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Original Article

Changes in anthropometric and metabolic parameters related to gastroesophageal reflux disease in non obese cases

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ABSTRACT

Aims: Central obesity associated with visceral adiposity is considered to increase reflux symptoms. This study aimed to investigate the effects of anthropometric and visceral fat-related measurements on reflux symptoms in non-obese patients with gastroesophageal reflux disease (GERD) and a body-mass index (BMI)<30 kg/m².

Methods: The study included 120 non-obese patients with dyspeptic complaints, BMI<30, and GERD diagnosed according to the Frequency Scale for the Symptoms of Gastroesophageal Reflux Disease (FSSG), and 50 non-obese controls with BMI<30 kg/m², no dyspeptic complaints, and no GERD according to the FSSG. Demographic data and extraesophageal symptoms were assessed using a questionnaire. Serum biochemical findings were recorded. Body composition and anthropometric measurements were evaluated using bioelectrical impedance analysis (TANITA).

Results: Significant differences were found between the GERD and healthy control groups in waist circumference, BMI, low-density lipoprotein cholesterol (LDL), body fat percentage, fat mass, total body water (TBW), metabolic age, visceral fat, obesity grade, reflux score, acid reflux score, and total FSSG score ($p<0.05$). Sore throat, apnea, and teeth grinding also differed significantly between the groups ($p<0.05$). In the patient group, acid reflux scores were positively correlated with BMI ($r=0.298$, $p=0.001$), LDL ($r=0.387$, $p<0.001$), and visceral fat ($r=0.180$, $p=0.049$), and negatively correlated with TBW ($r=-0.273$, $p<0.003$).

Conclusion: Reflux symptom frequency and severity are closely associated with body fat composition in non-obese as well as obese patients. Increased abdominal and visceral fat may increase GERD risk independently of obesity.

Keywords: Non-obese, GERD, FSSG, bioelectrical impedance

INTRODUCTION

Gastroesophageal reflux (GER) increasingly draws attention in recent years due to its prevalence, heterogeneity of its clinical presentation, morbidity and economic consequences. Considering multiple previous consensus definitions, gastroesophageal reflux disease (GERD) is defined as symptoms and complications resulted from retrograde flow of gastric contents into the esophagus, oral cavity (including larynx) and/or lungs.[1] Obesity is a potential risk factor for several chronic disorders including gastroesophageal reflux disease. Although various pathological mechanisms, including increased intragastric pressure, decreased lower esophageal sphincter (LES) pressure and esophageal acid exposure, can underly GERD, obesity has also been suggested to play an important role in the development of GERD.[2-3] Obesity can facilitate transient LES relaxation and cause GERD by increasing intraabdominal pressure and altering the structure and function of gastroesophageal junction.[4] Other possible obesity-linked mechanisms that may underlie GERD include increased levels of circulating inflammatory cytokines and delayed gastric motility.[5] Recently, visceral fat accumulation associated with abdominal obesity has been implicated in the development of gastrointestinal disorders as visceral fat accumulation increases intraabdominal pressure leading to the release of several bioactive substances known as adipocytokines such as TNFα, IL-6, resistin, leptin and adiponectin. Several mediators released by visceral adipose tissue, including adiponectin, leptin, TNFα, IL-6 impact the stomach and esophagogastric junction.[6] Proinflammatory cytokines IL-1 and TNFα have been reported to induce gastrin release from antral segments of the stomach.[7] Therefore, as with adipocytokines, proinflammatory mediators can enhance or perpetuate local inflammation at the gastroesophageal junction following local injury associated with esophageal acid exposure.[2]

In the assessment of obesity, body fat percentage, fat amount and fat distribution are far more important than total body weight alone. Bioelectrical impedance analysis is among

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the most efficient methods to estimate body fat ratio. This analytical method is based on electrical conductivity differences between fat-free tissue and fat tissue.[8-10]

Making an accurate diagnosis of GERD can be challenging in routine clinical practice. In clinical assessment, the self-assessment scale is an important part of diagnostic process. Japanese studies have demonstrated that FSSG might be beneficial for monitoring GERD in symptomatic patients.[11] A diagnosis of GERD is made in patients with a FSSG score of 8 or higher. FSSG is a diagnostic tool previously validated by public health and primary care physicians for use in Chinese populations to diagnose GERD.[12]

This study aims to demonstrate that the presence of insulin resistance (HOMA-IR>2.7) and alterations in anthropometric and metabolic parameters may be associated with an increased incidence of GERD in non-obese (BMI<30 kg/m²) populations. This study aims to assess potential associations between GERD and alterations in anthropometric and metabolic parameters in non-obese (BMI<30 kg/m²) populations.

