

Original Research Article

THE ROLE OF THE DECAF SCORE IN PREDICTING CLINICAL OUTCOMES AMONG PATIENTS ADMITTED WITH ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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ABSTRACT

Background: Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) are a leading cause of hospitalization and death. The DECAF score (Dyspnoea, Eosinopenia, Consolidation, Acidaemia, atrial Fibrillation) is a simple bedside predictor of in-hospital mortality, but data from India remain limited. The objective is to evaluate the admission DECAF score for predicting in-hospital mortality and secondary outcomes (need for non-invasive ventilation [NIV], invasive mechanical ventilation [IMV], intensive care unit [ICU] admission, and length of stay) in patients hospitalized with AECOPD.

Materials and Methods: This prospective observational study enrolled 70 consecutive patients aged ≥ 40 years admitted with AECOPD to a tertiary-care hospital in eastern India between May 2023 and May 2025. The DECAF score was calculated at admission, and patients were classified as low (0–1), moderate (2), or high (3–6) risk and followed until discharge or death. Discrimination was assessed by receiver operating characteristic (ROC) analysis.

Results: The mean age was 62.33 ± 9.98 years, and 64.3% were men. Acidaemia and atrial fibrillation were each present in 54.3% of patients, eosinopenia in 48.6%, consolidation in 47.1%, and severe dyspnoea (eMRCd 5b) in 40.0%. In total, 18.6%, 32.9% and 48.6% were low, moderate, and high risk. NIV was required in 81.4%, ICU admission in 48.6% and IMV in 17.1%; in-hospital mortality was 15.7%. All deaths, all IMV and all ICU admissions occurred in the high-risk group ($p < 0.001$). The area under the ROC curve for mortality was 0.845 (95% CI 0.745–0.928). A cut-off of ≥ 3 gave 100% sensitivity, 61.0% specificity and 100% negative predictive value. Length of stay did not differ across risk groups ($p = 0.984$).

Conclusion: The DECAF score showed good discrimination for in-hospital mortality and closely tracked the need for ventilatory and critical care support. A score below 3 reliably identified low-risk patients, supporting its use for early triage in resource-limited settings.

Keywords: COPD; acute exacerbation; DECAF score; in-hospital mortality; risk stratification; non-invasive ventilation; India

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) affected an estimated 392 million people worldwide

in 2019 and is among the three leading causes of death globally.^[1,2] The burden falls disproportionately on low- and middle-income countries, where exposure to tobacco smoke, biomass

fuel and ambient air pollution is common, and where access to diagnosis and long-term care is often limited.^[1,3] In India, COPD is the leading contributor to the chronic respiratory disease burden, and its impact is compounded by underdiagnosis, delayed presentation, and a scarcity of structured outpatient care.^[3–5]

Acute exacerbations are the defining events in the natural history of COPD. They accelerate the decline in lung function, impair quality of life, and account for most COPD-related healthcare costs.^[6–8] Exacerbations that require hospitalisation carry substantial short- and long-term mortality.^[9–11] Early and accurate identification of patients at high risk of death or deterioration is therefore essential, both to direct ventilatory and critical care resources to those who need them and to identify low-risk patients who may be safely managed with less intensive care.^[8,12] Several general and pneumonia-specific severity scores, such as APACHE II, CURB-65 and BAP-65, have been applied to AECOPD, but each has limitations in this population [13–15]. The DECAF score was developed specifically for patients hospitalised with AECOPD and combines five routinely available admission indices: stable-state dyspnoea on the extended Medical Research Council dyspnoea (eMRCD) scale, eosinopenia, radiological consolidation, acidaemia and atrial fibrillation.^[16,17] In its derivation cohort of 920 patients, DECAF predicted in-hospital mortality with an area under the receiver operating characteristic curve (AUROC) of 0.86.^[16] and it was subsequently validated in internal and external cohorts with AUROCs of 0.83 and 0.82.^[18] Meta-analyses have confirmed good pooled discrimination.^[19,20] and a randomised trial showed that patients with low DECAF scores can be safely treated at home.^[21]

Most validation data, however, come from high-income settings. Patients in India and similar settings differ in age, exposure profile, comorbidity burden and timing of presentation, and the performance of the score in these populations cannot be assumed.^[22–26] We therefore evaluated the ability of the admission DECAF score to predict in-hospital mortality and the need for non-invasive ventilation (NIV), invasive mechanical ventilation (IMV), intensive care unit (ICU) admission and length of hospital stay among patients hospitalised with AECOPD at a tertiary-care centre in eastern India.

