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Nutrition, Body Weight and Deterioration of Familial Combined Hyperlipidemia

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A B S T R A C T

Lipid and apolipoprotein serum levels as a consequence of excessive nutrition in the overweight individuals with familial combined hyperlipidemia (FCHL) in comparison with the obese ones are studied only sporadically. In this study, the effect of overweight and obesity in subjects with FCHL on serum lipids and apolipoproteins was investigated. The participants were 36 overweight and 10 obese men. 17 normolipidemic healthy men served as the control group. The mean age of all subjects included was 49±9 years. Lipid and apolipoprotein serum levels were determined by standard methods. The increased body weight in overweight men with FCHL correlates with increased cholesterol and triacylglycerol serum levels ($p<0.001$), atherogenous ratio values, apolipoprotein serum levels – apo C-III, apo C-II and apo B_{100} ($p<0.001$) as well as decreased HDL cholesterol serum levels ($p<0.05$). Lipid metabolism in men with FCHL is deteriorated by a high energy intake and its low output. The overweight and not only obesity, in association with FCHL, is an important risk factor for premature development of ischemic events.

Key words: *overweight, obesity, hypercholesterolemia, hypertriacylglycerolemia, apolipoproteins, lipids*

Introduction

Familial combined hyperlipidemia is a common familial lipid disorder characterized by a variable pattern of elevated levels of plasma cholesterol and/or triacylglycerols and some apolipoproteins (e.g. apo B_{100})[1,2]. It is present in 10% to 20% of patients with premature coronary artery disease. The genetic etiology of the disorder, including the number of genes involved and the magnitude of their effects is unknown. Goldstein et al. gave the designation »familial combined hyperlipidemia« to the most common genetic form of hyperlipidemia identified in a study of myocardial infarction survivors. The affected individuals characteristically showed an elevation in both cholesterol and triacylglycerols in the blood. The combined disorder was shown to be distinct from familial hypercholesterolemia (Fredrickson type II), and from familial hypertriacylglycerolemia[1,3].

In this study, we investigated the extent of changes in lipid and apolipoprotein (apo) serum levels as well as atherogenous ratios in overweight men with familial combined hyperlipidemia (FCHL) and with behavioral pattern of excessive nutrition and low energy output. These parameters are only rarely studied in both the overweight and obese subjects with FCHL due to a low incidence of the disease. Therefore, we compared lipid and apolipoprotein serum levels observed in overweight and obese subjects with those observed in the control group. In the overweight subjects, apolipoprotein B_{100} (apo B_{100}), apolipoprotein C-III (apo C-III) and apolipoprotein C-II (apo C-II) serum levels are evaluated only very rarely.

Material and Methods

Forty-six subjects with FCHL were included in the present study. These subjects (6.5%) were selected from a group 710 re-screened adult clerks. Thirty-six of them were overweight (5.1%) (OW) and 10 were obese (1.4%) (OB). The control group included 17 men (2.4%) (CONTR) with normal body weight and not suffering from any genetic lipid disorder. The lipid and apolipoprotein serum levels were assessed after overnight fasting. The genetic causes of FCHL were not analyzed because of an exclusively epidemiological character of the present study. The presence of a hereditary disorder was concluded on the basis of positive history of FCHL in the first line family relatives using genealogical analysis. The criteria for FCHL included serum levels of triacylglycerols higher

Received for publication January 26, 2006

than 2.3mmol/l and the total cholesterol higher than 6.2mmol/l after overnight fasting. Furthermore, LDL cholesterol and the atherogenous ratios were also calculated. Daily food consumption was estimated on the basis of questionnaires.

From the anthropometrical variables, the following ones were determined: the body weight, body height, surface of the body and body mass index (BMI kg/m^2). BMI we used as a criterion to distinguish among normal weight, overweight and obesity[4]. Peripheral blood was collected after overnight fasting (12 hour).

