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Clinical significance of the DECAF score in acute exacerbations of chronic obstructive pulmonary disease

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Abstract:

BACKGROUND AND AIM: The prognosis of chronic obstructive pulmonary disease (COPD) depends on the frequency and severity of exacerbations. The DECAF score (dyspnea, eosinopenia, consolidation, acidemia, and atrial fibrillation) is a cost-effective tool used to predict exacerbation outcomes. This study examines its relationship with six-month mortality following COPD exacerbations (ECOPD).

METHODS: Patients (n=260) hospitalized at our center between December 15, 2021 and June 15, 2022 due to ECOPD were evaluated prospectively. Patients were classified into low-risk (DECAF score <2) and moderate-high-risk (DECAF score ≥2) groups. Patients who died within six months were compared with survivors in terms of their demographic and clinical characteristics. Independent risk factors associated with mortality were identified using logistic regression analysis.

RESULTS: The study included 260 patients, 78.8% of whom were male, with a median age of 67 years. The moderate-high-risk group comprised 43.5% (n=113) of patients, and the low-risk group comprised 56.5% (n=147). Patients with a DECAF score ≥2 had a significantly higher six-month mortality rate (p=0.022). Regression analysis showed that mortality risk increased with the presence of congestive heart failure (CHF), higher blood urea nitrogen (BUN) levels, longer hospital stay, and lower hematocrit percentage and body mass index (BMI) values. However, the DECAF score did not emerge as an independent risk factor for six-month mortality following ECOPD.

CONCLUSIONS: Clinical parameters including BMI, length of hospital stay, hematocrit, BUN levels, and the presence of CHF can be used to determine six-month mortality risk in patients hospitalized due to ECOPD. However, the DECAF score was not found to be an independent predictor of six-month mortality.

Keywords:

DECAF score, exacerbations of chronic obstructive pulmonary disease, mortality

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Introduction

As a result of systemic responses to abnormal airway and parenchymal inflammation, chronic obstructive pulmonary disease (COPD) presents as a systemic disease with various extrapulmonary consequences.[1] COPD ranks as the third most common cause of mortality worldwide and causes approximately 3 million deaths each year. Low- and middle-income countries account for more than 90% of the global mortality burden. The prognosis of COPD depends on exacerbations resulting from physiological deterioration and increased airway inflammation. Exacerbations increase hospitalization rates and admissions to healthcare services.[2] Determining the severity of exacerbations and managing them attacks play an important role in the follow-up of patients with COPD. There is a growing body of evidence for determining prognosis in stable COPD; however, there remains a clinical need for practical scoring systems that can be used to predict mortality risk in patients hospitalized due to exacerbations of COPD (ECOPD).

Consequently, Steer et al.[3] developed the DECAF (dyspnea, eosinopenia, consolidation, acidemia, and atrial fibrillation) score, which consists of five parameters, to predict in-hospital mortality risk in patients hospitalized with ECOPD. According to this easily applicable scoring system, 0–1 points indicate low risk, 2 points indicate moderate risk, and 3–6 points indicate high risk. It has been shown that in-hospital mortality risk increases with higher scores.[4] There are also studies demonstrating that the DECAF score is an effective and cost-effective tool for predicting mortality, disease severity, and the possibility of early discharge in patients with COPD.[5,6] The DECAF score facilitates accurate prognostic assessment by identifying low-risk patients eligible for home care or early discharge, as well as high-risk patients requiring intensive management. The aim of this study was to evaluate the clinical significance of the DECAF score in predicting six-month mortality risk following ECOPD in hospitalized patients.

Materials and Methods

This prospective cohort study adhered to the principles outlined in the Declaration of Helsinki and received ethical approval from the Clinical Research Ethics Committee of the Keçiören Training and Research Hospital (Approval Number: 2012-KAEK-15/2433, Date: 23.11.2021). Written informed consent was obtained from each participant in accordance with ethical standards. The clinical and demographic characteristics of 260 patients hospitalized at our center between December 15, 2021 and June 15, 2022 due to ECOPD were evaluated prospectively. Patients with previously documented airway obstruction confirmed by pulmonary function test (PFT) (post-bronchodilator forced expiratory volume in one second to forced vital capacity ratio [FEV_1/FVC] <70%, indicating persistent airflow limitation) and a smoking history of at least 20 pack-years and/or biomass exposure were included in the study.

