

Arterial Blood Gas and Routine Laboratory Parameters as Predictors of In-Hospital Outcome in Acute Exacerbation of Chronic Obstructive Pulmonary Disease: A Prospective Observational Study from a Tertiary Care Centre in Eastern Uttar Pradesh, India

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Abstract

Background: Acute exacerbation of chronic obstructive pulmonary disease is a leading cause of emergency medical admission in India. We examined whether arterial blood gas and routine laboratory parameters, at admission and after 24 hours of bilevel positive airway pressure ventilation, predict in-hospital outcomes.

Methods: A prospective observational study enrolled 100 consecutive patients admitted with acute exacerbation of chronic obstructive pulmonary disease to

a tertiary teaching hospital in Gorakhpur, India, over one year. Arterial carbon dioxide and oxygen tensions, total leukocyte count, serum sodium, serum potassium and alanine aminotransferase were recorded. Outcomes were Glasgow Coma Scale score, duration of bilevel ventilation, intubation, intensive care and hospital stay, and in-hospital mortality.

Results: Eighty patients had an arterial carbon dioxide tension above 70 mmHg at admission. Compared with patients at or below this level, they more often had a

Glasgow Coma Scale score of 8 or less (32.5% versus 0%, $p=0.001$), needed bilevel ventilation beyond two days (51.3% versus 15.0%, $p=0.005$) and required intubation (38.8% versus 0%, $p<0.001$). All 14 deaths occurred in this group, although the mortality difference did not reach significance (17.5% versus 0%, $p=0.07$). An arterial oxygen tension below 40 mmHg at admission was associated with 33.3% mortality versus 1.6% ($p<0.001$), and its persistence after 24 hours of bilevel ventilation with 66.7% versus 2.4% ($p<0.001$). Persistent hypercapnia after 24 hours was associated with higher mortality (21.7% versus 2.5%, $p=0.007$). Hyponatremia was associated with longer intensive care stay (3.66 versus 2.54 days, $p<0.001$). Leukocyte count, alanine aminotransferase, sodium and potassium were not associated with mortality.

Conclusion: Severe hypoxemia at admission and failure of blood gases to improve after 24 hours of bilevel ventilation were the strongest markers of in-hospital death, while severe hypercapnia identified patients needing prolonged or invasive ventilatory support. Routine laboratory parameters added little prognostic information in this cohort.

Keywords: Chronic Obstructive Pulmonary Disease, Acute Exacerbation, Arterial Blood Gas Analysis, Hypercapnia, Hypoxemia, Noninvasive Ventilation, Hospital Mortality, Hyponatremia, India

Introduction

Chronic obstructive pulmonary disease (COPD) is one of the leading causes of death and disability worldwide, and its burden falls disproportionately on low- and middle-income countries¹. In India, the pooled prevalence of COPD among adults above 30 years is about 7%, and a large share of the disease is driven by non-tobacco exposures such as biomass fuel smoke,

occupational dust and previous pulmonary tuberculosis². Acute exacerbations of COPD (AECOPD), defined as an event of worsening dyspnea and/or cough and sputum over less than 14 days, are the main reason these patients present to hospital^{3,4}. Admission for an exacerbation carries substantial in-hospital mortality and cost, and outcome varies widely between patients^{5,6}.

Arterial blood gas (ABG) analysis is central to the assessment of AECOPD. Ventilation-perfusion mismatch and ventilatory pump failure produce hypoxemia and hypercapnia⁷, and the ABG identifies patients with acute hypercapnic respiratory failure who benefit from bilevel positive airway pressure (BiPAP), a form of noninvasive ventilation (NIV) that reduces the need for intubation and improves survival⁸⁻¹⁰. Blood gas variables also feature in prognostic tools; for example, acidemia is a component of the DECAF score for in-hospital mortality¹¹. Routine laboratory tests, including leukocyte count, serum electrolytes and markers of end-organ dysfunction, are widely available and have been proposed as additional prognostic markers¹².

Most outcome data on AECOPD come from high-income settings, where the patient population, the causes of COPD and access to intensive care differ from those in North India. Data from eastern Uttar Pradesh, where biomass exposure and post-tubercular lung disease are common, are scarce. The objective of this study was to assess the association of ABG parameters (arterial partial pressures of carbon dioxide [PaCO_2] and oxygen [PaO_2]) at admission and after 24 hours of BiPAP, and of routine laboratory parameters (total leukocyte count [TLC], alanine aminotransferase [ALT], serum sodium and potassium), with Glasgow Coma Scale (GCS) score, duration of BiPAP, endotracheal intubation, length of

intensive care unit (ICU) and hospital stay, and in-hospital mortality in patients admitted with AECOPD.

