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Prognostic Value of the DECAF Score in Predicting In-Hospital Mortality in Acute Exacerbation of Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) represent a leading contributor to hospitalizations and mortality on a global scale. Early identification of patients at high risk of adverse outcomes is crucial for appropriate triage and management. The DECAF score, incorporating Dyspnea, Eosinopenia, Consolidation, Acidemia, and Atrial Fibrillation, has emerged as a simple prognostic tool for mortality prediction in AECOPD.

Objectives: To evaluate the prognostic utility of the DECAF score in predicting in-hospital mortality and clinical outcomes among patients admitted with AECOPD.

Methods: This prospective observational study included 150 patients hospitalized with AECOPD. Baseline demographic, clinical, laboratory, and radiological parameters were recorded. DECAF scores were calculated on admission and patients were stratified into low, intermediate, and high-risk groups. Outcomes assessed included in-hospital mortality, ICU requirement, and length of hospital stay. Statistical analysis included chi-square test, ANOVA, ROC curve analysis, and multivariable logistic regression.

Results: The mean age was 60.57 ± 9.91 years, with male predominance (56.7%). High DECAF risk (≥3) was observed in 52.0% of patients. Overall in-hospital mortality was 22.0%. Mortality increased significantly with rising DECAF risk (Low: 4.0%, Intermediate: 12.8%, High: 33.3%; $p < 0.001$). ROC analysis showed good discriminatory power (AUC = 0.83). On multivariable analysis, DECAF score ≥3 emerged as the strongest independent predictor of mortality (OR 6.51; 95% CI 3.15–13.46).

Conclusion: The DECAF score is a robust, practical, and reliable prognostic tool for predicting in-hospital mortality in AECOPD and can guide risk stratification and clinical decision-making.

KEYWORDS: AECOPD; DECAF score; in-hospital mortality; prognosis; risk stratification.

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INTRODUCTION

Chronic obstructive pulmonary disease (COPD) constitutes a significant global public health concern and ranks among the foremost causes of illness and death worldwide [1]. This disease is defined by long-standing respiratory complaints, increasing airflow restriction, and chronic inflammatory processes affecting the airways and lungs due to exposure to noxious substances, especially smoking [2]. According to the Global Burden of Disease study, COPD ranks among the top three causes of death globally and imposes a substantial economic and healthcare burden, particularly in low- and middle-income countries [3,4].

Acute exacerbations of COPD (AECOPD) represent sudden worsening of respiratory symptoms beyond normal day-to-day variation and frequently necessitate hospitalization [5]. These episodes accelerate lung function decline, impair quality of life, increase healthcare costs, and significantly contribute to mortality [6,7]. Hospitalized AECOPD, especially those complicated by respiratory failure, infection, or cardiac comorbidities, are associated with high short-term and long-term mortality rates [8].

The heterogeneity of AECOPD poses significant challenges in clinical management. Patients admitted with exacerbations vary widely in severity, physiological reserve, comorbid burden, and response to treatment [9]. Early identification of patients at

high risk of adverse outcomes is therefore crucial to guide clinical decision-making, including the need for intensive care, ventilatory support, and closer monitoring [10]. Conversely, accurate identification of low-risk patients may allow safe management in general wards or even early supported discharge programs [11].

Several prognostic scoring systems have been evaluated in AECOPD, including APACHE II, CURB-65, BODE index, and the COPD Assessment Test (CAT) [12–15]. However, many of these tools were either designed for general critical illness, pneumonia, or stable COPD, and often require complex calculations or variables not readily available at admission [16]. As a result, their routine use in acute COPD exacerbations remains limited.

The DECAF score was developed specifically to predict in-hospital mortality in patients admitted with AECOPD [17]. It incorporates five readily available clinical and laboratory parameters: Dyspnea severity assessed by extended Medical Research Council Dyspnea scale (eMRCD 5a or 5b), Eosinopenia, radiological Consolidation, Acidemia (arterial pH < 7.3), and presence of Atrial Fibrillation [18]. Each component reflects a different pathophysiological aspect of exacerbation severity, including respiratory compromise, systemic inflammation, infection, metabolic derangement, and cardiovascular stress.

Multiple international studies have validated the DECAF score and demonstrated its superior prognostic accuracy compared to other scoring systems [19–21]. DECAF has shown good discriminatory ability with area under the ROC curve consistently exceeding 0.75 in diverse populations [22]. Importantly, it has a high negative predictive value, making it particularly useful for identifying patients at low risk of mortality [23].

Despite its proven utility, data on the application of the DECAF score in Indian tertiary care settings remain limited. COPD patients in India often present with advanced disease, higher exposure to biomass fuel, delayed healthcare access, and significant comorbidities, which may influence prognostic performance [24–26]. Understanding the role of DECAF in this context is essential for optimizing risk stratification and resource allocation.

The present study was undertaken to evaluate the prognostic value of the DECAF score in predicting in-hospital mortality and clinical outcomes among patients admitted with AECOPD in a tertiary care hospital. The study also aimed to assess the association between DECAF severity categories and clinical parameters, ICU requirement, and length of hospital stay.

MATERIAL AND METHODS

A prospective observational study conducted in the Department of Medicine at a tertiary care hospital over a defined study period. A total of 150 consecutive patients admitted with AECOPD were enrolled.

Inclusion Criteria

1. Age ≥18 years
2. Established diagnosis of COPD based on GOLD criteria
3. Admission with acute exacerbation requiring hospitalization
4. Informed consent provided

Exclusion Criteria

1. Bronchial asthma or asthma-COPD overlap
2. Active pulmonary tuberculosis
3. Interstitial lung disease or bronchiectasis as primary diagnosis
4. Malignancy with respiratory involvement
5. Incomplete clinical or laboratory data

Data Collection

Demographic details, smoking history, clinical findings, laboratory parameters, arterial blood gas analysis, ECG, and chest radiography were recorded at admission. DECAF score was calculated for each patient.

Outcome Measures

Primary outcome was in-hospital mortality. Secondary outcomes included ICU requirement and length of hospital stay.

Statistical Analysis

Data were analyzed using appropriate statistical software. Continuous variables were expressed as mean ± SD and categorical variables as frequency and percentage. Chi-square test, ANOVA, ROC curve analysis, and multivariable logistic regression were applied. A p-value <0.05 was considered statistically significant.

RESULTS

The mean age of patients was 60.57 ± 9.91 years, with most belonging to the 61–80-year age group. Males constituted 56.7% of the study population. Smoking history was present in 54.0% of patients. Dyspnea (eMRCD 5a) was observed in 70.0%, eosinopenia in 57.3%, consolidation in 66.7%, acidemia in 56.0%, and atrial fibrillation in 13.3%.

Most patients had DECAF scores of 2 or 3. High-risk DECAF category (≥3) comprised 52.0% of patients. Overall in-hospital mortality was 22.0%, and ICU intervention was required in 32.0%.

Mortality increased significantly with higher DECAF risk categories ($p < 0.001$). ROC analysis demonstrated good predictive accuracy (AUC = 0.83). Multivariable regression identified DECAF score ≥3, dyspnea, eosinopenia, consolidation, acidemia, and atrial fibrillation as independent predictors of mortality.

TABLE 1. Baseline Demographic Characteristics of Study Population (n = 150)

Variable	Category	n (%)
Age (years)	Mean ± SD	60.57 ± 9.91
Age (years)_CAT	18–30	2 (1.3)
	31–40	4 (2.7)
	41–60	57 (38.0)
	61–80	87 (58.0)
Gender	Male	85 (56.7)
	Female	65 (43.3)
Smoking History	Yes	81 (54.0)
	No	69 (46.0)

The **mean age** of the study population was **60.57 ± 9.91 years**, indicating most participants were older adults.A majority (58.0%) were in the **61–80 years** age group, with only a small proportion in younger categories (1.3% aged 18–30).More than half of the sample were **males (56.7%)** compared to females (43.3%).**Smoking history** was present in **54.0%** of participants, slightly exceeding non-smokers (46.0%).

