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ABOUT the CLINICAL SCIENCE of NUTRITION

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Clinical Science of Nutrition is a triannual journal that is published in English in April, August, and December.

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Aims, Scope, and Audience
The journal aims to contribute to the literature by publishing high impact content and become one of the leading publications of the field while functioning as an open discussion forum on significant issues of current interest. Clinical Science of Nutrition also aims to have significant input in emphasizing the increasing importance of clinical nutrition in Turkey and the region, identifying the effects of differences between societies on study results in a clearer way and converting clinical applications into scientific publications as well as forming a bridge between West and East.

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The journal covers all aspects of nutrition and dietetics including prevalence of malnutrition and its effects on clinical results; nutritional support and delivery methods and their advantages and disadvantages; nutritional support products and their side effects; immune system and nutritional support; ERAS protocol and nutritional support; home parenteral and enteral nutrition; nutrition support teams and their necessity, challenges and potential solutions of nutritional support.

The journal's target audience includes academicians, practitioners, specialists and students interested in nutrition and dietetics.

You can find the current version of the Instructions to Authors at https://clinscinutr.org/EN

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Original Article

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Increased Dietary Inflammatory Index Score Is Associated with Type 2 Diabetes Mellitus in Obese Women: A Case–Control Study

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ABSTRACT

Objective: Recent evidence indicates that diet-induced inflammation is related to chronical diseases including type 2 diabetes mellitus. This study aimed to examine the relationship between the dietary inflammatory index, which quantifies the inflammatory burden of the diet, and type 2 diabetes mellitus risk among obese women.
Methods: This case–control study, including 40 obese cases with type 2 diabetes mellitus and 40 obese controls without type 2 diabetes mellitus, aged between 30 and 50, was conducted from September 2019 to March 2020 in Ankara, Turkey. The Dietary Inflammatory Index was calculated based on the food frequency questionnaire. The logistic regression model was used to estimate the association between Dietary Inflammatory Index and the risk of type 2 diabetes mellitus. Linear regression model was used to estimate beta coefficients for glucose metabolism markers.
Results: Subjects with higher Dietary Inflammatory Index scores (i.e., with a more pro-inflammatory diet) had a higher risk of type 2 diabetes mellitus (odds ratio = 8.57; 95% CI: 1.24, 59.44, $P = .03$). In addition, as a continuous variable, the Dietary Inflammatory Index scores had a significant positive relationship with insulin ($\beta = 0.259$, $P = .026$) and homeostatic model assessment of insulin resistance ($\beta = 0.265$, $P = .015$) after multivariable adjustment.
Conclusion: The present study suggests that higher Dietary Inflammatory Index scores, corresponding to more pro-inflammatory diets, were positively associated with type 2 diabetes mellitus risk among obese women. In addition, the Dietary Inflammatory Index scores and insulin resistance were positively related. As a result of the findings, an anti-inflammatory diet can help prevent insulin resistance and reduce the risk of diabetes.

Keywords: Dietary Inflammatory Index (DII), diabetes, inflammation, obesity

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major cause of morbidity and mortality worldwide. An aging population, economic development, urbanization, an increasingly sedentary lifestyle, and unhealthy dietary habits have led to an increase in the T2DM prevalence around the world.[1] According to the International Diabetes Federation (IDF) report, there were 463 million people with diabetes around the world in 2019 and that this number will increase to 700 million by 2045.[2] There has been growing evidence that in addition to the genetic, metabolic, and lifestyle factors that cause T2DM, inflammation also plays an essential role in the pathogenesis of T2DM.[3] Inflammation is characterized by increased levels of pro-inflammatory cytokines such as C-reactive protein (CRP), tumor necrosis factor-alpha (TNF-α), and interleukins (ILs).[4] Especially in chronic low-grade inflammation, pro-inflammatory cytokines that increase in serum levels cause β-cell damage and chronic hyperglycemia, and T2DM occurs as a result.[5] Inflammation can develop due to many environmental and behavioral factors. However, the diet has been accepted as an essential modulator of chronic inflammation in recent years.[6] Indeed, many studies have shown that many nutritional factors and dietary patterns affect the serum levels of inflammatory markers such as IL-6, TNF-α, and CRP.[6,7]

The results of diet on inflammation and of inflammation on diabetes propose that diet may also improve the T2DM

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risk through inflammation.[8] In this context, previous studies have associated many nutrients that are considered to have an anti-inflammatory effect, such as dietary fiber and carotenoids, with low T2DM risk.[9,10] In contrast, the foods considered to have a pro-inflammatory effect, such as red meat containing high levels of saturated fatty acids, have been associated with high T2DM risk.[11]

Dietary Inflammatory Index (DII) is a literature-derived index developed to evaluate the inflammatory potential of diet. According to the index scoring, the higher DII score indicated a pro-inflammatory diet, whereas the lower score indicated an anti-inflammatory diet. The final score is obtained from not only a certain nutrient or food but from the overall diet.[12] The DII has been validated by inflammatory markers such as CRP, IL-6, or TNF-α in various studies.[13,14] It could be used in any human population with dietary data collected from different assessment methods such as food frequency questionnaire (FFQ), 24-hour dietary recall, and 3- to 7-day food record.[13-15] After the development of the DII, its relationship with various diseases such as cardiovascular diseases,[16] renal diseases,[17] mental health,[18] metabolic syndrome,[19] and, in particular, cancer[20] has been investigated. However, besides only a few studies existing on the relationship between the DII and glycemic markers in the literature,[21,22] to the best of our knowledge, no case–control study has focused on the relationship between the DII and T2DM risk in obese women.

The present study aimed to investigate the association between the inflammatory potential of the diet, as measured by the DII, and T2DM risk among obese adult women. The hypothesis of this study is that a higher DII score (indicating a pro-inflammatory diet) increases the risk of diabetes.

Main Points

- Diet-induced inflammation is related to type 2 diabetes mellitus (T2DM) risk.
- Dietary inflammatory index (DII) is a literature-derived index developed to measure the inflammatory potential of diet.
- It was hypothesized that using the DII in clinical practice may be useful to reduce the risk for diseases related to chronic inflammation including T2DM.
- To the best of our knowledge, this is the first case–control study that investigated the association between the DII and the risk for diabetes among obese women.
- A positive association was found between the dietary inflammatory potential measured by the DII and T2DM.
- In addition, the DII scores and insulin resistance were positively related.

METHODS

Participants

This case–control study was conducted in University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital between September 2019 and March 2020. Voluntary subjects were female, aged 30-50 years with a body mass index (BMI) range of 30-35 kg/m^2. The case group consisted of 40 obese patients with T2DM, whereas the control group consisted of 40 obese patients without T2DM. Type 1 diabetes patients, type 2 diabetes patients receiving insulin treatment, and those with chronical disease were excluded from the study. The graphical abstract of the study is given in the supplementary Figure 1.

Written informed consent was obtained from each participant. The study protocol was approved by the Dışkapı Yıldırım Beyazıt Training and Research Hospital Clinical Research Ethics Committee on August 26, 2019, with the decision number 70/04.

Sociodemographic attributes of the individuals such as age, education level, and employment status and general information regarding their health statuses such as family history of diabetes, smoking, and alcohol intake were obtained by the researcher through face-to-face interviews and were recorded on the questionnaire form.

Anthropometric Measurements

Body weight of the participants was measured using a digital weight scale (Seca 769). The measurements were done with as few and as thin clothes as possible and without shoes in the morning while the participants were hungry. Body height was measured using the height ruler attached to the digital weight scale with the feet placed side by side and head in the Frankfort plane position. Body mass index was calculated after the measurements using the following formula: body weight (kg)/height (m)^2. Considering the World Health Organization (WHO) criteria,[23] individuals between 30 and 35 kg/m^2 were included in the study. Waist and hip circumference were appropriately calculated. The waist-to-hip ratio was calculated by proportioning the participants' waist circumference to their hip circumference. According to the WHO criteria, the participants with a waist/hip ratio of ≥0.85 were accepted to be at risk of developing metabolic complications.[24]

Calculation of Dietary Intake and Dietary Inflammatory Index

A quantitative FFQ was used to collect data on food consumption by the researcher in face-to-face interviews. Consumption frequency of food intake and the portion

size in the last 3 months was queried. To help participants quantify the portions consumed, food photographs were used. Dietary data from the FFQ were converted into the daily intakes (g/day), and using The Nutrient Database (BeBİS, Ebispro for Windows, Germany; Turkish Version/ BeBİS 8.2) total energy, macro, and micronutrient intakes, which were utilized to calculate the DII, were computed. The DII is a valid and reliable tool to measure the dietary inflammatory potential. The development and the calculation steps of the DII have been previously documented in detail.[12,25] Briefly, the DII is based on a literature review of 1943 articles published between 1950 and 2010 linking dietary components to the following inflammatory markers: CRP, IL-1β, IL-4, IL-6, IL-10, and TNF-α. A total of 45 different food parameters including macronutrients and micronutrients, as well as some bioactive components, were identified as linking to inflammation. In the present study, a total of 44 food parameters (except trans fatty acid) used for the DII calculation were available from the FFQ. Higher DII scores indicate a more pro-inflammatory diet; lower DII scores indicate a more anti-inflammatory diet.

Serum Collection and Laboratory Measurements

As laboratory measurements, fasting blood glucose (FBG), glycosylated hemoglobin (HbA1c), and fasting insulin levels were measured from the blood samples. From these data, the homeostatic model assessment of insulin resistance (HOMA-IR) value (the indicator of insulin resistance) was calculated as previously described.[26]

Statistical Analysis

First, tertile cutoff points for the DII score were defined based on the distribution among controls. Then, all the participants were categorized according to these cutoffs. Dietary Inflammatory Index tertiles were defined as $t_1 <$ –0.625; t_2 (–0.625) – (0.046); $t_3 >$0.046. Chi-square tests and Fisher's exact tests were used for categorical variables, and the Student's *t*-test, 1-way analysis of variance test, and Kruskal–Wallis test were used for continuous variables to evaluate differences between different groups. Linear regression was used to evaluate beta coefficients for glucose metabolism markers. Binary logistic regression was used to estimate crude and adjusted association between the DII score (continuous and categorical variable) and T2DM risk. Three regression models were used. The first model was the unadjusted logistic regression model. In the second model, age (continuous), physical activity, and standardized energy intake (kcal/day) were adjusted. In the final model, additionally, BMI was adjusted. Statistical analyses of the study were done using IBM SPSS (Statistical Package for Social Science, SPSS Company, Ill, USA) version 23. Statistical significance was defined as $P < .05$.

RESULTS

The distribution of baseline characteristics and some anthropometric measurements for both the case and control groups and the DII tertiles are shown in Table 1. Compared with controls, T2DM cases were more likely to have higher BMI. In the third tertile of DII score, the BMI measurements were also significantly higher than that observed in any other tertiles. The distribution of food consumption according to the tertiles is presented in Table 2. Compared to the individuals with lower DII scores, the intake of fiber, magnesium, iron, copper, vitamin A, vitamin C, vitamin E, beta carotene, folic acid, thiamine, and vitamin B_6 were significantly lower in the individuals with higher DII scores. Biochemical parameters and the beta coefficients regarding the DII scores are displayed in Table 3. As a continuous variable, the DII scores had a significant positive relationship with all glucose metabolism markers in the crude model. After adjustment for age, physical activity, standardized energy intake, and BMI, a significant positive association between the DII scores and HOMA-IR and insulin was observed. Table 4 shows ORs and 95% CI for T2DM by tertiles. After adjusting for potential confounding factors, the risk of T2DM was found to be 8 times higher in the highest tertile than in the lowest tertile.

DISCUSSION

In this case–control study, the possible role of the DII in diabetes pathogenesis was investigated, and a positive association was found between the dietary inflammatory potential measured by the DII and T2DM. These findings show that a more pro-inflammatory diet indicated by high DII scores may increase the risk for diabetes and that a more anti-inflammatory diet indicated by low DII scores may have a protective effect on the development of T2DM. To the best of our knowledge, this is the first case–control study that investigated the association between the DII and the risk for diabetes among obese women.

The role of increased inflammatory responses on T2DM pathogenesis is known, and dietary components are considered to affect the T2DM risk via inflammation.[8] There have been several studies evaluating the effects of dietary components or dietary models on T2DM risk via inflammation.[6,27] However, only a few studies have investigated the pro- and anti-inflammatory effects of the overall diet on T2DM.[28-30]

The DII was developed to measure the inflammatory potential of an overall diet based on an extensive literature search including studies that subject to dietary components and inflammatory markers.[12] To obtain the DII

Table 1. Baseline Characteristics and Some Anthropometric Measurements According to Case and Control Groups and Tertiles of the DII Score

Variable	Cases (n=40)	Controls (n=40)	*P*†	DII Tertiles			*P*‡
				T_1 (n=16) <-0.625	T_2 (n=27) -0.625 to 0.046	T_3 (n=37) >0.046	
Age (mean) (years)	43.5±4.2	36.5±5.7	**<.001**	38.0 ± 6.2	39.8 ± 6.3	41.0 ± 5.8	.249
Age groups (years)							
30-40	13 (32.5)	31 (77.5)	**<.001**	12 (75.0)	15 (55.6)	17 (45.9)	.148
41-50	27 (67.5)	9 (22.5)		4 (25.0)	12 (44.4)	20 (54.1)	
Occupation							
Housewife	22 (55.0)	19 (47.5)	.087	11 (68.8)	13 (48.2)	17 (45.9)	.168
Employed	18 (45.0)	16 (40.0)		5 (31.2)	10 (37.0)	19 (51.4)	
Unemployed	0 (0.0)	5 (12.5)		0 (0.0)	4 (14.8)	1 (2.7)	
Education level							
Primary school	8 (20.0)	6 (15.0)	.761	4 (25.0)	2 (7.4)	8 (19.2)	.534
High school	19 (47.5)	22 (55.0)		8 (50.0)	16 (59.3)	17 (45.9)	
University	13 (32.5)	12 (30.0)		4 (25.0)	9 (33.3)	12 (32.4)	
Marital status							
Married	29 (72.5)	32 (80.0)	.600	13 (81.2)	20 (74.1)	28 (75.7)	.976
Single	8 (20.0)	7 (17.5)		3 (18.8)	5 (18.5)	7 (18.9)	
Widow/divorced	3 (7.5)	1 (2.5)		0 (0)	2 (7.4)	2 (5.4)	
Smoking status							
Never	32 (80.0)	30 (75.0)	.911	11 (68.8)	23 (85.2)	28 (75.7)	.464
Former smoker	2 (5.0)	2 (5.0)		2 (12.5)	1 (3.7)	1 (2.7)	
Current smoker	6 (15.0)	8 (20.0)		3 (18.8)	3 (11.1)	8 (21.6)	
Family history of DM	27 (67.5)	8 (20.0)	**<.001**	7 (43.8)	8 (29.6)	20 (54.1)	.151
BMI (kg/m^2)	33.4 ± 1.6	32.7 ± 1.7	**.044**	32.3 ± 1.4	32.3 ± 1.4	33.8 ± 1.7	**<.001**
Waist circumference (cm)	101.2 ± 6.8	98.5 ± 6.1	.074	99.5 ± 6.1	96.4 ± 5.5	102.5 ± 6.5	**.001**
Hip circumference (cm)	118.1 ± 6.1	115.6 ± 6.2	.067	115.4 ± 6.2	113.6 ± 4.2	119.9 ± 6.2	**<.001**
Waist-to-hip ratio	0.86 ± 0.04	0.85 ± 0.05	.783	0.86 ± 0.05	0.85 ± 0.05	0.86 ± 0.04	.610
Waist-to-height ratio	0.64 ± 0.04	0.63 ± 0.04	.232	0.63 ± 0.04	0.61 ± 0.03	0.65 ± 0.04	**.002**
Physical activity							
Mild	5 (12.5)	3 (7.5)	.712	2 (12.5)	4 (14.8)	2 (5.4)	.544
Moderate	35 (87.5)	37 (92.5)		14 (87.5)	23 (85.2)	35 (94.6)	

Data were presented with mean ± SD or n (%), where appropriate.
ANOVA, analysis of variance; BMI, body mass index; DII, dietary inflammatory index; DM, diabetes mellitus.
†P values were obtained from Student's t-tests, χ^2 tests, and Fisher's exact test, where appropriate.
‡P values were obtained from ANOVA, χ^2 tests, and Fisher's exact test, where appropriate.

Table 2. Dietary Intakes According to Tertiles of the DII Score (n:80)				
	DII tertiles			
Variables	T_1	T_2	T_3	*P*†
Energy (kcal/day)	2092.2±436.6	2015.0±225.9	1985.9±356.5	0.576
Macronutrients				
Carbohydrates (g/day)	264.5±57.0	248.0±34.6	250.0±58.6	0.563
Fiber (g/day)	38.7±6.2	36.1±4.8	34.7±4.8	**0.041***
Protein (g/day)	80.3±12.2	82.1±10.0	79.0±11.3	0.553
Fat (g)	76.5±19.9	74.6±13.0	72.0±14.4	0.587
Saturated Fat (g)	29.5±6.4	31.1±4.8	29.1±8.6	0.536
Monounsaturated fatty acid (g)	25.7±7.3	23.8±3.8	23.7±5.0	0.411
Polyunsaturated fatty acid (g)	14.5 (10.8-20.4)	11.3 (10.8-15.5)	11.1 (9.6-13.9)	0.129
Omega-3 (g)	1.8 (1.5-2.8)	1.6 (1.3-2.1)	1.5 (1.2-1.9)	0.194
Omega-6 (g)	12.6 (9.5-17.0)	9.7 (9.1-13.3)	9.6 (8.2-11.9)	0.106
Micronutrients				
Magnesium (mg)	396.5±61.1	369.8±48.5	359.0±45.5	0.048
Phosphorus (mg)	1382.8±219.8	1419.9±203.0	1324.9±165.2	0.142
Iron (mg)	14.6±1.9	13.5±1.5	13.1±1.3	**0.006***
Zinc (mg)	11.6±1.8	11.3±1.7	10.8±1.5	0.184
Copper (µg)	2.2±0.4	2.0±0.2	1.9±0.3	**0.026***
Selenium (mg)	13.8 (10.8-14.0)	13.9(11.3-14.1)	13.8 (11.1-14.1)	0.142
Vitamin A (µg)	2004.9±625.9	1843.8±364.8	1547.3±416.4	**0.002***
Vitamin E (mg)	14.4±3.6	12.6±2.0	12.0±2.4	**0.011***
Beta-carotene (µg)	8423.1(6286.5-10118.5)	7449.3(6239.32-8875.2)	6282.9(5626.7-7292.2)	**<0.001***
Thiamine (mg)	1.2±0.2	1.1±0.1	1.1±0.1	**0.020***
Niacin (mg)	1.8±0.3	1.8±0.3	1.7±0.2	0.139
Riboflavin (mg)	29.5±4.1	28.6±3.5	27.7±4.4	0.293
Vitamin B6 (mg)	1.8 (1.5-1.9)	1.5 (1.4-1.6)	1.4 (1.3-1.6)	**0.002***
Vitamin B12 (µg)	3.4 (2.8-4.2)	3.5 (3.0-4.1)	3.4 (2.9-4.2)	0.748
Total folic acid (µg)	404.6 (339.9-443.1)	362.4 (340.4-391.3)	348.7 (323.3-370.1)	**0.005***
Vitamin C (mg)	168.3±29.7	145.8±28.1	138.4±27.9	**0.001***

Data were presented with mean±SD or median (IQR), where appropriate.
†P values were obtained from ANOVA and Kruskal-Wallis test, where appropriate.
*Different lowercase letters in a row indicate a statistically significant difference between group.

scores, the whole diet was taken into account, not just individual nutrients or food. Thus, the DII is considered advantageous in terms of measuring the overall inflammatory potential of the diet.[25] To date, there have been only a few studies investigating the association between the DII and T2DM, and their results are inconsistent.[28-30] A cross-sectional study that evaluated the relationship between the DII and T2DM among adults found that the

Table 3. Beta-coefficient for Glucose Metabolism Markers According to DII Score (n:80)									
	Model 1			Model 2			Model 3		
Dependent Variable	*β*	t	*P*	*β*	t	*P*	*β*	t	*P*
Fasting Blood Glucose	0.283	2.606	**0.011**	0.219	2.233	**0.029**	0.087	0.793	0.430
HbA1c	0.275	2.530	**0.013**	0.201	2.137	**0.036**	0.091	0.851	0.398
Insulin	0.430	4.202	**<0.001**	0.447	4.259	**<0.001**	0.259	2.270	**0.026**
HOMA-IR	0.488	4.933	**<0.001**	0.472	4.716	**<0.001**	0.265	2.492	**0.015**

Linear regression.
Model 1: Crude model.
Model 2: Adjusted for age, physical activity and standardized energy intake.
Model 3: Adjusted for age, physical activity, standardized energy intake and BMI.

Table 4. Odds Ratios (ORs) and 95% Confidence Intervals (95% CIs) for T2DM According to Tertiles of the DII Score (n:80)						
	Model 1		Model 2		Model 3	
DII tertiles	OR (95% CI)	*P*	OR (95% CI)	*P*	OR (95% CI)	*P*
1 (<-0.625)	Reference	-	Reference	-	Reference	-
2 (-0.625-0.046)	4.024 (0.930-17.411)	0.062	4.836 (0.750-31.173)	0.097	5.294 (0.790-35.475)	0.086
3 (>0.046)	8.000 (1.923-33.724)	**0.004**	10.772 (1.646-70.469)	**0.013**	8.566 (1.235-59.437)	**0.030**
DII (as continuous)	2.316 (1.276-4.205)	**0.006**	2.312 (1.094-4.890)	**0.028**	2.043 (0.955-4.372)	0.066

Logistic regression.
Model 1: Crude model.
Model 2: Adjusted for age, physical activity and standardized energy intake.
Model 3: Adjusted for age, physical activity, standardized energy intake and BMI.

subjects in the highest quintile according to the DII scores were at a higher risk of T2DM than those in the lowest quintile.[28] King et al[29] found that the DII was significantly related to both the presence and the severity of diabetes and that with a 1-point increase in the DII score, having >9% HbA1c value increased by 43%. However, in another study conducted in Iran, the DII was not associated with the T2DM incidence.[30] The study mentioned above found that the DII was moderately related to the T2DM risk; however, the results were not statistically significant after multivariable adjustment. The present study indicated that participants with the higher DII score had an 8-fold increased risk of T2DM compared to those with the lowest DII scores.

Regarding the nutrient intake, it was observed that participants who consumed a more pro-inflammatory diet (in the highest tertile) had lower intakes of dietary fiber and some anti-inflammatory vitamins and minerals. Consistent with the findings of the current study, a study conducted in Mexico also reported that the intake of fiber and various vitamins and minerals was lower in the highest quintile than in the lowest quintile.[28] The positive effects of dietary fiber on inflammation have been reported.[9] Indeed, it was shown that dietary fiber and CRP levels are inversely related, and the intake of high dietary fiber both as part of the diet and as a supplement decreased serum CRP concentrations (14% and 18%, respectively).[9] Moreover, previous studies have shown that some vitamins and minerals, particularly antioxidant vitamins, reduce inflammation markers.[31,32] Besides, although it is considered insignificant, the lowest intakes of the fatty acids, including monounsaturated fatty acid (MUFA), polyunsaturated fatty acid (PUFA), omega 3, and omega 6, which are considered to be anti-inflammatory, were in the highest tertile in the present study. Consistent with the findings of the current study, a cross-sectional study that investigated the DII and dietary habits in individuals with T2DM also reported similar results.[33] Additionally, there have also been several studies conducted with different populations that obtained similar results.[28,34]

One of the possible mechanisms of the association between the DII and diabetes risk may be that a pro-inflammatory diet causes insulin resistance by affecting

levels of various inflammatory cytokines. Previous studies have shown positive associations between various inflammatory markers (CRP, TNF, IL-6, etc.) and insulin resistance.[5,35] The DII, which measures the dietary inflammatory potential, may also be positively related to insulin resistance, which is connected with inflammatory processes through inflammation; thus, this association may affect the diabetes risk.[8,28,33] These findings that support this association were also obtained in the present study. It was observed that the DII scores were significantly related to HOMA-IR, the biochemical marker of insulin resistance, even after the multivariable adjustment. There have also been various studies that obtained findings similar to the present study.[8,36] A study investigated the dietary inflammatory potential using the ADII (Adjusted-Dietary Inflammatory Index), which was developed with some modifications in the DII scoring algorithm, found a significant association between the DII scores and HOMA-IR and that the association between diet and insulin resistance was slightly mediated by inflammation.[15] Another study conducted with South African women also found a positive relationship between the DII and all glucose metabolism markers, including HOMA-IR.[36] There have also been some studies that reported different findings than those of the present study. The ORISCAVLUX (Observation of Cardiovascular Risk Factors in Luxemburg) survey found no significant association between the DII scores and any of the glucose metabolism markers.[22] Another study found that high DII scores were related only to postprandial blood glucose among the glucose metabolism markers.[21] These different findings may have been obtained due to the study designs, study populations, and the type and number of parameters used to calculate the DII scores.

