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Relationship of Epicardial and Subcutaneous Fatty Acids With Serum Lipids and Vascular Cramps in Patients Undergoing Coronary Artery Bypass Graft

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Abstract

Objectives: Fatty acids may include saturated or unsaturated fat. It seems that the consumption of trans fatty acids (TFA) can raise the risk of coronary artery diseases (CADs). The composition of the fatty acids of epicardial and subcutaneous adipose tissues (SATs) is a proper biomarker for assessing the fat used in a long-term period; therefore, nutrition can affect this composition. In addition, the human serum paraoxonase enzyme is considered an estraz/lactonase whose activity decreases in coronary heart disease (CHD). The present study aimed to investigate the relationship between metabolic variables related to the fatty acid composition of epicardial and subcutaneous fatty tissues and the activity of the paraoxonase enzyme. Further, the current study sought to explore the regional differences between subcutaneous and epicardial fatty acids, and the relationship between these measurements, as well as metabolic variables and food.

Materials and Methods: This descriptive cross-sectional study included 42 patients within the age range of 35-65 years who underwent coronary artery bypass graft. The fatty acid profile was measured by means of gas chromatography equipment, followed by estimating the lipid parameters of serum samples using commercial kits and enzymatic method in the autoanalyzer. Finally, paraoxonase enzyme activity was evaluated by Sigma chemical paraoxon substrate.

Results: Based on the results, the amounts of saturated fatty acids (SFAs) such as meristic (14:0), palmitic (16:0), and stearic (18:0) acids of the epicardium were higher. However, the levels of unsaturated fatty acids including palmitoleic (16:1), oleic (18:1 n-9), linoleic (18:2 n-6), and linolenic acids of the epicardium were lower compared to the SAT. Further, hypertension had a positive relationship with 18:1 n-11 (r=0.349, *P*=0.024) while a negative relationship with 18:1 n-9 (r=0.319, *P*=0.041) and 18:2 n-6 (r=0.391, *P*=0.01) epicardial adipose tissues. Foods such as fruits and vegetables had a positive relationship with linolenic acid (18:3 n-9) and conjugated linolenic acid epicardium. Furthermore, paraoxonase enzyme activity reduced by increasing the number of vessel cramps. Moreover, body mass index was found to have a negative relationship with subcutaneous SFAs whereas a positive association with the subcutaneous palmitoleic acid (16:1 n-7).

Conclusions: In general, the findings revealed that the amounts of subcutaneous and epicardial fatty acids vary in individuals with different CADs and that both types of fatty acids and serum lipid profile are correlated with each other. Additionally, there is a relationship between fatty acids of these two tissues and serum lipid profiles and food, as well as between paraoxonase enzyme and vascular cramps.

Keywords: Epicardial adipose tissue, Fatty acids, Cardiac artery bypass graft, Paraoxonase

Introduction

The adipose tissue is considered a metabolic and highly complex endocrine organ which has various topical and systemic effects (1,2). It stores calories and regulates energy homeostasis of the human body (3). In addition, in over-nutrition conditions, the adipose tissue accumulates nutritional supplements such as neutral fats while, in nutrition deficit conditions, it provides other tissues with nutrients by means of the lipolysis (4). Further, this tissue acts as an energy store and it is an endocrine organ influenced by immunologic and metabolic conditions of the body (5). Several saturated fatty acids (SFAs) like lauric, myristic, palmitic, and some trans fatty acids (TFA) have potential negative impacts on health (6). Furthermore, fatty acids of the adipose tissue seem to be associated with a variety of diseases, especially cardiovascular disease (CVD) (7).

Cholesterol is a major lipid in the incidence of atherosclerotic plaques. The role of high free cholesterol in plaque expression is greater than cholesterol esters. Atherosclerosis can be prevented by reducing the lipid accumulation in arterial wall which is achieved by lifestyle improvements including quitting smoking, eating healthy, and exercising (8). Despite general similarities of the tissues, different types of adipose tissues, especially subcutaneous and visceral fat, have different characteristics (9-11). Visceral fat is metabolically very active as such high lipolysis in the visceral adipose tissue

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increases the flow of the free fatty acids to the liver, raises fat store, and thus leads to resistance of the liver to insulin (12). Individuals with more body fat like the accumulation of fat in abdominal subcutaneous and visceral regions are susceptible to developing metabolic and cardiovascular complications, especially when the extra fat is located in visceral and central areas (13).

