

LOW BODY MASS INDEX AND LOW SERUM ALBUMIN LEVEL AS RISK FACTORS FOR CARBON DIOXIDE RETENTION DURING ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Vishnu Sharma M¹, Anupama N^{2*}, Harsha D S³, Viswambhar⁴, AlakaChaitra Bhat³, Basavaraj S³, Rahul Antony Simon³

Department of Pulmonary Medicine, A J Institute of Medical Sciences, Kuntikana
Mangalore, Karnataka, India¹

Department of Physiology, Kasturba Medical College, Manipal University, Mangalore, Karnataka, India²

Department of Pulmonary Medicine, A J Institute of Medical Sciences, Kuntikana,
Mangalore, Karnataka, India³

Department of Pulmonary Medicine, Chettinadu Health Institute and Research Center,
Chennai, India⁴

*** Corresponding Author:** Anupama N

Abstract: This study was done to compare BMI and serum albumin level as risk factors for carbon dioxide retention during acute exacerbation of COPD. It was a hospital based case control study done on 60 male patients aged of 40-80yrs already diagnosed as COPD as per GOLD 2010 guidelines. All patients with Paco2 level >46 mmHg which was not a compensation for metabolic alkalosis were classified as cases and those with Paco2 level <46 mmHg were controls. BMI and Serum albumin levels were compared between the two groups. Data was analysed by calculating odds ratio. The study shows that patients with low BMI showed 20 times risk of having carbon dioxide retention that of normal BMI patients and patients with low albumin levels showed a risk of having carbon dioxide retention 46 times that of patient with normal albumin.

Keywords: Acute exacerbation of COPD, Carbon dioxide retention, BMI, Serum albumin, hypercapnea.

I. INTRODUCTION

Hypercapnea is known to occur during acute exacerbation of chronic obstructive pulmonary disease (COPD) which increases the morbidity and mortality in these patients. Hypercapnea is defined as an arterial Paco2 above 46 mm Hg that does not represent compensation for a metabolic alkalosis [1]. In a patient with COPD, chronic hypercapnea secondary to alveolar hypoventilation can be considered an adaptive response to obstructive lung disease by decreasing the work of breathing, preventing respiratory muscle fatigue and allowing a diminished sensation of dyspnoea [2]. Reduced ventilation may also be due to reduced ventilatory drive. This may lead to carbon dioxide retention when it is combined with reduced ventilation due to a high work of breathing because of severe obstruction and hyperinflation coupled with ventilatory muscle impairment. The abnormalities in alveolar ventilation and a reduced pulmonary vascular bed further worsen the abnormalities [3]. During exacerbations there is increase in hyperinflation and gas trapping, with reduced expiratory flow, thus accounting for the increased dyspnoea [4]. There is also worsening of ventilation perfusion abnormalities, which can result in hypoxemia [5]. When carbon dioxide elimination is impaired due to hypoventilation, hypercapnea occurs. Hypoventilation leads to both hypoxia and hypercapnea as a result of decrease in total volume of air inhaled and exhaled each minute.

Low serum albumin level is one of the poor prognostic indicators in COPD patients [6]. Over the past few years several studies have indicated that nutritional state is impaired in a high proportion of patients with chronic obstructive lung disease [7]. Weight loss is common with severe disease [8]. Malnutrition is a leading cause of impaired respiratory muscle contractility, affecting both strength and endurance [9]. In patients with severe malnutrition significant changes

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in respiratory and limb muscle contraction and relaxation characteristics and fatigability properties have been described with decompensation in lung function [10]. Protein-calorie malnutrition should be suspected in any patient with COPD and acute respiratory failure [7]. Studies indicate that people with low BMI are at a higher risk of developing COPD [11,12]. Body weight and body mass index (BMI) are independent risk factors for mortality in chronic obstructive pulmonary disease (COPD) patients [13,14]. The role of low BMI as a determinant of poor survival in these patients could have been due to several factors, such as respiratory muscle weakness, impaired gas exchange [15], and impaired immune response, all of which have been related to malnutrition in COPD patients. In the present study, we analysed patients with acute exacerbation of COPD with and without carbon dioxide retention. These two groups were correlated with regard to BMI and serum albumin level.

II. MATERIALS AND METHODS

It is a hospital based case control study, done on 60 patients with 30 cases and 30 controls conducted over a year on the patients admitted with acute exacerbation of COPD in department of pulmonary medicine of A.J. Institute of Medical Sciences; Mangalore. Ethical clearance was obtained from institutional ethical committee. Patients already diagnosed as COPD as per GOLD 2010 guidelines, on routine follow-up in out-patient department and admitted with acute exacerbation were included in the study after informed consent. Age matched male patients of 40-80 years were included in the study. History regarding patient particulars, severity of COPD, smoking were noted. Arterial Blood Gas analysis (ABG) was done on admission.

The patients excluded from this study are those who had evidence of pneumonia or bronchiectasis developed as a sequel of other disease, clinically or on chest radiography (PA view), patients with ischemic heart disease, patients with symptoms of obstructive sleep apnoea, patients in whom all investigations could not be completed, patients who are not willing to participate, patients in whom carbon dioxide levels did not return to normal on discharge, patients in whom all tests could not be completed, patients who died in the hospital. Patients were grouped under two categories depending upon the ABG. All patients with $Paco_2$ level >46 mmHg which was not a compensation for metabolic alkalosis were classified as patients with carbon dioxide retention (cases). All patients with $Paco_2$ level <46 mmHg were classified as patients without carbon dioxide retention (controls). Chest x-ray, blood routine investigations, ECG, echocardiography, serum electrolytes, serum total protein, albumin, globulin were done in all patients. ABG was repeated before discharge in all patients. BMI, severity of COPD and serum albumin were compared between these groups as a risk factor for carbon dioxide retention.

