

Research Article

Study on the Clinical Spectrum of Respiratory Conditions Presenting with Hypercapnic Respiratory Failure - A Cross-Sectional Observational Study

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ABSTRACT

Background: Hypercapnic respiratory failure (HRF) reflects failure of pulmonary ventilation and confers substantial short-term morbidity and mortality. Patterns of disease precipitating HRF vary geographically and influence outcome.

Methods: We performed a prospective cross-sectional study at a tertiary centre in Hyderabad (Sept 2022 - Feb 2023). Sixty-nine consecutive adults (≥ 18 y) with respiratory-cause HRF ($\text{PaCO}_2 > 45$ mmHg, $\text{pH} < 7.35$) were enrolled. Clinical variables, comorbidity, arterial blood-gases, radiology, echocardiography and hospital course were recorded. Non-invasive ventilation (NIV) was first-line; failure was defined as need for endotracheal intubation or in-hospital death. Descriptive statistics and logistic regression explored factors associated with NIV failure and mortality.

Results: Mean age was 55 ± 16 y; 50.7 % were male. Leading aetiologies were acute exacerbation of COPD (AECOPD, 26 %), bronchiectasis (9 %), and OSA/OHS (7 %); mixed phenotypes accounted for 42 % (Figure 2). Hypertension (39 %) and diabetes (25 %) were common comorbidities. Median hospital stay was 7 days (IQR 6-8). NIV succeeded in 85.5 % (59/69). NIV failure (14.5 %) was strongly associated with prior-year HRF admission (OR 4.3, $p = 0.029$) and in-hospital mortality (100 % vs 3 %, $p < 0.001$). Overall mortality was 11.6 %; AECOPD contributed 62.5 % of deaths.

Conclusion: In this South-Indian cohort, AECOPD—often co-existing with other airway diseases—was the commonest precipitant of HRF and the principal driver of mortality. NIV was effective in the majority; previous HRF admission heralded NIV failure and death, highlighting a target group for enhanced post-discharge care.

Keywords: Hypercapnia, COPD, Non-Invasive Ventilation, Bronchiectasis, Obesity Hypoventilation.

INTRODUCTION

Respiratory failure, defined as derangements in arterial oxygen or carbon-dioxide tensions that threaten organ function, remains a leading trigger for emergency admission [1]. Hypercapnic respiratory failure (type II) arises when alveolar ventilation is insufficient to clear metabolic CO_2 . Globally, its epidemiology is shaped by chronic airway diseases, obesity, neuromuscular disorders and acute pulmonary insults [2]. Hospitalisations for acute exacerbations of COPD (AECOPD) alone exceed three million annually and carry in-patient mortality of 2–8 % (up to 15 % in ICU) [3]. South-Asian series report rising contributions from post-tuberculous bronchiectasis and obesity hypoventilation syndrome (OHS) [4], yet contemporary Indian data remain sparse. Non-invasive ventilation (NIV) revolutionised the management of acute hypercapnic respiratory failure (AHRF). Randomised trials demonstrate NIV reduces the need for intubation and improves survival in AECOPD

Compared with standard therapy [5]. Nevertheless, NIV fails in 10–30 % [6]. Predictors include pneumonia, severe acidosis, encephalopathy and prior hospitalisation [7]. Local validation is mandatory before extrapolating these predictors to heterogeneous Indian populations, where comorbidity profiles and health-system resources differ.

Evidence from Telangana is limited to small retrospective audits. We therefore undertook a prospective study at the Nizam's Institute of Medical Sciences to (i) describe the current clinical spectrum of respiratory disorders presenting with HRF; (ii) quantify hospital outcomes including NIV success, length-of-stay and mortality; and (iii) identify factors associated with adverse outcome. Generating such real-world data will inform risk-stratified pathways and optimise use of finite critical-care resources.

MATERIALS AND METHODS

Design & Setting

Single-centre, prospective, cross-sectional study in the Department of Respiratory Medicine, NIMS-Hyderabad (Sept 2022–Feb 2023). Ethical approval obtained (IEC-NIMS/2022-RM/047).

