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STUDY ON SERUM ELECTROLYTES AND URIC ACID IN PATIENTS WITH ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is the third leading cause of death and a major health problem worldwide. COPD is complicated by acute exacerbations that are associated with healthcare expenditures and high morbidity. Patients with severe COPD exacerbation have factors that influence serum electrolyte imbalance, such as hypoxia, respiratory acidosis, metabolic abnormalities such as serum electrolyte imbalance, uremia, and liver function abnormalities. Serum electrolyte imbalance such as hyponatremia, hypokalemia, hyperbilirubinemia, and elevated levels of transaminases, blood urea, and serum creatinine are either caused by the disease process or the therapy initiated.

Objectives of this study: The objectives of this study is to estimate and compare the levels of serum electrolytes (sodium and potassium) and uric acid levels in patients with AECOPD and controls.

Materials and Methods: Fasting blood samples from all the subjects were collected for the estimation of serum electrolytes like sodium and potassium and uric acid in fully automated biochemistry analyser.

Results: We included a total of 100 patients (Group 1, n=50 & Group 2, n= 50) based on inclusion and exclusion criteria. We found significantly decreased levels of serum levels of sodium and potassium in patients with AECOPD compared to the controls. There was no statistically significant differences in uric acid levels between the two groups.

Discussion and Conclusion: Electrolyte imbalances can cause respiratory muscle weakness and impair airway function in COPD patients. Serum sodium and potassium levels were lower in acute exacerbations of COPD who deceased during the admission, and there were no statistically significant differences in uric acid levels between the two groups. The levels of these parameters should be measured and corrected during AECOPD treatment to decrease mortality.

Key-words: electrolytes, sodium, potassium, uric acid, chronic obstructive pulmonary disease and acute exacerbation.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death and a major health problem worldwide [1]. COPD is complicated by acute exacerbations that are associated with healthcare expenditures and high morbidity. Patients with severe COPD exacerbation have factors that influence serum electrolyte imbalance, such as hypoxia,

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respiratory acidosis, metabolic abnormalities such as serum electrolyte imbalance, uremia, and liver function abnormalities [2]. Serum electrolyte imbalance such as hyponatremia, hypokalemia, hyperbilirubinemia, and elevated levels of transaminases, blood urea, and serum creatinine are either caused by the disease process or the therapy initiated [3].

Electrolyte imbalances can also cause respiratory muscle weakness and impair airway function in this group of patients [4]. Hypercapnia occurs during COPD exacerbations; the sudden decrease in ventilation leads to acute respiratory acidosis or deteriorates pre-existing chronic respiratory acidosis. Owing to the high prevalence of comorbidities [5] and corresponding multi-drug therapies in these patients, mixed acid-base and hydro-electrolyte disorders are becoming increasingly common, particularly in critically ill and elderly populations [6].

Uric acid has a particular importance in the human body owing its antioxidant nature; however, it can have the opposite effect, acting as a proinflammatory factor [7, 8]. A limited number of studies have investigated whether hyperuricemia is associated with a high risk of mortality in patients with COPD [9]. The associations between hyperuricemia and serum electrolyte imbalance and COPD-related mortality are still unclear. Hence we have taken up this study to evaluate serum electrolytes and uric acid levels in patients with AECOPD.

OBJECTIVES OF THE STUDY: The objectives of this study is, to estimate and compare the levels of serum electrolytes (sodium and potassium) and uric acid levels in patients with AECOPD and controls.

MATERIALS AND METHODS

Source of Data: A Hospital Based observational study conducted in the department of General Medicine in association with department of Respiratory Medicine.

Study design: Prospective observational study.

Study subjects: Patients with AECOPD and controls.

Sample size: we included a total of 100 patients on hemodialysis, they were grouped into two groups Group 1 cases (50 patients) and Group 2 (50 patients).

Inclusion criteria: we included the patients with AECOPD.

Exclusion Criteria: other causes of dyselectrolytemia were excluded from the study like chronic renal failure, diabetic ketoacidosis, adrenocortical insufficiency, cerebral salt wasting.

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Sample collection and Laboratory Analysis: Fasting blood samples from all the subjects were collected for the estimation of serum electrolytes like sodium and potassium and uric acid in fully automated biochemistry analyser.

Statistical Analysis: Significance of differences of average sodium and potassium levels in two groups were evaluated statistically using Student's' test. (p value <0.05 was significant)

RESULTS

We included a total of 100 patients (Group 1, n=50 & Group 2, n= 50) based on inclusion and exclusion criteria.

Table 1: Shows comparisons of demographic profile and laboratory parameters of study subjects.

