ORIGINAL ARTICLE

Respiratory Dysfunction Is Common in Patients with Achalasia and Improves After Pneumatic Dilation

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Abstract

Background Dysphagia, regurgitation, and chest pain are common achalasia, with a variable report of pulmonary symptoms possibly due to micro-aspiration. Pneumatic dilation (PD) may improve pulmonary function. Data on pulmonary dysfunction among achalasia patients are scanty, and the effect of PD is unknown.

Aim To evaluate pulmonary dysfunction in patients with achalasia based on clinical and radiologic evaluation and spirometry and to study the effect of PD at 1-month follow-up.

Methods Patients with achalasia (diagnosed using high-resolution manometry and the Chicago classification) were evaluated prospectively by spirometry before ($n = 38$) and 1 month after PD ($n = 31$). All patients received a chest X-ray, and patients with respiratory abnormality before PD received high-resolution computed tomography of the thorax.

Results Of the 38 patients, 17 and 21 had type I and II achalasia, respectively. The respiratory symptoms, such as pharyngeal symptoms [27/38 (71 %) vs. 8/31 (26 %); $P = 0.0001$], cough [23/38 (60.5 %) vs. 5/31 (16 %), $P = 0.0001$], and dyspnea [8/38 (21 %) vs. 0/31 (0 %),

$P = 0.006$], improved after treatment with PD. Spirometry showed abnormalities in 17/38 (45 %) patients before and in 8/15 (53 %) after PD. Median FEV_1, FVC, PEFR, and percentage of predicted MEF_{25-75}, improved from 78 % (36–85), 74 % (48–100), 62 % (18–72), and 48 % (15–66) before to 83 % (58–94), 86 % (55–99), 69 % (38–81), and 59 % (33–78) after PD, respectively ($P < 0.05$ for all).

Conclusion Respiratory symptoms and spirometry abnormalities are common in patients with achalasia and improved after successful PD.

Keywords Motor dysphagia · Esophagus · Pulmonary function test · Spirometry · Aspiration · Esophageal emptying

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Introduction

Achalasia is one of the most common esophageal motility disorders causing dysphagia [1]. It is characterized by impaired peristalsis in the esophageal body and failure of relaxation of the lower esophageal sphincter (LES). A proportion of patients with achalasia may present primarily with respiratory symptoms, which may cause diagnostic difficulty [2, 3]. Common pulmonary symptoms in achalasia are cough, which is frequently nocturnal, dyspnea, choking, recurrent respiratory infections, wheezing, stridor, and even acute respiratory obstruction [4–10]. Mechanical obstruction leading to stridor and acute tracheal obstruction are serious and life-threatening complications [11]. In addition, pharyngeal and laryngeal symptoms, such as sore throat, globus sensation, and hoarseness, may also occur [12, 13].

Respiratory dysfunction in achalasia may result from micro- and macro-aspiration [4] of the contents of the

dilated esophagus. Such aspiration events commonly occur during the night, when the patient is in a supine position. The severity of the pulmonary dysfunction is likely to depend on disease duration, frequency of aspiration episodes, and nature of the aspirated material [14]. Recurrent aspiration episodes over a long period of time may lead to serious consequences, such as diffuse aspiration bronchiolitis, obliterative bronchiolitis, aspiration pneumonia, lung abscess, empyema, and even lung fibrosis, some of which may be irreversible [14–16]. Early detection and appropriate treatment may prevent long-term respiratory morbidity.

Pneumatic dilation (PD), which lowers LES pressure, is a well-established modality for the management of achalasia [17]. Since esophageal emptying is improved after PD [18], it may reduce the risk of aspiration. Timed barium esophagogram (TBE) is a good modality to quantify the response of PD in terms of esophageal emptying [19, 20]. Whether this benefit translates into improvement in respiratory symptoms and lung injury is not fully known. Also, data on respiratory symptoms and the exact pattern of spirometry abnormalities in achalasia are scanty [8, 12]. Moreover, the effect of PD on pulmonary dysfunction is not known. Accordingly, we undertook a study with the following aims: (1) to evaluate the occurrence and spectrum of pulmonary dysfunction in patients with achalasia based on clinical and radiologic evaluations and spirometry and (2) to study the effect of PD on these variables at the 1-month follow-up.

