

Smoking may be a cause of hypertriglyceridemia

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Abstract

Background: We tried to understand whether or not there is a significant relationship between smoking and plasma triglycerides values.

Methods: Patients with plasma values of triglycerides lower than 100 mg/dL were collected into the first, lower than 150 mg/dL into the second, lower than 200 mg/dL into the third, and 200 mg/dL or higher into the fourth groups, respectively.

Results: The study included 457 cases (266 females), totally. The female ratio was decreased from the first towards the fourth groups (64.1% versus 49.4%, $p < 0.01$), gradually, whereas the mean ages of the groups, body mass index (BMI), and low density lipoproteins increased just up to the plasma triglycerides value of 200 mg/dL, significantly ($p < 0.05$ for all). On the other hand, the mean fasting plasma glucose and prevalence of white coat hypertension, hypertension, diabetes mellitus, and chronic obstructive pulmonary disease increased parallel to the plasma triglycerides values from the first towards the fourth groups, gradually. As one of the most surprising results, prevalence of smoking also increased parallel to the plasma values of triglycerides from the first towards the fourth groups, gradually (16.3% versus 42.5%, $p < 0.001$).

Conclusions: Plasma triglycerides may actually be some acute phase reactants indicating the disseminated endothelial damage, inflammation, fibrosis, and eventual atherosclerosis all over the body. There may be highly significant relationships between plasma triglycerides values and aging, BMI, and smoking up to the plasma triglycerides value of 200 mg/dL, but smoking may be much more important for plasma triglycerides values of 200 mg/dL or greater.

Key words: Smoking, triglycerides, acute phase reactant, chronic endothelial damage, accelerated atherosclerosis

Introduction

Chronic endothelial damage may be the most common kind of vasculitis and the leading cause of early aging and premature death in human beings (1-4). Much higher blood pressure (BP) of the afferent vasculature may be the major underlying cause by inducing recurrent injuries on endothelium, and probably whole afferent vasculature including capillaries are mainly involved in the process. Therefore the term of venosclerosis is not as famous as atherosclerosis in the literature. Secondary to the chronic endothelial damage, inflammation, edema, and fibrosis, vascular walls thicken, their lumens narrow, and they lose their elastic nature which reduces blood supply to terminal organs and increases systolic BP further. Some of the well-known components of the inflammatory process are sedentary life style, animal-rich diet, overweight, smoking, alcohol, hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, impaired fasting glucose, impaired glucose tolerance, white coat hypertension (WCH), and chronic inflammatory processes including rheumatologic disorders, chronic infections and cancers for the development of irreversible consequences including obesity, hypertension (HT), diabetes mellitus (DM), cirrhosis, peripheral artery disease (PAD), chronic obstructive pulmonary disease (COPD), chronic renal disease (CRD), coronary heart disease (CHD), mesenteric ischemia, osteoporosis, and stroke (5-9). Although early withdrawal of the underlying causes may prevent terminal consequences, after development of cirrhosis, COPD, CRD, CHD, PAD, or stroke, endothelial changes cannot be reversed completely due to their fibrotic nature. The underlying causes and terminal consequences were researched under the titles of metabolic syndrome, aging syndrome, or accelerated endothelial damage syndrome in the literature, extensively (10-13). Although its normal limits have not been determined clearly yet, higher plasma triglycerides values may be significant indicators of the metabolic syndrome (14). Due to the growing evidence about the strong association between higher plasma triglycerides and prevalence of CHD, Adult Treatment Panel (ATP) III adopts lower cutoff points for triglycerides abnormalities than did ATP II (15, 16). Although ATP II determined the normal triglycerides value as lower than 200 mg/dL in 1994, the World Health Organisation in 1999 (17) and ATP III in 2001 reduced its normal limit as lower than 150 mg/dL (15). Although these cutoff points are usually used to define limits of the metabolic syndrome, there are suspicions about the safest limits of plasma triglycerides values in the literature. Beside that, smoking may be found among one of the most common causes of vasculitis all over the world. It is a major risk factor for the development of atherosclerotic endpoints including CHD, PAD, COPD, cirrhosis, CRD, and stroke (18, 19). We tried to understand whether or not there is a significant relationship between smoking and plasma triglycerides values in the present study.