Body mass index is calculated weight in kg divided by square of height in meters [16]. They were further classified as per Indian Council for Medical Research classification. Underweight <18 , normal 18-23, overweight 23-25, obese >25 . Serum albumin levels is classified as low i.e., <3.5 g/dl and Normal 3.5-5.5g/dl [17]. Classification of severity of COPD is done according to the GOLD 2010 guidelines (table 1). Patients were classified into mild, moderate, severe and very severe as per their FEV₁ before admission in OPD follow-ups.

Table I: Classification of severity of airflow limitation in COPD GOLD 2010

Classification of severity of airflow limitation in COPD		
In patients with FEV ₁ /FVC $<70\%$		
GOLD 1	MILD	FEV ₁ $\geq 80\%$ predicted
GOLD 2	MODERATE	$50\% \leq FEV_1 < 80\%$ predicted
GOLD 3	SEVERE	$30\% \leq FEV_1 < 50\%$ predicted
GOLD 4	VERY SEVERE	FEV ₁ $< 30\%$ predicted or FEV ₁ $< 50\%$ predicted plus chronic respiratory failure

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III. STATISTICAL ANALYSIS

The data was entered into the Microsoft Office Excel 2007 and odds ratio was calculated for the variables under study.

IV. RESULTS

BODY MASS INDEX

24 patients (80%) in the case group had low BMI. In the control group only 5 patients had low BMI. Whereas 25 patients (83.33%) had normal BMI.

Table II: 2x2 table for BMI

BMI	Cases	Controls
Low	24(a)	5(b)
Normal	6(c)	25(d)

Odds Ratio = $ad / bc = 20$

The above odds ratio shows that patients with low BMI showed 20 times risk of having carbon dioxide retention that of normal BMI patients

SERUM ALBUMIN

28 patients (93.33%) of Case group had low serum albumin. In control group only 7 patients had low serum albumin. Whereas 23 patients (83.33%) had normal serum albumin.

Table III: 2x2 table for serum albumin

Albumin Levels	Cases	Controls
Low	28(a)	7(b)
Normal	2(c)	23(d)

Odds Ratio = $ad/bc = 46$

The above odds ratio shows that patients with low albumin levels showed a risk of having carbon dioxide retention 46 times that of patient with normal albumin

V. DISCUSSION

The incidence of under nutrition in patients with COPD depends on disease severity. Several studies indicate that the prevalence of nutritional abnormalities increases as disease severity increases [18]. Weight loss is also common with severe disease. Malnutrition is a leading cause of impaired respiratory muscle contractility [9] and this may promote carbon dioxide retention.

In our study 80% of patients with carbon dioxide retention had low BMI. Majority (83.3%) of the control group had normal BMI. Patients with low BMI had [19] (Odds ratio = 20) times the risk of carbon dioxide retention to that of normal BMI patients. Several studies have stressed the prevalence of malnutrition in COPD patients. Studies have shown that low BMI is one of the independent poor prognostic factors in COPD with a clear association between decreased BMI and increased mortality. Skeletal muscle dysfunction is associated with underweight. COPD patients with low BMI have skeletal muscle dysfunction. Several studies have highlighted the association between low BMI and respiratory muscle weakness and impaired gas exchange. Patients with low body weights have greater gas trapping, lower diffusing capacity, and less exercise capacity than do persons with similar respiratory mechanics but normal body weights [15]. Loss of body cell mass is associated with a reduction in the mass of the diaphragm and of the respiratory muscles, resulting in declines in strength and endurance. These effects can contribute to undesirable clinical sequelae that include hypercapnic respiratory failure, difficulty with weaning from mechanical ventilation, and

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nosocomial lung infections [20]. One of the previous studies done on 90 patients with respiratory failure showed a correlation between body weight loss and increase in carbon dioxide retention [19]. One study by Driver AG et al on patients with COPD and respiratory failure showed that in majority of these patients body weight was significantly lower than controls [21]. Patients with greater weight loss had greater degree of decompensation of lung function [7]. Another study showed that weight loss is associated with declining lung function in many individuals with COPD [22]. In our study 93.33% of the patients with carbon dioxide retention had low serum albumin. In the control group 23.33% had low serum albumin. Our study shows that patients with low albumin levels have 46 times more the risk of having carbon dioxide retention compared to patients with normal serum albumin. Serum albumin levels is one of the commonly used indicators of nutritional status. Nutritional status impairment in COPD patients has been highlighted by various studies [6, 21]. Malnutrition in COPD patients has been shown to be most important cause of respiratory muscle dysfunction [9]. This may further contribute to hypoventilation and carbon dioxide retention. One of the previous studies on patients with COPD respiratory failure concluded that protein calorie malnutrition should be suspected in this group of patients [7]. Another study done by Schols A et al demonstrated the relation between impaired gas exchange in COPD patients and serum albumin [21].

VI. CONCLUSION

Patients with low BMI and low serum albumin level have greater risk of having carbon dioxide retention than patients with normal BMI during acute exacerbation of COPD. Further, Cohort study with a large sample is required to confirm the result.

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