ORIGINAL ARTICLE
COPD

Swallowing dysfunction in patients hospitalised due to a COPD exacerbation

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ABSTRACT

Objectives: This cross-sectional study aimed to investigate the prevalence of self-reported and clinically screened swallowing dysfunction (dysphagia) in COPD patients with severe exacerbations and to identify any associated factors. Findings were then compared to a control group.

Methods: Participants included 30 patients hospitalised due to a COPD exacerbation. The control group consisted of 30 adults hospitalised with acute cardiac symptoms. Data were derived from spirometry, the 150 mL timed water swallow test, a cookie swallow test and a dyspnoea questionnaire (modified Medical Research Council (mMRC)). Scores from the 10-item Eating Assessment Tool (EAT-10) were calculated to assess patient perception of swallowing dysfunction.

Results: Self-reported swallowing dysfunction and clinical signs thereof were more common in COPD patients than in the control group (67% *versus* 23% and 80% *versus* 37%, respectively; $p \leqslant 0.001$). Clinical signs of swallowing dysfunction in the group with acute exacerbation of COPD were associated with self-reported swallowing dysfunction ($p=0.02$) and xerostomia ($p=0.04$). Dyspnoea (mMRC $\geqslant 2$) was more common among the COPD patients (90% *versus* 47%, $p<0.001$). There was a significant negative correlation between lung function and self-reported dysphagia ($r=-0.39$, $p=0.03$), but not between lung function and clinically screened dysphagia ($r=-0.23$, $p=0.21$).

Conclusion: COPD patients hospitalised with an acute exacerbation experienced significantly more self-reported and clinically screened swallowing dysfunction compared to a control group of patients with cardiac symptoms. Both patient groups experienced dyspnoea, but it was twice as common in the group with acute exacerbation of COPD. Both groups also experienced xerostomia.

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Introduction

Awareness of swallowing abnormalities in COPD is growing. A relationship between presence of swallowing dysfunction and COPD both in the stable and symptomatic phase has been identified [1, 2]. In prior studies we have found a 49% prevalence of reduced swallowing capacity [3] and a 33% prevalence of subjective swallowing symptoms [4] in stable-phase COPD patients. This is consistent with data from other research groups [2]. Complications secondary to swallowing impairments have been shown to contribute to disease exacerbations [1]. Robinson *et al.* [5] found that 56% of hospitalised COPD patients had a positive swallow screen with water. There are no data available on how COPD patients hospitalised due to an exacerbation (AECOPD) perceive their swallowing. Knowledge of swallowing function in COPD could be important for clinicians when making decisions regarding preventative and protective measures, treatment and need for fluids and nutrition.

The primary aim of this study was to assess the prevalence of self-reported symptoms and clinical signs of swallowing dysfunction in COPD patients hospitalised with an acute exacerbation and identify associated factors.

However, how swallowing is affected when the patient is more symptomatic is not fully understood. It could possibly be argued that any swallowing symptoms in severely symptomatic COPD patients are an effect of "feeling sick". Our secondary aim was therefore to study whether AECOPD patients were more likely to suffer from suboptimal swallowing than a control group of patients hospitalised due to acute cardiac symptoms. The disease burden of the control group is associated with significant physical and psychological distress, and dyspnoea is a common symptom [6].

Materials and methods

Participants

This was a cross-sectional study of patients admitted with a COPD exacerbation to the ward for pulmonary diseases at a secondary hospital in Sweden between May and September 2018. We used a convenience sample of 30 AECOPD patients. Inclusion criteria were a spirometry-verified COPD diagnosis, which means a ratio of the forced expiratory volume in 1 s (FEV_1)/vital capacity (VC) or forced vital capacity (FVC) <0.70; an unscheduled admittance to the hospital due to a worsening of COPD symptoms, later confirmed by lab tests, arterial blood gas and in some cases with radiography and/or sputum culture; and the ability to independently fill out the questionnaire and participate in the tests. The exclusion criteria were history of known dysphagia and severe comorbidities, *e.g.* neuromuscular diseases, dementia, metastatic cancer and severe sequelae from stroke.