METHODS

This study was conducted with the approval of the Clinical Researches Ethics Committee at Kırıkkale University (Date: 29.11.2016, Decision No: 23/14). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. Necessary permissions were obtained for the use of study materials, and it was essential that participants gave their free consent. This article is based on the internal medicine postgraduate dissertation no.466521.

This study included 120 patients who attended Gastroenterology Outpatient Clinics of the School of Medicine at Kırıkkale University for dyspeptic complaints and gave consent to take part in this study. Study participants were male and female patients aged between 18 and 65 years and had a BMI of<30 kg/m². 50 individuals who attended internal medicine outpatients clinics (general medicine, gastroenterology, endocrinology and rheumatology outpatients clinics) for reasons other than dyspepsia were included in this study as a control group. Laboratory data including fasting glucose, insulin, uric acid, TSH, ALT, HDL, LDL, triglyceride and total cholesterol levels were noted in addition to descriptive data such as age, gender, occupational status, smoking status and the presence of hypertension. HOMA-IR values were derived from fasting glucose and insulin levels (HOMA-IR=fasting glucose level (mg/dl) x fasting insulin level (uIU/ml)/405).

The F-scale FSSG (Frequency Scale for the Symptoms of Gastroesophageal Reflux Disease) was used in each participant to standardize the evaluation of gastroesophageal reflux disease. Each item in this scale was rated from 1 to 5 as never (1), rarely (2), occasionally (3), often (4) and sometimes (5) and items assessing dyspeptic symptoms (5 items) were excluded from the scale whereas participants responded to 7 questions assessing reflux symptoms. Participants who scored 8 points or more in the 7-item scale were included in the GERD group. Total scores were calculated as follows: reflux symptoms: (max:7x5=35 points) and acid reflux symptoms (dyspepsia) (max:5x5=25 points); Total score (max:7x5=35 points)+(max:5x5=25 points). An additional survey form was filled for extraesophageal symptoms (chronic cough, chest pain, hoarseness, sore throat, apnea, choking sensation, teeth grinding).

TANITA (bioelectrical impedance) was used for anthropometric assessments and height, weight, body-mass index (BMI), fat percentage, fat mass (in kg), fat-free mass (FFM), muscle mass (in kg), total body water (TBW), TBW percentage, bone mass weight (bone mineral mass), basal metabolic rate (BMR), metabolic age, visceral fat rating and obesity grade were noted. All measurements were performed using the same device. In addition, waist circumference was measured using a non-stretching measuring tape in a standing position and using the ribs and longest horizontal line that passes across both iliac crests as references.

People with a BMI of ≥30 kg/m², diabetes mellitus, thyroid dysfunction, chronic obstructive pulmonary disease, chronic kidney failure, coronary artery disease or liver cirrhosis, people who were on proton pump inhibitors, NSAIDs, anticoagulants, antiplatelet or any herbal medicine or people who had a history of abdominal surgery (cholecystectomy, gastric surgery, colon surgery, C-section, reflux surgery, hiatal hernia repair, etc.) were excluded from the study.

