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Functional Esophagogastric Junction Obstruction with Intact Peristalsis: A Heterogeneous Syndrome Sometimes Akin to Achalasia

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Abstract

Background—Some patients with suspected achalasia are found on manometry to have preserved peristalsis, thereby excluding that diagnosis. This study evaluated a series of such patients with functional esophagogastric junction (EGJ) obstruction.

Methods—Among 1,000 consecutive high-resolution manometry studies, 16 patients had functional EGJ obstruction characterized by impaired EGJ relaxation and intact peristalsis. Eight patients with post-fundoplication dysphagia and similarly impaired EGJ relaxation were studied as a comparator group with mechanical obstruction. Intrabolar pressure (IBP) was measured 1 cm proximal to the EGJ. Sixty-eight normal controls were used to define normal IBP. Patients' clinical features were evaluated.

Results—Functional EGJ obstruction patients presented with dysphagia (96%) and/or chest pain (42%). IBP was significantly elevated in idiopathic and post-fundoplication dysphagia patients versus controls. Among the idiopathic EGJ obstruction group treated with pneumatic dilation, BoTox™, or Heller myotomy, only the three treated with Heller myotomy responded well. Among the post-fundoplication dysphagia patients, three of four responded well to redo operations.

Conclusion—Functional EGJ obstruction is characterized by pressure topography metrics demonstrating EGJ outflow obstruction of magnitude comparable to that seen with post-fundoplication dysphagia. Affected patients experience dysphagia and/or chest pain. In some cases, functional EGJ obstruction may represent an incomplete achalasia syndrome.

Keywords

Esophagus; Achalasia; Dysphagia; Manometry

Introduction

The physiological defects in achalasia are attributable to loss of function by myenteric plexus ganglion cells, particularly inhibitory neurons.¹ However, because achalasia is rarely diagnosed on the basis of histopathology, this neural defect is usually inferred from its functional consequences rather than directly demonstrated. Hence, achalasia is diagnosed by demonstrating impaired esophagogastric junction (EGJ) relaxation and absent peristalsis without an obstructing lesion to otherwise explain these anomalies.² However, it is also clear that some dysphagic patients with suspected achalasia exhibit heterogeneity with respect to how completely the achalasia syndrome is expressed; while many have a flaccid esophagus, others have spastic contractions of the esophagus, many have preserved esophageal longitudinal muscle contraction, and some may have preserved peristalsis with manometric evidence of outflow obstruction.^{3,4} The latter group, with functional EGJ obstruction, is particularly interesting, as this group would be detectable only by manometry; there would be no anticipated endoscopic or fluoroscopic abnormalities. However, esophageal manometry is a test that has been historically plagued by limitations with respect to accuracy and reproducibility calling the very existence of this syndrome into question.^{5–7}

Recent years have witnessed an evolution in esophageal manometry to a methodology that is now more accurately termed high-resolution esophageal pressure topography. With this technology, intraluminal pressure is plotted as a continuum both in time and spatially along the length of the esophagus facilitating an objective, quantitative analysis beyond that which was possible with conventional manometry. Furthermore, esophageal pressure topography plots can be “interrogated” using customized algorithms to calculate objective numerical indices of EGJ relaxation, peristaltic function, or intrabolar pressure (IBP), all with excellent reproducibility.^{8–10} As such, high-resolution esophageal pressure topography would seem an appropriate methodology with which to evaluate functional EGJ obstruction. Is this a novel clinical entity, perhaps an incomplete expression of an achalasia syndrome? Or is this simply a manifestation of the imperfect specificity of the tools available to quantify EGJ relaxation? The aim of this study was to address these questions by identifying patients with isolated functional EGJ obstruction and exploring both the physiological consequences of that finding in terms of esophageal IBP and the associated clinical syndromes with which these individuals present.

Methods

Patient Population and Clinical Assessment

A series of 1,000 consecutive high-resolution esophageal pressure topography studies performed between February 2004 and January 2007 at Northwestern Memorial Hospital was reviewed to identify patients with functional EGJ obstruction defined by the combination of intact peristalsis and impaired EGJ relaxation, defined as a mean EGJ integrated relaxation pressure (IRP) of 15 mmHg or greater.¹¹ Manometric studies and clinical records of patients with functional EGJ obstruction were then analyzed in detail to further characterize the syndrome. Endoscopic records were retrieved to identify and exclude patients with mechanical EGJ obstruction. Patients who had previously undergone anti-reflux surgery and were undergoing evaluation for dysphagia were analyzed as a comparator group to the idiopathic functional obstruction patients with respect to consequences on peristaltic function and esophageal IBP. In addition to the study group, 68 asymptomatic volunteer subjects without hiatus hernia were studied with the identical manometry protocol to serve as the control arm for the esophageal pressure topography analysis of esophageal IBP. The study protocol was approved by the Northwestern University Institutional Review Board.