Control subjects were 30 patients selected from acute medical admissions to the hospital's cardiac ward during the same period. Primary diagnoses were congestive heart failure, cardiac arrhythmias or ischaemic heart disease. Inclusion criteria for the cardiac group were hospital admission due to acute cardiac symptoms and the ability to independently fill out the questionnaire and participate in the tests. Exclusion criteria were the same as for the AECOPD group. An additional exclusion criterion for the cardiac group was a COPD diagnosis.

All patients in the study were ambulatory, either independently or with a walker, and were taking their full nutrition by mouth. All patients were assessed when they were medically stable, not on the day of admission. The same tests were applied to both patient groups, apart from the spirometry, which was only performed on the COPD patients.

Procedure

Data were collected by four supervised speech and language pathology students after obtaining written informed consent. All measures except spirometries were performed on the same day. The test protocol consisted of a questionnaire designed for this and a previous study [3] collecting information on previous swallowing function, comorbidities, smoking history, lung function, medications, xerostomia and number of COPD exacerbation-related hospital admissions during the previous year. Lung function and COPD disease severity were obtained through post-bronchodilator spirometry during the hospital stay. A ratio of FEV_1/VC or FVC <0.7 was used to define COPD. Swedish reference values [7] were applied for the FEV_1. Airflow limitations were classified according to the Global Initiative for Chronic Obstructive Lung Disease standard [8]. Body mass index (BMI) was calculated using the formula weight (kg)/height (m^2). Two self-rating tools (modified Medical Research Council (mMRC) and the 10-item Eating Assessment Tool (EAT-10)) were used to evaluate the impact of dyspnoea and swallowing symptoms [9, 10]. Respiratory rate and oxygen saturation were measured prior to the swallowing tests.

Swallowing protocol

Swallow function data were collected through a timed water swallow test and a cookie swallow while the patient was sitting upright. The 150 mL (water) Swallowing Capacity Test (SCT) was originally

described by Nathadwarawala *et al.* [11] in 1992. It has since been confirmed that swallowing speed appears to be a good predictor of ability to tolerate thin liquids among different patient populations [12, 13]. A swallowing capacity index of 10 $mL \cdot s^{-1}$ is regarded to be the lower normal level. Patients were subsequently categorised into one of two groups: a swallowing capacity considered to be within normal limits ($\leqslant$15 s) or >15 s. The swallowing capacity was calculated as the volume (mL) of swallowed water divided by time (s). If the patient was unable to complete the test, the residual volume was measured. Swallowing dysfunction was considered present if reduced swallowing speed or clinical signs of affected swallowing safety (coughing/choking) were present. A solid bolus, typically in the form of a cracker or a cookie, is often part of a bedside swallowing evaluation [14]. Patients were asked to eat a dry cookie at his/her own pace and report any sensation of bolus retention in the mouth or pharynx. The investigator performed an oral inspection using a flashlight. Any coughing or change in voice quality were also noted.

Swallowing questionnaire

EAT-10 [10] is a widely used questionnaire for self-reported (subjective) dysphagia and has been translated and validated in many languages, including Swedish. It has been used previously with this patient group [15–17]. It consists of 10 statements for each of which patients are asked to rate themselves on a five-point scale for severity. An overall score of $\geqslant$3 suggests the presence of dysphagia. Participants were divided into two groups: an EAT-10 score 0–2 (no swallowing problems) and an EAT-10 score 3–40 (swallowing problems).

The mMRC scale

The mMRC dyspnoea scale is a self-rating tool for evaluating breathlessness and is considered a clinically meaningful method of quantifying disease severity in COPD patients [9, 18]. Scoring ranges from 0 to 4. The patient is asked to choose one of the following statements: "I only get breathless with strenuous exercise" (grade 0), "I get short of breath when hurrying on level ground or walking up a slight hill" (grade 1), "On level ground, I walk slower than people of the same age because of breathlessness, or I have to stop for breath when walking at my own pace on level ground" (grade 2), "I stop for breath after walking about 100 yards or after a few minutes on level ground" (grade 3) or "I am too breathless to leave the house or I am breathless when dressing" (grade 4). Patients were defined as being symptomatic if they had an mMRC score $\geqslant$2 [9].

Ethical approval

Written consent forms were obtained from all participants. The study protocol was approved by the regional ethical review board in Uppsala, Sweden (Dnr 2014/405).