Statistical Analysis

The SPSS 20.0 (IBM Corporation, Armonk, New York, United States) and Medcalc 14 (Acacialaan 20, B-8400 Ostend, Belgium) statistical software packages were used in data analysis. Normality of data distribution and homogeneity of variances were assessed by the Shapiro-Wilk test and Levene's test, respectively. In comparisons of quantitative data between two independent groups, bootstrapping was used with Independent-Samples T test whereas Monte Carlo simulations were used with the Mann-Whitney U test. Variables were tested using the backward elimination method. In comparisons of categorical variables, Pearson's Chi-square and Fisher's Exact tests were used with Monte Carlo simulations and Exact test results. The odds ratio was used to identify the most important risk factor among significant risk factors. The logistic regression test was used with backward method to determine cause and effect associations between dichotomous or multinomial categorical response variable and explanatory variables. The sensitivity and specificity of the cutoff was analyzed in a receiver operating curve (ROC) analysis based on the associations between the classification determined by the cutoff value calculated based on patient and control groups' variables and true classification. Spearman's rho test was used to analyze correlations between variables. Quantitative variables were presented as mean±standard deviation and median (range, maximum- minimum) and categorical variables were presented as counts (n) and percentages (%). Variables were analyzed at 95% confidence level and p-values less than 0.05 were considered significant.

RESULTS

The study included a total of 170 participants with 44 male (25.9%) and 126 female (74.1%) and the mean age in the study population was 30.45±10.77 years. The mean age in the patient group (31.65±11.45 years) was comparable to that of the control group (30.22±7.27 years) (p=0.054). Significant intergroup differences were found in BMI (p=0.022) and waist circumference (p=0.001) whereas intergroup differences were not significant in height, weight, gender, occupational

status, smoking status, alcohol consumption, and in the rate of hypertensive participants (p>0.05) (Table 1).

Table 1. Sociodemographic data

	Controls (n=50) mean±SD	Patients (n=120) mean±SD	Total (n=170) mean±SD	p-value
Age (years)	30.22±7.27	31.65±11.45	30.45±10.77	0.054
Height (cm)	167.68±10.14	164.47±10.16	165.41±10.23	0.064
Weight (kg)	63.12±11.29	64.25±10.66	63.92±10.82	0.537
BMI (kg/m$^{2)}$	22.43±3.43	23.79±3.60	23.43±3.59	0.022
Waist circumference (cm)	74.63±5.62	79.94±10.60	78.38±9.71	0.001
	n (%)	n (%)	n (%)	
Gender				
Female	39 (78.0)	87 (72.5)	126 (74.1)	0.565
Male	11 (22.0)	33 (27.5)	44 (25.9)	
Occupational status				
Unemployed	38. (76.0)	87 (72.5)	125 (73.5)	0.706
Employed	12 (24.0)	33 (27.5)	45 (26.5)	
Smoking status				
Non-smoker	37 (74.0)	72 (60.0)	109 (64.1)	0.114
Smoker	13 (26.0)	48. (40.0)	61 (35.9)	
Alcohol consumption				
No	48 (96.0)	110 (91.7)	158 (92.9)	0.513
Yes	2 (4.0)	10 (8.3)	12 (7.1)	
Hypertension				
No	49 (98.0)	115 (95.8)	164 (96.5)	0.72
Yes	1 (2.0)	5 (4.2)	6 (3.5)	

Independent T test (bootstrapped)-Person's Chi-square test (exact)-Fisher exact test (exact). SD: Standard deviation, BMI: Body-mass index

LDL levels (p=0.035), fat (p=0.016), fat mass (p=0.012), metabolic age (p=0.002), visceral fat (p<0.001), obesity grades (p=0.015), reflux scores (p<0.001), acid reflux scores (p=0.020) and total scores were statistically significantly higher in patient group compared to control group whereas TBW (%) was statistically significantly lower in patient group compared to control group (p=0.018). There were no statistically significant intergroup differences in glucose, insulin, HDL, triglyceride, total cholesterol, uric acid, TSH, ALT levels and FFM, muscle mass, TBW, bone mass, BMR and HOMA-IR values (p>0.05) (Table 2).