The exclusion criteria were metastatic malignancy with a life expectancy of less than one year, a diagnosis of asthma, severe neurological or psychiatric disorders impairing effective communication, and the requirement for invasive mechanical ventilation and/or sedative-hypnotic therapy in a level III intensive care unit.

The following demographic and clinical data of the study group were recorded: age, sex, smoking history, body mass index (BMI), occupation, comorbidities, exposures (asbestosis, biomass), electrocardiography (ECG) findings, arterial blood gas results (obtained within the first three days of hospitalization), complete blood count, pulmonary function test results, blood urea nitrogen (BUN), albumin levels, C-reactive protein (CRP), creatinine levels, chest radiograph findings, echocardiography results, history of Coronavirus Disease 2019 (COVID-19) infection, vaccination status (COVID-19, pneumococcal, influenza vaccines), length of hospital stay, and in-hospital mortality.

Patients with a positive COVID-19 polymerase chain reaction (PCR) test from a nasal or oronasal swab at any time before hospitalization were considered to have a history of COVID-19 infection.

Hemoglobin concentrations below 12 g/dL in women and below 13 g/dL in men were defined as anemia.[7] According to the 2016 World Health Organization (WHO) definition, hemoglobin levels >16.5 g/dL in men and >16 g/dL in women were classified as polycythemia.[8]

Treatment adherence was evaluated using the Morisky–Green–Levine Scale (MGLS), a self-administered tool consisting of four dichotomous (yes/no) questions. Medication adherence was considered high if all responses

were "no," moderate if one or two responses were "yes," and low if three or four responses were "yes".[9] Approval from Donald E. Morisky[9] was obtained for use of this questionnaire. This scale was validated in Turkish by Yılmaz et al.[10] in 2004.

The number of emergency department visits, hospitalizations, and intensive care unit (ICU) admissions due to COPD exacerbations within one year and six months before and after enrollment was recorded. Patients with more than two emergency department admissions in the previous year were considered frequent exacerbators.

The extended Medical Research Council (eMRC) dyspnea scale was used to assess the degree of dyspnea.[4] Patients were classified into groups A, B, and E according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) assessment tool.[1]

The DECAF score was calculated as the sum of the points listed below:

Dyspnea
- eMRCD 5a (too breathless to leave the house unassisted but independent in washing and/or dressing) – 1 point,
- eMRCD 5b (breathlessness severe enough to prevent leaving the house unaided and requiring assistance with washing and dressing) – 2 points.

Eosinopenia
- Eosinophils $<0.05\times10^9$/L – 1 point.

Consolidation
- Presence of consolidation – 1 point.

Acidemia
- Moderate or severe acidemia (pH <7.3) – 1 point.

Atrial fibrillation
- Atrial fibrillation, including a history of paroxysmal atrial fibrillation – 1 point.

A total score of 0–1 points was considered low risk, and 2–6 points were considered moderate-high risk.[5]

Statistical method
The demographic and clinical data of patients who died or survived within six months following ECOPD were compared using SPSS Statistics version 21 for Windows (IBM, New York, USA). The distribution of variables was evaluated using visual and analytical methods. Variables with compatible histograms and probability plots were considered normally distributed when the p-value in the Kolmogorov-Smirnov test was >0.05. Descriptive statistics for normally distributed variables were presented as mean ± standard deviation (SD). The significance of differences in means between groups was assessed using the Student's t test. Descriptive statistics for other numerical variables were presented as medians with interquartile ranges (IQR), and nominal variables were expressed as numbers and percentages. The Mann–Whitney U test was used to evaluate differences in median values, while the Pearson chi-square test or Fisher's exact test was applied to compare nominal variables.

Multiple logistic regression analysis was performed to determine independent risk factors for six-month mortality following ECOPD. Variables with a p-value <0.10 were included in the model. Among variables that were highly correlated with each other, clinically significant variables were selected. Model fit was assessed using the Hosmer-Lemeshow test. Final model results were obtained using the backward variable elimination method. As the aim of the study was to determine the odds of factors affecting six-month mortality after exacerbation (a binary outcome at a single time point), logistic regression analysis was preferred over Cox proportional hazards modeling. In addition, the small number of deaths may have violated the proportional hazards assumption. Results were considered statistically significant when the p-value was <0.05.

Statistical power was calculated using the final sample size, observed mortality ratios in the moderate-high-risk group (DECAF score ≥2), and a significance level of 0.05. The estimated power of the study was 0.63.