The present study has several strengths to be noted. First, this is the first case–control study investigating the association between the DII and the risk for T2DM among obese women. In addition, the study control group was selected attentively. Controls were selected among obese individuals to understand any effect that may be caused by obesity and to see better the effect of diabetes in the association between the DII and diabetes among obese women individuals. Also, the researchers tried to ensure homogeneity in the study by keeping the inclusion criteria of age and BMI within narrow intervals. Since the participants were women, an age limitation was implemented to eliminate the effects of menopause-related physiological changes. Moreover, although FFQ may lead to measurement error, even in healthy individuals, its use in the present study enabled the researchers to reach many of the food parameters required to calculate the DII. In many previous studies, fewer parameters were used.[28,29] Despite these strengths, some limitations should be acknowledged. First, since the study was based on observational data, the cause–effect relationships cannot be inferred. Therefore, the findings need to be further evaluated in future longitudinal studies. Second, its small sample size may have affected the statistical power to determine some effects. Third, although a detailed FFQ was used to determine the intake of food parameters utilized to calculate the DII scores, using FFQ that is based on the individuals' memory, may cause possible misreporting. Finally, the study population consists of exclusively women. This limitation may be considered minor since no gender differences in biological mechanisms have been reported to date.

In conclusion, the current study provided evidence that women consuming a pro-inflammatory diet with higher DII scores had a higher risk of diabetes compared to women with lower DII scores consuming a more anti-inflammatory diet. In addition, the DII scores and insulin resistance were positively related. Given these findings, adopting an anti-inflammatory diet may be an important approach in preventing insulin resistance and reducing the risk of diabetes. Further longitudinal studies are needed to determine the causality between the DII and diabetes. Such studies may test whether the DII would be useful in practice, and especially whether a diet model created considering the DII parameters would reduce inflammation and the risk of diabetes. Finally, the DII may be an essential tool to characterize the diet of populations. It may be expanded further in clinical researches to reduce the risk of diseases related to chronic inflammation.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of University of Health Sciences, Dışkapı Yıldırım Beyazıt Training and Research Hospital (Date: August 26, 2019, Number: 70/04).

Informed Consent: Written informed consent was obtained from all participants who participated in this study.

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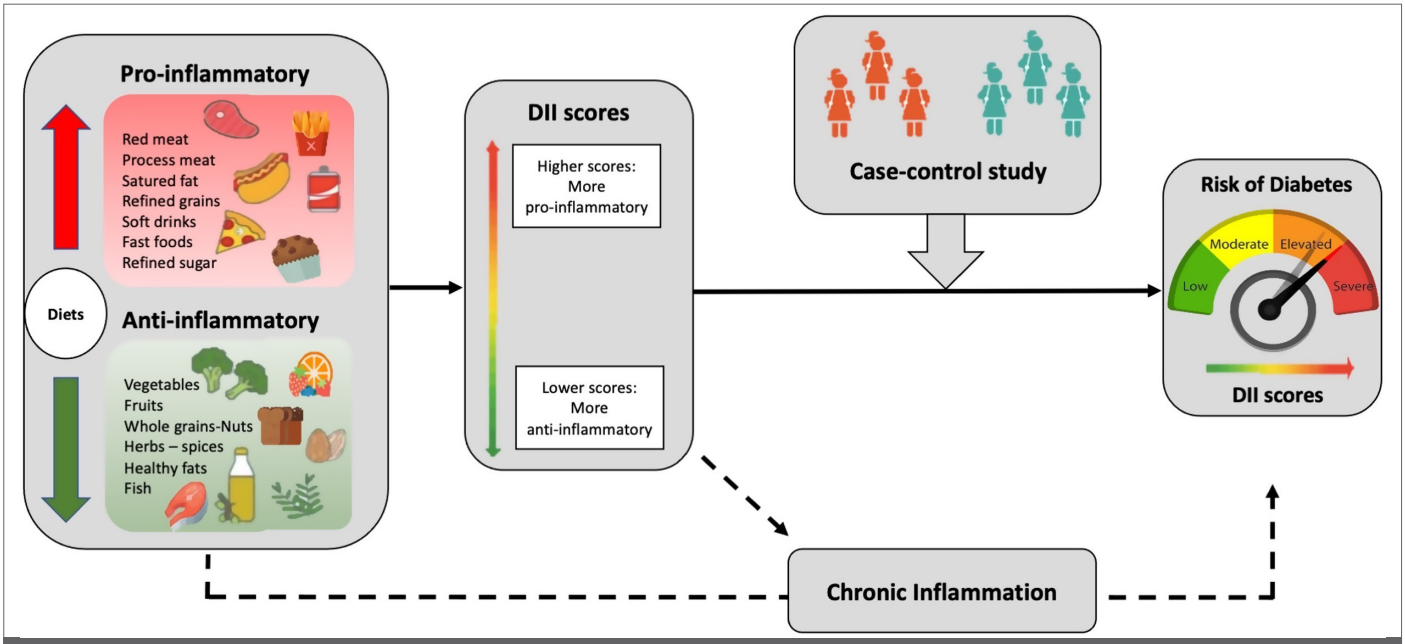

Supplementary Figure 1. Graphical abstract of the study.

CLINICAL SCIENCE OF NUTRITION

Original Article

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Mini Nutritional Assessment-Short Form and Frailty Screening According to 2 Different Frailty Scales

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ABSTRACT

Objective: Mini Nutritional Assessment-Short Form is a malnutrition screening scale that evaluates the patient from different perspectives and is thought to be used in frailty screening. The present study aimed to evaluate the reliability of Mini Nutritional Assessment-Short Form for frailty screening by using 2 frailty scales.

Methods: It was a cross-sectional study that included patients admitted to the geriatric medicine outpatient clinic of a university hospital. Mini Nutritional Assessment-Short Form was performed on all patients. The FRAIL Scale and Clinical Frailty Scale were used as reference frailty scales.

Results: While 62.2% (n = 61) of the 98 participants were female, the median age was 72 (interquartile range: 10.0). The FRAIL Scale (Spearman rho: −0.64, $P < .001$) and Clinical Frailty Scale (Spearman rho: −0.55, $P < .001$) were both correlated inversely and moderately with Mini Nutritional Assessment-Short Form. For both frailty scales, Mini Nutritional Assessment-Short Form cut-off for frailty identification was 11 (for FRAIL Scale, sensitivity: 68.00%, specificity: 87.67%, area under the curve: 0.83, $P < .001$; for Clinical Frailty Scale, sensitivity: 76.47%, specificity: 83.95%, area under the curve: 0.84, $P < .001$), and the cut-off was 13 for robust and pre-frail/frail identification (for FRAIL Scale, sensitivity: 71.70%, specificity: 73.33%, area under the curve: 0.80, $P < .001$; for Clinical Frailty Scale, sensitivity: 71.74%, specificity: 67.31%, area under the curve: 0.74, $P < .001$).

Conclusion: For quick evaluation of frailty and nutritional status concurrently, Mini Nutritional Assessment-Short Form may be an appropriate option.

Keywords: Frail elderly, frailty, malnutrition

INTRODUCTION

Malnutrition is a state resulting from insufficient intake of nutrients and energy that could lead to vitamin and mineral deficiencies, altered body composition (decreased fat-free mass), and body cell mass, resulting in diminished physical and mental function and impaired clinical outcomes from disease.[1] Individuals with malnutrition are at risk for sarcopenia, frailty, and increased mortality.[2] Various screening tools have been developed to diagnose the risk of malnutrition.[3] One of the tools is the Mini Nutritional Assessment-Short Form (MNA-SF) which evaluates the patient in many aspects, such as body mass index (BMI), weight loss, decreased food intake, neuropsychological problems, psychological stress or acute illness, and mobility.[4]

Frailty is a condition that decreases the appropriate response to stressors with the decrease of multiple physiological systems and increases the risk for adverse health outcomes. As frailty progresses, the risk of developing adverse health outcomes increases.[5] The risk of frailty climbs up with advancing age. Frailty ratios vary between 4% and 59% in community-dwelling older adults.[6] It is necessary to evaluate frailty in terms of physical, social, cognitive, and psychological aspects, but the number of scales evaluating frailty in various aspects is low.[7]

Frailty and malnutrition are geriatric syndromes and are common in the aged population. It is valuable that they are screened concurrently for the management of patients.[8,9] Based on this, it has been investigated that MNA-SF, a malnutrition screening tool, can be used in frailty screening

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due to conditions that increase the level of frailty, such as weight loss, low BMI, mobility, dementia, depression, psychosocial stress, and decreased food intake. Using Fried's frailty phenotype (FFP), the cut-off score of 11 for MNA-SF was appropriate for frailty identification.[10] The present study was designed to examine the relationship between MNA-SF and frailty and aimed to evaluate the reliability of MNA-SF for frailty screening by using 2 frailty scales, Clinical Frailty Scale (CFS) and FRAIL Scale (FS).

METHODS

Study Design and Participants

As a cross-sectional study, it was conducted with patients who met the study's inclusion criteria from patients who applied to geriatrics outpatient clinics between 03.01.2022 and 04.03.2022. Inclusion criteria were agreeing to participate in the study, being 65 years and older, and being able to cooperate in the tests. A comprehensive geriatric assessment was performed on all patients. Multimorbidity is the presence of 2 or more chronic diseases.[11] Polypharmacy has been accepted as the usage of 5 or more daily medications.[12] All tests were completed on the same day. Demographic, clinical, and laboratory information of the patients were also recorded.

SCREENING TOOLS

Mini Nutritional Assessment-Short Form

It consists of 6 items as BMI, weight loss in the last 3 months, psychological stress or acute illness in the last 3 months, mobility status, neuropsychological problems such as dementia and depression, and decreased food intake in the last 3 months due to loss of appetite, digestive problems, or chewing or swallowing difficulties. Weight loss and BMI are scored between 0 and 3, and other items are scored between 0 and 2. The maximum score is 14. A score of 12 or more is considered normal nutrition status, 7-11 is considered malnutrition risk, and 7 or under is considered malnutrition.[4] The Turkish validity and reliability study was performed by Sarikaya and colleagues.[13]

Main Points

- Frailty and malnutrition are common, interrelated conditions.
- Screening for malnutrition and frailty together can help healthcare professionals.
- Mini Nutritional Assessment-Short Form is valid and reliable for frailty screening according to reference scales.
- Mini Nutritional Assessment-Short Form may be an appropriate option for quick evaluation of frailty and nutritional status concurrently.

FRAIL Scale

It consists of 5 items and is scored according to the answers given by the patient. Fatigue is interpreted according to the response to "How much time during the previous four weeks did you feel tired?". Answer options were "1 = All of the time, 2 = Most of the time, 3 = Some of the time, 4 = A little of the time, and 5 = None of the time." Answers 1 and 2 are scored as 1 point. Resistance is evaluated according to "By yourself and not using aids, do you have any difficulty walking up ten steps without resting?". "Yes" response is scored as 1 point. Ambulation scored according to the response: "By yourself and not using aids, do you have any difficulty walking several hundred yards?". "Yes" answer is scored as 1 point. Illnesses are evaluated by illness number. "Did a doctor ever tell you that you have hypertension, diabetes, cancer (other than a minor skin cancer), chronic lung disease, heart attack, congestive heart failure, angina, asthma, arthritis, stroke, and kidney disease?" question is asked to the patients. Having 5 or more illnesses is 1 point. Loss of weight item is interpreted based on weight loss in 1 year. First "How much do you weigh with your clothes on but without shoes?" is asked. Second "One year ago in (MO, YR), how much did you weigh without your shoes and with your clothes on?" is asked. After the answers are received, the weight loss ratio is calculated. Percent change > 5 (representing a 5% loss of weight) is scored as 1 point. Zero-point is considered robust, 1 and 2 points pre-frail, and 3 or more points frail.[14] Turkish reliability and validity study was performed by Hymabaccus.[15]

Clinical Frailty Scale

It was developed for the Canadian Study of Health and Aging. A scoring system is based on clinical judgment by interpreting cognition, physical activity, functional dependence, and disease symptoms. Points are regarded as 1: very fit, 2: well, 3: managing well, 4: vulnerable, 5: mildly frail, 6: moderately frail, 7: severely frail, 8: very severely frail, and 9: terminally ill.[16] Reliability and validity study on the Turkish geriatric population was conducted by Özsürekci and colleagues.[17]

Some of the reasons for selecting these frailty scales as references are that their Turkish validity and reliability have previously been proved, they show high performance in predicting adverse health outcomes, they do not require an instrument, and they can be performed quickly.

Ethical Approval

The Non-interventional Clinical Researches Ethics Board of Hacettepe University Faculty of Medicine approved the study (Date: 22.03.2022, Decision Number: 2022/03-14). All subjects signed consent forms.

Statistical Analysis

Data were analyzed using Statistical Package for the Social Sciences 24.0 (IBM Corp., Armonk, NY, USA). By making descriptive statistics, categorical variables were expressed as numbers and percentages, and numerical variables were expressed as mean and SD or median and interquartile range (IQR) according to the normal distribution status. Pearson or Spearman tests were used according to the normal distribution status to evaluate the correlation. Using receiver operating characteristics (ROC), the cut-off value, sensitivity, and specificity values of MNA-SF were determined, which are suitable for identifying frailty. A *P*-value of <.05 was accepted to be statistically significant.

RESULTS

While 62.2% (n = 61) of the 98 participants were female, the median age was 72 (IQR: 10.0). The mean BMI was 30.11 ± 5.72. In frailty scales, the median of FS was 1.0 (IQR: 3.0) and the median of CFS was 3.0 (IQR: 1.0) (Table 1).

Mini Nutritional Assessment-Short Form cut-off points were examined for frailty identification with the ROC curve. For both frailty scales, the MNA-SF cut-off for frailty identification was 11, and the cut-off was 13 for robust and pre-frail/frail identification. The area under the curve, sensitivity, and specificity values are indicated in Table 2 and Figure 1.

DISCUSSION

The relationship of MNA-SF with 2 different frailty scales was revealed in the present study. The FS and CFS show a negative correlation with MNA-SF and the MNA-SF cut-off point of 11 was appropriate in terms of sensitivity and specificity in identifying frailty for both frailty scales. When FS and CFS are used as reference scales, MNA-SF appears to be valid and reliable in identifying frailty.

Table 1. Demographic, Clinical, and Laboratory Characteristics of Patients

	N = 98 (n, %)
Age (years) (median, IQR)	72.0 (10.0)
Sex (female)	61 (62.2)
Education (≤5 years)	64 (65.3)
Body mass index (kg/m^2) (mean, SD)	30.11 ± 5.72
Smoking	37 (37.8)
Multimorbidity (≥2 diseases)	67 (68.4)
Polypharmacy (≥5 medicines)	52 (53.1)
Drug number (median, IQR)	5.0 (3.0)
MNA-SF (median, IQR)	13.0 (4.0)
FRAIL scale (median, IQR)	1.0 (3.0)
CFS (median, IQR)	3.0 (1.0)
Comprehensive geriatric assessment	
Urinary incontinence	38 (38.8)
Falls	21 (21.4)
Katz ADL (median, IQR)	6.0 (1.0)
MMSE (median, IQR)	28.0 (4.3)
GDS-15 (median, IQR)	2.0 (6.0)
SARC-F (median, IQR)	1.0 (3.0)
Grip strength (kg) (mean, SD)	Females: 17.87 ± 5.09, Males: 27.84 ± 6.99
Gait speed (m/s) (median, IQR)	0.94 ± 0.35

ADL, activities of daily living; CFS, Clinical Frailty Scale; GDS-15, Geriatric Depression Scale-15; IQR, interquartile range; kg, kilogram; m, meter; MMSE, mini-mental state examination; MNA-SF, Mini Nutritional Assessment-Short Form; N, number; s, second; SARC-F, Strength, Assistance in walking, Rise from a chair, Climb stairs, Falls; FRAIL, Fatique, Resistance, Ambulation, Illnesses, Loss of weight.

Table 2. Sensitivity and Specificity of MNA-SF

MNA-SF Cut-Off	Frailty Scale	Diagnosis	AUC	*P*	Sensitivity (%)	Specificity (%)
11.0	FRAIL	Frail	0.83	<.001	68.00	87.67
	CFS	Frail	0.84	<.001	76.47	83.95
13.0	FRAIL	Pre-frail/frail	0.80	<.001	71.70	73.33
	CFS	Pre-frail/frail	0.74	<.001	71.74	67.31

AUC, area under the curve; CFS, Clinical Frailty Scale; MNA-SF, Mini Nutritional Assessment-Short Form.

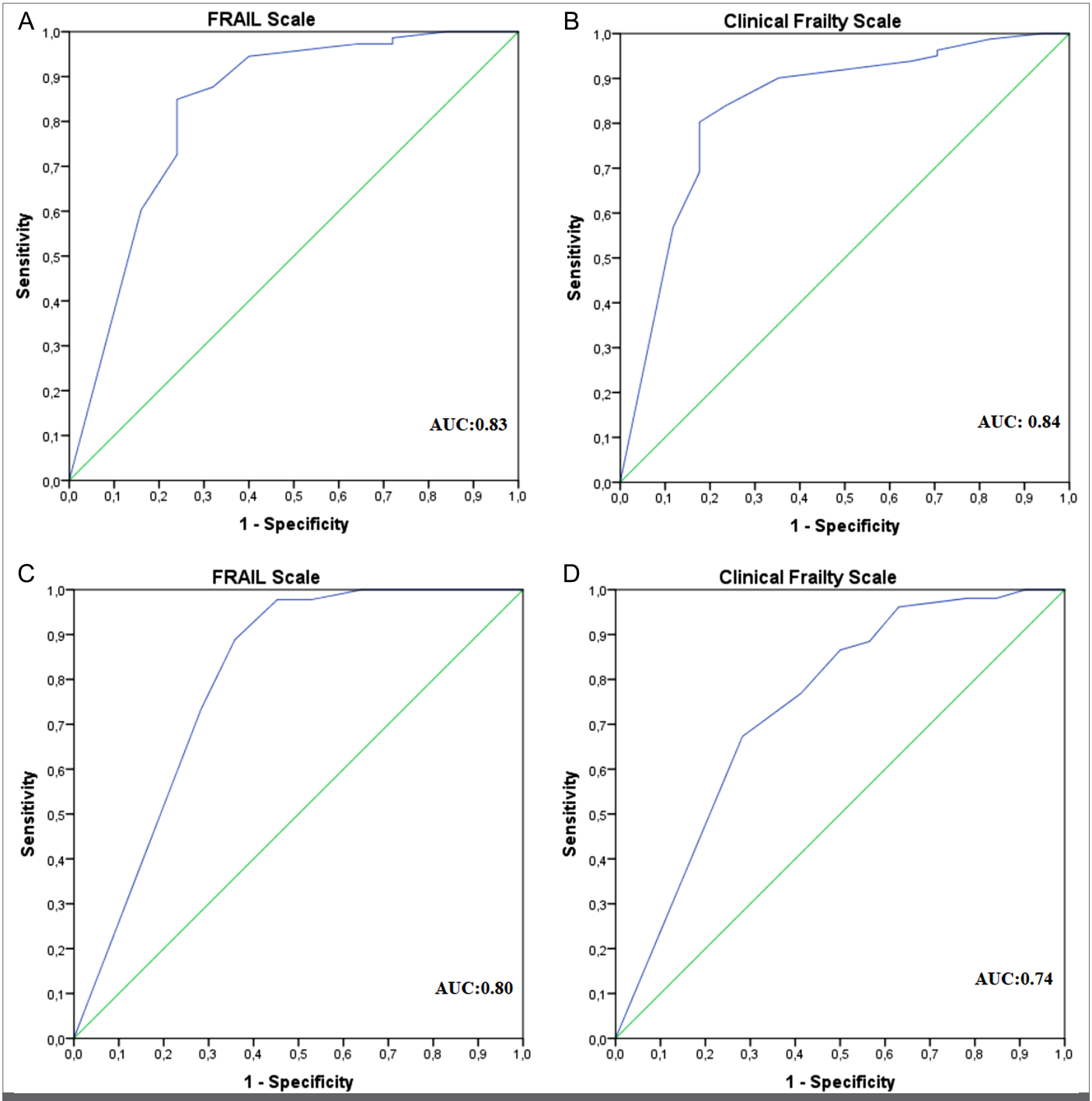

Figure 1. Receiver operating characteristic curve (ROC) analysis of the MNA-SF to detect (A, B) frailty and (C, D) pre-frailty/frailty. AUC, area under the curver; MNA-SF, mini-nutritional assessment-short form

Frailty is a complex condition with physical, social, psychological, and cognitive components.[18] To evaluate this multi-component condition, many scales have been developed. There are many variables like weight loss, weakness, slow walking, cognition, number of medications, use of medications, social relations, number of hospitalizations, functional independence, number of chronic diseases, disease symptoms, vision and hearing functions, falls, age, and gender in these scales.[19] The most significant characteristic connected to malnutrition among these frailty variables is weight loss.[20] Weight loss is one of the indicators of malnutrition and frailty coexistence[8,21,22] that has a negative impact on the patients' clinical course.[23,24] This strong relationship is also seen in the present study. The majority of malnourished or at malnutrition risk patients are pre-frail or frail.

Mini Nutritional Assessment-Short Form is a valid malnutrition screening scale developed from Mini Nutritional Assessment to use the time effectively.[4] It can be used in hospitalized, frail (outpatient/home care/institutionalized), and community-dwelling older adults.[25] Besides malnutrition, it predicts other adverse health outcomes. It can predict post-operative delirium,[26] prolonged length of hospital stay, complications, and mortality.[27] Considering the role of MNA-SF in evaluating these different adverse health outcomes and evaluating the patient in many different aspects with 6 different parameters, MNA-SF was proposed as a potential tool for frailty screening. As a result of the study conducted by Soysal et al,[10] MNA-SF could be used for frailty assessment due to the evaluation made with reference to FFP. In the present study, the MNA-SF cut-off point for frailty identification was 11, and the MNA-SF cut-off for pre-frail/frail identification was 13 for both frailty scales. The same cut-off results were obtained in the previous study with FFP by Soysal et al.[10] This state reveals that MNA-SF cut-off points do not differ between frailty scales. In addition, there is a moderate negative correlation between frailty scales and MNA-SF.

Items of MNA-SF play an essential role in MNA-SF's ability to recognize frailty. Weight loss and decline in food intake are among the questions asked while performing MNA-SF. These 2 items are closely related to frailty as well as malnutrition.[28,29] As age progresses, health problems that reduce mobility such as impaired strength and balance, joint diseases, heart failure, dementia, depression, Parkinson's disease, and cerebrovascular events increase. The mobility limitation is also becoming more frequent for these reasons.[30] This item, used in MNA-SF, gives a strong opinion on frailty. The examination of neuropsychological problems is valuable for evaluating conditions closely related to frailty, such as the presence of dementia and depression.[31,32] Other parameters that increase frailty include acute hospitalization and psychological stress.[33,34] So, MNA-SF has essential questions for assessing frailty, and the present study shows the relationship between MNA-SF and frailty.

The study has some limitations. First, it is a single-center cross-sectional study. The second disadvantage is the limited number of patients. On the other hand, its strengths are the use of frailty scales whose relationship with MNA-SF has rarely been evaluated before, including a frailty scale that evaluates cognitive function.

In conclusion, it has been shown that MNA-SF can be used in frailty screening by comparing it with 2 different scales. Mini Nutritional Assessment-Short Form may be a suitable choice for a quick assessment of frailty and nutritional status simultaneously.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Hacettepe University (Date: 22.03.2022, Number: 2022/03-14).

Informed Consent: Written informed consent was obtained from all patients who participated in this study.

