Original Article

# What is ideal the time to do post prandial triglycerides (PPTG) in routine clinical practice?

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## Abstract

**Background:** Postprandial triglyceride levels (PPTG) in plasma is a better indicator of atherogenesis. One has to subject the patients to multiple times to draw the blood sample i.e. 2, 4, 6, 8, 10, 12 hours after meal which is not only cumbersome but not feasible many time as patient may not be willing for such an investigation. **Methods and Materials:** Place of study was out patient department of Medicine, SVS Hospital, Mahabubnagar, Telangana State. Fifty cases of known ischemic heart disease (Group 1) and another 50 age and sex matched individuals (Group 2) were included in the present study. Blood withdrawn was analyzed for blood sugar, blood urea, serum creatinine, serum lipid profile. Blood drawn after 2 hours was tested for blood sugar and lipid profile; later on the lipid profile was tested after 4, 6, 8, 10 and 12 hours. **Observations:** The present study reveals there has been an elevation in plasma TG following normal meals in both groups though diseased group demonstrated a significantly high rise in PPTG levels. The rise was noted mainly up to 4 or 6 post prandial period; later there was a decline in the levels. **Summary and conclusions:** 1) The present study demonstrated an elevated PPTG levels were associated with CAD. 2) 4 hours after meals can safely be considered as PPTG levels for all practical methods as it avoids multiple blood drawings to the fussy patients and can performed an OPD (outpatient department) procedure.
**Keywords:** CAD, Triglycerides, PPTG, Risk factor.

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Received Date: 13/03/2016 Revised Date: 22/04/2016 Accepted Date: 28/05/2016

Access this article online

Quick Response Code:

Website: www.statperson.com

DOI: 01 June 2016

## INTRODUCTION

Coronary artery disease (CAD) is common ailment and is multi factorial; though mainly due to dyslipidemias and atherosclerosis[1]. Hokanson and Austin demonstrated triglyceridemia as independent risk factor, even if serum total cholesterol and low dense lipoproteins (LDL) are not elevated[2]. Zilversmit established post prandial triglyceride (PPTG) as a better indicator for atherogenesis[3]. There had been multiple studies to highlight this post prandial phenomenon[3,4,5,6]. But Post prandial exams are complex and time consuming and they are not commonly conducted in clinical practice, preventing the screening of population and acceptable most of rural population as they had get back home before dark as transport facilities do not exist in our Indian set up.

## METHODS AND MATERIALS

Place of study was out patient department of Medicine, SVS Hospital, Mahabubnagar, Telangana State. Fifty cases of known ischemic heart disease (Group 1) and another 50 age and sex matched individuals (Group 2) were included in the present study. Blood withdrawn was analyzed for blood sugar, blood urea, serum creatinine, serum lipid profile. Blood drawn after 2 hours was tested for blood sugar and lipid profile; later on the lipid profile was tested after 4, 6, 8, 10 and 12 hours. The data then transferred to excel sheet and accessed for any change in the two groups. The data was analyzed with paired‘t’ test and linear regression. Coefficients of variation (CV) were determined by taking the SD divided by the mean using the root mean square approach.

How to site this article: Venkateshwarlu Nandyala, Gandiah P. What is ideal the time to do post prandial triglycerides (PPTG) in routine clinical practice? *International Journal of Recent Trends in Science and Technology* June 2016; 19(2): 167-170. http://www.statperson.com (accessed 03 June 2016).

## RESULTS AND OBSERVATIONS

Table 1 describes the age wise distribution in both the group. Youngest was 30 years of age while the oldest was 63. There was no difference between the two groups.

**Table 1:** Distribution of subjects by Age group

| Age group in years | Group-1: Patients with C.A.D. | | Group 2: Controls | |
|---|---|---|---|---|
| | Number | % | Number | % |
| 30 – 40 | 18 | 36 | 16 | 32 |
| 41 – 50 | 16 | 32 | 20 | 40 |
| 51 – 60 | 14 | 28 | 12 | 24 |
| 61 – 70 | 2 | 4 | 2 | 4 |
| Total | 50 | 100 | 50 | 100 |
| Mean ± SD | 45.16 ± 9.4 | | 45.6 ± 9.34 | |
| Range | 30 - 61 | | 30 – 63 | |

**F = 0.25 p = 0.97**

Table II shows various biochemical parameters in the two groups. Obviously the study group showed significant difference in FBS, PPBS, fasting and post meal (4 hours) TG between the groups. Other parameters did not show any variation in this study.

