A DISSERTATION

ON

STUDY OF POSTPRANDIAL HYPERTRIGLYCERIDEMIA AS AN INDEPENDENT RISK FACTOR FOR ISCHEMIC HEART DISEASE

Submitted to
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BRANCH I

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CERTIFICATE

This is to certify that the dissertation entitled **"Study of Postprandial Hypertriglyceridemia as an Independent Risk Factor for Ischemic Heart Disease"** is a bonafide work done by **Dr. V. MEENAKSHISUNDARAM** in **M.D BRANCH I GENERAL MEDICINE** at Government Mohan Kumaramangalam Medical College, Salem, to be submitted to The Tamil Nadu Dr. M.G.R Medical University, in fulfilment of the University Rules and Regulation for the award of M.D. Degree Branch I General Medicine, under my supervision and guidance, during the academic period from January 2007 to July 2008.

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DECLARATION

I solemnly declare that this dissertation **"Study of Postprandial Hypertriglyceridemia as an Independent Risk Factor for Ischemic Heart Disease"** was prepared by me at Government Mohan Kumaramangalam Medical College and Hospital, Salem under the guidance and supervision of **Prof. Dr.R. NATARAJAN, M.D.,** Professor of General Medicine, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in fulfillment of the University regulations for the award of the degree of M.D. Branch I General Medicine.

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CONTENTS

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.

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. Researchers are noticing that Diseases prevalent in western hemisphere are now becoming more and more the prevalent causes of death in Asia.

The most important cause of Coronary Artery Disease is ‘Atherosclerosis’.

The risk factors for atherosclerosis are,

1. Smoking
2. Diabetes mellitus
3. Dyslipidemia
4. Sedentary life style
5. Positive family history
6. Hypertension
7. Obesity

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 nonfasting lipid measurements may be more relevant to artherogenesis.

Typical diets are associated with measurable postprandial lipemia 18 hours per day. A major source of circulating triglycerides is dietaryfat, which after hydrolysis into free fattyacids and glycerides, is transported through the intestinal villi and absorbed by enterocytes where these particles are synthesised into chylomicron - associated Triglycerides for entry into the blood compartment and ultimately storage in adipose tissue.

Postprandial lipids and their associated partially hydrolysed 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. 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

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.

Two articles recently published in the Journal of the American Medical Association directly address these issues by comparing fasting with nonfasting (Postprandial) triglycerides with respect to the prediction of future cardiovascular events.

The first report derived from the Women's Health study cohort showed nonfasting triglycerides maintained a strong independent relationship with future cardiovascular events in fully adjusted analyses.

The second report derived from a prospective cohort study showed nonfasting triglycerides were also found to significantly predict future vascular events in both sexes after multivariate analysis.

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.

In an Asia Pacific cohort studies collaboration study that included date from 26 cohorts, postprandial triglyceride concentrations were a more potent predictor of incident vascular events than were fasting triglycerides.

Our study is aimed at establishing the association between postprandial hypertriglyceridemia and atherosclerosis.

AIMS AND OBJECTIVES

- To study relation between risk factors for atherosclerosis and fasting and postprandial triglyceride levels in patients of unstable angina.

- To establish that post-prandial triglycerides level is a better indicator as a ‘risk factor’ for atherosclerosis.

- To study risk factors for atherosclerosis in diabetic and non-diabetic patients having normal fasting triglyceride.

LIMITATIONS OF THE STUDY

- The sample size should be large for avoiding the significant difference of disparity between group of patients, with large sample size there would be near equal distribution in each group.

- The cut off value of normal postprandial triglyceride is not yet internationally standardized. We had chosen the value, less than 160mg% as “Normal postprandial” triglyceride levels. Many authorities suggest that it should be less than 170mg%.

- The fat proportion of the diet on day the of collecting blood for postprandial triglyceride level assessment needs to be formulated.

MATERIALS AND METHODS

MATERIALS:

1. SELECTION OF PATIENTS:

This study was carried out in reference to 100 patients, diagnosed to have ischemic heart disease who were admitted at Govt. Mohan Kumramangalam Medical College Hospital, Salem for unstable angina. The study was approved by the Department of medicine.

This study was conducted on patients between January 2007 and June 2008.

2. INCLUSION CRITERIA:

1. Unstable angina diagnosed on classical anginal chest pain or anginal chest pain equivalent

With

ECG showing ST segment depression in two consecutive chest leads or Limb leads and Normal S.CPK-MB levels.

2. Fasting S. Triglycerides <150 mg%

3. Fasting S. Cholesterol < 180 mg%

3. EXCLUSION CRITERIA:

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

METHODS:

1. HISTORY

A complete and detailed history of patient with address, occupation, past history of diabetes, hypertension, family history, habits of smoking and alcohol was noted.