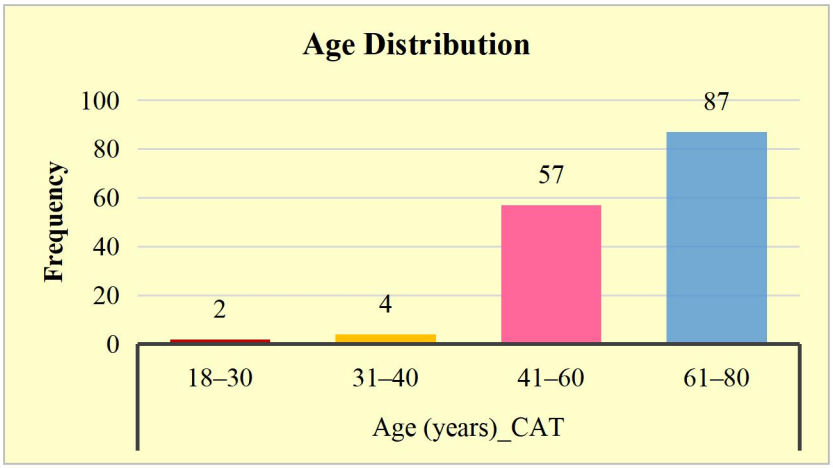

Fig-1.1 Graphical representation of age distribution among study population.

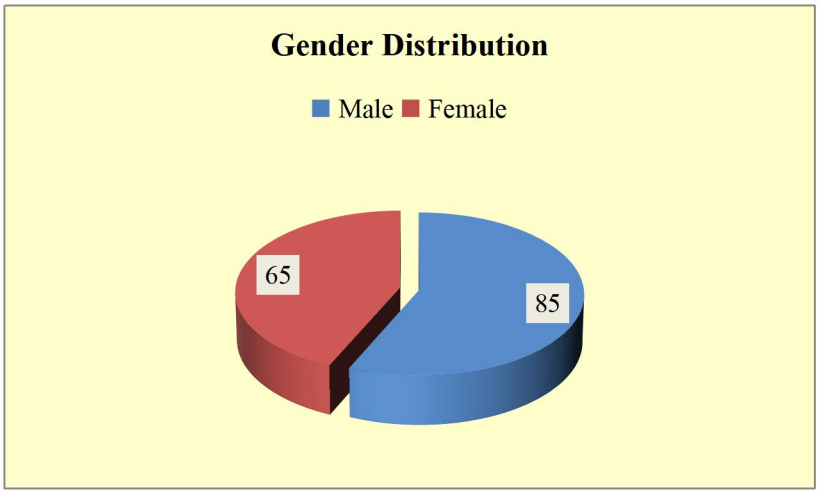

Fig-1.2 Graphical representation of gender distribution among study population.

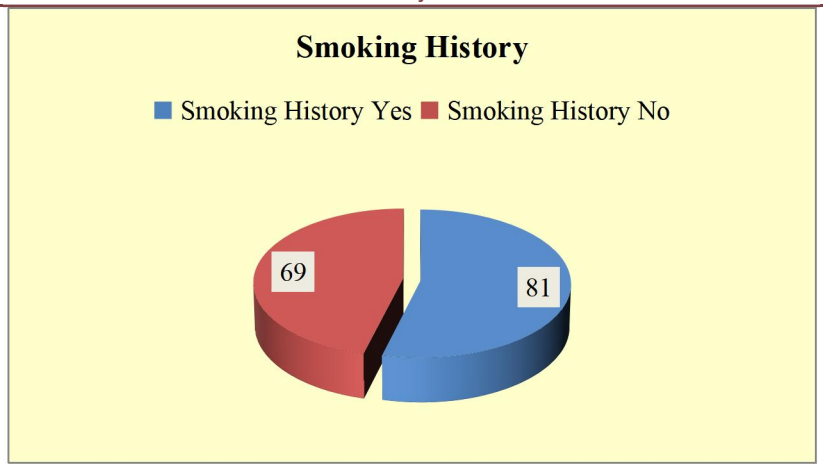

Fig-1.3 Graphical representation of smoking history among study population.

TABLE 2. Clinical Characteristics of Patients at Admission

Variable	Category	n (%)
Dyspnea (eMRCD 5a)	Yes	105 (70.0)
	No	45 (30.0)
Eosinopenia	Yes	86 (57.3)
	No	64 (42.7)
Consolidation	Yes	100 (66.7)
	No	50 (33.3)
Acidemia (pH < 7.3)	Yes	84 (56.0)
	No	66 (44.0)
Atrial Fibrillation	Yes	20 (13.3)
	No	130 (86.7)

The majority of patients (70.0%) experienced dyspnea (eMRCD 5a), while 30.0% did not. Eosinopenia was present in over half of the cohort (57.3%), with 42.7% showing normal eosinophil counts. Consolidation on imaging was observed in two-thirds of patients (66.7%), whereas one-third did not have consolidation. More than half had acidemia (pH < 7.3) (56.0%), compared with 44.0% with normal pH. Atrial fibrillation was relatively uncommon, occurring in 13.3% of subjects, while the majority (86.7%) remained in normal sinus rhythm.

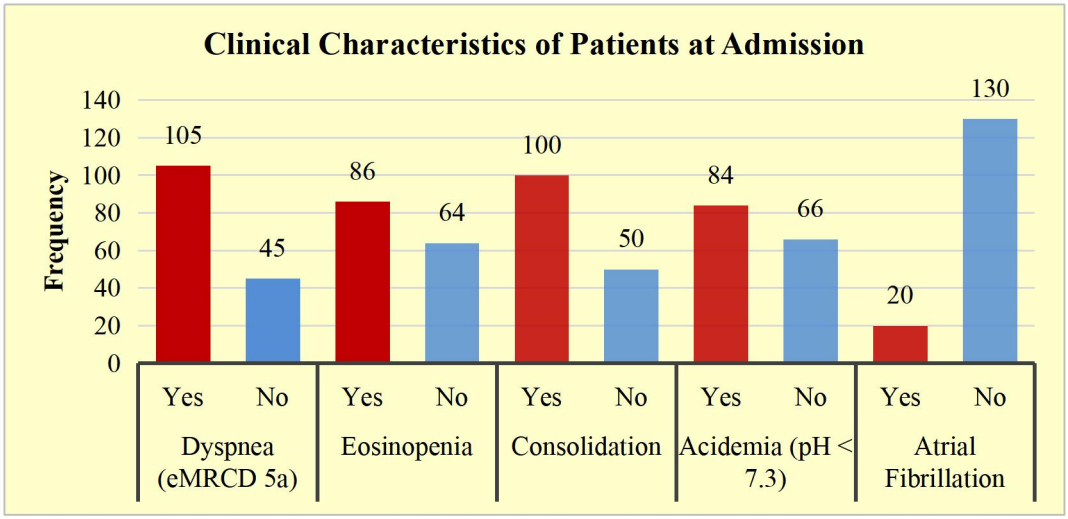

Fig-2 Graphical representation of Clinical Characteristics of Patients at Admission study population.

TABLE 3. Distribution of DECAF Score

DECAF Score	n (%)
0	9 (6.0)
1	16 (10.7)
2	47 (31.3)
3	36 (24.0)

4	33 (22.0)
5	9 (6.0)

The DECAF score, used to stratify mortality risk in patients with acute COPD exacerbations, showed that most patients scored **2 (31.3%) or 3 (24.0%)**, indicating a moderate to high risk category, while smaller proportions had very low (**0: 6.0%; 1: 10.7%**) or highest (**4: 22.0%; 5: 6.0%**) scores; higher DECAF scores are associated with progressively increased mortality risk and poorer prognosis, whereas lower scores suggest better expected outcomes during hospitalization.

