To Correlate Postprandial Triglyceride Levels and CIMT after a Fatty meal in Type 2 Diabetes Mellitus patients with and without microalbumininuria

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Abstract
Diabetes mellitus is one of the main threats to human health in 21st century. Diabetes is a heterogenous group of metabolic disorder due to defect in insulin secretion or action or both. CAD in diabetes carries a worse prognosis than in nondiabetic subjects.

Keywords: CIMT-Carotid Intima Media Thickness, CAD-Coronary Artery Disease

Introduction
Indians with high genetic predisposition, higher upper body obesity even with low BMI and high level of insulin resistance. Insulin resistance is the main cause for diabetes in obese as well as nonobese individuals and it may be due to postreceptor defect in insulin action leading to decrease responsiveness to insulin.

The risk of diabetes complication is directly related to the duration of the disease. A Finnish study dispelled the notion that duration of diabetes was the best predictor of CAD in type 2 diabetic subjects.

Early complications of diabetes mellitus are retinopathy and nephropathy. Microalbuminuria predicts renal damage due to diabetes and it is a main independent predictive factor of cardiovascular morbidity and mortality. A common pathophysiologic process underlying the association the between microalbuminuria and cardiovascular disease were such as endothelial dysfunction, inflammation or increased transvascular leakage of macromolecules.

It has been reported that the patients with insulin resistance had significantly higher concentrations of triglycerides and hscrp. Hscrp was positively correlated to BMI and triglycerides. CRP and apo-B could have close relationships with other components of metabolic syndrome such as postprandial triglycerides and carotid intima media thickness in type 2 diabetic patients. So high CRP could be a marker for an excessive postprandial response leading to an increased risk for chronic vascular complications and atherogenesis. Insulin resistance was associated with postprandial triglycerides in type 2 diabetes even after correction of hyperglycemia. The postprandial triglycerides levels were directly related to carotid intima media thickness.

Fat intake seems the nutritional determinant of postprandial triglyceride response in type 2 diabetic patients. Therefore, postprandial
hypertriglyceridemia is assessed by giving fatty meal to patients with type 2 diabetes mellitus. So the present study was taken up to study the correlation of inflammatory marker hscrp with postprandial triglyceride levels, carotid intima media thickness in type 2 diabetes mellitus patients with and without microalbuminuria. Serum triglyceride levels are important in development of atherosclerosis. TGL level is increased in postprandial periods. To investigate the role of post prandial triglycerides in atherosclerosis, and the possible correlation between post prandial triglycerides levels and carotid intima media thickness have been investigated. In addition to that an effort has been made to find out the relationship of inflammatory parameter C-reactive protein to the intima media thickness of carotid artery as well as variety of risk factors for CAD in type 2 diabetes patients with and without microalbuminuria.

Materials and methods
Selection of patients for 2 groups.

**Group1**: 25 Patients with microalbuminuria. Patients with urinary albumin excretion of 30 to 300 micrograms/mg of creatinine were selected. It was done twice within a period of 3 months.
Age groups 40 to 50 yrs.
Patients with systemic infection, UTI, congestive cardiac failure were excluded.

**Group2**: 22 Patients with type 2 diabetes mellitus without microalbuminuria.
Age groups 40 to 50 yrs.
Patients with micro and macro albuminuria, previous history of cardio vascular and cerebrovascular complications were excluded.

Fasting blood samples were collected after twelve hours fasting and two hourly and fourth hourly postprandial samples were collected after giving a fatty meal consisting of 100g of bread and 50g of butter to patients for estimation of plasma glucose, lipid profile, hscrp. Fasting urine samples were tested for microalbuminuria. Patients were taken to radiology department for carotid doppler to measure the intima media thickness of carotid artery on both sides left and right.

**Results**
Fasting and postprandial glucose levels were high in both the groups and there was no significant difference among the groups. FPG > 125 mg/dl. Postprandial plasma glucose greater than 200 mg/dl. Hba1c was more than 8% in both the groups. Fasting triglycerides and postprandial triglycerides were elevated but no difference among the groups. FTGL > 200 mg/dl. Postprandial triglycerides > 250 mg/dl. More than 76% of patients in both groups had LDL value > 100 mg/dl. In hscrp there was no significant difference among the groups but the values were higher than 3 micrograms/ml in both groups. 90% of the patients of both groups had right common carotid artery and left common carotid artery intima media thickness more than 1 mm.

**Discussion**
Our study showed no significant difference among the groups in case of CIMT, but it was increased in both groups irrespective of presence or absence of microalbuminuria. We did not find any correlation between CIMT and fasting triglyceride levels. In our study more than 93% of patients in both groups had high postprandial triglycerides (> 245 mg/dl) after a fat load. But they had no correlation with CIMT among the groups. Yet increased postprandial triglyceride level might be a contributing factor for increase in CIMT. There was a significant elevation of hscrp (> 3 micrograms/ml) to be considered as high risk and mean CIMT was greater than 1 mm in both groups. So, association of carotid atherosclerosis with high CRP levels can be considered but not with urine albumin excretion.
Conclusion
So based on the above observations of our study the possible association of high hsCRP levels with increased CIMT could be considered in the assessment of peripheral arterial disease and on the risk factor analysis for coronary artery disease.

References
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