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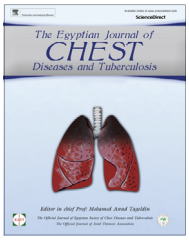

ORIGINAL ARTICLE

Value of the DECAF score in predicting hospital mortality in patients with acute exacerbation of chronic obstructive pulmonary disease admitted to Zagazig University Hospitals, Egypt

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KEYWORDS

DECAF;
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Acute exacerbation;
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Abstract *Background:* Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) are both common and often fatal. Lack of an accurate prognostic tool that can accurately predict inhospital mortality and help clinicians triaging patients to the appropriate level of care is a challenge. Toward this aim, the Dyspnea, Esinopenia, Consolidation, Acidemia and atrial Fibrillation (DECAF) Score is needed to be assessed against other available scores.

Patients and methods: Two hundred patients with primary diagnosis of AECOPD were included. They were subjected to thorough medical history taking, full clinical examination, plain chest X-ray, routine laboratory investigations, ECG, ABG_s analysis, assessment of DECAF Score, Acute Physiology and Chronic Health Evaluation (APACHE II) score, COPD and Asthma Physiology Score (CAPS) and CURB-65 score. Inhospital mortality was recorded.

Results: Twenty-five (12.5%) patients died in hospital. The DECAF Score showed an excellent discrimination for inhospital mortality (AUROC = 0.83) and performed significantly better for the prediction of inhospital mortality than: APACHE II Score (AUROC = 0.68, DECAF vs APACHE II $p = 0.03$); and the COPD and Asthma Physiology Score (CAPS) (AUROC = 0.65, $p = 0.01$). Furthermore, DECAF was a significantly stronger predictor of inhospital mortality than CURB-65 for the subgroup of patients with radiological consolidation (AUROC = 0.87 vs 0.65, $p = 0.02$).

Conclusion: The DECAF Score is a simple and effective clinical tool that can risk stratify hospitalized patients with AECOPD and could therefore help clinicians managing this fatal condition.

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Introduction

Acute exacerbations of chronic obstructive pulmonary disease (AECOPD) are both common and often fatal [1]. In-hospital mortality of 4–30% has been reported in patients with AECOPD requiring hospitaliation [1–3]. A robust clinical prediction tool, developed from a large prospective cohort of unselected admissions, could assist decisions regarding: location of care; early escalation of care; appropriateness for end-of-life care; and suitability for early supported hospital discharge and therefore could help to reduce morbidity and mortality and direct the most efficient use of resources [1].

In stable COPD, prognostic indices have been thoroughly investigated and tools predicting mortality risk, such as the BODE Score, are well established [4]. However, prognostic research in exacerbations requiring hospitalization has been limited, and there appears to be little common ground between predictors of mortality in stable disease and during AECOPD [5]. Moreover, none of the prognostic scores developed in stable disease have been tested on hospitalized patients, and most require clinical measurements not routinely available at hospital admission. Of the prognostic scores proposed for use in AECOPD requiring hospitalization, most were derived in highly selected, [6–9] rather than unselected, patients [10,11]. AECOPD are often complicated by radiographic consolidation especially in patients receiving ventilatory support [12]. Currently, in patients hospitalized with AECOPD complicated by consolidation, the CURB-65 (Confusion, Urea, Respiratory Rate, Blood pressure, Age > 65) community acquired pneumonia prognostic score [13] is often used to risk assess and guide antibiotic therapy [14]. However, it has been recently shown that the use of CURB-65 in patients with AECOPD and consolidation is suboptimal [15]. Hence, the Dyspnea, Esinopenia, Consolidation, Acidaemia and atrial Fibrillation (DECAF) score was first introduced by Steer et al. [1]. The tool is simple to administer at the bedside, using indices routinely available on admission. The value of the DECAF Score as a clinical prediction tool that can accurately risk stratify all patients with AECOPD is needed to be assessed against other available scoring systems in our locality.

Patients and methods

Patients

This study was carried out at the Respiratory ICU and Chest Department, Zagazig University Hospitals during the period from October 2010 to April 2013. It included 200 AECOPD patients with a mean age of 69.3 ± 8; they were 102 males and 98 females. Criteria for exclusion were: previous inclusion in the study; malignancy; or a primary reason for admission other than AECOPD.