Methods

Study Protocol

Consecutive patients (age ≥18 years) with achalasia were recruited for this prospective study over 1-year period (August 2011 to July 2012). Patients were evaluated using a standard questionnaire (Eckardt's score) [21]. Esophagogastroduodenoscopy was performed in all patients to rule out esophago-gastric malignancy. The diagnosis and type of achalasia (Chicago classification) [22, 23] was established by high-resolution manometry (HRM) using a 16-channel water-perfused catheter. A pulmonary physician then evaluated all patients with confirmed diagnosis of achalasia for respiratory symptoms, including a spirometry and chest X-ray. Chest computed tomography (CT) with expiratory films was performed in those patients with abnormal spirometry. Esophageal emptying was evaluated by TBE, performed as per a standard protocol [24]. Patients with mega-esophagus (esophageal width >6 cm), pregnant women, chronic smokers, and those with prior history of PD, Heller's myotomy, significant chronic respiratory illnesses (e.g., asthma, obstructive airway disease),

pulmonary tuberculosis) were excluded. Patients were treated by PD according to a standard protocol [25]. Each patient was re-evaluated based on Eckardt's score, HRM and TBE parameters, respiratory symptoms, chest X-ray (if previous X-ray was abnormal), and spirometry again at 1 month following PD. The study protocol was approved by the Ethics Committee of the Institute, and all patients gave informed consent.

Clinical Evaluation Including Assessment of Pulmonary Symptoms

All patients were evaluated for duration and severity of all symptoms, previous treatment obtained, co-morbid illnesses, and feasibility of PD. Baseline demographics, including prior history of respiratory diseases, were recorded. Dysphagia, chest pain, regurgitation, and weight loss were assessed according to Eckardt's score (first 3 symptoms scored as 0 = none, 1 = occasional, 2 = daily, 3 = at each meal). Weight loss was scored as 0 = none, 1 = <5 kg, 2 = 5–10 kg, 3 = >10 kg. The maximum possible score was 12, and a score <3 on follow-up was considered to be indicative of clinical response. Respiratory symptoms and signs, such as cough, nocturnal cough, sputum production, dyspnea, recurrent chest infections, wheezing, stridor, acute respiratory obstruction, sore throat, and hoarseness, and the results of the chest physical examination were recorded by a pulmonologist before and after PD; the pulmonologist was unaware of the results of the gastrointestinal evaluation, including Eckardt's score, esophagogastroduodenoscopy findings, HRM and TBE parameters, and treatment with PD. The same questionnaire for the presence/absence of symptoms and signs was administered before and after PD for qualitative evaluation. Chest X-ray (posterior–anterior view) and spirometry results were obtained in all patients.

High-Resolution Esophageal Manometry

Esophageal manometry was performed using a water perfusion system (G.S. Hebbard system, Australia). Drugs that might interfere with esophageal motility and LES pressure (prokinetics, calcium channel blockers, anti-cholinergics) were discontinued at least 7 days prior to the study. After an overnight fast, an HRM catheter, smeared with lubricating jelly, was passed trans-nasally without any sedation or anaesthesia into the stomach while the patient was in a supine position. Intra-gastric pressure was used as the baseline for all pressure measurements. On HRM, LES was located as a zone of high pressure in the Clouse plot. Patients were given ten wet swallows of 5 mL of water, with a 15–30-s interval between each swallow, to assess esophageal body motility and LES relaxation.

Achalasia was diagnosed on HRM using standard criteria [22]. These patients were further sub-typed according to the dominant characteristics of distal esophageal pressurization (the isobaric contour) after swallowing, set at 30 mmHg and a mean integrated relaxation pressure (IRP) [26]. Type I achalasia was defined as "no distal esophageal" pressurization of >30 mmHg in eight or more of the ten wet swallows; type II as pan-esophageal pressurization to >30 mmHg in at least two of the ten wet swallows; type III (spastic) as preserved fragments of distal peristalsis or premature (spastic) contractions in two or more of the ten swallows [22]. The value of IRP had to be >10 mmHg to diagnose type I achalasia and >15 mmHg to diagnose type II and III achalasia [23].