The total cholesterol (TC), triacylglycerols (TG) and HDL-cholesterol (HDL-C) serum levels were assessed by using standard Pliva-Lachema sets (Brno, Czech Republic). The LDL cholesterol (LDL-C) and non-HDL cholesterol (non-HDL-C) serum levels were calculated using the Friedewald´s[5] and De Backer´s[6] formula, respectively. Vitamin C serum levels were assayed colorimetrically according to the method of Roe and Kuether[7].

Apolipoprotein C-II and C-III serum levels were determined by the method of radial immunoassay according to Mancini[8]. Antibodies and standards purchased from Daiichi Company (Tokyo, Japan) were used for their determination. Serum levels of apo B_{100} were detected by the electroimmunoassay according to the method of Curry[9] using the standards and antibodies from Behringwerke Company (Marburg, Germany).

Statistics

The data were analyzed using the univariate and bivariate analysis, χ^2-test, Pearson correlation test as well as partial correlation including canonical correlation and McNemar's test. The relative risk according to Sato's computation was also calculated. The standard deviation, median, percentile distribution, including the determination of 95% confidence limit (CL95%), and confidence interval (CI 5–95%) were calculated using the descriptive statistics (p value <0.05 was accepted as significant).

Results

The mean body weight and its CL95% were increased by 2.43 kg/m^2 and 3.1 kg/m^2, respectively, in overweight subjects, in comparison with the control group (11.8%). The mean body mass index in the overweight subjects was 27.8±1.4 kg/m^2. The food intake in overweight and obese subjects was compared to the control group and is described in Table 1. The p value was calculated for OW versus CONTR.

In overweight vs. control subjects, the mean serum levels of TC, LDL-C, TG and non-HDL-C were significantly increased by: 71.7%; 91.8%; 283.5%, and 104.2% ($p<0.001$, Table 2), respectively. Serum levels of HDL-C were significantly decreased by –5.8% ($p<0.05$). The mean values of TC/HDL-C, LDL-C/HDL-C, and high TG/low HDL-C known as the atherogenous ratios were significantly increased by 82.4%; 103.8%; and 308.1% ($p<0.001$), respectively. The mean value of the TG/apo C-II atherogenous ratio was increased by 96.6% ($p<0.003$). The mean value of the HDL-C/LDL-C atherogenous ratio was significantly decreased by –50.8% ($p<0.01$, Table 2).

TABLE 1
THE FOOD INTAKE AND NUTRITION STATE IN HEALTHY MEN (CONTROL GROUP) AND OVERWEIGHT AND OBESE SUBJECTS WITH FCHL

Food intake	CONTR	OW	OB	p
Glycides (%)	51	50–54	52–47	<0.05
Fat (%)	25	30–28	35–42	<0.01
Proteins (%)	24	20–18	13–11	<0.01
Fiber (g)	25–28	15–18	8–11	<0.01

CONTR – control group, OW – overweight subjects, OB – obese subjects, p – probability

The mean serum levels of apo B_{100} were significantly increased not only the obese but also in overweight subjects by 63.9% ($p<0.001$) and 58.5% ($p<0.01$), respectively. In overweight subjects the apo C-II serum level was increased by 93.3% and apo C-III by 161.8% ($p<0.001$, Table 2).

In Table 3 we summarize the confidence intervals (CI 5–95%) of all variables studied. In the overweight subjects and not only in the obese ones, differences of the TC serum levels significantly increased by 69.8% ($p<0.01$). Differences of the LDL-C, TG and non-HDL-C serum levels in CL95% also significantly increased by 100.7%, 357.7% and 108.1% ($p<0.001$). Significant differences in extent of 82.4% we detected in the TC/HDL-C ratio. Significant differences in extent of 116.7% in CL95% we detected in the LDL-C/HDL-C ratio.

In the overweight and obese men with FCHL we found in CL95% against control group significantly highly increased apo B_{100} ($p<0.01$), apo C-II ($p<0.03$; $p<0.01$) and extremely high apo C-III serum levels ($p<0.001$).

