The Effects of a High-Fat, Low-Carbohydrate Diet on the Prognosis of Patients with an Acute Attack of Chronic Obstructive Pulmonary Disease

Akut Atak Geçiren Kronik Obstrüktif Akciğer Hastalığı (KOAH) Hastalarında Yüksek Yağ, Düşük Karbonhidrat İçeren Bir Diyetin Prognoza Etkileri

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ABSTRACT Objective: Acute exacerbations are the most frequent cause of medical visits, hospitalizations, admissions and death among patients with chronic obstructive pulmonary disease (COPD). Our objective was to investigate the effects of a high-fat low-carbohydrate (CHO) diet on the prognosis of patients hospitalized for an acute exacerbation of COPD. **Material and Methods:** Thirty male patients with COPD, hospitalized for acute exacerbation were randomized into two groups. Standard hospital diet was administered to group I for 10 days and an experimental diet comprising of 50% standard hospital diet and 50% of a special enteral product containing high-fat low-CHO was administered to group II. Pulmonary function tests, anthropometric measurements and blood gas analysis were performed on the first and tenth days of the study. All patients completed a questionnaire regarding socio-economic status and nutritional habits. **Results:** Pulmonary function tests revealed statistically significant differences in tidal volume (TV) and forced vital capacity (FVC) between the measurements of day 1 and day 10 of the two groups with better results in group II ($p< 0.05$). There was no significant difference between groups on blood gas analysis parameters on days 1, 3, 5, 7, and 9 ($p> 0.05$), yet there was an improvement (increase in PaO2, decrease in PaCO2). **Conclusion:** A high-fat low-CHO diet can improve the prognosis of COPD patients with acute exacerbation. Since nutritional interventions can improve treatment outcomes and quality of life in COPD patients, multi-center studies with a larger sample size and long-term nutrition support are needed.

Key Words: Pulmonary disease, chronic obstructive; diet therapy

ÖZET Amaç: Kronik obstrüktif akciğer hastalığı (KOAH) olan hastalarda görülen akut ataklar, hastaların sıklıkla hastaneyi ziyaretine, hastaneye yatışına ve ölümlere neden olmaktadır. Bu çalışmada, akut atak nedeni ile gelen KOAH hastalarında yüksek yağ, düşük karbonhidrat (KHO) içeren bir diyetin prognoza olan etkilerinin araştırılması amaçlanmıştır. **Gereç ve Yöntemler:** Akut atak nedeni ile gelen 30 KOAH'lı erkek hasta randomize iki gruba ayrıldı. On gün süreyle 1. grup hastane yemeği ile 2. grup ise %50 hastane yemeği ve %50 yüksek yağ ve düşük KHO içeren enteral bir ürün ile beslendi. Hastaların 1. ve 10. günlerde kan gazları, akciğer fonksiyon testleri ve antropometrik ölçümleri değerlendirildi. **Bulgular:** Akciğer fonksiyon testlerinde [tidal volüm (TV) ve zorlu vital kapasite (FVC)] 2. grubun 1. ve 10. gün ölçüm değerleri arasında anlamlı fark bulundu ($p< 0.05$). Grup 2'de 1, 3, 5, 7 ve 9. günlerde kan gazı ölçümlerinde anlamlı olmayan, fakat (PaO_2 değerinde artış, $PaCO_2$ değerinde düşme) olumlu yönde farklılıklar saptandı ($p> 0.05$). **Sonuç:** Yüksek yağ ve düşük KHO içeren diyetin akut atak geçirmekte olan KOAH hastalarının prognozunun seyrinde olumlu etkileri olabilir. Daha fazla sayıda hasta ve daha uzun süreli beslenme desteği ile planlanacak çalışmalara ihtiyaç vardır.

Anahtar Kelimeler: Kronik obstrüktif akciğer hastalığı; diyet tedavisi

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Chronic Obstructive Pulmonary Disease (COPD) is one of the major causes of mortality and morbidity worldwide and the disease burden is expected to increase in the next 20 years.[1-3] There is a

reciprocal and interactive relationship between pulmonary functions and the nutritional status of the patient. Nutritional status may influence pulmonary functions and may be a factor leading to respiratory insufficiency or deteriorating it, whereas pulmonary disease may itself result in nutritional deficiency. Wheight loss has been shown in COPD patients and weight gain can be achieved by an appropriate nutritional support.[4-6]