Methods

All studied patients were subjected to the following:

1- Thorough medical history.
2- Full clinical examination (general and local examination).
3- Plain Chest X- ray (postero-anterior or antero-posterior view according to circumstances).
4- Routine laboratory investigations:
   - Complete blood picture (CBC)
   - Liver functions
   - Kidney functions
   - Serum electrolytes (Na, K, Cl)
5- Arterial blood gases' analysis (ABGs).
6- Diagnosis of AECOPD according to Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria [16] supported by spirometric evidence of airflow obstruction (forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) < 0.70) when clinically stable; with clinical criteria of exacerbation including increased dyspnea, increased sputum volume or sputum purulence.
7- Stable-state dyspnea was assessed using the extended Medical Research Council (MRC) dyspnea (eMRCD) Score [15]; this subdivides patients too breathless to leave the house unaided (traditional MRCD5) into those able independently to manage washing and/or dressing (eMRCD5a) and those requiring assistance with both (eMRCD5b).
8- Assessment of Acute Physiology And Chronic Health Evaluation II (APACHE II) Score [17], COPD And Asthma Physiology Score (CAPS) [7] and CURB-65 Score [13].
9- Assessment of the DECAF Score according to Steer et al. [1].

Variable	Score
-Dyspnea	
eMRCD 5a	1
eMRCD 5b	2
-Esinopenia ($<0.05\times10^3$/dl)	1
-Consolidation	1
-Acidemia (pH <7.30)	1
-Atrial Fibrillation	1
Total DECAF Score	6

DECAF, Dyspnea, Esinopenia, Consolidation, Acidaemia and atrial Fibrillation; eMRCD, extended MRC dyspnea.

10- The presence of atrial fibrillation was confirmed by ECG at the time of hospital admission.
11- Assessment of outcome which was either inhospital death or discharge.

Statistical analysis

Statistical analysis was performed with the SPSS statistical software package version 19 (SPSS Inc., Chicago, IL, USA). *P*-value < 0.05 was considered significant.

Results

Two hundred patients with AECOPD were recruited with a mean age of 69.3 ± 8; they were 102 males and 98 females. In total, 25 (12.5%) patients died during their hospital stay.

Table 1 shows the socio-demographic data of all studied patients. There were statistically significant differences between survivors and non survivors regarding age, being housebound, FEV_1, eMRCD and being on long term oxygen therapy.

Table 2 shows that the presence of cerebrovascular disease, atrial fibrillation and renal comorbidity was statistically significantly different in non survivors when compared with survivors.

Table 3 shows statistically significant differences between survivors and those who died in hospital as regards BMI, respiratory rate, diastolic blood pressure and the presence of radiological consolidation, purulent sputum, ineffective cough and lower limb edema.

Table 4 shows comparison between survivors and non survivors as regards admission arterial blood gases' values. There were statistically significant differences between the two studied groups regarding pH and p_aCO_2.

Table 5 shows statistically significant differences between the two studied groups as regards serum levels of potassium, urea, creatinine, glucose, CRP and albumin. Also, hemoglobin level and esinophil count were significantly lower in non survivors than survivors.

Table 6 shows predictors of inhospital mortality in hospitalized patients with AECOPD. Extended Medical Research Council Dyspnea score 5b (eMRCD5b) was the most powerful predictor while, BMI was the least powerful one. Parameters were ordered according to the regression coefficient (B).

Table 7 shows inhospital mortality rates according to each grade of the DECAF score with relevant sensitivity and specificity: DECAF 0–1 ('low risk'; inhospital mortality = 3.37%); DECAF 2 ('moderate risk'; mortality = 7.7%); and DECAF 3–6 ('high risk'; mortality = 37%).

Table 8 & Fig. 1 show comparison between area under receiver operator characteristic (ROC) curves (AUROC) of DECAF (AUROC = 0.83), APACHE II (AUROC = 0.68) and CAPS (AUROC = 0.65) scores for predicting inhospital mortality. There were statistically significant differences when comparing DECAF vs APACHE II ($p = 0.03$) and DECAF vs CAPS ($p = 0.01$).

Table 1 Socio-demographic data of the studied patients.

Parameter	Survivors (no = 175)	Non survivors (no = 25)	*P*-Value
Age in years	68.5 ± 8	75.3 ± 10	<0.001
Sex: M/F	90/85	12/13	0.83
Housebound, No.(%)	53 (30.3%)	20 (80%)	<0.001
No. of AECOPD in previous year	2.75 ± 1	3 ± 0.7	0.29
FEV1 (% of predicted)	45 ± 14.3	38 ± 11.6	0.02
eMRCD, median (range)	4 (3–5a)	5a (5a–5b)	<0.001
Long-term oxygen therapy, no (%)	20 (11.4)	9 (36%)	0.003
Core-pulmonale, No. (%)	18 (10.3%)	3 (12%)	0.73
Home nebulized therapy, no (%)	25 (14.3%)	4 (16%)	0.77

Table 2 Comorbidity in the studied patients.