The significant increases in triacylglycerol serum levels after overnight fasting in overweight subjects are associated with increases in the BMI mean value (27.8± 1.4 kg/m^2). Even more pronounced increases in TG serum levels were observed in obese men. Elevation in triacylglycerol serum levels is also associated with a significant decrease in the mean, median and CL95% values of HDL-C serum levels ($p<0.05$). The mean HDL-C/LDL-C ratio significantly decreased in overweight subjects ($p<0.01$). The differences in TG/HDL-C ratio values between the overweight and obese subjects versus the control group were extremely high ($p<0.001$).

Consistently with the changes in serum lipid levels, apolipoprotein serum levels were also more significantly elevated in obese men than in overweight subjects. In 8.7% of overweight subjects we detected significantly increased apo B_{100} serum levels ($p<0.01$) but only a moderate increase in TC. This finding (after overnight fasting) is consistent with a picture of the hereditary hyperapobetalipoproteinemia[10].

TABLE 2
DISTRIBUTION OF THE LIPID AND APOLIPOPROTEIN SERUM LEVELS IN OVERWEIGHT AND OBESE SUBJECTS VERSUS CONTROLS

Biological variables	OW		OB		CONTR		OW vs. CONTR		OB vs. CONTR	
	X±2SD	MD	X±2SD	MD	X±2SD	MD	Δ%	p	Δ%	p
BMI (kg/m²)	27.8±1.38	28.50	32.80±2.81	32.80	25.37±0.64	25.10	9.58	<0.05	29.29	<0,05
TC (mmol/l)	6.80±1.25	7.40	6.76±0.92	6.70	3.96±0.46	3.72	71.72	<0.001	70.71	<0.001
LDL-C (mmol/l)	4.20±1.27	5.10	3.86±1.06	3.94	2.19±0.26	2.04	91.78	<0.001	76.26	<0.001
HDL-C (mmol/l)	1.29±0.14	1.32	1.27±0.24	1.25	1.37±0.23	1.35	–5.84	<0.05	–7.30	<0.05
TG (mmol/l)	3.26±1.41	2.51	4.21±2.15	3.60	0.85±0.21	0.94	283.53	<0.001	395.29	<0.001
non-HDL-C (mmol/l)	5.41±1.27	6.10	5.49±0.87	5.49	2.65±0.29	2.50	104.15	<0.001	107.17	<0.001
TC/HDL-C	5.27±0.36	5.61	5.32±0.23	5.36	2.89±0.13	2.42	82.35	<0.001	84.08	<0.001
LDL-C/HDL-C	3.26±0.57	4.62	3.04±0.22	3.15	1.60±0.07	1.51	103.75	<0.001	90.00	<0.001
HDL-C/LDL-C	0.31±0.05	0.22	0.33±0.02	0.32	0.63±0.02	0.66	–50.79	<0.01	–47.62	<0.01
TG/HDL-C	2.53±0.74	1.90	3.31±0.90	2.88	0.62±0.04	0.70	308.06	<0.001	433.87	<0.001
TG/apo C-II	0.57±0.03	0.48	0.56±0.12	0.46	0.29±0.05	0.31	96.55	<0.003	93.10	<0.003
Apo B_{100}/ LDL-C	25.40±3.79	17.05	28.58±2.26	28.43	30.73±0.65	30.88	–17.34	<0,05	–7.00	ns
apo B_{100} (mg/dl)	106.7±11.5	104.00	110.3±19.20	112.00	67.30±9.59	63.00	58.54	<0.01	63.89	<0.001
apo C-II (mg/dl)	5.74±2.00	5.20	7.58±1.75	7.80	2.97±0.18	3.00	93.27	<0.001	155.22	<0.001
apo C-II (mg/dl)	11.52±4.58	10.50	16.34±4.96	15.80	4.40±1.01	4.00	161.82	<0.001	271.36	<0.002
Vitamin C (μmol/l)	48.30±19.80	43.90	43.40±17.7	42.80	50.60±9.06	49.40	–4.55	ns	–14.23	<0.05

