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RESEARCH ARTICLE

High-resolution manometric features of achalasia of the cardia

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ABSTRACT

Background: Achalasia of the cardia is a common esophageal motility disorder. High-resolution manometry is the gold standard for the diagnosis of achalasia. The body of the esophagus and the lower esophageal sphincter (LOS) exhibits various motility characteristics that are typical to achalasia. **Aims and Objectives:** The study was conducted to study the physiology of the LOS in achalasia with a 16-channel water perfusion high-resolution esophageal manometer. **Materials and Methods:** High-resolution esophageal manometry was performed in 13 adult patients of achalasia and the functional patterns were evaluated. **Results:** 12 of 13 cases of achalasia had a high basal LOS pressure (BLOSP), while one had reduced pressure. All cases of achalasia had an elevated LOS nadir pressure, indicating an incomplete relaxation of the LOS. All 13 cases of achalasia showed the absence of peristalsis. **Conclusion:** A reduced BLOSP is rare in achalasia and so is a normal BLOSP; however, they have been described in literature, and hence, the BLOSP as such cannot be considered as a diagnostic feature of achalasia on manometry.

KEY WORDS: Achalasia; High-Resolution Manometry; Lower Esophageal Sphincter; Lower Esophageal Sphincter Relaxation

INTRODUCTION

Achalasia (Greek = Does not relax) of the cardia is a motility disorder of the esophagus. Its etiology is not known. It is characterized by degeneration of the ganglion cells of the Auerbach's plexus,[1,2] especially those that produce nitric oxide, which, in turn, affects the relaxation of the lower esophageal sphincter (LOS) during deglutition.[3,4] In the absence of a swallow, the smooth muscles of the LOS are tonically contracted. They relax when the neurons of the enteric nervous system release their inhibitory neurotransmitters.[5] Loss of inhibitory innervation in the LOS can cause basal LOS pressures (BLOSP) to rise. The rise, in turn, can interfere with normal relaxation of the LOS. On the other hand, the esophageal body does not exhibit a resting tone; hence, loss of neurons has little effect on the resting pressure of the body of the esophagus. Since inhibitory neural inputs are necessary for normal peristalsis, loss of these neurons can cause loss of peristalsis.[6] In classic achalasia, swallows are usually followed by either absent activity of the esophageal body or simultaneous esophageal contractions of low amplitude (<40 mm Hg).[7]

High-resolution manometry (HRM) is the gold standard for the diagnosis of achalasia but is undertaken only when structural causes of dysphagia have been ruled out endoscopically and radiologically. It is also performed as a pre-operative evaluation of patients who are being considered for surgery for reflux disease.[8,9] Although a few Indian studies have been conducted with HRM systems to classify and subtype achalasia[10] and to assess pre- and post-pneumatic dilatation status of the LOS,[11] there are very few that have dealt with

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the physiology of the LOS in achalasia with the help of a multichannel water perfusion HRM system. The present study was conducted to address the issue.

MATERIALS AND METHODS

The study was conducted in the Gastrointestinal Physiology Laboratory in the Department of Physiology in a tertiary care setting after obtaining the approval of the Institute Ethical Committee. It was a single-center observational cross-sectional study. The period of the study was from January 2008 to February 2009. A total of 13 (n = 13; 10 males) patients of achalasia were included in the study. The inclusion criteria were based on clinical, endoscopic, or radiographic (or a combination of these) evidence of achalasia. The mean age of the patients was 41.8 years (range = 27–54 years). All the patients were referred by medical/surgical gastroenterology centers of several hospitals for HRM, following endoscopy and barium swallow for ruling out the possibility of any structural lesion(s). All patients had symptoms suggestive of achalasia, namely dysphagia. Patients who had already been operated or pneumatically dilated for achalasia were excluded from the study. Patients with dysphagia who were clinically diagnosed as achalasia but failed to show the manometric features of the disease were also excluded from the study.

Esophageal manometry was performed using the high-resolution 16-channel water perfusion gastrointestinal manometric assembly with electronic pressure transducers (The Royal Melbourne Hospital, Victoria, Australia), along with its diagnostic software (Trace! 2005), which was used for the analysis of data. Written informed consent was obtained from all patients and they were asked to report to the laboratory in the morning after an overnight fast. Patients who were on drugs that may affect motility were told to withhold their medication for 48 h before the test. The HRM catheter was introduced into the esophagus through the nose, with the patients sitting upright on the bed. Once the LOS was localized, the patients were instructed to lie down in the right lateral recumbent position. Thereafter, the BLOSP was recorded for 3 min by asking the patients not to swallow. After obtaining the BLOSP, the patients were given a total of 10 swallows, with each swallow containing 5 ml of drinking water. The time gap between successive swallows was 30 s. Esophageal peristalsis patterns, and the minimum and maximum LOS pressures to swallows, were recorded. A similar set of 10 swallows (5 ml each, 30 s apart) was repeated after asking the patients to change their position from lying down to sitting upright. The software was accordingly corrected to eliminate the effects of the hydrostatic column associated with the upright posture.