2. CLINICAL ASSESSMENT

- 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 (m2)

Classification of obesity was done as per the National Institutes of Health Definition:

Normal range: 18.5-24.9

Over weight: 25-29.9

Obesity:

Class I - 30-34.9

Class II - 35- 39.9

Class III - > 40

Waist circumference was measured at umbilical level. Hip circumference was measured at maximum girth at hip.

Waist hip ratio cut off points > 1.0 for male and > 0.85 for female was considered.

3. EGG:

Recording of ECG was done with 12 leads recording in standard fashion with B.P.L machine.

4. BIOCHEMICAL ASSESSMENTS

- Fasting samples of blood glucose,
- Fasting Lipid profile was measured which includes measurements of
    - S.Cholesterol,
    - S.tryglyceride,
    - HDL,
    - LDL
    - VLDL.
    - Post prandial 2 hr glucose
    - 4hr blood samples for triglycerides levels.

5. PREREQUISITE FOR BIOCHEMICAL INVESTIGATION

1. Fasting lipid profile defined as at least 12 hour fast overnight in which only water is permitted.

2. Fasting blood sample collected in pre-sterile plain and pre-sterile sugar bulbs for fasting lipid profile and fasting blood glucose.
3. After which the patient is allowed to take routine breakfast.
4. Patients are not allowed to take Chocolate, ice cream on the day of investigation.
5. Patients asked to take routine standard meal in their lunch and after four hour of lunch, blood sample was collected for measurement of S. triglycerides.

6. DEFINITIONS

1. Normal Fasting Triglycerides < 150 mg%
2. Normal Post-prandial Triglycerides < 160 mg%
3. Criteria for Diagnosis of Diabetes Mellitus made if one of the following is present,
    a. Symptoms of diabetes plus random blood glucose concentration ≥11.1 mmol/L (200 mg/dL)
    b. Fasting plasma glucose ≥ 7.0 mmol/L (126 mg/dL)
    c. Two-hour plasma glucose ≥ 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test.

Enzymatic methods were used on fresh samples to measure Plasma levels of nonfasting triglycerides (postprandial triglycerides), total cholesterol and high density lipoprotein (HDL) Cholesterol. Low density Cholesterol was calculated by using the Friedewald formula.

7. DATA ANALYSIS

Data analysis done in order of:

A. Profile of Patients

B. Segregation of patients into two groups according to

1. Postprandial Triglyceride levels

Normal : up to 160 mg%

High : > 160 mg%

2. According to presence or absence of Diabetes Mellitus

3. Obese and non-obese.

4. According to Waist-Hip ratio,

C. Statistical analysis was performed and data analyzed in form of calculation of Mean and standard deviation. Calculation and analysis of variance and t-test were used to test statistically significant differences in groups. The significance of test was decided on the basis of P-value. Two tailed P-values < 0.05 were considered significant, chi-square test was applied to test the relationship of two attributes.

REVIEW OF LITERATURE

1. ATHEROSCLEROSIS

- Definition
- Pathogenesis
- Risk Factors
- Lipid metabolism and dyslipidemia

2. DIABETES AND DYSLIPIDEMIA

3. METABOLIC SYNDROME

4. POSTPRANDIAL TRIGLYCERIDEMIA

1. ATHEROSCLEROSIS

Atherosclerosis is the leading cause of death and disability in the developed world. Despite our familiarity with this disease, some of its fundamental characteristics remain poorly, recognized and understood. Although many generalized or systemic risk factors predispose to its development, atherosclerosis affects, various regions of the circulation preferentially and yields distinct clinical manifestations depending on the particular circulatory bed affected.

DEFINITION

Atherosclerosis is disease .of large and medium sized arteries and characterized by endothelial dysfunction, vascular inflammation, and build of lipids, cholesterol, calcium, cellular debris within intima of vessel wall.

This build up results in plaque formation, vascular remodeling, acute and chronic luminal obstruction, abnormalities of blood flow and diminished supply of oxygen to tissue.

PATHOGENESIS AND HISTOLOGY

The initial lesion in atherosclerosis involves the intima of the artery and begins in childhood with the development of fatty streaks.

Early arterial accumulation of cholesterol causes a reduction in arterial distensibility, which occurs before other vessel wall changes become apparent; the loss of distensibility, which increases with age, is related to development of atherosclerosis. A loss of vessel distensibility also occurs with accumulation of cholesterol in the vessel wall, even in young subjects.