All manometry analyses were performed using "Trace 1.2" software applied to the data tracings viewed in the color pressure topography mode and referenced to intragastric pressure (G.S. Hebbard system, Australia). The same technique was used for both pre- and post-PD manometry.

Timed Barium Esophagogram

The TBE was performed with the same technique [24] in all patients after HRM using 150 mL of 45 % w/v barium suspension (Microbar suspension; Eskay, Mumbai, India). Still images were obtained in the left posterior oblique standing position at 1, 5 and 10 min after completing the swallow (Axiom Icons-R200; Siemens, Munich, Germany). The maximum height and width of the barium column in all these images were measured. The same protocol was used for repeat TBE, done at 1 month after PD.

Spirometry

All patients with achalasia were evaluated by the pulmonologist for pulmonary function tests (PFTs) by spirometry. Spirometry was performed using a dry rolling seal spirometer (model 2010; Ganshorn Medizin Electronic GmbH, Niederlauer, Germany; software version LF 8.5). A minimum of three acceptable and reproducible maneuvers were recorded by an experienced technician using the guidelines of the American Thoracic Society (ATS) [27]. Spirometry indices like forced vital capacity (FVC), forced expiratory ventilation in 1 s (FEV_1), FEV_1/FVC ratio, mid expiratory flow (MEF_{25-75}), and peak expiratory flow rate (PEFR) were recorded. The pulmonary function of each patient was assessed according to the absolute lung volumes on spirometry in reference to the lower limit of normal (LLN) for that age, height, and gender. The LLN for each of the above variables was calculated as the difference between the predicted value and 1.645-fold the standard error of the estimate of the relevant regression equation [28]. The patients were categorized for pulmonary abnormalities according to the algorithm shown in Fig. 1. Small airway disease was defined as a normal FEV_1, FVC and FEV_1/FVC ratio in association with a reduction of the MEF_{25-75} to <60 % of the predicted value. The diffusion capacity of the lung for carbon monoxide (DLCO) was measured by the single breath test (model 2010; Ganshorn Medizin Electronic GmbH; version Power Cube-Diffusion, software LF8) in all patients as per standardized ATS guidelines [29].

Pneumatic Dilation

After complete baseline evaluation, PD was performed in fasting state using a Rigiflex balloon dilator (Microvasive, Milliford, MA). Most of the PD procedures were performed as daycare procedures under endoscopic guidance without fluoroscopy. Balloon sizes used for initial PD were 30 mm for females and 35 mm for males based on our previous study findings [25]. The dilator was passed into the stomach over a guide wire placed endoscopically. The dilator was positioned under endoscopic vision such that it was placed across the gastro-esophageal junction. The balloon was then inflated for 60 s at 6–8 psi. The patients were observed on the day of dilation for pain, fever, or any complication. A normal diet was allowed commencing on the day following dilation.

Follow-Up

All of the patients were re-evaluated after 1 month of PD in the Luminal Gastroenterology Clinic of the Department of Gastroenterology. Clinical scoring (Eckardt's score), HRM, and TBE were repeated. The evaluation of respiratory symptoms and spirometry were repeated by the pulmonologist using the same protocol.

Evaluation of Result of Dilation

Clinical response was defined as an Eckardt's score of <3 at the 1-month follow-up. Response according to HRM and TBE was defined as a "decrease in LES pressure to <22.5 mmHg" and "a more than a 50 % reduction in the barium column on a 5-min film," respectively, as shown in our previous studies [25, 30].

Statistical Analysis

Continuous unpaired and paired data were compared using the Mann–Whitney U and Wilcoxon rank-sum tests,