**Table 2:** Mean ± SD values of studied parameters in Diabetes with complication and controls

| PARAMETER | Group-1: Patients with complication | | Group 2: Controls cases | | 'p' value |
|---|---|---|---|---|---|
| | MEAN | SD | MEAN | SD | |
| FBS | 205.2 | 71.4 | 85.1 | 9.07 | < 0.001* |
| PPBS | 294.68 | 103.48 | 114.44 | 13.8 | < 0.001* |
| Urea | 29.64 | 5.2 | 26.8 | 2.91 | 0.42 |
| Creatinine | 1.07 | 0.19 | 0.84 | 0.16 | 0.084 |
| Total Cholesterol | 196.6 | 32.9 | 156.8 | 19.08 | 0.076 |
| Triglyceride Fasting | 223.43 | 61.8 | 126.08 | 24.86 | < 0.001* |
| HDL | 31.8 | 7.5 | 44.92 | 5.23 | 0.078 |
| LDL | 120 | 30.2 | 86.6 | 21.7 | 0.35 |
| VLDL | 44.6 | 12.3 | 25.2 | 4.97 | 0.092 |
| Triglyceride Post Meal (4 hours) | 407.2 | 109.8 | 211.3 | 40.8 | < 0.001* |

The following table III highlighted the serum TG levels at regular intervals of 2, 4, 6, 8 and 12 hours following a meal.

**Table 3:** Showing the mean values of triglycerides at various intervals

| Parameter | Group 1 | Group 2 |
|---|---|---|
| Fasting triglyceride levels | 223.4 ± 61.8 | 126.08 ± 24.86 |
| 2 hours after meals | 407.2 ± 109.8 | 211.3 ± 40.8 |
| 4 hours after meals | 411.24 ± 98.65 | 216 ± 35.84 |
| 6 hours after meals | 378 ± 78.68 | 208 ± 28.88 |
| 8 hours after meals | 308 ± 104.22 | 194.22 ± 24.44 |
| 10 hours after meals | 242.6 ± 66.86 | 134.66 ± 22.88 |
| 12 hours after meals | 219.8 ± 64.40 | 124.78 ± 22.88 |

The graphic distribution of the values of TG levels reveal a raise in levels of TG up to 4 hours steadily and then a plateau or decline in the levels and by 8 – 12 hours it was almost like the fasting levels. The same pattern was observed in both groups though the levels were significantly more in diabetic patients with CAD.

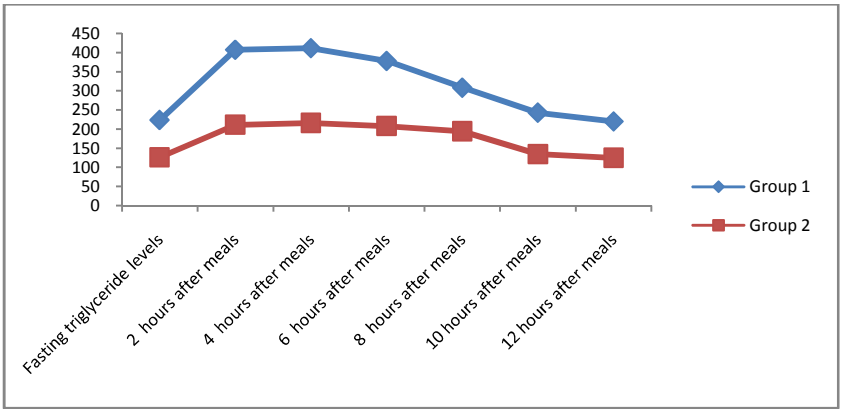


**Figure 1:** Showing the graphic representation of the mean values of triglycerides at various intervals

## DISCUSSION

The present study reveals there has been an elevation in plasma TG following normal meals in both groups though diseased group demonstrated a significantly high rise in PPTG levels. The rise was noted mainly up to 4 or 6 post prandial period; later there was a decline in the levels. Stampfer *et al*. (1996) earlier studied 266 diseased cases and 308 control individuals showed that plasma TG levels measured 3 to 4 h after a meal were better than fasting plasma TG levels at predicting future cases of myocardial infarction[6]. The relationship between elevated TG concentration and the risk of CAD has been well documented (Krieger 1998, Sharrett *et al*. 2001)[7,8,9]. Some studies highlighted elevated PPTG as "forgotten" risk factor by Austin[10] and Gotto[11]. However, postprandial TG measurements are related to the development of atherosclerosis[12,13]. Zilversmit opined the increased levels of non-fasting TG may indicate the presence of increased levels of atherogenic remnant lipoproteins[3], which can penetrate the endothelial cell layer and reside in the sub endothelial space which is a CAD risk factor[14,15,16,17,18].