OW – overweight subjects, OB – obese subjects, CONTR – control group, X – mean, SD – standard deviation, MD – median, Δ% – percentage differences, p – probability, ns – not significant

TABLE 3
DISTRIBUTION OF THE VARIABLES IN CI 5–95% INCLUDING DIFFERENCES IN THE CI95% IN THE OVERWEIGHT AND OBESE VERSUS CONTROL GROUP

Biological variables	OW (CI 5–95%)	OB (CI 5–95%)	CONTR (CI 5–95%)	OW vs. CONTR Δ%	p	OB vs. CONTR Δ%	p
BMI (kg/m²)	25.10–29.40	30.09–36.80	24.58–26.30	11.79	<0.05	39.92	<0.02
TC (mmol/l)	4.77–7.93	5.45–8.52	3.44–4.67	69.81	<0.01	82.44	<0.01
LDL-C (mmol/l)	2.14–5.40	2.20–5.54	2.01–2.69	100.74	<0.001	105.95	<0.001
HDL-C (mmol/l)	1.22–1.60	0.98–1.64	1.08–1.72	–6.98	<0.05	–4.65	ns
TG (mmol/l)	2.31–5.63	2.37–6.22	0.62–1.23	357.72	<0.001	405.69	<0.001
Non-HDL-C (mmol/l)	3.31–6.45	4.21–7.18	2.36–3.10	108.06	<0.001	131.61	<0.001
TC/HDL-C	3.91–4.96	5.56–5.20	3.19–2.72	82.35	<0.01	91.18	<0.01
LDL-C/HDL-C	1.75–3.38	2.24–3.38	1.86–1.56	116.34	<0.001	116.54	<0.001
HDL-C/LDL-C	0.57–0.30	0.45–0.30	0.54–0.64	–53.13	<0.01	–53.13	<0.01
TG/HDL-C	1.89–3.52	2.42–3.79	0.57–0.72	388.89	<0.001	426.39	<0.001
TG/apo C-II	0.68–0.69	0.46–0.64	0.11–0.19	263.16	<0.003	236.84	<0.003
Apo B_{100}/ LDL-C	44.09–23.00	38.18–25.54	29.45–31.71	–27.47	<0.05	–19.46	<0.05
Apo B_{100} (mg/dl)	94.35–124.20	84.0–141.50	59.20–85.30	45.60	<0.01	65.89	<0.01
Apo C-II (mg/dl)	3.45–8.21	5.18–9.75	5.40–6.46	27.09	<0.03	50.93	<0.01
Apo C-III (mg/dl)	6.27–17.44	8.93–24.00	3.17–6.03	189.22	<0.001	298.01	<0.001
Vitamin C (μmol/l)	23.50–76.68	20.20–74.58	36.86–67.70	13.26	<0.05	10.16	<0.05
N	10	36	16				

OW – overweight subjects, OB – obese subjects, CONTR – control group, CI 5–95% – confidence interval, Δ% – differences in CL95%, p – probability, N – sample size, ns – not significant

Not only in obese subjects but also in overweight men we found significant correlation between the mean serum levels of apo C-II and TG (r=0.33, p<0.04). This is a sign of a tight metabolic relationship between the two variables. Significant changes were also detected in the TG/apo C-II ratio. Its value in CL95% was nearly three--times higher in obese and overweight subjects than in the control group (p<0.003, Table 3).

The TG/HDL-C ratio shows a significant inverse correlation with body mass index: r=–0.48 (p<0.002) in overweight adults. In addition, the apo B_{100}/LDL-C ratio directly correlates with body mass index: r=0.47 (p< 0.002).

