author on reasonable request, subject to institutional and ethical permissions.

Related manuscript disclosure: This manuscript is a focused esophagogastric junction outflow obstruction subgroup analysis from a parent observational cohort evaluating high-resolution manometry after corrosive esophageal injury.

The present paper addresses a distinct mechanistic question focused on esophagogastric junction outflow physiology and does not reproduce the broad high-resolution manometry phenotype table or the symptom-by-diagnosis correlation table from related manuscripts derived from the same parent cohort.

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