

Modified DECAF Score in Acute Exacerbation of COPD: A Predictor of In-Hospital Stay and Mortality

Dr. Pulkit Singh¹, Dr. Monty Bansal¹, Dr. (Prof.) Deepak Sharma^{1*}, Dr. (Prof.) Ashok Kumar Dash¹, Dr. Pinjari Dawood¹

¹ Department of General Medicine, School of Medical Sciences and Research (SMS&R), Sharda University, Greater Noida, Uttar Pradesh, India

*Corresponding Author: Dr. Deepak Sharma, Department of General Medicine, Sharda University, Greater Noida – 201306, Uttar Pradesh, India. Email: deepak.sharma4@sharda.ac.in, Phone Number: 9810730971

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ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a major cause of in-hospital mortality and prolonged hospitalization. The modified DECAF score, replacing atrial fibrillation from the original DECAF with frequency of hospitalization in the preceding year, offers a potentially superior tool for early risk stratification. This study evaluated the utility of the modified DECAF score in predicting in-hospital mortality and length of hospital stay (LOS) in patients admitted with AECOPD.

Methods: This was a cross-sectional observational analytical study conducted at Sharda Hospital, Greater Noida, from May 2024 to November 2025. One hundred and three patients admitted with AECOPD fulfilling inclusion criteria were enrolled. Modified DECAF scores were calculated at admission using five components: extended MRC dyspnea grade (eMRCD), eosinopenia, consolidation on chest radiograph, acidemia (pH <7.30), and frequency of hospitalization in the last year. Statistical analysis included chi-square tests, Pearson correlation, Kaplan-Meier survival analysis, and Cox proportional hazards modelling.

Results: The mean age was 65.9 ± 11.8 years (range 46–98 years), with 64% males. In-hospital mortality was 27.2% (28/103). The mean modified DECAF score among survivors was 2.54 ± 1.23 versus 3.86 ± 1.30 among non-survivors. The modified DECAF score was strongly associated with in-hospital mortality (log-rank $p < 0.001$). Dyspnea severity (eMRCD) and acidemia were individually significant predictors of mortality ($p < 0.001$ and $p = 0.046$, respectively). Eosinopenia, consolidation, and prior hospitalization did not independently reach statistical significance. The modified DECAF score was not significantly associated with length of hospital stay ($p = 0.084$), although there was a trend toward longer stays at higher scores.

Conclusions: The modified DECAF score is a clinically useful and easily calculable bedside tool for stratifying in-hospital mortality risk in AECOPD. Dyspnea severity and acidemia are the dominant individual predictors of short-term mortality. Routine application of this score at admission can guide resource allocation, monitoring intensity, and early initiation of appropriate therapies.

Keywords: AECOPD, Modified DECAF Score, In-hospital Mortality, Length of Stay, Dyspnea, Acidemia, Eosinopenia, India.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory condition characterized by persistent airflow limitation and chronic respiratory symptoms including breathlessness, cough, and

expectoration. It currently ranks as the fourth most common cause of global mortality and is projected to become the third leading cause by 2030.(1,2) Acute exacerbations of COPD (AECOPD) represent a major driver of disease burden, associated with accelerated lung

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function decline, reduced quality of life, increased healthcare utilization, and substantial short- and long-term mortality.(3–5)

Patients hospitalized with AECOPD represent a particularly high-risk group. In-hospital mortality rates range widely across studies and settings, from approximately 6% to over 30%, depending on disease severity, patient comorbidities, and available healthcare resources.(6–8) Those requiring mechanical ventilatory support face even higher mortality, with three-year rates approaching 50%.(7) Despite the gravity of AECOPD, robust clinical tools for bedside risk stratification specifically designed and validated for hospitalized AECOPD patients remain limited.

The DECAF score, developed by Steer et al., integrates five routinely available clinical and laboratory parameters—dyspnea (eMRCd), eosinopenia, consolidation on chest radiograph, acidemia, and atrial fibrillation (AF)—into a single composite score.(9) Multiple studies have validated DECAF as a strong predictor of in-hospital mortality in AECOPD, outperforming other widely used scoring systems such as CURB-65, APACHE II, CAPS, and BAP-65. A modified version of the DECAF score replaces AF with the frequency of hospital admission in the preceding year, aiming to better capture chronic disease burden and relapse risk.(10)

In India, where the epidemiology of COPD is influenced by high rates of tobacco use, biomass fuel exposure, and limited access to early pulmonary care, context-specific data on AECOPD outcomes and predictors remain sparse. Most available evidence originates from high-income countries, and the applicability of Western-derived scores to Indian patient populations needs validation.

