**Original Research Article**

# Study of post-prandial hypertriglyceridemia as an independent risk factor for ischemic heart disease in Government Dharmapuri Medical College, Dharmapuri

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## Abstract

**Introduction:** Coronary heart disease is the leading cause of death in Western countries and it is now an increasing problem in developing countries too, due to changes in life style and dietary habits. Heart Disease is responsible for more deaths and disability among Western Population, both male and female, than any other killer disease, and it is quickly establishing itself as the leading cause of death and disability among Indians as well.

**Aim of the Study:** To study relation between risk factors for atherosclerosis and fasting and postprandial triglyceride levels in patients of Coronary artery disease. To establish that post-prandial triglycerides level is a better indicator as a 'risk factor' for atherosclerosis.

**Materials and methods:** A complete physical and cardiovascular system examination performed. Blood pressure measurements were performed with mercury sphygmomanometer in a standardized fashion. Waist circumference was measured at umbilical level. Hip circumference was measured at maximum girth at hip. Recording of ECG was done with 12 leads recording in standard fashion with B.P.L machine. Fasting samples of blood glucose, Fasting Lipid profile was measured.

**Results:** In our study, out of 56 male 34 (62.5%) were suffering from diabetes mellitus while 30 female out of 44 were (68.18%) diabetic. So out of 100 patients 64 patients were suffering from

diabetes. In our study, 57 patients were hypertensive. 32 male patients out of 56 male patients were hypertensive (57.14%) and 25 female patients out of 44 patients (56.81%) were hypertensive. In the present study, postprandial hypertriglyceridemia was found in 16 patients out of 23 patients (69.5%) in the age group 35-45, 31 patients out of 46 patients (67.39%) in the age group 46-55, 9 out of 21 patients (42.8%) in the age group 56,-65, and 8 patients out of 10 (80%) in the age > 66 years.

**Conclusion:** In our study with reference to patient of ischemic heart disease, postprandial Hypertriglyceridemia was found in 64% patients, having normal fasting triglyceride level. There is statistically a significant correlation between postprandial triglyceride and ischemic heart disease, even in patients having normal fasting triglyceride level. It means that patients having high postprandial triglyceride levels have higher risk of Ischemic heart disease. The relative risk is 1.44.

## Key words

Coronary heart disease, Fasting Lipid profile, Hip circumference, Postprandial Hypertriglyceridemia.

## Introduction

This sudden increase in incidence of Heart disease is seen in our people, as they have adopted a more sedentary westernized life style, together with the intake of high-fat, high-salt diet and processed foods that have come to be associated with technological affluence [1]. The association between atherosclerotic diseases and elevated fasting plasma LDL-C and reduced fasting plasma HDL-C is well established. However, many individuals without fasting lipid abnormalities develop atherosclerotic diseases and several lines of evidence suggest that no fasting lipid measurements may be more relevant to atherogenesis [2]. Typical diets are associated with measurable postprandial lipdemia 18 hours per day. A major source of circulating triglycerides is dietary fat, which after hydrolysis into free fatty acids and glycerides, is transported through the intestinal villi and absorbed by enterocytes where these particles are synthesized into chylomicron - associated Triglycerides for entry into the blood compartment and ultimately storage in adipose tissue [3]. Postprandial lipids and their associated partially hydrolyzed chylomicron remnants appear to promote early atherogenesis and adversely affect endothelial function, associate with atherogenic small LDL particles, and correlate with both pro-thrombotic and proinflammatory biomarkers, including factor VII, plasminogen activator inhibitor-1, and C-reactive protein [4]. Thus measurement of postprandial Triglycerides - particularly because they peak 3-4hr after ingestion of a fat-rich meal- might well provide more relevant information on vascular risk than measurements based on fasting concentrations. Diabetic patients have delayed clearance of Triglyceride from blood. Most type 2 diabetic patients have postprandial triglycerides [5] above optimal concentrations for several hours after meals. Moreover, optimal fasting concentrations are not always a good predictor of postprandial triglycerides. So there should be some association of postprandial lipid Metabolism and atherosclerosis, which remains to be proved [6]. Two articles recently published in the Journal of the American Medical Association directly address these issues by comparing fasting with no fasting (Postprandial) triglycerides with respect to the prediction of future cardiovascular events [7]. The first report derived from the Women's Health study cohort showed no fasting triglycerides maintained a strong independent relationship with future cardiovascular events in fully adjusted analyses. The second report derived from a prospective cohort study showed no fasting triglycerides were also found to significantly predict future vascular events in both sexes after multivariate analysis [8]. These data not only derive from typical western populations in the US and Europe but also extend to otherwise low risk populations in which overt hyperlipidemia is less prevalent [9].

