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Effects of Cigarette Smoking on Postprandial Triglyceride in Healthy Smokers

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Abstract

Background: Recent studies revealed that smoking causes metabolic syndrome and insulin resistance which are characterized by increased postprandial triglyceride in smokers compared with nonsmoker people. This study aimed to evaluate the effects of smoking on postprandial triglyceride in healthy smokers.
Methods: In a case-control study 78 participants aged 30-60 years (35 female and 43 male) who referred to the clinics of Khatam-Al-Anbia Hospital, Zahedan and had normal lipid levels and fasting blood sugar were recruited. Of 78 total subjects 39 were smokers and the remaining were non-smokers. Their body mass indices were between 19 to 29.9 kg/m2. Each of these patients consumed 60 g butter (containing 716 kcal energy per 100 grams and 81.06% fat). Then blood triglyceride levels were evaluated one and six hours after consumption. The results were statistically analyzed using T- test and ANOVA.
Results: Triglyceride levels among men and women showed no significant differences (P = 0.403). In both smokers and non-smokers, fasting triglyceride levels were not significantly different, but was significantly different one and six hours after butter consumption (P <0.001). Furthermore, triglyceride levels significantly increased from fasting than one hour and six hours after the butter consumption between smokers and non-smokers (P <0.001.(
Conclusion: In this study it was shown that in smokers postprandial triglyceride levels increased more than non-smokers, triglycerides after a meal remained longer in blood and triglyceride clearance delayed as well. The increase in triglyceride levels after a meal and smoking may consider as an important factor in the development of cardiovascular diseases.

Keywords: Cigarette smoking, Postprandial triglyceride, Coronary artery disease

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Introduction

The risk of cardiovascular diseases increases in smokers, which partly can be justified by changes in fasting lipoprotein levels. Recent studies showed that smokers are susceptible for metabolic disorders characterized by insulin resistance, which increases postprandial triglyceride and followed by increased small dense HDL and LDL (1). In a study, the effect of smoking on lipid profile after a meal containing fat (63 grams of fat with 837 kcal energy) on 12 healthy male and 12 smokers with the same fasting lipid profile, body mass index and lifestyle, were evaluated in 3, 4 and 8 hours after blood sampling. In the smoker group explicit increase in postprandial triglyceride, chylomicrons, and VLDL had observed. Apo-protein B-100 and lipolytic enzymes after a meal were similar between the two groups. Therefore, smokers have been subjected to the Impaired TG tolerance syndrome due to impaired chylomicrons clearance and its remnants (1). A preliminary study showed that increased triglyceride after meals in the smokers was 50% more prevalent than non-smokers, while both groups had normal fasting triglyceride (2). In patients with metabolic syndrome as well hypertension and hypertriglyceridemia, postprandial is more than normal people and even metabolic syndrome itself (3). Recent studies have suggested more noticeable role of increased postprandial lipids as a predictor and risk factor for cardiovascular diseases than fasting lipid (4). In the case of hypertriglyceridemia, large amounts of triglyceriderich lipoprotein produce and hydrolyze into smaller particles on the vascular surface before entering intima. These small particles are the postprandial chylomicron residues and considered as the most atherogenic lipid particles (5,6). The postprandial triglyceri-derich lipoprotein due to increase in the activity of activated factor VIII and decrease in PAI (plasminogen activating inhibitor) blood levels are thrombogenic (7). Studies have shown that older healthy people longer suffer from increased postprandial triglyceride after having a fatty meal than younger, while the total cholesterol in both groups showed no significant difference. This indicates the effective role of lipid metabolic disorder in development of atherogenesis (8, 9). Daily changes in triglycerides in normal healthy people are related to insulin sensitivity, body mass index and diet. In routine laboratory assessments usually fasting triglyceride measures but not triglyceride which produces during the postprandial time (11, 10). The transient increase in postprandial triglyceride levels in healthy people with normal lipid profile increase the large VLDL particles with structural changes that make those ready to absorb by cells especially fibroblasts (12). The postprandial hyperlipidemia and insulin resistance in normoglycemic people who are the first degree relatives of type 2 diabetic patients have been observed (13). The postprandial triglyceride assessment performs via different methods as the oral fat-loading method which uses oral fat with definite amount calories (usually butter) is the most popular one (14). As there are limited studies in this filed and on the other hand studies related to the postprandial lipid and smoking have not been done in Iran, we aim to conduct a study on the effects of smoking on the postprandial triglyceride levels.