In ROC analysis, out of the analyzed variables, total score displayed the highest performance in distinguishing patient and control groups with an AUC value of 0.928±0.019 (p<0.001), followed by visceral fat (AUC=0.673±0.045; p<0.001), metabolic age (AUC=0.652±0.044; p=0.001), waist circumference (AUC=0.631±0.042; p=0.002), fat mass (AUC=0.621±0.043; p=0.005), obesity grade (AUC=0.619±0.046; p=0.012), fat (AUC=0.617±0.043; p=0.008), TBW (%) (AUC=0.617±0.043; p=0.008), BMI (AUC=0.614±0.046; p=0.015) and LDL (AUC=0.602.±.0.045; p=0.025). The most appropriate cutoff values for these variables were determined as 79 cm, 22.8 kg/m², 107 mg/dl, 29 years, 10,3, 29,7, 19,4, 1, 25 and 50.7 respectively (Table 3).

The prevalence of sore throat, apnea and teeth grinding was significantly higher in the patient group compared to the control group (p=0.006, p=0.019 and p=0.029, respectively). Participants experiencing sore throat were 3.2 times more likely to be in the patient group (OR=3.2; 95% CI: 1.3-7.3). Likewise, participants experiencing teeth grinding were 3.3 times more likely to be in the patient group (OR=3.3; 95% CI: 1.1-10.1). Nevertheless, the prevalences of chronic cough, non-cardiac chest pain, hoarseness, bronchospasm and insulin resistance did not statistically significantly differ between the patient and control groups (p=0.557, p=0.255, p=0.528, p=0.069 and p=0.255, respectively). Multiple logistic regression analysis was performed using backward elimination method (backward stepwise [wald]) in a model including statistically significant variables for patients experiencing dyspepsia in this population aged 18 to 65 years. Variables identified as statistically significant by our logistic model included LDL, fat, total score and sore throat (p model<0.001) (Table 4).

Spearman's correlation analysis showed moderate, significant negative correlations between reflux scores and total scores and height in the control group (r=-0.365; p=0.009). Significant positive correlations were found between reflux scores and total scores and triglyceride (r=0.305; p=0.031), total cholesterol (r=0.354; p=0.012) and LDL (r=0.281; p=0.048) levels and significant negative correlations were found between reflux scores and total scores and TSH levels (r=-0.347; p=0.014). Other biochemical or body composition parameters did not significantly correlate with reflux scores (p>0.05). In the patient group, significant negative correlations were found between the height and reflux scores (r=-0.197; p=0.031), acid reflux scores (r=-0.252; p=0.006) and total scores (r=-0.261; p=0.004). Significant positive correlations were found between total cholesterol levels and reflux scores (r=0.295; p=0.001), acid reflux scores (r=0.293; p=0.001) and total scores (r=0.333; p<0.001) and between LDL levels and reflux scores (r=0.378; p<0.001), acid reflux scores (r=0.387; p<0.001) and total scores (r=0.427; p<0.001). ALT levels positively correlated with reflux scores alone (r=0.190; p=0.038). In anthropometric assessments, positive correlations were found between acid reflux scores and total scores and waist circumference (r=0.244-0.275), BMI (r=0.269-0.298), fat percentage (r=0.217-0.262), fat mass (r=0.188-0.241), metabolic age (r=0.232-0.255), obesity grade (r=0.269-0.278) and visceral fat; significant negative correlations were found between TBW percentage and acid reflux scores (r=-0.273; p=0.003) and total scores (r=-0.232; p=0.011).

DISCUSSION

The prevalence of obesity is increasing worldwide because of inadequate physical activity and overeating. The surge in the number of people living with obesity gave rise to an increase in the prevalence of metabolic syndrome. In addition to established associations between metabolic syndrome and several disorders such as ischemic heart disease and diabetes mellitus, numerous studies have also demonstrated associations between metabolic syndrome and gastrointestinal disorders.[2] Although several studies have reported conflicting results, many studies conducted in Japan and other developed countries have demonstrated strong associations between GERD symptoms and obesity and high BMI.[3,13] Nevertheless, associations between high BMI and obesity and reflux symptoms should be further discussed due to the conflicted results mentioned above.

