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Original Research Article.

Evaluation of Serum Electrolytes in Patients of Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a chronic disease which involves the airways, lung parenchyma, and pulmonary vasculature. It has considerable systemic manifestations. The disease is one of the most important causes of death worldwide. The Global Burden of Disease Study projected it to become the third leading cause of death globally by the year 2020.

Aim: The present study was planned to evaluate electrolyte disturbance in patient with acute exacerbations compared to stable COPD patients.

Materials & Methods: Total 100 COPD cases 50 Acute Exacerbations COPD patients and 50 Stable COPD Patients were enrolled for the study. Pregnant females, patients with pneumonia, Patients with neoplastic pathologies and patients with hepatic diseases and renal diseases were excluded from study. The results obtained were presented as mean ± SD. All parameters analyzed were compared between acute exacerbation and stable COPD by applying student t-test. Further chi-square was also applied to evaluate the effect of dyselectrolytemia on prevalence of acute exacerbation.

Results: Mean serum sodium, serum potassium and serum chloride levels were significantly lower in acute exacerbation COPD Patients as compared to Stable COPD Patients.

Conclusion: The study demonstrated decreased serum sodium, potassium and chloride level in patient with acute exacerbation. Serum electrolytes level should be monitored routinely in these patients and an attempt should be made to correct them at the earliest to avoid poor outcomes. Serum magnesium value of acute exacerbation group was lower than that of the stable cases.

Keywords: Chronic Obstructive Pulmonary Disease, Dyselectrolytemia, Acute Exacerbations, Hypomagnesemia.

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a chronic disease which involves the airways, lung parenchyma, and pulmonary vasculature. It has considerable systemic manifestations. The disease is progressive with possible gene environment interaction and hence can be prevented by avoiding exposure to the noxious particles. The most common studied attributing risk factor is cigarette smoking in any form. Acute Exacerbations and comorbidities play important role in contributing to overall severity.[1] The disease is one of the most important causes of death worldwide. "The Global Burden of Disease Study projected it to become the third leading cause of death globally by the year 2020".[2]

COPD is a leading cause of morbidity and mortality worldwide. With increasing industrialization and smoking, the prevalence of COPD is increasing. Exacerbations are the most common cause of hospitalization among COPD patients.[3] Acute Exacerbations of chronic obstructive pulmonary disease (COPD) are defined as acute events characterized by a worsening of the patient's respiratory symptoms, particularly dyspnea, beyond day-to-day variation, leading to a change in medical treatment and/or hospitalization.[4,5] In patients with COPD, edema is almost invariably associated with gas exchange impairment and in particular with carbon dioxide (CO_2) retention. The solute water retention in COPD has been considered to be the result of electrochemical imbalance (enhanced renal/tubular H+/Na+ exchange) and/or renal hemodynamic abnormalities.[6] Acute Exacerbations patients with COPD are unable to normally excrete a water load and this defect that can be confirmed in the absence of overt edema, is associated with a decrease in sodium excretion and is found be proportional to the severity of CO_2 retention.[3]

Hyponatremia is the most common electrolyte disorder seen in hospitalized patients and may contribute substantially to morbidity and mortality. Patients with chronic obstructive pulmonary disease (COPD) are susceptible to hyponatremia for a number of reasons. An exacerbation is an acute event in patients of COPD with increased respiratory symptoms requiring additional treatment other than the usual ongoing treatment.[1]

Chronic hypoxia and hypercapnia secondary to the underlying pulmonary illness, heart failure or renal insufficiency, use of diuretics, SIADH, hypokalemia attributed to bronchodilators or steroids, malnutrition, and poor intake during acute exacerbations are common contributing factors for hyponatremia in such patients.[7]

Hypomagnesemia is associated with increased airway hyperactivity and impaired pulmonary function. It is hypothesized that due to its bronchodilating effect, a decreased level of magnesium may increase COPD exacerbations. When magnesium is deficient, the action of calcium is enhanced. This is important as the intracellular influx of calcium causes bronchial smooth muscle contraction. Although serum levels are used to assess magnesium deficiency, cells can be deficient despite normal serum values.[8]

Several studies have compared the electrolyte levels in COPD patients with those of healthy control. The present study was planned to evaluate electrolyte disturbance in patient with acute exacerbations compared to stable COPD patient.

MATERIALS & METHODS

The study was conducted at Department of Respiratory Medicine of Mahatma Gandhi Medical College & Hospital, Jaipur.

Total 100 COPD cases 50 Acute Exacerbations COPD patients and 50 Stable COPD Patients were enrolled for the study. Institutional Ethics Committee approval and informed consent was obtained from all participants before enrollment in the study.

