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Evaluation of different parameters regarding nutritional status in patients suffering from COPD

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Abstract
Nutritional depletion is a prevalent finding in patients who have COPD. Several studies have demonstrated that under nutrition is an independent predictor of all cause and respiratory morbidity and mortality in COPD and has an additive effect with other factors that increase mortality. Investigators have identified a positive correlation between body weight and the Forced Expiratory Volume in the 1st second. Even among stable COPD patients there is a high proportion of under nutrition. COPD patients are at risk of weight loss and nutritional deficiencies because of a 15 to 25% increase in resting energy expenditure from breathing; a higher energy cost of daily activities; reduced caloric intake 2 relative to need because of dyspnea; and the catabolic effect of inflammatory cytokines such as TNF-α5. Hence based on above reported findings the present study was planned to estimate the nutritional status of stable COPD patients

The present study was planned in Narayan Medical College and Hospital, Jamuhar Sasaram, Bihar, India. Total 100 cases of the COPD were enrolled in the present study. All patients were classified based on combined COPD assessment by revised Global Initiative for Chronic Obstructive Lung Disease GOLD 2011. The study group was consist of male and female patients age more than 40 years and less than 80 years, diagnosed to have COPD by spirometry. The body mass index (BMI) was calculated as weight (in Kg)/height2 (in metres). Mid-arm circumference (MAC) measurement was done in the non-dominant arm of the patient using a flexible measuring tape. Blood samples were analyzed for albumin, total protein, blood urea, serum creatinine. Normal ranges of our biochemistry laboratory are as follows: Albumin: 3.5-5.3g/l, total protein: 6.0-8.3g/l, blood urea: 7- 35 mg/dl and creatinine: 0.6-1.2 mg/dl.

From the above observations, we may conclude that patients with COPD are generally malnourished and the prevalence is more in a developing country like India. Nutritional interventions in the form of dietary supplement have shown improvement in anthropometry, exercise capacity and HRQL but did not show any significant improvement in pulmonary function.

Keywords: COPD, nutritional status, albumin, chronic obstructive pulmonary disease, etc

Introduction

Chronic obstructive pulmonary disease (COPD) is a lung disease characterized by chronic obstruction of lung airflow that interferes with normal breathing and is not fully reversible. The more familiar terms 'chronic bronchitis' and 'emphysema' are no longer used, but are now included within the COPD diagnosis. COPD is not simply a "smoker's cough" but an under-diagnosed, life-threatening lung disease.

A COPD diagnosis is confirmed by a simple test called spirometry, which measures how deeply a person can breathe and how fast air can move into and out of the lungs. Such a diagnosis should be considered in any patient who has symptoms of cough, sputum production, or dyspnea (difficult or labored breathing), and/or a history of exposure to risk factors for the disease. Where spirometry is unavailable, the diagnosis of COPD should be made using all available tools. Clinical symptoms and signs, such as abnormal shortness of breath and increased forced expiratory time, can be used to help with the diagnosis. A low peak flow is consistent with COPD, but may not be specific to COPD because it can be caused by other lung diseases and by poor performance during testing. Chronic cough and sputum production often precede the development of airflow limitation by many years, although not all individuals with cough and sputum production go on to develop COPD.

The formal diagnosis of COPD is made with spirometry; when the ratio of forced expiratory volume in 1 second over forced vital capacity (FEV_1/FVC) is less than 70% of that predicted for a matched control, it is diagnostic for a significant obstructive defect. Criteria for assessing the severity of airflow obstruction (based on the percent predicted postbronchodilator FEV_1) are as follows:

- Stage I (mild): FEV_1 80% or greater of predicted
- Stage II (moderate): FEV_1 50-79% of predicted
- Stage III (severe): FEV_1 30-49% of predicted
- Stage IV (very severe): FEV_1 less than 30% of predicted or FEV_1 less than 50% and chronic respiratory failure

Developments in the 20th century included the widespread use of spirometry, recognition of airflow obstruction as a key factor in determining disability, and the improvement of pathologic methods to assess emphysema. Participants in the Ciba symposium of 1958 proposed definitions of chronic bronchitis and emphysema, incorporating the concept of airflow obstruction.

Chronic bronchitis is defined clinically as the presence of a chronic productive cough for 3 months during each of 2 consecutive years (other causes of cough being excluded). Emphysema, on the other hand, is defined pathologically as an abnormal, permanent enlargement of the air spaces distal to the terminal bronchioles, accompanied by destruction of their walls and without obvious fibrosis.

