

HACOR SCORE ALONG WITH RADIOLOGICAL CORRELATION TO PREDICT NIV FAILURE IN COPD PATIENTS WITH ACUTE EXACERBATION

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ABSTRACT

Background: Non-invasive ventilation (NIV) is crucial for managing COPD exacerbations. However, NIV failure occurs in 15-24% of cases, and delayed intubation increases mortality. This study aimed to use the HACOR score to predict COPD severity, assess NIV efficacy, identify NIV failure, and correlate findings with pathology through radiological investigations. **Materials and Methods:** This prospective observational study included 90 patients with COPD admitted to the medical wards and intensive care units (ICUs). The HACOR score was assessed at admission and 1–2, 12, and 24 h after NIV initiation. NIV failure was defined as the need for intubation or death. Radiological investigations were performed and correlated with the findings. **Result:** The NIV success rate was 84.4%, and 91.1% of the patients survived. Males had higher HACOR scores than females (OR 0.297, 95% CI: 0.90–0.97; $p = 0.038$). Non-survivors had higher HACOR scores above 5 at 24 h (63% vs. 22% in survivors; OR 5.926, 95% CI: 1.29–27.20; $p = 0.012$). Biomass exposure and NIV failure were significant predictors of adverse outcomes. Higher heart rate, respiratory rate, lower pH, and lower PaO₂/FiO₂ ratios at baseline and 24 h predicted adverse outcomes. Early intubation was associated with better survival rates than late intubation (56% vs. 0%, $p = 0.049$). **Conclusion:** The HACOR score effectively predicts NIV failure in patients with COPD, and radiological findings further enhance its prognostic value. Early identification of high-risk patients can improve clinical outcomes by facilitating timely intervention.

INTRODUCTION

Non-invasive ventilation (NIV) is an important intervention for managing acute exacerbations of chronic obstructive pulmonary disease (COPD), providing benefits in respiratory function by augmenting alveolar ventilation and reducing breathing effort.^[1] NIV typically results in decreased respiratory rate, lower PaCO₂ levels, and improved sensorium in patients.^[2] However, NIV success is not guaranteed, with failure rates in COPD patients ranging from 15% to 24%.^[3] Patients who start with NIV but later require intubation and invasive mechanical ventilation face worse prognoses and higher in-hospital mortality than those directly intubated.^[4] Delayed intubation is linked to increased hospital mortality, highlighting the need for early recognition of patients unlikely to benefit from NIV. Diaphragmatic Dysfunction (DD) is an important factor in COPD, particularly during acute exacerbations (AECOPD), which are more prevalent

in patients with COPD than in healthy individuals of similar age and sex.^[5] The pathogenesis of DD is multifactorial, involving systemic inflammation, prolonged steroid use, and mechanical consequences of lung hyperinflation. During AECOPD, dynamic hyperinflation elevates end-expiratory lung volume and residual volume, pushing the tidal volume (V_t) along the pressure-volume curve. This increases the demand on the respiratory system to generate higher intrathoracic pressures for ventilation. Premature collapse of terminal airways and air trapping introduce intrinsic positive end-expiratory pressure (PEEP), creating additional mechanical load for respiratory muscles before effective inspiratory flow can occur.^[6]

Radiological evaluation through chest radiography or chest CT is essential for assessing disease severity and guiding NIV use in COPD. Imaging studies help quantify structural changes and correlate them with clinical outcomes. Prior studies have attempted to identify NIV failure predictors, such as illness severity, heart rate, respiratory rate, consciousness

level, and arterial blood pH.^[7,8] However, no single variable has shown a reliable predictive value in this regard. Combining multiple bedside parameters into a composite scoring system offers a more practical approach to evaluating NIV success likelihood in COPD patients.^[9]

The HACOR score effectively predicted NIV failure in the early hours of initiation. With high sensitivity and specificity, it helps clinicians identify patients at risk of NIV failure using bedside data.^[10] This practical tool enables intensified monitoring and timely treatment modifications for COPD exacerbations, potentially reducing adverse outcomes. This study aimed to use the HACOR score to predict COPD severity, assess NIV efficacy, identify NIV failure, and correlate findings with pathology through radiological investigations. This approach aims to guide clinical decisions and reduce mortality in high-risk patients.