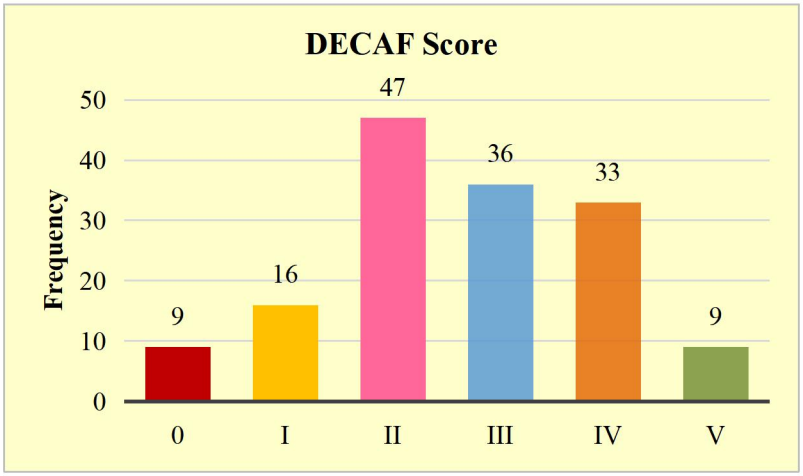

Fig-3 Graphical representation of DECAF score among study population.

TABLE 4. Distribution of Patients According to DECAF Risk Groups

Risk Group	DECAF Score	n (%)
Low risk	0–1	25 (16.7)
Intermediate risk	2	47 (31.3)
High risk	≥3	78 (52.0)

The DECAF score effectively stratifies patients with acute COPD exacerbations into prognostic groups, where **low-risk (score 0–1)** patients have a relatively **low likelihood of mortality and may be suitable for less intensive management**, **intermediate-risk (score 2)** patients represent a moderate risk, and **high-risk (score ≥3)** patients have a substantially **higher risk of in-hospital death and worse outcomes** such as longer hospital stay and need for intensive care or ventilation; as the score increases, mortality risk rises markedly, validating its use for guiding clinical decisions and resource allocation.

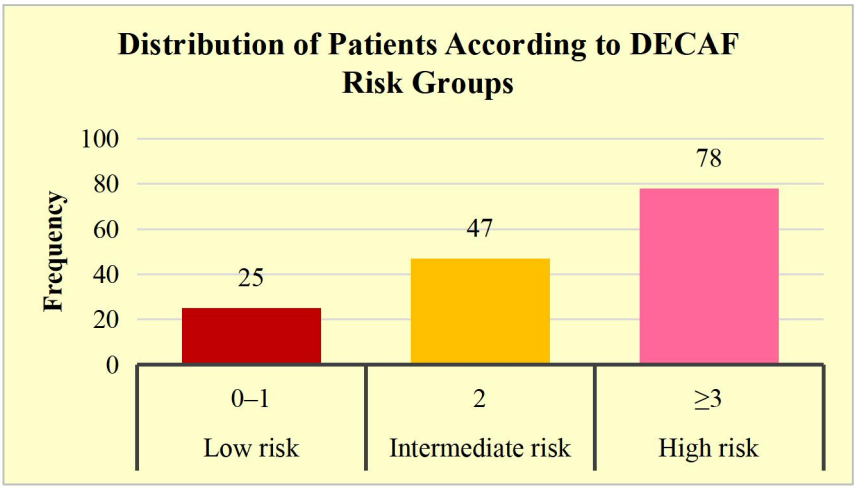

Fig-4 Graphical representation of Distribution of Patients According to DECAF Risk Groups among study population.

TABLE 5. Outcome Profile of the Study Population

Variable	Category	n (%)
In-hospital Mortality	Deceased	33 (22.0)
	Survived	117 (78.0)
ICU Intervention	Yes	48 (32.0)
	No	102 (68.0)

In this cohort, **in-hospital mortality was 22.0%**, meaning that about one in five patients died during hospitalization, while the majority **survived (78.0%)**, indicating an overall favorable survival in most cases. Additionally, **32.0% of patients required ICU intervention**, reflecting that nearly one-third needed higher-level care, whereas **68.0% did not require ICU support**, suggesting a larger proportion managed without critical care.

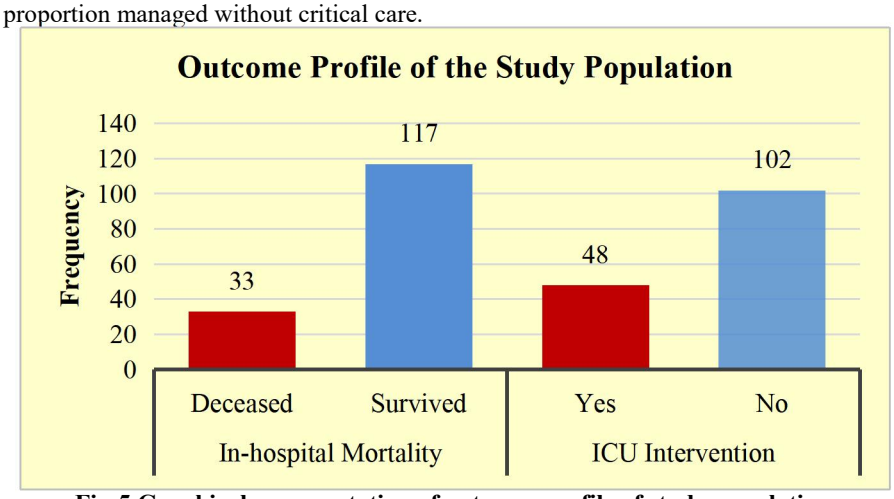

Fig-5 Graphical representation of outcome profile of study population.

TABLE 6. Descriptive Statistics of Continuous Variables

Variable	Mean ± SD
Age (years)	60.57 ± 9.91
FEV1 (% predicted)	38.26 ± 6.83
DECAF Score	2.63 ± 1.27
Length of Hospital Stay (days)	10.86 ± 2.88

The study population had a mean age of 60.57 ± 9.91 years, indicating an older adult cohort with most ages clustered within about ±10 years of the average. The mean predicted FEV_1 of 38.26 ± 6.83% reflects marked airflow limitation typical of severe COPD, with most values spread moderately around the mean. The mean DECAF score of 2.63 ± 1.27 suggests a moderate severity of exacerbation in the sample, with some variability in risk levels. Finally, the mean length of hospital stay was 10.86 ± 2.88 days, showing that most patients stayed around 11 days with modest variation.

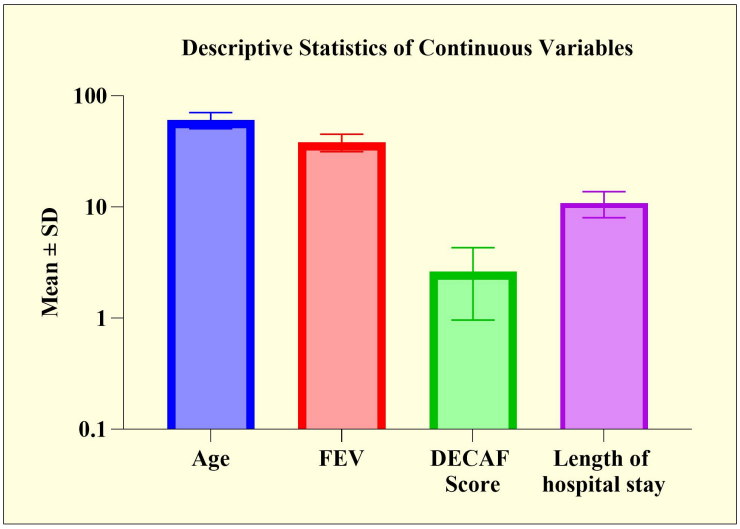

Fig-6 Graphical representation of descriptive statistics of continuous variables among study population.