In Figure 1, a regression curve between apo C-II and apo C-III is shown. This regression confirms a close relationship (p<0.01) between both variables in overweight subjects. The correlation coefficient has the value: r= 0.41 (p<0.01) at the t-value: 2.63.

In Figure 2, a regression curve is shown between apo C-III and HDL-C serum levels in overweight subjects. We found an inverse relationship (p<0.01) between both variables: r=–0.41 at the t-value: 6.367.

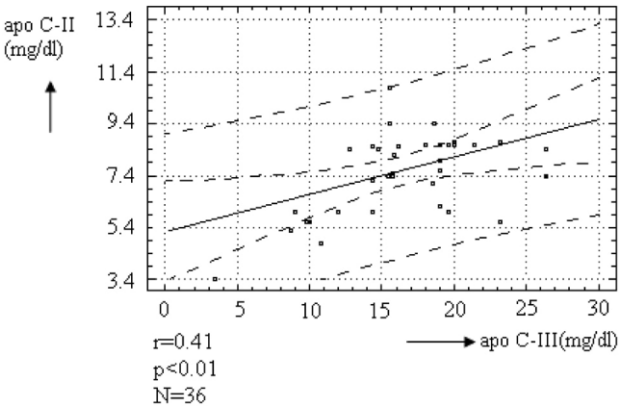

Fig. 1. Regression of the apo C-II and apo C-III in the overweight (OW) subjects. r – correlation coefficient, p – probability, N – sample size.

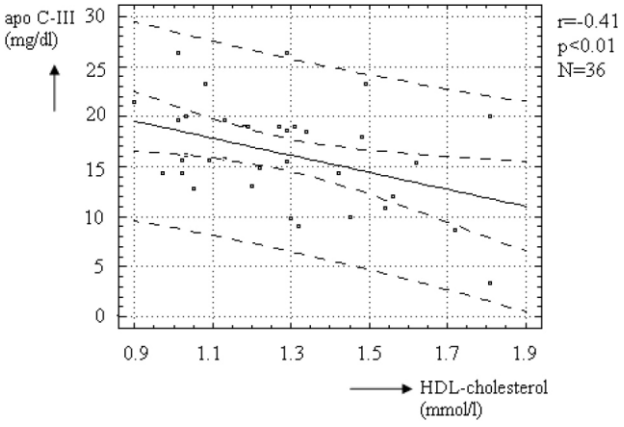

Fig. 2. Regression of the apo C-III and HDL-C in the overweight (OW) subjects. r – correlation coefficient, p – probability, N – sample size.

Discussion

Traditionally, in our geographic region, moderate body weight excess is accepted as a sign of good health. This misleading view, together with low social status and behavioural patterns of excessive eating are the main causes of consuming a diet rich in fat (mainly saturated) and poor in fruits and vegetables what is associated with a body weight gain. Usually, the frequency of FCHL is relatively low but is increased in overweight and obese subjects[11,12].

Over-nutrition deteriorates genetically caused diseases of the lipid metabolism and this is evidently notable on the mean and CL95% of the lipids and apolipoproteins serum levels in overweight and obese subjects.

FCHL, as expressed by the changes in lipid and apolipoprotein serum levels, is besides genetic causes deteriorated also by an undesirable overeating. Significant elevation in LDL-C and TG serum levels and raised TG/HDL ratio after overnight fasting in overweight and obese men demonstrates this relationship.