## Materials and methods

This study was carried out in reference to 100 patients, diagnosed to have ischemic heart disease who were admitted at Govt. Dharmapuri Medical College Hospital, Dharmapuri for Coronary artery disease. This study was conducted on patients between November 2016 and May 2017.

### Inclusion criteria

- Unstable angina diagnosed on classical angina chest pain or angina chest pain equivalent with ECG showing ST segment depression in two consecutive chest leads or Limb leads and Normal S.CPK-MB levels.
- Fasting S. Triglycerides <150 mg%
- Fasting S. Cholesterol < 180 mg%.

### Exclusion criteria

- Patient on treatment with Tablet Rosiglitazone in past one month.
- On treatment with lipid lowering drugs.
- Suspected cases of Prinzmetal's angina Rheumatic heart disease.
- Oral contraceptive pills or other hormone therapy.
- Abnormal liver and Renal function test.

A complete and detailed history of patient with address, occupation, past history of diabetes, hypertension, family history, habits of smoking and alcohol was noted. A complete physical and cardiovascular system examination performed. Blood pressure measurements were performed with mercury sphygmomanometer in a standardized fashion. Height measured in standing position without shoes with a standard tape meter. Body mass index was calculated with formula of $B.M.I = wt\ (kg)/Ht\ (m^2)$. Waist circumference was measured at umbilical level. Waist hip ratio cut off points > 1.0 for male and > 0.85 for female was considered. Recording of ECG was done with 12 leads recording in standard fashion with B.P.L machine. Fasting samples of blood glucose, Fasting Lipid profile was measured which includes measurements of Cholesterol, S. tryglyceride, HDL, and LDL VLDL. Post prandial 2 hour. Glucose 4 hour blood samples for triglycerides levels. Fasting lipid profile defined as at least 12 hour fast overnight in which only water is permitted.

## Results

In the present study, there were total 100 patients, out of whom 56 were male and 44 were female. Only 17 female patients are aged less than 46 years. There were 46 patients in age group 46-55, out of which 27 were male, and 19 were female. In the age group 56-65 years, 16 were male and 5 were female, while only 7 male and 3 female were in > 65 years age group. The mean age was $47.10 \pm 5.2$ years (**Table – 1**).

**Table – 1:** Distribution according to age and sex.

| Age (Years) | Male | Female | Total |
| --- | --- | --- | --- |
| 35-45 | 6 | 17 | 23 |
| 46-55 | 27 | 19 | 46 |
| 56-65 | 16 | 5 | 21 |
| > 66 | 7 | 3 | 10 |
| **Total** | **56** | **44** | **100** |

In our study, out of 56 male 34 (62.5%) were suffering from diabetes mellitus while 30 female out of 44 were (68.18%) diabetic. So out of 100 patients 64 patients were suffering from diabetes (**Table – 2**).

**Table – 2:** Presence of diabetes mellitus.

| D.M. | Male | Female | Total |
| --- | --- | --- | --- |
| Presence | 34 | 30 | 64 |
| Absence | 22 | 14 | 36 |
| **Total** | **56** | **44** | **100** |

In our study, 57 patients were hypertensive. 32 male patients out of 56 male patients were hypertensive (57.14%) and 25 female patients out of 44 patients (56.81%) were hypertensive (**Table – 3**).

In this study, out of 100 patients, 64 patients showed Triglyceride levels >160 mg% after four

hour of meal. 34 out of 56 male patients (60.7%) and 30 out of 44 female (68.18%) showed post prandial hypertriglyceridemia (**Table – 4**).

**Table – 3:** Presence of hypertension.

| Hypertension | Male | Female | Total |
|---|---|---|---|
| Presence | 32 | 25 | 57 |
| Absence | 24 | 19 | 43 |
| **Total** | **56** | **44** | **100** |

**Table – 4:** Distribution according to PP4TG.

| PP4TG | Male | Female | Total |
|---|---|---|---|
| Normal | 22 | 14 | 36 |
| High | 34 | 30 | 64 |
| **Total** | **56** | **44** | **100** |

In the present study, postprandial hypertriglyceridemia was found in 16 patients out of 23 patients (69.5%) in the age group 35-45, 31 patients out of 46 patients (67.39%) in the age group 46-55, 9 out of 21 patients (42.8%) in the age group 56,-65, and 8 patients out of 10 (80%) in the age > 66 years (**Table – 5**).