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CLINICAL SCIENCE OF NUTRITION

Original Article

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A Retrospective Analysis of All-Cause Mortality After Percutaneous Endoscopic Gastrostomy in a Single Center

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ABSTRACT

Objective: Percutaneous endoscopic gastrostomy is an effective and safe way of delivering enteral nutrition. Neurological diseases and malignancies are the leading indications. This study aimed to determine the mortality rates after percutaneous endoscopic gastrostomy placement by comparing age groups.
Methods: This retrospective cohort study included patients with percutaneous endoscopic gastrostomy placements between 2019 and 2022 in a single center. The date of percutaneous endoscopic gastrostomy placements and deaths were recorded. Patients were categorized according to age as follows: <65 years, 65-74 years, 75-84 years, and 85 years and over.
Results: A total of 476 patients were included. The median age was 79.0 (range 18-97), with 59.9% being female. The leading indications were neurological diseases (91.0%), and malignancies (5.0%). Of the 476 patients, 14.7% were <65 years, 20.6% were between 65 and 74 years, 37.4% were between 75 and 84 years, and 27.3% were 85 years and over. About 13.2% of patients died within 2 weeks and 67.2% of patients died within 12 months. About 8.6% of patients aged <65 years and 17.7% of patients aged 85 years and over died within 2 weeks. On the other hand, 60.0% of patients aged <65 years and 67.2% of patients aged 85 years and over died within 12 months. Approximately half of the patients (48.3%) died within 3 months.
Conclusion: In this study, almost half of the patients died within 3 months. The mortality rate of patients aged 85 years was higher in the short term. Current data for all institutions should be defined, and future strategies should be targeted. High-quality, controllable nutrition support teams are essential.

Keywords: Mortality, nutrition support team, percutaneous endoscopic gastrostomy

INTRODUCTION

Percutaneous gastrostomy tube placement is an effective and safe way of delivering enteral nutrition. Percutaneous endoscopic gastrostomy (PEG) is usually applied when the patient is expected to need enteral nutrition for longer than 4-6 weeks.[1] The classic indication for PEG tube placement is dysphagia secondary to neurological disorders, head and neck or esophageal cancer, and dementia.[2] According to the European Society for Clinical Nutrition and Metabolism (ESPEN) guidelines on home enteral nutrition (HEN), PEG may be required for swallowing disorders due to neurological diseases, malignancies, cachexia, chronic obstructive pulmonary disease, heart disease, chronic infections, and malabsorption/maldigestion. If life expectancy is expected to be less than 1 month, HEN is usually not recommended to be initiated.[3]

The choice of appropriate patients for PEG placement with accurate indication and in time is the cornerstone of this issue. There are guides published by the ESPEN and The Society of Clinical Enteral Parenteral Nutrition (KEPAN).[3-5] Old age, dementia, low body mass index, high anesthetic risk, hypoalbuminemia, and high Charlson comorbidity index have been defined as potential predictive factors for mortality after PEG placement.[1] In general, it is known that survival after placement of a PEG in geriatric patients is poor. In a meta-analysis, survival was

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reported as 81% after 1 month, 56% after 6 months, and 38% after 1 year.[6] There are so many factors affecting survival rates that they change widely from country to country and even from one hospital to another in the same city. Therefore, the existence of a nutrition support team (NST) is crucial. For example, in a recently published retrospective, large-sample cohort study, the post-PEG mortality rate has decreased by approximately 40% over the last 10 years with the existence of NST.[7]

This retrospective study aimed to determine the mortality rates after PEG placement by comparing the age groups. In this way, we planned not only to encourage health-care professionals to see the mistakes and difficulties but also to promote high-quality NSTs.

METHODS

This retrospective, cohort study was carried out in a single-center hospital. The Eskişehir City Hospital ethics committee approved the study (Decision date: April 19, 2023; decision number: ESH/GOEK 2023/19). The medical records of 544 PEG placements between 2019 and 2022 were taken into consideration. Patients who applied for tube changes were excluded. In that time period, an effective NST was absent. The decisions for PEG placement in the series depended on the request of the primary physician.

Patients were divided into groups according to age categories, defined by the World Health Organization as follows: <65 years, 65-74 years, 75-84 years, and 85 years and over. The survival of all participants was recorded in the Turkish national death registry from the last PEG procedure date until they died or at the end of January 2023.

Statistical Analysis

Statistical Package for the Social Science 23.0 (IBM SPSS Corp., Armonk, NY, USA) was used for statistical analysis. The normality tests of variables were performed using visual (histograms and probability plots) and analytical methods (Kolmogorov–Smirnov/Shapiro–Wilk's test). Categorical variables are summarized as counts and percentages. Mean ± SD and median (25 percentile-75 percentile) were used to present normally and non-normally distributed variables, respectively. The chi-square test or Fisher exact test, where appropriate, was used to compare proportions. Kaplan–Meier survival analysis was performed, and groups were compared with the log-rank test. Kaplan-Meier survival plots were presented as a figure according to age categories. The *P*-value of <.05 was considered statistically significant.

Main Points

- The leading indications for percutaneous endoscopic gastrostomy (PEG) were neurological diseases (91.0%) and malignancies (5.0%).
- Almost a quarter of patients (26.7%) died within 1 month, and half of the patients (48.3%) died within 3 months.
- This study highlighted the high all-cause mortality rates after PEG regardless of the disease or indication.
- The choice of appropriate patients for PEG placement with an accurate indication and in time is crucial.

RESULTS

After excluding duplicate cases, a total of 476 patients were included in the final analysis. The median age was 79.0 (range 18-97), with a 59.9% (n = 285) female rate. The median (25 percentile-75 percentile) follow-up duration was 74 (26-248) days. The majority of patients (91.0%) received PEG placement due to chronic neurological diseases (mainly including stroke, dementia, and Parkinson's disease). Malignancy was the second leading indication (5.0%) (Table 1). Of the 476 patients, 14.7% were <65 years, 20.6% were between 65 and 74 years, 37.4% were between 75 and 84 years, and 27.3% were 85 years and over. All patients were followed up for at least 1 year, other than death.

Two-week, 1-month, 3-month, 6-month, and 12-month mortality rates of patients according to age categories are presented in detail in Table 2. About 13.2% of patients died within 2 weeks and 67.2% of patients died within 12 months. Approximately half of the patients died within 3 months. About 8.6% of patients, aged <65 years, and 17.7% of patients, aged 85 years and over, died within 2 weeks. On the other hand, 60.0% of patients, aged <65 years, and 67.2% of patients, aged 85 years and over, died within 12 months. Kaplan–Meier survival graphs of patients according to age categories are given in Figure 1.

DISCUSSION

In this study, we presented the mortality rate after PEG placement, performed between 2019 and 2022, in a single center. About 27.3% of patients were aged 85 years and older. Sixty-three of the patients (13.2%) died within 2 weeks after PEG. Therefore, almost a quarter of patients (26.7%) died within 1 month and half of the patients (48.3%) died within 3 months. Six-month mortality rates were 55.7%, 44.9%, 60.1%, and 70.8% for patients aged <65 years, between 65 and 74 years, between 75 and 84 years, and 85 years and over, respectively. This study highlighted the high all-cause mortality rates after PEG regardless of the disease or indication.

Table 1. Baseline Characteristics of Patients	
	Total (n = 476)
Age, Median (minimum–maximum)	79 (18-97)
Sex, female, n (%)	285 (59.9)
Age categories, n (%)	
• <65	70 (14.7)
• 65-74	98 (20.6)
• 75-84	178 (37.4)
• ≥85	130 (27.3)
Indications	
• Neurological diseases (stroke, dementia, and Parkinson's)	433 (91.0)
• Malignancies	24 (5.0)
• Others	19 (4.0)
Follow-up duration (days), median (25 percentile-75 percentile)	74 (26-248)

In our study, the major indication for PEG placement was chronic neurological diseases, including stroke, dementia, and Parkinson's disease. In a review, 12 studies had neurological disease as the main indication for PEG, and 4 studies had dementia as the main indication for PEG.[2] Similarly, Hasırcı et al[8] presented the data of 386 patients between 2008 and 2020 with a mean age of 70 ± 12.8. The main indication for PEG was neurological disease (84%). That was close to our data. They also found the mortality rates for 1 week, 1 month, and 6 months to be 12%, 29%, and 49%, respectively. These rates were lower than those found in our study.

Table 2. Mortality Rates According to Age Categories from 2 Weeks to 12 Months					
	2 Weeks	**1 Month**	**3 Months**	**6 Months**	**12 Months**
All patients	63 (13.2)	127 (26.7)	230 (48.3)	282 (59.2)	320 (67.2)
Age, <65	6 (8.6)	15 (21.4)	32 (45.7)	39 (55.7)	42 (60.0)
Age, 65-74	5 (5.1)	17 (17.3)	37 (37.8)	44 (44.9)	52 (53.1)
Age, 75-84	29 (16.3)	45 (25.3)	84 (47.2)	107 (60.1)	129 (72.5)
Age, ≥85	23 (17.7)	50 (38.5)	77 (59.2)	92 (70.8)	320 (67.2)

In our study, 91% of patients had chronic neurological disease. We had no data about the rate of severe dementia, which may affect the mortality rates. Therefore, higher mortality rates may be due to PEG placement, lately. A Cochrane review found no evidence that tube feeding improves survival or quality of life in patients with severe dementia.[9] Therefore, redundant PEG placement for patients who are expected to survive less than 1 month is a challenging issue. Another systematic review and meta-analysis indicated that tube feeding is associated with an increased mortality rate and tube-related complications.[10]

We found the 30-day mortality rate after PEG to be 26.7%. The 30-day mortality rates were 21.4%, 17.3%, 25.3%, and 38.5% for patients aged <65 years, between 65 and 74 years, between 75 and 84 years, and 85 years and over, respectively. In a recently published systematic review, the 30-day mortality rate varied from 2.4% to

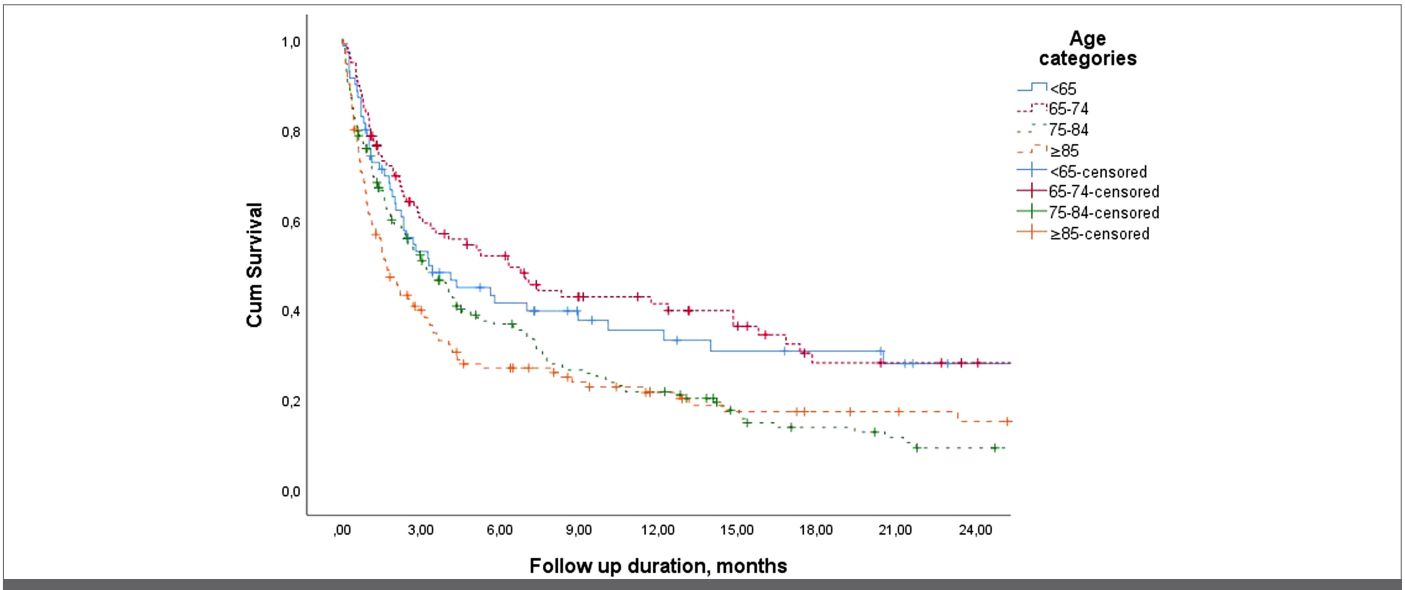

Figure 1. Kaplan–Meier survival analysis of patients.

23.5%.[2] Lima et al[11] evaluated the data of 277 patients. The indications for PEG placement were almost neurological diseases (89.5%) like ours. They found the 30-day mortality rate to be 13%. Duzenli et al reported the rate of 30-day mortality as 12.6% in 253 patients. Another study revealed a high mortality rate after PEG placement, especially within the first month (over 13%). Also, the most frequent indications were neurological diseases and malignancies, like ours.[1] In a study from Turkey about the evaluation of 644 PEG placements, the 30-day mortality rate was 9.7%, and the 1-year mortality rate was 36.4%. Neurological disorders and malignancy were the leading causes.[12] Our 30-day mortality rates were higher than in all of these studies. On the other hand, a study from palliative care (118 patients) reported the 90-day mortality rate after discharge as 40%.[13] In our study, the 90-day mortality was 48.3%.

The all-cause mortality rates after PEG placement should be researched by all institutions, and deficiencies and mistakes should be investigated. High-quality NSTs certified for this area should be built, and control mechanisms should be constituted. Therefore, a coordination and follow-up procedure will reduce not only the complication rates but also the mortality rates after PEG insertion.[3] A retrospective national cohort analysis of 87 862 patients from England reported the 30-day mortality after PEG tube placement from 2007 to 2019. The 30-day mortality rate was found to be 8.9%. It had fallen by 60% over 13 years. According to them, multidisciplinary NSTs provided better patient selection and pre- and post-procedural care.[14] In a recently published retrospective, large-sample cohort study, the post-PEG mortality rate has decreased by approximately 40% over the last 10 years despite apparently similar patient characteristics and rates of complications.[7]

As we stated above, the indications for PEG placement and timing are the main topics. For example, patients with malignancies can receive more benefits than others. In a retrospective study, the all-cause mortality was 15% at 30 days and 28% at 90 days. Malignancy was found to be associated with increased mortality at 90 days. They claimed that patients with malignancies may benefit from an earlier referral for PEG. They also found that older age, diabetes, heart failure, C-reactive protein level, and body mass index are associated with the risk of adverse outcomes, and they suggested considering these in preoperative PEG risk assessment in routine health care.[15] Mortality rates after PEG insertion usually depends on the indication and selection of patients. Although several studies show some improvement in the nutritional state, the effects on functionality, mortality, and quality of life remain unclear.[4] It would be rational to develop disease-specific targets and algorithms after analyzing national data.

Study Limitations

Due to its retrospective design, insufficient and limited data were obtained from medical records. We could not give information about in-hospital and after-discharge mortality separately. There were no data about the patient's risk of malnutrition. We could not find information about PEG-related or hospital-related complications. Future prospective cohort studies should be designed, and all factors affecting the mortality rate should be analyzed in detail. On the other hand, this study provides data from a populous hospital to play a role.

In conclusion, almost a quarter of patients (26.7%) died within 1 month and half of the patients (48.3%) died within 3 months. Current situations for all institutions should be defined, and future strategies should be targeted. High-quality NSTs are essential.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Eskişehir City Hospital (Date: April 19, 2023, Number: ESH/GOEK 2023/19).

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Original Article

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How Ginger Influences Blood Lipid Levels in Individuals Who Were Suggested Lifestyle Change by Systematic Coronary Risk Evaluation?

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ABSTRACT

Objective: This study was performed to investigate the effect of powdered ginger supplemented to the diet on blood lipid indices in individuals with moderate physical activity that are recommended only lifestyle intervention by Systematic Coronary Risk Evaluation.
Methods: In this exploratory experiment, individuals were divided into 2 groups as ginger supplementation (n = 20) and control (n = 20). The ginger supplementation group received 1 ginger capsule (400 mg ginger extract, 80 mg gelatin) twice a day for a month. Blood lipid levels (total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol) and systolic blood pressure were measured, and Systematic Coronary Risk Evaluation values were calculated.
Results: The systolic blood pressure and Systematic Coronary Risk Evaluation values in the ginger supplementation group were decreased ($P < .05$). The systolic blood pressure values of the ginger supplementation group before and after the study were higher than the control group ($P < .05$). Systolic blood pressure values in both groups were decreased compared to previous levels. Before and after the study, the Systematic Coronary Risk Evaluation values of the ginger supplementation group were found to be lower than that of the control group ($P < .05$). The study showed that 85% of the ginger supplementation group remained in the category requiring a lifestyle change and possibly drug treatment, whereas 15% of the individuals moved to the category in which no intervention was required for lipid levels. However, 90% of the participants of the control group remained in the lifestyle change category.
Conclusion: The results of the current study implicate that consumption of ginger might be beneficial to reduce the risk of cardiovascular disease and further studies are needed to explore this effect in more detail.

Keywords: Blood lipid levels, coronary risk score, ginger

INTRODUCTION

Although progress has been made regarding the elucidation of coronary risk factors in the world, cardiovascular diseases (CVDs) are still among the most common causes of death. In 2012, 46.2% (17.5 million) of deaths were due to noncommunicable diseases (NCDs) worldwide, with 7.4 million deaths due to heart attack (ischemic heart disease) and 6.7 million due to stroke. Cardiovascular diseases are responsible for 37% of deaths due to NCDs under the age of 70 years. Deaths caused by CVDs are estimated to reach 22.2 million by 2030.[1] Hyperlipidemia is an important risk factor for the prediction of CVD. There is a strong and sustained relationship between total cholesterol (TC) or low-density lipoprotein cholesterol (LDL-C) level and CVD. In general, a 1% increase in LDL-C raises the risk of CVD by 2%-3%.[2] Controlling TC and LDL-C levels through dietary measures is the primary target of preventing CVD. Because skewed plasma lipid and lipoprotein levels, obesity, and high blood pressure are the main cardiovascular risk factors, the role of diet in CVD has been established; therefore, the diet should be modified in terms of components that increase and reduce these risk factors.[3] Ginger is a reliable, easy-to-tolerate

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herb used in the kitchen and also has an important place in the Ayurveda, Chinese, German, and Arabic medicine systems;[4,5] it contains phenolics such as shogaol and gingerol as potential active substances; sesquiterpenes such as bisabolene, zingiberene, zingiberol, sesquiphellandrene, curcurmene; and also[6] dehydrogingerdione, galanolactone, gigasulfonic acid, geraniol, neral, monoacyldiglycerides, glycolipids and gingerol as other active compounds. The active ingredient of ginger is in the essential oil part, and the main active ingredients are bisabolene, zingiberene, and zingiberol. Various mechanisms have been proposed to explain the positive effect of ginger on blood lipid levels. It has been suggested that ginger impairs and weakens the gastrointestinal absorption of cholesterol.[7-9] Phytochemicals (E)-8 β and 17-ep oxylabd-12-ene-15, 16-dial in ginger were reported to inhibit cholesterol synthesis in the liver in rats. Moreover, ginger supports the uptake and catabolism of LDL-C from the circulation; it inhibits LDL-C oxidation and aggregation.[7] It has been demonstrated that the addition of ginger to the diet reduces the expression of retinol-binding protein and fatty acid-binding protein genes in the liver and adipose tissue of rats.[10] In a study, 400 mg/kg ethanolic ginger extract was given to rats for 6 weeks. At the end of the study, the LDL receptor messenger ribonucleic acid level increased in the liver and decreased expression of 3-hydroxy-3-methylglutarylcoenzyme A reductase protein has been observed.[11] It has been claimed that ginger inhibits cellular synthesis of cholesterol by suppressing hydroxymethylgultaryl Co-A reductase, which is the rate-regulating enzyme in cholesterol metabolism. It inhibits cholesterol 7 alpha-hydroxylase, which is the key enzyme in the conversion of cholesterol to bile acids. It has been suggested that ginger increases the fecal excretion of cholesterol.[7,12] Although recent preclinical studies have shown that ginger reduces serum TC, LDL-C, and TG levels and simultaneously increases HDL-C levels, the results are still contradictory.[4-6]

Main Points

- This study showed that 85% of the individuals receiving 400 mg/day ginger extract remained in the category needing to make a lifestyle change and possibly will need drug treatment, whereas 15% of the individuals shifted to the category that did not require any intervention for lipid levels.
- Incorporating ginger into the diet reduces systolic blood pressure and Systematic Coronary Risk Evaluation values, possibly contributing to the prevention of cardiovascular disease.
- Consumption of ginger, 400 mg ginger extract might be beneficial to reduce the risk of cardiovascular disease risk factors such as systolic blodd pressure

For this reason, this intervention study was conducted to reveal the effect of incorporating ginger into the nutrition plan on anthropometric measurements, blood lipid levels, and Systematic Coronary Risk Evaluation (SCORE) values solely in individuals who were recommended lifestyle intervention depending on the results of SCORE.

METHODS

Participants and Study Design

To conduct a comprehensive power analysis, we first obtained the necessary information from the study of Alizadeh Navaei et al.[5]. In this case, the predetermined values for beta (β) and alpha (α) were 0.80 and 0.05, respectively, and the number of participants was calculated as 20 per group. Thus, this study was carried out with 40 volunteers showing moderate physical activity who applied to the Cardiology Outpatient Clinic of a Erciyes University between February 2017 and December 2018.

The ethics committee permission was obtained from a local ethics committee, the Erciyes University Clinical Research Ethics Committee (Date: March 3, 2017, Approval No: 2017/120), and informed consent forms were signed by the individuals. The mean age of the individuals was ≥40 (46.15 ± 3.70 years). The participants consisted of 50% women and 50% men. There were no participants with menopause, and only lifestyle intervention was recommended based on SCORE results.

In our study, we employed a simplified method for randomizing our patients into 2 distinct groups: the ginger supplementation group and the control group. This randomization was based on a specific criterion, namely, the last digit of patient barcode numbers. More specifically, patients whose barcode numbers ended with an odd digit were allocated to the intervention group, while those with an even final digit were assigned to the control group.

In previous studies, several doses of ginger were given to participants. According to a meta-analysis,[13] use of a low dose for a short time was effective in improving the lipid profile rather than higher ginger doses. According to the literature[5,14] the individuals in the ginger supplementation group (n = 20; 10 women and 10 men) received 1 ginger capsule (400 mg ginger extract, 80 mg gelatin capsule, Sepe Natural Ginger, Sepe Organic Natural Products Ind. & Trade Co., Turkey) twice a day after meals in the morning and evening for 1 month. Ginger capsules (32 mg gingerol as the active ingredient) were provided by the researcher and given to the participants free of charge. No supplements were given to the control group (n = 20; 10 women and 10 men), and they were asked to maintain

moderate physical activity. Both groups received a diet containing 300 mg of cholesterol per day during the study period. In order to observe the effect of ginger supplementation, no dietary intervention was made, the habits of the patients were questioned, and recommendations were made regarding the foods in the 300 mg nutrition model only. Therefore, dietary compliance was not followed. At the beginning and end of the study, anthropometric measurements and biochemical parameters of the participants were evaluated.

Sociodemographic Features

The sociodemographic information of the participants was obtained through face-to-face interviews with a questionnaire form created by reviewing the literature.[10] Information such as age, education, and smoking status were included in the questionnaire.

Anthropometric Measurements

At the beginning and end of the study, the body weight, height, waist circumference, and hip circumference of the participants were measured and body mass index (BMI), waist–hip ratio were calculated (kg/m²). Based on World Health Organization (WHO) adult BMI classification, those with a BMI below 18.5 kg/m^2 were classified as underweight, those with 18.5-24.9 kg/m^2 as normal, those with 25-29.9 kg/m^2 as slightly obese, and those above 30 kg/m^2 as obese.[16]

Fasting body weights and heights of individuals with light clothing and without shoes were measured in the morning using weight-length counter (150 IB, N, Turkey). The height of the participants was measured while the individuals were standing without shoes with their feet next to each other, with the head in the plane of Frankfort (the eye triangle and the auricle aligned). Waist and hip circumference values were measured while the individuals were standing, with arms open on both sides and feet adjacent to each other.