Statistical analysis

Descriptive and comparative statistical calculations were made using SPSS statistics (version 24; IBM, Armonk, NY, USA). Demographic data are reported as means, median, standard deviation and range. There were missing data for the 150 mL swallow capacity test (n=4). No values were imputed; hence patients were excluded from analyses for which they had missing values. Mean values for EAT-10 scores, age and respiratory rate for the two groups were compared using an independent-samples t-test. The difference between group means was analysed using the Mann–Whitney U-test for non-normally distributed continuous data and Chi-squared test for categorical variables. Spearman's rank correlation coefficient was used to examine the correlation between lung function (FEV_1 % predicted) and patients' subjective and objective dysphagia.

Variables associated with subjective and objective dysphagia were identified using simple and multivariable binary logistic regression analysis. Simple binary logistic regression was performed for the following potential factors: age, BMI, lung function, xerostomia and dyspnoea. Subjective dysphagia was included in the analysis for objective dysphagia and *vice versa*. Factors shown as statistically significant were then included in a multivariate model. A p-value of <0.05 was regarded as significant.

Results

The demographic and clinical characteristics of the AECOPD group and the control group are described in table 1. Medications pharmacologically known to cause dry mouth (*e.g.* antibiotics, inhalation corticosteroids, diuretics) were prescribed for 93% of the COPD patients and 63% of the controls. There was no significant difference in age between the two groups, but BMI was significantly lower in the AECOPD group (p=0.005).

TABLE 1 Characteristics of the study population and control group

	AECOPD	Controls
Subjects	30	30
Sex		
Female	20 (67)	9 (30)
Male	10 (33)	21(70)
Age years		
Mean	75±7.7	71±13
Median	76	71
Comorbidities (patient-reported)		
Diabetes	7 (23)	9 (30)
Atrial fibrillation	5 (17)	0
Congestive heart failure	3 (10)	0
Hypertension	3 (10)	2 (7)
Angina	3 (10)	3 (10)
Asthma	0	4 (13)
Reflux	5 (17)	11 (37)
GOLD stage		
1	0 (0)	
2	5 (17)	
3	15 (50)	
4	10 (33)	
Spirometry		
FEV_1 % pred	36±15	
FEV_1/FVC	43.5±13.1	
Respiratory rate breaths·min^{-1}	21±6	20±6
Smoking		
Current	6 (20)	2 (7)
Former	23 (77)	14 (47)
Never	1 (3)	14 (47)
BMI kg·m^{-2}	24±6.4	28±5.6
Xerostomia	21 (70)	15 (50)
Dyspnoea symptom burden#	27 (90)	15 (50)

Data are presented as n, n (%) or mean±SD unless otherwise stated. AECOPD: acute exacerbation of COPD; GOLD: Global Initiative for Obstructive Lung Disease; FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; BMI: body mass index. #: modified Medical Research Council score ⩾2.

Dyspnoea (mMRC ⩾2) was more common among the AECOPD patients than the cardiac patients (table 1). COPD patients with more severe airflow limitation experienced a higher percentage of self-reported (p=0.03) and clinically screened (p=0.02) swallowing dysfunction compared to those with milder airflow limitation. Oxygen therapy *via* nasal prongs were given to 11 (37%) of the AECOPD patients at the time of the swallowing tests (range 0.5–2 L·min^{-1}). The mean oxygen saturation measured with pulse oximeter was 92% in the AECOPD group and 95% in the control group (p⩽0.001). The total mMRC mean score was 3.4±1.0 and 2.1±1.5, respectively (p⩽0.001). There was no significant difference in respiratory rate between the two groups (p>0.05).

All patients were on a total oral diet with no restrictions. Screening results are shown in table 2.

TABLE 2 The comparison of screening results between the two groups

	COPD group	Cardiac group	p-value
Subjects n	30	30	
EAT-10 score ⩾3	67	23	<0.001
Swallowing speed >15 s	70	20	<0.001
Cookie swallow (cough, bolus retention)	40	23	0.40
mMRC score ⩾2	90	47	<0.001

Data are presented as %, unless otherwise stated. EAT-10: 10-item Eating Assessment Tool; mMRC: modified Medical Research Council.

