Iranian Heart Journal; 2018; 19 (1)

Original Article

Relationship Between the Serum Levels of Nonfasting Triglyceride and the Severity of Coronary Artery Disease

Mostafa Ariafar[*1], MD; Fatemeh Dashti[1], MD; Shahla Assadi Hovyzian[2], MS

ABSTRACT

Background: Cardiovascular diseases are a major cause of death in both men and women around the world. Studies have shown that hypertriglyceridemia is an important risk factor for such diseases. While triglyceride levels after a meal increase, the relationship between postprandial triglyceride levels and the severity of coronary artery disease is still unproven.
This study aimed to determine the plasma levels of triglyceride after a meal in patients with different types of coronary heart disease.

Method: In this epidemiological study, 416 patients from among those referred to Golestan Hospital in Ahvaz were selected based on the results of angiography and were classified to type 1 to 4 groups comprising 69, 99, 83, and 165 individuals respectively, and their age and sex were recorded. From all the individuals, a blood sample was taken 2 hours after a meal. The data were analyzed using the Tukey test.

Results: The mean age of the patients was 58.4 years. There was only a significant difference between the patients with type 1 (normal patients) and those with type 4 (with severe coronary artery disease) (142 ± 67.1 mg/dL vs 188 ± 99.3 mg/dL; $P < 0.001$). Furthermore, no significant difference in the mean triglyceride level was observed in both sexes between the different types ($P > 0.05$).

Conclusions: The results showed that the mean triglyceride serum level after a meal in our patients with severe coronary artery disease (type 4) was higher than that in the other groups. Hypertriglyceridemia after a meal may be a major factor in coronary heart disease. ***(Iranian Heart Journal 2018; 19(1):21-29)***

KEYWORDS: Triglyceride, Nonfasting, Coronary artery disease, Angiography.

[1] Ahvaz Atherosclerosis Research Center , Ahvaz Jundishapur University of Medical Sciences, Ahvaz, IR Iran.
[2] Atherosclerosis Research Center, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, IR Iran.

*** Corresponding Author:** Mostafa Ariafar, MD; Golestan Blvd, Ahvaz Atherosclerosis Research Center, Ahvaz, IR Iran.
Email: Armx51@yahoo.com **Tel:** 0912 3224704

Received: August 29, 2017 **Accepted:** December 8, 2017

Cardiovascular diseases are the leading cause of morbidity and mortality in both sexes throughout the world, including Iran. [1] The prevalence of cardiovascular diseases is on the rise as a result of lifestyle changes during recent decades. [2] Risk factors such as obesity, dyslipidemia, hypertension, and smoking are associated with a high incidence rate of cardiovascular diseases. [3] Studies have shown that hypertriglyceridemia is

an important risk factor for cardiovascular diseases, especially coronary artery disease (CAD). [4]

A small cross-sectional study by Albrink and Man [5] reported the association between triglyceride and CAD. The authors showed that hypertriglyceridemia more than hypercholesterolemia was associated with CAD in men. Hypertriglyceridemia in patients with early-onset ischemic heart disease occurs alone more than hypercholesterolemia, and coronary events in patients with fasting triglyceride levels more than 1.13 millimoles per liter (150 mg/dL) increase. [6] Additionally, many studies have proven the role of total cholesterol and low-density lipoprotein cholesterol (LDL-C) in atherosclerosis. Nonetheless, the role of hypertriglyceridemia as an independent agent in predicting ischemic heart disease has yet to be fully elucidated. Typically, triglyceride has been measured in the fasting state, which is the lowest level of triglyceride in the day. In a recent large study conducted on 40 000 individuals, the authors demonstrated that nonfasting triglyceride was associated with an increased risk of cardiovascular diseases. [7, 8]