The present study was designed to evaluate the modified DECAF score as a predictor of in-hospital mortality and length of stay in patients admitted with AECOPD at a tertiary care center in Uttar Pradesh, India.

MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional observational analytical study conducted in the Department of General Medicine at Sharda Hospital, School of Medical Sciences and Research (SMS&R), Sharda University, Greater Noida, Uttar Pradesh, India. The study period extended from May 2024 to November 2025 (18 months).

Ethical Considerations

The study protocol was approved by the Institutional Research Ethics Board of SMS&R, Sharda University,

prior to commencement. Written informed consent was obtained from all participating patients or their legally authorized representatives in both English and Hindi. Confidentiality and anonymity were maintained throughout the study. There are no conflicts of interest to declare.

Study Population and Sample Size

Adult patients aged ≥ 25 years admitted to the OPD/IPD of the Department of General Medicine with a clinical diagnosis of AECOPD per GOLD criteria were eligible. Sample size was calculated using Cochran's formula ($N_0 = Z^2pq/e^2$), with $Z = 1.96$, anticipated prevalence $p = 0.074$, and margin of error $e = 10\%$, yielding a minimum sample size of 103.

Inclusion and Exclusion Criteria

Inclusion criteria comprised: (1) age ≥ 25 years and (2) clinical diagnosis of AECOPD per GOLD criteria. Exclusion criteria were: (1) age < 25 years; (2) unwillingness to participate; (3) known heart failure; (4) chronic kidney disease; (5) bronchial asthma; (6) interstitial lung disease; and (7) coronary artery disease or history of myocardial infarction.

Modified DECAF Score

The modified DECAF score was calculated at admission for each enrolled patient and comprises five binary or ordinal components: (1) eMRCd dyspnea grade ($\leq 4 = 0$; $5a = 1$; $5b = 2$); (2) eosinopenia (blood eosinophils $< 0.05 \times 10^9/L = 1$); (3) consolidation on chest radiograph (present = 1); (4) acidemia (arterial pH $< 7.30 = 1$); and (5) frequency of hospitalization in the last year (≥ 1 admission = 1). The total score ranges from 0 to 6 (Table 2).

Data Collection and Investigations

A standardized proforma was used to record demographic information, presenting complaints, comorbidities, and examination findings. Investigations included complete blood count, arterial blood gas (ABG) analysis, chest radiograph (CXR), electrocardiogram, and spirometry or high-resolution CT chest where indicated. Urine output, vital signs, and clinical status were monitored during admission. Outcomes including in-hospital mortality and length of hospital stay were recorded at the time of discharge or death.

Statistical Analysis

Data were analyzed using SPSS version 21 (IBM Corporation, Armonk, NY, USA). Continuous variables are expressed as mean \pm standard deviation (SD); categorical variables as frequencies and percentages. Chi-square tests were used for categorical comparisons. Pearson's correlation coefficient was used for continuous

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variable correlations. Kaplan-Meier survival analysis was performed to assess the relationship between modified DECAF strata and survival time, and differences between strata were evaluated using the log-rank (Mantel-Haenszel) test. Cox proportional hazards modelling was applied for multivariable analysis. A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Demographics and Baseline Characteristics

One hundred and three patients meeting AECOPD inclusion criteria were enrolled. The mean age was 65.9 ± 11.8 years (range 46–98 years), with 64% (66/103) male and 36% (37/103) female. Twenty-eight patients (27.2%) died during hospitalization; the mean age of non-survivors was 67.8 ± 12.6 years versus 65.3 ± 11.6 years among survivors. Age did not significantly differ between groups (Table 1).

Table 1. Baseline Characteristics of the Study Population (N = 103)

Characteristic	Value	p-value*
Age (years), mean ± SD	65.9 ± 11.8	—
Range	46–98	—
Age groups, n (%):		—
40–59 years	—	
60–79 years	—	
≥80 years	—	
Sex, n (%):		—
Male	66 (64%)	—
Female	37 (36%)	—
Mortality (in-hospital), n (%)	28 (27.2%)	—
Mean age – survivors (years)	65.3 ± 11.6	0.352†
Mean age – non-survivors (years)	67.8 ± 12.6	

*p-value for comparison between survivors and non-survivors; †Independent samples t-test. SD, standard deviation.