Results

The study group comprised 260 consecutive patients, 78.8% (n=205) of whom were male, with a median age of 67 years (IQR: 15). According to the DECAF score, 56.5% (n=147) of patients were in the low-risk group (score <2), and 43.5% (n=113) were in the moderate-high-risk group (score ≥2). Examination of DECAF score components showed that 33.8% had eMRC 5a dyspnea, 15% had eMRC 5b dyspnea, 43.5% had eosinopenia, 40.8%

had consolidation, 7.3% had acidemia, and 8.5% had atrial fibrillation or flutter. In-hospital mortality occurred in five patients. All in-hospital deaths (n=5) occurred in patients with a DECAF score ≥2, and this finding was statistically significant (p=0.015). However, because the number of in-hospital deaths was low, further statistical analyses to determine clinical factors associated with in-hospital mortality could not be performed. Six-month mortality after ECOPD was higher in patients with a DECAF score ≥2 (6.8% vs. 15.7%, p=0.022).

The 255 discharged patients were divided into two groups: the mortality group (patients who died within six months after ECOPD, n=27) and survivors. The clinical and demographic characteristics of these groups were compared. The proportion of patients with a moderate-high risk DECAF score (≥2) was significantly higher in the than in the survivor group (39.9% vs. 63%, p=0.022). Median BMI was significantly lower in the mortality group (21.8 kg/m^2 vs. 26.15 kg/m^2, p<0.001). Length of hospital stay (13 days vs. 10 days, p=0.001), rate of intensive care unit admission (25.9% vs. 10.1%, p=0.025), and length of ICU stay (17 days vs. 9 days, p=0.039) were also higher in the mortality group. The groups were similar with respect to GOLD stages and inhaler medication adherence assessed by the MGLS (Table 1).

When comorbidities were compared, only congestive heart failure (CHF) was significantly more frequent in the mortality group (29.6% vs. 5.7%, p<0.001). The groups were similar in terms of other comorbidities, including benign prostatic hyperplasia, lung cancer, extrathoracic malignancies, diabetes mellitus, hypertension, coronary artery disease, and pulmonary thromboembolism. No statistically significant difference was observed between groups in dyspnea severity assessed by the eMRC score (p=0.246). The rate of type 2 respiratory failure was 50.4% in the survivor group and 55.6% in the mortality group (p=0.615).

Comparison of laboratory findings showed that anemia was significantly more common in the mortality group (59.3% vs. 23.7%, p <0.001). Mean hemoglobin, hematocrit, and albumin levels were lower, while median BUN levels were significantly higher in the mortality group (p<0.001, p<0.001, p<0.001, and p=0.009, respectively) (Table 2). When echocardiographic findings were examined, the ejection fraction (EF) was lower (57.5% vs. 60%, p=0.003) and systolic pulmonary artery pressure (sPAP) was higher in the mortality group (38.5 mmHg vs. 35 mmHg, p=0.048) (Table 2). The FEV_1/FVC value was significantly lower in the mortality group, whereas other respiratory function test parameters were statistically similar between groups (Table 2).

Variables with a p-value <0.10 in univariate analyses were included in the logistic regression model for mor-

Table 1: Comparison of demographic and clinical characteristics between groups

	Survivors (n=228) Median (IQR)		Mortality group (n=27) Median (IQR)		p
	n	%	n	%	
Sex (male)	177	77.6	24	88.9	0.176
Age (years)	14	66	15	72	0.096
BMI (kg/m^2)	8.6	26.15	5.3	21.8	**<0.001**
ICU admission	23	10.1	7	25.9	**0.025**
Length of hospital stay					
Lenght of ward stay (days)	6	10	9	13	**0.001**
ICU stay (days) (n=30)	6	9	24	17	**0.039**
GOLD stage					0.260
B	119	52.2	11	40.7	
E	109	47.8	16	59.3	
Smoking status					
Current smoker	70	30.7	8	29.6	0.524
Former smoker	124	54.4	17	63	
Never smoker	34	14.9	2	7.4	
Exposure history					
Biomass exposure	89	39	10	37	0.840
Asbestos exposure	142	62.3	17	63	0.945
Occupational exposure	162	71.1	17	63	0.385
Treatment adherence (MGLS)					
Low	35	15.4	4	14.8	0.643
Moderate	81	35.5	12	44.4	
High	112	49.1	11	40.7	
Vaccination status					
COVID-19	220	96.5	27	100	1.00
Influenza	85	37.3	11	40.7	0.726
Pneumococcal	92	40.4	12	44.4	0.682
Hospital admissions in previous year					
≥2 emergency visits	146	64	20	74.1	0.301
≥1 ward hospitalization	91	39.9	16	59.3	0.054
≥1 ICU hospitalization	50	21.9	6	22.2	0.972
History of COVID-19 infection	65	28.5	3	11.1	0.053
DECAF score					**0.022**
<2	137	60.1	10	37	
≥2	91	39.9	17	63	