TABLE 3 Distribution of mean scores from the two diagnostic/disease groups relating to each item of the 10-item Eating Assessment Tool (EAT)

	COPD group	Cardiac group	p-value
1) My swallowing problem has caused me to lose weight	0.43	0.07	0.04
2) My swallowing problem interferes with my ability to go out for meals	0.43	0.13	0.17
3) Swallowing liquids takes extra effort	0.27	0.13	0.47
4) Swallowing solids takes extra effort	1.13	0.20	<0.001
5) Swallowing pills takes extra effort	0.40	0.13	0.16
6) Swallowing is painful	0.10	0.13	0.76
7) The pleasure of eating is affected by my swallowing	0.83	0.10	<0.001
8) When I swallow food sticks in my throat	0.60	0.33	0.19
9) I cough when I eat	0.57	0.23	0.11
10) Swallowing is stressful	0.53	0.10	0.049

EAT-10: self-reported swallowing symptoms

The results of the questionnaire are detailed in table 3. The total mean±SD score of the EAT-10 was 5.3±5 (range 0–17) in the AECOPD group and 1.6±3.5 (range 0–8) in the cardiac group. The mean EAT-10 scores for each item for both groups are shown in table 3. The most prominent scores in the AECOPD group were item 4 (swallowing solids takes extra effort), item 7 (the pleasure of eating is affected by my swallowing) and item 8 (food sticks in my throat). The first two items (4 and 7) were shared by the cardiac group; however, the third most common problem in that group was item 9 (I cough when I eat). When mean EAT-10 scores of the patients with AECOPD were compared with the control group there was a significant difference ($p \leqslant 0.002$) for questions 1, 4, 7 and 10.

Swallowing tests: clinical signs of dysphagia

Results of the swallowing tests are detailed in table 2. Four participants could not finish the SCT due to coughing/choking. They were included in the analyses since they were above the cut-off limit (⩾15 s). Mean (range) swallowing speed was 28.8 (5–121) s for the AECOPD group and 9.2 s for the controls. Mean±SD capacity was 8.9±7 $mL \cdot s^{-1}$ *versus* 29±26 $mL \cdot s^{-1}$.

Dyspnoea: factors associated with swallowing dysfunction

Statistically significant factors associated with clinical signs of swallowing dysfunction in the AECOPD group were self-reported swallowing dysfunction ($p=0.02$) and xerostomia ($p=0.04$) in a simple logistic regression analysis, while age, lung function (FEV_1 % pred), BMI and dyspnoea were nonsignificant factors ($p>0.05$). Self-reported swallowing dysfunction was significantly associated with clinical signs ($p=0.02$) and xerostomia ($p=0.02$). In the subsequent multivariate model, self-reported swallowing dysfunction was significantly associated with clinical signs and *vice versa* (OR 12.4, 95% CI 1.02–149). There was a significant negative correlation between lung function and self-reported dysphagia ($r=-0.388$, $p=0.03$), but not between lung function and clinical signs of dysphagia ($r=-0.234$, $p=0.21$).

Discussion

The main results of this study were that the prevalence of swallowing dysfunction, both patient-reported and screened in patients hospitalised with a COPD exacerbation, was high. This suggests that dysfunctional swallowing is a prevalent problem and significantly more so in severely symptomatic COPD patients than in hospitalised patients in general, in whom a recent study by Spronk *et al.* [19] found a prevalence of 31% in two centres (n=205). In addition, the AECOPD group was almost three times more likely to suffer from self-reported dysphagia compared to the control group. Swallowing dysfunction in COPD can be caused by the disease itself [2–4], but this study indicates that the additional stress of an acute exacerbation has a significant negative effect on swallowing function. Our findings suggest that, unlike that which has been reported in stable-phase COPD [4, 17], AECOPD patients' perception of their swallowing dysfunction is more congruent.

Interestingly, it was not the questionnaire item describing coughing that was the most prominent item, but difficulty swallowing solids effectively and a subsequent mental burden in the form of decreased pleasure in eating. Both groups experienced dyspnoea, but it was almost twice as common in the AECOPD group.

Several factors may predispose patients with COPD exacerbations to dysfunctional swallowing: disrupted breathing–swallowing pattern exacerbated by tachypnoea and hypercapnia, dysfunction of laryngeal–pharyngeal musculature, reduced endurance and fatigue due to increased work of breathing [20]. Dysphagia may be a cause or an effect of the exacerbation, but in either event it may contribute to the severity of the exacerbation [21].

