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## RESEARCH ARTICLE

# EVALUATION OF CORRELATION BETWEEN POST PRANDIAL DYSLIPIDEMIA AND ANGIOGRAPHIC SEVERITY OF CORONARY ARTERY DISEASE IN TYPE 2 DI-ABETES MELLITUS WITH SPECIAL REFERENCE TO TRIGLYCERIDE LEVELS

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**ABSTRACT**

**Background:** The dyslipidemia of diabetes in the postprandial phase is a major determinant of the atherogenicity. Postprandial high LDL and TG may account for up to fivefold increase in atherosclerosis in the diabetic patients.
**Objective:** In this study we investigated the correlation of postprandial dyslipidemia with angiographic severity of CAD in type -2 diabetic patients.
**Methods:** 50 type-2 diabetic patients (37 male, 13 female) were screened for glycemic control ($HBA_{1C}$), WHR(waist hip ratio), BMI (body mass index), fasting and postprandial lipid Profile. CAD was evaluated in terms of vessels involved, site of lesion, percentage of coronary stenosis and morphology of the lesions.
**Result:** The mean TG levels in diabetic with CAD and without CAD were 168.41±20.32 and 145±16.33, in fasting state ($p < 0.001$) and 190.25±19.08 and 154.54±21.50 in postprandial state, respectively ($p<0.0001$). Postprandial hypertriglycerdemia is associated with more severe CAD in terms of number of vessels involved, and type, number and morphologic severity of lesions on angiography.
**Conclusion:** We concluded that there is a definite correlation between severity of CAD and postprandial hypertriglyceridemia in diabetic patients.



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## INTRODUCTION

Diabetic dyslipidemia is believed to play an important role in the pathogenesis of accelerated atherosclerosis in diabetic patients (Fontbonne, 1991). The predominant lipid abnormalities seen in diabetes mellitus are an elevated serum triglyceride (TG) level and a low HDL-C level (Fontbonne *et al*., 1989; Taskinen, 1992). It is being increasingly believed that atherosclerosis is a postprandial phenomenon, at least with respect to lipids (Patsch *et al*., 1992).

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Triglycerides increase the risk of CAD by increasing the LDL level, decreasing HDL level, disrupting the function of artery walls, and activating the thrombogenic factors and plasminogen activators. Recent studies indicated that fasting triglyceride concentration has low independent effect on risk of CAD (Miller *et al*., 2011). As the prevalence of diabetes is high in our area (in and around Bikaner), and various risk factors like postprandial dyslipidemia for diabetes mellitus and CAD are more prevalent, this study was carried out to evaluate postprandial triglyceride levels in patients with type 2 diabetes mellitus and also to analyze the significance of postprandial triglyceridemia and its correlation with angiographic severity of coronary artery disease.

## SUBJECTS AND METHODS

This study was conducted on 50 type-2 diabetic patients (37 male, 13 female). Patients with hepatic disease, renal disease, hypothyroidism, Cushing's disease and inherited disorders of lipid Metabolism were excluded. Patients were divided into two groups (1) Diabetic patients with CAD (2) Diabetic patients without CAD. All patients were evaluated as per performa including clinical history, screening for various complications of diabetes; WHR and BMI, and glycemic control ($HBA_{1C}$). Blood was taken after 12 hours overnight fasting and after 2 hours of fat rich mixed meal. The samples were analyzed by semiauto-analyzer using enzymatic kits for Total cholesterol, Triglyceride, HDL-cholesterol (HDL), LDL-cholesterol (LDL), VLDL-cholesterol (VLDL).

Angiography was performed in eligible patients by radial artery approach preferentially by 5 Fr catheter. Nonionic contrast agents (Optiray) was used. Angiography study was done in at least 2 projections i.e. right anterior oblique (RAO) and left anterior oblique (LAO) to furnish the postprandial and lateral views of the least. The RAO caudal and LAO caudal projection are best for visualizing the proximal middle $LC_X$ and obtuse marginal branches. Cannulation of the origin of the RCA was also performed in the LAO position.

Total occlusion was identified on the cineangiogram as an abrupt termination of the epicardial vessel, osteal lesions are defined as those arising within 3mm of origin of the vessel or branches and CAD severity grading was done according to percentage of stenosis (> 50% being significant). The data obtained from the observation were analyzed statistically mean, SD, percentage and compared by Chi-square test, "p" value <0.05 was taken as significant.