BMI: Body mass index, GOLD: Global Initiative for Chronic Obstructive Lung Disease, ICU: Intensive care unit, MGLS: Morisky–Green–Levine Scale

Table 2: Comparison of laboratory findings between groups

	Survivors (n=228) n (%) Median (IQR) Mean±SD	Mortality Group (n=27) n (%) Median (IQR) Mean±SD	p
CBC values			
HGB (g/dL)	14.35±2.26	12.6±2.4	**<0.001**
Hematocrit (%)	44.68±6.87	39.3±7.1	**<0.001**
Thrombocyte count ($10^3/\mu L$)	258 (112.50)	241(184)	0.845
Eosinophils ($10^3/\mu L$)	0.08 (0.21)	0.03 (0.16)	0.565
Biochemical parameters			
Albumin (g/L)	36.51±5.08	33±4.4	**<0.001**
Creatinine (mg/dL)	0.90 (0.33)	0.97 (0.53)	0.617
BUN (mg/dL)	16 (8)	22 (18)	**0.009**
CRP (mg/L)	29.5 (120)	51 (109)	0.225
Anemia			
HGB <12 g/dL for females <13 g/dL for males	54 (23.7)	16 (59.3)	**<0.001**
Polycythemia			
HGB>16 g/dL for females >16.5 g/dL for males	45 (19.7)	2 (7.4)	0.118
Arterial blood gas analysis			
pH	7.42 (0.08)	7.42 (0.10)	0.812
pO_2 (mmHg)	57 (22.75)	55.6 (25.20)	0.440
pCO_2 (mmHg)	44.55 (17.3)	46.7 (16)	0.321
Echocardiography (n=222)			
EF (%)	(n=198) 60 (0)	(n=24) 57.5 (8.75)	**0.003**
sPAP (mmHg)	(n=198) 35 (13)	(n=24) 38.5 (19)	**0.048**
Pulmonary function tests			
FEV_1 (L)	0.95 (0.51)	0.810 (0.51)	0.153
FEV_1 (%)	34.5 (19)	32 (22)	0.444
FVC (L)	1.7 (0.89)	1.45 (0.74)	0.201
FVC (%)	49 (23.75)	46 (19)	0.614
FEV_1/FVC (%)	58 (16)	52 (14)	0.026

Data were expressed as % (n), median (interquartile range, IQR), or mean± standard deviation (SD), as appropriate for data type and distribution. BUN: Blood urea nitrogen, CRP: C-reactive protein, EF: Ejection fraction, FEV_1: Forced expiratory volume in the first second, FVC: Forced vital capacity, HGB: Hemoglobin, sPAP: Systolic pulmonary artery pressure

tality (Table 3). BMI, hematocrit, BUN, and CHF were identified as independent risk factors for six-month mortality after ECOPD. In contrast, DECAF risk groups were not found to be clinical predictors of six-month mortality (odds ratio [OR]: 0.432, confidence interval [CI]: 0.117–1.586, p=0.206) (Table 3).

The final model was constructed using the backward elimination method. The Hosmer-Lemeshow test indicated an acceptable model fit for the final model (p=0.396). In this model, six-month mortality risk increased with lower hematocrit and BMI values (p=0.016 and p=0.001, respectively). Higher BUN levels, longer hospital stay, and the presence of CHF were also identified as predictors of six-month mortality (p=0.008, p=0.047, and p=0.007, respectively) (Table 4). In this logistic regression analysis, CHF showed the strongest association with six-month mortality, with an odds ratio of 6.041 (CI: 1.650–22.131).