Comorbidity	Survivors (no = 175)	Non survivors (no = 25)	*P*-Value
Cerebrovascular disease	20 (11.3%)	9 (36%)	0.003
Ischemic heart disease	54 (30.9%)	7 (28%)	1.00
Hypertension	65 (37.1%)	10 (40%)	0.83
Diabetes	26 (14.9%)	4 (16%)	0.77
Atria fibrillation	15 (8.6%)	10 (40%)	<0.001
Renal comorbidity	9 (5.1%)	5 (20%)	<0.001

Table 3 Comparison between the studied groups as regards admission clinical and radiological data.

Parameter	Survivors (no = 175)	Non survivors (no = 25)	*P*-Value
Purulent sputum	105 (60%)	9 (36%)	0.03
Ineffective cough	20 (11.4%)	10 (40%)	<0.001
Lower limb edema	45 (25.7%)	9 (36%)	0.34
Acute confusion	18 (10.3%)	10 (40%)	<0.001
Herat rate/min	100.3 ± 21.2	102.5 ± 22.1	0.63
Systolic blood pressure (mm Hg)	140 ± 28	136.2 ± 29.5	0.53
Diastolic blood pressure (mm Hg)	77.5 ± 15.7	70.3 ± 17.9	0.036
Respiratory rate/min	24.5 ± 5.7	28.1 ± 6.6	0.004
Temperature (°c)	37 ± 0.6	36.8 ± 0.4	0.11
BMI (kg/m^2)	25 ± 5.9	21 ± 6	0.001
Radiological consolidation	55(31.4%)	17(68%)	<0.001

Table 4 Comparison between the studied groups as regards admission arterial blood gas values.

Parameter	Survivors (no = 175)	Non survivors (no = 25)	*P*-Value
pH	7.42 ± 0.04	7.34 ± 0.09	<0.001
p_aO_2 (mm Hg)	65.3 ± 9.5	63.1 ± 12.3	0.30
p_aCO_2 (mm Hg)	43.5 ± 8.3	49 ± 16.3	0.008
HCO_3 (mEq/L)	28 ± 5.9	29 ± 6.4	0.21
Oxygen saturation (%)	92 ± 5	92 ± 6	0.99

Table 5 Comparison between the studied groups as regards admission laboratory investigations.

Parameter	Survivors (no = 175)	Non survivors (no = 25)	*P*-value
Sodium (mEq/l)	136.4 ± 4.6	136.8 ± 4.8	0.69
Potassium (mEq/l)	4.3 ± 0.5	4.6 ± 0.6	0.007
Chloride (mEq/l)	98.6 ± 8.9	98 ± 6.2	0.75
Urea (mmol/l)	6.5 ± 2.1	10.3 ± 3.8	<0.001
Creatinine (mg/dl)	0.92 ± 0.21	1.04 ± 0.43	0.02
Albumin (g/dl)	3.9 ± 0.45	3.5 ± 0.52	<0.001
Glucose (mg/dl)	126 ± 18	135 ± 25	0.03
Hemoglobin (g/dl)	13.8 ± 1.8	12.6 ± 2.2	0.003
CRP (mg/dl)	5.6 ± 3.6	8.8 ± 5.8	<0.001
White cell count ($\times 10^3$/dl)	11.9 ± 3.6	13.3 ± 4.2	0.08
Neutrophil count ($\times 10^3$/dl)	9.2 ± 3.4	11.3 ± 4.1	0.005
Esinophil count ($\times 10^3$/dl)	0.1 ± 0.1	0.03 ± 0.03	<0.001

Fig. 2 shows a statistically significant difference between AUROC of DECAF and CURB-65 scores (0.87 vs 0.65; $p = 0.02$) in a subgroup of patients with consolidation.

Discussion

Despite improvements in care, death during hospitalization for AECOPD is a challenging issue. In the UK in 2008, almost 1 in 12 people admitted with a COPD exacerbation died during their hospital stay [18]. In the U.S. in 1996, about 1 in 40 people hospitalized with COPD exacerbations died in-hospital [2]. This could be reflecting a different threshold for hospital admission and care between the countries.

This study showed a mortality rate of 12.5% among patients hospitalized with AECOPD, a result that lies within the range of 4–30% that has been reported in patients with AECOPD requiring hospitalization [1–3]. The variability in published mortality rates for patients with COPD admitted for acute respiratory failure suggests that significant heterogeneity exists within this population. It is likely that differences in patient characteristics, more than in quality of care, account for much of the variability. The relatively small size of many of the previous studies makes them more susceptible to these considerations [19].