MATERIALS AND METHODS

Study design, setting and participants: This prospective observational study was conducted in the Department of Respiratory Medicine of a tertiary-care teaching hospital in eastern India, with support from the Departments of General Medicine and Critical Care, from May 2023 to May 2025. The centre is a referral hospital serving urban and rural populations. Consecutive adult patients admitted with AECOPD were screened. Treatment decisions were

made by the treating team according to standard practice and were not influenced by the study.

Patients were eligible if they were aged ≥ 40 years, had a diagnosis of COPD based on clinical history and available spirometric evidence.^[8,27] and were admitted with an acute exacerbation, defined as an acute worsening of respiratory symptoms beyond normal day-to-day variation requiring a change in regular medication.^[8] Patients with bronchial asthma or asthma–COPD overlap, active pulmonary tuberculosis, interstitial lung disease or bronchiectasis as the primary respiratory diagnosis, acute cardiogenic pulmonary oedema without evidence of AECOPD, terminal illness or malignancy, or those unwilling or unable to provide consent were excluded.

Sample size: The sample size was estimated for a single proportion, $n = Z^2pq/d^2$, assuming an in-hospital mortality of 15% based on published data (reported range 10–20%), a 95% confidence level ($Z = 1.96$) and an absolute precision of 8%.^[16,24,25] This gave a minimum of 77 patients. Within the study period, 70 eligible patients were enrolled, and all were included in the analysis.

Ethical considerations: The study protocol was approved by the Institutional Ethics Committee (approval no. [IEC/ref. no.], dated [date]) before commencement, and the study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants or their legally authorised representatives. No investigations were performed solely for research purposes, and all data were anonymised.

Clinical assessment and data collection: At admission, demographic details, smoking history, biomass fuel exposure, duration of COPD, previous hospitalizations for exacerbation, maintenance inhaled therapy, and comorbidities (including cardiovascular disease, diabetes mellitus, and hypertension) were recorded. Stable-state breathlessness in the period before the current exacerbation was graded on the eMRCD scale.^[17] Routine investigations included a complete blood count with differential, renal and liver function tests, serum electrolytes, and arterial blood gas analysis. Eosinopenia was defined as an absolute eosinophil count $< 0.05 \times 10^9$ /L and acidaemia as an arterial pH < 7.30 .^[16] Chest radiographs were obtained at admission, and consolidation was identified by a qualified radiologist using standard radiographic criteria. A 12-lead electrocardiogram (ECG) was recorded in all patients to identify atrial fibrillation, including atrial flutter.

DECAF score: The DECAF score was calculated from admission data according to the original scoring system (Supplementary Table S1).^[16] One point each was assigned for eMRCD grade 5a, eosinopenia, consolidation, acidaemia, and atrial fibrillation, and two points for eMRCD grade 5b, giving a total score of 0–6. Patients were classified as low risk (0–1), moderate risk (2) or high risk (3–6).^[16,18]

Outcomes and follow-up: The primary outcome was in-hospital mortality. Secondary outcomes were the need for NIV, the need for IMV, ICU admission and length of hospital stay. All patients were followed from admission until discharge or in-hospital death. No patient was lost to follow-up.

Statistical analysis: Data were analysed using Python (version 3.11.6). Normality of continuous variables was assessed, and continuous data were summarised as mean $\pm$ standard deviation (SD) or median (interquartile range, IQR) as appropriate. Categorical data were expressed as frequencies and percentages. Outcomes were compared across DECAF risk categories using the chi-square test or Fisher's exact test for categorical variables, and one-way analysis of variance or the Kruskal–Wallis test for continuous variables. The discriminatory ability of the DECAF score for in-hospital mortality was assessed with ROC analysis, and the AUROC was reported with a 95% confidence interval (CI) obtained by bootstrap resampling (5,000 replicates). Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV), with Wilson 95% CIs, were calculated at DECAF cut-offs of ≥ 2 , ≥ 3 and ≥ 4 , and the optimal cut-off was identified with the Youden index. All tests were two-tailed, and $p < 0.05$ was considered statistically significant. The study is reported in accordance with the STROBE statement.