The ratio of produced CO_2 to consumed O_2 gives the respiratory quotient (RQ) value ($RQ= VCO_2/VO_2$). Maximum amount of CO_2 is produced after CHO, whereas minimum amount of CO_2 is produced after fat metabolism. In healthy individuals, increases in metabolic rate and the amount of produced CO_2 are compensated by increased alveolar ventilation, therefore arterial CO_2 pressure is maintained within normal limits. However, a diet rich in CHO may lead to problems in patients with a limited respiratory reserve. Studies have shown that a diet with high CHO increases respiratory failure, whereas a diet with high fat and low CHO may decrease the duration of mechanical ventilation in acute severe exacerbations of COPD.[7]

Although a diet high in CHO and energy, low in fat and protein content results in hypercapnia and respiratory failure, excess protein may also be harmful due to the limited ability of these patients to respond to increased protein metabolism. In addition, by increasing CO_2 sensitivity and consequently minute ventilation and metabolic rate, excess protein in the diet of COPD patients causes dyspnea. Thus, energy requirements and distribution of fat, CHO and protein in the diet of patients with COPD must be designed carefully.[4,8]

There is a limited number of studies on the effect of different types of diet in patients with acute exacerbation of COPD, on pulmonary function tests and blood gas results.[4,8] This study was planned to investigate the effect of a diet containing high fat and low CHO on patients hospitalized with acute exacerbation of COPD.

MATERIAL AND METHODS

Following the local Ethics Committee approval, the study was performed prospectively with 30 male patients (aged 30-70 years) hospitalized with acute exacerbation of COPD in acute exacerbation and without any other chronic diseases in the Ondokuz Mayıs University, Faculty of Medicine, Pulmonology Department.

COPD was diagnosed, according to the criteria of the American Thoracic Society.[9] Acute exacerbation was defined by the presence of an increase, in at least two of the three following symptoms: dyspnea, cough and sputum production. Admission to the hospital was considered necessary based on the clinical status of the patient or the presence of accompanying factors like respiratory failure. After obtaining informed consent from patients, study group I was matched by age, body mass index (BMI) and physical activity with the control group II.

Basal metabolic rate (BMR) was calculated by the Harris-Benedict equation. Calculating activities and stress factor, daily energy requirement was estimated as BMR x 1.2.[10] Group I was administered standard hospital diet, whereas Group II was supplied half of their energy (50%) by hospital diet (same as group I) and half (50%) from a specific enteral product (pulmocare and hospital diet composed of 50% fat and 28% CHO). Pulmocare contains 1.5 kcal/mL energy, 62.5 g/L protein, 55.5% fat and 28% CHO. The standard hospital diet for patients with COPD included 57% carbohydrates, 30% fat, 16% protein and provided 1800 kcal/day energy. Patients were followed daily, whether they consumed the given diet or not and they ate 97% of the given food.

Pulmonary function tests, anthropometric measurements [height, body weight, triceps skinfold thickness (TSFT), waist circumference, hip circumference and mid-upper arm circumference], biochemical measurements (albumin, total protein) and pulmonary function tests [forced expiratory volume (FEV_1), forced vital capacity (FVC), tidal volume (TV) and vital capacity (VC)] were performed on the 1st and 10th days of the study. Arterial blood

gas analyses were obtained every other day throughout the study. Food composition table was used to calculate the dietary content (energy, protein, fat and other nutrients) of these patients.[11] World Health Organization (WHO) and National Center for Health Statistics (NCHS) standards were used to evaluate the antropometric measurements (TSFT, mid-upper arm circumference).[12] Nutritional status was assessed from anthropometric and biochemical data. All anthropometric measurements were performed by a single observer (G.Tümer). Weight was measured while patients were in light clothes in the morning, before eating or drinking anything and barefoot, with a portable personal scale, minimum load (NAN-mod 1985, minimum load 2 kg, maximum load 200 kg) to the nearest 1 kg. Height was measured with a portable stadiometer (NAN-mod1985) to the nearest 0.1 cm, with a telescopic measuring rod, range: 820-2000 mm. Waist circumference was measured by using a non- elastic tape, from the midpoint between the 12^{th} rib and the crista iliaca. Hip circumference was measured by specifying the highest lateral part of the standing patient. Mid- upper arm circumference and triceps skinfold thicknes was measured by using a Lange Skinfold Caliper®. Left arm was kept at a 90-degree angle and the measurement was made from the midpoint between acromion and olecranon. Biochemical analysis was performed by Hitachi Modular autoanalyzer via spectrophotometric method. Spiro Analyzer ST -250 Fukuda Sangyo and MIR devices were used for spirometric pulmonary function tests. Blood gas analysis was performed by AVL COMPACT Blood Gas Analyzer, using glavenometric and polarographic methods.