Subjective dysphagia symptoms documented with the EAT-10 has been shown to predict aspiration risk in COPD patients with a good level of accuracy [15]. However, it is important to identify patients who may have a difficulty swallowing regardless of whether aspiration is present. Aside from being one of the most basic needs in humans, eating can be a great pleasure and an important social interaction and the symptomatic COPD patients in our study indicated that the pleasure of eating was affected by their swallowing difficulties. Utilising a questionnaire for early detection of swallowing disorders has proven successful [22]. The EAT-10 presents limitations for its use with the COPD population, as the items are presented as statements rather than explorative open questions. However, no screening questionnaires validated for the COPD population were available at the time of this study.

There are various biomechanical and kinematic variations in the swallowing mechanism, depending on the viscosity of the bolus, which is why a swallowing screen usually consists of both liquids and solids. The viscosity of a bolus has an effect both on the safety and efficacy of the swallow. Increasing bolus viscosity often results in increased safety of swallowing, but may also result in increased oral and/or pharyngeal residue, which may result in post-swallow airway invasion [23]. In our study, 40% of the COPD patients experienced some problems with the cookie swallow in the form of cough or bolus retention and item 8 in the EAT-10 test (food sticks in my throat) got one of the most prominent scores. Reasons for this might be xerostomia (dry mouth) caused by more usage of medications and need of oxygen. Other reasons might be the underlying nature of the disease where skeletal muscle dysfunction is common and have an adverse effect on endurance and fatigability and/or reduced laryngopharyngeal sensitivity [24–26].

Looking at performance on the 150 mL SCT, the AECOPD group had a significantly slower swallowing speed than the control group (28.9 s *versus* 9.2 s, $p \leqslant 0.001$), although no significant difference was found in respiratory rate. Subsequently, the mean volume swallowed per second was also significantly smaller in the AECOPD group (8.9 $mL \cdot s^{-1}$ *versus* 26.1 $mL \cdot s^{-1}$, $p \leqslant 0.001$). The swallowing speed and time taken per swallow in relation to respiratory rate suggest a compensatory mechanism to airway protection in the AECOPD group.

Approximately 25% of patients with COPD will develop cachexia, a multifactorial syndrome with involuntary progressive weight loss [27], and an unrecognised swallowing impairment may be a factor [17]. However, in our study the mean±SD BMI was 24±6.4 $kg \cdot m^{-2}$. The results of the multivariate analysis showed no significant relationship between swallowing dysfunction and BMI, which separates the COPD patients from other patient cohorts in which poor nutritional status is associated with high dysphagia risk [28]. This is also in accordance with a previous study on 571 COPD patients in a stable phase [4].

Our finding of a strong association between screened (objective) and perceived (subjective) swallowing dysfunction differs from several previous studies that have explored the relationship between patients' self-perceptions of swallowing and the subsequent findings of objective assessments [29, 30]. However, our results can perhaps be explained by the high prevalence of swallowing dysfunction in this group of severely symptomatic COPD patients.

Despite a growing number of articles suggesting that dysphagia is a frequent and potentially serious problem in stable COPD patients, they are not systematically screened when admitted to the hospital due to an exacerbation, a condition which significantly affects muscle strength, for example. A nurse-led swallow screening test as a method to detect a swallowing problem early can potentially minimise swallowing-related complications such as aspiration, discomfort, stress and suboptimal nutritional intake [31]. If the patient fails the screen, a more thorough evaluation should be performed, usually by a speech and language pathologist.

Our study had some limitations. First, the sample size was small and there may have been a possible selection bias because the testing was done when the patient was medically stable, which varied (range 1–5 patients). Second, an instrumental assessment would have strengthened the study, but was not an option. However, when identifying swallowing dysfunction in patients of varying diagnoses, a SCT or questionnaire is often the first step.

Conclusion

The results from this study suggest that COPD patients while hospitalised with an acute exacerbation experience significant self-reported and clinically screened swallowing dysfunction. Further investigations into whether nurse-led screening could be helpful in identifying AECOPD patients at risk for dysphagia are warranted.

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