RESULTS

Baseline characteristics and DECAF profile: Seventy patients were included. The mean age was 62.33 ± 9.98 years, and 45 (64.3%) were men [Table 1; Supplementary Figures S1 and S2]. Baseline functional impairment was marked, with a mean eMRCD grade of 4.1 ± 0.84 ; 28 patients (40.0%) had severe dyspnoea (eMRCD 5b) (Supplementary Figure S3). Of the individual DECAF components, acidemia and atrial fibrillation were each present in 38 patients (54.3%), eosinopenia in 34 (48.6%) and radiological consolidation in 33 (47.1%) [Table 1 & Figure 1].

The DECAF scores ranged from 0 to 5, and the most frequent scores were 2 (32.9%) and 3 (31.4%). No patient scored 6. On risk stratification, 13 patients (18.6%) were low risk, 23 (32.9%) moderate risk and 34 (48.6%) high risk [Table 1; Figure 2; Supplementary Table S2].

Clinical outcomes: NIV was required in 57 patients (81.4%), ICU admission in 34 (48.6%) and IMV in 12 (17.1%). Eleven patients died in the hospital, giving an in-hospital mortality of 15.7% (Table 2; Supplementary Figure S4). The mean length of stay was 8.89 ± 3.29 days (median 8.5, IQR 6–12, range 4–14 days) [Supplementary Figure S5].

Association of DECAF risk category with outcomes: The need for ventilatory and critical care support rose steeply with the DECAF risk category [Table 2; Figure 3]. No low-risk patient required NIV, IMV or ICU admission. All moderate-risk patients (100%) received NIV, but none required IMV or ICU care. In the high-risk group, all patients (100%) required NIV and ICU admission, and 12 (35.3%) required IMV. All 11 deaths occurred in the high-risk group, giving a mortality of 32.4% in this group, compared with 0% in both the low- and moderate-risk groups. Each of these associations was statistically significant (all $p < 0.001$). Mortality increased with each point of the score, from 0% at scores 0–2 to 27.3% at a score of 3, 30.0% at 4 and 100% at 5 (Supplementary Table S2). In contrast, length of stay was similar across the low-, moderate- and high-risk groups (8.92 ± 3.38 , 8.78 ± 3.30 and 8.94 ± 3.36 days, respectively; $p = 0.984$) [Table 2; Supplementary Table S3; Supplementary Figure S6].

Prognostic performance for in-hospital mortality: The DECAF score showed good discrimination for in-hospital mortality, with an AUROC of 0.845 (95% CI 0.745–0.928) [Figure 4]. Diagnostic indices at different cut-offs are shown in [Table 3]. At a cut-off of ≥ 2 , sensitivity and NPV were both 100%, but specificity was only 22.0%. A cut-off of ≥ 3 , which was optimal by the Youden index ($J = 0.61$), retained 100% sensitivity and 100% NPV and improved specificity to 61.0% (PPV 32.4%). At ≥ 4 , specificity rose to 88.1% but sensitivity fell to 45.5%, so six of 11 deaths would have been missed.

Table 1: Baseline demographic and clinical characteristics of the study population (N = 70)

Characteristic	Value
Age, years, mean $\pm$ SD	62.33 $\pm$ 9.98
Sex, n (%)	
Male	45 (64.3)
Female	25 (35.7)
eMRCD dyspnoea grade, mean $\pm$ SD	4.1 $\pm$ 0.84
DECAF components, n (%)	
Severe dyspnoea (eMRCD grade 5b)	28 (40.0)
Eosinopenia ($<0.05 \times 10^9$ /L)	34 (48.6)
Consolidation on chest radiograph	33 (47.1)
Acidaemia (arterial pH <7.30)	38 (54.3)
Atrial fibrillation on ECG	38 (54.3)
Total DECAF score, median (IQR)	2 (2–3)
DECAF score, n (%)	
0	4 (5.7)
1	9 (12.9)
2	23 (32.9)

3	22 (31.4)
4	10 (14.3)
5	2 (2.9)
DECAF risk category, n (%)	
Low (0–1)	13 (18.6)
Moderate (2)	23 (32.9)
High (3–6)	34 (48.6)

SD, standard deviation; IQR, interquartile range; eMRCd, extended Medical Research Council

dyspnoea scale; ECG, electrocardiogram. No patient had a DECAF score of 6.