An investigator blinded to manometric analysis reviewed the medical records of each patient with functional EGJ obstruction to evaluate the predominant symptom at time of the manometry study, subsequent medical or surgical treatment rendered, and clinical response to that treatment. A successful treatment was defined as one with satisfactory symptom response such that no further intervention was recommended for 12 months as documented at a follow-up clinic visit. An unsuccessful treatment response was defined as one followed by the need for an additional intervention within 12 months or poor symptomatic response documented during the follow-up visit.

Manometry Protocol

Patients underwent a standardized manometry protocol after a brief interview and exam to assess symptoms. A solid-state manometry assembly with 36 pressure sensors spaced at 1 -cm intervals (OD 4.2 mm) was used (Manoscan™, Sierra Scientific Instruments Inc., Los Angeles, CA). The recording characteristics of this device have been previously described.¹² The transducers were calibrated at 0 and 100 mmHg using externally applied pressure prior to the study. The manometric assembly was placed transnasally and positioned to record from the hypopharynx to the stomach with approximately five intragastric sensors. Studies were performed in a supine position after at least a 6-h fast. The protocol included a 3-min baseline period and ten 5-ml water swallows.

Esophageal Pressure Topography Analysis

All pressure topography analysis was done using ManoView™ software with data tracings viewed in the color pressure topography mode. End-expiratory EGJ pressure was measured during the 3-min baseline recording using the eSleeve™ tool spanning the entire EGJ. In instances that there was a double-peaked EGJ pressure profile during inspiration, the proximal peak was taken to be the lower esophageal sphincter (LES) and the distal the crural diaphragm (CD). Hiatus hernia was defined using the criterion of ≥ 1.5 cm separation between the LES and CD during the baseline recording. In instances of hiatus hernia, end-expiratory LES and CD measurements were made by restricting the eSleeve™ domain to each of these elements respectively.

EGJ relaxation was analyzed using the ManoView™ IRP tool.¹¹ The default settings on the IRP tool establish a 6 cm $\times$ 10 s domain after the swallow and calculates the lowest mean eSleeve™ pressure for four contiguous or non-contiguous seconds of relaxation within that window. However, in the setting of hiatus hernia, the IRP could conceivably be indicative of either LES relaxation or CD relaxation. Thus, the default IRP setting spanning both the LES and the CD components was reported as IRP_{EGJ}, and the IRP boundaries were adjusted to a 2-cm domain capturing each EGJ element independently. Separate measurements were then made of IRP_{LES} and IRP_{CD}. All IRP measurements through the EGJ were referenced to concurrent intragastric pressure.

After characterizing EGJ relaxation, a detailed analysis was done of the associated distal esophageal IBP, reflecting the pressure within the fluid compartmentalized between the EGJ and distal esophageal contraction. IBP was measured using a new ManoView™ tool denoted in the software as IBP2. IBP2, hereafter designated max-IBP, is the greatest IBP obtained for a contiguous or non-contiguous 3-s period within the same temporal boundaries used to calculate the IRP. Typically, the 3 s of greatest IBP occurred close to the end of the relaxation window as the peristaltic contraction arrived at the distal esophagus. All IBP measurements were referenced to atmospheric pressure. Hence, the value of max-IBP could exceed the value of IRP_{EGJ}, as they were measured at different times within the relaxation window and the latter was referenced to intragastric pressure. The derivation of these measures is illustrated in Fig. 1.

Manometry studies were then further analyzed to characterize distal peristaltic weakness or dysfunction. This was done using: (1) the isobaric contour tool set at 30 mmHg to ascertain whether the peristaltic wavefront was intact, (2) the contractile front velocity (CFV) to ascertain normal propagation velocity, and (3) the distal contractile amplitude to identify hypertensive contractions. Each swallow was characterized as: (1) normal (intact isobaric contour, CFV <10 cm/s, mean contractile amplitude <180 mmHg), (2) hypotensive (>3 cm break in the 30 mmHg isobaric contour between the distal segment and the EGJ), (3) absent peristalsis (complete failure of contraction), (4) hypertensive (CFV <10 cm/s and mean contractile amplitude >180 mmHg), (5) spastic (rapidly propagated contraction with CFV $\geq$ 10 cm/s), (6) elevated IBP (>30 mmHg IBP_{max}), or (7) panesophageal pressurization (simultaneous esophageal pressurization to greater than 30 mmHg extending from the upper esophageal sphincter to the EGJ).²