## RESULTS

There were 39 patients of diabetes with CAD and 11 were without CAD, clinical characteristics are shown in table-1. It is observed that patients of diabetes with CAD were older, had higher BMI, poor control of diabetes and more dyslipidemia. Table-2 shows correlation between postprandial lipid levels and angiographic severity of CAD. It is observed that higher levels of postprandial total cholesterol, LDL and triglyceride are associated with more severe CAD in terms of number of vessels involved. We also observed that postprandial hypertriglyceridemia was associated with more severe angiographic CAD as compared to fasting hypertriglyceridemia (table-3). Table-4 shows comparison of various morphologic types of lesions observed during angiography between patients with fasting and postprandial triglyceride levels of <150 and >150mg%.

**Table 1. Clinical Characteristics of Diabetic Patients with or without CAD**

| 0 | Diabetic with CAD (n=39) | | Diabetics without CAD (n=11) | |
|---|---|---|---|---|
| | Mean | SD | Mean | SD |
| AGE | 53.66 | 10.12 | 44.18 | 10.53 |
| BMI | 23.27 | 2.08 | 20.57 | 2.39 |
| WHR | 0.876 | 0.035 | 0.884 | 0.053 |
| $HBA_1C$ | 9.64% | 1.62% | 8.07% | 1.44% |
| LIPID PROFILE(FASTIG) | | | | |
| TC | 180.58 | 23.42 | 152.72 | 22.12 |
| LDL | 109.20 | 19.54 | 85.54 | 23.46 |
| VLDL | 35.10 | 12.44 | 25.09 | 5.70 |
| HDL | 41.10 | 8.43 | 48.81 | 6.41 |
| TG | 168.41 | 20.32 | 145 | 16.33 |
| LIPID PROFILE (P.P) | | | | |
| TC | 196.66 | 22.40 | 163.45 | 22.77 |
| LDL | 122.35 | 21.03 | 96.63 | 29.94 |
| VLDL | 38.10 | 12.10 | 29.09 | 5.92 |
| HDL | 34.94 | 6.60 | 40.72 | 3.19 |
| TG | 190.25 | 19.08 | 154.54 | 21.50 |
| SEX | No. | % | No. | % |
| Male(n=37) | 32 | 84.37% | 5 | 15.63% |
| Female (n=13) | 7 | 53.85% | 6 | 46.15% |
| Blood Pressure | No. | % | No. | % |
| Hypertensive(n=32) | 27 | 84.37% | 5 | 15.63% |
| Normotensive(n=18) | 12 | 66.66% | 6 | 33.33% |
| Smoking | No. | % | No. | % |
| Smokers(n=23) | 21 | 91.30 | 2 | 8.70 |
| Nonsmokers (n=27) | 18 | 66.67 | 9 | 33.33 |

**Table 2. Correlation between postprandial lipid levels and severity of CAD (Total No=39)**

| Lipid profile | Lipid level | CAD severity | | | | | | Total vessels involved | | $\chi^2$ | p-value |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | SVD | | DVD | | TVD | | | | | |
| | | No. | % | No. | % | No. | % | No. | % | | |
| TC (mg%) | ≤200(n=23) | 11 | 47.8 | 10 | 43.48 | 2 | 8.69 | 37/69 | 53.62 | 5.40 | <0.02 |
| | >200(n=16) | 3 | 18.75 | 4 | 25 | 9 | 56.25 | 38/48 | 79.1 | | |
| LDL | ≤100(n=7) | 4 | 57.1 | 3 | 42.9 | 0 | - | 10/21 | 47.6 | 3.01 | <0.1 |
| (mg%) | >100(n=32) | 10 | 31.25 | 11 | 34.37 | 11 | 34.37 | 65/96 | 67.70 | | |
| HDL (mg%) | ≤45(n=34) | 11 | 32.35 | 12 | 35.29 | 11 | 32.35 | 68/102 | 66.66 | 2.82 | <0.1 |
| | >45(n=5) | 3 | 60 | 2 | 40 | 0 | - | 7/15 | 46.67 | | |
| TG (mg%) | ≤150(n=1) | 1 | 100 | 0 | - | 0 | - | 1/3 | 33.33 | 7.14 | <0.01 |
| | >150(n=38) | 13 | 34.2 | 14 | 36.8 | 11 | 28.94 | 74/84 | 88.1 | | |