Identifying upon admission those at higher risk of dying during their hospitalization could be useful for triaging patients to the appropriate level of care, deciding aggressiveness of therapies, and timing safe discharges. So, Steer et al. [1] derived the DECAF Score—**D**yspnea, **E**sinopenia, **C**onsolidation, **A**cidemia, and atrial **F**ibrillation—tying to accurately predict inhospital mortality for patients with AECOPD [20]. The DECAF Score is a simple prognostic tool, incorporating clinical and laboratory information available routinely on admission in patients hospitalized with AECOPD [1].

In accordance with the results obtained by Son et al. [21] and Steer et al. [1] the current work illustrated that the DECAF Score showed an excellent discrimination for inhospital mortality (AUROC = 0.83). Furthermore, the DECAF Score performed significantly better for the prediction of inhospital mortality than: the Acute Physiology and Chronic Health Evaluation (APACHE) II prognostic index (AUROC = 0.68, DECAF vs APACHE II $p = 0.03$); and the COPD and Asthma Physiology Score (CAPS) (AUROC = 0.65, $p = 0.01$); which have been proposed as useful predictive instruments in AECOPD (figure 1and Table 8).

In this study, DECAF was a significantly stronger predictor of inhospital mortality than CURB-65 for a subgroup of patients with radiological consolidation (AUROC = 0.87 vs 0.65, $p = 0.02$) (Fig. 2). Similar results were previously obtained by Steer et al. [1].

Roche et al. [11] derived a predictive score from 794 patients attending an emergency department with AECOPD.

Table 6 Predictors of hospital mortality in patients hospitalized with AECOPD.

Variable value	B	Odds ratio (95% CI)	*p*
eMRCD5a	1.68	5.3 (1.9–14.9)	<0.001
eMRCD5b	2.04	7.8 (2.7–22.3)	<0.001
Consolidation	1.53	4.6 (1.9–11.4)	<0.001
pH < 7.30	1.47	4.3 (1.8–10.3)	<0.001
Esinopenia < 0.05×10^3/dl	1.3	3.7 (1.6–8.9)	0.003
AF 0.008	1.16	3.2 (1.4–7.5)	0.008
Albumin < 3.5 g/dl	1.02	2.8 (1.2–6.5)	0.02
Ineffective cough	0.93	2.5 (1.0–6.0)	0.033
Cerebrovascular disease	0.89	2.4 (1.0–5.7)	0.039
BMI < 18.5 kg/m^2	0.86	2.4 (1.0–5.5)	0.047

Table 7 DECAF score and hospital mortality.

DECAF Score	No	Hospital mortality, no (%)	Sensitivity	Specificity
0	44	1 (2.8%)	1	0
1	45	2 (4.8%)	0.96	0.25
2	65	5 (7.7%)	0.88	0.49
3	30	7 (23.3)	0.68	0.83
4	12	7 (58.3%)	0.40	0.97
5	4	3 (75%)	0.12	0.99
6	0	0	–	–

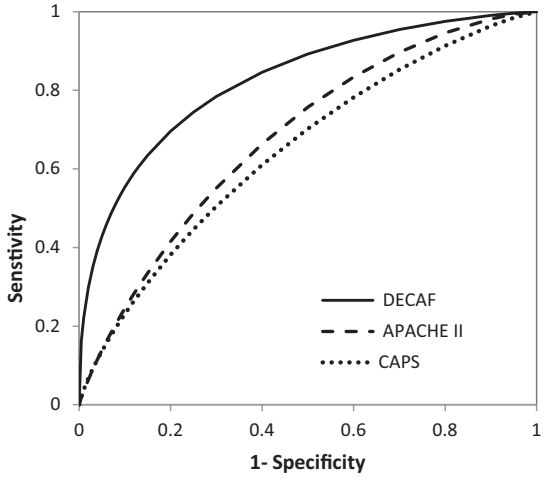
Table 8 Comparison of AUROC between DECAF, APACHE II and CAPS scoring systems.

Score	AUROC	*P* value in comparison with DECAF
DECAF	0.83	–
APACHE II	0.68	0.03
CAPS	0.65	0.01

Their prognostic tool showed good discrimination for inhospital mortality (AUROC = 0.79) but, it included subjectively assessed signs of clinical severity. The DECAF Score performed more strongly in this study (AUROC = 0.83) compared to the score described by Roche et al. moreover, the parameters included in the DECAF Score are objective with little potential for variable interpretation.