The nonfasting level is defined as a change in concentration after a meal depending on the extent and duration of the absorption stage. For example, an increase in plasma triglyceride occurs in response to fat intake, which is a dynamic condition with continuous fluctuations in blood lipid and blood sugar throughout the day where continuous restructuring of lipoprotein and other metabolic adaptations compared to the relatively stable conditions of fasting occur. [9] The serum triglyceride level typically remains high for 3 to 6 hours after a meal. [10–12] The evaluation of the response to nonfasting lipid to identify abnormalities in the metabolism of fat may be more important than the level measured in the fasting state. [9] The metabolism of nonfasting triglyceride levels is adjusted by dietary patterns, food composition, conditions associated with lifestyle (physical activity, smoking, and alcohol consumption), physiological factors (age, gender, genetic background, and postmenopausal status), and cardiometabolic conditions such as fasting triglyceride, type 2 diabetes mellitus, resistance to insulin, and obesity. [9] Particularly, triglyceride levels after a meal in blood are significantly higher in men than in women before menopause, while glucose and response to insulin are identical. [13]

Patients afflicted with moderate hyper-triglyceridemia with conditions such as familial hypertriglyceridemia and familial combined hyperlipidemia and metabolic syndrome are often prone to precocious atherosclerosis. [14] Atherosclerosis may be a nonfasting phenomenon whereby lipoprotein plays a dominant role in its onset. [15–17] Consequently, a rise in nonfasting triglyceride levels—which reflects the increase in lipoprotein levels—may predict the risk of myocardial infarction, ischemic heart disease, and death. [8] In addition, in patients who have undergone coronary artery bypass surgery, with the reduction in serum triglyceride levels, a deceleration in the progression of atherosclerosis in normal vascular grafts has been reported. [18, 19] The triglyceride level after meals is directly correlated with the angiographic progression of atherosclerosis of coronary arteries and carotid arteries. [20]

Atherogenic dyslipidemia, including both the occurrence of low high-density lipoprotein (HDL) cholesterol and high triglyceride associated with increased apoprotein and very low low-density lipoprotein (VLDL), is an important component of metabolic syndrome and a strong predictor of cardiovascular diseases. [21–24] The ratio of triglyceride to HDL is allied to high LDL phenotype B, small particles of HDL, and insulin resistance. [25–27] Gaziano et al [28] conducted a case-control study on this subject and showed that this ratio was a strong predictor of heart-attack risk. The ratio of triglyceride to HDL is correlated with coronary artery atherosclerosis, [29, 30] heart rate

recovery after exercise, [31] incidence of coronary heart disease, [32] and cardiovascular diseases.

The National Institutes in Favor of Health Conference evaluated the dominant role of triglyceride in cardiovascular diseases and offered diagnosis and treatment recommendations for countries with a high population of hypertriglyceridemia. [33, 34] In the United States, it was disconcerting that the level of triglyceride up to 1976 in line with the growth of epidemics such as obesity, insulin resistance, and type 2 diabetes mellitus was on the rise. [35, 36] In evaluating the different types of fats, triglyceride is the most problematic in increasing the risk of cardiovascular diseases. A previous investigation demonstrated that with a 20% to 40% reduction in triglyceride levels, the rate of CAD is reduced by 30% to 40%. [37]

Given the clinical significance of nonfasting triglyceride in the severity of CAD and the absence of previous research on this issue in Iran, we designed the present investigation at Ahvaz Jundishapur University of Medical Sciences. We evaluated patients candidated for coronary artery angiography in Golestan Hospital in terms of nonfasting serum and triglyceride levels. Based on the results of angiography, we investigated the relationship between nonfasting triglyceride levels and the severity of CAD.

METHOD

In this epidemiological study, 416 patients with suspected CAD who were referred to the Angiography Ward of Golestan Hospital were selected by convenience sampling. Informed consent was obtained from all the patients, and then demographic information such as age and gender was recorded by referring to the hospital records of the patients. Diabetic patients were identified from the other patients. At the time of angiography, a standard meal containing about 500 calories—including 60% carbohydrates, 20% protein, and 20% fat—was given to the patients. Two hours after the meal, a blood sample was taken from the patients. The samples were prepared with Pars test kits and were measured by using a Photometer-5010 device and a calorimetric device (GPO-PAD) at a wavelength of 546 nm. The patients were categorized based on the severity of their CAD according to the result of their angiography. The angiography results based on the vessel score method and the severity of CAD were divided into 4 general categories: normal patients (Group 1), patients with mild CAD (Group 2), patients with moderate CAD (Group 3), and patients with severe CAD (Group 4). The SPSS statistical software was used for the statistical analyses. The quantitative variables were compared between the groups using the ANOVA test, followed by the χ^2 test. The Tukey test was also employed for the qualitative variables.