**Table 3. Fasting and postprandial (PP) triglyceride levels and its correlation with angiographic severity of CAD (Total No=39)**

| Severity of CAD | | Levels of Triglyceride (mg%) | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 100-150 | | 151-200 | | 201-250 | | ≥251 | |
| | | Fasting | PP | Fasting | PP | Fasting | PP | Fasting | PP |
| No. of cases | | 4 | 1 | 29 | 23 | 5 | 9 | 1 | 6 |
| | SVD | 2 (50) | 1 (100) | 14 (48.27) | 8 (34.8) | 0 | 0 | 0 | 0 |
| | DVD | 2 (50) | 0 | 11 (37.9) | 10 (43.48) | 3 (60) | 4 (44.44) | 0 | 0 |
| Severity of CAD | TVD | 0 | 0 | 4 (13.79) | 5 (21.7) | 2 (40) | 5 (55.55) | 1 (100) | 6 (100) |
| Number of cases (%) | Total Vessels | 6/12 | 1/3 | 48/87 | 43/69 | 12/15 | 23/27 | 3/3 | 18/18 |
| | involved | (50) | (33.33) | (55.15) | (62.31) | (80) | (85.18) | (100) | (100) |

**Table 4. Comparison of various angiographic morphological Types of Lesions With Fasting &Postprandial Triglyceride Levels**

| Type of Lesion | FASTING TG LEVEL (mg%) | | | | POST PRANDIAL TG LEVEL | | | |
|---|---|---|---|---|---|---|---|---|
| | <150(n=4) | | >150(n=35) | | <150(n=1) | | >150 (n=38) | |
| | No. | Lesions /patient | No. | Lesions /patient | No. | Lesions /patient | No. | Lesions /patient |
| Total Lesions | 5 | 1.25 | 68 | 1.94 | 1 | 1 | 76 | 2 |
| Concentric lesions | 3 | 0.75 | 38 | 1.08 | 1 | 1 | 42 | 1.10 |
| Multiple irregular lesions | - | - | 9 | 0.26 | - | - | 9 | 0.24 |
| Diffuse Lesions | 1 | 0.25 | 7 | 0.20 | - | - | 9 | 0.24 |
| Other Lesions | 1 | 0.25 | 14 | 0.40 | - | - | 16 | 0.42 |

It shows higher number of lesions are associated with higher levels of triglyceride specifically postprandial.

## DISCUSSION

Our study evaluated postprandial dyslipedemia with special reference to triglyceride levels in cases of diabetes with coronary arteries disease proven by angiography. Moreover, these results also show us a prevalence of serum triglycerides in these patients. In this study we observed that the mean level of fasting and postprandial triglycerides were significantly higher in patients with CAD than without CAD. Furthermore, postprandial triglyceride abnormality was significantly more than fasting triglyceride in CAD patients. Our results also illustrated that postprandial triglyceride has more association with CAD and its severity as evaluated by angiography in terms of number of vessels involved; type, number and morphologic severity of the lesions and this test had more sensitivity for CAD detection in comparison to fasting triglyceride measurement. These observations represent a higher value of postprandial triglyceride measurement rather than fasting triglyceride in diabetics associated with CAD. Triglycerides have insulation and energy saving role in fat tissues of human body. Total glyceride and/or LDL level more than 90% or HDL level lower than 10% indicates dyslipidemia (Despres, 2009). Triglycerides increase the risk of CAD by increasing the LDL level, decreasing HDL level, disrupting the function of artery walls, and activating the thrombogenic factors and plasminogen activators (Harchaoui, 2009). Recent studies indicated that fasting triglyceride concentration has low independent effect on risk of CAD (Nordestgaard *et al*., 2007). This phenomenon could be derived from lots of daily changes in plasma triglyceride concentrations and the presence of a strong reverse relation between the concentration of serum triglycerides and HDLs (Austin, 1991; Carlson *et al*., 1979). Changing the lifestyle and new therapeutic approaches can increase the life quality of diabetics with CAD risk (Miller *et al*., 2011). Several underlying machanisms have been postulated for the exaggerated PP triglyceride response in diabetes mellitus. Although, this has not been resolved completely, delayed clearance of triglyceride rich lipoprotein (TRL) secondary to decreased LPL activity is believed to be the most important mechanism with some contribution from excessive hepatic triglyceride production (Taskinen, 2003).