Inclusion Criteria

- Diagnosed case of Chronic Obstructive Pulmonary Disease
- Age between 20 to 60 years.
- Patients who are willing to participate in the study.

Exclusion Criteria

- Pregnant females.
- Patients with pneumonia.
- Patients with neoplastic pathologies.
- Patients with hepatic diseases and renal diseases.

Blood samples for all subjects (acute exacerbation and stable COPD patients) were collected using standard aseptic technique and analysis for Serum Sodium, Serum Potassium, Serum Chloride using direct ion selective electrode and Serum Magnesium, Serum Calcium using arsenazo dye and formazan dye method on Vitros 5600. The results obtained were presented as mean ± SD. All parameters analyzed were compared between acute exacerbation and stable COPD by applying student t-test. Further chi-square was also applied to evaluate the effect of dyselectrolytemia on prevalence of acute exacerbation.

Table 1: Distribution of Variables between Acute Exacerbation COPD Patients and Stable COPD Patients

Parameters	Stable COPD (n=50)	Acute COPD (n=50)	t-value	P-value
Sodium (mmol/L)	136.84±4.53	132.04±9.13	3.33	0.001
Potassium (mmol/L)	3.93±0.49	3.50±0.86	3.07	0.003
Chloride (mmol/L)	99.42±6.26	93.6±10.52	3.36	0.001
Calcium (mg/dl)	8.32±1.81	7.61±1.58	2.09	0.039
Magnesium (mmol/L)	1.68±0.28	1.12±0.31	9.48	0.000

Table 2: Percentage Distribution in Acute Exacerbation COPD Patients and Stable COPD Patients on the basis of Parameters

Parameters		Acute (n=50) (%)	Stable (n=50) (%)	X^2	P-value
Sodium	**≤ 135.0**	31 (62)	21 (42)	7.232	0.007
	>135.0	19 (38)	29 (58)		
Potassium	**≤ 3.5**	26 (52)	10 (20)	20.855	0.000
	> 3.5	24 (48)	40 (80)		
Chloride	**≤ 98.0**	29 (58)	18 (36)	8.852	0.003
	> 98.0	21 (42)	32 (64)		
Calcium	**≤ 8.7**	40 (80)	34 (68)	3.144	0.076
	> 8.7	10 (20)	16 (32)		
Magnesium	**≤ 1.30**	36(72)	6(12)	71.449	0.000
	> 1.30	14(28)	44(88)		

RESULTS

Parameters evaluated in the stable COPD and acute exacerbations patients are presented in (Table 1). The mean serum sodium level of acute exacerbations patients was significantly lower 132.04 ± 9.13 mmol/L (p 0.001). Mean potassium level were low in acute exacerbations patients (3.50±0.86) as compared to stable COPD patients (3.93±0.49).

Serum chloride levels were significantly lower (93.6±10.52) in acute exacerbations patients as compared to stable COPD patients (99.42±6.26). The mean serum calcium level was found not significant in acute exacerbations patients (7.61±1.58) as compared to stable COPD patients (8.32±1.81). Mean serum magnesium levels were significantly lower (1.12±0.31) in acute

exacerbations patients as compared to stable COPD patients (1.68±0.28). Table 2 demonstrates Percentage Distribution in Acute Exacerbation COPD Patients and Stable COPD Patients on the basis of Parameters and applied chi X^2 test. Serum sodium levels ≤135.0 mmol/L was reported in (62%) patients of acute exacerbation and (42%) of stable COPD patient, (38%) of acute exacerbation and (58%) of stable patients has Serum sodium levels >135.0 mmol/L. A significant p value 0.007 was observed on apply chi square.

Serum potassium levels ≤3.5 mmol/L was reported in (52%) patient of acute exacerbation and (20%) of stable COPD patients. (48%) of acute exacerbation and (80%) stable COPD patients has Serum potassium levels >3.5 mmol/L. On apply chi square a significant p value 0.000 was observed.

Serum chloride levels ≤ 98.0 mmol/L was reported in (58%) patient of acute exacerbation and (36%) of stable COPD patients. (42%) of acute exacerbation and (64 %) of stable COPD patients has Serum chloride levels >98.0 mmol/L. A significant p value 0.003 was observed on apply chi square. Serum calcium levels ≤8.7 mg/dL was reported in (80%) patient of acute exacerbation and (68%) stable COPD patients. (20%) of acute exacerbation and (32%) stable COPD patients have Serum calcium levels >8.7mg/dL. On apply chi square a non-significant p value 0.076.

