Relationship of manometric findings to symptomatic response after pneumatic dilation in achalasia cardia

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Background: Achalasia cardia is usually treated by pneumatic dilation or surgical esophagomyotomy. The role of esophageal manometry for objective assessment of symptom response is controversial. Aim: To study the relationship between symptoms and manometric parameters before and after pneumatic dilation in patients with achalasia cardia. Methods: Sixteen patients with achalasia cardia underwent esophageal manometry before and after undergoing pneumatic dilation. At each time, lower esophageal sphincter (LES) pressure and mean basal esophageal-gastric pressure gradient (MIEP-MIGP) were measured. Results: Good symptom response was obtained in 12 of 16 patients. Median (range) LES pressure fell from 42 (17-51) mmHg to 18 (8-39) mmHg in those patients with a good response, and from 51 (25-68) mmHg to 29.5 (23-42) mmHg in those who responded poorly. Mean intragastric pressure fell below mean intragastric pressure in both the groups. Conclusions: Esophageal manometry does not correlate with symptom improvement after pneumatic dilation in achalasia cardia. Dysphagia may persist in spite of reversal of the MIEP-MIGP gradient. [Indian J Gastroenterol 1998; 17: 19-21]

Key words: Esophageal motility disorder, esophageal manometry, pressure measurement

Achalasia cardia, a functional disorder of the esophagus, is characterized by dysphagia for both solids and liquids. Manometric findings in this condition include absence of peristalsis in the esophageal body, incomplete relaxation of the lower esophageal sphincter (LES) in response to wet swallows, and usually an elevated basal LES pressure. Presently, the choice of therapy lies between pneumatic dilation, surgical esophagomyotomy and currently, intrasphincteric injection of botulinum toxin.1-4 These procedures aim at reducing the obstruction at the LES, i.e., reducing the basal LES pressure.

There is a controversy regarding whether assessment of response to these therapeutic modalities should be based on symptoms alone, or additionally, on assessment of LES pressure. Studies on the relationship of change in LES pressure after dilation to improvement in clinical symptoms have shown conflicting results.5,6 We therefore undertook a study to evaluate the changes in esophageal manometric findings before and after balloon dilation and to determine the relationship of manometric findings to patients’ symptom response.

Methods
We prospectively studied 16 consecutive patients (9 men; age range 15-88 years, median 33) with primary achalasia cardia diagnosed on the basis of compatible clinical history, manometric findings and contrast X-rays. Five patients had previously received nifedipine therapy (3 mo to 1 year ago) without lasting relief; these patients were similar in other respects to the remaining 11 patients.

Esophageal manometry was performed after an overnight fast using a low-compliance polyvinyl catheter and a Gaertec water-perfusion manometry system (GR 885; Aspen Medical, Dingwall, Scotland). The manometry catheter had six side-holes, each 0.8 mm in diameter, placed 5 cm apart along the length of the catheter and at 60° radial deviation to each other. Each port was perfused with deaerated water (0.6 mL/min) using a low-compliance capillary infusion system, and was connected to an external transducer. The pressure readings were recorded on a computer and analyzed using the Gaertec esophageal manometry software. No patient had received drugs which could interfere with esophageal motility (viz. prokinetic drugs, calcium-channel antagonists, beta-adrenergic receptor antagonists, nitrates, or drugs inhibiting gastric acid secretion) in the 72 h preceding the study.

Manometric recordings were done with the patients supine. The catheter assembly was passed transnasally without sedation or anesthesia. Intragastric pressure was used as the zero reference point for all pressure measurements. LES pressure was determined using a station pull-through technique. LES relaxation in response to wet swallows (5 mL water for each swallow) was then studied; failure of the LES pressure to reduce through at least 90% of basal pressure was considered as incomplete relaxation. Thereafter, esophageal body motility in response to ten consecutive wet swallows at least 30 seconds apart was studied. Amplitude and duration of each pressure wave were measured at four recording sites (5, 10, 15 and 20 cm above the LES) and abnormalities in pressure wave-forms and in peristaltic progression were diagnosed using standard criteria.9

We devised a manometric measure to quantitate the degree of obstruction at the LES. Basal mean intragastric
pressure (MIGP) and intraesophageal pressure (MIEP) were determined as means of pressures in the esophagus and stomach in four recording ports, each averaged over at least three respiration cycles. From these measurements, a pressure gradient between esophageal and gastric pressures was calculated (MIEP-MIGP, the same as MIEP since MIGP was used as zero reference point); this, we believed, provided a measure of esophageal retention. In normal subjects, MIEP is less than MIGP. In the presence of esophageal retention, MIEP may be expected to rise, thus causing a rise in the gradient. All manometric recordings were analyzed by a person (AM) who was not aware of the patients’ symptoms or type of dilator used.

Balloon dilation was performed within a week after esophageal manometry. Dilation was done under fluoroscopic and endoscopic guidance, using an indigenous dilator10 (Suntech, Inquip) in the initial 9 patients and a Rigiflex dilator (Microvasive, Boston Scientific, Watertown, USA) in the latter 7 patients, depending on the availability of dilators. All patients were treated on outpatient basis and no topical anesthesia or intravenous sedation was used. With the indigenous dilator a pressure of 250-300 mmHg (approximately 6 psi) was used after the position of the balloon was ensured by endoscopy.10 The Rigiflex dilator (balloon diameter 30 mm) was inflated till complete obliteration of the waist was achieved (usually to a pressure of 9 to 10 psi), and pressure was maintained at this level for 60 seconds. This was repeated after an interval of 1-2 minutes during the same session. Patients were observed in the hospital for 2-4 hours after the procedure.