Fatty Streaks – The first phase in atherosclerosis histologically presents as focal thickening of the intima with an increase in smooth muscle cells and extracellular matrix. These smooth muscle cells, which are possibly derived from hematopoietic stem cells, migrate and proliferate within the intima. This is followed by accumulation of intracellular lipid deposits or extracellular lipids or both, which produce the fatty streak. Biglycan, a small dermatan sulfate proteoglycan, can be detected in the intima of atherosclerotic ‘coronary’ artery segments; it can bind and trap lipoproteins, including apolipoprotein E, VLDL remnants, LDL, and HDL. The fatty streak

also consists of macrophages with a variable number of T lymphocytes.

As these lesions expand, more smooth muscle cells migrate into the intima. The smooth muscle cells within the deep layer of the fatty streak are susceptible to apoptosis which is associated with further macrophage infiltration and cytoplasmic remnants that can calcify, perhaps contributing to the transition of fatty streaks into atherosclerotic plaques.

Fibrous plaque - The fibrous plaque evolves from the fatty streak via accumulation of connective tissue with an increased number of smooth muscle cells ladened with lipids and often a deeper extracellular lipid pool.

Advanced lesions - More advanced lesions become revascularized from both the luminal and medial aspects, and often contain a necrotic lipid rich core, which can eventually become calcified.

Advanced lesions are associated with coronary artery remodeling. Positive remodeling is· defined as a positive correlation between plaque and external elastic membrane area due to a compensatory increase in local vessel size in response to increasing plaque burden (dilated coronary atherosclerosis), while negative remodeling refers to a smaller external elastic membrane area at the lesion site due to the local shrinkage of vessel size.

PATHOGENESIS

Multiple factors contribute to the pathogenesis of atherosclerosis including

1. Endothelial dysfunction
2. Dyslipidemia
3. Inflammatory and immunologic factors
4. Plaque rupture
5. Smoking
6. Infection

ENDOTHELIAL DYSFUNCTION

Endothelial dysfunction induced by dyslipidemia is an initial step in atherosclerosis. A positive family history aggravates the endothelial dysfunction associated with hypercholesterolemia and age, suggesting additive effects of genetic and environmental risk factors.

Endothelial dysfunction is induced by oxidized LDL, can be worsened by cigarette smoking, and can be reversed with correction of hyperlipidemia by diet or by therapy with a statin.

LIPID METABOLISM AND DYSLIPIDEMIA

Lipoproteins are complexes of lipids and proteins that are essential for the transport of cholesterol, triglycerides, and fat-soluble vitamins. The metabolic consequences associated with changes in diet and lifestyle have increased the number of hyperlipidemic individuals, and hyperlipidemia and atherosclerosis are strongly associated.

Lipoproteins are large, mostly spherical complexes that transport lipids primarily triglycerides, cholesterol, and fat-soluble vitamins through body fluids plasma, interstitial fluid, and lymph to and from tissues. Lipoproteins play an essential role in the absorption of dietary cholesterol, long-chain fatty acids, and fat-soluble vitamins, the transport of triglycerides, cholesterol, and fat-soluble vitamins from the liver to peripheral tissues, and the transport of cholesterol from peripheral tissues to the liver.

TRANSPORT OF DIETARY LIPIDS (EXOGENOUS PATHWAY)

The endogenous pathway of lipoprotein metabolism permits efficient transport of dietary lipids. Dietary triglycerides are hydrolyzed by lipases within the intestinal lumen and are emulsified with bile acids to form micelles.

Dietary cholesterol and retinol are esterified by the addition of a fatty acid in the enterocyte to form cholesteryl esters and retinyl esters, respectively. Longer-chain fatty acids (>12 carbons) are incorporated into triglyceride and packaged with apo B-48, cholesteryl esters, retinyl esters, phospholipids, and cholesterol to form chylomicrons.

Nascent chylomicrons are secreted into the intestinal lymph and delivered via the thoracicduct directly to the systemic circulation, where they are extensively processed by peripheral tissues before reaching the liver. The particles encounter lipoprotein lipase (LPL), which is anchored to proteoglycans that decorate the capillary endothelial surfaces of adipose tissue, heart and skeletal muscle.

The triglycerides of chylomicrons are hydrolyzed by LPL, and free fatty acids are released. ApoC-II, which is transferred to circulating chylomicrons, acts as a cofactor for LPL in this reaction. The released free fatty acids are taken up by adjacent myocytes or adipocytes and either oxidized or reesterified and stored as triglyceride. Some free fatty acids bind albumin and are transported to other tissues, especially the liver. The chylomicron particle

progressively shrinks in size as the hydrophobic core is hydrolyzed and the hydrophilic lipids (cholesterol and phospholipids) on the particle surface are transferred to HDL. The resultant smaller, more cholesterol ester-rich particles are referred to as chylomicron remnants. The remnant particles are rapidly removed from the circulation by the liver in a process that, requires apoE.