Table 2: Clinical outcomes in the overall cohort and by DECAF risk category

Outcome	Overall (N = 70)	Low risk (n = 13)	Moderate risk (n = 23)	High risk (n = 34)	p-value
NIV, n (%)	57 (81.4)	0 (0.0)	23 (100.0)	34 (100.0)	<0.001 ^a
IMV, n (%)	12 (17.1)	0 (0.0)	0 (0.0)	12 (35.3)	<0.001 ^a
ICU admission, n (%)	34 (48.6)	0 (0.0)	0 (0.0)	34 (100.0)	<0.001 ^a
In-hospital mortality, n (%)	11 (15.7)	0 (0.0)	0 (0.0)	11 (32.4)	<0.001 ^a
Length of stay, days, mean ± SD	8.89 ± 3.29	8.92 ± 3.38	8.78 ± 3.30	8.94 ± 3.36	0.984 ^b

NIV, non-invasive ventilation; IMV, invasive mechanical ventilation; ICU, intensive care unit; SD, standard deviation. ^a Fisher's exact test (2 × 3). ^b One-

way analysis of variance. The median (IQR) length of stay for the whole cohort was 8.5 (6–12) days.

Table 3: Diagnostic performance of the DECAF score for predicting in-hospital mortality at different cut-offs

Cut-off	TP/FP/FN/TN	Sensitivity, % (95% CI)	Specificity, % (95% CI)	PPV, % (95% CI)	NPV, % (95% CI)
≥2	11/46/0/13	100.0 (74.1–100.0)	22.0 (13.4–34.1)	19.3 (11.1–31.3)	100.0 (77.2–100.0)
≥3 ^a	11/23/0/36	100.0 (74.1–100.0)	61.0 (48.3–72.4)	32.4 (19.1–49.2)	100.0 (90.4–100.0)
≥4	5/7/6/52	45.5 (21.3–72.0)	88.1 (77.5–94.1)	41.7 (19.3–68.0)	89.7 (79.2–95.2)

Area under the ROC curve = 0.845 (95% CI 0.745–0.928; bootstrap, 5,000 replicates). TP, true positive; FP, false positive; FN, false negative; TN, true negative; PPV, positive predictive value; NPV,

negative predictive value; CI, confidence interval (Wilson method). ^a Optimal cut-off by the Youden index (J = 0.61).

Table 4: Comparison of the present study with selected DECAF validation studies

Study	Setting / design	n	In-hospital mortality	AUROC (95% CI)
Steer et al., 2012 [16]	UK; derivation cohort	920	10.4%	0.86 (0.82–0.89)
Echevarria et al., 2016 [18]	UK; internal / external validation	880 / 845	7.7% (overall)	0.83 (0.78–0.87) / 0.82 (0.77–0.87)
Nafae et al., 2015 [23]	Egypt; prospective	200	12.5%	0.83
Sangwan et al., 2017 [24]	India; prospective pilot	50	18.0%	0.905 (0.791–1.000)
Bansal and Gaude, 2020 [25]	India; prospective	228	21.1%	NR
Meena et al., 2023 [26]	India; prospective	100	13.0%	0.933
Hu et al., 2024 [20]	Meta-analysis (22 studies)	—	—	Pooled 0.83 (0.79–0.86)
Present study	India (eastern); prospective	70	15.7%	0.845 (0.745–0.928)

AUROC, area under the receiver operating characteristic curve; NR, not reported in the abstract. Values are as reported in the original publications.

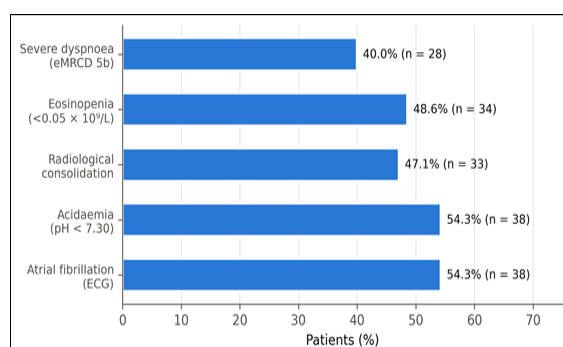


Figure 1: Prevalence of the individual DECAF components at admission (N = 70). eMRCd, extended Medical Research Council dyspnoea scale; ECG, electrocardiogram.