Modified DECAF Score Components

Table 2 summarizes the components and their scoring. The mean modified DECAF score for the entire cohort

was 2.9 (median = mode = 3). The distribution was: score 1, 17.5% (18/103); score 2, 20.4% (21/103); score 3, 28.2% (29/103); score 4, 20.4% (21/103); score 5, 8.7% (9/103); score 6, 3.9% (4/103).

Table 2. Components of the Modified DECAF Score

Variable	Category	Score
Dyspnea (eMRCD)	≤eMRCD 4 or 5a	0 or 1
	eMRCD 5b	2
Eosinopenia	Blood eosinophils <0.05×10 ⁹ /L	1
Consolidation (CXR)	Present	1
Acidemia	Arterial pH <7.30	1
Frequency of hospitalization (last 1 year)	≥1 admission	1
Total possible score	0–6	

Individual Component Analysis and Association with Outcomes

The analysis of individual DECAF components is presented in Table 3. Dyspnea severity was the strongest individual predictor of in-hospital mortality ($\chi^2 = 15.923$, $df = 2$, $p < 0.001$). Among non-survivors, 48.3% had eMRCD 5A and 41.4% had eMRCD 5B symptoms, compared with 35.1% and 14.9%, respectively, among survivors. Acidemia was present in 55.2% of non-survivors versus 33.8% of survivors, a difference that reached statistical significance ($\chi^2 = 3.978$, $p = 0.046$).

Eosinopenia was present in 63.1% of the cohort and was more frequent among non-survivors (72.4%) than survivors (59.5%), though this difference did not reach statistical significance ($p = 0.220$). Similarly, consolidation was present in 52.4% of patients overall and in 62.1% of non-survivors compared to 48.6% of survivors ($p = 0.220$). Prior hospitalization within the past year was documented in 57.3% of patients and in 65.5% of non-survivors versus 54.1% of survivors, without reaching significance ($p = 0.290$).

Regarding length of hospital stay (LOS), none of the individual DECAF components reached statistical

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significance as predictors of LOS. Mean LOS was 8.8 days for patients with \leq eMRC 4, 10.7 days for eMRC 5A, and 10.3 days for eMRC 5B ($p = 0.537$). Eosinopenic patients had a mean LOS of 10.8 days versus 8.3 days for non-eosinopenic patients ($p = 0.481$). The difference in LOS between patients with and without consolidation was also non-significant (10.7 vs. 8.9 days, $p = 0.650$).

Table 3. Individual DECAF Component Analysis and Association with Outcomes (N = 103)

Variable	Overall n (%)	Survivors n (%)	Non-survivors n (%)	p-value
Dyspnea \leq eMRC 4	40 (38.8%)	37 (50.0%)	3 (10.3%)	<0.001†
Dyspnea eMRC 5A	40 (38.8%)	26 (35.1%)	14 (48.3%)	
Dyspnea eMRC 5B	23 (22.3%)	11 (14.9%)	12 (41.4%)	
Eosinopenia	65 (63.1%)	44 (59.5%)	21 (72.4%)	0.220†
Consolidation on CXR	54 (52.4%)	36 (48.6%)	18 (62.1%)	0.220†
Acidemia (pH <7.30)	41 (39.8%)	25 (33.8%)	16 (55.2%)	0.046†
Prior hospitalization (last year)	59 (57.3%)	40 (54.1%)	19 (65.5%)	0.290†

Bold values indicate statistical significance ($p < 0.05$). †Chi-square test; CXR, chest radiograph; eMRC, extended Medical Research Council dyspnea scale; LOS, length of stay.

Modified DECAF Score and Outcomes

The distribution of the modified DECAF score and its association with outcomes is shown in Table 4. Mean modified DECAF scores were significantly different between survivors (2.54 ± 1.23) and non-survivors (3.86 ± 1.30), with $p < 0.001$. Higher DECAF strata were associated with progressively higher mortality rates, from

0% at score 1 to 75.0% at score 6. Kaplan-Meier survival analysis demonstrated a highly significant association between modified DECAF strata and in-hospital survival (log-rank $\chi^2 = 30.068$, $df = 6$, $p < 0.001$).

Mean LOS increased with higher DECAF scores from 5.9 days at score 1 to 12.7 days at score 5, though the overall association did not reach statistical significance ($p = 0.084$). Cox proportional hazards modelling confirmed the independent predictive value of the modified DECAF score for mortality.