Statistical Analysis

The manometric parameters and clinical variables obtained from medical records were summarized using mean, median, 95th percentile, and standard deviation. IRP and IBP measures were also summarized as the overall mean among the ten test swallows or the worst three values among the ten test swallows to accentuate the observed variability. Unpaired two-sample two-tail Student's *t* test was used to compare the mean values of manometric parameters and clinical variables among the asymptomatic control, idiopathic functional obstruction, and post-fundoplication groups.

Results

Study Population

Of the 1,000 patients evaluated, 78 had evidence of impaired EGJ relaxation on the basis of elevated IRP but did not meet diagnostic criteria for achalasia, a typical example of which is illustrated in Fig. 2. From this group, 38 patients exhibited a high frequency of peristaltic defects, but insufficient to meet criteria for achalasia, and were excluded. Additionally, 16 patients were excluded because of mechanical EGJ obstruction: five with paraesophageal hernia, four with esophageal cancer, three with eosinophilic esophagitis, two with strictures, one with a gastropasty, and one with obstructing gastroesophageal varices. This left 16 patients with idiopathic functional obstruction as exemplified in Fig. 1 and eight patients with post-fundoplication dysphagia. Age and sex were similar between these groups. Healthy controls, however, were younger than the patient groups (Table 1). For comparison, 129 of the 1,000 patients evaluated in the consecutive series had achalasia; 30 had a prior diagnosis, and 99 were newly diagnosed. The pressure topography characteristics of the achalasia patients were reported on earlier.³

Dysphagia was the dominant symptom among the functional EGJ obstruction patients (Table 1). All patients presented with a dominant complaint of either dysphagia or chest pain. The post-fundoplication group was notable for complaining of chest pain significantly more than the idiopathic group. Seven patients were found to have hiatus hernia: four post-fundoplication and three idiopathic. The idiopathic functional obstruction patients exhibited similar manometric characteristics compared to the post-fundoplication dysphagia patients, a model of mechanical obstruction (Table 2). Both groups exhibited impaired (but similar) EGJ relaxation pressures. Furthermore, distal esophageal max-IBP was significantly greater than in the control subjects in both subject groups, especially in post-fundoplication dysphagia patients. In addition to max-IBP, the other discriminating variable between the patient groups was EGJ pressure, which was greater in the idiopathic EGJ obstruction patients. The index that most accentuated the IBP abnormality in the patient groups was the max-IBP (worst 3; Table 2 and Fig. 3).

Specific to patients with hiatus hernia, the IRP analysis was done both to encompass the entire EGJ as well as restricted to the LES and CD components (Table 3). Although the IRP_{EGJ} was numerically greater in the post-fundoplication dysphagia group, this difference was not significant. More notable was that one of the three idiopathic EGJ obstruction patients with a hernia had EGJ relaxation pressures entirely dependent on the CD rather than the LES, whereas in others, the IRP_{LES} was greater (Fig. 4). This suggests that, in some hiatus hernia patients, the CD and not the LES is responsible for functional EGJ obstruction.

The composite characteristics of all individual swallows for functional obstruction subjects are provided in Table 4. The majority of swallows demonstrated functional esophageal obstruction as defined by an elevated max-IBP. Swallows displaying hypertensive peristalsis were seen more frequently in patients with idiopathic functional obstruction. Rarely, pressure topography findings associated with achalasia such as pan-esophageal pressurization or spasm were evident on isolated swallows. On the whole, only 4.6% of swallows demonstrated absent peristalsis.

Clinical Outcomes

The clinical response of idiopathic functional obstruction patients to conventional achalasia therapies (BoTox™, pneumatic dilation, and Heller myotomy) was assessed. Regarding the idiopathic group, the hiatus hernia patient with CD functional obstruction was eliminated from analysis leaving 15 patients subject to an average of 1.1 interventions per patient over a mean follow-up period of 16 months; three patients were lost to follow-up. Overall, nine patients were treated with one or more of these therapies (three pneumatic dilation, three Heller myotomy, two BoTox™, and one standard dilation) and generally exhibited a poor response to therapy with an overall success rate of only 33% for the final intervention. There was no instance in which non-surgical therapy was effective, whereas Heller myotomy was successful in all three individuals so treated (one of whom is illustrated in Fig. 1). Among the eight post-fundoplication dysphagia patients, four underwent redo operations to which three responded favorably.