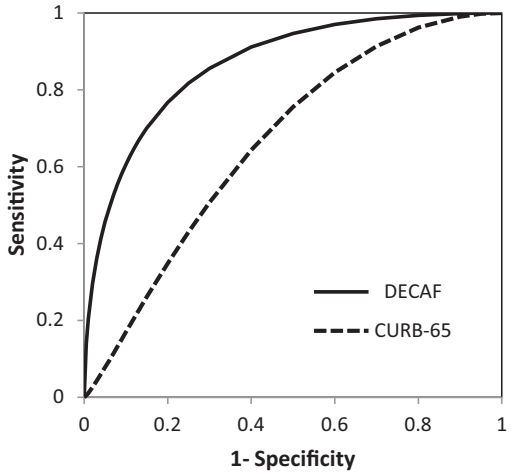
More than one third of our patients showed co existing radiological consolidation with a statistically significant difference when comparing survivors with non survivors ($p < 0.001$) (Table 3). Moreover, consolidation was one of the strongest predictors of inhospital mortality in this study (Table 6) ($p < 0.001$). So, it was logic to be incorporated as a component of the DECAF Score.

AECOPD are often complicated by radiographic consolidation [12,18]. Practice varies over whether such individuals



DECAF: Dyspnea, Esinopenia, Consolidation, Acidemia, atrial Fibrillation; APACHE II: Acute Physiology and Chronic Health Evaluation II; CAPS: COPD and Asthma Physiology Score

Figure 1 Receiver operator characteristic curves of DECAF, APACHE II and CAPS Scores for inhospital mortality in all patients.



DECAF: Dyspnea, Esinopenia, Consolidation, Acidemia, atrial Fibrillation; CURB: Confusion, Urea, Respiratory rate, Blood pressure.

Figure 2 Receiver operator characteristic curves of DECAF and CURB-65 Scores for inhospital mortality in patients with consolidation.

are included under the diagnosis of AECOPD, but most studies of prognosis in AECOPD requiring hospitalization have not excluded patients with complicating consolidation [6,7,10,11,22]. Furthermore, CT scanning in AECOPD often shows consolidation not visible by plain radiography [23,24] and the severity of airway obstruction and spectrum of pathogens in pneumonic and non-pneumonic exacerbations are similar [25]. Pneumonic AECOPD are not simply treated as pneumonia, but require specific management of the AECOPD, including controlled oxygen therapy, corticosteroids, nebulized bronchodilators and, if respiratory acidemia is present, non-invasive ventilation [26]. Therefore, the practice of not excluding such patients was adopted in this work.

Most of the predictors associated with higher inhospital mortality in Table 6 are consistent with previously published studies in AECOPD: increasing age [2,11]; dyspnea severity[11,15]; low BMI [1,22]; low pH [3,22]; cough effectiveness[27]; coexistent consolidation [15,25]; and chronic comorbidities both cardiovascular and non-cardiovascular including atrial fibrillation and cerebrovascular disease [1,28].

Holland et al. [29] reported that esinopenia ($< 0.04 \times 10^3$/dl) was associated with a higher inhospital mortality in AECOPD, but the study population was small ($n = 65$). Our results show that esinopenia is a strong predictor of inhospital mortality ($p = 0.003$) (Table 6). It has previously been shown in an animal model that esinopenia accompanies the response to acute infection and inflammation [30], independent of adrenal glucocorticosteroids [31], and may be a useful marker of sepsis in patients who are receiving intensive care [32,33]. In AECOPD, the strong prognostic influence of eosinopenia may reflect the severity of the accompanying acute inflammatory response [1].

The DECAF Score shows promise for the risk stratification of patients hospitalized with AECOPD [1]. In the present work, the death rates for each grade of the DECAF Score (Table 7) suggest the following risk categories: DECAF 0–1 ('low risk'; inhospital mortality = 3.37%); DECAF 2 ('moderate risk'; mortality = 7.7%); and DECAF 3–6 ('high risk'; mortality = 37%). Findings of the current study suggest that

near half (44.5%) of the patients hospitalized with AECOPD can be classified as low risk (DECAF 0–1) of inhospital mortality and might therefore potentially be suitable for early supported discharge. On the other hand, a high DECAF Score ($\geqslant 3$) might be used as a guide to early escalation of care.

In conclusion, the DECAF Score is simple, effective and quick to calculate clinical tool that can accurately risk stratify hospitalized patients with AECOPD and could therefore help clinicians managing this common and fatal condition.

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