Fig. 1 Algorithm for the diagnosis of pulmonary function test (*PFT*) abnormality on spirometry. The simplified algorithm according to American Thoracic Society (ATS) guidelines was used to assess lung function in achalasia patients. It presents classic patterns for various pulmonary disorders. FEV_1 The forced expiratory ventilation in 1 s/ forced vital capacity ratio (FEV_1/FVC) and FVC were considered first in comparison to lower limit of normal (*LLN*). MEF_{25-75} Mid expiratory flow rate
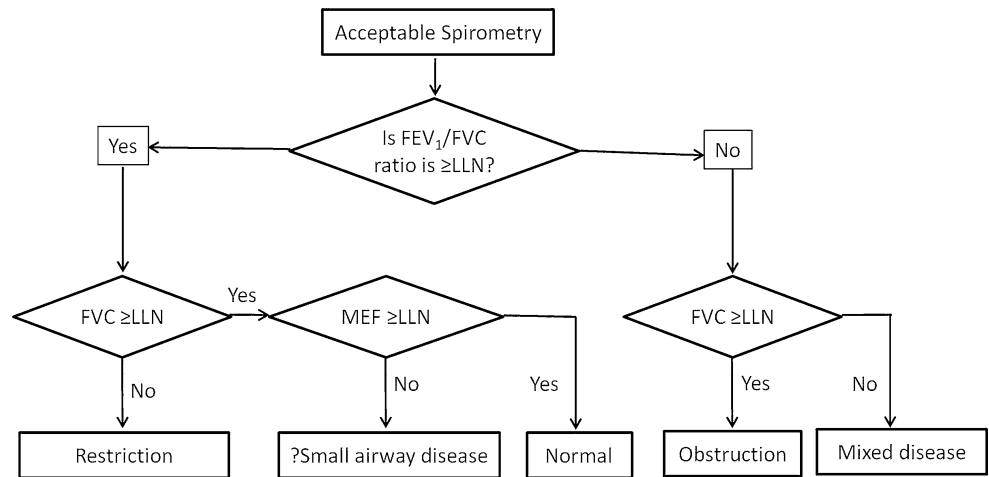
respectively. Categorical variable were compared using Fisher's exact test. Data were analyzed using the statistical package SPSS (version 15.0 for Windows; SPSS, Chicago, IL), and *P* values of <0.05 were considered to be significant.

Results

Of the 46 consecutive patients with achalasia initially screened, eight were excluded due to secondary achalasia or mega-esophagus or for declining to consent (Fig. 2). Table 1 shows the pre-PD baseline patient characteristics. Of the 38 patient ultimately enrolled in the study, [20 (53 %) males; median age 34.5 years, range 18–62 years] who underwent PD, one required urgent surgery due to perforation. At the 1-month follow-up, 31 patients were re-evaluated for response to PD, respiratory symptoms, and PFTs, and six had been lost to follow-up.

Baseline Characteristics

Of the 38 patients entering the study, type I and type II achalasia was diagnosed in 17 (45 %) and 21 (55 %) patients, respectively; no patient had type III achalasia. All patients had an IRP of >15 mmHg (Table 3). There was no significant difference in age, duration of dysphagia, and body mass index between the two patient groups (Table 1). Dysphagia was the most common presenting symptom (100 %) followed by regurgitation, weight loss and chest pain, occurring in 30 (79 %), 18 (47 %), and 11 (29 %) patients, respectively. Median duration of dysphagia was 36 (range 4–180) months. Grade 3 dysphagia was present in most patients in both groups [14/17 (82 %) and 17/21 (81 %) in type I and II, respectively].
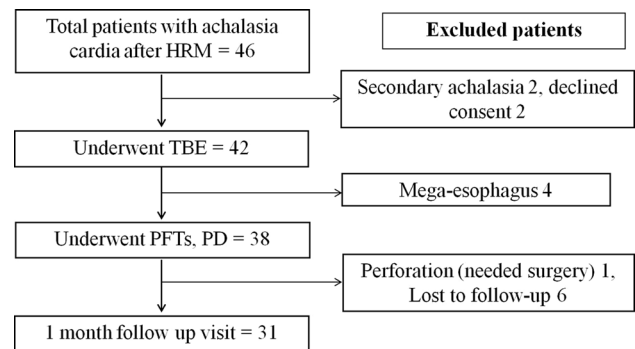
Fig. 2 Schematic summary of the protocol and exclusions. Achalasia was confirmed after high-resolution manometry (*HRM*) in 46 patients. Before pneumatic dilation (*PD*), 8 patients were excluded from the study for the reasons as shown. After PD, 31 patients were re-assessed after 1 month; 1 patient was excluded from analysis as he required surgery and 6 were lost to follow up. *TBE* Timed barium esophagogram