STATISTICAL ANALYSIS

SPSS 10.0 for Windows Software was used for statistical analyses. Anthropometric measurements, biochemical tests and pulmonary function tests of both groups, obtained on days 1 and 10, were tested by Wilcoxon Paired Two Samples Test. Nonparametric counterpart of obtained significance test of the difference between two pairs. The significance test between two parametric mean and Mann-Whitney U tests were used to compare the mean energy, CHO, protein, fat and other nutrient intakes. The difference between biochemical and pulmonary function tests between groups were compared with Mann-Whitney U Tests. One-way analysis of variance (ANOVA) was used to evaluate the blood gas changes of groups within time. The difference between biochemial and antropometric measurements, and pulmonary function tests between groups, were compared with Spearman's Rank Correlation Tests.

RESULTS

The demographic characteristics of the patients were shown in Table 1. The groups were similar regarding age, weight, height and BMI ($p > 0.05$). In both groups, 60% of patients had lost weight within the previous year ($p > 0.05$) (Table 1).

Table 2 shows the mean energy and nutrient intakes of patients for 10 days. There was no significant difference between groups in mean energy, protein and vitamin A intake ($p > 0.05$). CHO consumption was significantly lower in group II than in group I, whereas fat, phosphorus, iron, vitamin C, thiamin, riboflavin, niacin, copper, magnesium, zinc and calcium intakes in group II were significantly higher than in group I ($p < 0.05$). Considering respiratory function tests (RFT), there were significant increases in FVC and TV in group II between the first and tenth days ($p < 0.05$). However, there was no significant difference between FEV and VC ($p > 0.05$) (Table 3). No significant difference in blood gas parameters was observed between the groups on days 1, 3, 5, 7, 9 ($p > 0.05$) (Table 4).

Table 5 shows the correlation of forced expiratory volume (FEV_1) values, with several variables such as smoking habits, blood albumin levels, selected nutrient intakes and antropometric measurements. In group I, there was a negative correlation between FEV_1 values and the mean CHO intake on day 1 and a positive correlation between FEV_1 values and mean fat intakes on day 10 ($p < 0.05$). There was no significant correlation between FEV_1 and other nutrient intakes in group II ($p > 0.05$). There was a significant ($p < 0.05$) positive correlation between BMI on day 1 and FEV_1 on day

TABLE 1: Demographic characteristics of the patients.

	Group I (n= 15)	Group II (n= 15)	p
Age (years)	63.6 ± 4.3 (56.00-70.00)	60.9 ± 7.0 (47.00-70.00)	0.209
Height (cm)	167.0 ± 6.5 (157.00-178.00)	165.7± 5.5 (157.00-177.00)	0.548
Weight (kg)	71.5 ± 15.0 (45.00-96.00)	75.5 ± 15.4 (48.00-96.00)	0.474
BMI (kg/m^2)	25.6 ± 4.8 (18.02-33.61)	27.4 ± 5.1 (19.47-37.03)	0.319
Waist circ. (cm)	96.0 ± 15.7 (78.50-128.00)	103.0 ± 11.8 (85.10-128.00)	0.183
Hip circ. (cm)	94.7 ± 6.1 (84.20-107.50)	96.9 ± 7.3 (84.00-106.00)	0.371
Waist/hip ratio	1.0 ± 0.1 (0.80-1.20)	1.1 ± 0.1 (0.90-1.20)	0.207
Mid-upper arm circ. (cm)	25.3 ± 4.0 (18.60-33.10)	26.8 ± 2.7 26.8 ± 2.8	0.233
TSFT (mm)	13.1 ± 6.3 (5.50-29.00)	16.7 ± 8.0 (5.20-29.00)	0.178
Weight loss* (kg)	4.4 ± 4.5 (0.00-12.00)	4.0 ± 4.0 (0.00-10.00)	0.799
Smoking (package/year)	52.3 ± 27.1 (7.50-108.00)	39.1 ± 23.6 (20.00-112.50)	0.166

*: Weight loss in the previous year, BMI: Body mass index, TSFT: Ttriceps skinfold thickness, .

Values were given as mean ± SD (minimum- maximum); p< 0.05.

10 in group II (p< 0.05), whereas there was a negative but an insignificant association between the waist/hip ratio on day 1 and day 10 and FEV_1 on day 1 and day 10 (p> 0.05)

DISCUSSION

In the current study, we evaluated the effects of a high-fat and low-CHO diet on blood gases, RFTs, anthropometric measurements and biochemical variables in hospitalized COPD patients with acute exacerbation.