The waist circumference was measured with the non-stretchable tape measure between the lower rib bone and the caudal fin when the individual breathes out. Measurement was made by taking care that the tape measure is parallel to the ground and does not press on the skin. Hip circumference was determined by measuring the circumference from the side of the individual from the highest point of the hip. The waist−hip ratio was determined by dividing the waist circumference values by the hip circumference values.[15] The reference values determined by the WHO were used in the evaluation of waist circumference and waist−hip ratio measurements. Accordingly, a waist circumference of more than 80 cm in women and 94 cm in men was considered to be risky, and a waist circumference of 88 cm in women and 102 cm in men was considered obese.[16]

Blood Pressure Measurements

Before systolic blood pressure measurement, the researchers ensured that the participants did not smoke cigarettes, drink tea or coffee, consume caffeine and other nutrients within 30 minutes of measurement, and the measurement was performed by the nurse after at least a 5-minute resting period. The individual was asked to sit with their back leaned against the chair with the arm bare during the measurements. Measurements were made at least twice with an interval of 2 minutes and the average value was calculated.[17]

Biochemical Analysis

A 4 mL blood sample was taken from the participants after 12 hours of fasting. The TG, HDL-C, LDL-C, and TC levels were determined by an auto-analyzer (Architect/Aeroset 16000, Abbott, USA).[18]

Systematic Coronary Risk Evaluation

An electronic version, "HeartScore," specially adapted to Turkey is used for the SCORE calculation. HeartScore is the electronic and interactive version of the SCORE risk table of the European Clinical Practice Prevention of CVD, prepared by the Fourth United European Associations Working Group on the prevention of CVD in Clinical Practice.[19] A record for each individual was created in HeartScore. The SCORE values for each individual were calculated by entering the age, gender, smoking status, TC value and systolic blood pressure of the individuals to the system. Individuals with a 10-year calculated risk of cardiovascular death <1% were considered to be at low risk, from ≥1% to <5% were considered to be at moderate risk, from ≥5% to <10% were considered to be at high risk, and ≥10% were considered to be at very high risk.[19,20] Considering the LDL-C values, the low-risk group with a SCORE value of <1%, the medium-risk group with a value of ≥1%-<5%, and the high-risk group with a value of ≥5%-<10% were evaluated.[19] Individuals with SCORE values within the interval of recommended lifestyle intervention were included in the study.

International Physical Activity Questionnaire Short Form

In order to determine the physical activity levels of the participants, the IPAQ Short Form (88), consisting of 7 questions, was applied. Individuals were asked questions about heavy physical activity, moderate physical activity, duration and frequency of walking in the last 7 days. The sum of the values obtained via multiplying the number of days of activity type, daily duration, and the activity coefficient (heavy activity coefficient 8, medium activity

coefficient 4, and walking coefficient 3.3). Individuals with <600 metabolic equivalent of task (MET), those with 600-3000 MET, and those with >3000 MET were considered as inactive, moderately active, and very active, respectively. Individuals with moderate physical activity were included in the study.[14] Individuals were asked not to make radical changes in their physical activity status for a month.

Nutrition Plan

Training was provided to the participants in both groups to implement the nutrition plan including 300 mg of cholesterol per day.[19] The nutritional habits, sociocultural conditions, working conditions, and lifestyles of individuals were taken into consideration for preparing the nutrition plan for each individual. In nutrition education, portion sizes and amounts (grams) of foods were arranged according to the book named "Food and Food Photo Catalog: Measures and Quantities."[16] At the beginning of the study, 30-40 minutes of training was provided to each individual.

Exclusion Criteria

Individuals who were younger than 40; being obese (≥30kg/m²); having one of the diseases such as diabetes, familial dyslipidemia, atherosclerotic heart disease, and chronic kidney failure; using vitamin–mineral tablet, alcohol, or medication; having mild or heavy activity determined with the short form IPAQ[14] were excluded. And individuals who did not need any intervention or who were recommended drug treatment as a result of joint evaluation of SCORE and serum LDL-C levels (7) were not included in the study.

Statistical Analysis

Türkiye National cloud-based statistics software (TURCOUSA) was used for statistical evaluation of the data. The normality of the data was determined with Shapiro–Wilk test. When the data were normally distributed, Student's *t*-test was used for group comparisons and paired *t*-test was used for determination of the differences between the data obtained before and the after the study for each group. When the data were not normally distributed, Mann–Whitney *U*-test was used for group comparisons and the paired samples Wilcoxon test was used for the data obtained before and the after the study for each group. Categorical variables were analyzed with chi-square test. In addition, logistic regression analysis was performed. The results of the analysis were evaluated in the 95% CI and the significance level was accepted as $P < .05$.

RESULTS

The mean ages of the control and ginger supplementation groups were similar (44.80 ± 3.70 and 46.20 ± 5.08,

Table 1. Anthropometric Measurement Values and Smoking Status of the Participants in the Groups Before and After the Study

Anthropometric Measurements	Ginger Supplementation Group (n = 20)	Control Group (n = 20)	
	x ± S	x ± S	P**
Age (years)	44.8 ± 3.7	46.0 ± 5.1	.325
Body weight (kg)			
Before	70.74 ± 9.4	71.6 ± 8.3	.074
After	71.2 ± 9.273	71.52 ± 8.2	.558
P	.754	.902	
BMI (kg/m²)			
Before	26.1 ± 2.7	26.1 ± 2.6	.069
After	26.2 ± 2.7	26.0 ± 2.5	.490
P	1.000	.800	
Waist circumference (cm)			
Before	86.6 ± 8.1	86.3 ± 7.8	.163
After	86. 2 ± 8.1	86.0 ± 7.7	.083
P	.922	.952	
Hip circumference (cm)			
Before	99.1 ± 4.7	98.9 ± 4.1	.179
After	98.5 ± 5.1	98.6 ± 3.9	.069
P	.943	.945	
Waist–hip			
Before	0.87 ± 0.07	0.87 ± 0.09	0.748
After	0.88 ± 0.07	0.87 ± 0.09	0.666
P	.968	.921	
	n = 20	n = 20	Total (n = 40)
	n (%)	n (%)	n (%)
Smoking rate			
Nonsmoker	3 (15)	5 (25)	8 (20)
Regularly	8 (40)	14 (70)	22 (55)
Occasionally	6 (30)	0 (0)	6 (15)
Stopped	3 (15)	1 (5)	4 (10)
Total	20 (100)	20 (100)	40 (100)
	$\chi^2 = 9.1$, $P^* = .133$		

Student's *t*-test was used for group comparisons. Chi-square test was used for smoking status.
BMI, body mass index.
*Within the group. **Between the groups.

respectively) ($P > .05$), In terms of anthropometric measurements, there was no statistically significant difference between the groups before and after the study (Table 1, $P > .05$). In addition, the rate of nonsmokers in the control group (25%) was higher than the ginger supplementation group (15%) but the difference between groups was not significant ($P > .05$) (Table 1).

In the beginning of the study, concerning the TC, LDL-C, HDL-C, and TG levels, there were no significant differences between control and ginger supplementation groups. The systolic blood pressure in the ginger supplementation group before the study was higher than the systolic blood pressure value in the control group ($P < .05$). The SCORE value in the ginger supplementation group before the study was found to be lower ($P < .05$) than the SCORE value in the control group (Table 2, Figure 1, and Figure 2).

At the end of the study, the HDL-C level in the control group was found to be lower than that of the ginger supplementation group ($P < .05$). The systolic blood pressure decreased in both groups, but there was no significant difference between groups. Compared to the control group, the SCORE values in the ginger supplementation group decreased significantly ($P < .05$) (Table 2, Figure 2).

When the values obtained in the beginning and at the end of the study were compared, there were no significant differences in TC, LDL-C, HDL-C, and TG levels in the ginger supplementation group, whereas systolic blood pressure and SCORE values decreased. In the control group, TC and LDL-C levels decreased, but there were no statistically significant differences in HDL-C, TG, systolic blood pressure, and SCORE values.

At the end of the study, 85% of the individuals in the ginger supplementation group remained in the category needing to make a lifestyle change and possibly will need drug treatment, whereas 15% of the individuals shifted their category that did not require any intervention for lipid levels. In the control group, 90% of the participants remained in the category that require to make a lifestyle change, and if it is not controlled, possibly need drug treatment; the remaining 10% shifted to the category that did not require any intervention for lipid levels. Although not statistically significant ($P > .05$), the frequency (15%) of the participants in the ginger supplementation group who shifted to the category that did not need intervention for lipid levels was 10% higher than the control group (Table 3).

In addition, the frequency of individuals whose LDL-C was ≥100, <155 decreased from 90% to 70%. It was determined that the proportion of individuals with very high (≥190) LDL-C levels did not change. While the SCORE of 10% of the individuals remained the same, the LDL-C levels decreased to ≥70, <100 levels (Table 4).

When examining whether the ginger intervention improved blood pressure and blood lipid parameters, analyses showed that the intervention was likely to improve systolic blood pressure (B = 4.33, $P = .03$ for model 0; B = 5.05, $P = .03$ for model 1; Table 5).

Table 2. Serum Lipid Levels, Systolic Blood Pressure, and SCORE Values of the Groups Before and After the Intervention

Variables	Ginger Group (n = 20) x ± SD/Median (IQR)	Control Group (n = 20) x ± SD/Median (IQR)	P**
TC (mg/dL)			
Before	203.7 ± 24.5	194.5 (158-269)	**.570**
After	200.8 ± 25.7	183.5 (165-248)	**.256**
P*	.835	**.018**	
LDL-C (mg/dL)			
Before	127.9 ± 20.0	121.6 (103.4-198.4)	**.787**
After	124.8 ± 20.4	116.2 (92.4-170.8)	**.402**
P*	.869	**.021**	
HDL-C (mg/dL)			
Before	52.8 ± 11.9	43.5 (31-79)	**.140**
After	52.8 ± 12.2	40.9 (31.1-79)	**.027**
P*	.269	.064	
TG (mg/dL)			
Before	114.8 ± 45.6	116.5 (69-213)	**.285**
After	115.9 ± 41.9	129.5 (51-210)	**.323**
P*	.551	.247	
Systolic blood pressure (mm/Hg)			
Before	133.6 ± 17.7	112 (100-166)	**.005**
After	124.8 ± 15.6	117 (100-150)	**.062**
P*	**.012**	.361	
SCORE (%)			
Before	1.0 ± 0.0	1.4 ± 0.8	**.037**
After	0.8 ± 0.4	1.4 ± 0.8	**.005**
P*	**.036**	.682	

Student's *t*-test, Mann–Whitney *U*-test, and the paired samples Wilcoxon test were used.
*Within the group. **Between the groups.

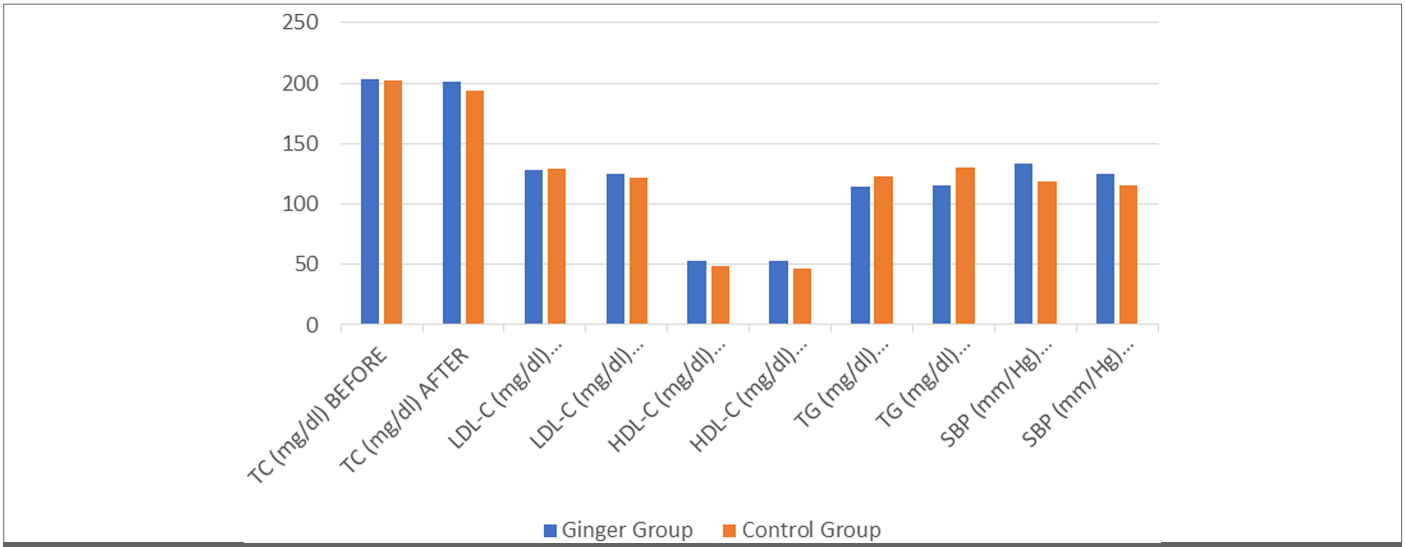

Figure 1. Serum lipid levels and systolic blood pressure values of the groups before and after the intervention.

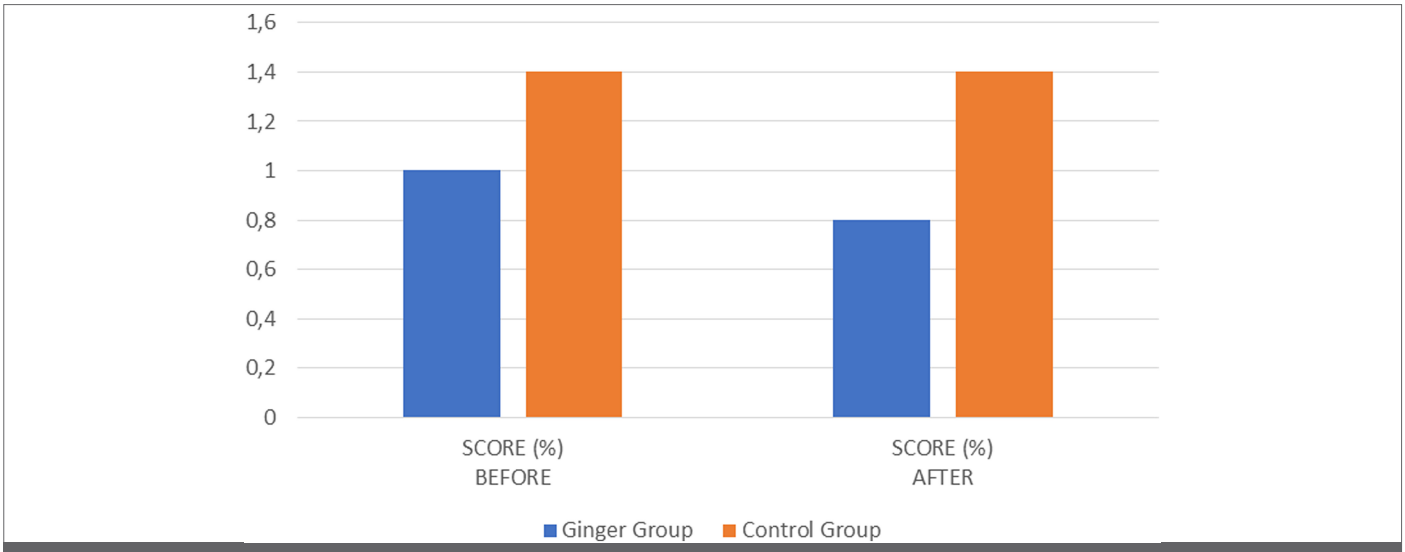

Figure 2. SCORE values of the groups before and after the intervention. SCORE, Systematic Coronary Risk Evaluation.

DISCUSSION

The CVD is a general nomenclature given to the group that includes diseases of the heart or blood vessels (arteries and veins) and is one of the leading causes of morbidity and mortality. According to the data of the WHO, CVDs rank first among global causes of death (31%) and 17.5 million people died in 2012. In Turkey, considering the 10

Table 3. Distribution of Interference Strategies Determined as a Function of SCORE and LDL-C at the End of the Study

	Ginger Group	Control Group	Total	
Interference Strategies	**n (%)**	**n (%)**	**n (%)**	
Lifestyle intervention, consider drug if uncontrolled	17 (85.0)	18 (90.0)	35 (87.5)	$\chi^2 = .663$ $P = .500$
No lipid intervention	3 (15.0)	2 (10.0)	5 (12.5)	
Total	20 (100)	20 (100)	40 (100)	

Chi-square test was used.
LDL-C, low-density lipoprotein cholesterol; SCORE, Systematic Coronary Risk Evaluation.

Table 4. Distribution of the Groups by SCORE and LDL-C Values Before and After the Study

	SCORE (%)							
	Ginger Supplementation Group (n = 20)				Control group (n = 20)			
	Before		After		Before		After	
	<1	1-5	<1	1-5	<1	1-5	<1	1-5
LDL-C	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)	n (%)
70-100 mg/dL				2 (10)				2 (10)
100-155 mg/dL		18 (90)	2 (10)	14 (70)		17 (85)		15 (75)
155-190 mg/dL		2 (10)		2 (10)		2 (10)		3 (15)
>190 mg/dL						1 (5)		

LDL-C, low-density lipoprotein cholesterol; SCORE, Systematic Coronary Risk Evaluation.

diseases that constitute the disability-adjusted life years, ischemic heart diseases (8%) occupy the second place.[21] The 40.4% of the death was due to CVDs in 2014, and the death rate due to CVDs in men was 35.83%, while in women it was 44.61%, with an increase of 0.8% when compared to the rate of 2013. Because increased plasma lipid and lipoprotein levels, obesity, and high blood pressure are explained as the main risk factors for CVD, the type of diet followed has proven to be beneficial for the amelioration of the lipid profiles. It has been shown that 10 g ginger reduced platelet aggregation in coronary artery patients when given at 4 and 10 g/day ginger for 3 months.[5]

The risk of CVD in smokers is 2-3 fold higher than that in nonsmokers. Smoking is responsible for half of the preventable deaths in long-term smokers, and 50% of these deaths are due to CVD. Smoking disrupts the lipid and lipoprotein profiles and causes endothelial dysfunction that results in the progression of atherosclerosis.[22] Cigarette smoking lowers HDL-C and increases the oxidation of LDL-C. The causal role of smoking in CVD has been demonstrated in over 20 million patients with a long time follow-up study.[23] It is known that there were 1.1 billion smokers in the world in 2012.[1] According to the WHO report in 2017, smoking rates in individuals over 15 years of age ranged from 21.9% to 25.9% in Turkey.[25] In the present study, the percentage of regular smokers was higher in the control group (70%) than in the ginger supplementation group (40%), which was considerably above the percentage stated in the WHO 2017 Turkey report. While there were no occasional cigarette smokers in the control group, the proportion of occasional smokers in the ginger supplementation group was 30% (Table 1).

Studies in individuals without CAD have shown that smoking cessation reduces the risk of death and reinfarction by 7%-47%.[25,26] Stopping smoking in patients without apparent disease ensures that the risk of CAD decreases to a nonsmoking level within 10 years.[27] In this study determining the risk of CVD with the SCORE system in healthy adults, although it was not statistically significant, the rate of those who used to smoke was higher in the ginger supplementation group than in the control group (Table 1). In this study, the number of cigarettes smoked per day was found to be significantly higher in the control group (15.21 ± 8.40) than in the ginger supplementation group (6.65 ± 6.1; $P < .05$) (Table 1). Smoking is a health problem that increases the risk and mortality of CVD and is therefore a unique problem.[25]

Exercise or regular physical activity has been shown to positively affect plasma lipid profiles, cardiovascular and pulmonary functional capacity, glucose tolerance, and blood pressure and prevent obesity.[23,25,28] In addition, increasing physical activity can prolong the total life expectancy of 1.3-3.5 years and life expectancy without CVD.[16,29] In a study investigating the effects of a 4-week intervention program consisting of diet restriction and walking activity on the lipid profile in sedentary individuals, significant decreases in TC (35 ± 37 mg/dL), TG (30 ± 68 mg/dL), and LDL-C (29 ± 41 mg/dL) levels were determined.[28]

Healthy individuals who have low physical activity are at twice the risk of CAD than the individuals who do regular physical activity. According to Turkey's Chronic Disease Risk Factors Incidence Study, 23% of men had adequate activity, 22% a moderate level, and 55% a low level of physical activity. These rates are even lower in women.[1] For these reasons, in order to prevent physical activity from affecting the risk of CVD and SCORE values in the present study, individuals with moderate physical activity were included in the study by applying the IPAQ Short Form[13] (ginger supplementation group: 1981.38 ±

Table 5. Effect of Ginger Intervention on Improvement of Blood Lipid and Blood Pressure Parameters

Improvements			Systolic Blood Pressure				Diastolic Blood Pressure				Blood HDL Levels			
			CI (B)	Lower	Upper		CI (B)	Lower	Upper		CI (B)	Lower	Upper	
Ginger intervention	Model 0		4.33	1.15	16.32	**0.03**	2.25	0.63	7.97	0.21	1.91	0.52	7.00	0.33
	Model I		5.05	1.71	21.82	**0.03**	2.25	0.64	7.97	0.20	2.33	0.54	10.16	0.26
		Blood LDL levels					**Blood triglycerides levels**				**Blood total cholesterol levels**			
	Model 0		1.08	0.26	4.43	0.92	1.86	0.52	6.61	0.34	0.67	0.15	2.92	0.59
	Model I		0.88	0.19	4.00	0.87	1.57	0.38	6.44	0.53	075	0.15	3.68	0.72

No adjustment has been made in model 0. Model I was adjusted for the parameters of age, gender, smoking, and physical activity levels. Logistic regression analysis was used.
HDL, high-density lipopotein; LDL, low-density lipoprotein.

837.97 MET and control group: 1536.75 ± 831.58 MET) ($P = .100$).

In a rat study, it was observed that serum TC decreased significantly whereas TG level was not affected by 500 mg/kg aqueous extract of ginger given for 4 weeks via oral route. Ginger suppresses the accumulation of bile and cholesterol in the body by increasing the secretion of bile salts and the activity of pancreatic lipase, thereby making a positive contribution to weight management.[30,31]

As BMI increases, the risk of CVD increases and if the waist–hip ratio also increases, this risk becomes more pronounced. Weight loss reduces the risk of developing CVD.[22] It has been claimed that ginger activates adrenaline secretion by activating beta-adrenoceptors, thereby increasing thermogenesis. Thus, the increase in lipolysis in adipose tissue results in decreases in body weight.[32] It has also been suggested that ginger increases fat catabolism by activating the peroxisome proliferator-activated receptors found in skeletal muscle. Thus, by increasing calorie burning, it provides weight loss.[30,32] In contrast, in our study, it was determined that there was no statistically significant difference ($P > .05$) between the groups in terms of anthropometric measurements as well as within each group before and after the intervention (Table 1).

Although the potential active ingredient of ginger 6-gingerol has been reported to prevent fat accumulation and weight gain,[33] and also 6-shogaol to decrease adiposity,[33,34] in the present study, the lack of statistically significant changes within and between groups in terms of all anthropometric measurements including body weight and waist–hip ratio may be due to the use of the powdered form of ginger, rather than its isolated active form. In addition, these results may be attributed to including the participants who are not obese (with 26.1 ± 2.7 kg/m² BMI).

Hyperlipidemia that can be corrected by diet is a predictor and an important risk factor for CVD. There is a strong, persistent, and high relationship between TC or LDL-C levels and CVD.[16] Controlling TC and LDL-C levels with dietary measures is the primary target of preventing CVD risk. Recently, although, some preclinical studies have shown that ginger reduces serum TC, LDL-C, and TG levels and simultaneously increases HDL-C levels,[4,5] in the present study, no statistically significant change was detected in the serum lipid levels of the ginger supplementation group at the end of the study (Table 2, Figure 1) ($P > .05$).

The amount of cholesterol taken by the diet is of great importance in the balance and regulation of cholesterol

synthesis.[33,34] In a study investigating the relationship between dietary cholesterol change and endogenous cholesterol synthesis, it was determined that when the diet contains 0.05% cholesterol, 70%-80% of TC was synthesized in the liver, small intestines, and adrenal glands.[35] In another study, it was shown that serum cholesterol level decreased by 0.13 mmol/L with the decrease of 100 mg of cholesterol in the diet.[33] Similarly, at the end of our study, it was found that the TC and LDL-C levels of the control group decreased significantly ($P < .05$) when compared to the levels prior to the study. These results may be attributed to the fact that the individuals in control group may be more compliant with the nutrition group containing 300 mg of cholesterol before the study than the ginger supplementation group.