Patients were assessed 2-4 weeks later for the presence of symptoms like dysphagia, chest pain, heartburn and regurgitation. They were asked to quantify symptom relief on a 0-100 analog scale; relief was graded as poor (0-74), good (75-94) and excellent (95). At this time, a repeat esophageal manometry was also performed. Patients were thereafter followed up clinically; median follow-up duration was 6 months (range 1.5 to 12 mo).

Statistical methods
Willcoxon’s signed rank test was used for intragroup comparisons. For all tests, a p value of less than 0.05 was considered significant.

Results
The duration of symptoms ranged from 2 to 96 mo (median 21). None of the patients had sigmoid esophagus or distal esophageal diverticulum on barium esophagogram. At manometry, seven patients had resting LES pressures in the range regarded as high (>45 mmHg).9

Symptom response
Fourteen patients reported good or excellent symptom relief immediately after dilation. Of these, 13 patients had no dysphagia, whereas one patient had occasional difficul
ty with solids. The two patients who did not get much relief after dilation underwent repeat dilation within a month. Of the patients who initially improved, two had recurrence of symptoms after one and nine months, necessitating repeat dilation. Hence, 12 of 15 patients had lasting relief after a single dilation; these patients were considered as good responders and those needing repeat dilation were considered as poor responders. All the four patients who failed to get relief or had recurrence of symptoms had undergone dilation with the indigenous dilator.

Manometric results
The median LES pressure fell from median (range) predilation level of 44.3 (17-68) mmHg to 21 (8-42) mmHg after dilation (p<0.05). MIEP-MIGP fell from 4.3 (-1.8 to +12) mmHg to -1.9 (-5.8 to +3.0) mmHg (p<0.05). Of the 15 patients who initially had a positive MIEP-MIGP gradient, 11 had a negative gradient after dilation. LES relaxation and esophageal body motility were unchanged.

Relationship of manometric findings to symptom improvement
LES pressure prior to dilation was higher, though not significantly, in patients who responded poorly or not at all, than in good responders (Table). LES pressures fell by 51% (p<0.01) in the good responders and by 36% (p=ns) in the poor responders; the percent fall in the two groups was not significantly different. No pressure cut-off which could separate the two groups at either point of time could be identified.

Complications
Mild chest discomfort during the procedure was common, but it subsided on deflation of the balloon. One patient had a perforation after the use of the Rigiflex dilator, and recovered with conservative management. This patient also had the lowest LES pressure after dilation (8 mmHg) and was the only one to complain of disabling heartburn after dilation. However, pHmetry did not indicate increase in total reflux time as compared to the normal reference range in our laboratory.

Discussion
Of the 16 patients studied, 12 had lasting symptom relief after one session of pneumatic dilation, while two more
had initial good relief. This is comparable to previously reported figures.5,6,7,11,12

After dilation, basal LES pressure showed a significant fall, as expected. Though pre- and post-dilation LES pressures were higher in the poor responders, symptomatic improvement had no relationship with the fall in LES pressure. Previous studies on this subject have yielded conflicting results. Eckardt et al2 and Csendes et al5 found that post-treatment LES pressure had a good relationship with symptom improvement. Eckardt et al2 reported that all patients in whom an LES pressure of <10 mmHg was attained after pneumatic dilation remained in remission for at least two years. Such low LES pressures, however, may lead to unacceptably high frequency of symptomatic gastroesophageal reflux. In our study, the patient with the lowest post-dilation LES pressure (8 mmHg) developed heartburn.

On the other hand, Gelfand and Kozarek7 found that a decrease in LES pressure of greater than 50% corresponded well with satisfactory response, but five of their 20 patients with a satisfactory result dropped their sphenicter pressures through less than 50%. They found that post-dilation LES pressure did not always correlate with symptom response. Our finding of lack of a relationship between the fall in LES pressure and symptom improvement agrees with theirs. It is possible that failure to detect a difference in post-dilation LES pressure between good and poor responders in our study may reflect a beta error.

Since there was no relationship of symptom relief with LES pressure, we also looked at the MIEP-MIGP gradient, which we believe represents esophageal retention. We found that, even in the poor responders, the gradient became negative after dilation. This means that the obstruction at the LES had been overcome and esophageal retention was no longer occurring. In these patients, the persistence of dysphagia in the face of normal gradient might suggest that it is related to peristaltic abnormalities in the esophageal body rather than to obstruction at the LES. It has previously been shown that fall in LES pressure after pneumatic dilation does not correlate well with esophageal emptying as measured by scintigraphy.13 Thus, we believe that persistent symptoms may be a function not only of residual LES pressure but also of esophageal body motility.

We conclude that pneumatic dilation is an effective treatment for achalasia cardia. Successful dilation results in relief of symptoms and, in a majority of patients, reduction in LES pressure; the latter, however, does not always correlate well with symptoms and does not help to identify good response.

References

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