Malnutrition is observed in nearly 25-30% of moderate to severe COPD cases. Both fat and lean mass (muscle) decrease and loss is attributed to the decrease in protein synthesis.[7,13] In the present study, mean weight loss in the previous year was 4.4% in group I and 4% in group II, respectively. Decreasing BMI in patients with COPD is one of the causes of disease progression, decrease in lung functions and increased mortality in moderately hypoxic COPD patients.[14] Respiratory difficulty while food intake, loss of appetite, dyspnea, bloating and malabsorption due to hypoxemia can be mentioned as the causes of malnutrition in COPD patients. Normally, 36-72 kcal is required for respiration in healthy people, but it is about 430-420 kcal in patients with COPD.[15]

In 44 COPD patients with weight loss, the average expiration rates and lung diffusion capacities had decreased during the 5-week study. On the other hand, there was no change in the maximum expiration rate and there was a decrease only in lung diffusion capacity in 32 patients without weight

TABLE 2: Average daily consumption of selected energy and nutrients.

Enery and Nutrient	Group I (n=15)	Group II (n=15)	p
Energy (kcal)	1723.3 ± 297.6 (1315.2- 2289.7)	1883.4 ± 278.2 (1492.9- 2424.6)	0.139
Carbohydrate (g)	241.4 ± 40.5 (192.6-334.8)	181.4 ± 36.9 (136.1- 278.1)	0.001*
Carbohydrate (%)	56.4 ± 3.9 (48.2-61.6)	38.9 ± 2.9 (33.3 - 46.7)	0.001*
Fat (g)	49.9 ± 10.9 (33.7-65.9)	89.0 ± 11.2 (74.1- 107.9)	0.001*
Fat %	25.9 ± 3.2 (22.0-32.5)	42.8 ± 2.7 (37.6- 47.9)	0.001*
Protein (g)	76.8 ± 18.3 (54.6-120.2)	83.5 ± 10.6 (72.3-104.6)	0.231
Protein%	17.8 ± 1.5 (15.7-19.7)	18.3 ± 1.0 (15.7-19.6)	0.251
Phosphorus (mg)	513.3 ± 12.0 (281.0-700.0)	2671.7 ± 932.0 (1500.0-4750.0)	0.001*
Calcium (mg)	769.2 ± 229.4 (354.6-1153.2)	1290.9 ± 90.7 (1135.4-1390.5)	0.001*
Iron (mg)	9.1 ± 1.2 (7.3-10.8)	17.3 ± 2.8 (14.2-22.7)	0.001*
Vitamin A (IU)	3055.8 ± 1639.5 (1357.6-6477.3)	3429.4 ± 1247.3 (2293.6-7259.0)	0.488
Thiamin (mg)	0.98± 0.3 (0.7- 1.8)	2.1 ± 0.3 (1.6- 2.6)	0.001*
Riboflavin (mg)	1.6 ± 0.3 (0.9-2.1)	3.0 ± 0.3 (2.5- 3.6)	0.001*
Niacin (mg)	12.8 ± 3.2 (8.1- 20.2)	23.7 ± 3.6 (19.7-29.9)	0.001*
Vitamin C (mg)	83.7 ± 30.1 (48.1-146.6)	145.1 ± 30.0 (93.1-175.3)	0.001*
Copper (mg)	1.4 ± 0.2 (0.9-1.7)	2.0 ± 0.2 (1.6-2.8)	0.001*
Zinc (mg)	8.2 ±1.3 (5.8-10.3)	17.0 ± 3.2 (13.4-26.0)	0.001*
Magnesium (mg)	239.1 ± 45.5 (178.0-315.3)	346.8 ± 42.8 299.3-417.5)	0.001*

Values were given as mean ± SD (minimum- maximum).

loss.[16] Weight loss occurs both in fat and lean body mass.[13] Recent studies have shown that lean body mass is the main determinant of mortality, regardless of fat mass.[17-19] Low lean body mass leads to negative effects on skeletal muscle function, exercise capacity and global health status of the COPD patient and eventually increases the number of exacerbations.[20] In the present study, TSFT was over 50th percentile. Mid-upper-arm circumference was below 25th percentile and the average weight loss during the previous year was 4.2% (in both groups). when both groups were taken into account toget-

TABLE 3: Respiratory function tests of patients on day 1 and on day 10.