Normally, there is adipose tissue around epicardial coronary arteries. The thickness of this tissue is an appropriate criterion for estimating visceral obesity (14). The results of the study by Baker et al demonstrated that the epicardium adipose tissue as a paracrine tissue and, especially endocrine, is a risk factor for CVD (15).

The epicardial fat storage site, as a sort of visceral fatty tissue, is located around the coronary artery of the pericardium and the right anterior ventricle (16). Additionally, epicardial adipose tissue (EAT) is a unique type of fat located between the myocardial surface and pericardial visceral layer. In addition, physiological actions of this tissue involve myocardial protection against hypothermia and mechanical protection for coronary blood flow (17).

Further, EAT acts as localized storage of myocardial triglyceride which is critical for mechanical action of the heart (18,19). The triglyceride storage provides the cardiac muscle with free fatty acids and purifies extra blood fatty acids and thus produces cytokines (20). The rates of fatty acid participation in EAT and its decomposition are significantly higher in EAT compared to the other fats. Epicardial fat seems to act as a buffer in order to protect the heart against circulatory fatty acids (21). Conversely, visceral fat is correlated with an increased risk of coronary artery diseases (CAD). Indeed, thickness and volume of EAT increase in cardiovascular patients compared to patients with normal arteries (22). Furthermore, the distribution of body fat is related to the angiogenesis (atherogenesis) while the accumulation of visceral abdominal fat in the white adipose tissue is a risk factor for higher rates of CAD (23). Other studies found that the amount of TFAs is higher in the visceral adipose tissue compared to abdominal fat storage (e.g., subcutaneous or retroperitoneal) which possibly indicates further TFA uptake (24). Different fat storages may be associated with various metabolic risks; visceral abdominal tissue is related to CVDs such as diabetes mellitus, insulin resistance, dyslipidemia, and hypertension as the risk factors (25).

Individuals with similar body mass index (BMI) and excessive accumulation of visceral adipose tissue have a high risk of developing metabolic syndrome compared to those extremely fat individuals with less accumulation of this tissue (26). Some milk fatty acids like butyric, oleic, and conjugated linoleic acids, as well as polyunsaturated fatty acids (PUFAs) are likely to have potential anti-carcinogenic and anti-atherogenic effects. Additionally, fatty acids including several SFAs such as lauric, myristic, and palmitic acids and a number of TFAs may negatively impact health (27). Although higher levels of high-density lipoprotein (HDL) decreases the risk, men with low glyceride levels are less exposed to the risk of developing coronary heart disease (CHD); Contrarily, men with high glyceride levels have a higher risk of developing CHD (28).

Atherosclerosis is accompanied by narrowed and lost elasticity of the artery wall and therefore causes increased blood pressure. The results of epidemiological studies indicated that a vegetable and fruit-rich diet can protect against CAD (29).

The findings of Brouwer et al suggested that the effects of animal and industrial products and TFA on the surface of some human lipoproteins may increase low-density lipoprotein (LDL)/HDL cholesterol ratio irrespective of their origin or structure (30). In addition, Parodi concluded that not all SFAs are regarded as a major risk factor in hypercholesterolemia (31). Vegetable hydrogenated oil, meat, and milk of ruminants are mainly considered the most important sources of TFAs for humans (6). In general, the majority of SFAs are hypercholesterolemia while stearic acid and monounsaturated fatty acids, neutral, or hypercholesterolemia properties, as well as PUFAs are hypocholesterolemia (32). Human serum paraoxonase enzyme is an esterase/lactonase which can hydrolyze a wide range of substrates including organophosphates such as paraoxon and esters (e.g., phenyl phosphates). Paraoxonase activity decreases in CHD (33). Serum paraoxonase-1 is a major protein in relation to HDL, which prevents LDL oxidation and reduces the oxidative stress in blood vessels. Previous research represented that the activity of such an enzyme is conversely associated with the outbreak of CVD (34). Further, plasma short-chain fatty acids are generally detected by several techniques such as enzymatic method (only acetic acid), gas chromatography, liquid chromatography, and gas chromatography-mass spectrometry (35).

Based on the above-mentioned discussions, the current study sought to explore the regional differences between subcutaneous and epicardial fatty acids, the relationship between these measurements, as well as metabolic variables and food. It further attempted to investigate the relationship between paraoxonase enzyme and blood lipids and vascular cramps in patients undergoing coronary artery bypass grafting.