Discussion

Exacerbations are the most important cause of mortality and morbidity in COPD, as they reduce quality of life, accelerate the decline in lung function, and lead to frequent hospitalizations.[1] Therefore, effective and reliable clinical tools for determining mortality risk after ECOPD are essential. Many scoring systems, such as DECAF, CURB-65 (Confusion, urea, respiratory rate, blood pressure, age), BAP-65 (blood urea nitrogen, altered mental status, pulse, age), PSI (Pneumonia Severity Index), PEARL (previous admissions, eMRC, age, right ventricular failure, left ventricular failure), PLR (platelet-to-lymphocyte ratio), and NLR (neutrophil-to-lymphocyte ratio), have been developed and investigated to address this clinical need. Recent studies have reported that the DECAF score is more effective in predicting 30- and 90-day mortality than the CURB-65, PSI, and BAP-65 scores.[11–13] However, our study, which evaluated multiple demographic and clinical confounders simultaneously, revealed contrasting results for longer-term mortality.

It has also been reported that the DECAF score predicts in-hospital mortality related to ECOPD with high sensitivity and specificity.[5,12,13] In-hospital mortality rates among patients with ECOPD range from 4.4% to 25%.[14–16] Due to the exclusion of patients admitted to the level 3 ICUs, the in-hospital mortality rate in the present study was 1.9%. Nevertheless, all in-hospital deaths occurred in patients with a DECAF score ≥2.

Previous studies have highlighted the DECAF score as a cost-effective tool for predicting disease severity and guiding early discharge decisions in patients with COPD. However, a meta-analysis evaluating studies on these scoring systems indicated a high risk of bias associated with the DECAF score.[17] In our study, although the proportion of patients with DECAF scores ≥2 was significantly higher in the mortality group in univariate analyses, regression analyses demonstrated that the DECAF

Table 3: Initial regression analysis model

Variable	B	SE	Wald	Odds ratio	95% CI	p
ICU admission vs. ward	0.122	0.841	0.021	1.130	0.218-5.868	0.885
Length of hospital stay (days)	0.069	0.038	3.269	1.072	0.994-1.155	**0.071**
BMI (kg/m^2)	-0.171	0.068	6.321	0.843	0.737-0.963	**0.012**
Hct (%)	-0.101	0.043	5.364	0.904	0.830-0.985	**0.021**
BUN (mg/dL)	0.084	0.031	7.614	1.088	1.025-1.155	**0.006**
Albumin (g/L)	-0.074	0.058	1.618	0.928	0.828-1.041	0.203
DECAF score	-0.084	0.664	1.601	0.432	0.117-1.586	0.206
FEV_1/FVC ratio	-0.008	0.030	0.063	0.992	0.935-1.053	0.801
CHF	1.736	0.721	5.799	5.676	1.381-23.319	**0.016**
Ward hospitalization in previous year	0.179	0.628	0.081	1.196	0.349-4.095	0.775
sPAP (mmHg)	0.021	0.020	1.026	1.021	0.989-1.062	0.311
History of COVID-19 infection	-1.428	0.803	3.158	0.240	0.050-1.158	**0.076**

Hosmer–Lemeshow test p value=0.934. B: Beta coefficient, SE: Standard error, CI: Confidence Interval, BMI: Body mass index, BUN: Blood urea nitrogen, CHF: Congestive heart failure, FEV_1/FVC: Forced expiratory volume in 1 second to forced vital capacity ratio, Hct: Hematocrit, sPAP: Systolic pulmonary artery pressure

Table 4: Final regression analysis model

Variable	B	SE	Wald	Odds ratio	95% CI	p
Length of hospital stay (days)	0.068	0.034	3.855	1.071	1.001–1.145	**0.047**
BMI (kg/m^2)	-0.201	0.060	11.019	0.818	0.727–0.921	**0.001**
Hct (%)	-0.102	0.042	5.762	0.903	0.831–0.982	**0.016**
BUN (mg/dL)	0.074	0.028	7.148	1.076	1.020–1.136	**0.008**
CHF	1.799	0.662	7.381	6.041	1.650–22.131	**0.007**
History of COVID-19 infection	-1.392	0.737	3.570	0.249	0.059–1.053	0.059

Hosmer–Lemeshow test p value=0.396. B: Beta coefficient, SE: Standard error, CI: Confidence Interval, BMI: Body mass index, BUN: Blood urea nitrogen, CHF: Congestive heart failure, Hct: Hematocrit

score could not predict six-month mortality after ECOPD. Instead, the regression analysis emphasized the importance of BMI, length of hospital stay, hematocrit, BUN levels, and the presence of CHF, parameters that have also been incorporated into other prognostic scoring systems.