RESULTS

Data obtained were then analyzed for various manometric features. The results of the analysis can be summarized as per Table 1. A plot obtained for a patient during a swallow is depicted in Figure 1.

DISCUSSION

The normal LOS nadir pressure is <8 mm Hg, whereas the normal BLOSP ranges between 10 and 20 mm Hg. The manometric diagnosis of achalasia includes an incomplete relaxation of the LOS to swallows, i.e. a raised LOS nadir pressure (>8 mm Hg).[12] All 13 cases of achalasia had raised LOS nadir pressures (mean = 13.01 mm Hg), thereby preventing the LOS from relaxing completely after a swallow. Normal nadir pressures of the LOS to swallows have been described in achalasia, but such cases have been termed in

Table 1: Summary for various manometry findings in achalasia

Patient serial number	Age (years)	Sex (M/F)	Mean BLOSP (mm Hg)	LOS nadir pressure (mm Hg)	Peristalsis (present/absent)
1	43	M	22.0	12.4	Absent
2	48	M	22.6	18.5	Absent
3	38	F	26.5	13.5	Absent
4	49	M	30.1	12.8	Absent
5	27	M	31.4	12.1	Absent
6	41	M	29.4	16.6	Absent
7	37	M	26.6	14.5	Absent
8	54	M	8.7	8.9	Absent
9	49	F	26.1	11.4	Absent
10	36	M	25.7	13.6	Absent
11	41	F	23.1	12.9	Absent
12	44	M	29.1	10.2	Absent
13	47	M	30.0	11.8	Absent

LOS: Lower esophageal sphincter, BLOSP: Basal LOS pressure

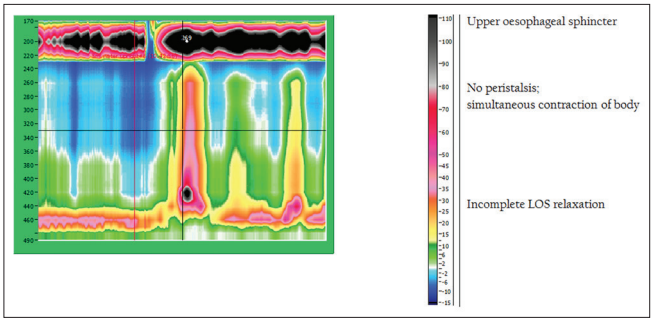

Figure 1: Plot of esophageal manometry. LOS: Lower esophageal sphincter

literature as "apparent complete relaxations of the LOS." The duration of LOS relaxation in this group was found to be significantly shorter ($P \leq 0.01$) than in normal subjects. Although nadir pressures were normal in these patients, and the relaxation of the LOS to swallows was complete, the relaxation was functionally inadequate, perhaps due to the shortened duration.[13] This functional inadequacy may be due to the failure of the gastroesophageal junction pressure to drop during the flow of fluid or a bolus across it.[14] However, in this study, none of the cases presented with such a feature on HRM.

The BLOSP in 12 of 13 patients was raised (mean = 25.48 mm Hg). However, one patient had a low BLOSP (8.7 mm Hg). The value of BLOSP is not used as a diagnostic criterion for achalasia on HRM, though it can be a supportive evidence. Although it is rare for the BLOSP to be reduced in achalasia, yet it has been described in literature and so has been a normal BLOSP.[15] It must be remembered at this point, however, that a high BLOSP is a supportive evidence for a diagnosis of achalasia and not its defining manometric feature.[12] The absence of peristalsis and simultaneous contraction of the body of the esophagus are both proven manometric features of achalasia, and these features were found in all the 13 patients. The etiology of the altered functioning of the LOS in achalasia has yet not been addressed. However, there is evidence suggesting involvement of immunoglobulin-G antibodies against the ganglion cells of the Auerbach's plexus, thereby indicating a possible autoimmune etiology.[16] A previous viral infection has also been implicated, but not proven.[17] Environmental factors have also been implicated, leading to an inflammatory response in the enteric nervous system, causing the disease.[18]

CONCLUSION

The sample size of this study was limited to 13 cases of achalasia. However, the manometric features of achalasia are only two, and both are highly specific for the disease. These features are present in all cases of achalasia. The presence of any one of these features does not lead to a manometric diagnosis of achalasia. Hence, it is unlikely that a large sample size would offer different manometric findings in this disease. A high BLOSP is a supportive evidence for a diagnosis of achalasia, and not a defining manometric feature for this condition, which, in turn, is an incomplete LOS relaxation and failure of peristalsis. A reduced BLOSP is rare in achalasia and so is a normal BLOSP. However, they have been described in literature, and hence, the BLOSP as such cannot be considered as a diagnostic feature of achalasia on HRM.

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