The HTG after overnight fasting, as a dominant finding, is significantly expressed in overweight and obese subjects by significant elevation of apo C-III serum levels. This is an indirect signal of increased production of triglyceride rich-lipoprotein (TG-RLPs) in the liver. Elevated apo C-III serum levels are a reliable indirect sign of the production of these large lipoprotein particles. The TG-rich lipoprotein particles usually display a delayed catabolism that is among other reasons also caused by a direct inhibitory effect of apo C-III on the lipoprotein lipase activity[13,14]. The TG-RLPs remnants are delayed in circulation. Therefore they are more susceptible to oxidation. Simultaneously with their increased uptake to by macrophages, they are deposits into the arterial wall. This explains why these large lipoprotein particles are atherogenous. The storage of TG in the adipocytes of subcutaneous fat mass and predominantly viscerally is the main cause of the body weight gain. However, accumulation of TG associates with significant increase in the apo C-III serum levels and their increased production has a malicious impact on the catabolism of TG-RLPs. Significant elevation of these variables potentiates the risk of premature development of atherosclerosis leading to an increased frequency of ischemic events in the heart, brain and peripheral arteries[15]. Our data suggest that not only obesity but also overweight, particularly in combination with genetic causation of the FCHL, is associated with serious changes in lipid and apolipoprotein serum levels[2,12].

High TG-RLPs serum levels are an indirect marker of endothelial dysfunction and through this pathway, they amplify the global risk of ischemic events[12]. Therefore, they play a significant role in triggering the early phases of atherosclerosis and mainly atherothrombosis[16,17,18] through the pathways of plaque destabilization. Usually, HTG as-

sociates with low serum levels of HDL-C and is an important clinical sign for dyslipidemia. The mechanism of this type of disorder is sophisticated. In overweight and obese subjects it is also potentiated by physical inactivity and an intake of high amounts of fat, mainly saturated and trans-fatty acids (Table 1). We assume that the significantly increased value of TG/apo C-II ratio in CL95% may be used as a useful indirect sign for delayed catabolism of TG-RLPs (Table 3). It seems to be evident that there is a significant disproportion between apo C-II and triacylglycerol serum levels in subjects with overweight. Nearly three times more increased apo C-III serum levels against the control group means that activation of the lipoprotein lipase by apo C-II is not satisfactorily efficient in the lipolytic pathway of lipoprotein particles in spite of its increased serum levels. With high probability we may exclude genetic defects and apo C-II deficit in the overweight and obese subjects as one of the FCHL causes. Generally, genetic defect in apo C-II is a very rare event because mostly it is not compatible with survival.

Inhibitory effect of apo C-III on the activity of lipoprotein lipase and lipolysis of TG-RLPs is also experimentally proven in transgenic mice[19–21]. Increased apo C-III serum levels generally associate with production of TG-RLPs via inhibition of their clearance[22].

The high serum levels of apo C-III, as a marker of premature acute ischemic events, show a 3.4-times higher ability to predict ischemic heart disease (IHD). Apo C-III has a specific effect in patients with HTG because it decreases apo E-mediated remnant removal by apo E displacement from VLDL particles[23]. This mechanism contributes to HTG via blocking the binding of VLDL or IDL particles on the LDLR-1, LDLR-2, eventually also VLDLR and SR-BI receptor.

Not only obesity but also overweight closely associates with development of metabolic syndrome and has a close relationship to the development of insulin resistance[24,25]. This explains why also the overweight subjects with genetically caused FCHL, increased blood pressure and low HDL cholesterol, are threatened with premature development of diabetes mellitus type 2, IHD, stroke and atherosclerosis of the peripheral arteries.

We propose to introduce to the clinical practice the calculation not only the LDL-C/HDL-C ratio in CL95% but also the high TG/low HDL-C ratio for its high potential to predict premature development of IHD and of stroke[26,27].

HTG detected after overnight fasting in the overweight subjects with FCHL associates with a significant increase in the apo B_{100} serum level. Significant elevation in the apoB_{100}/LDL-C ratio value is a valuable pathognomic marker of a disproportion between apo B_{100} and LDL-C serum levels, and an indirect sign of the LDL particles type B production, as well as a reliable predictor of the premature development of IHD and myocardial infarction[11,28,29]. The TG serum levels equal or over 1.7 mmol/l in the overweight and obese subjects serve as an indirect predictor of atherogenous LDL particles production (ALP) and hence a cause of endothelial dysfunction[16,30].