Materials and Methods

Study design and setting

This prospective observational study was conducted in the Department of General Medicine, Baba Raghav Das Medical College, Gorakhpur, Uttar Pradesh, over one year. The hospital is a tertiary referral centre serving eastern Uttar Pradesh, western Bihar and adjoining districts.

Participants

Consecutive patients aged 40 years or above admitted with AECOPD were screened. COPD was diagnosed clinically and confirmed by spirometry (post-bronchodilator FEV₁/FVC below 0.70) performed after recovery from the exacerbation, once patients had returned to their baseline modified Medical Research Council dyspnea grade; severity was graded according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria³. Patients with household biomass fuel exposure of at least one hour daily for at least one year were eligible. Patients were excluded if they declined consent, had another primary respiratory diagnosis (bronchial asthma, primary interstitial lung disease, or pneumonia without underlying COPD), had a severe concurrent systemic illness unrelated to AECOPD likely to influence outcome independently, or were transferred or left against medical advice before data collection was complete.

The sample size was estimated with the formula $n = Z^2P(1 - P)/d^2$, using an expected COPD prevalence of 7% [2], a Z value of 1.96 and an absolute precision of 5%, which gave 100 patients. A pilot study of 10 patients was used to refine the case record form; these patients were not included in the analysis.

Data collection

A single trained investigator recorded sociodemographic data, smoking history, biomass and occupational exposures, comorbidities, previous exacerbations, presenting complaints, clinical examination findings and GCS score at presentation on a pre-tested case record form. Radial arterial samples for ABG analysis were drawn within the first 24 hours of admission and repeated after 24 hours of BiPAP, and analysed on a calibrated blood gas analyser in the hospital laboratory. Venous samples drawn within 24 hours of admission were analysed for TLC, serum sodium, serum potassium and ALT in the same laboratory. Management, including controlled oxygen, bronchodilators, systemic corticosteroids, antibiotics, BiPAP and the decision to intubate, was determined by the treating team according to standard practice; the study did not alter management.

Variable definitions and outcomes

Admission PaCO₂ was grouped as below 50, 50–70 and above 70 mmHg; for the main comparison, patients above 70 mmHg were compared with those at or below 70 mmHg. PaCO₂ after 24 hours of BiPAP was grouped as hypocapnia (below 35 mmHg), normocapnia (35–45 mmHg) and hypercapnia (above 45 mmHg). Severe hypoxemia was defined as PaO₂ below 40 mmHg. Sodium was classified as hyponatremia (below 135 mEq/L), normal (135–145 mEq/L) or hypernatremia (above 145 mEq/L); potassium as hypokalemia (below 3.5 mEq/L), normal (3.5–5.0 mEq/L) or hyperkalemia (above 5.0 mEq/L); ALT as normal (40 U/L or below) or raised (above 40 U/L); and TLC as below 4000, 4000–11000 or above 11000 cells/mm³. Outcomes were GCS score at presentation (3–8, 9–12, 13–15), duration of BiPAP (2 days or less versus more than 2 days),

endotracheal intubation, length of ICU and hospital stay, and in-hospital mortality.

Statistical analysis

Data were entered and summarised in SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Categorical variables are presented as numbers and percentages and continuous variables as mean $\pm$ standard deviation (SD). Associations between categorical variables were tested with Fisher's exact test for 2×2 tables and the Fisher–Freeman–Halton exact test (Monte Carlo estimate, 200,000 permutations) for larger tables, because several expected cell counts were below five. Means across three groups were compared by one-way analysis of variance (ANOVA) and between two groups by Welch's t test. Exact tests were computed with SciPy version 1.17 (Python). A two-sided p value below 0.05 was

considered significant. Multivariable modelling was not performed because the number of deaths ($n = 14$) was too small to support it.

Ethical considerations

The study followed the Declaration of Helsinki and the Indian Council of Medical Research National Ethical Guidelines for Biomedical and Health Research Involving Human Participants. It was approved by the College Research Council and the Institutional Human Ethics Committee of Baba Raghav Das Medical College, Gorakhpur, and registered with the Clinical Trials Registry of India. Written informed consent was obtained from each patient or legally authorised representative in Hindi or English. No investigation was performed solely for research purposes.