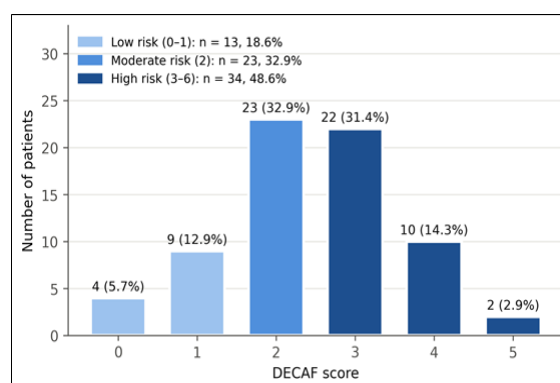


Figure 2: Distribution of admission DECAF scores, shaded by risk category (low 0–1, moderate 2, high 3–6). Numbers above the bars are n (%) of the 70 patients.

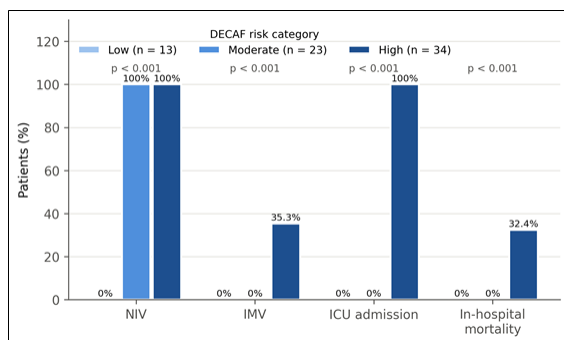


Figure 3: Need for non-invasive ventilation (NIV), invasive mechanical ventilation (IMV), intensive care unit (ICU) admission and in-hospital mortality by DECAF risk category. p-values are from Fisher's exact test.

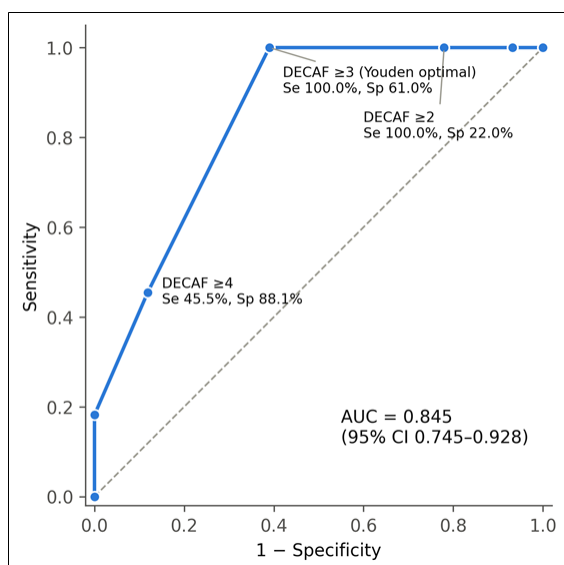


Figure 4: Receiver operating characteristic curve of the DECAF score for predicting in-hospital mortality. The dashed line is the line of no discrimination. Se, sensitivity; Sp, specificity; AUC, area under the curve; CI, confidence interval.

DISCUSSION

In this prospective study of patients hospitalised with AECOPD in eastern India, the admission DECAF score showed good discrimination for in-hospital mortality (AUROC 0.845). A score below 3 identified a group with no in-hospital deaths and no need for invasive ventilation or intensive care. The score also closely tracked the need for NIV, IMV and ICU admission, although it was not associated with length of stay. These findings support the use of DECAF as a simple triage tool in a setting where both disease severity and pressure on critical care resources are high.

The discrimination we observed is consistent with the AUROC of 0.86 reported in the derivation cohort and 0.82–0.83 in the UK validation cohorts.^[16,18] and with the pooled AUC of 0.83 in a recent meta-analysis of 22 studies.^[20] It is also in line with validation studies from India, Egypt and the United Arab Emirates [Table 4].^[21–24,26] The consistency of these results

across very different health systems and patient populations suggests that the five DECAF indices capture core determinants of short-term prognosis in AECOPD, which do not depend strongly on the setting.