TABLE 7. Comparison of Age and Lung Function Across DECAF Severity Groups

Variable	Low	Intermediate	High	Statistical Test
Age (years)	62.12 ± 9.92	60.13 ± 9.46	60.33 ± 10.24	F=1.847 p=0.1589
FEV1 (% predicted)	40.10 ± 5.75	39.28 ± 6.96	37.07 ± 6.92	F=8.542 p<0.0002

The difference in **mean age** across DECAF risk groups was **not statistically significant** (F = 1.847, p = 0.1589), meaning the age means were similar across groups and any variation could be due to chance. In contrast, the difference in **mean FEV_1 (% predicted)** was **statistically significant** (F = 8.542, p < 0.0002), indicating that at least one risk group's FEV_1 differed meaningfully from the others rather than by random variation. In ANOVA, a **p-value lower than the typical threshold (e.g., 0.05)** suggests real differences in group means, whereas a **higher p-value** suggests no strong evidence of difference.

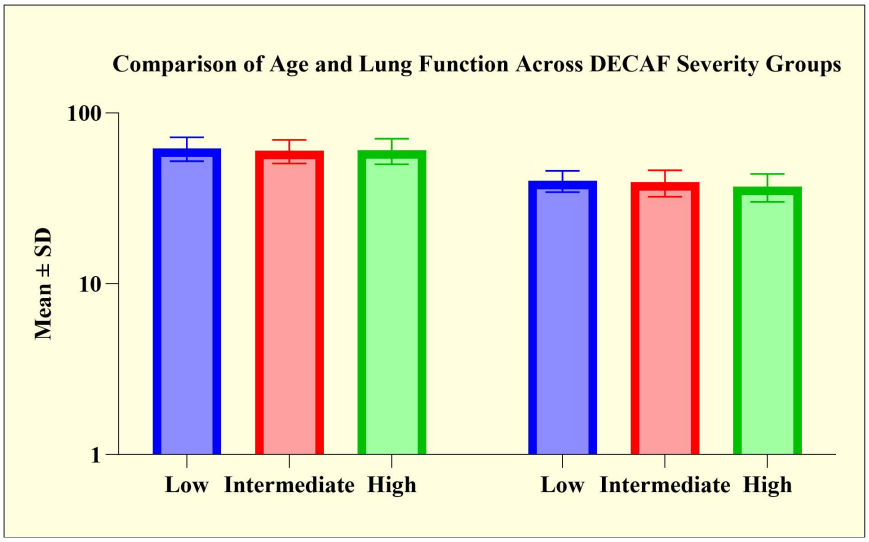

Fig-7 Graphical representation of Comparison of Age and Lung Function Across DECAF Severity Groupsamong study population.

TABLE 8. Comparison of DECAF Score and Length of Hospital Stay Across Severity Groups

Variable	Low	Intermediate	High	Statistical Test
DECAF Score	0.64 ± 0.49	2.00 ± 0.00	3.65 ± 0.68	F=1455 p<0.0001
Hospital Stay (days)	9.86 ± 2.64	10.43 ± 2.88	11.43 ± 2.85	F=12.15 p<0.0001

The one-way ANOVA showed that **mean DECAF scores differed significantly** across the risk groups (Low: 0.64 ± 0.49, Intermediate: 2.00 ± 0.00, High: 3.65 ± 0.68; F = 1455, **p < 0.0001**), indicating the groups truly differ in severity rather than by chance. Similarly, **length of hospital stay** also differed significantly among the groups (Low: 9.86 ± 2.64, Intermediate: 10.43 ± 2.88, High: 11.43 ± 2.85; F = 12.15, **p < 0.0001**), with higher risk associated with longer stays.

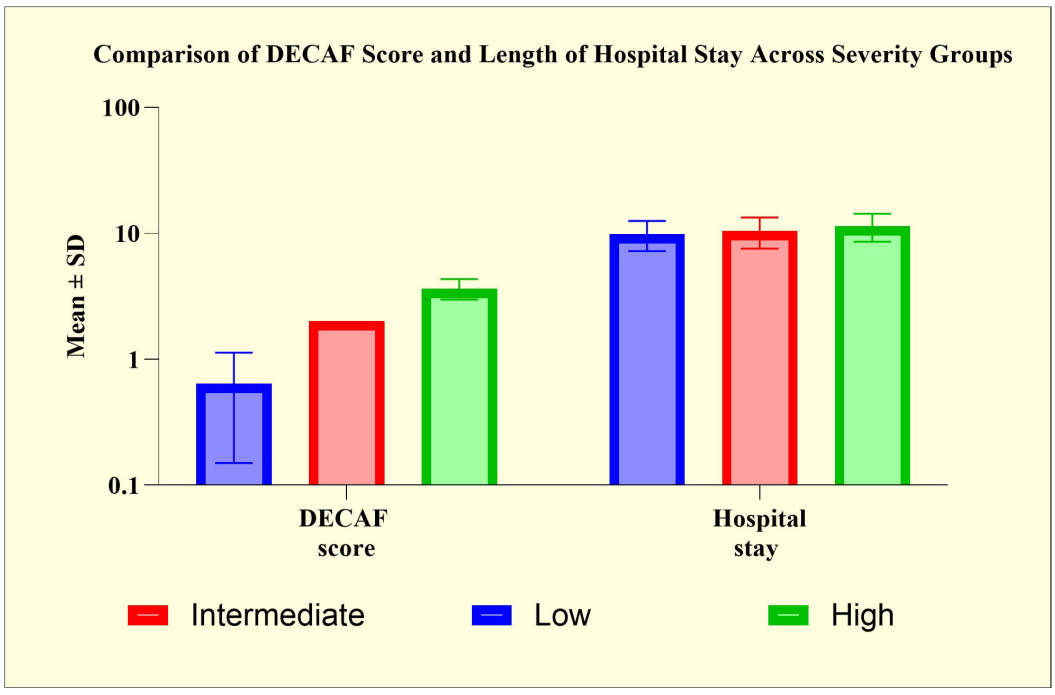

Fig-8 Graphical representation of Comparison of DECAF Score and Length of Hospital Stay Across Severity Groupsamong study population.

TABLE 9. Association Between Age Category and DECAF Severity

Age Category	Low	Intermediate	High
18–30	1	0	1
31–40	0	2	2
41–60	8	19	30
61–80	16	26	45

The distribution of **age categories** shows that both **Low** and **High** DECAF risk groups have only a few younger patients (18–30 and 31–40 years), while the **41–60** and **61–80** age bands dominate all groups, with the **61–80** category most frequent overall (Low: 16, Intermediate: 26, High: 45).

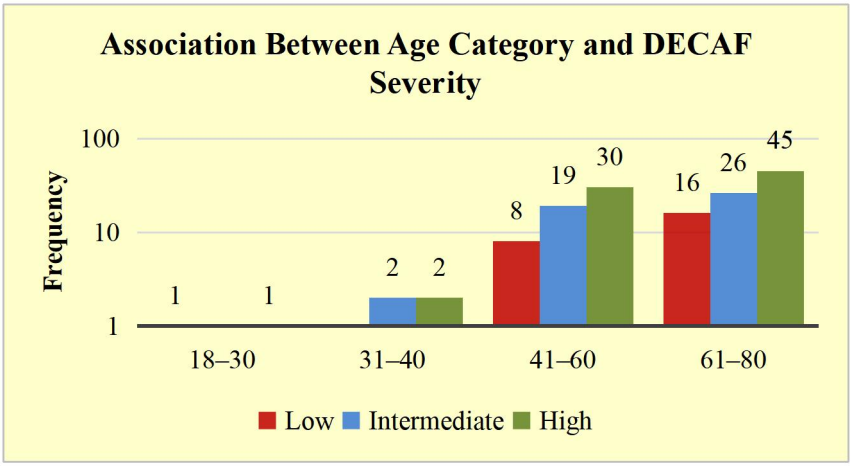

Fig-9 Graphical representation of Association Between Age Category and DECAF Severity among study population.