MATERIALS AND METHODS

This prospective observational study was conducted on 90 patients with COPD admitted to the medical wards and medical ICUs of the Department of Internal Medicine, Madras Medical College, and Rajiv Gandhi Government General Hospital, Chennai, between March 2023 and June 2024. The study was approved by the Institutional Ethics Committee (IEC NO. 21092023), and informed consent was obtained from all the patients before the study.

Inclusion criteria

The study included patients with COPD admitted to the respiratory ICU for NIV who met one of the following conditions: respiratory acidosis ($\text{PaCO}_2 > 45$ mmHg with arterial pH ≤ 7.35), severe dyspnoea with signs of respiratory muscle fatigue or increased work of breathing, or persistent hypoxaemia despite supplemental oxygen therapy.

Exclusion criteria

Patients with respiratory or cardiac arrest, diminished consciousness, uncontrolled psychomotor agitation, massive aspiration, or persistent vomiting; those unable to clear respiratory secretions; those with severe haemodynamic instability unresponsive to treatment; or those with life-threatening hypoxaemia preventing NIV tolerance were excluded from the study.

Methods:

The HACOR score, which comprises heart rate, acidosis (pH), consciousness (Glasgow Coma Scale), oxygenation ($\text{PaO}_2/\text{FiO}_2$), and respiratory rate, was assessed at admission and 1–2 h, 12 h, and 24 h after NIV initiation. NIV was delivered via a CPAP face mask, which was adjusted to minimise leaks. NIV began in pressure support mode, with PEEP at 5–10 cmH₂O and titrated to a maximum of 15 cmH₂O, while FiO_2 was adjusted to maintain SpO_2 above 92%. Additional therapies, including antibiotics,

nebulization, bronchodilators, and supportive measures, were administered as needed.

The clinical condition was regularly reassessed. NIV efficacy was determined by improvement in symptoms and laboratory parameters. Patients who improved were weaned off NIV, while those who deteriorated and met the criteria for invasive mechanical ventilation were intubated. NIV failure was defined as the need for intubation or death. Early NIV failure occurred within 48 h, and late failure occurred after 48 h. The criteria for NIV failure included respiratory distress with a rate above 35 breaths/min, $\text{PaO}_2/\text{FiO}_2 < 100$ mmHg, uncorrected acidosis (pH < 7.25), need for airway protection, haemodynamic instability, fall in consciousness (GCS < 8), respiratory or cardiac arrest, or death.

Radiological investigations were performed and correlated with the findings. For intubated patients, the HACOR score before intubation and the timing of invasive ventilation were recorded. The outcome measures included NIV duration, length of hospital stay, and patient status at discharge or death.

Statistical analysis

Data were entered into Excel and analysed using SPSS (version 29.0). The frequencies of qualitative and categorical variables are presented as numbers and percentages. Groups were compared using the chi-square test. For continuous variables, the mean and standard deviation (SD) were calculated, and comparisons were made using an unpaired Student's t-test.

RESULTS

The mean age was 47.93 ± 12.54 years, with males comprising 3/4th of the patients. Over half of the patients reported shortness of breath with sputum production. The mean BMI was 24.81 ± 1.74 kg/m², with 70% of the patients in the normal range. Approximately 7 in 10 patients had an MRC scale score of four, and 60% had high CAT scores. The main comorbidities were hypertension (40%), diabetes mellitus (26.7%), CKD, and CHD (13.4% each).

Half of the patients were smokers, and biomass exposure was common in women (52.2%). The NIV success rate was 84.4%. Intubation was performed in 14.4% of patients. 91.1% survived. The percentage of patients with HACOR scores < 5 increased from admission at 0, 1, 12, and 24 h (except from 1 to 12 h, where it fell from 61.1% to 58.9%). The most common diagnoses were community-acquired pneumonia, bronchitis, emphysema, and chronic bronchitis. The mean hospital stay was 23.53 ± 7.5 days, with a mean ICU stay of 9.09 ± 3.46 days. CT findings showed airway wall thickening with GGOs and consolidation (40%), bilateral hyperinflation (25.6%), and emphysematous changes with multilobar consolidation (10%) [Table 1].