Significant depletion of HDL-C has a negative influence on its anti-inflammatory and anti-atherosclerotic effects, from which thus the overweight subjects cannot benefit[31].

Very tight correlation between HTG (after overnight fasting) and BMI in the overweight, as well as obese persons indicates a very close metabolic association between with deposition of TG into adipose tissue and over-nutrition, high-energy intake and mainly low energy output. It is the typical »acquired« behavioral pattern of the people with sedentary lifestyle and low physical activity[2,32,33].

Significant increase in the TG-RLPs in the obese and overweight subjects frequently associates with serious disorders in platelet aggregability. HTG triggers hyperactivity of the platelets with a tendency to hypercoagulation states. In addition, significant increase in the plasma apo B_{100} levels predicts platelet-dependent thrombosis in patients with coronary artery disease[34].

Significant depletion of vitamin C serum levels in the HTG state in the overweight and obese subjects is an important sign of decreased anti-oxidative activity. Decreased vitamin C serum level is generally accepted as a predisposition for higher oxidative risk[17,35–37].

These changes detected in the overweight subjects with FCHL indicate that not only obesity but also the overweight is not a benign and tolerable biological, socio-cultural and socio-educational marker due to the above mentioned reasons.

Conclusion

The FCHL as a genetically caused disease is deteriorated by a high-energy intake and sedentary lifestyle because of serious changes in the lipid and apolipoprotein serum levels. In both the overweight and obese subjects, the HTG detected after overnight fasting, is associated with a significant increase in serum levels of apo C-III, apo C-II, apo B_{100} and TG, as well as the HDL-C depletion. Surprising is the finding of the increased apo C-II serum levels because it is in contradiction to the traditional view. It might be a compensatory attempt to decrease high serum levels of triacylglycerols. Increased apo C-III serum levels in association with HTG (after overnight fasting) are a sign of TG-RLPs overproduction. This is associated with delayed catabolism of lipoproteins and triggering oxidation of lipoprotein particles and production of small LDL atherogenous particles. Decreased HDL-C serum levels indicate a lipid disorder what can be extremely hazardous mainly in adults with FCHL and increased body weight.

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PREHRANA, TJELESNA TEŽINA I POGORŠAVANJE OBITELJSKI NASLJEDNE HIPERLIPIDEMIJE

SAŽETAK

Razina lipida i apolipoproteina u serumu kao posljedica pretjeranog unosa hrane kod osoba s prekomjernom tjelesnom težinom s obiteljski nasljednom hiperlipidemijom (FCHL) u usporedbi s pretilim osobama proučava se sporadično. U ovome istraživanju proučavao se utjecaj prekomjerne tjelesne težine i pretilosti kod osoba s FCHL na razinu serumskih lipida i apolipoproteina. Učesnici u ovom istraživanju bili su 36 muškaraca s prekomjernom tjelesnom težinom i 10 pretilih muškaraca. 17 zdravih muškaraca s normalnom razinom lipida činili su kontrolnu skupinu. Srednja starosna dob svih ispitanika bila je 49±9 godina. Razine lipida i apolipoproteina u serumu određivane su upotrebom standardiziranih metoda. Tjelesna težina i kod muškaraca s prekomjernom tjelesnom težinom s FCHL, a ne samo kod pretilih muškaraca, povezana je s povišenom razinom kolesterola i triglicerida u serumu ($p<0.001$), aterogenim promjenama, razinom apolipoproteina u serumu – apo C-III, apo C-II i apo B_{100} ($p<0.001$) te sa smanjenom razinom HDL kolesterola u serumu ($p<0.05$). Metabolizam lipida kod muškaraca FCHL pogoršava se s visokim unosom energije u odnosu na malu potrošnju. Prekomjerna tjelesna težina, a ne isključivo pretilost povezana s FCHL, značajan je rizični faktor za prerani nastanak ishemije.