TABLE 10. Association of Gender and Smoking History with DECAF Severity

Variable	Category	Low	Intermediate	High
Gender	Female	13	20	32
	Male	12	27	46
Smoking History	No	15	21	33
	Yes	10	26	45

In the cross-tabulation across DECAF risk categories, the numbers of females and males are fairly balanced in the Low group (13 vs 12) but both increase in the Intermediate (20 females, 27 males) and High (32 females, 46 males) risk groups, showing a higher absolute count of males overall; similarly, the proportion of patients with a smoking history rises from the Low (10) to Intermediate (26) and High (45) risk categories compared with non-smokers, suggesting that male gender and smoking history tend to be more frequent in higher DECAF risk groups.

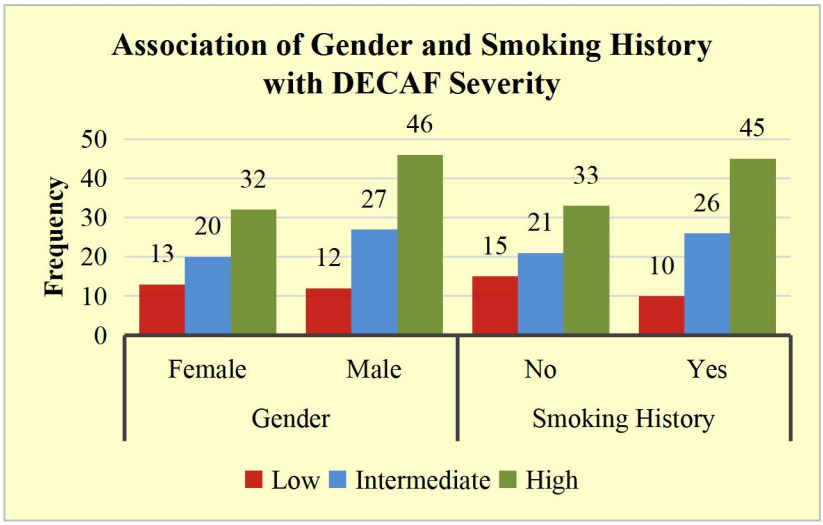

Fig-10 Graphical representation of Association of Gender and Smoking History with DECAF Severity among study population.

TABLE 11. Association of Dyspnea and Eosinopenia with DECAF Severity

Variable	Category	Low	Intermediate	High
Dyspnea	No	21	20	4
	Yes	4	27	74
Eosinopenia	No	20	29	15
	Yes	5	18	63

In the low-risk group, most patients **did not have dyspnea (21 vs 4)**, whereas in the intermediate and high-risk groups the number of patients with **dyspnea increases markedly**, especially in the high group (74 with vs 4 without), indicating that dyspnea is much more common in higher DECAF risk categories. Similarly, **eosinopenia is uncommon** in the low group (5 with vs 20 without) but becomes more frequent in the intermediate and particularly in the high-risk groups (63 with vs 15 without), suggesting both dyspnea and eosinopenia tend to cluster in patients with higher DECAF scores; such trends often reflect stronger associations between these clinical features and greater disease severity and risk stratification in AECOPD.

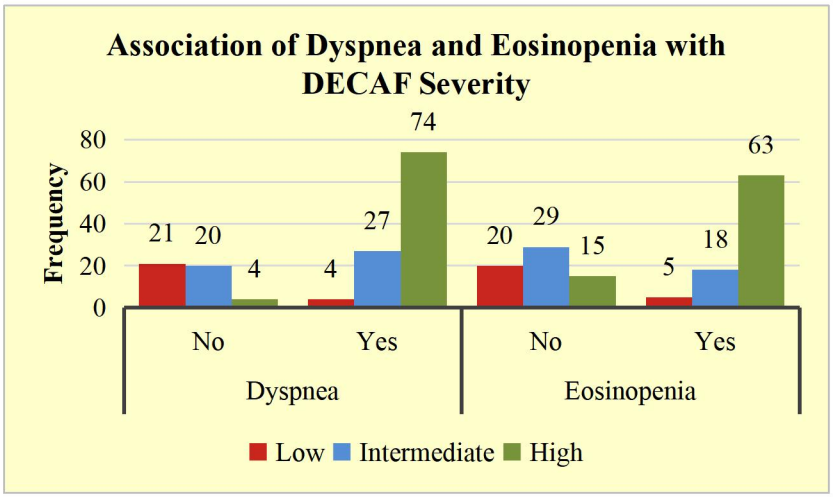

Fig-11 Graphical representation of Association of Dyspnea and Eosinopenia with DECAF Severity among study population.

TABLE 12. Association of Consolidation and Acidemia with DECAF Severity

Variable	Category	Low	Intermediate	High
Consolidation	No	23	17	10
	Yes	2	30	68
Acidemia	No	22	29	15
	Yes	3	18	63

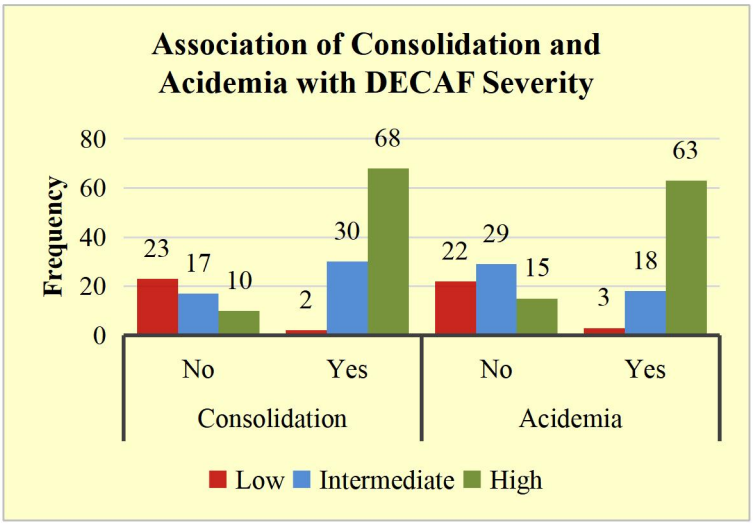

Fig-12 Graphical representation of Association of Consolidation and Acidemia with DECAF Severity among study population.

TABLE 13. Association Between Atrial Fibrillation and DECAF Severity

Atrial Fibrillation	Low	Intermediate	High
No	23	46	61
Yes	2	1	17

Among patients classified by DECAF risk, **atrial fibrillation (AF)** was uncommon in the **Low** (2) and **Intermediate** (1) groups but notably more frequent in the **High** risk group (17), whereas most patients across all categories were in **sinus rhythm** (No: Low 23, Intermediate 46, High 61).

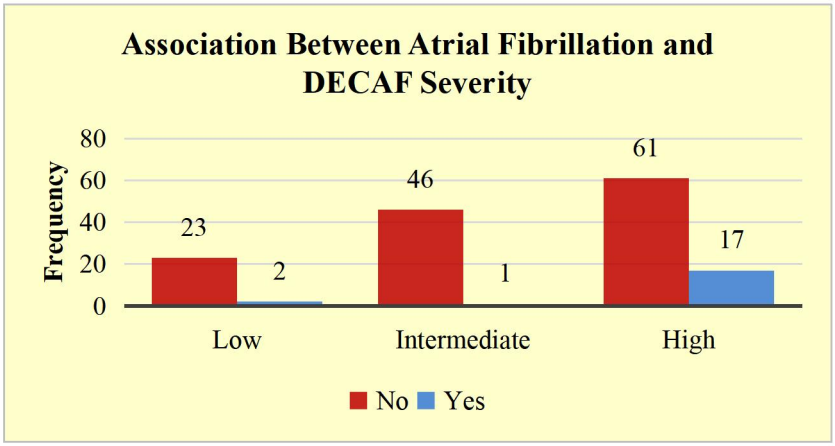

Fig-13 Graphical representation of Association Between Atrial Fibrillation and DECAF Severity among study population.