Participants

Adults ≥ 18 y with respiratory-cause HRF ($\text{PaCO}_2 > 45$ mmHg, $\text{pH} < 7.35$) were screened. Exclusions: post-operative/trauma HRF, non-respiratory metabolic causes, refusal of consent.

Data Collection

A structured proforma captured demographics, antecedent admissions, comorbidities, anthropometry, clinical parameters, ABG, radiograph/HRCT, and echocardiography. Severity indices (GCS, presence of pulmonary hypertension) were recorded.

Management Protocol

All patients received standard medical therapy plus NIV (bilevel positive-pressure ventilator; initial IPAP 12 cmH₂O, EPAP 6 cmH₂O titrated to achieve $\text{pH} > 7.30$ and PaCO_2 fall > 10 %). Indications for escalation to invasive ventilation: worsening acidosis, hypoxaemia on $\text{FiO}_2 \geq 0.6$, haemodynamic instability or encephalopathy (GCS < 8).

Outcomes

Primary—NIV success (avoidance of intubation and survival to discharge). Secondary—length-of-stay (LOS), in-hospital mortality.

Statistical Analysis

Data analysed with SPSS v20. Continuous variables expressed as mean \pm SD or median (IQR); categorical as proportion. Logistic regression assessed associations with NIV failure and mortality; $p < 0.05$ considered significant.

RESULTS

Patient Profile

A total of 69 patients were enrolled. Age distribution was bimodal (Figure 1) with mean 55 ± 15.9 y; gender distribution was even (M 51 %). Overweight/obesity ($\text{BMI} > 25$ kg m⁻²) was present in 39 % (Table 1).

Figure 1 illustrates age strata; Figure 2 summarises leading diagnoses.

Aetiology of HRF

Pure AECOPD accounted for 26 % but overlap phenotypes (AECOPD + bronchiectasis/asthma/OSA) comprised a further 19 %. Post-tuberculous bronchiectasis alone caused 9 %, while OSA/OHS syndromes contributed 7 %. Community-acquired pneumonia precipitated HRF in 3 % as sole diagnosis but co-occurred in mixed cases (Table 2).

Hospital Course and NIV Outcomes

Median LOS was 7 days (IQR 6–8) overall but prolonged in connective-tissue-disease ILD (11 days). NIV succeeded in 85.5 % (59/69). Ten patients met failure criteria; eight died and two were intubated then survived (Table 3). Overall mortality was 11.6 %.

Prior-year admission for HRF was recorded in 71 %. This variable predicted NIV failure (OR 4.3; 95 % CI 1.1–17.0) and mortality ($p = 0.029$). AECOPD explained 62.5 % of deaths; cardiac arrest and septic shock were immediate causes (Table 4).

Table 1. Baseline Characteristics of the Study Cohort (N = 69)

| Characteristic | Overall |
|---------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------|
| Age, mean \pm SD (y) | 55.3 \pm 16.0 |
| Male sex, n (%) | 35 (50.7) |
| BMI, mean \pm SD (kg·m ⁻²) | 26.1 \pm 6.3 |
| Weight status, n (%) | Underweight 6 (8.7); Normal 33 (47.8); Overweight 15 (21.7); Obese class 1 3 (4.3); Obese class 2 3 (4.3); Obese class 3 9 (13.0) |
| Hypertension, n (%) | 27 (39.1) |
| Diabetes mellitus, n (%) | 17 (24.6) |
| Hypothyroidism, n (%) | 4 (5.8) |
| Prior hospitalisation for HRF in past year, n (%) | 49 (71.0) |