Materials and Methods

Totally, 42 patients (34 males vs. 8 females), who referred to Shahid Madani teaching hospital of Tabriz for coronary artery bypass grafting surgery, were selected and signed the written consent for participation in the study. Then, the blood sample was collected and centrifuged before the surgery and their serum was stored at -70°C until analysis. During the surgery, samples of EAT and subcutaneous adipose tissue (SAT) were obtained from the adjacent epicardial artery and the cervical spine of 1-0.5 g, respectively. Next, all the samples were dissolved

in hexane and stored at -70°C for 3 months until further analysis. Fatty acids were measured using a Buck Scientific gas-chromatography machine (made in the United States) equipped with a flame ionization detector to a precision of 0.01 µg. Furthermore, blood lipids were estimated employing Pars test kits by the enzymatic method through an autoanalyzer. Additionally, the paroxyson activity of PON1 was evaluated with Sigma Chemical substrate. Totally, 20 µL of serum was added to Tris/HCL buffer (100 mmol, pH=8) containing 2mmol of paraoxon and 2 mmol of CaCl2. Then, paraoxon hydrolysis rate was achieved by releasing para nitrofenol at 37°C, and a wavelength of 412 nm was computed using a spectrometer (UV 1250 Shimadzu, Japan). Finally, the activity of the enzyme was calculated with a quenching factor of 18290 mol/L; the enzyme activity unit was expressed in nmol/min/mL serum. Individuals over 65 years old, as well as those with diabetes (i.e., blood glucose greater than 125 mg/dL) and high cholesterol (i.e., above 250 mg/dL) were excluded from the samples of the study. In addition, patients who met the following criteria were excluded from the study:

- Having a history of hospitalization for a cardiac disease more than 5 months before the study;
- Suffering from liver and kidney diseases, diabetes, and endocrine disorders;
- Being older than 65 years.

In addition, the profile of fatty acids and lipid parameters of serum samples were measured by gas chromatography equipment and commercial kits using enzymatic method through an autoanalyzer, respectively. The collected data were arranged, coded, and finally, analyzed by the SPSS software, version 20. Further, the *t* test was utilized for comparing the means of variables in both groups of fatty acids of EAT and SAT. The qualitative parameters of the two groups were assessed and compared employing Chi-square test. Eventually, due to the relationship between some fatty acids and BMI, the Pearson correlation was calculated between blood lipid parameters and the fatty acids of adipose tissue after the adjustment.

Detection of the Peaks of Standard Compositions

First, the standard compositions were separately injected in order to detect the peaks related to fatty acids.

The fatty acids of the adipose tissue samples were analyzed as follows.

t-16:1, t-18:1, t-18:2, total tFA, SFA, MUFA, and PUFA.

Trans Fatty Acids (TFA)

t-16:1 = [16:1-7t +16:1- 9t]
t-18:=[18:1-9t +18:1-10t + 18:1-11t + 18:1-12t]
t-18:2 = [18:2 -9t,12t+ -18:2-9c,12t + 18:2-9t,12c]
Total tFA = [t-16:1+ t-18:1 + t-18:2]
SFA - [12:0 + 14:0 + 16:0 + 18:0]
MUFA - [18:1-11c+ 18:1-9c + 16:1-9c+16:1-7c]
PUFA - [18:3-9c, 12c, 15c + 18:2-9c,12c]

Results

Repeatability of Sample Analysis

At this stage, the repeatability of the percentage measurements of the detected fatty acids was evaluated (36). This stage included assessing the sample preparation and injection procedures. The sample was tested in triplicate per day during three successive days. In this regard, more samples of subcutaneous and epicardial adipose tissues (SAT and EAT) were obtained from a patient. Then, each of these samples was cut into four smaller pieces and put in separate test tubes containing hexane and the procedures of preparation and injection of these samples were conducted in two successive days. BMIs of these patients were high while their lipid profiles were normal and 69% of them consumed solid fats.

Furthermore, fatty acids of EAT and SAT were measured by the gas chromatography method (Table 1).