RESULTS

The present study evaluated 416 patients with suspected CAD, comprising 234 males and 192 females. The groups were composed of type 1 with 69 individuals (22 males and 47 females at an average age of 50.9 and 53.6 years, respectively), type 2 with 99 patients (51 males and 48 females at a mean age of 56.9 and 59.6 years, respectively), type 3 with 83 patients (40 men at a mean age of 59.3 years and 43 women at a mean age of 62.8 years), and type 4 with 165 patients (111 males and 54 females at a mean age of 57.6 and 63.2 years, correspondingly). The mean age of type 1 to type 4 patients was 52.7, 58.2, 61.1, and 59.5 years—respectively. There was a significant statistical difference between the average age of type 1 patients and the other groups ($P < 0.001$), and the other types did not show significant differences with one another (Table 1).

The mean triglyceride level 2 hours after a meal was 141± 67 mg/dL in the patients with type 1, 161 ± 85 mg/dL in the patients with type 2, 170 ± 73 mg/dL in the patients with type 3, and 170 ± 87 mg/dL in the patients with type 4. In

order to determine the relationship between the triglyceride levels between the different types, we used the Tukey test to calculate the significant difference at 95% confidence interval. There was only a significant difference between type 1 and type 4 ($P < 0.001$). It can be concluded that the mean triglyceride level after a meal in the patients with type 4 was higher than that in the patients with the other types. Furthermore, the significant difference between the patients with type 1 (normal cardiovascular artery disease) and those with type 4 showed an abnormality in the patients with type 4.

The mean nonfasting triglyceride level by sex and in the men and women in the patients with type 1 to type 4 was 133 ± 60.6 mg/dL and 145 ± 70.2 mg/dL in type 1, 145 ± 45.4 mg/dL and 177 ± 111.2 mg/dL in type 2, 175 ± 81.7 mg/dL and 165 ± 65.6 mg/dL in type 3, and 192 ± 104.8 mg/dL and 180 ± 87.1 mg/dL in type 4—respectively (Table 1).

Table 1. Characteristics in the different group types

Group Type	[b] Normal Patients (1)			Patients With Mild CAD(2)			Patients With Moderate CAD (3)			Patients With Severe CAD (4)		
Sex	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
N	22	47	69	51	48	99	40	43	83	111	54	165
Age	50.9±11.1	53.6±9.6	52.7±10.1	56.9±11.2	59.5±11	58.2±11.1	59.3±12.3	62.8±10.9	61.1±11.6	57.6±11.7	63.2±8.5	59.5±11.1*
TG	133±60.6	145±70.2	142±67.1	145±45.4	177±111.2	161±85.1	175±81.7	165±65.6	170±73.5	192±104.8	180±87.1	188±99.3*
HDL	51.3±1.1	57.7±1.5	55.7±1.4	50.3±2.1	56±2.4	53.1±2.2	45±1.3	54±2.2	49.7±1.8	45±1.7	53.4±1.9	47.7±1.8
LDL	145±4	150±6	148.4±5.4	160±3	155±5	157.6±4	180±6	176±5	177.9±5.5	189±4	185±3	187.7±3.7
BMI	24.5±1.5	24.2±1	24.3±1.2	25.1±1.2	25±1	25.1±1.1	26.5±1.4	25.3±2	25.9±1.7	27.1±2.1	26.5±1.8	26.9±2
Chol	250±5	255±5	253.4±5	254±4	272.2±6	262.8±5	270±5	275±6	272.6±5.5	283±6	288±5	284.6±5.7

Group types are divided based on the vessel score method. Values are expressed as means ± SDs.
TG (mg/dL), Triglyceride; HDL (mg/dL), High-density lipoprotein; LDL (mg/dL), Low-density lipoprotein (mg/dL); BMI (kg/m^2), Body mass index; Chol, Cholesterol
Significant difference between the other groups and Group 1 for TG and age in the whole patient population (*P = 0.001)
No significant difference was obtained between the men and women in each group for age and TG ($P > 0.5$).