Our cohort was younger than the UK derivation cohort (mean age 62.3 vs 73.1 years), and a larger proportion were men (64.3% vs 46.1%).^[16] This profile is typical of Indian series.^[24–26,28] and reflects earlier and heavier exposure to tobacco, occupational dust and biomass smoke in India.^[4,5] The proportion of women (35.7%) is nonetheless substantial and is in keeping with the recognised contribution of household biomass exposure to COPD among Indian women.^[3,29]

Adverse DECAF components were considerably more common in our patients than in the derivation cohort, where around a third had pneumonic exacerbations.^[16] Almost half of our patients had consolidation, more than half had acidemia at admission and 40% had severe baseline dyspnoea. Pneumonic exacerbations are associated with a roughly two-fold higher risk of in-hospital death,^[17,30] and acidemia identifies patients with ventilatory failure who are at high risk of NIV failure and death.^[31,32] The high prevalence of atrial fibrillation (54.3%) is notable. It may reflect a heavy and often undiagnosed cardiovascular comorbidity burden in this population, and it may also partly reflect acute, exacerbation-related arrhythmia.^[33,34] Eosinopenia, found in nearly half of our patients, is an established marker of acute physiological stress and sepsis in critically ill patients.^[35] and an independent predictor of in-hospital death in AECOPD.^[16] Together, these findings suggest that many patients reached our centre late in the course of the exacerbation, as has been described in other resource-limited settings.^[4,28]

The in-hospital mortality of 15.7% is higher than the 7.7–10.4% reported in UK DECAF cohorts.^[16,18] but is similar to the 12.5–21% reported from Egypt and India (Table 4).^[23–26] Similarly, the high rates of NIV use (81.4%) and ICU admission (48.6%) reflect the severity of illness at presentation and the referral nature of the centre. The strong association between the DECAF risk category and IMV and ICU admissions is consistent with earlier reports that higher DECAF scores predict the need for ventilatory support and escalation of care.^[24] This supports the use of the score to anticipate resource needs as well as to predict death.

The score performed particularly well as a rule-out tool. At the Youden-optimal cut-off of ≥3, sensitivity and NPV were both 100%, so no patient with a score of 0–2 died. This agrees with the low mortality reported in low-risk DECAF groups in UK cohorts.^[16,18] In those cohorts, identifying low-risk patients made it possible to treat them safely at home, which was also cost-effective.^[21] In Indian hospitals, where ICU beds are scarce, a low DECAF score could help clinicians to manage patients on the ward and reserve intensive monitoring for higher-risk

patients. However, the specificity at this cut-off was moderate (61.0%) and the PPV only 32.4%. A high score should therefore prompt closer monitoring and early escalation, but it should not be read as a prediction of death in an individual patient. A cut-off of ≥ 4 gave higher specificity (88.1%) at the cost of missing more than half of the deaths, which makes it unsuitable for initial triage.

Length of stay did not differ across risk categories. This is likely to reflect non-clinical determinants of discharge, such as social circumstances, discharge practices and the time needed to wean from NIV, and the fact that patients who died early had shorter stays. Length of stay is therefore a poor surrogate for severity in this setting.

Strengths and limitations: The strengths of this study include its prospective design, consecutive enrolment, calculation of the score at admission before outcomes were known, and complete follow-up to discharge. It also has several limitations. First, it was a single-centre study at a tertiary referral hospital, so the case mix was severe and the results may not apply to secondary care or community settings. Second, the sample size (70 patients, 11 deaths) was slightly below the calculated target, which gives wide confidence intervals for the AUROC and diagnostic indices and prevented multivariable analysis. Third, NIV and ICU admission are partly decisions made by clinicians, and these decisions may have been influenced by the same clinical features that make up the DECAF score. Fourth, pre-admission spirometric severity was not recorded consistently, and outcomes after discharge, such as readmission and 30- or 90-day mortality, were not assessed. Finally, we did not compare DECAF directly with other scores such as BAP-65 or CURB-65.^[14,15] Larger multicentre Indian studies with post-discharge follow-up and head-to-head comparison of scores are needed.^[36]

CONCLUSION

In patients hospitalised with AECOPD at a tertiary-care centre in eastern India, the admission DECAF score showed good discrimination for in-hospital mortality and was strongly associated with the need for NIV, IMV and ICU admission. A score below 3 reliably identified patients at low risk of death. Because it is simple and uses routinely available data, DECAF can be recommended for early risk stratification and triage of patients with AECOPD, especially in resource-limited settings.

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