Parameters	AECOPD	Controls
Age (in years)	58.8±8.8	59.32±7.86
F/M (number)	22/28	20/30
SBP	108.2±6.88	110±3.42
DBP	78±4.23	82.6±4.30

Table 2: Shows serum electrolyte levels and uric acid in cases and controls

Parameters	AECOPD	Controls	P value
Sodium (meq/L)	126.2±4.7	143.44±3.62	HS
Potassium (meq/L)	3.6±0.78	4.3±1.18	HS
Uric acid (mg/dL)	5.4±1.32	5.4±1.42	NS

DISCUSSION

In patients with acute exacerbation of COPD, it has been observed that besides the signs of acute infection, there may be number of co-morbid conditions like type II respiratory failure and carbon dioxide narcosis, metabolic abnormalities such as dyselectrolytemia, uremia and liver function abnormalities. Though most of the abnormalities are correctable, attempt is not made to correct either due to over looking or due to lack of lab facility for 24 hrs monitoring. In our study, we measured serum electrolytes (sodium and potassium) in COPD exacerbation patients. We found a significantly low level of serum sodium and potassium in the COPD patients than that of the healthy controls (p value <0.05 in each case). Patients with COPD are susceptible to hyponatremia for a number of reasons like development or worsening of hypoxia, hypercapnia and respiratory acidosis and right side heart failure with development of lower limb

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edema, renal insufficiency, use of diuretics, SIADH (Syndrome of Inappropriate Antidiuretic Hormone Synthesis), malnutrition, and poor intake during acute exacerbations are common contributing factors in such patients. Activation of the renin angiotensin aldosterone system and inappropriately elevated plasma arginine vasopressin (AVP) in COPD may all these factors aggravate the electrolyte imbalance during acute exacerbation of COPD (Bauer et al, Vallyet al, Das et al) [10-12].

Hypokalemia is a common electrolyte disturbance in COPD. Serious symptoms, including strokes that lead to respiratory paralysis and tetany, may occur in severe hypokalemia . A study conducted by Goli et al. demonstrated that serum K levels were lower in AECOPD patients than in control subjects, which is consistent with our results. Similarly, a study by Ouf et al. found lower serum K, Na, Mg, Ca, and chlorine levels in patients with AECOPD before treatment than in control subjects and higher $PaCO_2$ and lower pH in COPD patients than in control group. The same study also demonstrated significantly decreased serum Na, K, Mg, and Ca levels in patients who required mechanical ventilation. Among our patients, those previously receiving home LTOT and NIMV had more acidic pH and higher $PaCO_2$ than who did not receive. In addition, significantly more hypokalemia was noted in our AECOPD patients whose hospital admission resulted in mortality. Both Mg and K levels were significantly lower in patients who received LTOT and NIMV than in those who did not. A possible mechanism for this electrolyte imbalance may be the indispensable diuretic treatment given for cor pulmonale, which develops in late COPD. Besides, beta-agonists, which are used for treating both exacerbations and stable disease, stimulate Na+/K+ ATPase, facilitating intracellular K and Mg uptake. Side effects of systemic corticosteroids that are frequently used to treat attacks may be another explanation for the electrolyte imbalance mechanism [13-15].

Uric acid is the end-product of nucleotide catabolism. It accounts for 60% of the plasma antioxidant capacity and is detected at high concentrations in epithelial fluids of the respiratory tract. It is also a proinflammatory factor; tissue hypoxia, chronic inflammation, and oxidative stress seen in COPD lead to elevations in uric acid levels. There are studies indicating a relationship between uric acid elevation and mortality in COPD. Bartziokas et al. study on 314 patients with acute COPD attacks identified decreased uric acid as an independent predictor of 30-day mortality and associated it with acute exacerbation and hospitalization over a one-year follow-up period. NIMV and intensive care need were also high during the 30-day monitoring period. This was attributed to increased xanthine oxidase

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activity caused by hypoxemia, cardiovascular comorbidity, and damage caused by tissue hypoxia in the lungs and other tissues by increasing circulating levels of uric acid. Vafaei et al. found a significant relationship between high uric acid levels and FEV_1, oxygen saturation, $PaCO_2$, body mass index, echocardiographic changes, and COPD severity. In our study, we did not find the significant differences between the two groups. The reason may be that we did not follow up the patients and the morality was not documented [16-18].

CONCLUSION: Electrolyte imbalances can cause respiratory muscle weakness and impair airway function in COPD patients. Serum sodium and potassium levels were lower in acute exacerbations of COPD who deceased during the admission, and there were no statistically significant differences in uric acid levels between the two groups. The levels of these parameters should be measured and corrected during AECOPD treatment to decrease mortality.

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