DISCUSSION

In the current study, the only significant difference in terms of triglyceride levels after meals was observed between Group 1 and Group 4 ($P < 0.001$) and no significant difference was seen between the other groups ($P > 0.001$). Dyslipidemia is one of the most important risk factors for cardiovascular diseases. Plasma triglyceride is produced in the intestines and the liver and a significant portion of it enters the bloodstream by chylomicron. Triglycerides are assembled from the synthesis of fatty acids and lipids and return to the liver in the form of VLDL secretion. Thereafter, they are transformed into free fatty acids, monoglycerides, glycerols, and small particles in chylomicron; most of these debris particles are cleared from the bloodstream by the hepatic LDL receptors. [38] Many of the triglyceride-rich lipoproteins that accumulate in plasma after a meal are actually VLDL, [39] which generally contains modified apolipoprotein and fat compounds. [40] When triglyceride-rich particles in the bloodstream bind to the endothelium of arteries, the existing lipoprotein lipase by attaching to fat particles starts the hydrolysis of the triglycerides and reduces the size of the bonded pieces. Triglyceride hydrolysis by lipoprotein lipase leads to some changes in chylomicrons and VLDL. [41]

Chylomicrons and VLDL remnants exist in the plasma of patients with hypertriglyceridemia; they are smaller than triglyceride-rich lipoproteins and hence permeate into the intima of vessels and seem to be trapped by the vessel wall. [42, 43] Small dense LDL particles are more susceptible to oxidation and impair the endothelium function; as a result, patients with hypertriglyceridemia are assumed to have endothelial dysfunction. [44, 45] High triglyceride levels are associated with a reduction in HDL and small dense LDL in humans. [39]

Stensvold et al [46] concluded that an increase in nonfasting triglyceride was an independent cause of mortality due to coronary heart disease and cardiovascular diseases among Norwegian women. The current study also showed that in patients with severe CAD, postprandial triglyceride levels were higher than postprandial triglyceride levels in patients with normal CAD, which can be considered a risk factor. Teno et al [47] reported a significant relationship between nonfasting hyper-triglyceridemia and the intima layer thickness of the carotid artery and concluded that fasting hypertriglyceridemia could be an independent risk factor for early atherosclerosis even if nonfasting triglyceride was normal. Likewise in the current study, the mean triglyceride level after a meal in Group 4 was higher than that of all the other groups; in addition, the normal individuals (Group 1) had the lowest triglyceride level after a meal (Fig. 1).