Airflow limitation in emphysema is due to loss of elastic recoil and decrease in airway tethering, whereas chronic bronchitis leads to narrowing of airway caliber and increase in airway resistance. Although some patients predominantly display signs of one of these diseases or the other, most fall somewhere in between the spectrum of these two conditions.

Past guidelines of COPD have been pessimistic at best, indicating that the disease process is irreversible and that therapy has little to offer. However, a more optimistic view has come to be widely accepted. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) guidelines define COPD as a disease state characterized by airflow limitation that is not fully reversible, is usually progressive, and is associated with an abnormal inflammatory response of the lungs to inhaled noxious particles or gases [1].

Oral and inhaled medications are used for patients with stable COPD to reduce dyspnea, improve exercise tolerance, and prevent complications. Most of the medications used in COPD treatment are directed at the potentially reversible mechanisms of airflow limitation. Pathologic changes in chronic obstructive pulmonary disease (COPD) occur in the large (central) airways, the small (peripheral) bronchioles, and the lung parenchyma. Most cases of COPD are the result of exposure to noxious stimuli, most often cigarette smoke. The normal inflammatory response is amplified in persons prone to COPD development. The pathogenic mechanisms are not clear but are most likely diverse. Increased numbers of activated polymorphonuclear leukocytes and macrophages release elastases in a manner that cannot be counteracted effectively by antiproteases, resulting in lung destruction.

The primary offender has been found to be human leukocyte elastase, with synergistic roles suggested for proteinase-3 and macrophage-derived matrix metalloproteinases (MMPs), cysteine proteinases, and a plasminogen activator. Additionally, increased oxidative stress caused by free radicals in cigarette smoke, the oxidants released by phagocytes, and polymorphonuclear leukocytes all may lead to apoptosis or necrosis of exposed cells. Accelerated aging and autoimmune mechanisms have also been proposed as having roles in the pathogenesis of COPD [2-3].

Cigarette smoke causes neutrophil influx, which is required for the secretion of MMPs; this suggests, therefore, that neutrophils and macrophages are required for the development of emphysema. Studies have also shown that in addition to macrophages, T lymphocytes, particularly CD8+, play an important role in the pathogenesis of smoking-induced airflow limitation. To support the inflammation hypothesis further, a stepwise increase in alveolar inflammation has been found in surgical specimens from patients without COPD versus patients with mild or severe emphysema. Indeed, mounting evidence supports the concept that dysregulation of apoptosis and defective clearance of apoptotic cells by macrophages play a prominent role in airway inflammation, particularly in emphysema [4]. Azithromycin (Zithromax) has been shown to improve this macrophage clearance function, providing a possible future treatment modality [5]. In patients with stable COPD without known cardiovascular disease, there is a high prevalence of micro albuminuria, which is associated with hypoxemia independent of other risk factors [5]

Airway hyper responsiveness (ie, Dutch hypothesis) stipulates that patients who have nonspecific airway hyperreactivity and who smoke are at increased risk of developing COPD with an accelerated decline in lung function. Nonspecific airway hyperreactivity is inversely related to FEV1 and may predict a decline in lung function. The data regarding the possible role of airway hyperresponsiveness as a risk factor for the development of COPD in people who smoke are unclear. It is important to note, however, that 60% demonstrate bronchial hyperresponsiveness [7] Moreover, bronchial hyperreactivity may result from airway inflammation observed with the development of smoking-related chronic bronchitis. This may contribute to airway remodeling, leading to a more fixed obstruction, as is seen in persons with COPD.

Nutritional depletion is a prevalent finding in patients who have COPD. Several studies have demonstrated that under nutrition is an independent predictor of all cause and respiratory morbidity and mortality in COPD and has an additive effect with other factors that increase mortality. Investigators have identified a positive correlation between body weight and the Forced Expiratory Volume in the 1st second. Even among stable COPD patients there is a high proportion of under nutrition. COPD patients are at risk of weight loss and nutritional deficiencies because of a 15 to 25% increase in resting energy expenditure from breathing; a higher energy cost of daily activities; reduced caloric intake 2 relative to need because of dyspnea; and the catabolic effect of inflammatory cytokines such as TNF-α5. Hence based on above reported findings the present study was planned to estimate the nutritional status of stable COPD patients.