Results

Characteristics of the study population

A total of 100 patients were enrolled. Most were aged 51–70 years (59%), and 53% were men (Table 1). Dyspnea was present in all patients; 26% presented with altered sensorium. Fewer than half (44%) had ever smoked; previous pulmonary tuberculosis (14%) and fuelwood smoke exposure (8%) were the commonest additional exposures. At admission, 98 patients were hypercapnic (PaCO_2 above 45 mmHg) and 80 had PaCO_2 above 70 mmHg. Overall, 31 patients (31%) were intubated and 14 (14%) died in hospital.

Table 1: Baseline characteristics of the study population ($n = 100$)

Characteristic	n (%)
Age group (years)	
18–30	1 (1.0)
31–50	18 (18.0)
51–70	59 (59.0)
71–90	21 (21.0)
Above 90	1 (1.0)
Sex	
Male	53 (53.0)
Female	47 (47.0)
Presenting complaints	
Shortness of breath	100 (100.0)
Cough	32 (32.0)
Altered sensorium	26 (26.0)
Fever	16 (16.0)
Edema	12 (12.0)
Hemoptysis	5 (5.0)
Smoking history	
Ever smoker	44 (44.0)
Never smoker	56 (56.0)
Additional exposure or comorbidity	
None	69 (69.0)
Previous pulmonary tuberculosis	14 (14.0)
Fuelwood smoke	8 (8.0)
Obstructive sleep apnea	4 (4.0)
Interstitial lung disease	2 (2.0)
Sandblasting (silica)	1 (1.0)

Characteristic	n (%)
Kyphosis	1 (1.0)
Malignancy	1 (1.0)
Admission PaCO ₂ (mmHg)	
Below 50	3 (3.0)
50–70	17 (17.0)
Above 70	80 (80.0)
Outcomes	
Endotracheal intubation	31 (31.0)
In-hospital death	14 (14.0)

PaCO₂: arterial partial pressure of carbon dioxide.

Admission PaCO₂ and clinical outcomes

No patient with an admission PaCO₂ of 70 mmHg or below had a GCS score of 8 or less, was intubated or died (Table 2). Patients with PaCO₂ above 70 mmHg more often had a GCS score of 8 or less (32.5% vs 0%, $p = 0.001$), needed BiPAP for more than two days (51.3% vs 15.0%, $p = 0.005$) and were intubated (38.8% vs 0%, $p < 0.001$). Within the lower group, BiPAP beyond two days was needed by none of the 3 patients with PaCO₂ below 50 mmHg and 3 of 17 (17.6%) with PaCO₂ 50–70 mmHg. All 14 deaths occurred in patients with PaCO₂ above 70 mmHg (17.5% vs 0%); however, this difference did not reach statistical significance ($p = 0.07$).

Table 2: Clinical outcomes according to PaCO₂ at admission

Outcome	PaCO ₂ ≤70 mmHg (n = 20)	PaCO ₂ >70 mmHg (n = 80)	p value
GCS score 3–8 at presentation	0 (0.0)	26 (32.5)	0.001
BiPAP for more than 2 days	3 (15.0)	41 (51.3)	0.005
Endotracheal intubation	0 (0.0)	31 (38.8)	<0.001
In-hospital death	0 (0.0)	14 (17.5)	0.07

Values are n (%). p values by Fisher's exact test. BiPAP: bilevel positive airway pressure; GCS: Glasgow Coma Scale; PaCO₂: arterial partial pressure of carbon dioxide.

Oxygenation and response to BiPAP

Severe hypoxemia (PaO₂ below 40 mmHg) was present in 39 patients at admission, of whom 13 (33.3%) died, compared with 1 of 61 (1.6%) patients with PaO₂ of 40 mmHg or above ($p < 0.001$) (Table 3). After 24 hours of BiPAP, 18 patients remained severely hypoxemic; 12 of them (66.7%) died, compared with 2 of 82 (2.4%) whose PaO₂ had risen to 40 mmHg or above ($p < 0.001$). After 24 hours of BiPAP, 60 patients remained hypercapnic, and their mortality (21.7%) was

higher than that of patients who had become normocapnic or hypocapnic (1 of 40, 2.5%; $p = 0.007$). None of the 9 patients who became hypocapnic died.