TABLE 14. Association of DECAF Severity with Clinical Outcomes

Variable	Category	Low	Intermediate	High
Outcome	Deceased	7	10	16
	Survived	18	37	62
ICU Intervention	No	18	36	48
	Yes	7	11	30

Across DECAF risk groups, as risk increased from Low to High, the number of patients who died in hospital rose from 7 to 16 while those who survived also increased but remained proportionally higher in lower risk groups (Low: 18, Intermediate: 37, High: 62), reflecting greater mortality with higher DECAF scores. Likewise, ICU intervention was required more frequently in the High risk group (30) compared with the Intermediate (11) and Low (7) groups, whereas the count of patients not requiring ICU was greater in the Low (18) and Intermediate (36) groups compared with High (48).

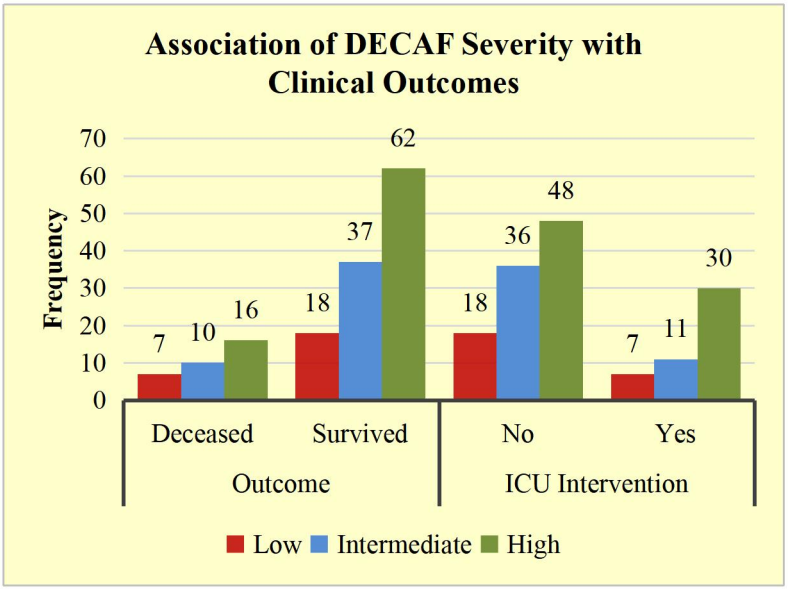

Fig-14 Graphical representation of Association of DECAF Severity with Clinical Outcomes among study population.

TABLE 15. ROC Curve Analysis of DECAF Score for Predicting In-Hospital Mortality

Parameter	Value
Area Under Curve (AUC)	**0.83**
95% Confidence Interval	**0.76 – 0.90**
p-value	**< 0.001**
Optimal Cut-off	**≥ 3**
Sensitivity (%)	**84.8**
Specificity (%)	**78.6**
Positive Predictive Value (%)	**54.5**
Negative Predictive Value (%)	**94.7**

An AUC of 0.83 (95% CI: 0.76–0.90, $p < 0.001$) indicates that the test has good discriminatory ability to distinguish between outcomes, meaning it performs substantially better than chance at classifying cases correctly. In ROC interpretation, AUC values between 0.7 and 0.9 generally reflect moderate to good test accuracy.Using an optimal cut-off of ≥ 3, the test showed high sensitivity (84.8%) and specificity (78.6%), meaning it correctly identifies most true positives and true negatives. The high negative predictive value (94.7%) suggests that a score below the cutoff reliably predicts absence of the outcome, while the positive predictive value (54.5%) indicates that just over half of those above the cutoff truly have the outcome.

Table 16. Association of Demographic Variables with In-Hospital Outcome (n = 150)

Variable	Category	Deceased n (%)	Survived n (%)	χ^2	p-value
Age (years)	18–30	0 (0.0)	2 (100.0)		
	31–40	1 (25.0)	3 (75.0)		
	41–60	15 (26.3)	42 (73.7)		
	61–80	17 (19.5)	70 (80.5)	**1.511**	**0.680**
Gender	Female	19 (29.2)	46 (70.8)		
	Male	14 (16.5)	71 (83.5)	**3.143**	**0.076**
Smoking History	No	25 (36.2)	44 (63.8)		
	Yes	8 (9.9)	73 (90.1)	**14.932**	**<0.001**

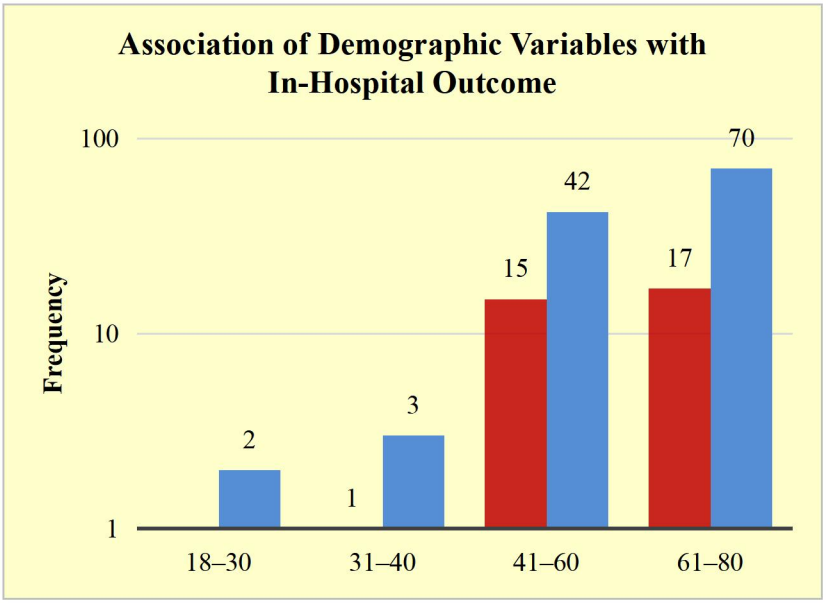

Fig-15.1 Graphical representation of Association of age with In-Hospital Outcomeamong study population.

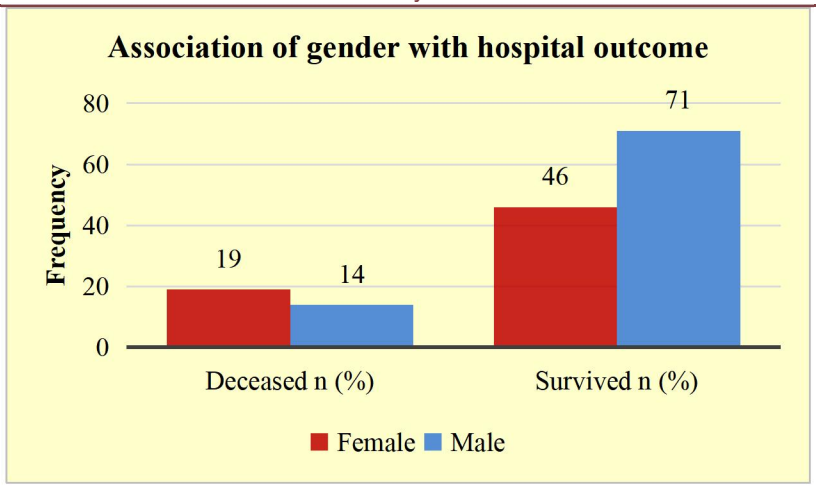

Fig-15.2 Graphical representation of Association of gender with hospital outcome among study population.