Clinical Presentation

Pharyngeal symptoms, such as excessive throat clearing, sore throat, hoarseness and post-nasal drip, were collectively the commonest symptoms occurring in 27 (71 %) patients. The next most common symptom was cough, present in 23 (60.5 %) patients, of which 19 (50 %) had both daytime as well as nocturnal cough. Dyspnea (8/38, 21 %) and wheezing (3/38, 8 %) were the other respiratory symptoms. Abnormal findings on chest auscultation (rhonchi or crepitations) were present in 9/38 (24 %) patients. Regurgitation and respiratory symptoms were more common among type I achalasia patients while chest pain was more frequent in type II achalasia patients (Table 2). The median duration of respiratory symptoms was 12 (range 1–72) months, which was shorter than that of dysphagia (36 months, range 4–180 months).

Table 1 Baseline characteristics of pre-pneumatic dilation patient and disease characteristics according to type of achalasia

Baseline patient characteristics	Type I achalasia ($n = 17$)	Type II achalasia ($n = 21$)	Total ($n = 38$)	P value
Age (years)	46 (18–56)	34 (18–62)	34.5 (18–62)	0.93
Male, n (%)	9 (53)	11 (52)	20 (53)	0.77
Body mass index (kg/m^2)	18.9 (15.2–27.3)	19.5 (11.7–25.1)	19.3 (11.7–27.3)	0.79
Grade of dysphagia (1/2/3)[a]	2/1/14	1/3/17	3/4/31	0.54
Duration of dysphagia (months)	36 (4–180)	36 (4–144)	36 (4–180)	0.66
Duration of respiratory symptoms (months)	18 (1–60)	12 (6–72)	12 (1–72)	0.91

Values are presented as the median with the range in parenthesis unless indicated otherwise

[a] Absolute number of patients at grade 1, 2, 3, respectively

Table 2 Clinical presentation of patients according to type of achalasia and comparison with post-pneumatic dilation variables

Clinical presentation parameters and respiratory symptoms[a]	Pre-PD				1 month post-PD ($n = 31$)	P value[c]
	Type I achalasia ($n = 17$)	Type II achalasia ($n = 21$)	Total ($n = 38$)	p value[b]		
Eckardt's score	8 (4–11)	7 (4–10)	7 (4–11)	0.26	2 (1–5)	<0.001
Dysphagia	17 (100)	21 (100)	38 (100)	1.00	26 (84)	0.02
Regurgitation	15 (88)	15 (71)	30 (79)	0.26	18 (58)	0.07
Chest pain	3 (17)	8 (38)	11 (29)	0.28	12 (39)	0.45
Weight loss	8 (47)	10 (48)	18 (47)	1.00	7 (23)	0.44
Pharyngeal symptoms	14 (82)	13 (62)	27 (71)	0.31	8 (26)	0.02
Day-time cough	10 (59)	11 (52)	21 (55)	0.94	5 (16)	0.001
Nocturnal cough	11 (65)	10 (48)	21 (55)	0.34	3 (10)	<0.001
Dyspnea	5 (29)	3 (14)	8 (21)	0.46	0	0.02
Wheeze	2 (12)	1 (5)	3 (8)	0.58	0	0.25
Abnormal chest auscultation	6 (35)	3 (14)	9 (24)	0.25	2 (6)	0.09

PD Pneumatic dilation

[a] Eckardt's score is expressed as the median with the range given in parenthesis; all other variables are given as the absolute number of patients, with the percentage given in parenthesis

[b] P value for significance of difference between type I and type II achalasia before PD