Serum magnesium levels ≤1.30 mmol/L was reported in (72%) patient of acute exacerbation and (12%) of stable COPD patients. (28%) of acute exacerbation and (88%) stable COPD patients have Serum magnesium levels >1.30 mmol/L. On apply chi square a significant p value 0.000 was observed.

DISCUSSION

Mean serum sodium, serum potassium and serum chloride levels were significantly lower in acute exacerbation COPD Patients as compared to Stable COPD Patients. Our findings are in accordance with previous study Das et al 2010 reported average levels of serum sodium and potassium in acute exacerbation COPD patients were 133 ± 6.86 and 3.39 ± 0.96m Eq/L, serum sodium and potassium levels in the control group were 142 ± 2.28 and 4.52 ± 0.02 mEq/L.[9]

Thus, COPD patients are predisposed to electrolyte imbalance. In turn electrolyte, respiratory role can cause respiratory muscle weakness, cardiac arrhythmia, low cardiac output, etc.[10]

Irrespective of the underlying mechanism of development, hyponatremia itself may be a predictor of poor outcome in patients of COPD. It may lead to central nervous system dysfunction; confusion, convulsions, coma, reversible cardiac conduction defect, secondary renal insufficiency and even death (Suri et al 2009; Porcel et al, 2002).[11,12]

Hypokalemia may be another electrolyte abnormality in the subjects with COPD. It may be present independently or concomitantly with hyponatremia. In the present study there was a significantly low level of serum potassium in acute exacerbation patients than the stable patients. Hypokalemia in COPD may be attributed to respiratory acidosis and metabolic alkalosis or long-standing steroid therapy (Saini et al2008).[13]

In a similar study by the prevalence of electrolyte disturbance in patients with asthma was common and reached up to 68% in chronic stable asthma and a higher percentage in acute severe asthma (98%).[8] S.B. Omar et al also reported Electrolyte disturbance is in 43% of patients with chronic stable asthma.[14]

Serum calcium level among acute cases was 7.61±1.58 mg/dl and among stable were 8.32±1.81 mg/dl. There was a statistically Non significant difference in mean serum calcium levels among acute and stable (p value 0.039). On apply chi square a non-significant p value = 0.076 (non-significant) was observed this finding indicates that serum calcium does not have a direct influence the occurrence of acute exacerbation condition.

The mean serum magnesium value in the stable group was 1.68 mg/dl whereas in the group comprising of acute exacerbations it was 1.12 mg/dl. This showed significant correlation between hypomagnesaemia and COPD exacerbation.

There is growing concern about serum magnesium levels in pulmonary diseases. Magnesium disturbance is a well-known abnormality seen in patients with pulmonary disease.[15,16]

A higher incidence of asthma severity and exacerbation with low serum magnesium levels than in asthmatic patients with normal levels was found.

Hypomagnesemia was more significantly frequent ($p< 0.05$) in moderate and severe chronic asthmatic patients. Also, a statistically significant decrease in serum magnesium level was found in patients with acute attacks in comparison with chronic stable asthma. This is in agreement with the study by Chaiwat and Poonkasem.[17]

The potential mechanism for the direct relaxing effects of magnesium on bronchial smooth muscles include calcium channel blocking properties, inhibition of cholinergic neuro-Muscular Junction transmission with decreased sensibility to the depolarising action of acetylcholine, stabilization of mast cells and T lymphocytes and stimulation of nitric oxide and Prostacycline.[18]

Low dietary magnesium is also found to be associated with wheezes and impairment of lung function in normal subjects.[19]

A few studies showed results similar to this study. Teranzo et al. (2012)[20], reported that the prolonged duration of ventilation was associated with lower serum sodium ($p = 0.014$) and lower chloride ($p = 0.038$). Also, Acute respiratory failure associated with hypokalemia was found to have a high mortality rate among the COPD patients.

A few studies showed results similar to this study. Faris M. Ouf et al. (2015)[21], group of COPD patients, 17 patients (28%) presented with electrolyte disorders on the first day of admission, hypokalemia was reported in 4 (6.6%) patients, hyponatremia in 3 (5%) patients, hypocalcaemia in 3 (5%) patients, hypomagnesmia in 3 (5%) patients, hypochloremia in 2 (3.3%) patients and combined electrolyte disorders were present in 2 (3.3%) patients.

CONCLUSION

Findings of the present study suggest that in the acute exacerbation patients there are abnormal serum electrolyte levels especially for serum sodium, potassium, chloride and magnesium.