Table 2. Intergroup comparisons of biochemical data

	Controls (n=50) median (min-max)	Patients (n=120) median (min-max)	Total (n=170) median (min-max)	p-value
Glucose	90.50 (77-165)	92 (53-165)	91.50 (53-165)	0.508
Insulin	8.05 (1.90-93)	8.35 (1.32-108)	8.20 (1.32-108)	0.553
HDL	51.50 (3.10-99)	47.50 (3.10-90)	48.50 (310-99)	0.066
LDL	74.50 (42-129)	87 (11-243)	80 (1-243)	0.035
Triglycerides	81 (33-350)	97 (28-404)	95 (28-404)	0.184
Total cholesterol	146.50 (73-222)	161 (19-310)	159 (19-310)	0.216
Uric acid	4.05 (1.20-41)	4.30 (1.20-59)	4.20 (1.20-59)	0.471
TSH	1.68 (0.07-7.56)	1.54 (0.02-10)	1.58 (0.02-10)	0.476
ALT	12.50 (5-39)	13 (5-65)	13 (5-65)	0.213
Fat	19.30 (3-41.80)	24.75 (3-95.20)	22.55 (3-95.20)	0.016
Fat mass	12.80 (1.10-36.20)	15.15 (1.10-43.40)	14.20 (1.10-43.40)	0.012
FFM	45.95 (19-75.10)	46.05 (36.80-74.20)	45.95 (19-75.10)	0.613
Muscle mass	43.95 (35-70.50)	43.90 (34.90-516)	43.90 (34.90-516)	0.520
TBW	32.70 (25.10-52.60)	32.50 (25.10-52.60)	32.55 (25.10-52.60)	0.341
TBW percentage	55.80 (41.60-80.90)	52.50 (41.60-80.90)	53.80 (41.60-80.90)	0.018
Bone mass	2.40 (1.90-3.70)	2.40 (1.90-4)	32.55 (25.10-52.60)	0.442
BMR	5.858 (55.94-9.138)	5.851.50 (55.94-8.996)	5.858 (55.94-9.138)	0.586
Metabolic age	16 (12-64)	27 (12-66)	22 (12-66)	0.002
Visceral fat	1 (1-12)	3 (1-10)	3 81-12)	<0.001
Obesity grade	0.70 (-29.90-35.90)	10.95 (-29.90-35.90)	8.80 (-29.90-35.90)	0.015
HOMA-IR (mg/dl)	1.90 (0.42-19.06)	1.84 (0.27-22.93)	1.88 (0.27-22.93)	0.495
Acid reflux scores	13.50 (5-23)	15 (8-23)	14 (5-23)	0.020
Reflux scores	7 (7-7)	19 (9-35)	15 (7-35)	<0.001
Total scores	20.50 (12-30)	33 (17-53)	29 (12-53)	<0.001

Mann Whitney U Test (Monte Carlo). Min: Minimum, Max: Maximum, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, TSH: Thyroid-stimulating hormone, ALT: Alanine aminotransferase, FFM: Fat-free mass, TBW: Total body water, BMR: Basal metabolic rate, HOMA-IR: Homeostatic model assessment of insulin resistance

Table 3. ROC analysis of variables that showed significant differences between patient and control groups

	Control group (n=50)			Patient group (n=120)		AUC+SE	p-value
	Cutoff	n	%	n	%		
Waist circumference	≤79	43	86.0%	70	58.3%	0.631±0.042	0.002
	>79	7	14.0%	50	41.7%		
BMI	≤22.8	32	64%	48	40.0%	0.614±0.046	0.015
	>22.8	18	36.0%	72	60.0%		
LDL	≤107	44	88.0%	82	68.9%	0.602±0.045	0.025
	>107	6	12.0%	37	31.1%		
Metabolic age	≤29	41	83.7%	66	55.0%	0.652±0.044	0.001
	>29	8	16.3%	54	45.0%		
Obesity grade	≤10.3	36	73.5%	57	47.5%	0.619±0.46	0.012
	>10.3	13	26.5%	63	52.5%		
Fat	≤29.7	46	92.0%	77	64.2%	0.617±0.043	0.008
	>29.7	4	8.0%	43	35.8%		
Fat mass	≤19.4	46	92.0%	80	66.7%	0.621±0.043	0.005
	>19.4	4	8.0%	40	33.3%		
Visceral fat	≤1	31	63.3%	38	31.7%	0.673±0.045	<0.001
	>1	18	36.7%	82	68.3%		
Total score	≤25	49	98.0%	39	32.5%	0.928±0.019	<0.001
	>25	1	2.0%	81	------		
TBW percentage	>50.7	43	86.0%	69	57.5%	0.617±0.043	0.008
	≤50.7	7	14.0%	51	42.5%		