In EAT, SFAs (SFAs) such as myristic (C14:0), palmitic

Table 1. The Comparison of Pericardial and Subcutaneous Fatty Acids

N=42	Epicardial	Subcutaneous	*P* Value
12:0 (lauric acid)	1.01±0.75	0.90 ±0.69	0.329
14:0 (myristic acid)	2.90±0.98	2.47 ±1.01	0.002
16:0 (palmitic acid)	27.10±5.01	21.44 ±3.58	<0.001
16:1 t (trance palmitoleic acid)	1.38±0.87	1.43 ±0.48	0.735
16:1(palmitoleic acid)	5.10±2.31	7.43 ±3.34	<0.001
18:0 (stearic acid)	4.49±1.69	2.40 ±1.26	<0.001
18:1t (trance oleic acid)	4.58±1.07	4.65 ±0.98	0.702
18:1 n-9 (oleic acid)	34.14±6.44	38.15 ±5.72	0.002
18:1 n-11 (oleic acid)	2.65±1.49	2.46 ±1.32	0.452
18:2 t (trance linoleic acid)	1.58±0.79	2.01 ±0.91	0.004
18:2 n-6 (linoleic acid)	11.99±2.94	12.99±3.60	0.032
18:3 n-9 (linolenic acid)	0.58±0.23	0.67 ±0.29	0.041
CLA (conjugated linoleic acid)	0.73±0.41	0.72 ±0.28	0.769
20:4 n-6 (arachidonic acid)	0.60±0.39	0.59 ±0.31	0.887
20:5 n-3 (eicosapentaenoic acid)	0.15±0.13	0.16 ±0.12	0.532
22:6 n-3 (docosahexaenoic acid)	0.18±0.16	0.18 ±0.15	0.103

CLA: Conjugated linoleic acid; Docozahexaenoic acid; Data are presented as Mean± SD.

(C16:0), and stearic (C18:0) acids were higher compared to the SAT. However, unsaturated fatty acids including palmitoleic (C16:1), oleic (C18:1 ω9), linoleic (C18:2ω6), and linolenic (C18:3 ω3) acids were lower in relation to the SAT. Based on the results, oleic, palmitic, and linoleic fatty acids were the highest amounts of fatty acids in both tissues, respectively.

The percentages of the fatty acids of epicardial and subcutaneous peripheral adipose tissues in CHD patients, as well as the difference in fatty acid contents between the two adipose tissues are illustrated in Figure 1. The statistical comparison demonstrates that the total SFA is higher in EAT ($P<0.05$) while MUFAs ($P<0.01$), along with ω6 and ω3 PUFAs ($P<0.01$) are fewer compared to the SAT. Additionally, the content of TFA is identical in both tissues.

Based on the results, there was no relationship between fatty acids and age while family history had a negative relationship with 12:0 fatty acid ($P<0.05$). In addition, no relationship was observed between smoking and subcutaneous and epicardial fatty acids. Further, hydrogenated oil intake and hydrogenated vegetable oil represented no significant association with subcutaneous and epicardial fatty acids ($P<0.05$). However, the BMI index demonstrated a reverse and significant relationship with SFAs of the SAT. Eventually, family history had a reverse relationship with subcutaneous lauric acid 12:0 ($P<0.05$).

Furthermore, there was a negative relationship between subcutaneous 22:6 n-3 fatty acid and the protein (i.e., meats and eggs) group whereas a positive significant association between subcutaneous 18:3 n-9 fatty acid and fruit and vegetable groups ($P<0.05$). Additionally, a positive significant relationship was found between subcutaneous 14:0 fatty acid and nuts group, as well as between subcutaneous 18:1t fatty acid and confections group ($P<0.05$), The details related to the correlation between fatty acids of adipose tissue and various food groups are provided in Table 2.As regards the epicardial fatty acids, a statistically positive and significant relationship was observed between epicardial 12:0 fatty acid and protein, along with confection and nut groups ($P<0.05$). However, the relationship between epicardial 18:2t fatty acid and grain group was significant and negative ($P<0.01$). Conversely, epicardial CLA fatty acid demonstrated a positive relationship with fruit and vegetable group ($P<0.05$). Finally, there was no significant relationship between subcutaneous and epicardial fatty acids and dairy and drink groups (Table 2).