**Table – 5:** Distribution according to age and PP4TG.

| Age (Years) | Normal PP4TG | High PP4TG | Total |
|---|---|---|---|
| 35-45 | 7 | 16 | 23 |
| 46-55 | 15 | 31 | 46 |
| 56-65 | 12 | 9 | 21 |
| > 66 | 2 | 8 | 10 |
| **Total** | **36** | **64** | **100** |

**Table – 6:** Distribution according to PP4TG and HDL.

| PP4TG | Normal HDL | Low HDL | Total |
|---|---|---|---|
| Normal | 22 | 14 | 36 |
| High | 20 | 44 | 64 |
| **Total** | **42** | **58** | **100** |

In the present study, out of 100 patients 64 had postprandial hypertriglyceridemia. Out of 64, 44 had low fasting S. HDL and 20 had normal fasting S. HDL. While 36 patients had normal postprandial Triglyceride level, out of 36, 14 had low fasting S.HDL and 22 had normal fasting S.HDL. There was no relation found between HDL level and High PP4TG levels (P value was 0.34) (**Table – 6**).

## Discussion

In the present study, 69% patients were aged less than 55 years. While 75% female and 62% male were aged less than 55 years. These finding shows that middle age patients are more likely to be selected for study like the present study (P value <0.05). In Hiroyasu, et al. study, 55% were male and 45% were female, average age was 55.1 ± 6.3 years. In that study also majority of patients were from middle age group. (P value <0.05) [10]. In our study, 40% of patients were overweight, 52% of patients were obese, according to National Institutes of Health Definition. The mean BMI was 30.08.The mean BMI in normal PP4TG group was 28.90 and high PP4TG group was 30.87, so there is strong correlation found between High BMI and High PP4TG. In Hiroyasu, et al. study, the mean BMI was 28.08. The mean WHR in normal PP4TG group was 0.968 and high PP4TG group was 1.028, so there is strong correlation found between High WHR and High PP4TG.In Couillard, et al. study, on postprandial triglyceride response in visceral obesity showed that obesity and waist hip ratio are associated with impaired postprandial TG clearance [11]. In our study, 64% were diabetic of which 62.5% of male were suffering from Diabetes Mellitus, whereas 68.18% females were diabetic. The mean FBS in normal PP4TG group was 80.30 and high PP4TG group was 168.90, so there is strong correlation found between High FBS and High PP4TG.The mean PP2BS in normal PP4TG group was 174.30 and high PP4TG group was 226.46, so there is strong correlation found between High PP2BS and High PP4TG.In Hiroyasu et al study, 52.1% patients were diabetic [12]. A study done by Matte Axelsen, et al., on postprandial Hypertriglyceridemia and type-2 diabetes showed postprandial lipid intolerance despite having normal fasting

triglyceride level and increased risk of macro-angiopathy [13].In this study, out of 100 patients, 64 patients showed serum triglyceride level more than 160 mg% after 4 hour of meal. 34 out of 56 male patients (60.7%) and 30 out of 44 females (68.18%) showed postprandial hypertriglyceridemia these data tells that patient having ischemic Heart disease, even if they have normal fasting triglyceride levels, they might have impaired postprandial lipid metabolism. The mean PP4TG was 181.47mg% (P Value <0.05) suggest that there is an association between coronary artery disease and PP4TG levels and the relative risk was 1.75 [14]. In Hiroyasu, et al. study, 58% male and 64% female patients showed postprandial hypertriglyceridemia (P value <0.05). In Borge G. Nordestgaard, et al. study on non-fasting Triglycerides and risk of Myocardial infarction, Ischemic Heart disease and Death in Men and Women showed that non-fasting triglyceride levels independently predict myocardial infarction, ischemic heart disease and death. In our study 57 patients were hypertension. 32 male patients (57.14%) were Hypertensive and 25 female (56.81%) were hypertensive [15]. The mean systolic blood pressure in normal PP4TG group was 129.88, high PP4TG group was 141.84 and the mean diastolic blood pressure in normal PP4TG group was 84.16 and high PP4TG group was 88.51, so there is strong correlation found between hypertension and High PP4TG [16].