ROC analysis [Hanley & McNell-Youden Index (J)] ROC: Receiver operating characteristic, AUC: Area under the curve; SE: Standard error, BMI: Body-mass index, LDL: Low-density lipoprotein, TBW: Total body water

Table 4. Multiple logistic regression analysis of study data

					%95 CI for OR	
	B	SE	p-value	OR	Lower limit	Upper limit
LDL	2.318	0.916	0.011	10.160	1.690	61.220
Fat (>29.7)	-2.201	0.902	0.015	9.030	1.540	52.940
Total score (>25)	-5.838	1.200	<0.001	342.970	32.630	3.605.150
Sore throat (+)	-1.485	0.722	0.040	4.410	1.070	18.170

Dependent variable: Groups, predicted (patient)=88.2, predicted (control)=86.3, predicted =86.3, p model <0.001

Multiple logistic regression-method: backward stepwise (Wald). CI: Confidence interval, OR: Odds ratio, B: Regression coefficients, SE: Standard error, LDL: Low-density lipoprotein

Based on available evidence, waist circumference, which is used to assess central obesity, is associated with esophageal acid exposure as much as BMI does. In a case-control study that included 1.679 patients, a multifactorial analysis demonstrated that metabolic syndrome was an independent risk factor for GERD.[14] In a cross-sectional study in one hundred patients, Kallel et al.[15] demonstrated that metabolic syndrome components, abnormal waist circumference and elevated fasting blood glucose levels were independent risk factors for GERD using 24-hour pH monitoring. Although several studies in patients with obesity have demonstrated associations between metabolic syndrome and GERD, these studies have failed to demonstrate a direct association between BMI and GERD, supporting the hypothesis that GERD is associated with central obesity rather than BMI.[16] In a study conducted in 2008 with 475 patients, Danjo et al.[17] reported that FSSG scores could reflect the severity of endoscopic findings in GERD and FSSG could be used to monitor clinical course of GERD and assess treatment efficacy. In a study conducted in 2015, Fujiwara et al.[18] compared several variables including height, weight, BMI, body fat ratio, visceral fat, waist circumference, triglyceride, ALT, AST/ALT, HOMA-IR and FSSG scores (reflux scores, acid reflux scores, total scores) in 50 patients with NAFLD to those of a control group of 228 healthy participants and FSSG was found to be associated with BMI. They reported that FSSG scores were higher in participants with a BMI of >30 kg/m^2. Statistically significant intergroup difference was found in FSSG scores and the sensitivity and specificity of FSSG scores were determined as 81.7% and 90%, respectively. These findings suggest that our findings are valuable for explaining associations between intergroup differences and gastroesophageal reflux. In another study conducted in 2012 in 96 patients with NAFLD and 139 controls to investigate the prevalence of GERD symptoms and potential risk factors for GERD in patients with NAFLD, Fujikawa et al.[19] found that total scores, reflux scores and acid reflux scores were higher in patients with NAFLD compared to healthy controls. In 2009, Danjo et al.[17] conducted a study to compare GERD symptoms scales FSSG and QUEST to each other and found that reflux scores were higher in the GERD group compared to functional dyspepsia, stomach ulcer and duodenal ulcer group. In our study, total reflux scores were higher in the GERD group compared to the healthy control group. Our results demonstrate that FSSG is a reliable diagnostic tool for gastroesophageal reflux disease. The prevalence of GERD was found to be higher in overweight participants and in those with obesity compared to non-obese participants in a study conducted by Icitovic et al.[5] in 19.819 participants, in 2016. Our study was conducted in non-obese participants with a BMI less than 30 kg/m^2. BMI values statistically significantly differed between the subgroup of participants with higher reflux symptom scores and the control group. In the patient group, a significant positive correlation was found between acid reflux scores and BMI (r=0.298; p=0.001). In our study group, any increases in BMI, even the increases with the normal range, were found to be associated with increased reflux symptom scores, independently from obesity. Our findings support literature reports from previous studies and indicate that BMI can significantly contribute to the increases in reflux symptom scores. In 2007, a study conducted by Corley et al.[20] in 316 patients with GERD and 317 controls demonstrated associations between waist circumference and GERD symptoms and reported that abdominal obesity might contribute to GERD. In 2007, Corley et al.[21] investigated potential associations between GERD symptoms and waist circumference and BMI by ethnicity in a cross-sectional study conducted in 80.110 persons in the US and increased abdominal diameter was identified as an independent risk factor for gastroesophageal reflux symptoms. As for waist circumference, we found a statistically significant difference between the subgroup of patients with higher reflux scores and the control group. In the patient group, a significant positive correlation was found between acid reflux scores and waist circumference (r=0.275; p=0.002). Likewise, a significant positive correlation was found between total scores and waist circumference in the patient group (r=0.244; p=0.007). Previous studies have demonstrated that intraabdominal pressure driven by central obesity stimulates proinflammatory mediators and adipocytokines and these are important factors in the pathophysiology of GERD. In our study a positive correlation was found between waist circumference and reflux scores. Our study provides further evidence on the assumption that any increase in waist circumference may be an important factor in the development of reflux symptoms, even in non-obese people. Positive correlations between serum lipids levels and NEGERD were demonstrated by Matsuzaki et al.[22] in a study conducted in Japan, in 2010. Associations were found between the severity of gastroesophageal reflux symptoms and serum triglyceride levels in men and LDL levels in female. These findings indicate that the severity of GERD increases with the severity of dyslipidemia. In a study conducted in 2012, Fujikawa et al.[19] found that serum total cholesterol and triglyceride levels were risk factors for GERD symptoms in Japanese patients with NAFLD. Our study has supported these study reports. Serum LDL levels were found to be higher in the GERD group compared to the healthy control group in our study. A significant positive correlation was found between acid reflux scores and LDL levels in the patient group (r=0.387; p<0.001). There are literature reports indicating that inflammatory cytokines might be increased in conditions associated with increased levels of dlDL and other lipid parameters.[23] Proinflammatory mediators such as TNFα and IL-6 stimulated by increased levels of LDL can increase acid production by dilating LES and promoting gastrin secretion.[7] Significant associations between LDL levels and reflux scores in our study provided further evidence for these pathophysiological mechanisms. In a study conducted in 2015, Abdelkader et al.[24] compared overweight participants