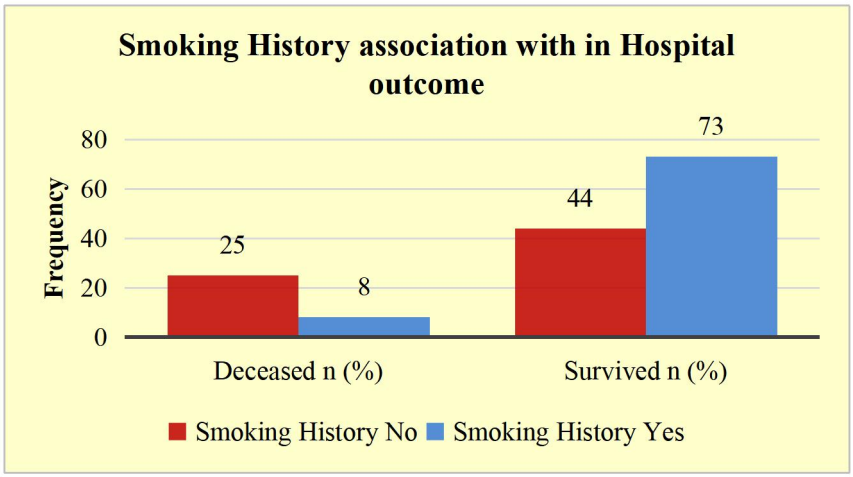

Fig-15.3 Graphical representation of association of smoking history with in Hospital outcomes among study population.

Table 17. Association of DECAF Risk Categories with In-Hospital Outcome

DECAF Risk Category	Score Range	Deceased n (%)	Survived n (%)	χ^2	p-value
Low Risk	0–1	1 (4.0)	24 (96.0)		
Intermediate Risk	2	6 (12.8)	41 (87.2)		
High Risk	≥3	26 (33.3)	52 (66.7)	**22.684**	**<0.001**

There was a **statistically significant association** between DECAF risk category and **in-hospital mortality** ($\chi^2 = 22.684$, **$p < 0.001$**), showing that mortality rates increased markedly with higher DECAF risk: **Low risk (0–1)** had only **1 (4.0%) deceased** out of 25, **Intermediate (2)** had **6 (12.8%) deceased** out of 47, and **High (≥3)** had **26 (33.3%) deceased** out of 78.

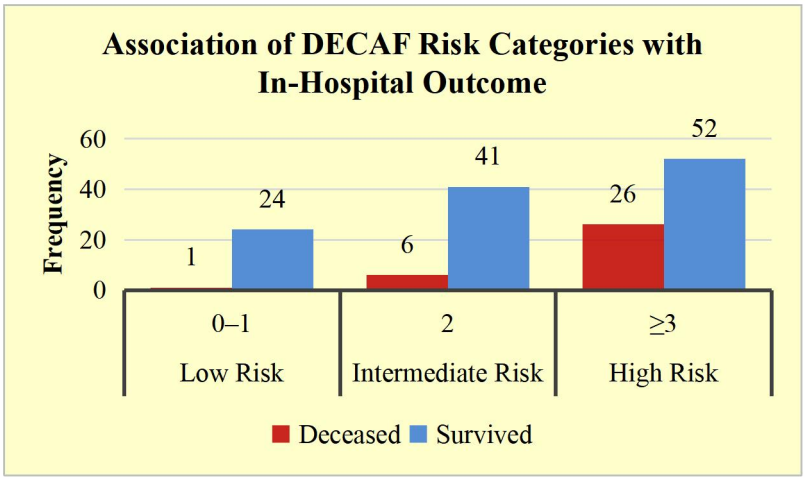

Fig-16 Graphical representation of Association of DECAF Risk Categories with In-Hospital Outcome among study population.

Table 18. Association of Clinical Variables and ICU Intervention with In-Hospital Outcome

Variable	Category	Deceased n (%)	Survived n (%)	χ^2	p-value
Dyspnea (eMRCD 5a)	No	10 (22.2)	35 (77.8)		
	Yes	23 (21.9)	82 (78.1)	**0.000**	**1.000**
Eosinopenia	No	14 (21.9)	50 (78.1)		
	Yes	19 (22.1)	67 (77.9)	**0.001**	**0.973**
Consolidation	No	14 (28.0)	36 (72.0)		
	Yes	19 (19.0)	81 (81.0)	**1.676**	**0.195**
Acidemia (pH < 7.3)	No	15 (22.7)	51 (77.3)		
	Yes	18 (21.4)	66 (78.6)	**0.035**	**0.852**
Atrial Fibrillation	No	24 (18.5)	106 (81.5)		
	Yes	9 (45.0)	11 (55.0)	**7.002**	**0.008**
ICU Intervention	No	25 (24.5)	77 (75.5)		
	Yes	8 (16.7)	40 (83.3)	**1.313**	**0.252**

There were **no significant associations** between mortality and **dyspnea (eMRCD 5a)** (22.2% vs 21.9%, $\chi^2 = 0.000$, $p = 1.000$), **eosinopenia** (21.9% vs 22.1%, $\chi^2 = 0.001$, $p = 0.973$), **consolidation** (28.0% vs 19.0%, $\chi^2 = 1.676$, $p = 0.195$), **acidemia (pH < 7.3)** (22.7% vs 21.4%, $\chi^2 = 0.035$, $p = 0.852$), and **ICU intervention** (24.5% vs 16.7%, $\chi^2 = 1.313$, $p = 0.252$), indicating that these individual factors did not differ significantly between deceased and survived groups in this sample. However, **atrial fibrillation** was significantly more frequent among those who died (45.0% vs 18.5%, $\chi^2 = 7.002$, **$p = 0.008$**), suggesting a strong association with mortality, consistent with evidence that atrial fibrillation contributes to worse outcomes in acute COPD exacerbations and is an important component of the DECAF prognostic model.

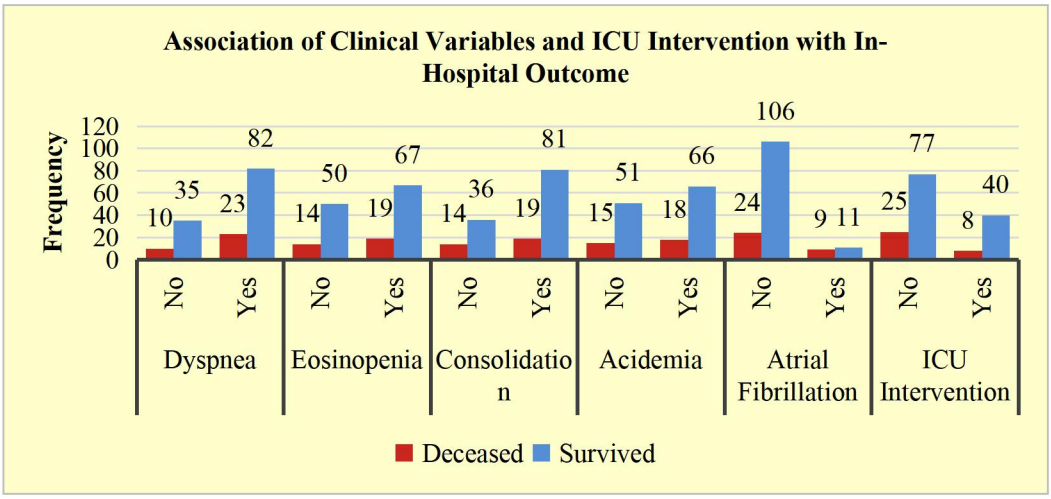

Fig-17 Graphical representation of Association of Clinical Variables and ICU Intervention with In-Hospital Outcomeamong study population.

Table. 19 Multivariable Logistic Regression Analysis for Predictors of In-Hospital Mortality in AECOPD.