[c] P value for significance of difference between pre- and post-PD

Objective Pulmonary and Esophageal Assessment

Seventeen of the 38 (44.7 %) patients had abnormal spirometry findings before PD; these tended to be commoner among patients with type I (10/17, 59 %) than type II achalasia (7/21, 33 %) ($P = 0.09$). PFT abnormalities were classified into restrictive (7/38, 18 %), small airway (6/38, 16 %), obstructive (2/38, 5 %), and mixed types (2/38, 5 %). Chest X-rays were normal in all except four of the 38 (10.5 %) patients (Table 3). Radiography findings included right basal consolidation, bilateral, or right-sided non-homogenous opacities. High-resolution CT scans of the thorax were performed in 17 patients who had abnormal PFT; the scans showed abnormal findings in 12 of these patients (70.6 %), inclduing ground glass opacity with mosaic attenuation, centrilobular nodules with a tree-in-bud pattern (Fig. 3), septal thickening with mosaic attenuation, and necrotizing pneumonia (Fig. 4). Tracheal compression with dilated esophagus (Fig. 5) was evident in two patients on the CT scan. The frequency of abnormal findings on the chest X-ray was comparable among patients with type I and II achalasia. Similarly, there was no significant difference in the median height (128 and 109 mm) and width (82 and 35 mm) of the barium column in the TBE at the 5-min film between type I and type II achalasia.

Table 3 Pulmonary function tests, timed barium esophagogram, and high-resolution manometry according to type of achalasia before PD and comparison with post-PD tests

Variables[a]	Pre-PD				1 month post-PD ($n = 31$)	P value[c]
	Type I achalasia ($n = 17$)	Type II achalasia ($n = 21$)	P value[b]	Total ($n = 38$)		
Abnormal chest X-ray	2 (12)	2 (10)	0.76	4 (11)	3 (10)	0.78
Abnormal PFT	10 (59)	7 (33)	0.34	17 (45)	8 (26)	0.13
Ht_5 in TBE (mm)	128 (51–230)	109 (0–240)	0.77	115 (0–240)	24 (0–208)	<0.001
Wd_5 in TBE (mm)	42 (28–62)	35 (0–66)	0.45	38.5 (0–66)	16 (0–42)	<0.001
Resting LES pressure (mmHg)	41 (10–56)	41 (15–57)	0.81	41 (10–57)	18 (9–34)	<0.001
Esophageal body pressure (mmHg)	21 (8–38)	45 (32–182)	<0.001	37 (8–182)	16 (5–90)	<0.001
IRP	34 (16–51)	37 (16–78)	0.45	36.5 (16–78)	16 (4–40)	<0.001

TBE timed-barium esophagogram, *IRP* integrated relaxation pressure, *LES* lower esophageal sphincter, *PFT* pulmonary function tests

Values are presented as the median with the range in parenthesis or as the absolute number of patients with the percentage in parenthesis, as appropriate

[a] Ht_5 and Wd_5, Height and width of barium column in 5-min film

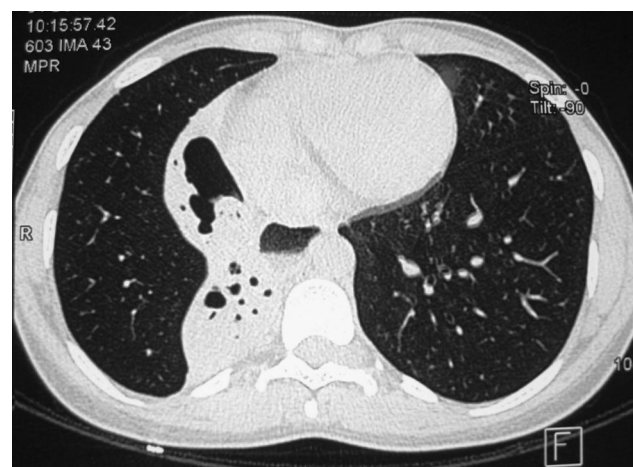[b] P value for significance of difference between type I and type II achalasia before PD

[c] P value for significance of difference between pre- and post-PD

Fig. 3 High-resolution computed tomography (CT) of 23-year-old female showing multiple diffuse centrilobular areas of increased attenuation with a characteristic tree-in-bud appearance


Fig. 4 High-resolution CT of a 20-year-old female with achalasia showing right side necrotizing pneumonia and esophageal dilatation with air-fluid level

Effect of PD

Following PD, 24/31 (77 %) patients had clinical response (Table 3) as evidenced by a reduction in the Eckardt's score from a median of 7 (range 4–11) to 2 (0–5). Respiratory abnormalities, such as pharyngeal symptoms, cough, and dyspnea, also improved following successful PD (Table 2). Similarly, abnormal chest auscultatory findings improved in seven of nine patients. Of the 17 patients with abnormal PFT before PD, 15 could be re-assessed at the 1-month follow-up; abnormal PFT normalized in 7/15 (46.7 %) patients. A statistically significant ($P < 0.05$) improvement occurred in the

median FEV_1, FVC, PEFR, and MEF_{25-75} % of the predicted after PD in the patients with baseline PFT abnormality (Table 4). The median percentage of predicted DLCO did not change significantly after PD.