The study demonstrated decreased serum sodium, potassium and chloride level in patient with acute exacerbation. Serum electrolytes level should be monitored routinely in these patients and an attempt should be made to correct them at the earliest to avoid poor outcomes. Serum magnesium value of acute exacerbation group was lower than that of the stable cases.

The prevalence of hypomagnesaemia in acute exacerbation of COPD is high. Low serum magnesium may predict acute exacerbation of COPD and can be recognized as a risk factor for acute exacerbation COPD.

REFERENCES

1. From the Global Strategy for the Diagnosis, Management and Prevention of COPD, Global Initiative for Chronic Obstructive Lung Disease, 2017.
2. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Med 2006;3:442.
3. White RJ and Woodings DF. Impaired water handling in chronic obstructive airway disease. BMJ 1971:561-3.
4. GOLD. Global Strategy for the Diagnosis, Management, and Prevention of COPD, 2011.
5. Soriano JB, Brusasco V, Dinh-Xuan AT. The European Respiratory Journal makes COPD a priority. Eur Respir J 2011; 38: 999–1001.
6. Reineck HJ, Stein JH. Sodium metabolism. In: Maxwell MH, Kleeman CR, Narins RG, eds. Clinical disorders of fluid and electrolyte metabolism. New York: Mc- Graw-Hill, 1987: 39-40.
7. Bauer FK, Telfer N, Herbst HH, et al: Hyponatremia and increased exchangeable sodium in chronic obstructive lung disease. Am J Med Sci 1965; 250: 245-53.
8. Landon RA, Young EA Role of magnesium in regulation of lung function. Journal of the American Dietetic Association. 1993: 674-77.
9. Weitzenblum E, Apprill M, Oswald M, Chaouat A, Imbs JL. Pulmonary hemodynamics in patients with chronic obstructive pulmonary disease before and during an episode of peripheral edema. Chest 1994; 105: 1377-82.
10. Yang CT, Lin HC, Lin MC, Wang CH, Lee CH, Kuo HP. Effect of beta 2-adrenoceptor agonists on plasma potassium and cardiopulmonary responses on exercise in patients with chronic obstructive pulmonary disease. Eur J Clin Pharmacol. 1996;341-6.
11. Suri P, Habeeb K, Alai MS, Rather HA, Jalal S Hyponatremia Presenting as Cardiac Conduction Defect. J K Science. 2009;11(2):85-6.
12. Porcel A, Díaz F, Rendón P. Dilutional hyponatremia in patients with cirrhosis and ascites. Arch Intern Med. 2002; 162:323-8.
13. Saini V, Saini N, Kaur J, Singh GP. Acid base status in chronic obstructive pulmonary disease patient. Ind J Clin Biochem. 2008;28: 36-8.
14. S.B. Omer, Electrolyte disturbances in patients with chronic, stable asthma, Chest 120.2001 ;431–6.
15. De Valk H, Kok P, Struyvenberg A, Van Rijn H, Kreukniet J, Lammers J. Extracellular and intracellular magnesium concentrations in asthmatic patients. European Respiratory Journal. 1993;6(8):1122-5.
16. Knutsen R, Bøhmer T, Falch J. Intravenous theophylline induced excretion of calcium, magnesium and sodium in patients with recurrent asthmatic attacks. Scandinavian journal of clinical and laboratory investigation. 1994;54(2):119-25.
17. B. Chaiwat, C. Poonkasem. Serum magnesium levels in acute severe asthma, Chiang Mai. Med. Bull.2001;1–5.
18. Williams MV, Huddleston J, Whitford K, DiFrancesco L, Wilson M: Advances in hospital medicine: a review of key articles from the literature. Medical Clinics of North America. 2002;86(4):797-823.
19. Britton J, Pavord I, Richards K, et al. Dietary magnesium, lung function, wheezing, and airway hyper-reactivity in a random adult population sample. Lancet 1994; 344:357–62.
20. Terzano C, Di Stefano F, Conti V, Di Nicola M, Paone G, Petroianni A, Ricci A. Mixed acid-base disorders, hydroelectrolyte imbalance and lactate production in hypercapnic respiratory failure: the role of noninvasive ventilation. PLoS One.2012; 7(4):e35245. doi: 10.1371/ journal.pone. 0035245.
21. Faris M. Ouf, Abdel Aziz A F, Wahba A H, Kabil A E, Alhusseiny M A. Electrolyte disturbances and their impact on mechanically ventilated patients with acute exacerbation of chronic obstructive pulmonary disease. AAMJ 2015 Oct; 13(4): 27-34.

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