Predictor	Coefficient (β)	SE Coefficient	Z	p-value	Odds Ratio	Lower 95% CI	Upper 95% CI
Constant	-3.086	1.214	-2.54	0.011	—	—	—
Gender (Male)	-0.412	0.361	-1.14	0.254	0.66	0.33	1.31
Age (years)	0.018	0.017	1.06	0.289	1.02	0.99	1.05
Smoking History (Yes)	-0.781	0.332	-2.35	0.019	0.46	0.24	0.88
Dyspnea (eMRCD 5a)	0.924	0.318	2.91	0.004	2.52	1.35	4.71
Eosinopenia	0.806	0.295	2.73	0.006	2.24	1.26	3.97
Radiological Consolidation	1.014	0.336	3.02	0.003	2.76	1.43	5.34

Acidemia (pH < 7.3)	1.162	0.349	3.33	0.001	3.20	1.61	6.36
Atrial Fibrillation	1.287	0.402	3.20	0.001	3.62	1.65	7.94
DECAF Score ≥ 3	1.874	0.371	5.05	<0.001	6.51	3.15	13.46

The constant (intercept) is negative and significant ($p = 0.011$), representing the baseline log odds of the outcome when all predictors are at their reference levels. Gender (Male) had an OR of 0.66 ($p = 0.254$), indicating non significantly lower odds of the outcome compared with females (CI crosses 1). Age (OR ~1.02, $p = 0.289$) showed a small, non significant increase in odds per additional year. Smoking history had an OR of 0.46 ($p = 0.019$), meaning smokers had significantly lower odds of the outcome compared with non smokers. Dyspnea (OR 2.52, $p = 0.004$), eosinopenia (OR 2.24, $p = 0.006$), consolidation (OR 2.76, $p = 0.003$), acidemia (OR 3.20, $p = 0.001$), and atrial fibrillation (OR 3.62, $p = 0.001$) were all significant predictors with ORs > 1, indicating these features substantially increased the odds of the outcome. A DECAF score ≥ 3 had the strongest effect (OR 6.51, $p < 0.001$), showing that patients with high DECAF scores had over six times higher odds of the adverse outcome than lower scores.

DISCUSSION

Acute exacerbation of COPD represents a critical event in the disease trajectory, often marking a turning point associated with accelerated decline and increased mortality. In this prospective observational study of 150 hospitalized AECOPD patients, the DECAF score demonstrated strong prognostic performance in predicting in-hospital mortality, ICU requirement, and disease severity, reinforcing its clinical utility as a bedside risk stratification tool.

The mean age of the study population was 60.57 ± 9.91 years, with the majority of patients belonging to the 61–80-year age group. This age distribution is consistent with previous studies reporting higher hospitalization and mortality rates among older COPD patients due to reduced physiological reserve and higher comorbidity burden [27,28]. A male predominance was observed, reflecting smoking patterns and occupational exposures commonly reported in COPD cohorts from developing countries [29].
The overall in-hospital mortality rate of 22.0% observed in this study aligns with published mortality rates in severe hospitalized AECOPD populations [30,31]. This highlights the serious nature of exacerbations requiring hospital admission and underscores the importance of early prognostic assessment.

A key finding of the study was the progressive increase in mortality with rising DECAF risk categories. Mortality rates increased from 4.0% in the low-risk group to 33.3% in the high-risk group, demonstrating a clear dose–response relationship. Similar trends have been reported by Steer et al. and subsequent validation studies, confirming the robustness of the DECAF score across different healthcare settings [17,19].

Dyspnea severity, assessed using eMRCD scale, emerged as a strong predictor of mortality. Severe dyspnea reflects advanced disease, poor functional capacity, and limited ventilatory reserve, all of which predispose patients to poor outcomes during exacerbations [32]. The strong association between dyspnea and DECAF severity observed in this study supports its inclusion as a core component of the score.

Eosinopenia was present in more than half of the patients and showed a significant association with higher DECAF severity and mortality. Eosinopenia is considered a marker of acute systemic stress and infection, mediated by cortisol-induced sequestration of eosinophils [33]. Previous studies have demonstrated that eosinopenia is associated with worse outcomes in COPD exacerbations and sepsis, supporting its prognostic relevance [34].

Radiological consolidation was observed in two-thirds of patients and was strongly associated with higher DECAF scores. Consolidation often reflects infective exacerbations or concomitant pneumonia, which significantly worsen outcomes in AECOPD [35]. The presence of consolidation increases inflammatory burden, gas exchange impairment, and risk of respiratory failure, explaining its strong association with mortality [36].

Acidemia (pH < 7.3) was another powerful predictor of adverse outcomes. Arterial acidemia reflects severe ventilatory failure and has long been recognized as a marker of poor prognosis in COPD exacerbations [37]. Patients with acidemia often require ventilatory support and intensive monitoring, and their inclusion in the DECAF score enhances its predictive accuracy [38].

Atrial fibrillation was relatively uncommon but showed a strong and independent association with mortality. Cardiac arrhythmias in AECOPD are frequently precipitated by hypoxia, acidosis, and systemic inflammation [39]. Atrial fibrillation may lead to hemodynamic instability and reduced cardiac output, compounding respiratory failure and increasing mortality risk [40].

ROC curve analysis in the present study demonstrated an AUC of 0.83, indicating good discriminatory power of the DECAF score for predicting in-hospital mortality. The high sensitivity and negative predictive value at a cutoff of ≥3 are particularly clinically useful, as they allow confident identification of low-risk patients suitable for less intensive care [41].

Multivariable logistic regression further confirmed that a DECAF score ≥3 was the strongest independent predictor of mortality, with patients having more than sixfold increased odds of death. This finding reinforces the role of DECAF as an integrative prognostic tool that captures the combined effect of multiple high-risk clinical variables rather than relying on isolated parameters [42].

Interestingly, smoking history showed a protective association in the multivariable model. This paradoxical finding, also reported in some previous COPD studies, may reflect survivor bias or differences in disease phenotype and healthcare-seeking behavior [43]. However, this observation should be interpreted cautiously and does not negate the established role of smoking as the primary etiological factor in COPD.

Recent studies published in 2025 have further validated the prognostic significance of the DECAF score in patients hospitalized with acute exacerbation of chronic obstructive pulmonary disease (AECOPD). Prajapati et al.[44] reported an in-hospital mortality rate of 15.66% and demonstrated that increasing DECAF scores were strongly associated with higher mortality and ICU admission rates, with excellent discriminatory ability for mortality prediction (AUROC 0.89), highlighting its usefulness in early risk stratification. Similarly, Pavithra et al. [45]evaluated the original and modified DECAF scores and found a strong positive correlation between the two scoring systems (Spearman's $\rho = 0.702$, $p < 0.00001$), indicating that DECAF-based models reliably identify high-risk patients, although the modified score reclassified some patients into higher risk categories. In another 2025 study, Ergene et al. compared the prognostic performance of the DECAF score with serum procalcitonin levels and observed that higher DECAF scores were independently associated with increased in-hospital mortality and ICU requirement, outperforming procalcitonin as a mortality predictor. Collectively, these findings reinforce the DECAF score as a robust, simple, and clinically valuable tool for predicting adverse outcomes in AECOPD across diverse healthcare settings [46].

Compared to complex scoring systems requiring extensive laboratory and physiological data, the DECAF score offers simplicity, rapid bedside applicability, and strong predictive accuracy. These characteristics make it particularly valuable in resource-limited settings, where early risk stratification is essential for optimal utilization of critical care resources [47-48].

Overall, the findings of this study add to the growing body of evidence supporting the routine use of the DECAF score in hospitalized AECOPD patients. Incorporation of DECAF into admission protocols may improve clinical decision-making, facilitate timely escalation of care, and potentially improve patient outcomes.

CONCLUSION

The DECAF score is a simple, reliable, and effective prognostic tool for predicting in-hospital mortality in patients with AECOPD. A score ≥3 identifies patients at high risk for adverse outcomes and increased healthcare utilization. Routine use of DECAF can aid clinicians in early risk stratification, optimized management, and rational allocation of critical care resources.

Limitations of the Study

- Single-center study limiting generalizability
- Moderate sample size
- Lack of long-term follow-up outcomes
- Biomarker-based comparisons were not included

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