Table 1: Socio-demographic characteristics of the study population

		N (%)
Age (Years)		47.93 ± 12.54
Sex	Male	70 (77.8%)
	Female	20 (22.2%)
Chief complaints	Shortness of breath	39 (43.3%)
	Shortness of breath with sputum production	51 (56.7%)
BMI	Mean ± SD	24.81 ± 1.74
	18.4 – 24.9	63 (70%)
	25 – 29.9	27 (30%)
MRC scale	3	28 (31.1%)
	4	62 (68.9%)
CAT score	High	52 (57.8%)
	Very high	38 (42.2%)
Comorbidities	Diabetes mellitus	24 (26.7%)
	Hypertension	36 (40%)
	Chronic kidney disease	12 (13.3%)
	Old Pulmonary TB	6 (6.7%)
	Chronic heart disease	12 (13.4%)
Biomass exposure	Present	47 (52.2%)
	Absent/smoking	43 (47.8%)
NIV Success / Failure Rate	Success	76 (84.4%)
	Failure	14 (15.6%)
Intubation	Yes	13 (14.4%)
	No	76 (84.4%)
	Death during NIV	1 (1.1%)
Mortality	Survived	82 (91.1%)
	Dead	8 (8.9%)
HACOR (at 0 hour)	<5	54 (60%)
	>5	36 (40%)
HACOR (at 1 hour)	<5	55 (61.1%)
	>5	35 (38.9%)
HACOR (at 12 hours)	<5	53 (58.9%)
	>5	37 (41.1%)
HACOR (at 24 hours)	<5	67 (74.4%)
	>5	23 (25.6%)
Diagnosis	Atypical Pneumonia with emphysema	23 (25.6%)
	Emphysema with community-acquired pneumonia	25 (27.8%)
	Chronic bronchitis	7 (7.8%)
	Emphysema	5 (5.6%)
	Community-acquired pneumonia with bronchitis	30 (33.3%)
Days of hospital stay		23.53 ± 7.50
Days of ICU stay		9.09 ± 3.46
CT findings	Diffuse air wall thickening with reduced lumen calibre with GGOs progressing to consolidation (RUL/RLL)	36 (40%)
	Bilateral hyperinflated lung fields	23 (25.6%)
	Air wall thickening with reduced lumen calibre	14 (15.6%)
	Bilateral hyperinflated field with emphysematous changes with GGOs progressing to consolidation (Right U/L/II)	9 (10%)
	Bilateral hyperinflated with bilateral multilobar patchy consolidation	9 (10%)

Regarding diagnosis, atypical pneumonia with emphysema (OR 0.164, 95% CI: 0.02–1.38; $p = 0.045$), emphysema with community-acquired pneumonia (OR 1.225, 95% CI: 0.31–4.91; $p = 0.002$), and community-acquired pneumonia with bronchitis (OR 1.230, 95% CI: 0.51–3.0; $p = 0.035$) were associated with the HACOR score at 1–2 h.

Males had higher HACOR scores than females (OR 0.297, 95% CI: 0.90–0.97; $p = 0.038$). No association was found between age, chief complaints, BMI, MRC scale, CAT score, comorbidities, biomass exposure, NIV outcome, intubation status, mortality, chronic bronchitis, emphysema, or duration of hospital or ICU stay ($p > 0.05$) [Table 2].

Table 2: Association of risk factors for HACOR score at 1-2 hours

HACOR score at 1-2 hours		OR (95%CI)	P value
Age		1.649(0.59-0.79)	0.648
Sex	Male	0.297(0.90-0.97)	0.038
	Female		
Chief complaints	Shortness of breath	1.643(0.69-3.90)	0.259
	Shortness of breath with sputum production		
BMI	18.4 – 24.9	1.300(0.52-3.24)	0.573
	25 – 29.9		
MRC scale	3	1.044(0.42-2.60)	0.926
	4		
CAT score	High	1.164(0.50-2.73)	0.727

	Very high		
Comorbidities	Diabetes mellitus	0.525(0.19-1.44)	0.206
	Hypertension	1.360(0.58-3.21)	0.482
	Chronic kidney disease	2.366(0.69-8.14)	0.164
	Old Pulmonary TB	0.735(0.12-4.24)	0.73
	Chronic heart disease	0.735(0.12-4.24)	0.73
Biomass exposure	Present	2.036(0.87-4.80)	0.102
	Absent/smoking		
NIV Success / Failure Rate	Success	1.818(0.52-6.32)	0.342
	Failure		
Intubation	Yes	0.550(0.16-1.91)	0.342
	No		
Mortality	Survived	0.891(0.20-3.98)	0.88
	Dead		
Diagnosis	Atypical Pneumonia with emphysema	0.164(0.02-1.38)	0.045
	Emphysema with community-acquired pneumonia	1.225(0.31-4.91)	0.002
	Chronic bronchitis	1.062(0.63-3.11)	0.912
	Emphysema	1.257(0.49-3.20)	0.631
	Community-acquired pneumonia with bronchitis	1.230(0.51-3.0)	0.035
Days of hospital stay		17	0.472
Days of ICU stay		13.90(0.18-0.36)	0.234