Table 2. Respiratory Aetiologies Precipitating Hypercapnic Respiratory Failure

| Aetiology (primary or contributory) | n (%) | Median LOS days (IQR) |
|-------------------------------------|-----------|-----------------------|
| AECOPD (\pm combination*) | 33 (47.8) | 7 (6–8) |
| Bronchiectasis (\pm OLD PTB) | 18 (26.1) | 7 (6–7) |
| OSA/OHS (\pm other) | 15 (21.7) | 7 (6–8) |
| CAP (\pm other) | 11 (15.9) | 8 (6–12) |
| Kyphoscoliosis / Pleural effusion | 4 (5.8) | 5.5 (4–8) |
| CTD-ILD | 1 (1.4) | 11 (–) |

Table 3. Niv Outcome by Selected Baseline Characteristics

| Characteristic | NIV Failure (n=10) | NIV Success (n=59) | p-value |
|---------------------------------------|------------------------------------|--------------------------|---------------------------------------------|
| Sex | Male 5 (14.3%) Female 5 (14.7%) | 30 (85.7%) 29 (85.3%) | 1.00 ^a |
| Mortality | Yes 8 (100%) No 2 (3.3%) | 0 59 (96.7%) | <0.0001 ^b |
| Prior HRF hospitalisation (past year) | Yes 10 (20.4%) No 0 | 39 (79.6%) 20 (100%) | 0.053 ^a (0.029 ^c) |

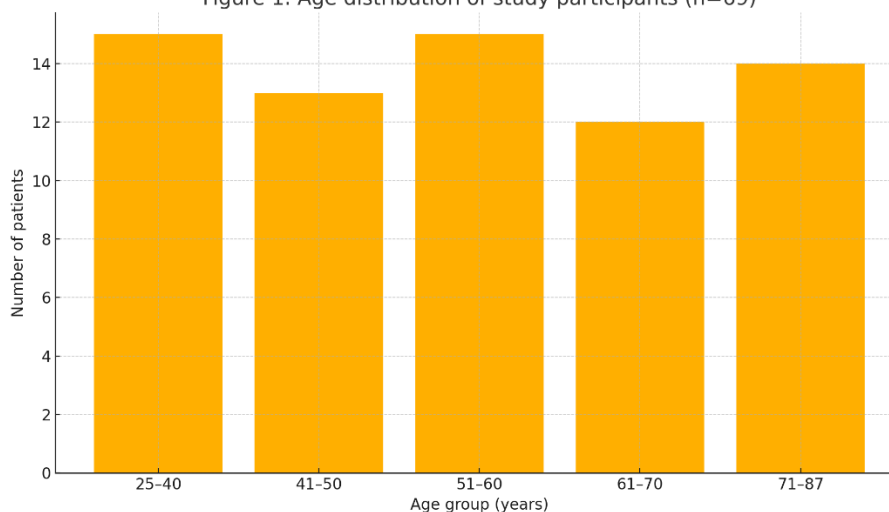
Table 4. Mortality by Major Aetiology Group

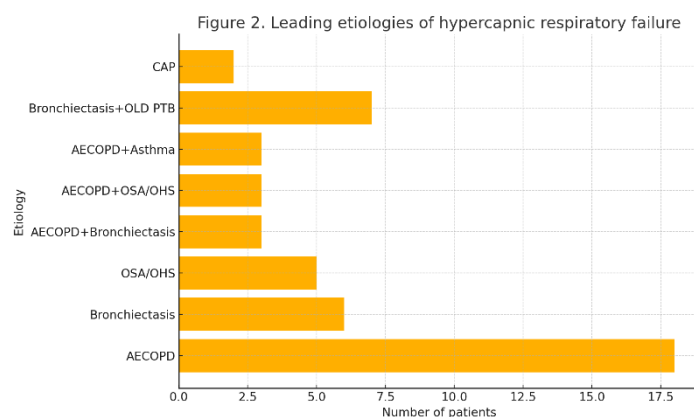
| Aetiology group | Deaths / Total | % | p vs all other groups ^a |
|---------------------------------------|----------------|------|------------------------------------|
| AECOPD (\pm combination) | 5 / 33 | 15.2 | 0.47 |
| CAP (\pm combination) | 2 / 11 | 18.2 | 0.64 |
| OSA/OHS (\pm combination) | 1 / 15 | 6.7 | 1.00 |
| Bronchiectasis (\pm OLD PTB) | 0 / 18 | 0 | 0.06 ^b |
| Other (Kyphoscoliosis, CTD-ILD, etc.) | 0 / 4 | 0 | — |