Discussion

The aim of this study was to characterize the clinical characteristics of patients with functional EGJ obstruction and preserved peristalsis. The major findings were that these patients experience dysphagia as a dominant symptom and that the physiology of idiopathic functional EGJ obstruction mirrors that of a known model of mechanical EGJ obstruction, post-fundoplication dysphagia. In both cases, swallowing is associated with significantly elevated distal esophageal IBP, arguing that these patients truly have EGJ outflow obstruction as opposed to a measurement artifact. Furthermore, a subset of patients with idiopathic functional EGJ obstruction with preserved peristalsis respond to treatment for achalasia, suggesting that, in some cases, this condition likely represents the incomplete expression of an achalasia syndrome. The extent of overlap between the diagnoses of functional EGJ obstruction and achalasia depends on how strictly one defines absent peristalsis. In the current study, we excluded 38 patients from the functional EGJ obstruction group because they exhibited such a high proportion of peristaltic defects that some might consider them achalasics; we did not but were not willing to rule out that possibility either. Another rationale for excluding these indeterminate patients was that the degree of their peristaltic dysfunction was so severe as to preclude the measurement of IBP.

Post-fundoplication dysphagia serves as the iatrogenic model of idiopathic functional esophageal obstruction: EGJ relaxation is impaired, flow through the EGJ is reduced, and distal esophageal IBP is increased.¹³ In a series of 34 post-fundoplication patients, IBP was found to be significantly increased, remaining elevated for at least 2 years after surgery.¹⁴ The development of secondary achalasia as a late consequence has also been observed following

anti-reflux surgery.¹⁵ Furthermore, post-fundoplication dysphagia has been shown to resolve in parallel with the reduction of IBP following conversion to partial fundoplication.¹⁶ In the current series, we applied a systematic analysis of IBP using an algorithm devised for pressure topography plots to demonstrate that the degree of IBP developed in idiopathic functional EGJ obstruction was similar to that observed in post-fundoplication dysphagia. Among several indices of IBP tested, we found that, comparing the maximal IBP in the post-deglutitive window for the three most abnormal swallows (max-IBP) was the best discriminator between normal controls and functional obstruction patients due to the large variation in IBP observed in a ten-swallow series.

Of the 16 patients with idiopathic functional obstruction, three were noted to have hiatus hernias. In one instance, it was the CD rather than the LES that appeared to be the focus of deglutitive resistance to bolus transit, suggesting the hernia itself to be the cause of dysphagia in this individual. In the remaining 15 patients, we had no explanation for their dysphagia other than functional EGJ obstruction. Nonetheless, we expect this to be a heterogeneous group with some individuals having a variant expression of achalasia and others likely having an undetected mechanical etiology of EGJ outflow obstruction. Certainly, the treatment efficacy that we experienced is consistent with that hypothesis. In fact, the only patients who experienced a satisfactory response to treatment were the three treated with laparoscopic Heller myotomy. While these data perhaps serve to demonstrate a proof of concept, they also emphasize the need to further characterize these patients to find better predictors of treatment response and physiological markers of treatment effect. Were failed therapies a consequence of misdiagnosis or inadequate treatment? Was treatment response paralleled by decreased IBP? Were there histopathological markers of achalasia in treatment responders? Clearly, we need to address these questions in future studies.

IBP is attributable to the balance between peristaltic forces acting to move the bolus through the esophagus and downstream resistance to that movement. As evident from data in Table 2 and Fig. 3, normal values of esophageal IBP are low, on the order of 10 mmHg, confirming that the esophagus and EGJ are normally relatively compliant. However, with functional EGJ obstruction, IBP values will often exceed 30 mmHg. This degree of IBP can be likened to balloon distention, a stimulus known to elicit symptoms of chest pain, pressure, and heartburn.^{17,18} The genesis of these symptoms is presumably by wall strain activating tension-sensitive afferent nerves in the esophageal submucosa and muscularis propria.^{19,20} Physiologically, the range of pressure thresholds stimulating vagal and spinal afferents varies from 5 to 50 mmHg,^{21,22} values consistent with those observed in functional EGJ obstruction patients, arguing that elevated IBP may be the primary stimulus for the perception of dysphagia. Future research into the relationship between sensory thresholds, IBP, allodynia, and hyperalgesia will likely shed further light on this.