and participants with obesity to normal-weight participants using a formula that included intraabdominal, subcutaneous abdominal and total body fat tissue areas by BMI. All three parameters mentioned above were found to be higher in the overweigh and obesity groups compared to the normal-weight group.[21] In another multicenter study conducted in 2015, Matsuzaki et al.[25] demonstrated that visceral fat tissue area might be an independent risk factor for reflux esophagitis. In 2017, Kutlu et al.[26] conducted a study aimed at assessing body compositions in our country and compared anthropometric measures in participants with obesity to those in non-obese participants. Fasting blood glucose, LDL, triglyceride, total cholesterol, visceral fat, BMI, waist circumference, BMR, FFM, bone mass, muscle mass measurements were found to be higher in the obesity group compared to the control group. A negative correlation was found between BMI and TBW levels. A direct link was found between BMI and visceral adiposity and fat and visceral fat accumulation and fat ratio were found to be increased as BMI increased.[7,26] In a study conducted in 2013, Peppa et al.[27] found a positive correlation body fat percentage and BMI whereas no significant differences were found between healthy and patient phenotypes in peripheral fat distribution and fat-free body mass (FFM) indices. Martin et al.[28] demonstrated that laparoscopic repair of gastrogastric fistula (GGF) following Roux-en-Y gastric bypass (RYGB) was associated with significant weight loss, resolution of reflux symptoms, and metabolic improvements, even after attempts at medical or endoscopic management. In a study by Salminen et al.,[29] sustained weight loss was observed at 10 years following laparoscopic Roux-en-Y gastric bypass (LRYGB) and laparoscopic sleeve gastrectomy (LSG), and the cumulative incidence of Barrett's esophagus (BE) was found to be substantially lower than that reported in previous studies. Patients with non-severe GERD generally respond well to lifestyle modifications and optimized pharmacological therapy, whereas patients with severe GERD often require long-term anti-reflux management. In patients with persistent symptoms despite these measures, a precision approach to treatment escalation is recommended. This approach should be guided by factors including the integrity of the anti-reflux barrier, the presence of visceral hypersensitivity, confirmation of PPI-refractory GERD, symptom profile, BMI, and esophageal and gastric motor function.[30] Our study was the first study assessing GERD and anthropometric values analyzed using bioelectrical impedance. In our study fat, fat mass, visceral fat, metabolic age and obesity grades were higher in the GERD group compared to the healthy control group. Furthermore, TBW percentage was lower in the GERD group compared to the healthy control group.