Methodology

The present study was planned in Narayan Medical College and Hospital, Jamuhar Sasaram, Bihar, India. Total 100 cases of the COPD were enrolled in the present study. All patients were classified based on combined COPD assessment by revised Global Initiative for Chronic Obstructive Lung Disease GOLD 2011. The study group was consist of male and female patients age more than 40 years and less than 80 years, diagnosed to have COPD by spirometry. The body mass index (BMI) was calculated as weight (in Kg)/height2 (in metres). Mid-arm circumference (MAC) measurement was done in the non-dominant arm of the patient using a flexible measuring tape. Blood samples were analyzed for albumin, total protein, blood urea, serum creatinine. Normal ranges of our biochemistry laboratory are as follows: Albumin: 3.5-5.3g/l, total protein: 6.0-8.3g/l, blood urea: 7- 35 mg/dl and creatinine: 0.6-1.2 mg/dl.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: Male and female patients age more than 40 years and less than 80 years, diagnosed to have COPD by spirometry.

Exclusion criteria: Patients with any other comorbidity such as diabetes mellitus, chronic systemic or localized infections, renal failure, hepatocellular failure, heart failure, malignancies, etc.; lactating and pregnant women; and those with a history of use of systemic steroids, antibiotics, or hospitalization for an exacerbation within past 4 weeks were excluded from the study.

Results & Discussion

In general, insufficient attention is paid to the nutritional assessment of patients with COPD in routine practice. It should, like spirometry and arterial blood gas analysis, be included in the initial clinical evaluation of these patients. Regular follow-up of nutritional status is also essential because this variable has been shown to have independent prognostic value, a more than sufficient reason for its assessment [8]. Consequently, simple, easy-to-use, cheap, and reproducible procedures for the assessment of nutritional status are needed. There is no single ideal nutritional marker, but a combination of several simple parameters can facilitate the diagnosis of malnutrition in these patients [9]. Several parameters are used to assess nutritional status and they can be basically categorized as either anthropometric or biochemical.

Abnormalities in skeletal muscle are common in COPD patients; contractility, strength, and resistance are reduced, while fatigability increases [10]. The etiology of muscular dysfunction in COPD is multifactorial and includes electrolyte abnormalities, atrophy due to lack of exercise, prolonged use of drugs such as corticosteroids [11-12], changes in the geometry of the thoracic cage, hypoxia, and malnutrition. Malnutrition decreases muscular strength and resistance, and reduces glycolytic and oxidative capacity in both type I and type II fibers. A weak respiratory musculature contributes to dyspnea and has a negative impact on exercise tolerance [13].

Biochemical changes affect the alveolar surfactant provoking a decrease in total phospholipids, phosphatidylglycerol, and phosphatidylcholine. This triggers a rise in surface tension and a corresponding decrease in the protective effectiveness of the surfactant. These changes are due to a reduction in the enzyme activity that regulates its synthesis, to a reduced availability of energy substrates, and to characteristics of the local oxidative metabolism. These abnormalities may be reversible on re-nourishment, and a normal state is recovered more rapidly than in the case of connective tissue [14].

Chailleux *et al.* [15] found that lower body mass index (BMI) was an independent negative determinant of survival in patients with COPD. The inverse relationship between BMI and incidence of COPD is in agreement with results of Higgens *et al*. [16] who reported that after an average follow-up period of 15 years, the incidence of COPD was highest in lean men and lowest in those who were overweight.

Mid-arm circumference and skin fold thickness reflect muscle mass and the body's fat stores respectively. A low triceps skin fold has been shown in patients with COPD patients, and it helps in early identification of a deteriorating clinical state. This measurement is not affected by fluid retention often seen in COPD patients, which might mask weight loss. Studies by Lewis *et al*. [17] and Sridhar *et al*. [18] had also shown no significant improvement in mid-arm circumference and skin fold thickness in patients.

Mean percentage fat levels showed a declining trend whereas mean percentage fat free mass levels showed inclining trends with increasing stage of COPD and both are having statistically significant association with COPD stages. Significant decrease of fat mass in our study which is observed among disease stages may be related to factors like anorexia, early satiety and dyspnoea during eating. Mid arm circumference and mid-calf circumference showed a significant decline with increasing severity of COPD. Muscle wasting increased with progression of disease. It may be due to accelerated muscle proteolysis, hormonal dysfunction and increased level of cytokine.