Table 4. Modified DECAF Score Distribution, Mortality, and Length of Hospital Stay (N = 103)

DECAF Score	n (%)	In-hospital Deaths n (%)	Mean LOS (days)	Median Survival (days)
0	1 (1.0%)	1 (100%)	3.0	3.0
1	18 (17.5%)	0 (0%)	5.9	45.0
2	21 (20.4%)	2 (9.5%)	10.0	23.0
3	29 (28.2%)	7 (24.1%)	11.5	30.0
4	21 (20.4%)	11 (52.4%)	9.9	16.0
5	9 (8.7%)	5 (55.6%)	12.7	34.0
6	4 (3.9%)	3 (75.0%)	10.8	12.5
p-value (log-rank)		<0.001	0.084	

*Log-rank test. LOS, length of stay.

Figure Legends

Figure 1. Distribution of modified DECAF scores in the study population. Bar chart displaying frequency (%) of patients across modified DECAF scores 0–6 (mean score 2.9, median 3).

Figure 2. Box and whisker plot showing mean modified DECAF scores of survivors (2.54 ± 1.23) and non-survivors (3.86 ± 1.30). The difference was statistically significant ($p < 0.001$).

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Figure 3. Kaplan-Meier survival curves stratified by modified DECAF score. Higher DECAF strata show significantly shorter survival times (log-rank $p < 0.001$).

Figure 4. Cox proportional hazards model demonstrating the modified DECAF score as an independent predictor of in-hospital mortality.

DISCUSSION

This cross-sectional observational study evaluated the utility of the modified DECAF score in predicting in-hospital mortality and length of stay among 103 patients hospitalized with AECOPD at a tertiary care center in Uttar Pradesh, India. The modified DECAF score demonstrated strong discriminative ability for mortality prediction, with mean scores significantly differing between survivors and non-survivors and Kaplan-Meier analysis confirming progressively worsening outcomes at higher score strata (log-rank $p < 0.001$). These findings align with a growing body of international evidence supporting DECAF-based scoring as a superior bedside prognostic tool in AECOPD.

Mortality and Modified DECAF Score

The in-hospital mortality of 27.2% in this cohort is higher than the 7.7% reported in the original validation study by Steer et al. from the UK.(9) This disparity likely reflects differences in patient populations, healthcare delivery systems, referral patterns, and the exclusion of high-risk conditions in the original cohort. Comparable or higher mortality rates have been reported in studies from low- and middle-income settings. Memon et al. reported 12.6% mortality using the DECAF score in a Pakistani cohort, while Gharraf et al. observed approximately 18% in Egypt.(10,33) The higher mortality in the present study likely reflects the severity of illness at presentation and the burden of comorbid conditions in this regional patient population.

The modified DECAF score performed strongly in our cohort, with mean scores differing by approximately 1.3 points between survivors and non-survivors. Higher DECAF strata corresponded to progressively increased mortality rates (0% at score 1; 75% at score 6; log-rank $p < 0.001$), consistent with its original description and subsequent validations. These observations reaffirm the clinical relevance of the scoring system and its applicability in the Indian setting. Comparative analyses in the literature have demonstrated that DECAF performs as well as, or better than, CURB-65 and BAP-65 for in-hospital mortality prediction in AECOPD.(13,33,34)

Dyspnea Severity

Dyspnea severity was the dominant individual predictor of mortality ($\chi^2 = 15.923$, $p < 0.001$). Among non-survivors, 89.7% had eMRC 5A or 5B symptoms, compared with 50% among survivors. This finding is consistent with the established literature showing that the eMRC dyspnea scale is a robust indicator of COPD prognosis. Hajiro et al. demonstrated that dyspnea classification predicted 5-year survival more accurately than FEV₁-based disease staging.(15) The strong correlation between severe dyspnea and in-hospital death reinforces the importance of immediate dyspnea assessment as a component of early risk stratification, even before laboratory results are available.

Acidemia

Acidemia at admission was significantly associated with in-hospital mortality ($\chi^2 = 3.978$, $p = 0.046$), consistent with established pathophysiological principles and the DECAF literature. Acidemia reflects the failure of alveolar ventilation, respiratory muscle fatigue, and an inability to maintain carbon dioxide homeostasis. Brochard et al. and Confalonieri et al. both demonstrated that pH at admission is a key predictor of non-invasive ventilation (NIV) failure and mortality in AECOPD.(26,27) The recognition of acidosis at admission should prompt immediate consideration of NIV, correction of reversible precipitants, and appropriate care escalation. Interestingly, acidemia did not significantly predict LOS, potentially because patients with severe acidosis who die early create an opposing trend against the longer stays seen in acidotic survivors requiring stabilization.