In summary, idiopathic functional EGJ obstruction with preserved peristalsis is associated with quantifiable outflow obstruction from the esophagus comparable in severity to post-fundoplication dysphagia. This functional defect was well demonstrated by elevated maximal IBP in the worst three of ten test swallows (max-IBP). Lastly, some patients with idiopathic functional EGJ obstruction may represent an early or variant expression of achalasia. To what degree this might progress, over what length of time, and with what frequency will need to be addressed by long-term follow-up studies.

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Figure 1.

Representative example of a patient with functional EGJ obstruction. Max-IBP (*yellow*) is the greatest IBP obtained for a contiguous or non-contiguous 3-s period within the same 10-s temporal boundary used to calculate the IRP (*white*). This patient later responded favorably to Heller myotomy. The IRP is calculated with ManoView™. Pressure is referenced to atmospheric with the 30 mmHg isobaric contour highlighted in *black*.

Figure 2.

High resolution esophageal pressure topography study consistent with classic achalasia exhibiting aperistalsis and impaired EGJ relaxation. Compare this study to that in Fig. 1 consistent with functional EGJ obstruction exhibiting intact peristalsis and impaired EGJ relaxation. The IRP is calculated with ManoView™ in both examples. In this example, pressure is referenced to intragastric pressure and the 15 mmHg isobaric contour is highlighted in *black*.

Figure 3.

Comparison of IRP, max-IBP, and max-IBP (worst 3) among subject groups. All of these manometric measures are significantly elevated in functional EGJ obstruction compared with controls. Among them, max-IBP (worst 3) best discriminated functional EGJ obstruction from controls.

Figure 4.

High-resolution esophageal pressure topography (*top*) and landscape (*bottom*) plots of a hiatus hernia patient with functional EGJ obstruction attributable to the CD (*left*) and another hiatus hernia patient with EGJ functional obstruction attributable to the LES (*right*). In each case, the corresponding IRP_{CD} and IRP_{LES} values are shown.

Table 1

Demographic Characteristics and Symptom Profile Among Subject Groups

	Subject group		
	Asymptomatic controls (n=68)	Post-fundoplication dysphagia (n=8)	Idiopathic functional EGJ obstruction (n=16)
Age	27±5	57±15 *	57±13 *
Gender, % male	53	38	31
Hiatus hernia (%)	0	4 (50%)	3 (19%)
Dysphagia (%)	0	8 (100%)	15 (94%)
Chest Pain (%)	0	6 (75%) *	4 (25%)
Heartburn± regurgitation (%)	0	6 (75%)	9 (56%)
Globus (%)	0	0	3 (19%)

*
p<0.05, vs. asymptomatic controls

**
p<0.05, vs. idiopathic functional obstruction

Table 2

EGJ and IBP Measures Among Subject Groups

Manometric measure	Subject group		
	Normal subjects (n=68)	Post-fundoplication dysphagia (n=8)	Idiopathic functional EGJ obstruction (n=16)
EGJ pressure (mmHg)	16±8	25±17 [*]	40±17 ^{*,**}
Mean IRP (mmHg)	9±3	21±7 [*]	22±5 ^{*t}
Max-IBP (all) (mmHg)	11±4	40±12 [*]	32±6 ^{*,**}
Max-IBP (3 worst) (mmHg)	13±3	47±15 [*]	40±14 [*]

^{*} $p < 0.05$, vs. asymptomatic control

^{**} $p < 0.05$, vs. post-fundoplication

Table 3

Relaxation Pressures of EGJ Components in EGJ Obstruction Patients with Hiatus Hernia

Manometric measure	Subject group	
	Post-fundoplication dysphagia (n=4)	Idiopathic functional EGJ obstruction (n=3)
IRP _{EGJ} (mmHg)	24.8±9.2	17.5±2.7
IRP _{LES} (mmHg)	24.3±9.4	16.9±2.3
IRP _{CD} (mmHg)	18.4±13.0	8.5±6.5

Table 4

Characterization of Individual Swallows Among Functional Obstruction Subtypes

Esophageal contraction	Subject Group	
	Post-Fundoplication Dysphagia	Idiopathic Functional EGJ Obstruction
Normal (%)	15(19%)	33 (21%)
Hypotensive peristalsis (%)	1 (1%)	9 (6%)
Absent peristalsis (%)	1 (1%)	10 (6%)
Hypertensive peristalsis (%)	0	13 (8%)
Spasm (%)	0	5 (3%)
Panesophageal pressurization (%)	0	4 (3%)
Max-IBP > 30 mmHg (%)	63 (79%)	86 (54%)