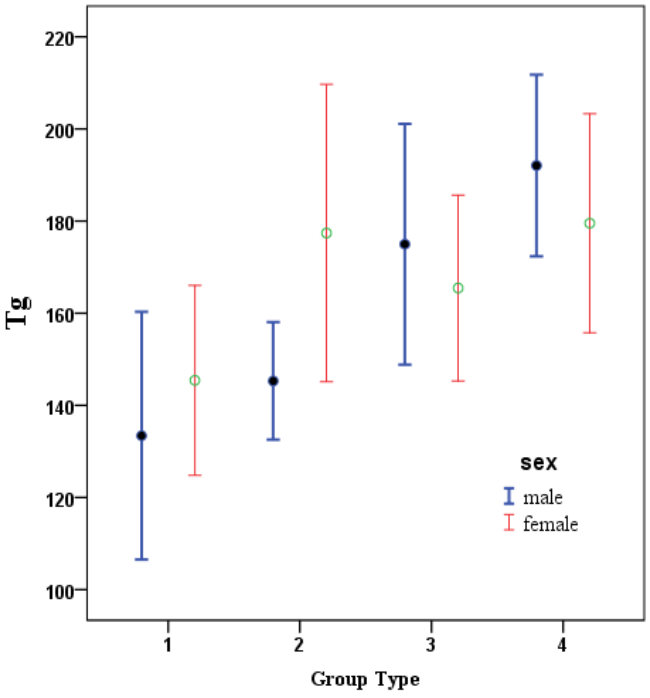

Figure 1. Mean triglyceride levels after meals between the different types

In a previous investigation, the measurement of nonfasting triglyceride levels illustrated that women were at higher risk (the highest compared with the lowest level) than men at risk of acute myocardial infarction; furthermore, nonfasting triglyceride measurement also helped to identify individuals at risk. [48] In the current study, there was no significant relationship between the men and women in the different groups ($P > 0.05$). In the patients with normal CAD and in those with mild CAD, the postprandial triglyceride levels in the women were higher than those in the men, whereas in the patients with moderate

CAD and in those with severe CAD, the triglyceride levels after a meal were higher in the men than in the women. We also found that the women with high levels of triglyceride after a meal were not at high risk for CAD compared to the men; the statistics between the groups were not statistically significant ($P > 0.05$). The results of a study by Afkhami et al [49] showed that nonfasting triglyceride levels in diabetic patients with a history of heart attack were more than those in diabetic patients without a history of myocardial infarction; moreover, nonfasting hypertriglyceridemia was indicated to be an important factor in the development of atherosclerosis. This finding is concordant with the difference shown in the normal group and the group with severe CAD in the current study. High-calorie easy-to-digest high-absorption foods as well as drinks can cause a significant increase in blood sugar and triglyceride after a meal. [50] Recent studies on healthy individuals have demonstrated that a meal with a great deal of saturated fat causes solely an immediate increase in triglyceride levels, oxidative stress, and inflammation—causing endothelial dysfunction, vasoconstriction, and high systolic blood pressure.

CONCLUSIONS

The measured nonfasting triglyceride level was significantly associated with an increased risk of myocardial infarction, ischemic heart disease, and mortality in the men and women. Recently, nonfasting triglyceride in anticipation of an increased risk of CAD as well as fasting triglyceride was considered important. [7, 8] However, in general, in tandem with a rise in fasting triglyceride levels, there is an elevation in nonfasting triglyceride levels. [51, 52] Moreover, nonfasting hypertriglyceridemia predicts a higher level of cholesterol, which is another risk factor for CAD. In addition, high nonfasting triglyceride levels are also associated with the risk of stroke. [53]

In a study by Stampfer et al, [54] nonfasting triglyceride levels were significantly higher in the patients with a higher risk, showing that nonfasting triglyceride levels were a strong and independent risk factor for myocardial infarction. Consequently, the treatment of hyperlipidemia after a meal can prevent atherosclerosis. We, therefore, recommend that longer cohort studies be undertaken to determine the postprandial hypertriglyceridemia level as a risk factor for CAD.

Acknowledgements
There is no acknowledgment.

REFERENCES

1. Sarrafzadegan N, Baghaei A, Sadri G, Kelishadi R, Malekafzali H, Boshtam M, et al. Isfahan healthy heart program: Evaluation of comprehensive, community-based interventions for noncommunicable disease prevention. Prev Control. 2006;2(2):73–84.

2. Azizi F, Rahmani M, Emami H, Mirmiran P, Hajipour R, Madjid M, et al. Cardiovascular risk factors in an Iranian urban population: Tehran lipid and glucose study (phase 1). Sozial-und präventivmedizin. 2002;47(6):408–26.

3. Helfand M, Buckley DI, Freeman M, Fu R, Rogers K, Fleming C, et al. Emerging risk factors for coronary heart disease: a summary of systematic reviews conducted for the US Preventive Services Task Force. Ann Intern Med. 2009;151(7):496–507.

4. Sarwar N, Danesh J, Eiriksdottir G, Sigurdsson G, Wareham N, Bingham S, et al. Triglycerides and the risk of coronary heart disease: 10 158 Incident cases among 262 525 participants in 29 Western prospective studies. Circulation. 2007;115(4):450–8.