Intubation was required in all non-survivors versus 7% of survivors (OR 2.333, 95% CI: 1.27–4.27; $p < 0.0001$). Patients with atypical pneumonia with emphysema (75% among non-survivors vs. 21% among survivors; OR 3.571, $p = 0.011$), emphysema with community-acquired pneumonia (88% vs. 22%; OR 1.321, $p = 0.005$), and pneumonia with bronchitis (38% vs. 33%; OR 1.222, $p = 0.046$) showed higher mortality. Non-survivors had higher HACOR scores above 5 at 24 h (63% vs. 22% in survivors; OR 5.926, 95% CI: 1.29–27.20; $p = 0.012$).

All non-survivors experienced NIV failure compared to 7% of survivors ($p < 0.0001$). No differences were found between survivors and non-survivors in age, sex, complaints, body mass index, MRC scale, CAT score, comorbidities (diabetes mellitus, hypertension, chronic kidney disease, old pulmonary tuberculosis, chronic heart disease), biomass exposure, hospital stay, ICU stay, or HACOR scores at baseline, 1 h, and 12 h ($p > 0.05$) [Table 3].

Table 3: Association of risk factors between the survivors' vs non-survivors' groups

			Mortality		OR (95%CI)	p-value
			Survived (n=82)	Dead (n=8)		
Age			47.30 ± 12.49	54.38 ± 11.93	1.524(0.16-2.09)	0.129
Sex	Male		64(78%)	6(75%)	1.185(0.22-6.38)	0.843
	Female		18(22%)	2(25%)		
Chief complaints	Shortness of breath		34(41%)	5(63%)	0.425(0.1-1.9)	0.252
	Shortness of breath with sputum production		48(59%)	3(38%)		
BMI	Mean		24.80±1.77	24.96±1.53	0.252(0.14-1.13)	0.801
	18.4 – 24.9		58(71%)	5(63%)	1.450(0.32-6.55)	0.628
	25 – 29.9		24(29%)	3(38%)		
MRC scale	3		27(33%)	1(13%)	3.436(0.40-29.36)	0.234
	4		55(67%)	7(87%)		
CAT score	High		48(59%)	4(50%)	1.412(0.33-6.04)	0.641
	Very high		34(41%)	4(50%)		
Comorbidities	Diabetes mellitus	No	59(72%)	7(88%)	0.366(0.04-3.15)	0.342
		Yes	23(28%)	1(13%)		
	Hypertension	No	51(62%)	3(38%)	2.742(0.61-12.28)	0.174
		Yes	31(38%)	5(63%)		
	Chronic kidney disease	No	71(87%)	7(87%)	0.922(0.10-8.23)	0.942
		Yes	11(13%)	1(13%)		
	Old Pulmonary TB	No	76(93%)	8(100%)	0.905(0.84-0.97)	0.428
		Yes	6(7%)	0		
	Chronic heart disease	No	77(94%)	7(88%)	2.200(0.23-21.56)	0.488
		Yes	5(6%)	1(13%)		
Biomass exposure	Present		45(55%)	2(25%)	3.649(0.70-19.16)	0.106
	Absent / smoking		37(45%)	6(75%)		
NIV Success / Failure Rate	Success		76(93%)	0	0.429(0.23-0.79)	< 0.0001
	Failure		6(7%)	8(100%)		
Intubation	Yes		6(7%)	8(100%)	2.333(1.27-4.27)	< 0.0001
	No		76(93%)	0		
Diagnosis	Atypical Pneumonia with emphysema	No	65(79%)	2(25%)	3.571(0.60-21.13)	0.011
		Yes	17(21%)	6(75%)		