Table 3: In-hospital mortality according to arterial blood gas values at admission and after 24 hours of BiPAP

Parameter	Patients, n	Deaths, n (%)	p value
PaO ₂ at admission			
Below 40 mmHg	39	13 (33.3)	<0.001
40 mmHg or above	61	1 (1.6)	
PaO ₂ after 24 h BiPAP			
Below 40 mmHg	18	12 (66.7)	<0.001
40 mmHg or above	82	2 (2.4)	
PaCO ₂ after 24 h BiPAP			
Below 35 mmHg	9	0 (0.0)	0.033 ^a
35–45 mmHg	31	1 (3.2)	
Above 45 mmHg	60	13 (21.7)	0.007 ^b

p values for PaO₂ by Fisher's exact test. ^aFisher–Freeman–Halton exact test across the three PaCO₂ groups. ^bFisher's exact test, above 45 mmHg versus 45 mmHg or below. BiPAP: bilevel positive airway pressure; PaO₂/PaCO₂: arterial partial pressure of oxygen/carbon dioxide.

Laboratory parameters

Raised ALT was common (74%), and leukocytosis (TLC above 11000 cells/mm³) was present in 58%; no patient had leukopenia. Hyponatremia was found in 49% and hyperkalemia in 38% of patients. None of these parameters was associated with admission PaCO₂ category ($p = 0.20$ for TLC, 0.28 for ALT, 0.92 for sodium and 0.73 for potassium). Mortality did not differ significantly by ALT, TLC, sodium or potassium category (Table 4). Patients with hypokalemia and hyperkalemia needed BiPAP for more than two days somewhat more often (55.0% and 50.0%) than those with normal potassium (33.3%), but this was not statistically significant ($p = 0.18$). Sodium category was not associated with BiPAP duration.

Table 4: In-hospital mortality according to laboratory parameters at admission

Parameter	Patients, n	Deaths, n (%)	p value
Alanine aminotransferase			
40 U/L or below	26	2 (7.7)	0.35
Above 40 U/L	74	12 (16.2)	
Total leukocyte count (cells/mm ³)			
4000–11000	42	5 (11.9)	0.77
Above 11000	58	9 (15.5)	
Serum sodium			
Below 135 mEq/L	49	7 (14.3)	0.31
135–145 mEq/L	45	5 (11.1)	
Above 145 mEq/L	6	2 (33.3)	
Serum potassium			
Below 3.5 mEq/L	20	4 (20.0)	0.53
3.5–5.0 mEq/L	42	4 (9.5)	
Above 5.0 mEq/L	38	6 (15.8)	

p values by Fisher's exact test (two groups) or Fisher–Freeman–Halton exact test (three groups).

Length of ICU and hospital stay

Mean ICU stay differed significantly across sodium categories and was longest in patients with hyponatremia (3.66 ± 1.29 days vs 2.54 ± 1.12 days with normal sodium; $p < 0.001$) (Table 5). ICU stay also differed across potassium categories ($p = 0.034$), being longest with hypokalemia. Patients with severe hypoxemia at admission stayed longer in the ICU than the remainder (3.12 ± 1.39 vs 2.25 ± 0.89 days, $p = 0.001$). Patients who remained hypercapnic after 24 hours of BiPAP had a longer hospital stay than those who became normocapnic (4.93 ± 1.99 vs 4.00 ± 0.98 days, $p = 0.004$).

Table 5: Length of ICU and hospital stay according to electrolyte and blood gas categories

Category	n	Mean ± SD (days)	p value
ICU stay by serum sodium			
Below 135 mEq/L	49	3.66 ± 1.29	<0.001 ^a
135–145 mEq/L	45	2.54 ± 1.12	
Above 145 mEq/L	6	2.33 ± 0.71	
ICU stay by serum potassium			
Below 3.5 mEq/L	20	2.96 ± 1.12	0.034 ^a
3.5–5.0 mEq/L	42	2.51 ± 1.21	
Above 5.0 mEq/L	38	2.10 ± 1.22	
ICU stay by PaO ₂ at admission			
Below 40 mmHg	39	3.12 ± 1.39	0.001 ^b
40 mmHg or above	61	2.25 ± 0.89	
Hospital stay by PaCO ₂ after 24 h BiPAP			
35–45 mmHg	31	4.00 ± 0.98	0.004 ^b
Above 45 mmHg	60	4.93 ± 1.99	

^aOne-way ANOVA. ^bWelch's t test. ICU: intensive care unit; SD: standard deviation; PaO₂/PaCO₂: arterial partial pressure of oxygen/carbon dioxide; BiPAP: bilevel positive airway pressure.