When the studies evaluating the effect of ginger on TC in the literature were examined, it was seen that ginger reduced serum cholesterol levels in some studies.[5,25,36] However, in the present study, it was determined that before–after study TC levels did not differ statistically ($P > .05$) between groups (Table 2, Figure 1), similar to some previous studies.[13,37] According to the results of the present study and previous contradicting studies, it is not possible to make a definitive conclusion about whether ginger reduces serum TC or not.

In this study, it was determined that the differences in LDL-C levels were not statistically significant ($P > .05$) between groups before and after the study (Table 2, Figure 1). Different results have been obtained in studies investigating the effect of ginger on LDL-C levels. Confirming our results, there are also studies reporting ginger has no effect on LDL-C levels.[5,38] According to the results of studies especially in humans, ginger was found to be ineffective on HDL-C levels.[5, 33,35,37] Similarly, in the current study, it was determined that HDL-C levels were also statistically insignificant ($P > .05$) in the ginger supplementation group before and after the study (Table 2, Figure 1). This may be due to the fact that dietary factors are less effective on HDL-C.[36,39] Compared to the ginger supplementation group, a significant decrease was observed in the control group at the end of the study. However, there was no significant difference between the measurements performed before and after the study in the control group. This difference may be due to the lower but not significant HDL-C level in the control group that was determined before the study.

Looking at studies investigating the effect of ginger on TG levels, some studies[5,36,37,39] found that ginger reduced serum TG levels. However, in some other studies,[13,38] similar to the current study, it was concluded that ginger does not have a significant effect on serum TG. As in the present study, a study showing that ginger is ineffective on serum lipid levels has been carried out in Iran.[39] In another study investigating the effects of ginger use on serum lipid profile in coronary artery patients, ginger was added to the diet at 4 g/day for 3 months as a powder and 10 g/day for 3 months and no effects of ginger supplementation on the lipid profile was determined.[6]

In a meta-analysis evaluating the clinical study in humans, it was concluded that adding ginger to the diet did not have any significant effect on TC as in the current study. In the mentioned meta-analysis, a statistically significant decrease was found in serum TG level. However, high heterogeneity was detected in the studies that were included in the meta-analysis, and it remained in this heterogeneous subgroup analysis with the ginger dose, duration and quality of work used. Depending on the location and growing conditions of ginger, the active ingredients are likely to vary.[40] For these reasons, more studies are needed to determine the optimum dose. In addition to the amount of ginger used, its type, method of preparation, and the characteristic features of the participants may play a very important role in the effect of ginger on blood lipid levels.

Hypertension has a very important place among the risk factors of CVD. When taken under control, it has been shown that 8.6% of the total burden of disease can be prevented, thus reducing the risk of CVD.[14]

Considering the limited number of human studies on the effect of blood pressure and ginger in the literature, it was found that ginger used in different doses (50 mg/kg, 100 mg/kg) in healthy adults significantly reduced blood pressure[41] by reducing total peripheral resistance via directing the blood flow to the vessels in the periphery. Another possible mechanism has been suggested that the blood pressure lowering effect is due to the serotonergic antagonistic feature of ginger.[42,43] The vasodilator activity of the ginger was attributed to 6-shogaol and 6-, 8-, and 10-gingerol content.[44] In this study, the systolic blood pressure of control group was lower than the ginger supplementation group in the beginning of the study ($P < .05$). At the end of the present study, the systolic blood pressure in both groups were found to be lower than prior to the study and this result was found to be statistically significant ($P < .05$) However, there was no statistically significant difference between ginger supplementation and control groups ($\bar{x} = 124.8 \pm 15.57$ and $\bar{x} = 115.3 \pm 12.2$, respectively) (Table 2, Figure 1). Considering the limited number of human studies on the effect of blood pressure and ginger in the literature, similar to the current study, it was found that ginger used in different doses (50 mg/kg, 100 mg/kg) in healthy adults significantly reduced blood

pressure[41] by reducing total peripheral resistance via directing the blood flow to the vessels in the periphery. Another possible mechanism has been suggested that the blood pressure lowering effect is due to the serotonergic antagonistic feature of ginger.[42,43] The vasodilator activity of the ginger was attributed to 6-shogaol and 6-, 8-, and 10-gingerol content.[44] Therefore, it is thought that the current study may lead to future studies on this subject since it is a study that has determined the possible effect of ginger on hypertension in humans. Results from the aforementioned studies and our study indicate that ginger may be beneficial in reducing hypertension.

The SCORE system aims to make a risk estimate in individuals who are apparently healthy, with no clinical or preclinical symptoms. Considering this feature of SCORE system, its benefits and its primary target of primary protection, it is seen that the SCORE system is quite suitable for use in the detection of risky individuals in the primary level.[16,45] At the end of this, there was a significant ($P < .05$) decrease in the SCORE value in the ginger supplementation group ($\bar{x} = 1.0 \pm 0.0$ and $\bar{x} = 0.8 \pm 0.4$, respectively) compared to the prestudy value, and the difference between ginger supplementation and control groups was also significant ($P < .05$) (Table 2, Figure 2). Although there was no statistically significant effect of ginger on serum lipid levels in the current study, the statistically significant decrease in the risk level of the group using ginger as a result of the SCORE evaluation is important in terms of revealing the positive effect of ginger. When the distribution of the groups according to the SCORE and LDL-C values was examined, 10% of the individuals using ginger had reduced SCORE value below 1% at the end of the study, whereas the control group did not show the SCORE value below 1%. In addition, the proportion of individuals whose LDL-C was ≥100, <155 decreased from 90% to 70%. It was determined that the proportion of individuals with very high (≥190) LDL-C levels did not change. While the SCORE of 10% of the individuals remained the same, the LDL-C levels decreased to ≥70, <100 levels (Table 4). It is thought that these results may have been due to the effect of ginger. The present study is likely to contribute to the literature by evaluating the effectiveness of ginger on serum lipid levels and SCORE values. The results of the studies in the literature regarding the effect of ginger on serum lipid levels appear to contradict each other. The inconsistent results in clinical trials may be due to the comparison of study groups that are not similar to each other, the use of different types and doses of ginger preparations, and the duration of ginger supplementations as well as neglecting other factors affecting food intake. Therefore, clinical studies are needed where different amounts of ginger are used for longer periods. It is obvious that there is a need for human studies that would reveal the mechanism of the possible effects of ginger on serum lipid levels.

In many previous studies, the following variables were not evaluated. Physical activity, diet, and smoking status of the individuals included in the present study were questioned at the beginning of the study and individuals with moderate physical activity and smoking status did not change their behaviors during the study. Individuals were provided with nutritional training to stay in moderate physical activity for a month, not to make radical changes in their diet, and were informed about the sources of dietary cholesterol. Although in this study there is no significant relationship between ginger and serum lipids, it is hoped that the research will contribute positively to the literature in terms of physical activity, smoking, and dietary factors.

As a result, this randomized controlled intervention clinical study showed that supplementing 400 mg of ginger twice a day for 1 month showed no statistically significant effect on serum lipid levels in individuals who were at risk by SCORE evaluation. However, compared to the beginning of the study in the group using ginger, it was revealed that the systolic blood pressure and SCORE values decreased significantly at the end of the study. Since ginger is a safe, inexpensive, easily accessible, medicinal plant that does not have any serious side effects when used in routine doses, making it available for the treatment of CVD can reduce the use of common cardiovascular drugs and make treatments much more cost-effective. In this exploratory study, the results of regression analyses suggest that the ginger intervention contributed to the positive improvement of systolic blood pressure levels (OR: 4.33 for model 0; OR: 5.05 for model I) ($P < .05$). Current findings indicate that incorporating ginger into the diet reduces systolic blood pressure and SCORE values, possibly contributing to the prevention of CVD. However, it is obvious that large-scale, long-term further detailed studies are needed for confirmation of the results of the current study.

Study Limitations

This randomized study was summarized from a PhD thesis. The ginger used in the study was provided by the researcher. The limitations of the study were that the study could not be carried out for a longer period of time due to the burden of its cost and that it could not be carried out on a larger participant population even though the number of samples was calculated. In addition, it was a limitation that the individuals were determined to be moderately active, no dietary intervention was made, and the compliance with them could not be followed. In this exploratory experiment, the higher blood pressure in the ginger supplementation group than that of the control

group at the beginning of the study, and thus after the study, was another limitation of the study.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Erciyes University (Date: March 3, 2017, Number: 2017/120).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

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Malnutrition Assessed by the GLIM Criteria Using 6 Different Approaches for Reduced Muscle Mass Criterion: Which Version Is Better Associated with Mortality in Community-Dwelling Older Adults?

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ABSTRACT

Objective: The Global Leadership Initiative on Malnutrition (GLIM) criteria suggest alternative methods for assessment of muscle mass, and which of these methods is more strongly associated with adverse outcomes remains an issue to be clarified. Our primary outcome was to report malnutrition prevalences defined by 6 different GLIM approaches and study their relationship with mortality.

Methods: This retrospective follow-up study included the data of outpatients admitted to a tertiary hospital. We used 6 different approaches for GLIM, based on methods used to identify reduced muscle mass: i) skeletal muscle mass (SMM)/height², ii) SMM/body mass index (BMI), iii) handgrip strength (HGS), iv) calf circumference (CC), v) CC adjusted for BMI, and vi) GLIM without third phenotypic criterion (P3). We evaluated survival in malnutrition with Kaplan–Meier log rank test. The Cox proportional hazards model was used to identify the relationships of different GLIM versions with mortality.

Results: The study population included 224 older individuals, with a median age of 72, and female predominance (68.8%). The prevalences with different GLIM versions ranged between 4.0% and 34.1%. During a median follow-up period of 31 months, 14 (6.3%) participants died. According to unadjusted analyses, only GLIM (SMM/h²), GLIM (HGS), GLIM (CC), and GLIM (without P3) were significantly associated with increased mortality risk [Hazard Ratio (95% CI) were 3.8 (1.1-13.7), 4.3 (1.4-12.8), 4.6 (1.3-16.7), and 7.3 (2.0-26.5), respectively]. After final adjustments were made for age and sex, it was revealed that none of the versions were the predictors of mortality in older outpatients.

Conclusion: The GLIM criteria have room for improvement as different options for muscle mass assessment are allowed, and this study aimed to fill the gap in the literature on whether malnutrition diagnosed by alternative GLIM definitions had predictive validity in community-dwelling older adults. Further outcome studies using larger cohorts and different pragmatic approaches are needed to detect the ideal GLIM definition for malnutrition assessment.

Keywords: Older adults, malnutrition, mortality, sarcopenia, survival

INTRODUCTION

Malnutrition is accepted as "a geriatric syndrome" that has significant relationships with adverse outcomes like sarcopenia, frailty, increased hospitalizations, and mortality.[1] The prevalence rates were primarily determined by settings, underlying diseases and methods used for assessment. A systematic review and meta-analysis using 22 different malnutrition screening tools have reported a pool prevalence of malnutrition in older adults ranging between 8.5% and 28.0% (for community-dwelling and hospitalized older adults, respectively), and the prevalence rates differed from 14.9% to 40.6%, depending on the method used for assessment of malnutrition.[2] Until 2019 (year of publication of the abovementioned systematic review and meta-analysis), there was a lack of consensus regarding the diagnostic criteria of malnutrition, leading to variations and inconsistencies between reports. Just in time, the Global Leadership Initiative on Malnutrition (GLIM) criteria were developed by the representatives of 4

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major clinical nutrition societies around the world with the aim of standardization of the clinical practice of malnutrition diagnosis.[3]

The GLIM criteria cover malnutrition diagnosis in quite a comprehensive way and require a 3-step approach: screening, diagnosis (searching for the presence of at least one phenotypic and one etiologic criterion), and grading of the severity. As a striking step, the panel implemented reduced muscle mass as one of the diagnostic criteria for malnutrition due to the close relationship between nutritional status and muscle health. Therefore, at the third phenotypic criterion, they recommended measurement of muscle mass with a validated tool, but proxy measurements were also welcomed in the absence of these tools.[3] A year after its publication, 2 of the main authors of the GLIM criteria assessed whether GLIM worked in older people and concluded that although predictive and criterion validity were acceptable, the lack of guidance on how to assess muscle mass hampered the validation and implementation of the GLIM criteria.[4] At the time of uncertainty on which diagnostic method is optimal for assessment of the third phenotypic criterion, it is assumed that further studies with different diagnostic tools will reveal the most useful version to properly detect malnutrition and predict adverse outcomes.

Another gap in the literature is that there are limited studies on GLIM-defined malnutrition and its outcomes in community-dwelling older adults, since the reports on GLIM-defined malnutrition have been mostly conducted on patients with specific diseases and different settings.[5-7] Therefore, the primary aim of this report is to find out the prevalence rates of GLIM-defined malnutrition with 6 different approaches in community-dwelling older adults and study the association of different GLIM definitions with mortality.

Main points

- The lack of guidance on how to assess the reduced muscle mass criterion of the Global Leadership Initiative on Malnutrition (GLIM) criteria hinders its validity and application in clinical practice. Therefore, studies with different diagnostic methods for the third phenotypic criterion are needed to identify the most useful version to properly detect malnutrition and predict adverse outcomes.
- The GLIM criteria with 6 different pragmatic approaches for reduced muscle mass criterion ended up with a broad range of malnutrition prevalence in older outpatients: The GLIM criteria without P3 criterion had the lowest (4.0%), and the GLIM with skeletal muscle mass adjusted for body mass index had the highest malnutrition prevalence (34.1%).
- In a study population of mostly overweight–obese older adults with a low mortality rate during 31-month follow-up, none of the GLIM versions were independently associated with mortality after adjusting for age and sex.

METHODS

Population and Setting

This study is a retrospective, longitudinal study conducted in a geriatric outpatient clinic of a tertiary health center between May 2018 and December 2021. We included community-dwelling older adults aged over 60 years who provided informal consent for participating in a comprehensive geriatric assessment (CGA). Exclusion criteria were i) moderate-to-severe dementia; ii) severe depression; iii) certain conditions that might prevent reliable muscle strength measurements (i.e., hand osteoarthritis, stroke, peripheral artery disease, etc.), bioelectrical impedance analysis (BIA) measurements (i.e., edematous state, metal implants, inability to stand on 2 feet, etc.), or calf circumference (CC) measurements (i.e., edematous state, amputation of lower extremities, etc.); iv) conditions other than dementia and depression that might prevent healthy communication (like severe hearing impairment); v) acute, unstable, or deteriorating clinical conditions that prevent CGA to be optimally performed; and vi) refusal to participate. We followed the Strengthening the Reporting of Observational Studies in Epidemiology statement.[8] The local ethics committee of Istanbul University Istanbul Faculty of Medicine gave approval to the study on November 25, 2022 (approval number: 1399682/2022).

Sample Size Calculation

We performed a sample size calculation based on the reported GLIM-defined malnutrition prevalences:[9,10] Using a power of 80%, a CI of 95%, and an error probability of 10%, we determined a sample size of 184 participants. Anticipating a dropout rate of 10%, a sample size of 202 participants was considered sufficient for this study.

Measurements

Interview on Baseline Characteristics and Comprehensive Geriatric Assessment

We obtained the demographical and clinical characteristics of the participants. We recorded information about marital status, education level, tobacco and alcohol use, chronic diseases, and medications. Expert geriatricians performed CGA and questioned falls in the previous year, fear of falling, sleep disturbance, constipation, urinary and fecal incontinence, and chronic pain, with closed-ended questions. We assessed functionality via Katz Activities of Daily Living (ADL)[11] and Lawton Instrumental ADL (IADL) scales.[12] We screened for sarcopenia and frailty via SARC-F questionnaire and FRAIL scales, respectively.[13,14] The scores obtained from these 5-itemed questionnaires

were interpreted as: increased sarcopenia risk for SARC-F ≥4 points, frail for FRAIL ≥ 3, pre-frail for FRAIL = 1 or 2, and robust for FRAIL = 0.

Assessment of Nutritional Status with Different Versions of the GLIM Criteria

We assessed malnutrition via Mini-Nutritional Assessment—Short Form (MNA-SF) and the GLIM criteria. We classified a MNA-SF score of less than 12 as undernutrition and less than 8 as malnutrition.[15] The GLIM evaluates malnutrition by 3 phenotypic and 2 etiologic criteria and necessitates the presence of at least 1 phenotypic and 1 etiologic criterion for diagnosis step of malnutrition assessment. Phenotypic criteria are i) nonvolitional weight loss (>5% within past 6 months, or >10% beyond 6 months), ii) low body mass index (BMI) (<20 kg/m² if <70 years, or <22 kg/m² if >70 years), and iii) reduced muscle mass by validated body composition techniques. Etiologic criteria are i) reduced food intake or assimilation and ii) disease burden/inflammation.[3] Since this is a retrospective study, and GLIM criteria were not published during the earlier periods of the data collection, we derived the data on weight loss of some participants from the items of MNA-SF and FRAIL scale that question weight loss during the past 3 months and past year. Likewise, we derived the data on the first etiologic criterion by using the first item of the MNA-SF (i.e., Has food intake decreased over the past 3 months due to loss of appetite, chewing or swallowing difficulties, or digestive problems?[15]). Although we could not assess disease burden for the second etiologic criterion, diagnosis of acute or chronic inflammatory diseases, or a C-reactive protein level >5 mg/L at admission were considered positive for second etiologic criterion.

The GLIM panel recommended measurement of muscle mass primarily by dual-energy absorptiometry (DXA) or other validated techniques like BIA. Considering that these instruments are not readily available in most settings, they also optionalized the use of anthropometric measurements or measurements of muscle strength as proxies of muscle mass.[3] Since our primary goal was to compare different versions of GLIM determined by alternative methods for the third phenotypic criterion, we used 5 different definitions based on the modality used for measurement, i.e., i) reduced muscle mass [with the definition of skeletal muscle mass (SMM) adjusted for height square], ii) reduced muscle mass (with the definition of SMM adjusted for BMI), iii) reduced muscle strength [with measurement of handgrip strength (HGS)], iv) reduced CC, and v) reduced CC adjusted for BMI. We measured muscle mass via BIA and used 2 SMM indices: SMM/h² and SMM/BMI. We used SMM/h² definition, since it was the most commonly used way to define reduced muscle mass globally; however, this adjustment method has also been criticized that it could overlook reduced muscle mass in obese or overweight individuals and SMM/BMI would be a better adjustment technique in terms of finding cases and reported to have better association with adverse outcomes.[16] Therefore, we also defined reduced SMM by adjustments for BMI. We used Tanita-BC532 bioelectrical impedance analyzer, which demonstrated a strong correlation with magnetic resonance imaging measurements.[17] We obtained fat free mass values and multiplied them with 0.566 to transform them into SMM. We measured body weight and height with a standardized stadiometer to the nearest 0.1 kg and 0.1 cm. We calculated BMI as body weight (kg) divided by height square (meters). The cutoffs used for SMMI were population-specific thresholds obtained from total SMM measurements via BIA and determined by calculating mean minus 1 standard deviation of young and healthy reference population (which was recommended by experts of GLIM for determining thresholds for mild-to-moderate reduced muscle mass).[18] Hence, low SMM/h² thresholds were 10.1 and 8.2 kg/m²,[18] and low SMM/BMI thresholds were 1.189 and 0.954 kg/BMI,[19] for males and females, respectively.

For measurement of muscle strength, we used a Jamar hydraulic hand dynamometer, applying a standardized protocol.[20] We asked the participants to keep their elbows at 90° flexion and their wrists in a neutral position and to apply their maximum strength 3 times with both hands separately and with 30-second rest intervals. We accepted the maximum HGS measured as the muscle strength value and used the population and sex-specific thresholds to identify reduced HGS, i.e., <35 kg and <20 kg, for males and females, respectively.[21] We measured CC at the level of widest circumference of nondominant leg via a nonelastic tape while the participants were standing. We used the population and sex-specific thresholds for reduced CC, i.e., <33 cm and <32 cm, for males and females, respectively.[22] As CC is highly affected from subcutaneous fat tissue and an evident difference of CC between different BMI categories has been put forth, Gonzalez et al[24] have suggested adjusting CC measurements for different BMI categories, except for normal BMI range of 18.5-24.9 kg/m².[23] They have suggested a practical formula as adding 4 cm to the measured CC value in those with BMI <18.5 kg/m² or subtracting 3, 7, or 12 cm from the CC value in BMI categories of 25-29, 30-39, and ≥ 40 kg/m², respectively, from the CC measurement.[24] We applied the aforementioned formula to obtain adjusted CC values and used the population and sex-specific thresholds mentioned previously to identify reduced adjusted CC. Apart from these measurements, we decided to define an alternative GLIM definition as "GLIM without any measurement regarding muscle mass," and aimed to find out whether we could show a significant association between "GLIM without

third phenotypic criterion" and mortality. The rationale behind this approach was that the third phenotypic criterion being the rate-limiting step for most settings that do not have any equipment for measurements regarding muscle mass or its proxies. Thus, we wanted to check how the relationship between GLIM and mortality would be affected when the reduced muscle mass criterion was not used.

In summary, we used 6 alternative GLIM definitions to assess malnutrition:

1. GLIM with P3 defined as SMM adjusted for height square
2. GLIM with P3 defined as SMM adjusted for BMI
3. GLIM with P3 defined as reduced HGS
4. GLIM with P3 defined as reduced CC
5. GLIM with P3 defined as reduced CC adjusted for BMI
6. GLIM without P3

The abovementioned measurements were performed by a single qualified physiotherapist. All participants gave informed consent prior to assessments. Deaths were ascertained by a death certification search at the end of December 2021, using Death Notification System (DNS) of Republic of Türkiye Ministry of Health. The DNS is a national electronic software program used by physicians for mandatory reporting of in- or out-of-hospital deaths nationwide.

Statistical Analysis

We presented the categorical data as numbers and percentages. We investigated the normality of numerical variables by using visual (histograms and probability plots) and analytical methods. Accordingly, we presented normal distributed variables as mean ± standard deviation and skew distributed ones as median (minimum and maximum). We compared 2 independent groups with *t*-test or Mann–Whitney *U*-test, where necessary. We used chi-square test with Yates correction and Fisher's exact test when appropriate for categorical data. For comparison of more than 2 categorical variable groups, we used chi-square test. In order to find out the coherence between different GLIM versions, we studied the overall concordance rate and reported the Cohen's kappa coefficient (κ). The κ values between 0.81 and 1 were considered as perfect, 0.6-0.8 indicated strong, 0.4-0.6 indicated moderate, 0.20-0.4 indicated low, between 0 and 0.20 indicated very slight agreement, and less than 0 indicated disagreement. We evaluated survival in malnutrition defined by different GLIM versions with Kaplan–Meier log rank test. We defined follow-up duration as "the time (months) between date of death (for deceased participants) or December 2021 (for alive participants) and date of the first evaluation." We performed Cox regression analysis to find out whether malnutrition defined by different GLIM versions was independently associated with mortality. We primarily performed a crude analysis (without any adjustments for confounding factors) between mortality and malnutrition as defined by different GLIM versions. Furthermore, we defined different models to perform regression analyses adjusted for confounding variables, which were found to be significantly associated with mortality in univariate analyses. Before including confounding variables in the same regression models, we checked whether multicollinearity existed and confirmed that there was no such strong relationship that would cause multicollinearity. We derived hazard ratio (HR) and 95% CI and used alpha of less than 0.05 as the level of significance. We used the Statistical Package for the Social Sciences Statistics for Windows 21.0 program for statistical analyses.

RESULTS

There were 224 participants included in the study; 68.8% were female. The median age was 72 (60-96). The median number of chronic diseases was 3 (0-8) and regular medications was 5 (0-17). Hypertension was the most prevalent chronic disease (72.0%), followed by diabetes mellitus (35.5%) and dyslipidemia (21.5%). According to the CGA findings, more than half of the study population suffered chronic pain (53.6%), and nearly half of them had fear of falling (47.3%) and urinary incontinence (46.0%). According to MNA-SF, undernutrition (MNA-SF <12) prevalence was 22.5%, and malnutrition prevalence was 2.3%. The baseline characteristics and CGA findings of the study population are given in Table 1.