Repeat chest X-ray normalized only in one of the four patients with baseline abnormality, others had persistence of symptoms as well as radiographic abnormality. The high-resolution CT scan of the thorax was not repeated on follow-up.

Discussion

The results of this study show that pulmonary manifestations are common among patients with achalasia and are
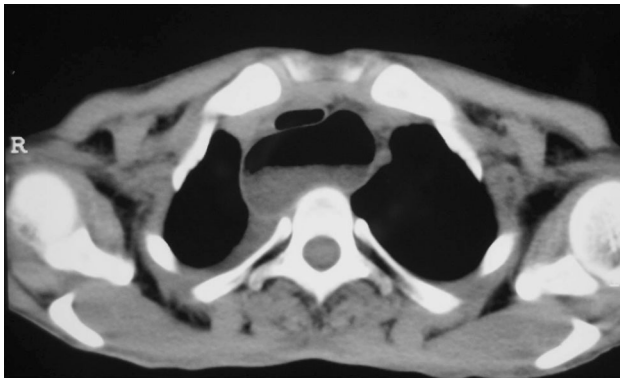
Fig. 5 High-resolution CT of 50-year-old male showing esophageal dilatation with air-fluid level and compression of tracheal lumen

Table 4 Pre- and post-PD pulmonary function tests in achalasia patients with baseline abnormal pulmonary function tests

Patients with abnormal PFT	Before PD ($n = 17$)	After PD ($n = 15$)	P value
FEV$_1$ % of predicted	78 (36–85)	83 (58–94)	0.01
FEV$_1$ (L)	1.8 (0.4–2.9)	2.0 (1.2–3.1)	0.03
FVC % of predicted	74 (48–100)	86 (55–99)	0.04
FVC (L)	2.2 (0.4–3.8)	2.5 (1.4–4.1)	0.03
FEV$_1$/FVC (%)	79 (44–98)	83 (60–98)	0.18
PEFR % of predicted	62 (18–72)	69 (38–81)	0.003
PEFR (L/s)	4.1 (0.7–6)	4.8 (2.2–7.5)	0.005
MEF$_{25–75}$ % of predicted	48 (15–66)	59 (33–78)	0.01
MEF$_{25–75}$ (L/s)	1.6 (0.4–2.7)	2.3 (1.0–3.5)	0.02
DLCO % of predicted	79 (52–138)	84 (52–159)	0.07

DLCO Diffusion lung capacity for carbon monoxide, *FEV$_1$* forced expiratory ventilation in 1 s, *FVC* forced vital capacity, *MEF$_{25–75}$* mid expiratory flow rate, *PEFR* peak expiratory flow rate

Spirometry variables are expressed as the percentage of that predicted for height, age, and gender, respectively, of the patient as well as absolute values obtained

prevalent in both type I and type II disease. Common respiratory symptoms among our patient cohort were sore throat, globus sensation, hoarseness, and throat clearing (reported by two-thirds of patients). Other common respiratory symptoms included cough, especially nocturnal, and dyspnea, both of which occurred in more than half of the patients. Previous retrospective studies [31, 32] reported only a 10–12 % prevalence of respiratory symptoms among patients with achalasia, but prospective studies [6, 8, 33] have reported about 50 % prevalence of chronic cough, nocturnal cough, dyspnea, or acute respiratory symptoms. Also, a few case reports [2, 4, 7, 34] have shown the occurrence of respiratory symptoms in achalasia for variable duration and improvement after treatment [8, 33], but none have evaluated whether the type of achalasia was a determining factor for respiratory involvement.