Table 5. Arterial Blood Gas (Abg) Indices at Presentation by Niv Outcome (Exploratory)

| ABG variable | NIV Failure (n=10) | NIV Success (n=59) | Mean difference | p-value ^a |
|----------------------------------------|--------------------|--------------------|-----------------|----------------------|
| pH | 7.17 \pm 0.09 | 7.29 \pm 0.04 | –0.12 | <0.0001 |
| PaCO ₂ (mmHg) | 74 \pm 16 | 60 \pm 15 | +14 | 0.004 |
| PaO ₂ (mmHg) | 48 \pm 20 | 53 \pm 18 | –5 | 0.45 |
| HCO ₃ [–] (mmol/L) | 36.7 \pm 5.5 | 31.2 \pm 8.1 | +5.5 | 0.052 |

Figure 1. Age distribution of study participants (n=69)





DISCUSSION

This prospective Indian cohort confirms AECOPD as the dominant driver of hypercapnic respiratory failure, mirroring global trends [8]. However, nearly half our patients harboured mixed pathologies, chiefly post-tuberculous bronchiectasis and OSA/OHS. Similar overlap has been described from South-East Asia [9] and underscores the lingering pulmonary sequelae of tuberculosis and burgeoning obesity epidemic.

Our NIV success rate (85 %) aligns with pivotal trials [5] and recent real-world audits (80–90 %) [10]. Notably, NIV failure clustered in patients rehospitalised within the preceding year, supporting findings by Cavelot et al. [11] that prior admission portends readmission and death. These individuals likely possess advanced underlying lung disease, impaired respiratory drive or social barriers to early care. Post-discharge interventions—pulmonary rehabilitation, domiciliary NIV, weight management for OHS—may mitigate recurrence [12].

Hospital mortality (11.6 %) sits between figures from European medical wards (≈ 10 %) [13] and ICU cohorts (19–26 %) [6,14]. All deaths followed NIV failure, reaffirming the importance of timely escalation protocols. Cardiac arrest and septic shock dominated terminal events, echoing German data where pneumonia complicated 28 % of COPD-related HRF deaths [15]. Early infection surveillance and sepsis bundles could therefore improve outcome.

Hypertension and diabetes were the commonest comorbidities, reflecting regional non-communicable disease burden. Although neither independently predicted mortality, metabolic syndrome augments systemic inflammation and may accelerate COPD progression [16]. Multidisciplinary clinics integrating respiratory and metabolic care warrant evaluation.

Strengths of our study include prospective design, uniform NIV protocol and comprehensive echocardiographic assessment. Limitations comprise single-centre scope, modest sample and six-month enrolment window, potentially under-representing seasonal respiratory infections. Moreover, long-term outcomes beyond discharge were not captured.

Future research should explore post-discharge NIV adherence, quality-of-life trajectories and cost-effectiveness of community respiratory outreach. Genetic and biomarker profiling could refine phenotyping of overlap syndromes. At policy level, smoking cessation drives and TB control remain pivotal to curb COPD and bronchiectasis epidemics.

CONCLUSION

In this Telangana tertiary-care cohort, AECOPD—often entwined with bronchiectasis or sleep-disordered breathing—was the leading precipitant of hypercapnic respiratory failure and accounted for most deaths. NIV provided effective first-line support, yet failure, strongly linked to prior HRF admission, carried universal mortality. Targeted post-discharge surveillance for high-risk survivors and aggressive management of comorbidities may curtail recurrent decompensation. These findings highlight the evolving Indian landscape of HRF and emphasise the need for integrated, phenotype-driven care pathways.

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