Parameters		Group I (n= 15)	Group 2 (n= 15) p*
FEV_1	Day I	1.25 ± 0.5 (0.61-1.99)	1.11 ± 0.6 (0.58-2.94) 0.517
	Day 10	1.21 ± 0.5 (0.53-2.01)	1.20 ± 0.3 (0.80-1.65) 0.942
	p	0.755	0.069
FVC	Day I	1.94 ± 0.6 (1.03-2.90)	1.70 ± 0.7 (0.80- 3.71) 0.307
	Day 10	1.94 ± 0.6 (1.00-3.12)	1.92 ± 0.4 (1.12-2.72) 0.953
	p	0.510	0.033Δ
TV	Day I	0.64 ± 0.3 (0.28-1.15)	0.43 ± 0.1 (0.25-0.63) 0.016
	Day 10	0.88 ± 0.6 (0.32-2.87)	0.56 ± 0.2 (0.27-0.97) 0.075
	p	0.190	0.002Δ
VC	Day I	2.0 ± 0.6 (1.10-3.06)	1.8 ±0.6 (0.90-3.43) 0.477
	Day 10	2.2 ± 0.6 (1.20-3.31)	2.1 ± 0.5 (1.20-3.00) 0.535
	p	0.551	0.053

FEV1: Forced expiratory volume FVC: Forced vital capacity TV: Tidal volume VC: Vital capacity,
Values were given as mean ±SD (minimum-maximum),
P : Significance tests of difference between two pairs,
P*: Significance tests of difference between two means,
Δ : p< 0.05.

her (Table 1).[12] So, the weight loss mainly originated from fat mass.

A close relationship exists between body composition and lung functions, especially with fat-free mass and pulmonary functions. Body fat is negatively correlated with FVC and abdominal circumference is negatively correlated with FVC and FEV_1.[14] Obesity contributes to the decrease in both FEV_1 and respiratory functions.[21,22] Santana et al, demonstrated a negative correlation between lung function tests (FEV_1, FVC) and abdominal region fat mass, and body fat percentage in older men.[23] Besides, increment in weight has some detrimental effects on the lungs such as function impairment in the extension of the chest and decrease in the downward extension of the diaphragm, due to increased intraabdominal pressure. Inefficient ventilation results with atelectasia in the peripheral lung fields and hypoxemia, which may increase the pulmonary vascular resistance. In our study, considering BMI as well as the waist/hip ratios of the patients in both groups, obesity was not present in our patients and no significant correlation was observed in the FEV_1 values (Table 5).

Openbrier et al studied COPD patients in terms of nutritional status and lung functions.[24] No dietary deficiency was observed in the group with a FEV_1 value of 57%, while somatic loss was observed in the group whose FEV_1 was 35%. FEV_1 is not a parameter that can change easily. An evident change is not expected despite treatment, especially in patients with long term COPD.[7] In our study, no significant change was observed on the first and the tenth day of FEV_1 for both groups (3.2% decrease in group I, 7.5% increase in group

TABLE 4: Blood gas parameters of the patients.

		Days					
		1	3	5	7	9	p*
Parameters							
	Group I	70.99 ± 26.29	65.25 ± 16.57	70.87 ±23.50	76.45 ± 26.68	74.95 ±17.50	0.692
		(37.30-132.60)	(33.50-94.70)	(43.80-122.10)	(46.20-151.30)	(50.10-111.70)	
PO2 (mmHg)	Group II	66.48 ±21.01	66.20 ± 14.78	55.69 ±15.84	75.50 ±35.39	69.26 ±20.00	0.211
		(37.80-113.20)	(36.60-85.10)	(32.10-89.70)	(43.60-179.70)	(33.70-102.70)	
	p	0.608	0.870	0.470	0.935	0.414	
	Group I	51.14± 14.89	50.95 ±12.33	49.20 ±10.41	55.21 ±23.4	48.27 ±10.15	0.757
		(31.80-90.70)	(31.20-70.50)	(30.50-61.30)	(30.00-132.30)	(31.90-67.60)	
PCO_2 (mmHg)	Group II	52.81 ± 13.71	53.21 ± 13.77	50.31 ±11.72	50.90 ± 11.69	51.08 ±12.29	0.962
		(28.50-76.90)	(30.10-72.20)	(33.70-68.20)	(34.80-66.00)	(32.40-75.80)	
	p	0.752	0.639	0.785	0.528	0.500	
	Group I	7.37 ± 0.04	7.40 ± 0.04	7.40 ± 0.04	7.39 ± 0.03	7.40 ± 0.04	0.140
		(7.29-7.43)	(7.33-7.49)	(7.34-7.45)	(7.34-7.45)	(7.34-7.50)	
PH (mmHg)	Group II	7.38 ± 0.04	7.41 ± 0.05	7.41 ± 0.05	7.41 ± 0.04	7.39 ± 0.04	0.252
		(7.30-7.45)	(7.26-7.49)	(7.30-7.48)	(7.32-7.47)	(7.26-7.46)	
	p	0.481	0.494	0.457	0.292	0.513	
	Group I	27.51 ± 6.70	29.63 ± 6.89	29.08 ± 6.35	30.06 ± 5.98	28.61±5.42	0.830
		(20.70-43.30)	(21.00-42.60)	(19.00-40.20)	(21.50- 41.70)	(20.90-40.40)	
HCO_3 (mmol/L)	Group II	29.44 ± 5.70	31.23 ± 5.93	30.29 ± 6.67	31.32 ± 7.83	30.01 ± 6.23	0.921
		(16.40-40.80)	(22.60-44.40)	(20.70-42.70)	(21.60-47.60)	(21.80-40.90)	
	p	0.403	0.503	0.614	0.625	0.515	