Studies available in literature have found associations between gastroesophageal reflux and increased body fat area, subcutaneous fat tissue thickness, abdominal fat tissue area although these results were obtained using conventional methods. In our study, statistically significantly higher levels of fat, fat mass, visceral fat, metabolic age, obesity grades measured using bioelectrical impedance and positive correlations in the subgroup of patients with higher reflux scores demonstrated that a tendency to gastroesophageal reflux might occur as body fat ratio increased in non-obese study participants. In our study, a significant negative correlation was found between TBW percentage and acid reflux scores in the patient group (r=-0.273; p=0.003). In the patient group, a significant negative correlation was also observed between TBW percentage and total scores (r=-0.232; p=0.011). TBW ratio decreased with weight gain and increased body fat ratios and their negative correlations with reflux scores and significantly lower TBW ratio in the subgroup of patients with higher reflux scores further supported other results of our study. In this study, we did not find any statistically significant intergroup differences in FFM, muscle mass, bone mass and BMR. Considering that our patient group consisted of non-obese participants and ages were matched, this was an expected result. In our study, there were no significant differences between the patient group and control group in glucose, insulin, uric acid, TSH, ALT, Homa-IR (mg/dl) levels. Lack of significant differences between the patient group and control group in biochemical parameters could be explained by the study population constituted of non-obese participants with BMI values of 29 kg/m² or lower. Being the first study using bioelectrical impedance to assess potential associations between GERD and anthropometric values was an important aspect of our study. GERD diagnosis was based solely on the FSSG scale, which evaluates patients' symptoms, and objective diagnostic methods such as upper gastrointestinal endoscopy or ambulatory pH monitoring were not routinely used. Therefore, the possibility of symptom-based misclassification cannot be completely excluded. The limitations of this study included the lack of biomarkers of adipose tissue inflammation, the absence of BMI categorization according to obesity grades, and the reliance on the FSSG scale alone for the diagnosis of GERD.

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CONCLUSION

This study suggests that visceral fat accumulation and a higher body fat ratio may be associated with gastroesophageal reflux symptoms, even in non-obese individuals. This study also identifies an association between GERD and fat tissue and body fat composition, which are implicated in and investigated in the pathophysiology of several diseases. Further studies with larger sample sizes are needed to clarify the potential associations between GERD symptoms and adipocytokines related to visceral fat accumulation, as well as to assess the relationship between GERD and BMI categorized according to obesity grade.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was conducted with the approval of the Clinical Researches Ethics Committee at Kırıkkale University (Date: 29.11.2016, Decision No: 23/14).

Informed Consent

Written informed consent was obtained from all individual participants prior to their inclusion in the study. Participants were fully informed about the study's aims, procedures, potential risks and benefits, and their rights-including the right to withdraw at any time without consequence. All

participants voluntarily signed a written informed consent form.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

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Author Contributions

Concept: ŞY; Design: ŞY; Control: ŞY, ÖGU; Data Collection and/or Processing: ŞY; Analysis and/or Interpretation: ŞY, ÖGU, DO; Literature Review: ŞY, BE; Article Writing: ŞY; Critical Review: All Authors.

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