Eosinopenia, Consolidation, and Prior Hospitalization

Eosinopenia, consolidation on chest radiograph, and prior hospitalization within the preceding year were more prevalent among non-survivors but did not individually reach statistical significance for mortality prediction. These results are in keeping with findings in several previous studies. Ho et al. found that eosinophilic COPD patients had comparable in-hospital mortality to their non-eosinophilic counterparts, while eosinopenia may serve as a surrogate for systemic inflammation rather than a direct mortality driver.(23) Singanayagam et al. noted that the independent prognostic value of consolidation diminishes after adjustment for physiological variables.(36) Prior hospitalization is an established predictor of future exacerbations and long-term mortality but has been less consistently predictive of short-term in-hospital outcomes.(30,31) These findings suggest these variables

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are most informative as part of the composite DECAF model rather than as standalone predictors.

Length of Hospital Stay

The modified DECAF score was not significantly associated with LOS in this study ($p = 0.084$), though a numerical trend toward longer stays at higher DECAF scores was observed. This is consistent with some international data: Steer et al. found that DECAF predicted LOS, but other studies, particularly from resource-limited settings, have found weaker associations. Small sample size and variability in clinical management practices may contribute to this finding. Future multicenter studies with larger sample sizes are needed to clarify the relationship between modified DECAF and LOS in the Indian context.

Comparison with Published Studies

Table 5 presents a summary comparison of the present study with key published series. The higher mortality observed in our cohort is notable, likely reflecting the severity of disease at presentation, limited access to early ambulatory care, and the burden of infectious comorbidities. The modified DECAF score's strong mortality discrimination is consistent across settings, supporting its generalizability.

Table 5. Comparison with Published Studies on DECAF and Modified DECAF Scores in AECOPD

Study (Year)	N	Country	Score Used	Mortality (%)	LOS Significant
Steer et al. (2012)	920	UK	DECAF	7.7%	Yes
Memon et al.	—	Pakistan	DECAF	12.6%	Yes
Gharraf et al. (2020)	100	Egypt	DECAF/Modified	~18%	Comparable
Zidan et al.	50	Egypt	DECAF	20%	—

(2015)					
Present study (2025)	103	India	Modified DECAF	27.2%	No (p=0.084)

Clinical Implications

These findings have several practical implications. First, the modified DECAF score should be calculated routinely at AECOPD admission as a simple triage tool. Second, patients with high eMRCD grades or acidemia should be prioritized for close monitoring, prompt NIV evaluation, and early escalation of care. Third, eosinopenia, while not an independent mortality predictor here, may guide antibiotic and corticosteroid decisions based on exacerbation phenotype. Fourth, patients with higher DECAF scores may require extended hospitalization and post-discharge follow-up planning.

Strengths and Limitations

The strengths of this study include prospective data collection, systematic application of the modified DECAF score, comprehensive clinical and laboratory assessment, and use of validated statistical methods including Kaplan-Meier and Cox hazard analyses. Limitations include the single-center design limiting generalizability, a relatively small sample size reducing power for subgroup analyses, restriction of outcome assessment to the in-hospital period, absence of biomarkers such as CRP or procalcitonin, and potential unmeasured confounders including frailty, medication adherence, and timing of NIV initiation.

CONCLUSION

This study demonstrates that the modified DECAF score is a clinically valuable and readily calculable tool for predicting in-hospital mortality in AECOPD patients. Dyspnea severity (eMRCD grade) and acidemia are the most significant individual predictors of short-term mortality in this setting, while eosinopenia, consolidation, and prior hospitalization contribute meaningfully within the composite score. Although LOS did not significantly correlate with the modified DECAF score, a numerical trend was observed. Routine implementation of the modified DECAF score at admission can facilitate early risk stratification, guide management decisions, and optimize resource allocation. Larger multicenter studies

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are needed to validate these findings, refine cutoff thresholds, and evaluate score-guided management protocols in Indian AECOPD populations.

Declarations

Ethics approval and consent to participate: The study was approved by the Institutional Research Ethics Board of SMS&R, Sharda University. Written informed consent was obtained from all participants or their legally authorized representatives.

Availability of data and materials: The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare no competing interests.

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