

ORIGINAL RESEARCH

Assessment of serum electrolytes, acid–base balance and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease (AECOPD)

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ABSTRACT

Background: When breathing is compromised and the amount of carbon dioxide exhaled by the respiratory system is less than the amount of carbon dioxide produced by the tissues, hypercapnia and respiratory acidosis result. The present study was conducted to assess serum electrolytes, acid–base balance and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease (AECOPD). **Methods:** 82 patients with (AECOPD) of both genders were selected. Blood investigations such as arterial blood gases (ABG), electrolyte levels (Sodium (Na) and potassium (K)) were performed. NIV was performed through oronasal mask with a pressure/ volume ventilator. Support pressure, positive end expiratory pressure (PEEP) was used. **Results:** Out of 50 cases of compensated respiratory acidosis, 10 were NIV positive and 40 were NIV negative. Out of 18 cases with mixed respiratory acidosis and metabolic alkalosis, 15 were NIV positive and 3 were NIV negative. Out of 14 cases with combined respiratory and metabolic acidosis, 12 were NIV positive and 2 were NIV negative. The difference was significant ($P < 0.05$). The mean sodium level was 135.2 mEq/l and 137.4 mEq/l, potassium level was 4.9 mEq/l and 3.2 mEq/l and bicarbonate level was 29.5 mEq/l and 23.7 mEq/l in NIV positive and NIV negative patients. The difference was significant ($P < 0.05$). Improvement was seen in 17 cases of compensated respiratory acidosis, 11 cases of mixed respiratory acidosis and metabolic alkalosis and 5 cases of combined respiratory and metabolic acidosis. The difference was significant ($P < 0.05$). **Conclusion:** While minor AECOPD episodes are usually curable, more severe respiratory failures are linked to a significant death rate and a protracted period of impairment for those who survive.

Keywords: chronic obstructive pulmonary disease, metabolic acidosis, positive end expiratory pressure

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INTRODUCTION

When breathing is compromised and the amount of carbon dioxide exhaled by the respiratory system is less than the amount of carbon dioxide produced by the tissues, hypercapnia and respiratory acidosis result.¹ Alveolar hypoventilation is not usually the outcome of lung illnesses that lead to anomalies in alveolar gas exchange. These illnesses frequently cause hypoxia and reflex receptors, which in turn cause ventilation and hypocapnia. Hypercapnia usually happens when respiratory muscles tire or when the condition progresses to a severe degree.² (Metabolic acidosis, Pediatric Metabolic Acidosis, and Pediatric Respiratory Acidosis). Acute or chronic

respiratory acidosis can occur. When there is acute respiratory acidosis, there is an acidemia (pH less than 7.35) and an increased PaCO₂ over the upper limit of the reference range (i.e., >45 mm Hg).³

Chronic respiratory acidosis is characterized by excessive serum bicarbonate levels (>30 mEq/L), normal or almost normal pH due to renal compensation, and elevated PaCO₂ levels over the upper limit of the reference range. An rapid lack of breathing results in acute respiratory acidosis.⁴ The central respiratory center may be depressed for any of the following reasons, leading to this ventilation failure: Diseases of the central nervous system or medication-induced respiratory depression.⁵

incapacity to breathe enough as a result of paralysis or a neuromuscular illness (such as muscular dystrophy, ALS, myasthenia gravis, or Guillain-Barré syndrome). airway blockage, typically associated with asthma or COPD (chronic obstructive pulmonary disease). Many illnesses, including COPD, may have chronic respiratory acidosis as a secondary condition. The present study was conducted to assess serum electrolytes, acid–base balance and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease (AECOPD).

MATERIALS & METHODS

The study was carried out on 82 patients with (AECOPD) of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Blood investigations such as arterial blood gases (ABG), electrolyte levels (Sodium (Na) and potassium (K)) were performed. NIV was performed through oronasal mask with a pressure/ volume ventilator. Support pressure, positive end expiratory pressure (PEEP) was used. Results thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Assessment of arterial blood gases

Acid–base disturbance	Need for non-invasive ventilation		P value
	NIV positive (37)	NIV negative (45)	
Compensated respiratory acidosis(50)	10	40	0.05
Mixed respiratory acidosis and metabolic alkalosis (18)	15	3	
Combined respiratory and metabolic acidosis (14)	12	2	
PO ₂	55.1	64.3	0.04
PCO ₂	64.7	48.2	0.05

Table I shows that out of 50 cases of compensated respiratory acidosis, 10 were NIV positive and 40 were NIV negative. Out of 18 cases with mixed respiratory acidosis and metabolic alkalosis, 15 were NIV positive and 3 were NIV negative. Out of 14 cases with combined respiratory and metabolic acidosis, 12 were NIV positive and 2 were NIV negative. The difference was significant (P< 0.05).