	Emphysema with community-acquired pneumonia	No	64(78%)	1(13%)	1.321(0.14-12.15)	0.005
		Yes	18(22%)	7(88%)		
	Chronic bronchitis	No	76(93%)	7(88%)	0.589(0.07-5.14)	0.629
		Yes	6(7%)	1(13%)		
	Emphysema	No	78(95%)	7(88%)	0.345(0.04-2.96)	0.312
		Yes	4(5%)	1(13%)		
	Community-acquired pneumonia with bronchitis	No	55(67%)	5(63%)	1.222(0.27-5.50)	0.046
		Yes	27(33%)	3(38%)		
Days of hospital stay (mean)			23.48 ±7.68	24.13 ±5.64	1.125(0.62-0.49)	0.817
Days of ICU stay (mean)			9.23 ±3.52	7.63 ±2.45	2.034(0.93-4.14)	0.211
HACOR (at 0 hour)	<5		49(60%)	5(63%)	0.891(0.20-3.98)	0.88
	>5		33(40%)	3(38%)		
HACOR (at 1 hour)	<5		51(62%)	4(50%)	1.645(0.38-7.06)	0.499
	>5		31(38%)	4(50%)		
HACOR (at 12 hours)	<5		48(59%)	5(63%)	0.847(0.19-3.79)	0.828
	>5		34(41%)	3(37%)		
HACOR (at 24 hours)	<5		64(78%)	3(37%)	5.926(1.29-27.20)	0.012
	>5		18(22%)	5(63%)		

Biomass exposure was a significant predictor of adverse outcomes, with exposed patients showing an increased risk (AOR: 0.000, 95% CI: 0.87–4.80; p = 0.009). NIV success was associated with improved outcomes, as NIV failure increased adverse events

(AOR: 0.764; 95% CI: 0.52–6.32; p = 0.010). No significant association was found between adverse outcomes and sex, MRC scale, chief complaints, or intubation timing (p > 0.05) [Table 4].

Table 4: Binary logistic regression for risk factors associated with mortality

Variable		Coef (B)	AOR (Exp (B))	95%CI	p- value
Sex	Male	-1.923	0.146	0.01-2.13	0.159
	Female				
MRC scale	3	-0.189	0.828	0.40-2.36	0.999
	4				
Chief complaints	Shortness of breath	17.081	0.262	0.1-1.9	0.996
	Shortness of breath with sputum production				
Biomass exposure	Present	-34.308	0.000	0.87-4.80	0.009
	Absent / smoking				
NIV Success/ Failure Rate	Success	33.158	0.764	0.52-6.32	0.010
	Failure				
Intubation	Early	-	0.000	0.16-1.91	1
	Late				

Chronic kidney disease (OR: 0.491, 95% CI: 0.25–0.45; p = 0.050) and old pulmonary tuberculosis (OR: 1.220, 95% CI: 0.99–1.19; p = 0.001) were associated with adverse outcomes. Higher heart rate at baseline (OR: 1.991, 95% CI: 1.04–1.95; p = 0.033) and 24 hours (OR: 1.742, 95% CI: 0.78–2.50; p = 0.003), respiratory rate at baseline (OR: 1.048, 95% CI: 0.01–1.14; p = 0.007) and 24 hours (OR: 0.846, 95% CI: 1.11–1.73; p = 0.005), lower pH at baseline (OR: 2.113, 95% CI: 1.36–2.92; p = 0.011) and 24 hours

(OR: 0.445, 95% CI: 0.01–1.20; p = 0.010), and lower PaO₂/FiO₂ ratios at baseline (OR: 0.918, 95% CI: 0.27–1.58; p = 0.001) and 24 hours (OR: 1.229, 95% CI: 0.21–2.70; p = 0.002) predicted adverse outcomes. A lower GCS at 24 h was also significant (OR: 0.382, 95% CI: 0.00–0.84; p = 0.026). No significant differences were found for diabetes, hypertension, chronic heart disease, blood pressure, or GCS at baseline (p > 0.05) [Table 5].