During a median follow-up period of 31 months, 14 (6.3%) participants died, with male participants demonstrating higher mortality rate than females (12.9% vs 3.2%, $P = .006$). The comparisons of each GLIM criteria between alive and deceased groups and the prevalences of malnutrition according to different GLIM versions can be found in Table 2. The prevalences with different GLIM versions ranged between 4.0% and 34.1%, as GLIM defined by SMM/BMI giving the highest and GLIM defined without P3 criterion giving the lowest prevalence. The prevalences of GLIM-defined malnutrition were significantly higher in deceased groups only when GLIM defined by HGS, CC, or without P3 criterion (*P*-values were .004, .048, and .018, respectively).

Among the different GLIM versions, the strongest agreement existed between GLIM defined by CC and GLIM defined without P3 criterion [$\kappa = 0.824$ (0.655-0.993); $P < .001$], followed by GLIM defined by CC and GLIM defined

Table 1. Baseline Characteristics of the Study Population

	Total (n = 224)	Female (n = 154)	Male (n = 70)	*P*
Age[#]	72 (60-96)	72 (60-96)	75 (61-93)	**.007**
Marital status				**.005**
Married	134 (59.8%)	81 (52.6%)	53 (75.7%)	
Single/divorced/widow	90 (40.2%)	73 (47.4%)	17 (24.3%)	
Education level				**.005**
Illiterate	63 (28.1%)	56 (36.4%)	7 (10.0%)	
Primary school	91 (40.7%)	57 (37.0%)	34 (48.5%)	
Secondary school	28 (12.5%)	18 (11.7%)	10 (14.3%)	
Post secondary education	42 (18.7%)	23 (14.9%)	19 (27.1%)	
Tobacco use	14 (6.3%)	10 (6.5%)	4 (5.7%)	**<.001**
Alcohol use	9 (4.0%)	3 (1.9%)	6 (8.6%)	**<.001**
Number of chronic diseases*	3 (0-8)	3 (1-8)	3 (0-7)	.411
Number of regular medications*	5 (0-17)	5 (0-17)	4 (0-14)	.060
Chronic diseases				
Hypertension	144 (72.0%)	103 (76.3%)	41 (63.1%)	**.051**
Diabetes mellitus	71 (35.5%)	47 (34.8%)	24 (36.9%)	.770
Dyslipidemia	43 (21.5%)	33 (24.4%)	10 (15.4%)	.144
Hypothyroidism	35 (17.5%)	32 (23.7%)	3 (4.6%)	**.001**
IHD	30 (15.0%)	16 (11.9%)	14 (21.5%)	.072
COPD	10 (5.0%)	6 (4.4%)	4 (6.2%)	.731
Comprehensive Geriatric Assessment				
Falls in the previous year	81 (36.3%)	54 (35.3%)	27 (38.6%)	.637
Fear of falling	105 (47.3%)	79 (52.0%)	26 (37.1%)	**.040**
Urinary incontinence	103 (46.0%)	86 (55.8%)	17 (24.3%)	**<.001**
Fecal incontinence	12 (5.4%)	11 (7.1%)	1 (1.4%)	.110
Chronic pain	120 (53.6%)	92 (59.7%)	28 (40.0%)	**.006**
Constipation	66 (30.0%)	45 (30.0%)	21 (30.0%)	1.0
Sleep disturbance	79 (33.3%)	59 (38.3%)	20 (28.6%)	.334
Undernutrition (MNA-SF < 12)	50 (22.5%)	34 (22.4%)	16 (22.9%)	.935
Malnutrition (MNA-SF < 8)	5 (2.3%)	3 (2.0%)	2 (2.9%)	.652
Frailty	38 (17.0%)	29 (19.0%)	9 (12.9%)	.261
ADL*	6 (0-8)	6 (1-6)	6 (0-6)	.268
IADL*	8 (0-8)	8 (0-8)	8 (0-8)	.708
SARC-F ≥ 4	44 (20.4%)	32 (21.5%)	12 (17.9%)	.547
Measurements				
Height (cm)*	156 (135-181)	152 (135-178)	166 (146-181)	**<.001**
Body weight (kg)*	72.2 (42.0-128.8)	71.0 (43.5-117.6)	74.3 (42.0-128.8)	.177
BMI (kg/m²)[#]	30.2 ± 5.3	31.3 ± 5.3	27.8 ± 4.6	**<.001**
Handgrip strength*	24 (6-50)	22 (10-44)	34 (6-50)	**<.001**
CC*	37 (29-47)	38 (31-47)	37 (29-45)	**.017**
Adjusted CC*	33 (24-40)	32 (24-40)	33 (25-37)	.392
Mortality rate	14 (6.3%)	5 (3.2%)	9 (12.9%)	**.006**

P < .05 are given in bold.

ADL, activities in daily living; BMI, body mass index; CC, calf circumference; COPD, chronic obstructive pulmonary disease; IADL, instrumental activities in daily living; IHD, ischemic heart disease; MNA-SF, Mini-Nutritional Assessment-Short Form; SARC-F, strength, assistance in walking, rise from a chair, limb stairs, and falls.

*Median.

[#]Mean ± standard deviation.

Table 2. Malnutrition Prevalence According to Different Versions of the GLIM Criteria and Comparisons Between Alive and Deceased Groups

	Total	Alive	Deceased	*P*
GLIM P1*	16 (7.1%)	12 (5.7%)	4 (28.6%)	**.011**
GLIM P2#	10 (4.5%)	7 (3.3%)	3 (21.4%)	**.018**
GLIM P3 (SMM/h²^	26 (11.6%)	23 (11.0%)	3 (21.4%)	.212
GLIM P3 (SMM/BMI)^	208 (92.9%)	195 (92.9%)	13 (92.9%)	1
GLIM P3 (HGS)^	80 (36.0%)	70 (33.7%)	10 (71.4%)	**.004**
GLIM P3 (CC)^	16 (7.2%)	13 (6.3%)	3 (21.4%)	.069
GLIM P3 (CC-adjusted) ^, ∫	83 (37.4%)	78 (37.5%)	5 (35.7%)	.894
GLIM E1√	17 (7.6%)	14 (6.7%)	3 (21.4%)	.078
GLIM E2◦	68 (33.2%)	62 (32.3%)	6 (46.2%)	.304
GLIM total (without P3 criterion)	9 (4.0%)	6 (2.9%)	3 (21.4%)	**.018**
GLIM total (with P3 defined with SMM adjusted for height square)	16 (7.2%)	13 (6.3%)	3 (21.4%)	.069
GLIM total (with P3 defined with SMM adjusted for BMI)	70 (34.1%)	64 (33.3%)	6 (46.2%)	.345
GLIM total (with P3 defined with reduced handgrip strength)	37 (17.2%)	31 (15.3%)	6 (46.2%)	**.004**
GLIM total (with P3 defined with reduced calf circumference)	14 (6.3%)	11 (5.3%)	3 (21.4%)	**.048**
GLIM total (with P3 defined with reduced adjusted calf circumference)	39 (18.1%)	36 (17.8%)	3 (23.1%)	.709

P < .05 are given in bold.
BMI, body mass index; CC, calf circumference; GLIM, Global Leadership Initiative on Malnutrition; h, height; HGS, handgrip strength; SMM, skeletal muscle mass.
*GLIM P1 (first phenotypic criterion): >5%within past 6 months, or 10% beyond 6 months.
#GLIM P2 (second phenotypic criterion): Low BMI (kg/m²): <20 if <70 years, or <22 if ≥70 years.
^GLIM P3 (third phenotypic criterion): Reduced muscle mass by validated body composition measuring techniques (SMM/h² thresholds were <10.1 kg/m² and <8.2 kg/m²; SMM/BMI thresholds were <1.189 kg/BMI and <0.954 kg/BMI; reduced handgrip strength thresholds were <35 kg and <20 kg; reduced calf circumference thresholds were <33 cm and <32 cm, for males and females, respectively).
∫Calf circumference was adjusted for body mass index. The adjusted CC was obtained by adding 4 cm to the measured CC value in those with BMI <18.5 kg/m² or subtracting 3, 7, or 12 cm from CC value in those with BMI 25-29, 30-39, ≥40 kg/m², respectively from the CC measure.
√GLIM E1 (first etiologic criterion): Reduced food intake or assimilation.
◦GLIM E2 (second etiologic criterion): Inflammation (acute disease/injury or chronic disease related).

by SMM/h² [κ = 0.786 (0.619- 0.953); *P* < .001], and GLIM defined by SMM/h² and GLIM defined without P3 criterion [κ = 0.756 (0.570-0.942); *P* < .001]. The findings of concordance analyses between different versions of GLIM are found in Table 3.

Mean survival time was significantly shorter in participants with malnutrition defined by GLIM (SMM/h²) (37.1 vs. 41.6 months; log rank, *P* = .027), GLIM (HGS) (38.8 vs. 40.0 months; log rank, *P* = .004), GLIM (CC) (36.5 vs. 41.6 months; log rank, *P* = .010), and GLIM (without P3 criterion) (34.6 vs. 41.6 months; log rank, *P* < .001) (Figure 1). We defined 4 models to identify which versions of GLIM defined malnutrition were independently associated with increased mortality. According to model 1 (crude analysis): GLIM (SMM/h²) [HR (95% CI) = 3.8 (1.1-13.7), *P* = .040], GLIM (HGS) [HR (95% CI) = 4.3 (1.4-12.8), *P* = .009], GLIM (CC) [HR (95% CI) = 4.6 (1.3-16.7), *P* = .019], and GLIM (without P3 criterion) [HR (95% CI) = 7.3 (2.0-26.5), *P* = .003] were significantly associated with mortality. In model 2 (adjusted for age), only GLIM (without P3 criterion) demonstrated persistence in relationship with mortality [HR (95% CI) = 4.0 (1.1-14.6), *P* = .039]. Adjustments made for sex (model 3) revealed that GLIM (HGS) [HR (95% CI) = 4.2 (1.4-12.5), *P* = .010] and GLIM (without P3 criterion) [HR (95% CI) = 5.9 (1.6-21.7), *P* = .007] were the only predictors of increased mortality risk. In model 4 (adjusted for age and sex), it was revealed that none of the GLIM versions were independently associated with mortality (Table 4, Supplementary Table 1).

Table 3. Concordance Between Alternative Versions of Global Leadership Initiative on Malnutrition Criteria Developed by Integrating Surrogates of Muscle Mass Measurement

	SMM/h²	SMM/BMI	HGS	CC	Adj. CC	Without P3
SMM/h²	1					
SMM/BMI	0.201 (0.089-0.313)	1				
HGS	0.431 (0.260-0.602)	0.530 (0.406-0.654)	1			
CC	**0.786** (0.619-0.953)	0.194 (0.086-0.302)	0.372 (0.200-0.545)	1		
Adj. CC	0.410 (0.243-0.577)	0.551 (0.431-0.671)	0.422 (0.265-0.578)	0.478 (0.313-0.643)	1	
Without P3	**0.756** (0.570-0.942)	0.125 (0.116-0.134)	0.334 (0.164-0.505)	**0.824** (0.655-0.993)	0.361 (0.194-0.528)	1

P < .001 for all, except *P* = .002. K values indicating strong agreement are given in bold.
Adj. CC, adjusted calf circumference; BMI, body mass index; CC, calf circumference; h, height; HGS, handgrip strength; P3, third phenotypic criterion; SMM, skeletal muscle mass.

DISCUSSION

In this study, we created different versions of the GLIM criteria determined by alternative definitions of third phenotypic criterion. Accordingly, we found out that there was a broad range of malnutrition prevalence according to different versions, with GLIM without P3 criterion had the lowest (4.0%) and GLIM (SMM/BMI) had the highest (34.1%). In crude analyses, GLIM without P3 criterion demonstrated the strongest relationship with mortality, followed by GLIM (CC) and GLIM (HGS). After adjustments made for age and sex, we found out that the relationship no longer persisted between GLIM-defined malnutrition and mortality.

According to the GLIM criteria, malnutrition prevalence in community-dwelling older adults was between 4.0%-34.1% in our study. This broad range of prevalence with different methods for third criterion is striking, as it shows that although several methods were optionalized for measurements regarding this criterion, results might be totally different from one another depending on the preferred method. In our country, GLIM-defined malnutrition prevalence in community-dwelling older adults was reported to be 24.5%-32.2% in previous studies by using BIA-derived reduced fat-free mass index (adjusted for h²)[10] or appendicular lean mass index (adjusted for h²)[9] for the third phenotypic criterion. The preference of different modalities for the third phenotypic criterion appears to be an important determinant of these reported prevalences.

In our study, malnutrition prevalence was lowest with GLIM without P3 criterion. Apart from the exclusion of reduced muscle mass factor, one of the main reasons behind this finding might be that our outpatient clinic had a significant number of healthy older adults attending to visit for follow-up of stable chronic diseases and for preventive medicine. The median number of chronic diseases was 3, which was lower than the number reported previously for older adults living in the community.[25,26] Additionally, the mean BMI of the study population was 30.2 kg/m², meaning most of the participants were overweight and even class I obese. In a study population consisted of mostly overweight individuals, a diagnostic tool using BMI, weight loss, and reduced food intake would be expected to detect low number of malnutrition cases. Contrarily, the Cox proportional hazards model revealed that when GLIM was used without the reduced muscle mass criterion, it demonstrated the highest mortality risk in crude analysis compared to the versions with third phenotypic criterion. Hence, although GLIM without P3 identified less individuals with malnutrition among the GLIM versions, it was also the strongest version that predicted increased mortality risk in seemingly healthier older adults living in the community. This finding might be useful in settings where equipment or qualified personnel do not exist for measurements for third phenotypic criterion to detect malnutrition cases with increased mortality risk. In community-dwelling older adults, GLIM without P3 was reported to be independently associated with mortality after adjustments made for age, sex, number of concomitant diseases, number of drugs, physical activity level, and cognitive status [HR (95% CI) = 3.1 (1.7-5.7)].[27] In fact, although settings were different or populations were more specific compared to ours (like mainly older outpatients with cancer who were actively receiving treatment for their diseases,[5] or hospitalized patients with hip fracture[6] or COVID-19[28]), there are other studies reporting that malnutrition defined by GLIM without P3 criterion had no significant relationship with increased mortality after adjustments for confounding variables.

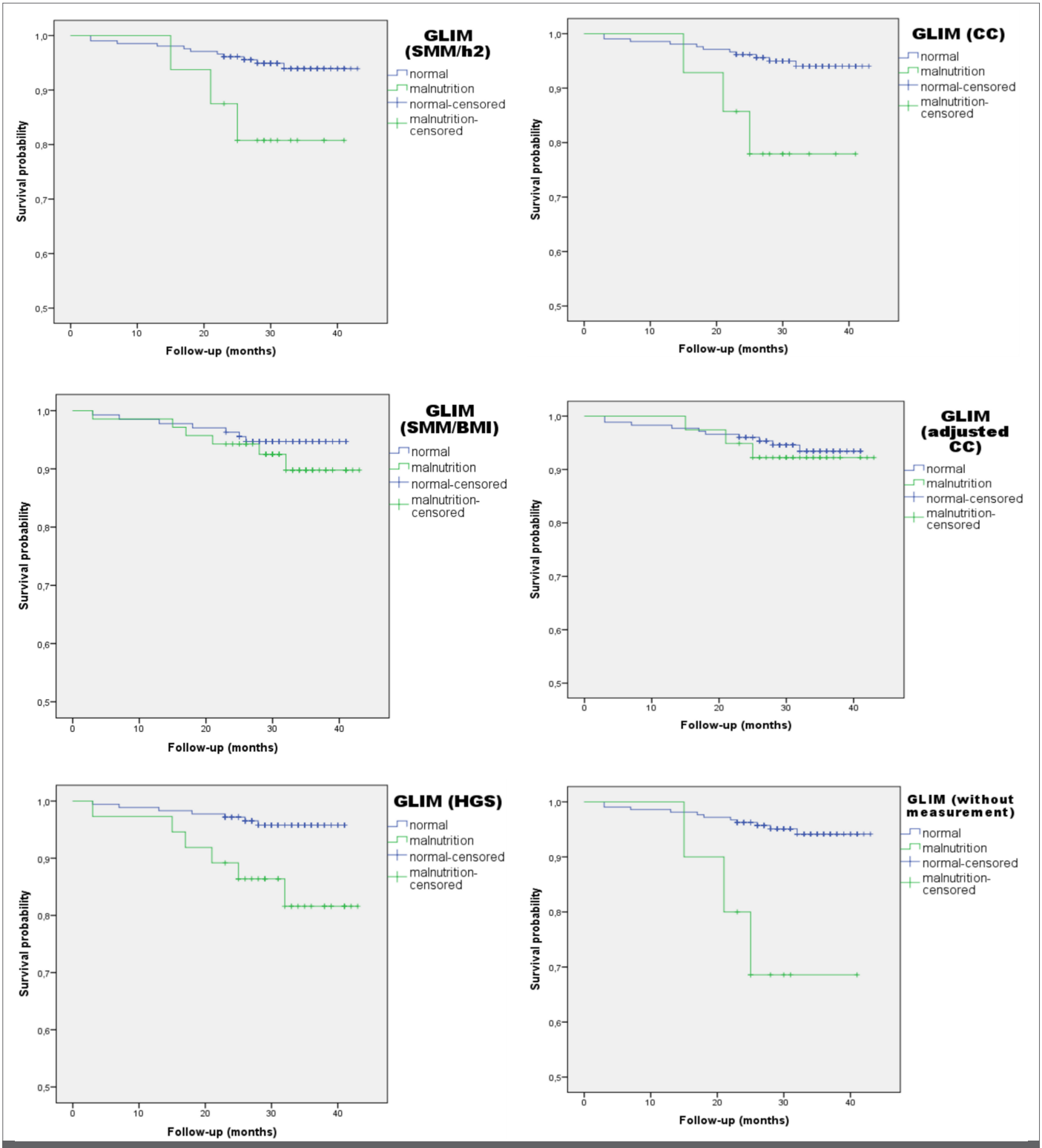

Figure 1. Kaplan–Meier survival curves with 6 different GLIM definitions for malnutrition diagnosis. BMI, body mass index; CC, calf circumference; GLIM, Global Leadership Initiative on Malnutrition; h, height; HGS, handgrip strength; P3, third phenotypic criterion; SMM, skeletal muscle mass.

Anthropometric measurements have been the most commonly used method for the third phenotypic criterion in previous studies with the GLIM criteria, and CC has been the most commonly used anthropometric measurement.[29] In our study, GLIM (CC) was the second version with the highest mortality risk in the unadjusted analysis, but the relationship did not persist in further models. Indeed, lower CC thresholds (which were

Table 4. Cox Regression Analyses Regarding Associations Between Malnutrition Defined by Different Versions of the GLIM Criteria and Mortality

Malnutrition Definition	Model 1	Model 2	Model 3	Model 4
GLIM (SMM/h²)	**3.8 (1.1-13.7)**	2.2 (0.6- 8.1)	2.3 (0.6-8.8)	1.5 (0.4-5.6)
	***P*=.040**	*P*=.221	*P*=.212	*P*=.594
GLIM (SMM/BMI)	1.7 (0.6-5.0)	1.1 (0.4-3.4)	1.9 (0.6-5.5)	1.2 (0.4-3.7)
	P=.363	*P* = .850	*P* = .272	*P* = .721
GLIM (HGS)	**4.3 (1.4-12.8**)	2.5 (0.9-7.6)	**4.2 (1.4-12.5)**	2.2 (0.7-6.9)
	***P* = .009**	*P* = .096	***P* = .010**	*P*=.174
GLIM (CC)	**4.6 (1.3-16.7**)	2.5 (0.7-9.2)	3.6 (0.98-13.0)	1.8 (0.5-6.9)
	***P* = .019**	*P* = .161	*P*=.054	*P*=.388
GLIM (adj. CC)	1.3 (0.4-4.9)	0.7 (0.2-2.6)	1.3 (0.4-4.6)	0.7 (0.2-2.7)
	P = .656	*P*=.566	*P* = .726	*P*=.619
GLIM (w/out P3)	**7.3 (2.0-26.5**)	**4.0 (1.1-14.6**)	**5.9 (1.6-21.7**)	3.0 (0.8-11.6)
	***P* = .003**	***P*=.039**	***P*=.007**	*P*=.117

Hazard ratios (95% confidence intervals) and p values with statistical significance are given in bold. Model 1 was the crude analysis performed with a single independent variable: Malnutrition defined by the GLIM criteria. Model 2 was adjusted for age; model 3 was adjusted for sex (female); model 4 was adjusted for age and sex (female). Age and sex were determined as confounding variables, as they were found to be significantly associated with mortality in univariate analyses.

adj. CC, adjusted calf circumference; BMI, body mass index; CC, calf circumference; GLIM, Global Leadership Initiative on Malnutrition; HGS, handgrip strength; P3, third phenotypic criterion; SMM, skeletal muscle mass.

suggested for the Turkish population as 31 and 30 cm for grading severe malnutrition in males and females, respectively[22]) would probably end up with stronger relationships in terms of mortality. Accordingly, we aimed to stratify the analyses for grading malnutrition, but unfortunately, we could not reach the exact data on weight loss questioned for the first phenotypic criterion. In the literature, 2 studies (1 with older patients with diabetes[30] and the other with older patients with cancer[31]) revealed that GLIM (CC)-defined malnutrition was independently associated with mortality for only severe, but not moderate, malnutrition after adjustments for confounding factors. We also used adjusted CC alternatively to exclude the confounding effect of BMI as an indicator of adiposity and identify if it would better predict mortality than unadjusted. Although this method identified more individuals with malnutrition, it demonstrated no significant relationship with mortality in any of the studied models. In fact, adjusting CC for BMI may have resulted in ignoring the interaction between muscle mass and fat mass and bypassing the negative (or may be positive) consequences of this close relationship. Furthermore, it might be necessary to come up with new thresholds for adjusted CC, as thresholds for unadjusted CC might not be applicable for the adjusted ones.

We used HGS for the P3 criterion alternatively and found that this definition was better associated with mortality than versions with muscle mass measurements. The GLIM (HGS) also identified more individuals with malnutrition compared to the GLIM (SMM/h²) (17.2% vs. 7.2%). In the literature, there are a plenty of studies reporting significant increase in mortality risk with reduced muscle mass criterion adjusted for height, based on either DXA or BIA measurements and conducted in different settings or study groups (patients with cancer,[32] heart failure,[7] or other cardiovascular diseases[33]). However, studies using HGS are less and more inconsistent. While in community-dwelling older adults, it was reported to be significantly associated with increased 5-year incidence of deaths,[27] it was not a predictor of mortality in older outpatients with heart failure[34] or cancer,[5] after adjustments made for confounding factors. It is obvious that more studies are needed to reveal whether GLIM (HGS) can be a strong alternative of reduced muscle mass measurement in community-dwelling older adults.

We also used BMI for SMM adjustments and defined another version for GLIM in order not to overlook the relative decrease in muscle mass in obese and overweight individuals.[35] Although GLIM-defined (SMM/BMI)

malnutrition was not an independent predictor of mortality, it identified more cases of malnutrition than any other GLIM version. Since our study population mostly consisted of overweight–obese individuals, SMM/BMI probably identified more individuals with reduced muscle mass in this group than other adjustment methods, hence ended up with more positivity on the third phenotypic criterion. The possible explanation for SMM/BMI not demonstrating a significant relationship with mortality might be "the obesity paradox," as being overweight or mildly obese has been reported to be protective in terms of mortality in older adults.[36] Likewise, several studies recently reported that obesity defined by fat percentage might also be protective in terms of mortality,[37] and when it accompanied to sarcopenia, it might be more favorable in terms of frailty, functionality, or physical performance than sarcopenia alone.[38,39] Hence, the study group may have benefited from the survival advantage of being overweight or mildly obese, even if they were malnourished according to GLIM (SMM/BMI). In fact, a lack of significant association with mortality does not mean that certain diagnostic method is not useful for routine practice, as detecting cases of malnutrition and timely intervention are expected to create significant impact on prognosis, even in obese older adults. As a matter of fact, this is the only study using SMM/BMI for third criterion of GLIM in the literature to the best of our knowledge, and more studies in different populations would reveal its exact relationship with mortality.