Values are given as mean ± SD (minimum-maximum),

p: Evaluations between all time intervals, significance test between two averages ,

p*: Evaluation of changes between days 1, 3, 5, 7, 9 in every group, one-way ANOVA ($p< 0.05$).

II, respectively). FVC is a parameter that can change easily and be more reactive to treatment, relative to FEV_1.[7] In this study, the increase between the first and the tenth day FVC measurements in the group who received high-fat, low-carbohydrate diet statistically significant ($p< 0.05$). Similarly, the increase in TV in group II was significant ($p< 0.05$) and this change was considered secondary to the high-fat low-CHO diet.

Although VC increased more in the high-fat, low-CHO group (14.3% versus 9%), the difference between the two groups was not significant.

VCO_2 increased by a diet high in CHO can easily be tolerated in healthy indivuduals, but dyspnea develops along with an increase in partial arterial carbondioxide pressure ($PaCO_2$), in patients with COPD as well as a likely reduction in exercise tolerance.[25] In a study, 12 male patients with COPD ingested a diet with normal CHO and normal fat, a diet with high CHO and low fat, and a diet including high fat and low CHO, in sequence. An increase in CO_2 was observed after these three diets during rest as well as during exercise. However, changes were observed in minute ventilation and VCO_2 increase following the three meals, and no significant difference was detected in minute ventilation increase.[26] Moreover, another accepted argument is that the amount of VCO_2 will not increase unless the maximum glucose oxidation rate is exceeded. Hence, appropriateness and content of the energy intake is important. In our study, partial arterial oxygen pressure (PaO_2) increased in both groups during the study period (5.3% in group I, 4% group II, respectively), while $PaCO_2$ pressure decreased in both groups (5.6% in group I, 3.2% in group II, respevtively). However, these changes

TABLE 5: Correlation of FEV_1 with COPD and several variables (smoking habits, serum albumin levels, some selected nutrient intakes and antropometric measurements).

		Group I (n= 15)		Group II (n= 15)	
		FEV_1(1)	FEV_1(10)	FEV_1(1)	FEV_1(10)
Smoking (package/year)		-0.242	-0.170	0.331	-0.011
Energy (Calories)		-0.073	0.438	-0.029	-0.021
CHO (g)		-0.521*	-0.007	0.163	0.200
Fat (g)		0.267	0.524*	-0.162	-0.089
Protein (g)		0.113	0.391	-0.182	-0.139
Vitamin C (mg)		-0.023	-0.020	0.173	0.240
Vitamin A (IU)		0.492	0.459	0.247	0.321
Magnesium (mg)		0.073	-0.010	0.172	0.238
Albumin (g/dL)	Day 1	-0.090	0.190	-0.100	-0.370
	Day 10	0.040	0.200	0.430	0.210
BMI (kg/m^2)	Day 1	0.177	0.261	0.426	0.551*
	Day 10	0.182	0.225	0.211	0.370
Waist/hip	Day 1	0.362	0.359	-0.307	-0.023
	Day 10	0.099	-0.025	-0253	-0.467
TSFT (mm)	Day 1	0.363	0.111	0.478	0.493
	Day 10	0.161	0.390	0.386	0.218

Spearman Rank Correlation Test. BMI: Body mass index; CHO: Carbohydrate; TSFT: Triceps skinfold thickness.