5. ALBRINK MJ, Man EB. Serum triglycerides in coronary artery disease. AMA Arch Intern Med. 1959;103(1):4–8.

6. Miller M, Seidler A, Moalemi A, Pearson TA. Normal Triglyceride Levels and Coronary Artery Disease Events: The Baltimore Coronary

Observational Long-Term Study 1. J Am Coll Cardiol. 1998;31(6):1252–7.

7. Bansal S, Buring JE, Rifai N, Mora S, Sacks FM, Ridker PM. Fasting compared with nonfasting triglycerides and risk of cardiovascular events in women. Jama. 2007;298(3):309– 16.

8. Nordestgaard BG, Benn M, Schnohr P, Tybjæ rg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. Jama. 2007;298(3):299–308.

9. Alcala-Diaz JF, Delgado-Lista J, Perez-Martinez P, Garcia-Rios A, Marin C, QuintanaNavarro GM, et al. Hypertriglyceridemia influences the degree of postprandial lipemic response in patients with metabolic syndrome and coronary artery disease: from the CORDIOPREV study. PLoS One. 2014;9(5):e96297.

10. Björkegren J, Karpe F, Milne RW, Hamsten A. Differences in apolipoprotein and lipid composition between human chylomicron remnants and very low density lipoproteins isolated from fasting and postprandial plasma. J Lipid Res. 1998;39(7):1412–20.

11. Karpe F, de Faire U, Mercuri M, Bond MG, Hellénius M-L, Hamsten A. Magnitude of alimentary lipemia is related to intima-media thickness of the common carotid artery in middle-aged men. Atherosclerosis. 1998;141(2):307–14.

12. Boquist S, Ruotolo G, Tang R, Björkegren J, Bond MG, de Faire U, et al. Alimentary lipemia, postprandial triglyceride-rich lipoproteins, and common carotid intima-media thickness in healthy, middle-aged men. Circulation. 1999;100(7):723–8.

13. Horton TJ, Commerford SR, Pagliassotti MJ, Bessesen DH. Postprandial leg uptake of triglyceride is greater in women than in men. Am J Physiol Endocrinol Metab [Internet]. 2002;283(6):E1192–202. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12424104

14. Nordestgaard BG, Benn M, Schnohr P, Tybjæ rg-Hansen A. Nonfasting triglycerides and risk of myocardial infarction, ischemic heart disease, and death in men and women. JAMA - J Am Med Assoc. 2007;298(3):299–308.

15. Kolovou GD, Anagnostopoulou KK, Daskalopoulou SS, Mikhailidis DP, Cokkinos D V. Clinical relevance of postprandial lipaemia. Curr Med Chem. 2005;12(17):1931–45.

16. Kolovou GD, Anagnostopoulou KK, Pavlidis AN, Salpea KD, Iraklianou SA, Tsarpalis K, et al. Postprandial lipemia in men with metabolic syndrome, hypertensives and healthy subjects. Lipids Health Dis. 2005;4(1):1.

17. Zilversmit DB. Atherogenesis: a postprandial phenomenon. Circulation. 1979;60(3):473–85.

18. Ericsson C-G, de Faire U, Grip L, Svane B, Hamsten A, Nilsson J. Angiographic assessment of effects of bezafibrate on progression of coronary artery disease in young male postinfarction patients. Lancet. 1996;347(9005):849–53.

19. Frick MH, Syvänne M, Nieminen MS, Kauma H, Majahalme S, Virtanen V, et al. Prevention of the angiographic progression of coronary and vein-graft atherosclerosis by gemfibrozil after coronary bypass surgery in men with low levels of HDL cholesterol. Circulation. 1997;96(7):2137–43.

20. Steinmetz A. Treatment of diabetic dyslipoproteinemia. Exp Clin Endocrinol diabetes. 2003;111(05):239–45.

21. Assmann G, Schulte H. Relation of high-density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM experience). Am J Cardiol. 1992;70(7):733–7.

22. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. Circulation. 2005;112(17):2735–52.

23. Manninen V, Tenkanen L, Koskinen P, Huttunen JK, Mänttäri M, Heinonen OP, et al. Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki

Heart Study. Implications for treatment. Circulation. 1992;85(1):37–45.

24. Ballantyne CM, Olsson AG, Cook TJ, Mercuri MF, Pedersen TR, Kjekshus J. Influence of low high-density lipoprotein cholesterol and elevated triglyceride on coronary heart disease events and response to simvastatin therapy in 4S. Circulation. 2001;104(25):3046–51.

25. Hanak V, Munoz J, Teague J, Stanley A, Bittner V. Accuracy of the triglyceride to highdensity lipoprotein cholesterol ratio for prediction of the low-density lipoprotein phenotype B. Am J Cardiol. 2004;94(2):219–22.

26. McLaughlin T, Reaven G, Abbasi F, Lamendola C, Saad M, Waters D, et al. Is there a simple way to identify insulin-resistant individuals at increased risk of cardiovascular disease? Am J Cardiol. 2005;96(3):399–404.

27. Jia L, Long S, Fu M, Yan B, Tian Y, Xu Y, et al. Relationship between total cholesterol/ highdensity lipoprotein cholesterol ratio, triglyceride/high-density lipoprotein cholesterol ratio, and high-density lipoprotein subclasses. Metabolism. 2006;55(9):1141–8.

28. Gaziano JM, Hennekens CH, O'Donnell CJ, Breslow JL, Buring JE. Fasting triglycerides, high-density lipoprotein, and risk of myocardial infarction. Circulation. 1997;96(8):2520–5.

29. Frohlich J, Dobiášová M. Fractional esterification rate of cholesterol and ratio of triglycerides to HDL-cholesterol are powerful predictors of positive findings on coronary angiography. Clin Chem. 2003;49(11):1873–80.

30. Drexel H, Aczel S, Marte T, Benzer W, Langer P, Moll W, et al. Is Atherosclerosis in Diabetes and Impaired Fasting Glucose Driven by Elevated LDL Cholesterol or by Decreased HDL Cholesterol? Response to Schulze and Hoffmann. Diabetes Care. 2005;28(5):1264–5.

31. Shishehbor MH, Hoogwerf BJ, Lauer MS. Association of triglyceride–to–HDL cholesterol ratio with heart rate recovery. Diabetes Care. 2004;27(4):936–41.

32. ørgen Jeppesen J, Hein HO, Suadicani P, Gyntelberg F. Triglyceride concentration and ischemic heart disease an eight-year follow-up in the Copenhagen male study. Circulation. 1998;97(11):1029–36.

33. Health NI of. Consensus Conference: Treatment of hypertriglyceridemia. Jama. 1984;251:1196–200.

34. Rapaport E, Bilheimer DW, Chobanian A V, Hajjar DP, Hawkins CM, Hutchins GM, et al. Triglyceride, high-density lipoprotein, and coronary heart disease. Jama. 1993;269(4):505–10.

35. Flegal KM, Carroll MD, Ogden CL, Johnson CL, H T, KM W, et al. Prevalence and Trends in Obesity Among US Adults, 1999-2000. JAMA [Internet]. 2002 Oct 9 [cited 2016 Dec 26];288(14):1723. Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.288.14.1723

36. Carroll MD, Lacher DA, Sorlie PD, Cleeman JI, Gordon DJ, Wolz M, et al. Trends in Serum Lipids and Lipoproteins of Adults, 1960-2002. JAMA [Internet]. 2005 Oct 12 [cited 2016 Dec 26];294(14):1773. Available from: http://jama.jamanetwork.com/article.aspx?doi=10.1001/jama.294.14.1773

37. O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary Strategies for Improving Post-Prandial Glucose, Lipids, Inflammation, and Cardiovascular Health. J Am Coll Cardiol. 2008;51(3):249–55.