Methods

Setting, Design and study population

In this study case-control study, 93 subjects from those patients who referred to the local clinics of Khatam-Al-Anbia Hospital, Zahedan, Iran, from 2001 to 2002 were randomly selected. Patients aged 30-60 years and included with following criteria: fasting blood sugar ≤ 100 mg/dl, fasting triglyceride <150 mg/dl, body mass index between 18.5 to 29.9 Kg/m^2, having moderate physical activity, no alcohol consumption, no systemic disease and agree to participate. The selected sample was divided to two groups: group 1 smokers and group 2 non-smokers. Group 1 included subjects with a history of smoking at least 10 cigarettes for 10 years. The case and control subjects were matched based on the age and body mass indices. First, blood samples were collected after 12 hours fasting to measure blood sugar and triglyceride. Then, each of these patients consumed 60 grams butter (containing 716 kcal energy per 100 grams and 81.06% fat). Blood triglyceride levels were checked one and six hours after consumption.

Meanwhile, the subjects should not eat food, but smokers in this period were free of smoking.

Statistical analysis

SPSS software was used for analysis. The statistical tests used include T-test as well as ANOVA. P-values less than 0.05 considered as statistically significant.

Results

Of the participants, 15 persons were excluded. Among the remaining 78 cases, 35 were male and 43 female. Out of 78, 39 were smokers and the rest were non-smokers. In female, 23 were non-smokers (59%) and 12 were smokers (30.8%). Age, body mass index, fasting blood sugar and triglyceride levels had no significant difference between two study groups. In group 2, the mean fasting triglyceride levels, one hour and six hours postprandial were 111.82, 140.84, and 159.15 mg/dl, respectively. The mean changes in triglyceride levels one hour postprandial minus fasting were 29.02 mg/dl and six hours postprandial than the fasting was 47.33 and six hours than an hour postprandial was 18.3 mg/dl (Table 1). In group 1, the mean of fasting triglyceride levels was 140.41, one hour postprandial was 186.77, and six hours postprandial was 246.28 mg/dl. The mean changes in triglyceride of one hour postprandial to fasting, of six hours postprandial than fasting and six hours than an hour postprandial were 46.36, 10.57 and 59.51 mg/dl, respectively (Table 1). Both groups showed a statistically significant difference (P<0.001) in the mean triglyceride of fasting, one hour and six hours postprandial (Table 1).

Table 1- Comparing the mean triglyceride levels of fasting, one hour and six hours postprandial

Variables / Groups	Fasting TG*	one hour postprandial TG*	six hours postprandial TG*
1	140.4 ± 11.5	186.7 ± 47.5	246.2 ± 48.5
2	111.8 ± 32.7	140.8 ± 57	159.1 ± 69.9

Group1, Smokers; group 2, Non-smokers; TG: triglyceride
Values are Mean± SD.
*Differences were significant (P<0.05)

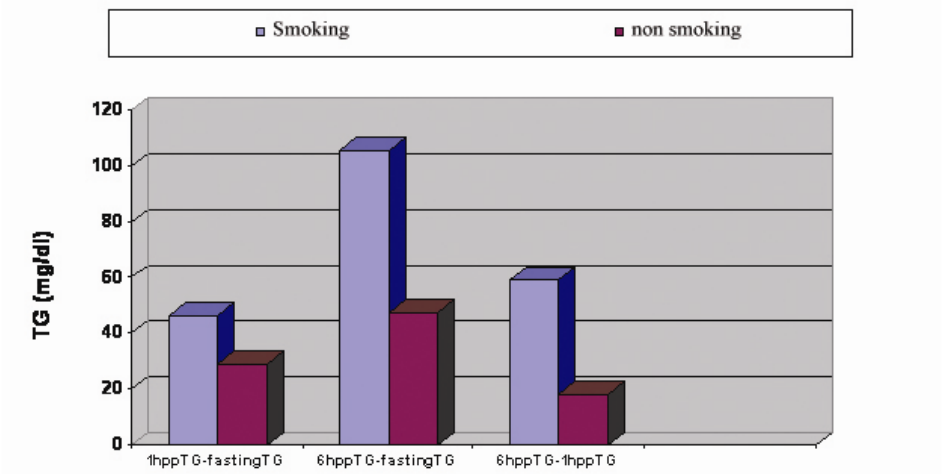

Figure 1- Comparing the mean triglyceride levels of fasting, an hour and six hours postprandial

The mean changes in triglyceride of one hour postprandial to fasting, of six hours postprandial than fasting and six hours than an hour postprandial were illustrated in Figure 1.