* : $p< 0.05$.

were insignificant. Although insignificant, the decrease in $PaCO_2$ pressure may be interpreted as a good indicator of the response to treatment. The patients included in this study had severe COPD. The short study period might be insufficient to observe the beneficial effects of the diet on blood gases, as well.

Blood gas parameters on days 1, 3, 5, 7 and 9 days of the study did not differ significantly betvveen the study groups during the study period, but there was an improvement on these values (Table 4). In a study, patients consumed diets containing high-CHO (83%) and high-fat (50%), first at low infusions and then at increasing levels. Following low energy and infusion, VO_2, VCO_2, minute ventilation, heat production and heart rate increased linearly.[27]

Electrolyte imbalance is common in COPD patients and can improve the prognosis of the disease. This imbalance in electrolytes not only disturbs skeletal function but may also result in cardiac arrhythmias. The treatment of hypophosphatemia, hyper/hypokalemia, hypocalcemia, and hypomagnesemia improves diaphragmatic function but overfeeding must also be introduced in caution. In our study we found significantly higher magnesium, zinc, copper, vitamin C, and phosphorous intakes in group II (Table 2). In a cohort study, the relationship between decreasing lung functions and magnesium, vitamin C and other antioxidant intakes in the diet were investigated in COPD patients. After 9 years, a positive relationship was found between high FEV_1 levels and high vitamin C and magnesium intakes. It was concluded that high vitamin C intake with diet or consumption of foods rich in vitamins could reduce the rate of decline in lung functions.[28] In the present study, the intake of vitamin C and magnesium was relatively ($p< 0.05$) higher in the high-fat, low-CHO group ($p< 0.05$). Positive effects of vitamin C may be observed in longer time periods.

CONCLUSION

Airway obstruction in COPD is irreversible. Therefore, diet treatment along with other clinical treatment options can be helpful in terms of

pulmonary function tests, blood gases and other factors, yet it cannot correct a chronic obstruction. In this study, statistically significant improvements in FVC and TV were observed with the administration of a high-fat, low-CHO diet in patients hospitalized for acute COPD attack as well as an increase in FEV_1 (although not significant). On the other hand, positive changes were observed in blood gas values. Therefore, the administration of a special dietary support (55% high-fat, 28% low-CHO) in patients hospitalized for acute COPD attack may accelerate recovery. In conclusion, future studies providing a special dietary support with a longer-time period and a larger sample size are needed to evaluate the blood gasses and lung function tests in COPD.

Diet should be an essential part of the treatment for COPD. A dietary intervention appropriate for the disease is important for the quality of the treatment. An adequate and balanced diet, along with consuming increased amounts of fresh vegetables, fruits, fish, reduced salt, antioxidant vitamins and unsaturated fatty acids will have a positive impact on the prevention and improvement of the severety of COPD. Determining the energy intake based on the patient's requirements and consumption of a high-fat, low-CHO diet during the acute attack period may be beneficial.