Table II Assessment of electrolytes finding

Electrolytes	Need for non-invasive ventilation		P value
	NIV positive (37)	NIV negative (45)	
Sodium (mEq/l)	135.2	137.4	0.75
Potassium (mEq/l)	4.9	3.2	0.04
Bicarbonate (mEq/l)	29.5	23.7	0.05

Table II, graph I shows that mean sodium level was 135.2mEq/l and 137.4mEq/l, potassium level was 4.9mEq/l and 3.2mEq/l and bicarbonate level was 29.5mEq/l and 23.7mEq/l in NIV positive and NIV negative patients. The difference was significant (P< 0.05).

Graph I Assessment of electrolytes finding

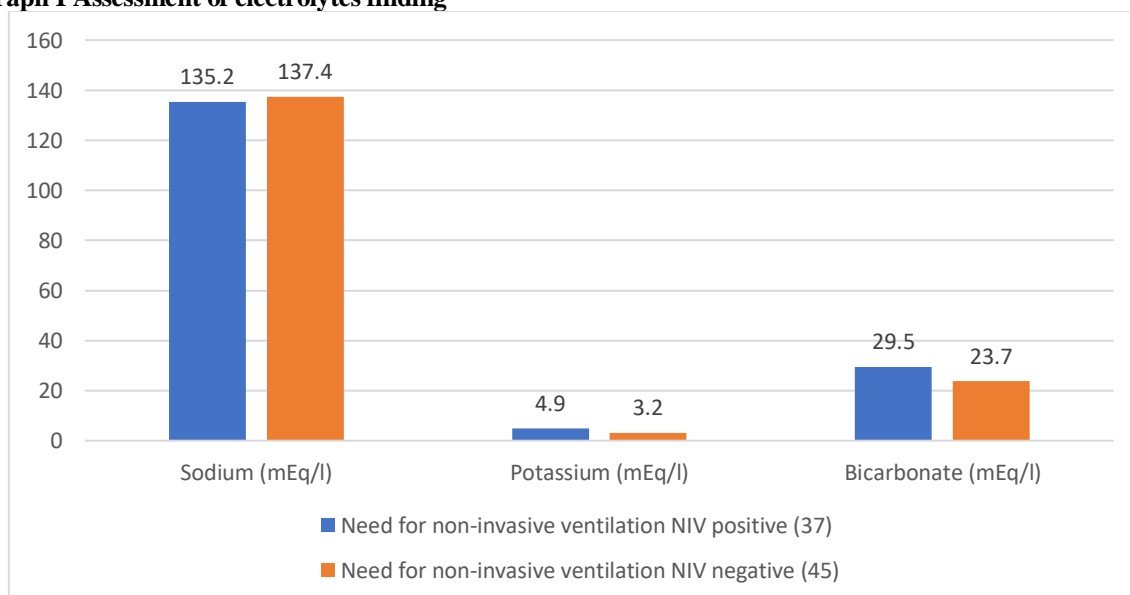


Table III Outcome

Acid–base disturbance	Improved (33)	Failed (4)	P value
Compensated respiratory acidosis	17	2	0.05
Mixed respiratory acidosis and metabolic alkalosis	11	1	
Combined respiratory and metabolic acidosis	5	1	

Table III shows that improvement was seen in 17 cases of compensated respiratory acidosis, 11 cases of mixed respiratory acidosis and metabolic alkalosis and 5 cases of combined respiratory and metabolic acidosis. The difference was significant ($P < 0.05$).

DISCUSSION

Acid/base (AB) balance abnormalities are linked to multi-organ impairment in hypercapnic respiratory failure, a complex clinical and functional disease.⁷ The kidney, respiratory system, red blood cells, blood proteins, and bicarbonate buffering system are among the physiological systems that regulate the acid-base balance.⁸ The hydro-electrolytic (HE) balance and the AB balance are intimately associated. When CO₂ levels rise (respiratory acidaemia), a metabolic alkalosis counteracts the increase.⁹ This is mostly accomplished by a complex excretion system involving ions in the urine. Therefore, knowledge of the intricate regulatory processes governing AB diseases is essential for their management.^{10,11} The present study was conducted to assess serum electrolytes, acid–base balance and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease (AECOPD).

We found that out of 50 cases of compensated respiratory acidosis, 10 were NIV positive and 40 were NIV negative. Out of 18 cases with mixed respiratory acidosis and metabolic alkalosis, 15 were NIV positive and 3 were NIV negative. Out of 14 cases with combined respiratory and metabolic acidosis, 12 were NIV positive and 2 were NIV negative. Schiavo et al¹² evaluated acid–base and hydroelectrolyte alterations in these subjects and the effect of non-invasive ventilation and pharmacological treatment. They retrospectively analysed 110 patients consecutively for acute exacerbation of hypercapnic chronic obstructive pulmonary disease. On admission all patients received oxygen with a Venturi mask to maintain arterial oxygen saturation at least $>90\%$, and received appropriate pharmacological treatment. Non-Invasive Ventilation (NIV) was started when, despite optimal therapy, patients had severe dyspnea, increased work of breathing and respiratory acidosis. Based on Arterial Blood Gas (ABG) data, we divided the 110 patients in 3 groups: A = 51 patients with compensated respiratory acidosis; B = 36 patients with respiratory acidosis + metabolic alkalosis; and C = 23 patients with respiratory acidosis + metabolic acidosis. 55 patients received only conventional therapy and 55 had conventional therapy plus NIV. The use of NIV support was lower in the patients belonging to group B than in those belonging to group A and C (25 %, vs 47 % and 96 % respectively; $p < 0.01$). A statistically significant association was found between pCO₂ values and serum

chloride concentrations both in the entire cohort and in the three separate groups.