## Conclusion

In our study with reference to patient of ischemic heart disease, postprandial Hypertriglyceridemia was found in 64% patients, having normal fasting triglyceride level. There is statistically a significant correlation between postprandial triglyceride and ischemic heart disease, even in patients having normal fasting triglyceride level. It means that patients having high postprandial triglyceride levels have higher risk of Ischemic heart disease [17]. The relative risk is 1.44.There is statistically a significant correlation found between postprandial Hypertriglyceridemia and

high waist-hip ratio and Diabetes Mellitus [18]. Our study showed patients of ischemic Heart disease have high postprandial triglyceride levels in spite of normal fasting triglyceride level. There is a positive correlation between high waist-hip ratio, diabetes mellitus and postprandial hypertriglyceridemia in ischemic heart disease patients.

## References

1. Borge G. Nordestgaard, Marianne Benn, Peter Schnohr, et al. Nonfasting triglycerides and Risk of Myocardial Infarction, Ischemic Heart Disease, and Death in Men and Women. JAMA, 2007; 298(3): 299-308.
2. Michael O'Riordan, Hien T. Nghiem, et al. Nonfasting Triglyceride levels Associated with an increased risk for Cardiovascular Disease from Heartwire. Available on http://www.medscape.com/viewarticle/789462
3. Austin MA, McKnight B, Edwards KL, et al. Cardiovascular disease Mortality in Familial forms of Hypertriglyceridemia: 920-year prospective study. Circulation, 2000; 101(24): 2777-2782.
4. Sarti, C. Gallagherj. The Metabolic Syndrome: Prevalence, CHD Risk, and treatment. Journal of Diabetes Complications, 2006; 20(2): 121-132.
5. Rutledge JC, Mullick AE, Gardner G, Goldberg U. Direct Visualization of lipid deposition and reverse lipid transport in a perfused artery; roles of VLDL and HDL. Circ. Res., 2000; 86(7): 768-773.
6. Zilversmit DB. Atherogenesis: a postprandial phenomenon. Circulation, 1979; 60(3): 473-485.
7. Kolovou GD, Anagnostopoulo KK, Daskalopaoulou SS, et al. Clinical relevance of postprandial lipaemia. Curr. Med. Chem., 2005; 12(17): 1931-1945.
8. Schnohr P, Jensen JS, Scharling H, Nordestgaard BG. Coronary Heart disease risk factors ranked by importance for the individual and community: a 21-year follow-up of 12000 men and women from the

copenhegen city heart study. Eur Heart J., 2002; 23(8): 620-626.

9. Kofoed SC, Gronholdt ML, Bismuth J., et al. Echolucent, rupture-prone carotid plaques associated with elevated triglyceride – rich lipoprotein, particularly in Women. J. Vasc. Surg., 2002; 36(4): 783-792

10. Cohnis, McNamara JR, Cohn SD, et al. Plasma lipoprotein Changes in the triglyceride – rich lipoprotein fraction of human subjects fed a fat-rich meal. J. lipid Res., 1988; 29(7): 925-936.

11. Hokanson JE, Austin MA. Plasma triglyceride level is a risk factor for cardiovascular disease independent of high-density lipoprotein. Cholesterol level: a metaanalysis of population –based prospective studies. J. Cardiovasc Risk, 1996; 3(2): 213-219.

12. Austin MA. Plasma triglyceride and Coronary Heart disease. Arterioscler Thromb., 1991; 11(1): 2-14.

13. Sarwar N, Danesh J, Eiriksdottir G., 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): 450-458.

14. Wittrup HH, Tybjaerg – Hansen A, Nordestgaard BG. Lipoprotein lipase Mutations, Plasma lipids and lipoproteins, and risk of ischemic Heart disease: a Meta-analysis. Circulation, 1999; 99(22): 2901-2907.

15. Manninen V, Elo Mo, Frick MH, et al. Lipid alterations and decline in the incidence of coronary Heart disease in the Helsinki Heart Study. JAMA, 1988; 260(5): 641-651.

16. Secondary Prevention by raising HDL, Cholesterol and reducing triglycerides in patients with coronary artery disease: the Bezafibrate Infarction Prevention (BIP) Study, circulation, 2000; 102(1): 21-27.

17. Robins. S.J. Collins D, Coitles J.T., et.al. Relation of gemfibrozil treatment and lipid levels with major coronary events. JAMA, 2001; 285(12): 1585-1591.

18. Stampfer. M.S. Krauss RM, Maj, et al. A prospective study of triglyaride level, low – density lipoprotein particle diameter, and risk of myocardial Infarction. JAMA, 1996; 276(11): 882-888.