REFERENCES

1. Polatlı M. [COPD and pneumonia]. Turkiye Klinikleri J Int Med Sci 2005;1(46):47-52.
2. Demir R. [Chronic obstructive pulmonary disease: Definition, epidemiology and risk effects]. Turkiye Klinikleri J Thorax Dis 2003;1(1):1-6.
3. Global Initiative for Chronic Obstructive Lung Diseases (GOLD). Global strategy for the diagnosis, management and prevention of chronic obstructive pulmonary disease: NHLAVHO Workshop Report, NIH publication No. 2701. Bethedsa: US Department of Health and Human, 2001.
4. Askanazi J, Weissman C, Rosenbaum SH, Hyman AI, Milic-Emili J, Kinney JM. Nutrition and the respiratory system. Crit Care Med 1982;10(3):163-72.
5. Romieu I, Trenga C. Role of nutrition in environmental lung disease. Semin Resp Crit Care Med 1999; 20(6):581-90.
6. Hatch GE. Asthma, inhaled oxidants, and dietary antioxidants. Am J Clin Nutr 1995;61(3 Suppl):625S-30S.
7. Reis AL. Rehabilitation in chronic obstructive pulmonary disease and other respiratory disorders. In: Fishman AP, Elios JA, Fishman JA, Grippi MA, Kaiser LR, Senior RM, eds. Fishman's Manual Pulmonary Disorders. Vol 1. 3rd ed. New York: McGraw-Hill; 1997. p.709-19.
8. Goldstein SA, Thomashow B, Askanazi J. Functional changes during nutritional repletion in patients with lung disease. Clin Chest Med 1986;7(1):141-51.
9. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. American Thoracic Society. Am J Respir Crit Care Med 1995;152(5 Pt 2): S77-121.
10. Ferreira IM, Brooks D, Lacasse Y, Goldstein RS. Nutritional support for individuals with COPD: a meta-analysis. Chest 2000;117(3): 672-8.
11. Baysal A, Keçecioğlu S, Arslan P, Yücecan S, Pekcan G, Güneyli U, et al. Besinlerin Bileşimleri. Türkiye Diyetisyenler Derneği Yayını. 3rd ed. Ankara: Yeniçağ Matbaacılık; 1991. p. 11-26.
12. Pekcan G. [Nutrition asessment]. In: Buzgan T, Kesici C, Çelikcan E, Soylu M, eds. Beslenme Bilgi Serisi. 1st ed. Ankara: Klasmat Matbaacılık; 2008. p. 213-25034-50.
13. Wouters EF. Nutrition and metabolism in COPD. Chest 2000;117(5 Suppl 1): 274S-80S.
14. Górecka D, Gorzelak K, Sliwiński P, Tobiasz M, Zieliński J. Effect of long-term oxygen therapy on survival in patients with chronic obstructive pulmonary disease with moderate hypoxaemia. Thorax 1997;52(8):674-9.
15. Brown SE, Wiener S, Brown RA, Marcarelli PA, Light RW. Exercise performance following a carbohydrate load in chronic airflow obstruction. J Appl Physiol 1985;58(4):1340-6.
16. Lazarus R, Gore CJ, Booth M, Owen N. Effects of body composition and fat distribution on ventilatory function in adults. Am J Clin Nutr 1998;68(1):35-41.
17. Schols AM, Broekhuizen R, Weling-Scheepers CA, Wouters EF. Body composition and mortality in chronic obstructive pulmonary disease. Am J Clin Nutr 2005;82(1):53-9.
18. Vestbo J, Prescott E, Almdal T, Dahl M, Nordestgaard BG, Andersen T, et al. Body mass, fat-free body mass, and prognosis in patients with chronic obstructive pulmonary disease from a random population sample: findings from the Copenhagen City Heart Study. Am J Respir Crit Care Med 2006;173(1):79-83.
19. Slinde F, Grönberg A, Engström CP, Rossander-Hulthén L, Larsson S. Body composition by bioelectrical impedance predicts mortality in chronic obstructive pulmonary disease patients. Respir Med 2005;99(8):1004-9.
20. Mostert R, Goris A, Weling-Scheepers C, Wouters EF, Schols AM. Tissue depletion and health related quality of life in patients with chronic obstructive pulmonary disease. Respir Med 2000;94(9):859-67.
21. Collins LC, Hoberty PD, Walker JF, Fletcher EC, Peiris AN. The effect of body fat distribution on pulmonary function tests. Chest 1995;107(5):1298-302.
22. Lazarus R, Sparrow D, Weiss ST. Effects of obesity and fat distribution on ventilatory function: the normative aging study. Chest 1997;111(4):891-8.
23. Santana H, Zoico E, Turcato E, Tosoni P, Bissoli L, Olivieri M, et al. Relation between body composition, fat distribution, and lung function in elderly men. Am J Clin Nutr 2001;73(4): 827-31.

24. Openbrier DR, Irwin MM, Rogers RM, Gottlieb GP, Dauber JH, Van Thiel DH, et al. Nutritional status and lung function in patients with emphysema and chronic bronchitis. Chest 1983;83(1):17-22.

25. Ferreira I, Brooks D, Lacasse Y, Goldstein R. Nutritional intervention in COPD: a systematic overview. Chest 2001;119(2):353-63.

26. Frankfort JD, Fischer CE, Stansbury DW, McArthur DL, Brown SE, Light RW. Effects of high- and low-carbohydrate meals on maximum exercise performance in chronic airflow obstruction. Chest 1991;100(3):792-5.

27. Heymsfield SB, Head CA, McManus CB 3rd, Seitz S, Staton GW, Grossman GD. Respiratory, cardiovascular, and metabolic effects of enteral hyperalimentation: influence of formula dose and composition. Am J Clin Nutr 1984;40(1):116-30.

28. McKeever TM, Scrivener S, Broadfield E, Jones Z, Britton J, Lewis SA. Prospective study of diet and decline in lung function in a general population. Am J Respir Crit Care Med 2002;165(9):1299-303.