Discussion

We were determined a significant difference between non-smokers and smokers in terms of fasting, 1 hour and 6 hours postprandial triglyceride levels (P<0.001). In smokers who had normal fasting triglyceride levels, it's mean value was higher than non-smokers. Furthermore, triglyceride levels in smokers at 1 hour and 6 hours postprandial had a higher increase than in non-smokers. The changes in triglyceride levels six hours than an hour postprandial was also significant (P<0.001) in smokers, which means that six hours

postprandial increased triglyceride levels were higher than an hour comparing to non-smokers.

Eliasson *et al.* (15) observed insulin tolerance syndrome and postprandial lipid intolerance in smokers as well as they showed higher levels of triglyceride and lower HDL in smokers due to impaired postprandial lipid clearance. They also observed the postprandial lipid intolerance coexisting with normal fasting lipid levels, which was close to our study. In the recent study that was conducted by Nordestgaard and colleagues, they showed that postprandial hyperlipidemia is associated with cardiovascular disease risk (4).

The present study could be considered a base for more extensive studies to evaluate various aspects of postprandial hyperlipidemia in individuals with different life habits. It also may leads to establish the lipid tolerance test as a future standard and popular test similar to the GTT (glucose tolerance test) for blood sugar.

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References

1. Mero N Syvanne M: Postprandial elevation of ApoB48 containing triglyceride rich particles and retinyl esters in normolipidemic males who smoke. *Arterioscler Thromb Vasc Biol* 1997; 17(10): 2096-102
2. Axelen-M, Eliasson-B. Lipid intolerance in smokers. *J Inter Med* 1995; 237(5):449-55.
3. Kolovou GD, Anagnostopoulou KK, Pavlidis AN, Salpea KD, Iraklianou SA, Tsarpalis K, Damaskos DS, Manolis A, Cokkinos DV. Postprandial lipemia in men with metabolic syndrome, hypertensive and healthy subjects. *Lipid Health Dis* 2005; 30: 21.
4. Nordestgaard BG, Langsted A, Freiberg JJ. Non-fasting hyperlipidemia and cardiovascular disease. *Curr Drug Targets* 2009; 10(4):328-35.
5. Mero N Syvanne M. Postprandial elevation of ApoB48 containing triglyceride rich particles and retinyl esters in normolipemic males who smoke. *Arterioscler Thromb Vasc Biol* 1997; 17(10): 2096-102.
6. Axelen-M; Eliasson-B. Lipid intolerance in smokers. *J .Inter-Med* 1995; 237(5):449-55.
7. Cohn Js et al: Postprandial Lipemia: Emerging evidence for atherogenicity of remnant lipoproteins. *Can J Cardiol* 1998:14 (Suppl B): 18B-27B.
8. Gaziano Jm et al. Triglyceride and coronary risk. *Curr Cardiol Rep* 1999; 1(2):125-30.
9. Cassader M, Gambino R et al. Postprandial triglyceride-rich lipoprotein changes in elderly and young subjects. *Aging (Milano)* 1996; 8(6):421-8.
10. Karpe F, Hellenius Ml, Hamesten A. Difference in postprandial concentration of very-low-density lipoprotein and chylomicron remnants between normotriglyceridemic and hypertriglyceridemic men with and without coronary heart disease. *Metabolism* 1999; 48 (3): 301-7.
11. Van Oostrom Aj, Castro Cabezas M, et al. Diurnal triglyceride profiles in healthy normolipidemic male subjects are associated to insulin sensitivity, body composition and diet. *Eur J Clin Invest* 2000; 30(11):964-71.
12. Bjorkegren J,Karpe F, et al. Transient triglyceridemia in healthy normolipidemic men increase cellular processing of large very low density lipoproteins by fibroblasts in vitro. *J Lipid Res* 1998; 39 (2): 423-36.
13. Mette Axelsen, Ulf Smith, et al. Postprandial hypertriglyceridemia and insulin resistance in normoglycemic First-Degree relatives of patients with type2 diabetes. *Annals of internal medicine* 1999; 131(1): 27-31.
14. Van Oostrom AJ, Alipour A, Sijmonsma TP, Verseyden C, Dallinga-Thie GM, Plokker HW, et al. Comparison of different methods to investigate postprandial lipaemia. *Neth J Med* 2009; 67 (1):13-20.
15. Eliasson B, Mero N. The insulin resistance syndrome and postprandial lipid intolerance in smokers. *Atherosclerosis* 1997; 129(1):79-88.