Although several methods were used for the third phenotypic criterion in GLIM, there are very limited studies that used more than one alternative in the same study for community-dwelling older adults. The most striking one was the SarcoPhAge study, as Sanchez-Rodriguez et al27 used 7 alternative approaches for the third phenotypic criterion (i.e., GLIM without P3, HGS, CC, mid-arm circumference, Yu's formula, Ishii's score chart, and Goodman grid), in addition to the original GLIM criteria (reduced muscle mass according to DXA-derived ALMI and FFMI). In a study population with 373 older adults, they reported a narrower range of prevalences for malnutrition, i.e., 13.9%-24.4%. Similar to our study, the lowest prevalence was obtained with GLIM without P3 criterion, and the highest was detected with the original GLIM criteria (i.e., reduced FFMI and ALMI). Different from our study, all the 8 approaches were independently associated with increased 5-year mortality risk despite confounding factors.[27] It is obvious from this conflicting result that more longitudinal studies from different populations with larger cohorts will determine which diagnostic method for third criterion is stronger to predict mortality and other adverse outcomes related to malnutrition.

This study harbors several limitations. First of all, it was conducted on outpatients living in the community who might be considered relatively healthier older adults. For reliable measurements, we had to exclude some of the most vulnerable individuals, such as patients with dementia or stroke, and this may have led to selection bias. Thus, the findings cannot be generalized to whole older adult population. Another limitation is the retrospective design of the study. Since GLIM criteria were not published during the commencement of the data collection, some items were indirectly assessed (like weight loss and reduced food intake and assimilation), and some might be assessed insufficiently (since disease burden was not assessed). In addition, although we reached out for information about mortality, we did not know the actual causes of deaths. Hence, the cause of mortality may have nothing to do with the nutritional status of the deceased individuals. Another major limitation can be considered as low mortality rate, since the relationship between malnutrition and mortality could have been stronger and more significant in a sample with a higher mortality rate. The major strength of the study is that it is one of the limited studies in the literature searching for the GLIM version that better predicted mortality among six different pragmatic approaches. Studies reporting the GLIM-mortality relationship are mostly conducted on populations with specific diseases (like cancer or surgery) and inpatients; therefore, we consider that a study searching for predictive validity of the GLIM criteria in outpatients with different comorbidity profiles will serve to fill the gap in the literature. Finally, we used the population-specific thresholds for all of the methods used to assess the third phenotypic criterion; hence, this represents a particular strength of the study that distinguishes it from many other similar studies that used nonspecific, conventional thresholds.

Our study revealed that the use of GLIM criteria in malnutrition practice among older outpatients living in the community resulted in a broad range of prevalences, depending on the definition used for the reduced muscle mass criterion. Moreover, none of the GLIM versions were independently associated with mortality, as increased age was the only significant predictor of mortality in an older population considered to be relatively healthy. The gap regarding the ideal GLIM version that both identifies malnutrition and predicts adverse outcomes better in this population will be filled with further longitudinal studies with larger cohorts and different approaches regarding the reduced muscle mass criterion.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of İstanbul University İstanbul Faculty of Medicine (Date: November 25, 2022, Number: 1399682).

Informed Consent: Written informed consent was obtained from older adults who participated in this study.

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Supplementary Table 1. Cox regression analyses showing association of different versions of GLIM and other independent factors with mortality

Independent variables	HR (95 % Confidence interval)	*P* value
GLIM (SMM/h²)		
Model 1		
GLIM (SMM/h²)	**3.8 (1.1 - 13.7)**	**0.040**
Model 2		
GLIM (SMM/h²)	2.2 (0.6 – 8.1)	0.221
Age	1.15 (1.1 – 1.2)	<0.001
Model 3		
GLIM (SMM/h²)	2.3 (0.6 – 8.8)	0.212
Sex (female)	0.3 (0.09 – 0.9)	0.028
Model 4		
GLIM (SMM/h²)	1.5 (0.4 – 5.6)	0.594
Age	1.15 (1.06 – 1.2)	<0.001
Sex (female)	0.4 (0.1 – 1.2)	0.088
GLIM (SMM/BMI)		
Model 1		
GLIM (SMM/BMI)	1.7 (0.6 – 5.0)	0.363
Model 2		
GLIM (SMM/BMI)	1.1 (0.4 – 3.4)	0.850
Age	1.15 (1.07 – 1.2)	<0.001
Model 3		
GLIM (SMM(BMI)	1.9 (0.6 – 5.5)	0.272
Sex (female)	0.3 (0.08 – 0.8)	0.022
Model 4		
GLIM (SMM/BMI)	1.2 (0.4 – 3.7)	0.721
Age	1.16 (1.06 – 1.2)	<0.001
Sex	0.4 (0.1 – 1.2)	0.095
GLIM (HGS)		
Model 1		
GLIM (HGS)	**4.3 (1.4 – 12.8)**	**0.009**
Model 2		
GLIM (HGS)	2.5 (0.9 – 7.6)	0.096
Age	1.15 (1.1 – 1.2)	<0.001
Model 3		
GLIM (HGS)	**4.2 (1.4 – 12.5)**	**0.010**
Sex (female)	0.3 (0.1 – 0.8)	0.021
Model 4		
GLIM (HGS)	2.2 (0.7 – 6.9)	0.174
Age	1.14 (1.05 – 1.2)	0.002
Sex (female)	0.4 (0.1 – 1.3)	0.138
GLIM (CC)		
Model 1		
GLIM (CC)	**4.6 (1.3 - 16.7)**	**0.019**
Model 2		
GLIM (CC)	2.5 (0.7 – 9.2)	0.161
Age	1.15 (1.1 – 1.2)	<0.001
Model 3		
GLIM (CC)	**3.6 (0.98 – 13.0)**	**0.054**
Sex (female)	0.3 (0.1 – 0.8)	0.019
Model 4		
GLIM (CC)	1.8 (0.5 – 6.9)	0.388
Age	1.15 (1.1 – 1.2)	<0.001
Sex (female)	0.4 (0.1 – 1.2)	0.095
GLIM (adj. CC)		
Model 1		
GLIM (adj CC)	1.3 (0.4 – 4.9)	0.656
Model 2		
GLIM (adj CC)	0.7 (0.2 – 2.6)	0.566
Age	1.16 (1.08 – 1.2)	<0.001
Model 3		
GLIM (adj CC)	1.3 (0.4 – 4.6)	0.726
Sex (female)	0.3 (0.1-0.8)	0.024
Model 4		
GLIM (adj CC)	0.7 (0.2 – 2.7)	0.619
Age	1.15 (1.07 – 1.3)	<0.001
Sex (female)	0.4 (0.1 – 1.2)	0.100
GLIM (without P3 criterion)		
Model 1		
GLIM (w/out P3)	**7.3 (2.0 – 26.5)**	**0.003**
Model 2		
GLIM (w/out P3)	**4.0 (1.1 – 14.6)**	**0.039**
Age	1.14 (1.1 – 1.2)	<0.001
Model 3		
GLIM (w/out P3)	**5.9 (1.6 – 21.7)**	**0.007**
Sex (female)	0.3 (0.1-0.9)	0.018
Model 4		
GLIM (w/out P3)	3.0 (0.8 – 11.6)	0.117
Age	1.13 (1.1 – 1.2)	0.001
Sex (female)	0.4 (0.1 – 1.2)	0.092

Abbreviations and acronyms: adj CC: adjusted calf circumference; BMI: body mass index; CC: calf circumference; GLIM: Global Leadership Initiative on Malnutrition; HGS: handgrip strength; HR: Hazard ratio; P3: third phenotypic criterion; SMM: skeletal muscle mass

*Model 1 is the unadjusted (crude) analysis between malnutrition defined by GLIM and mortality.

**Age and sex were identified as other independent variables in the Cox regression analyses in addition to malnutrition defined by the GLIM versions, as they were found to be significantly associated with mortality in univariate analyses.

CLINICAL SCIENCE OF NUTRITION

Original Article

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Screening for Nutritional Status in the Outpatient Setting Across Different Clinical Specialities in Turkiye: A Cross-Sectional NutritionDay Awareness Survey

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ABSTRACT

Objective: To screen the nutritional status and determine the prevalence of malnutrition (MN) or MN risk among newly diagnosed and follow-up patients in different outpatient speciality clinics across Turkey

Methods: A total of 3521 patients from 52 outpatient speciality clinics across Turkiye were included in this cross-sectional study. MN risk and/or MN were evaluated using Nutritional Risk Screening 2002 (NRS 2002) and Mini Nutritional Assessment (MNA) tools. Time of diagnosis (new admissions vs. follow-up patients) was compared with the nutritional status.

Results: Overall, 652 (18.7%) of 3492 patients were at risk of MN according to NRS 2002, while 381 (40.9%) of 931 geriatric patients assessed by MNA were either malnourished (scores <17, 14.7%) or at risk of MN (scores 17-23.5, 26.2%). MN risk was more prevalent in medical oncology patients (44.1%), as well as in new vs. follow-up patients (23.1% vs. 19.0%, *P* = .007), particularly in radiation oncology (30.5% vs. 15.7%, respectively), medical oncology (47.2% vs. 41.6%, respectively) and geriatric (69.6% vs. 46.5%) clinics. In geriatric outpatient clinics, NRS 2002 showed MN risk in 35.3% of the patients those were at MN risk according to MNA, which was only 45.9% for those with MN (MNA score lower than 17)

Conclusion: In conclusion, this screening study in the outpatient setting across different clinical specialities revealed poor nutritional status in 1 out of every 5 patients overall, and nearly 1 out of 2 patients admitted to medical oncology and geriatrics clinics, respectively. In geriatric patients, NRS 2002 seems to underestimate MN risk compared to MNA. Given the higher MN risk prevalence in cancer and geriatric patients, it is important to screen nutritional status in those patients, especially during the first admission.

Keywords: Malnutrition, nutritional assessment, outpatient care, medical oncology, radiation oncology

INTRODUCTION

Malnutrition (MN) is a serious health problem with major adverse health outcomes such as frequent infections, poor wound healing, impaired quality of life, and increased morbidity and mortality in addition to prolonged length of hospital stay (LOS) and increased healthcare costs.[1,2]

Malnutrition is considered to be prevalent across several healthcare settings, particularly in hospitalized patients,

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in elderly patients, and in patients with chronic comorbid conditions.[1-3]

The hospital studies reported that 30%-50% of adult inpatients were malnourished or at risk upon admission, particularly the older adults and those with complicating health conditions.[3,4] The prevalence of MN or risk of MN in the community setting was reported to range from 20% to 30%, while much higher rates (up to 70%) were considered in older adults.[5-7]

Malnutrition is a preventable condition through early identification of poor nutritional status via validated screening tools and timely provision of appropriate nutritional intervention tailored to the individual needs of at-risk or malnourished patients.[1,8-10] Nonetheless, MN remains an under-recognized and under-diagnosed condition with detrimental consequences in the clinical practice, due to insufficient awareness of clinicians and lack of uniform screening tools and diagnosis protocols.[2,9,11]

In this regard, the NutritionDay initiative, an annual worldwide cross-sectional multicenter audit promoted by the European Society for Clinical Nutrition and Metabolism (ESPEN) in 2006, has become performed annually as a single-day screening to determine the prevalence of MN in hospitalized patients via a simple nutritional screening tool.[12-14] Many NutritionDay audits have been conducted in the inpatient setting across 8000 hospital wards in nearly 300 000 patients globally, improving the knowledge and awareness of MN among hospitalized patients.[12-14]

However, issues related to the nutritional screening for systematic identification of MN risk and the provision of nutritional intervention in the ambulatory outpatient setting have been less extensively addressed and not as well documented as in the inpatient setting.[15]

Main Points

- This screening study in the outpatient setting across different clinical specialties revealed poor nutritional status in 1 out of every 5 patients overall, and nearly 1 out of 2 patients admitted to medical oncology and geriatrics outpatient clinics based on Nutritional Risk Screening 2002 and MNA screening tools, respectively.
- Given the higher prevalence of malnutrition risk in new vs. follow-up patients, screening for nutritional risk in every cancer patient and geriatric patient during the time of initial diagnosis seems crucial to achieve the improved long-term health outcomes via timely provision of appropriate multimodal nutritional intervention.
- In this regard, efforts to increase awareness among clinicians regarding the appropriate and timely use of nutritional screening tools are crucial to be able to recognize the malnutrition risk at an earlier and more responsive phase and to improve patient outcomes through appropriate nutritional support.

Therefore, this cross-sectional screening study aimed to determine nutritional status among newly diagnosed and follow-up patients in multiple outpatient speciality clinics across Turkey in collaboration with Turkish Society of Clinical Enteral and Parenteral Nutrition (KEPAN) as an awareness-raising project within the context of World Nutrition Day.

METHODS

Study Population

A total of 3521 adult patients who were evaluated for nutritional status via Nutritional Risk Screening 2002 (NRS 2002) and Mini Nutritional Assessment (MNA) during their admission to 52 outpatient speciality clinics across Turkiye were included in this cross-sectional study conducted between September 25, 2019, and October 25, 2019.

All of the participating centers were hospitals. Primary care centers were not included in the study. The study was performed in the surgery, medical oncology, radiation oncology, geriatrics, and neurology clinics of 32 different hospitals. Of these 20 were university hospitals, 8 were state hospitals, and 4 were private hospitals.

Written informed consent/assent was obtained from each patient. The study was conducted in accordance with the ethical principles stated in the "Declaration of Helsinki" and approved by Hacettepe University Non-interventional Clinical Research Ethics Committee (Date of Approval: September 17, 2019, Protocol No: 2019/22-21).

Assessments

Cancer type (in oncology patients), time of diagnosis (newly diagnosed patients, follow-up patients), and prevalence of MN risk and/or MN using the NRS 2002 and MNA tools were recorded. NRS score ≥3 indicated the risk of MN.[16] Mini Nutritional Assessment was used only in elderly patients in the geriatric clinics, with consideration of the absence of MN, risk of MN, and the presence of MN for scores over 23.5, between 17 and 23.5, and <17, respectively.[17]

Statistical Analysis

Statistical analysis was done using IBM Statistical Package for Social Sciences (IBM SPSS Corp., Armonk, NY, USA) Statistics for Windows, version 20.0 (PASW statistics 20). Descriptive statistics were reported including percentages for categorical variables. Chi-square ($\chi 2$) test and Fisher's exact test were used for the comparison of categorical data including the MNA and NRS 2002 results in

subgroups of outpatient clinics and time of diagnosis as well as their cross-classification. $P < .05$ was considered statistically significant.

RESULTS

Participating Clinics, Time of Diagnosis, and Assessment Tools

Of the 52 centers participated in the study, 21 were geriatrics clinics comprising 1006 (28.6%) of 3521 patients in the overall study population. NRS 2002 and MNA scores were not available in 29 and 75 geriatric patients, respectively, while data on time of diagnosis were not available in 561 patients (Table 1).

Overall, 40.1% of the patients were new, while 59.9% of patients were chronic follow-up patients. The percentage of follow-up patients was higher in geriatrics (91.9%), neurology (67.1%), and medical oncology (55.9%) clinics, whereas general surgery (67.0%) and radiation oncology (61.9%) clinics were associated with higher percentage of first admission patients ($P < .001$) (Table 1).

Nutritional Risk Screening 2002 and Mini Nutritional Assessment Scores According to Outpatient Clinics and Time of Diagnosis

Overall, 652 (18.7%) of 3492 patients had MN risk according to NRS 2002, while 381 (40.9%) of 931 geriatric patients indicated MN (14.7%) and/or MN risk (26.2%) according to MNA (Table 2).

Based on NRS 2002 results, medical oncology (44.1%) and radiation oncology (25.0%) clinics had higher MN risk prevalence ($P < .001$). Normal nutritional status was less prevalent in new admission patients than in follow-up patients according to both NRS 2002 (76.9% vs. 81.0%, $P = .007$) and MNA (30.4% vs. 53.5%, $P < .01$) assessments (Table 2, Figure 1).

Poorer nutritional status in new vs. follow-up patients was particularly noted for radiation oncology (30.5% vs. 15.7%, respectively) and medical oncology (47.2% vs. 41.6%, respectively) patients, as well as in geriatric patients assessed by MNA (69.6% vs. 46.5%, respectively) (Tables 2 and 3, Figure 1).

Cross-Classification of Nutritional Risk Screening 2002 and Mini Nutritional Assessment Scores

We performed cross-classification of NRS 2002 and MNA in 902 geriatric patients. NRS 2002 showed MN risk in 169 (18.7%) patients, while MNA revealed MN risk in 241 (26.7%) patients and MN in 122 (13.5%) patients. Of 511 patients with normal nutrition status on both tools, 94.8% (511/539) were those assessed by MNA and 69.7% (511/733) were those assessed by NRS 2002. Of 85 patients who were found to be at risk of MN on both tools, 35.3% (85/241) were those assessed by MNA and 50.3% (85/169) were those assessed by NRS 2002. In 56 patients, NRS 2002 scores indicated the risk of MN (33.1% of 169 patients), while MNA scores indicated the presence of MN (45.9% of 122 patients). Accordingly, only 35.3% of patients who were at risk of malnutrition and 45.9% of malnourished patients according to MNA were accurately identified with NRS 2002 ($P < .001$) (Table 4).

DISCUSSION

The main scope of this research was basically to define the risk of MN among different outpatient clinics and to

Table 1. Distribution of Outpatient Clinics, Time of Diagnosis, and Assessment Tools

	n[a]	Screened Patients, n (%)	Time of Diagnosis			Assessed by NRS 2002	Assessed by MNA
			Newly Diagnosed	Follow-up	Total		
Outpatient clinic							
Geriatrics	21	1006 (28.6)	58 (8.1)	659 (91.9)	717	977[b]	931[c]
General surgery	9	811 (23.0)	364 (67.0)	179 (33.0)	543	811	–
Neurology	6	748 (21.2)	245 (32.9)	500 (67.1)	745	748	–
Radiation oncology	8	552 (15.7)	341 (61.9)	210 (38.1)	551	552	–
Medical oncology	8	404 (11.5)	178 (44.1)	226 (55.9)	404	404	–
Total	52	3521 (100.0)	1186 (40.1)	1774 (59.9)	2960[d]	3492	931

[a]The number of participated centers, NRS 2002 scores, and MNA scores were not available in [b]29 and [c]75 geriatric patients, respectively; [d]data on time of diagnosis were not available in 561 patients overall.

Table 2. Nutritional Risk Screening 2002 and Mini Nutritional Assessment Scores According to Outpatient Clinics and Time of Diagnosis

	NRS 2002 Scores			
	<3 (normal)	≥3 (Malnutrition Risk)		*P*
Outpatient Clinic	**n (%)**	**n (%)**		
Geriatrics (n = 977)	801 (82.0)	176 (18.0)		**<.001**
General surgery (n = 811)	695 (85.7)	116 (14.3)		
Medical oncology (n = 404)	226 (55.9)	178 (44.1)		
Neurology (n = 748)	704 (94.1)	44 (5.9)		
Radiation oncology (n = 552)	414 (75.0)	138 (25.0)		
Total (n = 3492)	2840 (81.3)	652 (18.7)		
Time of diagnosis	n (%)	n (%)		
Follow-up patient (n = 1747)	1415 (81.0)	332 (19.0)		**.007**
Newly diagnosed (n = 1184)	910 (76.9)	274 (23.1)		
Total (n = 2931)	2325 (79.3)	606 (20.7)		
	MNA scores			
	>23.5 (normal)	17 to 23.5 (malnutrition risk)	<17 (malnourished)	*P*
	n (%)	n (%)	n (%)	
Geriatrics (n = 931), n (%)	550 (59.1)	244 (26.2)	137 (14.7)	
Time of diagnosis				
Follow-up patient (n = 622)	333 (53.5)	177 (28.5)	112 (18.0)	**.006**
Newly diagnosed (n = 46)	14 (30.4)*	17 (37.0)	15 (32.6)	
Total (n = 668)	347 (51.9)	194 (29.0)	127 (19.0)	

*$P < .01$; compared to normal nutritional status in follow-up patients (with Bonferroni corrected *P*-value: .016).

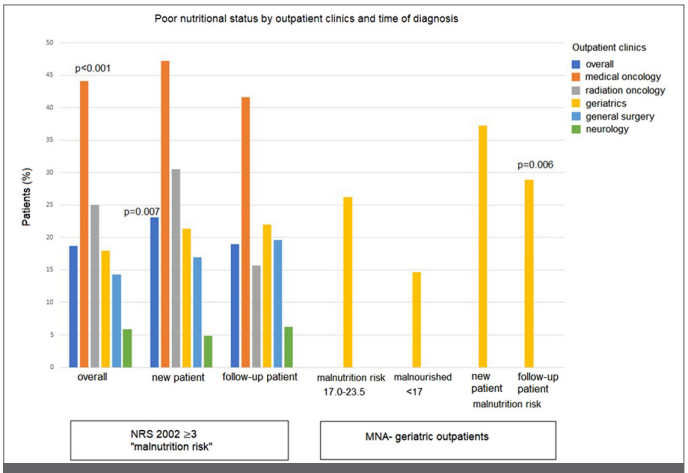

Figure 1. Poor nutritional status (NRS 2002 scores ≥3 and MNA scores 17.0-23.5 or <17) by outpatient clinics and time of diagnosis.

determine the differences between NRS and MNA to measure the MN risk in older adults in different sites of Turkey cross-sectionally. Our findings revealed poor nutritional status and the need for nutritional intervention in 1 out of every 5 patients overall, and nearly 1 out of 2 patients admitted to medical oncology and geriatrics outpatient clinics.