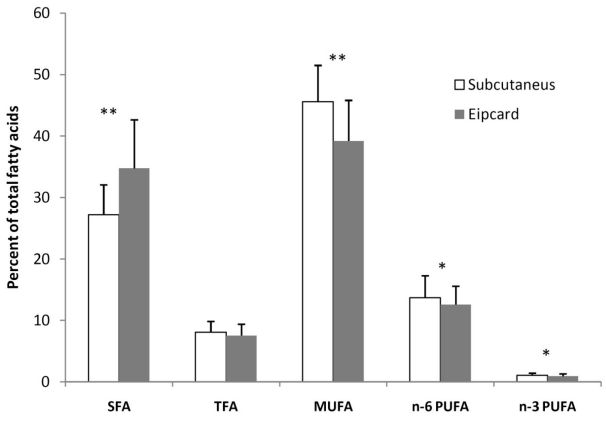

Figure 1. Mean ±SD of Fatty Acid Content Related to Epicardial and Subcutaneous Peripheral Adipose Tissues (%) in 42 Patients With Coronary Heart Disease.
Note. SFA: Saturated fatty acids; TFA: Trans-fatty acids; MUFA: Monounsaturated fatty acids; PUFA: PUFAs; *P<0.05; **P<0.01

The relationship between adipose tissue fatty acids, lipid levels, and hypertension was calculated after BMI adjustment. Statistically, these indices had no significant relationship with the contents of epicardial fatty acids ($P>0.05$). In addition, the activity of paraoxonase enzyme was examined in patients with 1, 2, and 3 clogged arteries. The obtained results indicated that the enzyme activity reduces with an increase in the number of clogged arteries (Table 3).

Discussion

Generally speaking, the results represented that the EAT in CAD patients contains higher amounts of SFA whereas lower amounts of PUFA and MUFA. Further, the levels of unsaturated ω3 and ω6 fatty acids were lower in the epicardial compared to the SAT. Furthermore, the percentage of ω3 fatty acids was extremely lower compared to ω6. According to Harris, ω3 fats are simple, safe, and cheap and reduce the risk of CHDs (37).

Therefore, a diet rich in ω-3 unsaturated fatty acids, in particular, long-chain fatty acids of eicosapentaenoic (C20:5 n-3, EPA) and docosahexaenoic (C22:6 n-3, DHA) acids derived from minerals or fish oils reduces CVDs in humans; additionally, it is beneficially effective in decreasing blood pressure and preventing the progression of atherosclerosis, decreased triglycerides, and de novo lipogenesis (38).

Based on the results of the present study, the relationship between SFAs and BMI was negative while MUFAs had a significant positive relationship with BMI. In addition, Growe et al found a positive relationship between age and 20:5 n-3 and 22:6 n-3 fatty acids in male and female participants after matching BMI, genetics, and smoking, which is in line with the findings of the current study (39).

Further, hypertension was found to have a positive relationship with subcutaneous 16:1 n-7 (palmitoleic acid) and epicardial 18:1 n-11 (oleic acid) whereas a negative association with 16:1 n-7 (palmitoleic acid) and 18:2 n-11 ($P<0.05$). Furthermore, there existed a negative relationship between hypertension and epicardial 18:2 n-6 (linoleic acid) ($P<0.01$).

HDL-C, among the fat parameters, represented a positive relationship with myristic acid (14:0), as well as palmitoleic acid (16:1 n-7). However, a negative

Table 2. Correlation Between the Fatty Acids of Adipose Tissue and Various Food Groups