We observed that the mean sodium level was 135.2 mEq/l and 137.4 mEq/l, potassium level was 4.9 mEq/l and 3.2 mEq/l and bicarbonate level was 29.5 mEq/l and 23.7 mEq/l in NIV positive and NIV negative patients. Improvement was seen in 17 cases of compensated respiratory acidosis, 11 cases of mixed respiratory acidosis and metabolic alkalosis and 5 cases of combined respiratory and metabolic acidosis. Krishna et al¹³ analyzed 74 patients for hypercapnic AECOPD. Based on arterial blood gas finding, we categorized our patients into three main groups: the first group comprised 44 (59.4%) patients who had compensated respiratory acidosis, and the majority of them (35 patients) received medical treatment only. The second group comprised 17 (22.97%) patients, who had mixed respiratory acidosis and metabolic alkalosis. Overall, 13 patients needed non-invasive mechanical ventilation with the medical treatment. The third group comprised 13 (17.5%) who had combined respiratory and metabolic acidosis. Of them, 11 patients needed non-invasive mechanical ventilation with the medical treatment.

The shortcoming of the study is small sample size.

CONCLUSION

Authors found that while minor AECOPD episodes are usually curable, more severe respiratory failures are linked to a significant death rate and a protracted period of impairment for those who survive.

REFERENCES

1. Kramer N, Meyer TJ, Meharg J, Cece RD, Hill NS. Randomized, prospective trial of non-invasive positive pressure ventilation in acute respiratory failure. *Am J Respir Crit Care Med.* 1995;151:1799–806.
2. Confalonieri M, Garuti G, Cattaruzza MS, Osborn JF, Antonelli M, et al. Italian non-invasive positive pressure ventilation (NPPV) study group. A chart of failure risk for non-invasive ventilation in patients with COPD exacerbation. *Eur Respir J.* 2005;25:348–55.
3. Plant PK, Owen JL, Elliott MW. Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. *Lancet.* 2000;355:1931–5.
4. Ucgun I, Oztuna F, Dagli CE, Yildirim H, Bal C. Relationship of metabolic alkalosis, azotemia and morbidity in patients with chronic obstructive pulmonary disease and hypercapnia. *Respiration.* 2008;76:270–4.

5. Terzano C, Di Stefano F, Conti V, Di Nicola M, Paone G, et al. Mixed acid–base disorders, hydroelectrolyte imbalance and lactate production in hypercapnic respiratory failure: the role of non-invasive ventilation. *PLoS ONE*. 2012;7:e35245.
6. Brijker F, Heijdra YF, van den Elshout FJ, Folgering HT. Discontinuation of furosemide decreases PaCO₂ in patients with COPD. *Chest*. 2002;121:377–82.
7. Brackett Jr NC, Cohen JJ, Schwartz WB. Carbon dioxide titration curve of normal man. Effect of increasing degrees of acute hypercapnia on acid–base equilibrium. *N Engl J Med*. 1965;272:6–12.
8. Mizock BA. The epatosplanchnic area and hyperlactatemia. A tale of two lactates. *Crit Care Med*. 2001;29:447–9.
9. Schiraldi F, Guiotto G. Equilibrioacido-base. *Ossigeno. Fluidi&elettroliti*. 2012. pp 45–53.
10. Brackett NC, Wingo CF, Muren O, Solano JT. Acid–base response to chronic hypercapnia in man. *N Engl J Med*. 1969;280:124–30.
11. Alfaro V, Torras R, Ibáñez J, Palacios L. A physical-chemical analysis of the acid–base response to chronic obstructive pulmonary disease. *Can J PhysiolPharmacol*. 1996;74:1229–35
12. Schiavo A, Renis M, Polverino M, Iannuzzi A, Polverino F. Acid–base balance, serum electrolytes and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease admitted to an internal medicine ward. *Multidisciplinary Respiratory Medicine*. 2016 Dec;11:1-6.
13. Krishna, V Venkateswara Rao, Syed Umer Ahmed, B. Vishwanath. Serum electrolytes, Acid–base balance and need for non-invasive ventilation in patients with hypercapnic acute exacerbation of chronic obstructive pulmonary disease. *International Journal of Health and Clinical Research*, 2021;4(2):186-189.