Previous cross-sectional NutritionDay studies in the inpatient setting revealed that 27%-40% of hospitalized patients were at risk for MN, and MN prevalence differed depending on the screening tool, hospital unit, and age of the patient.[12,13,18,19] A high prevalence of MN risk in the current study emphasizes that nutritional screening for early identification and multimodal intervention of poor nutritional status is also important in the outpatient setting, particularly among cancer patients and elderly.[2,15,20]

Table 3. NRS 2022 Scores According to Time of Diagnosis for Each Outpatient Clinic

Outpatient Clinic		NRS 2002 Scores	
		<3 (Normal Status)	≥3 (Malnutrition Risk)
		n (%)	n (%)
Geriatrics (n = 977)	Newly diagnosed (n = 56)	44 (78.6)	12 (21.4)
	Follow-up patient (n = 632)	493 (78.0)	139 (22.0)
	Total (n = 688)	537 (78.1)	151 (21.9)
General surgery (n = 811)	Newly diagnosed (n = 364)	302 (83)	62 (17.0)
	Follow-up patient (n = 179)	144 (80.4)	35 (19.6)
	Total (n = 543)	446 (82.1)	97 (17.9)
Medical oncology (n = 404)	Newly diagnosed (n = 178)	94 (52.8)	84 (47.2)
	Follow-up patient (n = 226)	132 (58.4)	94 (41.6)
	Total (n = 404)	226 (55.9)	178 (44.1)
Neurology (n = 748)	Newly diagnosed (n = 245)	233 (95.1)	12 (4.9)
	Follow-up patient (n = 500)	469 (93.8)	31 (6.2)
	Total (n = 745)	702 (94.2)	43 (5.8)
Radiation oncology (n = 552)	Newly diagnosed (n = 341)	237 (69.5)	104 (30.5)
	Follow-up patient (n = 210)	177 (84.3)	33 (15.7)
	Total (n = 551)	414 (75.1)	137 (24.9)
Total (n = 3492)	Newly diagnosed (n = 1184)	910 (76.9)	274 (23.1)
	Follow-up patient (n = 1747)	1415 (81)	332 (19.0)
	Total (n = 2931)	2325 (79.3)	606 (20.7)

Table 4. Cross-Classification of NRS 2002 and MNA Scores (n = 902)

		MNA Scores			
		Normal Status (n = 539)	At Risk of Malnutrition (n = 241)	Malnourished (n = 122)	*P*
NRS 2002 scores					
At risk of malnutrition (n = 169)	n	**28**	**85**	**56**	**<.001**
	% within NRS 2002	16.6	50.3	33.1	
	% within MNA	5.2	35.3	45.9	
Normal status (n = 733)	n	**511**	**156**	**66**	
	% within NRS 2002	69.7	21.3	9.0	
	% within MNA	94.8	64.7	54.1	

Indeed, oncology inpatients are considered to have at least 1.5 times higher rate of MN diagnosis compared with other hospitalized populations.[10,21] Medical oncology and radiation oncology patients in the present study were also at higher risk of MN than other patient populations, along with the further increase in the MN risk among newly diagnosed vs. follow-up patients. Similarly, in a multicenter NRS 2002–based screening study by KEPAN among 29 139 patients, the MN risk at the time of hospital admission (15% overall) was reported to increase up to 43.4% in medical oncology (19.5% in radiation oncology) clinics.[22] In another cross-sectional NRS 2002-based NutritionDay screening study by Turkish Society of Radiation Oncology, 33.8% patients including 36.0% of newly diagnosed patients were reported to be at risk for MN, indicating a need for nutritional intervention in 2 out of every 5 patients with newly diagnosed cancer.[10]

The presence of MN risk in nearly half of our newly diagnosed cancer patients is also notable given that MN prevalence ranges from 40% at cancer diagnosis to 70%-80% in advanced disease stages, and the anti-cancer treatments contribute to an additional deterioration of the nutritional status.[9,10,22-24] Besides, the early recognition of MN is also important since the clinical nutrition is considered more effective during earlier phase before the emergence of advanced cachexia.[9,10,25,26]

The presence of poor nutritional status in 40.9% (MN risk in 26.2% and MN in 14.7%) of geriatric patients in our study is in line with the prevalence of MN risk (24.0%-36.0%) and MN (13.0%-19.0%) among geriatric patients reported in previous studies using the MNA tool.[20,27] In a systematic review and meta-analysis of studies on the nutritional screening via the MNA tool in older adults across different healthcare settings, the prevalence of MN was reported to range from 3% (in the community setting) to 30% (in

rehabilitation and subacute care).[28] In a systematic review of studies including 22 MN screening tools validated for use in elderly population, the prevalence of MN risk was reported to range from 8.5% (in the community setting) to 28.0% (in the hospital setting) across screening tools.[3]

In general, the prevalence of MN is considered to be high in older adults and to further increase with age and the number of comorbidities, contributing also to the development of the geriatric syndromes in these patients.[5,29] Hence, nutritional screening at regular intervals is strongly recommended in older adults at initial diagnosis and hospital admission as well as during outpatient follow-up since early identification and management of poor nutritional status can lead to improved outcomes and quality of life.[3,5,6,8,20,28,29]

The NRS 2002 is the ESPEN-recommended screening tool for hospitalized patients with high sensitivity and specificity, particularly in critically ill patients, and its association with morbidity, mortality, and LOS was reported in many studies.[2,16,30,31] Our findings support the consideration of NRS 2002 as a suitable tool for screening nutritional risk in cancer patients at the time of initial diagnosis, which enables planning the appropriate nutritional care as an essential component of multimodal therapy in oncology practice.[2,9,10,32,33]

Considering elderly outpatients, while there are no uniform tools for assessing the risk of MN in this population, there is a range of recommended simple and validated comprehensive screening tools, such as NRS 2002 (a high sensitivity, negative predictive value) and MNA (a high clinical sensitivity and specificity).[2,5,32,34-36] However, NRS 2002 tool was able to identify the MN risk in only one-third of our geriatric patients who were at risk of MN on MNA and half of those who were malnourished on MNA. In this regard, the use of MNA as a screening tool in geriatric population seems to be more appropriate in terms of accurate identification of poor nutritional status which otherwise may easily be overlooked if screening is based solely on NRS 2002. Nonetheless, it should also be noted that the specificity of the MNA has been questioned in terms of a potential risk of "over-diagnosing" MN in the older adults.[29,37]

Hence, since none of the current screening tools per se is considered sufficiently reliable to determine the nutritional status in varying clinical situations and the prevalence of MN risk varies considerably depending on the screening tools, complementary use of more than 1 nutritional screening tool is suggested.[2,29,38,39] Accordingly, complementing MNA with the Global Leadership Initiative on MN (GLIM) criteria is suggested to provide more accurate prevalence of MN and more reliable data on prediction of the incident sarcopenia in older adults.[29,40,41] Also, in an analysis of the NutritionDay database in the inpatient setting, traditional screening tools (such as NRS 2002, Malnutrition Screening Tool [MST], and Malnutrition Universal Screening Tool [MUST]) applied at admission and repeatedly during hospitalization are considered to fail to identify a group of patients at risk due to reduced intake during hospitalization since these tools do not include monitoring for current food intake.[18] In a cross-sectional study in cancer outpatients, the prevalence of MN was reported to be higher with use of GLIM criteria (46.7%) compared to using the ESPEN criteria (21.2%), and the authors considered the association of new GLIM criteria with a greater sensitivity in early diagnosis and thus early intervention of MN in cancer patients.[42]

The prevalence of MN risk (14.3%) in our general surgery patients, similarly in new and follow-up patients, seems in line with the previous studies indicated the prevalence of MN to range from 14% to 25% in the medical and surgical gastroenterology patients with no difference between new and follow up patients.[43,44] Although the neurology clinics were associated with lowest MN risk prevalence in our study, MN in the neurology outpatient setting has been reported to differ significantly (0.8%-32%) with respect to underlying disease (higher for stroke, CNS infections and movement disorders than polyneuropathy, demyelinating diseases, epilepsy, or pseudotumor cerebri) as well as the presence of co-morbid diabetes.[45,46]

The major strength of this screening study seems to be the inclusion of 52 outpatient speciality clinics across Turkey and comprehensive analysis of MN risk or MN with use of standardized screening tools across centers. However, certain limitations to this study should be considered. First, due to the cross-sectional design, it is impossible to establish any cause-and-effect relationships. Secondly, nutritional screening was based on single-point assessment with no data on follow-up status with respect to multimodal cancer treatment or provision of nutritional support. Third, lack of detailed data on patient and treatment characteristics is another limitation which otherwise would extend the knowledge achieved in the current study. Nevertheless, this was a screening study conducted as an awareness-raising project within the context of World Nutrition Day, providing a snapshot of the nutritional status in outpatient setting across Turkey.

In conclusion, this screening study in the outpatient setting across different clinical specialties revealed poor nutritional status in 1 out of every 5patients overall and nearly 1 out of 2 patients admitted to medical oncology and geriatric outpatient clinics based on NRS 2002 and

MNA screening tools, respectively. In this regard, efforts to increase awareness among clinicians regarding the appropriate and timely use of nutritional screening tools are crucial to be able to recognize the MN risk at an earlier and more responsive phase and to improve patient outcomes through appropriate nutritional support.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Hacettepe University Non-interventional Clinical Research Ethics Committee (Date: September 17, 2019, Number: 2019/22-21).

Informed Consent: Written informed consent/assent was obtained from each patient following a detailed explanation of the objectives and protocol.

Peer-review: Externally peer-reviewed.

Author Contributions: OA contributed to conception/design of the research and acquisition, analysis and interpretation of the data; drafted the manuscript and critically revised the manuscript; other members of the KEPAN NutritionDay Study Group contributed to conception/design of the research and contributed to acquisition, analysis and interpretation of the data. OA agrees to be fully accountable for ensuring the integrity and accuracy of the work. All authors read and approved the final manuscript.

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Review Article

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An Alternative Approach to Nutrition: Intuitive Eating

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ABSTRACT

Intuitive eating is the act of eating in response to physiological hunger and satiety signals. In intuitive eating, environmental and emotional events are not affected, and it is possible to display intuitive eating behavior only by responding to bodily signals. Medical nutrition is a primary method in the treatment of obesity, the prevalence of which is increasing day by day. After medical nutrition therapy, drug therapy and surgical intervention come to mind. In addition to these medical interventions, intuitive eating and eating awareness practices, which are proposed approaches, are also methods with a high level of success in preventing obesity. In this review, the approaches to intuitive eating and the factors affecting intuitive eating behavior are discussed. Emotional eating theories, factors affecting emotional eating, and risk factors are emphasized, and the relationship between intuitive eating and emotional eating is examined. An inverse relationship was found between emotional eating and intuitive eating behaviors, and it was concluded that as the awareness of eating increased, the level of intuitive eating increased and emotional eating behavior decreased. Making peace with food is one of the basic principles of intuitive eating; it has been observed that the behavior of eating without any prejudice and without classifying foods as good or bad increases nutritional pleasure, and it has been concluded that individuals with eating behavior disorders, especially consuming foods that are described as "forbidden," reduce their eating attacks.

Keywords: Eating awareness, emotional eating, intuitive eating, obesity

INTRODUCTION

Eating behavior is one of the basic behaviors that living things display throughout their lives in order to survive. Eating behavior develops with age and may change according to mood. The behavior of eating for survival can give pleasure to the individual, while at the same time, it can cause inadequate or excessive nutrition problems and also invite health problems such as eating disorders. Eating behaviors are highly influenced by human emotions.[1]

Intuitive Eating

Intuitive eating is an approach developed in response to physiological hunger and satiety signals, without being affected by emotional and environmental stimuli.[2] Since intuitive eating is an approach that imposes on people how they should respond to their physiological signals, some addictions that people unintentionally create under the influence of external factors related to their bodies and foods are discussed under the name of intuitive eating.[3]

Basic Approaches to Intuitive Eating

Unconditional Consent to Eat

Unconditional consent to eat reflects readiness for consumption in response to homeostatic hunger signals and the desired food.[4] It is also expressed as the ability of an individual to eat the foods they want without hesitation when they are hungry and to refuse to label foods as "forbidden" or "bad."[5] Individuals who restrict the time of eating, amount and type of food consumed experience more feelings of deprivation and the eating situation can get out of control more easily. Dietary restriction can increase anxiety about food in the long run.[4]

Eating for Physical Rather Than Emotional Reasons

Individuals with intuitive eating behavior do not use their eating behavior to cope with their emotional fluctuations. Instead, the sole purpose of eating behavior is to satisfy the homeostatic hunger drive.[5] When they are hungry, they exhibit the behavior of eating to avoid the feeling of hunger, and when the feeling of satiety occurs, they stop the eating behavior.[6] Since the level of intuitive

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eating improves coping skills, individuals who eat intuitively are less likely to use food as a means of coping with emotions.[2]

Relying on Hunger and Fullness Signals to Determine When and How Much to Eat

According to intuitive eating, the innate homeostatic hunger and satiety signals of the human body control the eating behavior. Awareness of these innate internal signals can be replaced by external rules about when and how much to eat as social messages about the effect of food restrictions on weight loss in adulthood are adopted.[7]

Intuitive Eating and Physiological Signals

It is known that the intuitive eating instinct is innate, and if we respond to our body's signals, the body is naturally capable of maintaining the required body weight and adjusting the nutrients it needs. This innate ability of a person is called "body wisdom." As a result of a recent study, it has been shown that the rate of dieting and eating disorders is lower in individuals who respond to signals from their body about when and how much to eat. Likewise, it has been shown that the rate of binge eating disorder is lower for women, and the rate of chronic dieting for women is lower because they stop eating when they feel full.[8] People's insistence on eating the food they make, advertisements encouraging them to eat, large portions in some restaurants, and so on are some of the factors taught by the society that prevent the body from responding to the hunger–satiety signals, that is, trying to undermine the innate bodily instinct. If the people who take care of the child (parents, grandparents, caregivers, etc.) force the child to eat when the child does not want to eat and use the food they want to consume to calm the children, the children's confidence in signals such as hunger, satiety, and appetite decreases and the development of body wisdom is prevented.[9]

Main Points

- In this review, the effect of intuitive eating on emotional eating and eating awareness was investigated. The relationship between eating awareness and eating behavior disorders is emphasized.
- The relationship between intuitive eating and body weight control and its effect on dieting behavior are mentioned.
- As a result of the literature reviews, a significant relationship was found between the intuitive eating behavior score and body weight control.
- Studies have shown that individuals with eating awareness have a lower risk of experiencing eating disorders.

Factors Affecting Intuitive Eating

Gender

One of the most influential factors on intuitive eating is gender. As a result of a study that examined the relationship between the level of intuitive eating and gender and conducted with the participation of 260 university students, it was seen that male individuals had higher total intuitive eating scores compared to female individuals. At the same time, it was concluded that the rate of displaying emotional eating behavior among men is lower than that among women.[10]

A significant gender difference has been noted in intuitive eating and health awareness.[11] Studies have shown that intuitive eating behavior is associated with low body mass index (BMI) for both men and women.[12]

In a study conducted on 182 female and 68 male participants, the average of the intuitive eating scale scores of men was found to be higher than that of women.[13]

Obesity

As a result of studies examining the relationship between BMI and intuitive eating, it was concluded that there is an inverse relationship between intuitive eating behavior and BMI.[14]

In a study examining the relationship between intuitive eating behavior and body weight, it was determined that individuals with high intuitive eating scores had a lower BMI compared to individuals with low intuitive eating scores.[11,12] It is also suggested that intuitive eating plays a protective role in the development of obesity. As a result of another study conducted on students, it was found that people with high intuitive eating scores had lower BMI scores, lower health awareness, and higher behavior of eating for pleasure.[12]

Dieting Behavior

Intuitive eating affects dieting behavior positively because it increases awareness of eating and directs it to healthy foods.[15] On the other hand, because diet is compatible with restrictions on individuals, dieting behavior negatively affects intuitive eating.[15,16] Given the failure of calorie-restricted diets in reducing body weight in the long term, intuitive eating is a very convenient way of eating to maintain appropriate body weight as a solution to the eating behavior relationship that is disrupted by long-term restrictive diets.[16] Weight loss is not one of the main goals of intuitive eating, but intuitive eating allows approaching the ideal body weight.[12]

Psychological Factors

Like many eating-related behaviors, intuitive eating is also affected by psychological factors. Body dissatisfaction can

result in high BMI and excessive fat accumulation in the body as a result of unbalanced food intake. As the intuitive eating score increases, body satisfaction increases and eating disorder symptoms decrease.[3]

Emotional Eating

Emotional eating is an eating disorder that is developed to cope with negative emotions and is characterized by excessive food consumption.[17] Studies have suggested that eating attacks may be closely related to emotional eating behavior. It has been observed that this uncontrolled binge eating reaction triggered by negative emotions is more common in women with eating disorders and in obese individuals.[17,18]

Stress and negative emotions can prevent dieters from being loyal to self-imposed rules and restrictions on food intake. In the case of undereating, the body distinguishes self-imposed food restriction from real food scarcity and acts as if it were in starvation mode. In this case, while the metabolic rate slows down, hunger and appetite increase.[18]

Emotional Eating Theories

Although the relationship between negative emotions and binge eating is clearly associated, it is not known exactly which negative emotion triggers the urge to eat in individuals and by which mechanism. Theories about emotional eating are generally based on obesity research, since emotional eating behavior reduces success in dieting and prevents reaching the ideal body weight.[19]

Schachter's Internal–External Theory of Obesity

The internal–external theory of obesity put forward by Schachter[20] proposes that while the symptoms of negative emotions such as fear, anxiety, and unhappiness in the body cause a decrease in food consumption for individuals with the ideal body weight, this does not happen for individuals with a BMI above 30 kg/m^2 who are in a state of insensitivity to internal stimuli. In Schachter's theory, it is stated that some aspects of hunger are learned, but this learning situation is not valid for individuals with body weight above normal. In the external eating theory, individuals are not sensitive to their homeostatic hunger and satiety signals. The extrinsic eating theory leads to the resumption of the eating event. The eating perceptions of individuals with an external eating attitude are only revealed when they are in the same environment with the food. External eating behavior occurs as a result of being affected by sensory characteristics such as the smell and appearance of the food.[21]

Restriction Theory

The basis of the restriction theory is that as a result of the excessive desire to eat foods, the individual creates a restriction in his/her own mind against this desire. Individuals with restrictive eating behaviors constantly complain about overeating and restrict their eating habits. What is mentioned in this restriction is the restriction that individuals make by their effort to consume less than the amount they want to eat, not to take food as much as they need.[22]

Individuals who limit their long-term eating behavior cause this restriction to disappear beyond their control after a while, and this behavior leaves its place to excessive eating behavior. The transformation of restrictive eating into excessive eating behavior is generally seen in individuals who restrict the amount of energy they need to take daily.[23]

Escape Theory

The escape theory is the theory that is claimed to be used as an escape or self-defense from environments in which emotional eating causes negative awareness. It is thought that the escape theory is exhibited to avoid being in the same environment with stimuli that can change awareness or to distract attention from these stimuli. According to this theory, individuals who display emotional eating behavior tend to flee in order to avoid negative emotions and deterrent effects when they are confronted with information that threatens their selves. These individuals escape from this awareness by the act of turning to external stimuli that result in overeating.[24]

To make a general comment, it can be said that emotional eaters use their eating behaviors as a way of coping with negative emotions, and then this behavior gets out of control and becomes problematic.[25] The views advocated in the theories of emotional eating, that there is a tendency to avoid the negative effects of emotional eating, are also related to the way individuals cope with other independent situations. Strategies using these coping styles, especially emotion-directed and avoidance behaviors, were generally found to be closely associated with dieting, binge eating syndrome, and eating disorders.[26]

Physiological Mechanisms in Emotional Eating Behavior

The internal mechanisms underlying binge eating behavior in emotional situations have not been clarified yet. Physiological changes due to nutrients are believed to have an effect on mood. Some studies on this subject say that there is a positive change in mood after consuming foods with a high carbohydrate content. This effect is related to the secretion of serotonin from the brain after eating. Proteins are also suggested to have a positive effect on emotional state. This situation is related to the fact that the protein, which is found in limited amounts in food, increases the tryptophan level in the blood–brain

barrier and causes the secretion of serotonin in the human body. Another study reported that a high intake of tryptophan-derived hydrolyzed protein has a positive effect on mood in the case of acute stress.[17]

Risk Groups for Emotional Eating

Children and Adolescents

It is seen that uncontrolled overeating in adolescents and children is spreading rapidly. In addition to obesity being a great risk for this group, there is also the possibility of eating disorders such as anorexia nervosa. Children and adolescents are a group that deserves great attention since eating disorders are most commonly seen in adolescence.[27] Early detection of emotional eating in this group is very important for the prevention of eating disorders and obesity.[28] In a study on the subject, the relationship between parenting and emotional eating was examined and it was determined that the family had a significant effect on exhibiting emotional eating behavior. Literature studies reveal that the self-efficacy and emotional maturity of children with authoritarian parents are more developed compared to other children. Children of families with weak bonds with their children are also much more likely to display unhealthy eating behaviors, as they will experience more emotional distress.[29]

Obesity

It is known that emotional eating plays an active role in the etiology of obesity. It has been shown that the food consumed due to stress causes an increase in body weight. Obese individuals go to the method of suppressing the hunger they feel through foods in order to reduce emotional stress due to their previous experiences.[30] It is emphasized that teaching emotion regulation skills is important for the effective treatment of obesity in children.[31] In a study examining obese and normal individuals, the eating behaviors of individuals in response to anxiety-triggering emotions were compared, and as a result, the emotional eating scores of the obese individuals were found to be much higher than those of normal individuals.[32]

Other Problems

Negative emotions such as stress, depression, and anxiety can cause an increase or decrease in food consumption. Emotional eating can occur through various mechanisms such as eating to cope with negative emotions, psychological fluctuations accompanied by emotional changes, and confusing internal states of hunger and satiety.[33] Apart from the groups specified for emotional eating, individuals with eating disorders and those using weight loss treatments are also at risk for emotional eating. In addition, some studies have noted that individuals with binge eating disorder or bulimia nervosa are more alexithymic than normal individuals.[17]

Factors Associated with Emotional Eating

There are various ideas about the effect of emotions on eating behavior. For example, a study examining the extent to which negative emotional states are effective in overeating showed that negative emotions trigger food intake more than positive emotions do.[34]

Stress

Stress has both direct and indirect negative effects on health. There is some evidence that it adversely affects other physiological functional processes, with adverse cardiovascular function, suppressed immune response, and some contributing to cancer. Eating is one of the behaviors that are heavily affected by stress and impair health indirectly. Some individuals increase their food consumption when they feel stressed, which leads to obesity and related health problems.[34,35]

Stress also has an impact on what food people choose to eat. Studies on what kind of food people prefer when under stress have concluded that high-calorie desserts and fatty snacks are preferred more under stress.[35]

Depression

Another factor that triggers eating behavior is depression. Depressed individuals often engage in binge eating behavior as a way of regulating their negative mood.[36] Recent systematic reviews and meta-analyses have concluded that there is a positive association between depressive symptoms and obesity.[37] At the same time, it has been found that depressed individuals prefer foods with high energy content and their BMIs are higher than that of individuals with stable mood.[38]

Parent Modeling

Emotional eating behavior can be triggered through modeling. In particular, parents can seriously affect their children's eating behavior in food selection. The most important risk factor for childhood obesity is family obesity. The risk is particularly increased if both parents are obese. Some studies have determined that maternal obesity is more effective than paternal obesity, although it varies depending on prenatal and postnatal environmental and gender-oriented genetic mechanisms.[39]

Anger

As a result of some studies, it has been revealed that anger triggers various addictions. Anger can also cause behavioral reactions such as restlessness, drug use, and excessive food consumption. It is an important emotion that also affects the process and outcome of eating disorders

treatment. It has been revealed that emotions such as anger and fear increase impulsive eating and there is a tendency to high-calorie foods such as junk food to regulate the emotional state. Clinical studies have proven that anger is an important antecedent of binge eating.[40]

Boredom

Boredom is a major cause of eating disorders that many researchers and experts overlook. Although there are a few studies showing that adults tend to eat when they are distressed, detailed studies on this subject have not been conducted yet. In a study conducted with the participation of 139 students from different education levels, students' eating behaviors were investigated. It has been shown that students show more eating behavior when they are bored compared to other emotions.[41]

Happiness

The effects of positive emotions on food consumption are related to limiting and controlling food consumption. It has been determined that women with high eating control and who are encouraged to eat when they are happy show more controlled eating behavior. In a study conducted on individuals who can and cannot control their food intake, the link between positive emotions and eating behaviors of the individuals who consume chocolate snacks was investigated. Participants who were able to control their eating behaviors decreased their snack consumption as their positive emotions increased.[41,42]

Observation of less food intake as a result of positive emotions is seen in people who can control their food intake. In individuals who do not have strong eating control, positive emotions such as happiness may cause more food consumption.[42]

Eating Awareness

Definition of Eating Awareness

Eating awareness focuses not on what is eaten but on why and how the eating behavior occurs. It is defined as the eating behavior characterized by internalizing the homeostatic hunger and satiety signals, being aware of how positive or negative emotions affect the eating behavior, without being affected by the stimuli coming from the environment and focusing only on the food at the consumption stage, without making any judgments during the selection phase of the consumed food. By focusing on the eating behavior, accepting the food, and putting emotions into the background during food intake, the individual can more easily decide on the healthy option in food choices. Eating awareness reduces food cravings and helps to control weight.[43]

Eating awareness includes being aware of the triggers that affect the desire to eat, food choice, amount and shape of food intake. Those who eat consciously are aware of the effects of the foods they consume and their eating behaviors on the human body.[44]

Eating awareness improves health and prevents disease-related quality of life.[43]

Effect of Eating Awareness on Nutritional Status

Eating awareness aims not to show a reactive attitude towards foods and the emotions felt towards the foods consumed, and this situation minimizes the cases of remorse and self-restraint after eating. Some studies show that eating awareness can significantly reduce the behavior of eating in response to emotional states.[45]

Being able to perceive food with our senses can automatically increase food consumption. An individual with eating awareness, on the other hand, focuses on his/her own food in such a situation and excessive food consumption is prevented. Findings showed that eating awareness deautomates eating and reduces responses to food cravings, thus aiding in weight management.[46]

Eating awareness enables the individual to recognize the types of hunger and also prevents losing control over eating. It aims to realize food intake according to cellular hunger signals by being aware of cellular hunger rather than emotional hunger.[47]

Relationship Between Eating Awareness and Eating Behavior Disorders

Studies show that individuals with eating awareness have a more balanced and regular diet. At the same time, awareness of eating reduces stress, provides weight loss, and prevents the development of eating behavior disorders.[48] Eating awareness also reduces the difficulties experienced by individuals with problematic eating behavior in controlling their food intake.

A study with the participation of 318 adults stated that as the eating awareness scale score increases, the eating test attitude score decreases. As a result of the study, it was noted that having awareness of eating reduces the risk of eating behavior disorder.[49]

CONCLUSION

Intuitive eating has been demonstrated to be an alternative approach to medical treatments for body weight control. A significant difference was found between the emotional eating scales applied before and after the education of the groups in which eating awareness practices

were applied, and it was determined that as the eating awareness of the individuals increased, their emotional eating behaviors decreased. Likewise, increasing awareness of eating has also positively affected intuitive eating behavior and indirectly has a positive effect on body weight control.

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Kubilay Demirağ
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Macit Gülten
Mehmet Akif Topçuoğlu
Meltem Gülhan Halil
Meltem Koca
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Osman Abbasoğlu
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Pınar Taşar
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Seher Demirer
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Zarife Kuloğlu
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