	Milk & Dairy	Meat & Eggs	Cereals	Fruits & Vegetables	Nuts	Sugar Content	Drinking & Miscellaneous
Subcutaneous							
12:0 (lauric acid)	-0.107	-0.051	-0.011	0.172	0.003	0.169	0.126
14:0 (myristic acid)	-0.077	0.078	-0.067	0.004	0.316[b]	0.223	-0.140
16:0 (palmitic acid)	0.020	0.110	-0.112	0.028	0.249	0.179	0.378
16:1t (trance palmitoleic acid)	-0.072	-0.215	0.064	-0.117	-0.046	0.179	0.177
16:1n-7(palmitoleic acid)	-0.123	-0.006	-0.101	-0.176	0.102	-0.011	0.106
18:0 (stearic acid)	0.146	-0.234	0.033	0.158	-0.096	0.074	0.210
18:1t (trance oleic acid)	0.054	0.031	-0.096	-0.002	0.159	0.455[b]	0.052
18:1n-9 (oleic acid)	0.211	-0.191	-0.245	0.269	-0.030	-0.160	0.172
18:1n-11 (oleic acid)	-0.167	-0.246	-0.065	-0.179	0.284	0.158	-0.105
18:2t (trance linoleic acid)	0.077	-0.260	-0.132	-0.042	-0.058	-0.147	0.182
18:2n-6 (linoleic acid)	-0.096	-0.214	0.053	0.007	-0.057	-0.057	0.132
18:3n-9 (linolenic acid)	-0.160	0.108	0.185	0.346b	0.095	0.105	0.229
CLA (conjugated linoleic acid)	-0.278	-0.127	0.082	-0.073	0.166	0.042	0.004
20:4n-6 (arachidonic acid)	-0.077	-0.164	0.051	-0.135	0.007	-0.016	-0.076
20:5n-3 (eicosapentaenoic acid)	-0.082	-0.055	-0.085	-0.112	-0.098	-0.110	-0.107
22:6n-3 (docosahexaenoic acid)	-0.071	-0.338b	0.212	0.229	0.022	-0.194	0.136
Epicardial							
12:0 (lauric acid)	-0.128	0.358[b]	-0.129	-0.163	0.390[b]	0.529[c]	-0.181
14:0 (myristic acid)	0.065	0.126	0.010	-0.103	0.141	0.180	0.256
16:0 (palmitic acid)	-0.089	0.055	-0.022	-0.073	0.012	-0.229	-0.230
16:1t (trance palmitoleic acid)	-0.060	0.239	-0.127	-0.076	0.227	0.112	-0.244
16:1n-7(palmitoleic acid)	0.161	0.094	-0.102	-0.190	-0.022	0.152	0.195
18:0 (stearic acid)	-0.148	-0.046	-0.040	0.109	0.253	0.144	0.106
18:1t (trance oleic acid)	-0.167	0.032	0.062	-0.239	0.139	0.146	0.027
18:1n-9 (oleic acid)	0.168	-0.185	0.142	0.200	-0.203	-0.116	0.204
18:1n-11 (oleic acid)	-0.075	0.236	-0.145	-0.221	0.109	0.118	0.177
18:2t (trance linoleic acid)	0.045	-0.082	-0.490[c]	-0.011	0.055	0.206	0.139
18:2n-6 (linoleic acid)	-0.123	-0.065	0.128	0.067	-0.085	-0.035	-0.049
18:3n-9 (linolenic acid)	-0.197	-0.132	-0.247	0.078	0.284	0.295	0.276
CLA (conjugated linoleic acid)	-0.038	-0.083	0.008	0.333[b]	0.127	-0.011	0.182
20:4n-6 (arachidonic acid)	0.009	-0.101	-0.067	0.056	-0.039	-0.159	-0.165
20:5n-3 (eicosapentaenoic acid)	-0.105	0.033	-0.181	0.145	-0.029	-0.077	-0.152
22:6n-3 (docosahexaenoic acid)	-0.062	-0.044	-0.081	0.174	0.163	0.101	0.073

[a] Partial Pearaon's correlation (r) is adjusted for food groups. [b] $P<0.05$; [c] $P<0.01$.

Table 3. The Activity of Paraoxonase Enzyme and Lipid Profiles in Patients With Vascular Cramps With 1, 2, and 3 Veins

Blocked Vessels	Paraoxon Activity (nmol/min/mL)	Sinus Blood Cholesterol (mg/dL)	Sinus Blood TG (mg/dL)	LDL Sinus Blood (mg/dL)
1	93.16±17.42	133.83±47.56	91.83 ±41.18	77.33±39.15
2	81.16±17.19	139.37±46.06	102.31±53.99	77.62±44.71
3	71.39±20.57	133.81±31.41	101.25±53.37	79.12±37.50
P-value	0.157	0.917	0.911	0.993

TG: Triglyceride; LDL: Low-density lipoprotein; $P<0.05$.

relationship was detected between this parameter and subcutaneous linoleic acid (18:2 n-6), in addition to epicardial linoleic (18:2 n-6) and arachidonic (20:4 n-6) acids. Conversely, oleic acid (18:1 n-9) of the SAT had a positive relationship with total cholesterol ($P<0.045$) and low-density lipoprotein (LDL) cholesterol ($P<0.01$).