Discussion

In this prospective cohort of 100 patients admitted with AECOPD in eastern Uttar Pradesh, severe hypercapnia was almost universal, and blood gas values were more closely related to outcome than routine laboratory tests. Three findings stand out. First, a PaCO₂ above 70 mmHg at admission identified every patient who was later intubated and was strongly associated with depressed consciousness and prolonged BiPAP. Second, severe hypoxemia at admission, and even more its persistence after 24 hours of BiPAP, was the strongest marker of in-hospital death. Third, the failure of PaCO₂ to fall into the

normal range after 24 hours of BiPAP was associated with higher mortality and longer hospital stay.

The association between severe hypercapnia and the need for ventilatory support is consistent with the physiology of acute-on-chronic ventilatory failure and with the trials that established NIV as first-line therapy for hypercapnic AECOPD⁸⁻¹⁰. The intubation rate in our cohort (31%) is higher than that reported from high-income countries¹³, which probably reflects the severity at presentation: four in five patients had PaCO₂ above 70 mmHg and a quarter presented with altered sensorium, which may reflect late presentation to a tertiary centre.

Although all deaths occurred in the group with PaCO₂ above 70 mmHg, the association between admission PaCO₂ and mortality was not statistically significant. This is partly a matter of numbers, because only 20 patients had lower PaCO₂ values, but it also agrees with previous work in which the absolute PaCO₂ is a weaker predictor than acidemia. Patients with chronic hypercapnia retain bicarbonate and tolerate high PaCO₂ with near-normal pH¹⁴, and validated tools such as the DECAF score use acidemia rather than PaCO₂¹¹. We did not analyse pH or bicarbonate, and future work from this population should include them.

The response to BiPAP was more informative than the admission values. Patients whose PaO₂ remained below 40 mmHg after 24 hours of BiPAP had a mortality of two in three, and persistent hypercapnia was associated with about eight times the mortality seen when PaCO₂ normalised. This mirrors studies showing that failure of gas exchange to improve on NIV predicts NIV failure and in-hospital death^{15,16}. In practice, a repeat ABG at 24 hours (and ideally earlier) is a simple way to identify patients who need escalation of care, including timely intubation.

Routine laboratory parameters were of limited prognostic value. Raised ALT was present in three-quarters of patients, probably reflecting hypoxic or congestive liver injury, but it was not significantly associated with mortality. Leukocytosis, a marker of infective triggers, was similarly unrelated to death. Hyponatremia was common and was associated with a longer ICU stay, and hypokalemia with a modestly longer ICU stay; these disturbances may reflect diuretic and bronchodilator use, cor pulmonale and the severity of illness rather than independent causal effects. They

remain worth correcting, but in our data they did not predict mortality.

The population profile also deserves attention. More than half of the patients had never smoked, nearly half were women, and previous tuberculosis and fuelwood smoke were frequent exposures. This is in keeping with the importance of non-tobacco risk factors for COPD in India⁵ and supports prevention strategies that address household air pollution and post-tubercular lung disease.

Limitations

This was a single-centre study with a modest sample size and only 14 deaths, which precluded multivariable analysis; the associations reported are therefore unadjusted and may be confounded by overall illness severity. Continuous variables were analysed in categories, which reduces statistical power. PaO₂ was measured on varying fractions of inspired oxygen, and pH and bicarbonate were not analysed. Decisions to intubate were made by the treating team rather than by a fixed protocol. The findings should be confirmed in larger, multicentre studies using continuous variables and adjusted models.

Conclusion

In patients admitted with AECOPD, severe hypercapnia at admission identified those needing prolonged BiPAP and invasive ventilation, while severe hypoxemia at admission and failure of PaO₂ and PaCO₂ to improve after 24 hours of BiPAP were the strongest markers of in-hospital death. Routine laboratory parameters (TLC, ALT, sodium and potassium) did not predict mortality, although hyponatremia was associated with a longer ICU stay. Early ABG assessment, prompt BiPAP and a repeat ABG at 24 hours to guide escalation are recommended.

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