Table 5: Univariate and multivariate analyses for association of risk factors with NIV failure

Variable		Univariate analysis OR (95%CI)	p-value	Multivariate analysis OR (95%CI)	p-value
Comorbidities	Diabetes mellitus	1.467(0.98-2.24)	0.098	-	-
	Hypertension	1.830(1.01-1.49)	0.976	-	-
	Chronic kidney disease	1.529(1.76-2.97)	0.004	0.491(0.25-0.45)	0.050
	Old Pulmonary TB	1.027(1.01-1.04)	0.001	1.220(0.99-1.19)	0.001
	Chronic heart disease	2.193(0.99-1.23)	0.067	-	-
HACOR at 0 hour	HR (beats/min)	0.976(0.98-2.34)	0.000	1.991(1.04-1.95)	0.033
	RR (breaths/min)	1.045(0.25-1.47)	0.003	1.048(0.01-1.14)	0.007
	PH	2.765(1.11-2.07)	0.000	2.113(1.36-2.92)	0.011
	PaO ₂ /FiO ₂	1.806(0.98-1.45)	0.001	0.918(0.27-1.58)	0.001
	GCS	2.367(1.98-3.11)	0.062	-	-
	SBP, mmHg	1.478(0.67-2.88)	0.982	-	-
	DBP, mmHg	1.210(1.27-1.99)	0.43	-	-
HACOR at 24 hours	HR (beats/min)	2.306(0.54-1.32)	0.001	1.742(0.78-2.50)	0.003
	RR (breaths/min)	1.520(0.73-2.81)	0.024	0.846(1.11-1.73)	0.005
	PH	1.091(0.41-2.34)	0.030	0.445(0.01-1.20)	0.010

	PaO ₂ /FiO ₂	1.734(0.94-1.71)	0.011	1.229(0.21-2.70)	0.002
	GCS	0.297(0.01-3.87)	0.005	0.382(0.00-0.84)	0.026

The prevalence of pulmonary tuberculosis was higher in the late intubation group than in the early intubation group (25% vs. 22.2%, $p = 0.000$). Patients who underwent early intubation had a higher baseline systolic blood pressure (169.78 ± 16.11 mmHg vs. 146 ± 20.27 mmHg; $p = 0.043$). At 24 hours, early intubation showed lower pH (7.30 ± 0.03 vs 7.31 ± 0.03 ; $p = 0.046$), PCO₂ (30.33 ± 5.45 vs 33.25 ± 0.96 ;

$p = 0.033$), and GCS (15 vs 15; $p = 0.018$). The mortality rate was higher in the late intubation group than in the early intubation group (100% vs. 44%, $p = 0.049$). No differences were observed between the groups in terms of age, sex, comorbidities, baseline or 24-hour vital signs, PaO₂, diastolic blood pressure, HACOR score, or other parameters ($p > 0.05$) [Table 6].

Table 6: Early vs late intubation for patients with HACOR score at 1-2hours

		Early intubation (n=9)	Late intubation (n=4)	p-value
Age		46.11 $\pm$ 12.68	59.25 $\pm$ 4.35	0.073
Male		7(77.8%)	2(50%)	0.358
Comorbidities	Diabetes mellitus	2(22.2%)	1(25%)	0.347
	Hypertension	4(44.4%)	3(75%)	0.349
	Chronic kidney disease	2(22.2%)	1(25%)	0.347
	Old Pulmonary TB	2(22.2%)	1(25%)	0.000
	Chronic heart disease	1(11.1%)	1(25%)	0.169
HACOR at 0 hour	HR (beats/min)	72.89 $\pm$ 5.28	70.50 $\pm$ 9.33	0.561
	RR (breaths/min)	21.33 $\pm$ 2.45	21 $\pm$ 2.58	0.619
	PH	7.30 $\pm$ 0.03	7.29 $\pm$ 0.03	0.455
	Pco ₂	29.67 $\pm$ 7.09	31.75 $\pm$ 5.85	0.22
	PaO ₂ /FiO ₂	163.01 $\pm$ 72.30	117.57 $\pm$ 93.56	0.357
	GCS	14.67 $\pm$ 0.50	15	0.828
	SBP, mmHg	169.78 $\pm$ 16.11	146 $\pm$ 20.27	0.043
	DBP, mmHg	109 $\pm$ 9.90	95.50 $\pm$ 16.84	0.092
HACOR at 24 hours	HR (beats/min)	98.33 $\pm$ 19.98	89.75 $\pm$ 7.76	0.322
	RR (breaths/min)	18.56 $\pm$ 1.67	18.50 $\pm$ 1.29	0.423
	PH	7.30 $\pm$ 0.03	7.31 $\pm$ 0.03	0.046
	Pco ₂	30.33 $\pm$ 5.45	33.25 $\pm$ 0.96	0.033
	PaO ₂ /FiO ₂	146.83 $\pm$ 43.07	197.20 $\pm$ 50.30	0.575
	GCS	15	15	0.018
HACOR score before NIV		5 $\pm$ 2.24	6 $\pm$ 2.71	
HACOR score at 24 hours of NIV		7.89 $\pm$ 3.10	6.75 $\pm$ 6.24	
Mortality	No	5	0	0.049
	Yes	4	4	