Additionally, SAT n6-PUFA had a significant positive relationship with cholesterol level and blood LDL-C; in addition, myristic acid was found to have a positive relationship with blood HDL-c. Contrarily, there was a negative relationship between linoleic acid and blood HDL-C level. Linoleic acid is the principal n6-PUFA source of diet and is amply found in seeds, nuts, and plant oils. It is strongly emphasized that replacing SFA

with PUFA reduces CHD and possibly prevents diabetes (40). Mozaffarian and Wallace indicated that using PUFAs instead of SFAs decrease CHD in randomized controlled trials. Based on the results of the above-mentioned, study, the tendency toward utilizing more PUFA may reduce the risk of CHD (41). Nutrition with unsaturated fatty acids with a double bond in humans reduces LDL without decreasing the HDL compared to primates and provides the lowest LDL to HDL ratio when compared to saturated and unsaturated fats. Long chain fatty acids (18: 0) were demonstrated to have no effect on LDL, HDL, or total cholesterol/HDL (TC/HDL) ratios, whereas short chain fatty acids including 12: 0, 14: 0, and 16: 0 were reported to have a greater effect on LDL elevation (42).

Regarding the SAT, the results indicated that HDL cholesterol level was positively related to saturated 14:0 fatty acid (myristic acid) while it was negatively related to unsaturated 18:2 n-6 fatty acid (linoleic acid). In addition, EAT represented a positive relationship with 16:1 n-7 fatty acid (palmitoleic acid) whereas a negative relationship with 18:2 n-6 and 20:4 n-6 (arachidonic acid) fatty acids.

Further, LDL cholesterol was negatively associated with subcutaneous unsaturated 18:1 n-9 fatty acid.

The results of the present study revealed that oleic and linoleic acids had a negative relationship with blood pressure; conversely, oleic acid had a positive relationship with blood pressure in EAT. However, no significant relationship was found between 16:0 fatty acid of the adipose tissue and lipid profile in the current study. This may be related to fast synthesis of 16:0 in high carbohydrate intake instead of the plasma cholesterol concentration. Furthermore, smoking demonstrated no association with subcutaneous and epicardial fatty acids. Additionally, family history had a negative relationship with 12:0 lauric acid (subcutaneous). In addition, the relationship between hydrogenated oil intake and subcutaneous and fatty acids was negligible.

However, hypertension was positively associated with subcutaneous and epicardial 16:1 n-7 (palmitoleic acid) while negatively correlated with epicardial and subcutaneous 18:1 n-9 (oleic acid) and epicardial 18:2 n-6 (linoleic acid) tissues. Total cholesterol represented a positive relationship with subcutaneous 18:1 n-9 (oleic acid) whereas no relationship with epicardial fatty acids. Further, no relationship was observed between triglyceride and subcutaneous and epicardial fatty acids. Conversely, HDL-c was positively correlated with subcutaneous14:0 (myristic acid) and epicardial 16:1 n-7 (palmitoleic acid) while negatively associated with subcutaneous 18:2 n-6 (linoleic acid) and epicardial 20:4 n-6 (arachidonic acid). Based on the results, LDL-c was positively related to subcutaneous 18:1 n-9 (oleic acid) and 18:2 n-6 (linoleic acid); however, no relationship was found between LDL-C and EAT. Furthermore, TG/HDL-c indicated no relationship with subcutaneous fat while it was negatively related to epicardial 14:0 (myristic acid).

Additionally, the results revealed that paraoxonase enzyme activity reduced with the increased number of clogged arteries. This is in agreement with the findings of Graner et al (43). However, Rahmani et al found no significant relationship between paraoxonase activity and CAD (44).

Conclusions

In general, there was a relationship between paraoxonase enzyme and the degree of artery clog. In addition, the amounts of epicardial and subcutaneous fatty acids were found to vary in CAD patients. Further, the amount of SFAs was higher in the EAT compared to the SAT. Eventually, a positive relationship was observed between the levels of fatty acids and lipid profiles and foods.

Limitations of the Study

The current study focused on patients with CADs and therefore, it may not represent the general population. Furthermore, hypertension and lower level of lipid are considered independent factors which can modify cardiovascular risk factors. Therefore, caution should be taken when interpreting the absence of significant relationships between clinical risk factors and the fatty acids of the adipose tissue. Accordingly, patients with diagnosed diabetes or hypercholesterolemia were excluded to keep the possible effects of drug therapies to a minimum.

Ethical Issues

This study was approved by the Ethics Committee of Tabriz University of Medical Sciences under the ethical code of TBZMED. REC.86.2-7.7.

Conflict of Interests

The authors have no conflicts of interest.

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