38. Goldberg IJ, Eckel RH, McPherson R. Triglycerides and Heart Disease Still a Hypothesis? Arterioscler Thromb Vasc Biol. 2011;31(8):1716–25.

39. Bjorkegren J, Packard CJ, Hamsten A, Bedford D, Caslake M, Foster L, et al. Accumulation of large very low density lipoprotein in plasma during intravenous infusion of a chylomicron-like triglyceride emulsion reflects competition for a common lipolytic pathway. J Lipid Res. 1996;37(1):76–86.

40. Björkegren J, Hamsten A, Milne RW, Karpe F. Alterations of VLDL composition during alimentary lipemia. J Lipid Res. 1997;38(2):301–14.

41. Felts JM, Itakura H, Crane RT. The mechanism of assimilation of constituents of chylomicrons,

very low density lipoproteins and remnants-a new theory. Biochem Biophys Res Commun. 1975;66(4):1467–75.

42. Nordestgaard BG, Wootton R, Lewis B. Selective Retention of VLDL, IDL, and LDL in the Arterial Intima of Genetically Hyperlipidemic Rabbits In Vivo Molecular Size as a Determinant of Fractional Loss From the Intima–Inner Media. Arterioscler Thromb Vasc Biol. 1995;15(4):534–42.

43. Rutledge JC, Mullick AE, Gardner G, Goldberg IJ. Direct Visualization of Lipid Deposition and Reverse Lipid Transport in a Perfused Artery Roles of VLDL and HDL. Circ Res. 2000;86(7):768–73.

44. Anderson TJ, Meredith IT, Charbonneau F, Yeung AC, Frei B, Selwyn AP, et al. Endothelium-dependent coronary vasomotion relates to the susceptibility of LDL to oxidation in humans. Circulation. 1996;93(9):1647–50.

45. Grundy SM. Small LDL, atherogenic dyslipidemia, and the metabolic syndrome. Circulation. 1997;95(1):1–4.

46. Stensvold I, Tverdal A, Urdal P, Graff-Iversen S. Non-fasting serum triglyceride concentration and mortality from coronary heart disease and any cause in middle aged Norwegian women. Bmj. 1993;307(6915):1318–22.

47. Teno S, Uto Y, Nagashima H, Endoh Y, Iwamoto Y, Omori Y, et al. Association of postprandial hypertriglyceridemia and carotid intima-media thickness in patients with type 2 diabetes. Diabetes Care. 2000;23(9):1401–6.

48. Egeland GM, Igland J, Sulo G, Nygård O, Ebbing M, Tell GS. Non-fasting triglycerides predict incident acute myocardial infarction among those with favourable HDL-cholesterol: Cohort Norway. Eur J Prev Cardiol. 2015;22(7):872–81.

49. Afkhami M, Samsami M, Rashidi M. Postprandial triglyceride levels in type 2 diabetic patients with and without history of myocardial infarction. Bimon J Hormozgan Univ Med Sci. 2007;11(3):181–8.

50. O'Keefe JH, Bell DSH. Postprandial hyperglycemia/hyperlipidemia (postprandial dysmetabolism) is a cardiovascular risk factor. Am J Cardiol. 2007;100(5):899–904.

51. Patsch JR, Miesenböck G, Hopferwieser T, Mühlberger V, Knapp E, Dunn JK, et al. Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state. Arterioscler Thromb Vasc Biol. 1992;12(11):1336–45.

52. Marcoux C, Hopkins PN, Wang T, Leary ET, Nakajima K, Davignon J, et al. Remnant-like particle cholesterol and triglyceride levels of hypertriglyceridemic patients in the fed and fasted state. J Lipid Res. 2000;41(9):1428–36.

53. Varbo A, Nordestgaard BG, Tybjæ rg-Hansen A, Schnohr P, Jensen GB, Benn M. Nonfasting triglycerides, cholesterol, and ischemic stroke in the general population. Ann Neurol. 2011;69(4):628–34.

54. Stampfer MJ, Krauss RM, Ma J, Blanche PJ, Holl LG, Sacks FM, et al. A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction. Jama. 1996;276(11):882–8.