Recent evidence has shown that the risk of moderate and/or severe exacerbations is higher in patients with low BMI.[18,19] Similar to our study, Zhang et al.[20] examined 890 patients hospitalized for COPD exacerbations and found a significant negative correlation between one-year mortality and body mass index.

The relationships between biochemical markers and clinical outcomes in COPD have been investigated in numerous studies. Hemoglobin abnormalities, including anemia and polycythemia, are common in COPD and have been associated with increased symptoms, poorer quality of life, reduced exercise capacity, higher hospitalization rates, and increased mortality.[21–25] In addition, in-hospital, 30-day, six-month, and one-year mortality rates have been reported to be significantly higher in anemic patients hospitalized for ECOPD.[24,25] These findings indicate that anemia is not only an important marker of long-term survival but also a risk factor for short-term mortality in severe ECOPD. In our study, anemia was significantly more common in the mortality group, whereas the prevalence of polycythemia was similar between groups.

Inflammatory responses in COPD accelerate proteolysis, leading to hypoalbuminemia and increased BUN levels. BUN is associated with neurohumoral activity, cardiorenal function, and catabolic state. Therefore, elevated BUN levels have been linked to poor prognosis in patients with ECOPD.[26,27] In the present study, elevated BUN was identified as an independent predictor of six-month mortality after ECOPD. Our results also demonstrated that longer hospital stay was associated with six-month mortality. Similarly, Gunen et al.[28] reported that in-hospital mortality in ECOPD increases with prolonged hospitalization. Comorbidities have been emphasized as important components in the follow-up of patients with COPD, particularly cardiovascular comorbidities, which are significant predictors of one-year mortality after an exacerbation.[1,29] In the present study, among multiple comorbidities analyzed,

only CHF emerged as an independent risk factor for six-month mortality after ECOPD. Patients with CHF had sixfold higher odds of six-month mortality after ECOPD compared with those without CHF after adjustment for potential confounders. However, the wide confidence interval suggests limited precision, likely reflecting the small number of cases.

As a limitation of this study, its single center design means that the results may not reflect regional differences. Additionally, data regarding smoking history, exposures, occupation, medication adherence, comorbidities, and hospitalization history were largely based on patient self-report. Therefore, recall bias should be considered when interpreting these subjective data. Although the study provides important preliminary information on the DECAF score with a longer follow-up period than previous studies, the statistical power was moderate, which may have limited the ability to detect smaller effect sizes.

Conclusion

In conclusion, although the proportion of patients with medium-high risk DECAF scores (≥2) was significantly higher in the mortality group, logistic regression analysis for six-month mortality identified length of hospital stay (days), BMI (kg/m^2), hematocrit (%), BUN (mg/dL), and the presence of CHF as independent risk factors. A key strength of this study is that the clinical significance of the DECAF score was evaluated alongside numerous clinical and demographic variables that may influence mortality, including treatment adherence, vaccination status, history of COVID-19 infection, and hospital admissions in the preceding year. Furthermore, unlike studies focusing on shorter-term outcomes, the present study demonstrated that the DECAF score was not an independent factor of six-month mortality. If these preliminary findings are confirmed in large, multicenter studies, they may contribute to the development of future artificial intelligence-based models for predicting mortality after ECOPD.

Ethics Committee Approval

The study was approved by the Keçiören Training and Research Hospital Clinical Research Ethics Committee (No: 2012-KAEK-15/2433, Date: 23/11/2021).

Informed Consent

Written informed consent was obtained from the participants.

Conflicts of Interest

The authors have no conflicts of interest to declare.

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Author Contributions

Concept – A.Ç., S.Ş.D., D.K., Ö.S., S.D.; Design – A.Ç., S.Ş.D., D.K., Ö.S., S.D.; Supervision – A.Ç., S.Ş.D., D.K.; Resource – A.Ç., S.Ş.D., D.K.; Materials – A.Ç., S.Ş.D., D.K., Ö.S., S.D.; Data Collection and/or Processing - A.Ç., S.Ş.D., D.K., Ö.S., S.D.; Analysis and/or Interpretation - A.Ç., S.Ş.D., D.K.; Literature Review – A.Ç., S.Ş.D., D.K.; Writing – A.Ç., S.Ş.D., D.K.; Critical Review – A.Ç., S.Ş.D., D.K.

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