DISCUSSION

In our study, NIV failure was 15.6% (success 84.4%), aligning with the lower end of the reported ranges (15–24%). Similarly, Duan et al. reported NIV failure rates of 18.8–18.9%, which supports our findings.¹⁰ Our low failure rate might indicate effective patient selection and timely initiation of NIV. However, specific subgroups showed different outcomes. Waghmare et al. reported a higher NIV failure (38%) in post-tuberculosis obstructive airway disease, highlighting how coexisting fibrotic changes may limit NIV efficacy.^[11] In contrast, non-COPD chronic respiratory conditions, such as obesity hypoventilation or chest-wall disorders, show lower NIV failure (~13%), placing our COPD population between these extremes.¹² Moreover, NIV failure in AECOPD is significantly better than in conditions such as de novo hypoxaemic respiratory failure, where rates can reach 40–50%.^[10,12]

In our study, patients had a mean age of 47.93 ± 12.54 years, contrasting slightly with the higher mean age of 59.82 ± 11.42 years reported by Teh YH et al.^[13] Hypertension (40%), diabetes mellitus (26.7%), and CKD (13.3%) were the top comorbidities in our population, similar to Teh YH et al.'s findings.^[13]

Anand et al. reported a higher hypertension prevalence (60.8%) compared to our study (40%).^[14] These consistent comorbidity patterns across studies underscore common chronic disease profiles affecting COPD patients, reinforcing the generalizability of the findings.

In our study, the mean hospital stays for patients who died was 24.13 ± 5.64 days, nearly double the duration reported by Gunen et al. (11.5 ± 6.9).^[15] Despite this difference, both studies highlight the extended hospitalisation associated with severe illness leading to mortality. Teh et al. found a significant association between NIV outcomes and mortality, consistent with our results.^[13] This indicates a robust link between NIV failure and increased mortality risk, which has been validated across diverse populations and geographic locations. Our study revealed no association between age, sex, or BMI and mortality, paralleling the findings of Gunen et al. ($p > 0.05$).^[15] This lack of correlation across multiple populations suggests that other factors, such as illness severity, significantly outweigh demographic variables in predicting mortality outcomes. Consistent with findings by Sprooten et al., our study also found no gender-based association with mortality, further supporting the

limited influence of gender on survival outcomes in COPD patients.^[16]

In agreement with Teh YH et al., our findings demonstrated a significant association between Glasgow Coma Scale (GCS) scores and NIV failure, emphasising GCS as a reliable predictor of NIV outcomes.^[13] Hypertension and diabetes mellitus showed no correlation with NIV outcomes in our study, aligning with reports by Anand et al. ($p=0.251$ for HTN and $p=0.111$ for DM), Duan et al., and Jha et al. ($p=0.151$ for HTN and $p=0.745$ for DM).^[14,17,18] These results collectively suggest that these chronic conditions have a limited impact on NIV efficacy. Our study and that of Duan et al. both highlighted significant associations between heart rate, GCS, and NIV outcomes, underlining their consistent predictive value across diverse clinical environments.^[17] Varpaei et al. identified an association between baseline HACOR scores (9.34 ± 2.30) and NIV failure ($p<0.001$), whereas our study demonstrated associations at both baseline and 24 hours.¹⁹ Both studies confirmed that higher initial HACOR scores significantly predicted NIV failure, validating the reliability of the HACOR score as a prognostic tool.

CONCLUSION

Our study demonstrated that NIV failure in patients with acute exacerbations of COPD is associated with increased in-hospital mortality and prolonged hospitalisation. Baseline PaCO_2 levels did not predict NIV failure in this study. The HACOR score is a reliable predictor of NIV failure in COPD patients, and radiological findings further enhance its prognostic value. Patients with a HACOR score greater than 5 at 1–2 h after starting NIV were at a significantly higher risk of treatment failure. Early intubation within 48 h in these high-risk patients was linked to better survival than delayed intubation. These findings highlight the importance of close monitoring and early identification of patients likely to fail NIV, enabling prompt escalation of care and potentially improving clinical outcomes.

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