## SUMMARY AND CONCLUSIONS

The present study demonstrated an elevated PPTG levels were associated with CAD. Four hours after meals can safely be considered as PPTG levels for all practical methods as it avoids multiple blood drawings to the fussy patients and can performed an OPD procedure.

## REFERENCES


1. Rosenson RS. Hypertriglyceridemia and coronary heart disease risk. *Cardiol Rev* 1999; 7 : 342-8.
2. 13. Hokanson JE and Austin MA (1996) Plasma triglyceride level is a risk factor for cardio-vascular disease independent of high density lipoprotein cholesterol levels: a meta analysis of population based prospective studies. J Cardiovasc. Risk 3: 213 – 219.
3. Zilversmit DB. Atherogenesis: A postprandial phenomenon. *Circulation* 1979; 60: 473-85.
4. Bae JH, Schwemmer M, Lee IK, *et al*. Postprandial hypertriglyceridemia induced endothelial dysfunction in healthy subjects is independent of lipid oxidation. *Int J Cardiol* 2003; 87: 259-67.
5. Ernst JS, Marie CA, Judith R, *et al*. Comparison of fasting and postprandial plasma lipoproteins in subjects with and without coronary heart disease. *Am J Cardiol* 2001; 88: 1129-33.
6. Stampfer MJ, Krauss RM, Ma J, Blanche PJ, Holl LG and Sacks FM. (1996). A prospective study of triglyceride level, low-density lipoprotein particle diameter, and risk of myocardial infarction JAMA. s276: 882-888.
7. Krieger M. 1998. The best of cholesterols, the worst of cholesterols: a tale of two receptors. Proc Natl Acad Sci 95: 4077-4080.
8. Sharrett AR, Ballantyne CM, Coady SA, Heiss G, Sorlie PD, Caterllier D, Sharrett AR, and Patsch W.: (2001). Coronary heart disease prediction from lipoprotein cholesterol levels, triglyceride, lipoprotein (a), apolipoprotein A-I and B, and HDL density subfractions; Circulation 104: 1108-1113.
9. Gandiah P, Venkateshwarlu Nandyala, Bingi Srinivas, Kartikeya Raman Reddy, Najma Farheen, Yashwant Reddy; A study to show Postprandial hypertriglyceridemia as a risk factor for macrovascular complications in Type 2 DM, International J contemporary Medical Research 2016 (6): 1587-1590.
10. Austin MA, Hokanson JE and Edwards KL. 1998. Hypertriglyceridemia as a cardiovascular risk factor; Am J Cardiol 81: 7B-12B.
11. Gotto AM. (1998): Triglyceride: the forgotten risk factor; Circulation 97: 1027-1028.
12. Boquist S, Ruotolo G, Tang R, Bjorkegren J, Bond MG, de Faire U, Karpe F, Hamsten A: Alimentary lipemia, postprandial triglyceride- rich lipoproteins, and common carotid intima-media thickness in healthy, middle aged men. *Circulation* 100:723–728, 1999.
13. 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.

14. Patsch JR, Miesenbock G, Hopferwieser T, Muhlbergerger V, Knapp E, Dunn JK, Gotto Jr AM and Patsch W. (1992): Relation of triglyceride metabolism and coronary artery disease. Studies in the postprandial state; Arterioscler Thromb Vasc Biol 12: 1336-1345.
15. Rapp JH, Lespine A, Hamilton RL, Colyvas N, Chaumeton AH, Tweedie-Hardman J, Kotite L, Kunitake ST, Havel RJ and Kane JP (1994): Triglyceride-rich lipoproteins isolated by selected affinity anti-apolipoprotein B immunosorption from human atherosclerotic plaque; Arterioscler Thromb Vasc Biol 14: 1767-1774.
16. Proctor SD and Mamo JC (1998): Retention of fluorescent labelled chylomicron remnants within the intima of the arterial wall-evidence that plaque cholesterol may be derived from post-prandial lipoproteins. Eur J Clin Invest 28: 497-503.
17. Ginsberg HN (2002): New perspectives on atherogenesis: role of abnormal triglyceride-rich lipoprotein metabolism. Circulation 106: 2137-2142.
18. Kolovou GD, Mikhailidis DP, Kovar J, Lairon D, Nordestegaard BG, Ooi TC, perez-Martinez P, Bilianou H, Ananotopoulou K and Panotopoulos G (2011) Assessment and clinical relevance of non-fasting and postprandial triglycerides: An Expert Panel Statement; Curr Vasc Pharmacol 9: 258-270.

Source of Support: None Declared
Conflict of Interest: None Declared