Material and Methods

The study was performed in the Internal Medicine Polyclinic of the Dumlupinar University between August 2005 and March 2007. Consecutive patients above the age of 15 years were studied. Their medical histories including HT, DM, COPD, and already used medications were learnt, and a routine check up procedure including fasting plasma glucose (FPG), triglycerides, and low density lipoproteins (LDL) was performed. Current daily smokers with six pack-months and cases with a history of three pack-years were accepted as smokers. Patients with devastating illnesses including type 1 DM, malignancies, acute or chronic renal failure, chronic liver diseases, hyper- or hypothyroidism, and heart failure were excluded to avoid their possible effects on weight. Additionally, anti-hyperlipidemic drugs, metformin and/or acarbose users were excluded to avoid their possible effects on blood lipid profiles and/or weight (20, 21). Body mass index (BMI) of each case was calculated by the measurements of the Same Physician instead of verbal expressions. Weight in kilograms is divided by height in meters squared (15). Cases with an overnight FPG level of 126 mg/dL or greater on two occasions or already using antidiabetic medications were defined as diabetics (15). An oral glucose tolerance test with 75-gram glucose was performed in cases with a FPG level between 110 and 126 mg/dL, and diagnosis of cases with a 2-hour plasma glucose level of 200 mg/dL or greater is DM (15). Additionally, office blood pressure (OBP) was checked after a 5-minute rest in seated position with a mercury sphygmomanometer on three visits, and no smoking was permitted during the previous 2 hours. A 10-day twice daily measurement of blood pressure at home (HBP) was obtained in all cases, even in normotensives in the office due to the risk of masked HT after a 10-minute education session about proper BP measurement techniques (22). An additional 24-hour ambulatory blood pressure monitoring was not required due to its similar effectivity with the HBP measurements (3). Eventually, HT is defined as a mean BP of 135/85 mmHg or greater on HBP measurements, and WCH as an OBP of 140/90 mmHg or greater but a mean HBP measurement of lower than 135/85 mmHg (22). The spirometric pulmonary function tests were performed in required cases and the criterion for diagnosis of COPD is post-bronchodilator forced expiratory volume in one second/forced vital capacity of less than 70% (23). Eventually, patients with plasma triglycerides values of lower than 100 mg/dL were collected into the first, lower than 150 mg/dL into the second, lower than 200 mg/dL into the third, and 200 mg/dL or higher into the fourth groups, respectively. The female ratio, mean age, BMI, FPG, triglycerides, and LDL, and prevalence of smoking, WCH, HT, DM, and COPD were detected in each group and compared in between. Mann-Whitney U test, Independent-Samples T test, and comparison of proportions were used as the methods of statistical analyses.

Results

The study included 457 cases (266 females and 191 males), totally. The female ratio was decreased from the first towards the fourth groups (64.1% versus 49.4%, $p < 0.01$), gradually. Whereas the mean ages of the groups, BMI, and LDL increased just up to the plasma triglycerides value of 200 mg/dL, significantly ($p < 0.05$ for all). On the other hand, the mean FPG and prevalence of WCH, HT, DM, and COPD increased parallel to the plasma triglycerides values from the first up to the fourth groups, gradually. As one of the most surprising results, prevalence of smoking also increased parallel to the plasma values of triglycerides from the first towards the fourth groups, gradually (16.3% versus 42.5%, $p < 0.001$) (Table 1).

Table 1: Characteristic features of the study cases according to plasma values of triglycerides

Variable	Lower than 100 mg/dL	p-value	Lower than 150 mg/dL	p-value	Lower than 200 mg/dL	p-value	200 mg/dL or higher
Number	159		133		78		87
Mean age	<u>40.6 ± 17.6</u> (16-83)	<u>0.001</u>	<u>46.9 ± 15.9</u> (16-82)	<u>0.014</u>	<u>51.7 ± 11.8</u> (23-73)	Ns*	50.5 ± 12.3 (21-86)
Female ratio	<u>64.1%</u>	Ns	57.8%	Ns	56.4%	Ns	<u>49.4%</u>
Prevalence of smoking	<u>16.3%</u>	<u>0.05 ></u>	<u>23.3%</u>	Ns	<u>28.2%</u>	<u>0.01 ></u>	<u>42.5%</u>
Mean BMI†	<u>26.7 ± 5.6</u> (16.7-49.3)	<u>0.000</u>	<u>29.5 ± 6.0</u> (18.4-50.5)	Ns	30.0 ± 4.9 (19.2-49.0)	Ns	29.7 ± 4.7 (21.0-42.9)
Mean value of FPG‡	<u>102.7 ± 40.3</u> (59-341)	Ns	102.7 ± 26.6 (71-244)	<u>0.009</u>	<u>114.6 ± 43.6</u> (68-320)	Ns	<u>117.1 ± 42.1</u> (80-287)
Mean value of triglycerides	<u>70.3 ± 16.4</u> (27-99)	<u>0.000</u>	<u>120.8 ± 14.8</u> (100-149)	<u>0.000</u>	<u>174.6 ± 14.9</u> (150-199)	<u>0.000</u>	<u>304.8 ± 118.7</u> (200-1.144)
Mean value of LDL§	<u>109.7 ± 33.7</u> (43-269)	<u>0.000</u>	<u>132.1 ± 31.8</u> (64-228)	<u>0.048</u>	<u>140.9 ± 27.7</u> (75-210)	<u>0.009</u>	<u>128.2 ± 39.8</u> (10-239)
Prevalence of WCH	<u>23.2%</u>	<u>0.05 ></u>	<u>30.8%</u>	Ns	32.0%	Ns	<u>34.4%</u>
Prevalence of HT**	<u>11.9%</u>	<u>0.001</u> ≥	<u>23.3%</u>	Ns	25.6%	Ns	<u>25.2%</u>
Prevalence of DM***	<u>8.1%</u>	Ns	12.7%	Ns	<u>16.6%</u>	Ns	<u>22.9%</u>
Prevalence of COPD****	<u>9.4%</u>	Ns	11.2%	Ns	<u>15.3%</u>	<u>0.001</u> ≥	<u>28.7%</u>

*Nonsignificant ($p > 0.05$)

†Body mass index

‡Fasting plasma glucose

§Low density lipoproteins

|| White coat hypertension

**Hypertension

***Diabetes mellitus

****Chronic obstructive pulmonary disease

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