Table 1: Anthropometry indices, BIA and Biochemical indices

Variable	GOLD classification of COPD			
	Group A	Group B	Group C	Group D
No. of Cases	25	25	25	25
Anthropometry Indices				
Body Mass Index (BMI)	21.4 – 28.1	18.4 – 22.9	17.4 – 20.6	14.2 – 19.5
Mid arm circumference (MAC)	25.7 – 28.3	22.1 – 27.5	22.0 – 24.3	17.8 – 22.3
Mid Arm Muscle Circumference (MAMC)	24.8 – 26.5	22.5 – 24.8	18.9 – 21.6	16.1 – 20.4
Triceps Skin Fold (TSF)	12.5 – 17.3	10.2 – 14.5	7.3 – 13.5	4.5 – 8.9
Bioelectric impedance analysis				
Fat Mass (FM)	29.4 – 44.6	26.4 -38.9	23.4 – 35.7	15.7 – 31.4
Free Fat Mass (FFM)	31.4 – 39.8	32.4 – 38.6	31.4- 37.5	37.5 – 33.1
Total Body Water (TBW)	49.2 – 53.7	43.7 – 51.6	38.4 – 52.4	35.4 – 49.8
Biochemical Parameters				
Albumin	3.4 – 4.2	4.1- 4.9	3.3 – 4.2	3.3 – 4.0
Protein	6.2 – 7.1	5.2 – 7.6	6.1 – 7.3	5.5 – 7.2
Urea	31.2 – 45.8	28.4 – 43.6	24.5 – 44.9	24.6 – 43.5
Creatinine	0.5 – 1.1	0.6 – 1.1	0.4 – 1.2	0.3 – 1.1

King D *et al*. observed that underweight is a poor prognostic sign in chronic obstructive pulmonary disease (COPD) is at least in part associated with the severity of airflow obstruction. Nutritional supplementation in undernourished patients with COPD can lead to weight gain and improvements in respiratory muscle function and exercise performance [19]. In a study perform by Yuceege MB, *et al* on 60 out patients of COPD without comorbidity they found that malnutrition leads to increased hospital admission [20].

Sabino PG, *et al* had observed that overweight/obese patients had a higher fat-free mass (FFM) index, exercise capacity and maximal inspiratory pressure in comparison to normal weight and underweight patients, respectively [21]. Leila Y, *et al* had done cross-sectional study on 63 COPD patients with mean age (SD) of 67.6 (9.4) years. In their study reduction of body mass index (BMI), Mid-Arm Muscle Circumference (MAMC) and Fat-Free Mass (FFM) were observed alongside an increase in disease severity but it was not significant. Significant reduction of Fat Mass (FM) (P= 0.007), Fat Mass Index (FMI) (P= 0.03) and

biochemical indices like albumin (P= 0.001) and total protein (P= 0.04) were associated with an increase in disease stages. Nutritional status indices like MAMC, FFM and FM, other than BMI should be used for early diagnosis of malnutrition before weight loss occurs [22].

Nutritional depletion is a prevalent finding among patients who have COPD, in particular those who have advanced disease. The prevalence of weight loss in stable COPD is in the range of 20%, and it increases to 35% among those who are hospitalized [23-24]. Several studies have found that the body mass index (BMI) is an independent risk factor for COPD mortality [25]. Landbo and colleagues [26], in The Copenhagen City Heart Study, found BMI to be an independent predictor of all-cause and respiratory mortality among COPD patients with FEV1 less than 50% predicted. The impact of weight change on survival in COPD also was examined retrospectively by Schols and colleagues [27] in 400 COPD patients who participated in a pulmonary rehabilitation programme. A low BMI (less than 25 kg/m2) was associated with a significant increase in the risk for mortality (P < .001). In a prospective post hoc analysis of 203 COPD patients who received nutritional support, weight gain (greater than 2 Kg/8 wks) was a significant predictor of survival [27]. Studies using more complex tests to evaluate nutrition, such as midthigh [28] and midarm [29] muscle cross-sectional area obtained by CT also have shown significant association between malnutrition and mortality in COPD, with a predictive value that is superior to that of BMI. Taken together, the evidence suggests that weight loss can be considered an independent risk factor for mortality in patients who have COPD.

Assessment of the nutritional status in COPD is a vital step in management of COPD patients. Planning what we eat and balancing our meals help us to manage our health. Good nutrition helps the body fight infections by strengthening our immunity. Assessment of nutrition and filling of lacuna will not cure COPD but will surely postpone the associated comorbidity. Physicians should aware of different methods that can be used for nutritional assessment.

This was a single centre study and the number of subjects enrolled in our study was small. Hence, large scale validation is needed. In this study, staging of COPD was made by an older version of GOLD guideline, by spirometry finding alone.

Conclusion

From the above observations, we may conclude that patients with COPD are generally malnourished and the prevalence is more in a developing country like India. Nutritional interventions in the form of dietary supplement have shown improvement in anthropometry, exercise capacity and HRQL but did not show any significant improvement in pulmonary function.

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