Elevated postprandial triglyceridemia have been associated in clinical trials with both early coronary artery and carotid artery atherosclerosis for persons with hyperlipidemia, independent of establish risk factors (Wilson *et al*., 1991; Miller *et al*., 1981; Manochehri *et al*., 2016). Some earlier studies also show that diabetic patients having high triglyceride levels had more total lesions, concentric lesions and multiple irregular lesions in both fasting and postprandial state (Kasaoka *et al*., 1997; Madhu *et al*., 2005). Although numbers of patients studied were less in the present study and further studies are required with large number of the cases to firmly document correlation between severity of CAD and postprandial hypertriglyceridemia in diabetic patients. Some other risk factors like obesity, poor glycemic control, smoking and hypertension have also shown association with postprandial hypertriglyceridemia in diabetics, which might have independent or additive effects. Role of life style modification, dietary control and aggressive therapy for dyslipidemia and hypertension becomes more relevant in prevention and treatment of coronary artery disease in diabetic patients.

### Conclusion

Our study concludes that there is a definite correlation between severity of CAD and postprandial hypertriglyceridemia in diabetic patients. It would be rational to investigate for postprandial lipid levels in diabetic patients with CAD for better management. Conflicts of Interest Statement: The authors have NO affiliations with or involvement in any organization or entity with any financial interest or non-financial interest in the subject matter or materials discussed in this manuscript.

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## REFERENCES

Austin MA. 1991. Plasma triglyceride and coronary heart disease. Arteriosclerosis, Thrombosis, and Vascular Biology. 1991;(1): 2-14

Carlson LA, Bottiger LE. 1979. Risk factors for myocardial infarction in the Stockholm Prospective Study. Acta Medica Scandinavica. 1979; 206(1-6): 351-60.

Despres JP. 2009. Effect of Rimonabant on the High-Triglyceride/ Low - HDL-Cholesterol Dyslipidemia,

Intraabdominal Adiposity, and Liver Fat The ADAGIO-Lipids Trial. Arteriosclerosis, Thrombosis, and Vascular Biology. 2009; 29(3): 416-23.

Fontbonne A, Eschwege E, Cambien F, Richard J-L, Ducimetiere P *et al*. 1989. Hypertriglyceridemia as a risk factors for coronary heart disease mortality in subjects with impaired glucose toleranceor diabetes: Results from 11 year follow up of the Paris Prospective study. Diabetologia 1989;32:300-04.

Fontbonne A. 1991. Relationship between diabetic dys-lipoproteinemia and coronary heart disease risk in non-insulindependent diabetes. Diabetes Metab Rev 1991;7:179-89.

Harchaoui KEL, Visser ME, Kastelein JJ, *et al*. 2009. Triglycerides and Cardiovascular Risk. Current Cardiology Reviews. 2009; 5(3):216-22.

Kasaoka S, Okuda F, Sato A, Miure T, Kohno M *et al*. 1997. Effect of coronary risk and factors on coronary angliographic morphology in patients with ischemic heart disease. Jpn Arc J 1997;61 : 390-395.

Madhu SV, Mittal V, Ram BK, Srivastava DK. 2005. Postprandial lipid abnormalities in type 2 diabetes mellitus. J Assoc physicians India 2005;53:1043-6.

Manochehri M, Moghadam AJ. 2016. Studying the Relation of Postprandial Triglyceride with Coronary Artery Disease (CAD). Med Arch. 2016 Aug; 70(4): 261-264.

Miller M, Stone NJ, Ballantyne C, *et al*. 2011. Triglycerides and cardiovascular disease a scientific statement from the American Heart Association. Circulation. 2011; 123(20): 2292-2333.

Miller NE, Hammett F, Saltissi S, Rao S, Van Zeller H *et al*. 1981. Relation of angiographically defined coronary artery disease to plasma lipoprotein subfractions and apolipoproteins. Br Med J (Cin Res Ed). 1981;282(6278): 1741-4

Nordestgaard BG, Benn M, Schnohr P, Tybjaerg-Hansen A. 2007. Nonfasting triglyceride and risk of myocardial infarction, ischemic heart disease, and death in man and women. JAMA 2007; 298:299–308.

Patsch JR, Miesenbock G, Hopferwiser T, Muhlberger V, Knapp E *et al*. 1992. Relation of triglyceride metabolism and coronary artery disease: studies in the postprandial state. Arterioscler Thromb. 1992;12:1336-1345.

Taskinen M. 1992. Quantitative and qualitative lipoproteinabnormalities in diabetes mellitus. Diabetes 1992;41:12-17.

Taskinen M.R. 2003. Diabetic dyslipidaemia: from basic research to clinical practice. Diebetologia 2003;46:733-49

Wilson PWF, Anderson KM, Castelli WP. 1991. The impact of triglycerides on coronary heart disease: the Framingham Study. In: Gotto Jr AM, Paoletti R, eds. Atherosclerosis reviews, vol. 22. New York: Raven Press;59-63

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