Repeated aspiration of the contents of the dilated esophagus is an important contributory factor in the pathogenesis of respiratory symptoms [2, 4, 15, 35]. Furthermore, dilated esophagus can cause a direct compression of recurrent laryngeal nerve and trachea leading to hoarseness and respiratory symptoms [10, 36, 37]. It was observed that onset of dysphagia (median duration 36 months) preceded respiratory symptoms (median duration 12 months) by about 24 months in both types of achalasia, suggesting that respiratory abnormalities occur later in the course of disease due to chronic aspiration, as described in GERD, [38–40] and other esophageal motility disorders [41]. Aspiration can also result in severe necrotizing infection [42], as occurred in one of our patient who developed lung abscess. We believe that timely diagnosis can prevent these permanent changes and avoid further morbidity.

Regurgitation and respiratory symptoms both tended to be commoner in type I than type II achalasia. Stasis is expected to be more prolonged in type I achalasia, probably due to atonic esophageal body leading to increased risk of aspiration. The difference was not statistically significant for either the stasis, as seen in TBE, or for the prevalence of symptoms, most likely due to small sample size.

We found that PD had a favorable impact on various respiratory symptoms, which is expected due to improved esophageal clearance reducing aspiration. This observation has not been reported in any previously published study. Respiratory symptoms persisted in a few patients after PD despite improvement in dysphagia and esophageal emptying. These patients might have developed chronic changes, which were either irreversible or would take longer to improve after PD.

In our study, functional changes in the lungs were evaluated by spirometry in all patients before PD, which determined the prevalence of PFT abnormalities in achalasia. Functional abnormalities tended to be more prevalent in type I than type II achalasia (59 and 33 %, respectively). Abnormal spirometry was seen in 45 % of the patients, of which restrictive and small airway disease (19 and 16 %, respectively) were the most common dysfunction. Only two previous studies have evaluated functional abnormalities in the lungs in achalasia patients using spirometry [6, 8]. The first of these showed the prevalence of functional abnormality in the lungs to be 20 %; but the authors of this study did not evaluate small airway disease. In our study, those patients suspected to have small airway disease were further evaluated by a high-resolution thorax CT scan, including expiratory film, and were confirmed to have small airway disease. Regarding obstructive airway abnormality, none of the two patients with this pattern in our study showed reversibility with bronchodilators (data not shown), in contrast to results of previous studies, implying that obstruction was not simply because of bronchial hyper-responsiveness.

In our study, PD resulted in normalization of PFT in about half of the patients, demonstrating that PD had a positive impact on pulmonary dysfunction in achalasia. However, this was not statistically significant in terms of number of patients showing improvement, although individual spirometry parameters, namely, FEV_1, FVC, PEFR and MEF_{25-75}, showed significant improvement. This is probably due to a reduction in ongoing aspiration. There was resolution of restrictive abnormality only in one patient, while four patients continued to have restrictive changes on spirometry. Since the radiation dose from the chest CT scan is large, high-resolution CT was done only in patients with clinical or spirometry-based pulmonary dysfunction and repeat scanning post-PD was avoided.

The strength of our study includes objective evaluation of pulmonary symptoms by spirometry along with DLCO, chest X-ray, and high-resolution chest CT scan as required. Also, we re-assessed pulmonary symptoms and spirometry following PD to study the effect of treatment. The limitations of our study include a small number of patients for subgroup analysis and post-treatment follow-up evaluation and short-term follow-up. A body plethysmography is another option to better delineate the respiratory dysfunction.

We excluded patients with mega-esophagus; dysphagia and esophageal emptying are less likely to respond to PD in such patients and, hence, the respiratory dysfunction. Exclusion of these patients might have resulted in an overestimation of the improvement in pulmonary dysfunction, but their inclusion would have underestimated the response. Moreover, these patients are better treated by surgery rather than PD.

The benefit of treatment in this study was reflected by improvement in the clinical and functional pulmonary parameters after PD. Thus, treatment with PD is valuable option for achalasia without any increase in mortality; in addition, complications are infrequent. Based on our data, we conclude that functional pulmonary dysfunction occurs in about half of the patients with achalasia and that timely treatment could lead to reversal. We therefore recommend respiratory evaluation of patients with achalasia in routine and timely management before permanent pulmonary changes occur.

Conflict of interest None.

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