TRANSPORT OF HEPATIC LIPIDS (ENDOGENOUS PATHWAY)

The endogenous pathway of lipoprotein metabolism refers to the hepatic secretion and metabolism of VLDL to IDL and LDL. VLDL particles resemble chylomicrons in protein composition but contain apoB-100 rather than apoB-48 and have a higher ratio of cholesterol to triglyceride.

The triglycerides of VLDL are derived predominantly from the esterification of long-chain fatty acids. The packaging of hepatic triglycerides with the other major components of the nascent VLDL particle (apoB-100, cholesteryl esters, phospholipids, and vitamin E) requires the action of the enzyme microsomal Triglyceride transfer protein (MTP).

After secretion into the plasma, VLDL acquires multiple copies of apoE and apolipoproteins of the C series. The triglycerides of VLDL are hydrolyzed by LPL, especially in muscle and adipose tissue. As VLDL remnants undergo further hydrolysis, they continue to shrink in size and become IDL, which contain similar amounts of cholesterol and triglyceride.

The liver removes approximately 40 to 60% of VLDL remnants and IDL by LDL receptor-mediated endocytosis via binding to apoE. The remainder of lDL is remodeled by hepatic lipase (HL) to form LDL. During this process, most of the triglyceride in the particle is hydrolyzed, and all apolipoproteins except apoB-100 are transferred to other lipoproteins.

The cholesterol in LDL accounts for overhalf of the plasma cholesterol in most individuals. Approximatedly 80% of circulating LDL is cleared by LDL receptor-mediated endocytosis in the liver. Lipoprotein(a) [Lp(a)] is a lipoprotein similar to LDL in lipid and protein composition, but it contains an additional protein called apolipoprotein(a) [apo(a)]. Apo(a) is synthesized in the liver and is attached to apoB-100 by disulfide linkage. The major site of clearance of Lp(a) is the liver, but the uptake pathway is not known.

HDL METABOLISM AND REVERSE CHOLESTEROL TRANSPORT

All nucleated cells synthesize cholesterol, but only hepatocytes and enterocytes can effectively excrete cholesterol from the body. The predominant route of cholesterol elimination is by excretion into the bile, either directly or after conversion to bile acids. Cholesterol in peripheral cells is transported from the plasma membranes of peripheral cells to the liver and intestine by an HDL-mediated process termed “reverse cholesterol” transport.

DYSLIPIDEMIA

Lipid abnormalities play a critical role in the development of atherosclerosis. High levels of low density lipoprotein (LDL) and low levels of high density lipoprotein (HDL) are particularly important risk factors for atherosclerosis.

Circulating LDL rapidly accumulates in the cholesterol-enriched macrophages (called foam cells), but not the lipid core, of atherosclerotic plaque. Oxidative modification of LDL is a prerequisite for macrophage via unregulated scavenger receptors and accelerated accumulation of cholesterol. Macrophage uptake of LDL

cholesterol may initially be an adaptive response, which prevents LDL-induced endothelial injury. However, cholesterol accumulation in foam cells leads to mitochondrial dysfunction, apoptosis, and necrosis, with resultant release of cellular proteases, inflammatory cytokines, and prothrombotic molecules.

Oxidized LDL can cause disruption of the endothelial cell surface, promote inflammatory and immune changes via cytokine release from macrophages and antibody production, and increase platelet aggregation. It may also play a role in plaque instability. Levels of oxidized LDL are increased in patients with an acute coronary syndrome and are positively correlated with the severity of the syndrome. HDL, in contrast to LDL, has antiatherogenic properties that include reverse cholesterol transport, maintenance of endothelial function, protection against thrombosis, and maintenance of low blood viscosity through a permissive action on red cell deformability.

Several large prospective observational studies and intervention trials show a strong positive relationship between raised TGL and CAD – a dose response relationship. Hypertriglyceridemia is an

independent risk factor for coronary artery disease when it is associated with low HDL and/or raised LDL.

There is an inverse relation between HDL and CAD. The mechanism by which HDL confers decreased risk of CAD is poorly understood.

1. They indirectly initiate TGL catabolism and remnant removal.
2. They accept free cholesterol from cells and play a key role in cholesterol transport.

Lipoprotein (a) [LP (a)] is an independent risk factor for CAD. The level of LP (a) is genetically determined, occurs as a complex between LDL and plasma apolipoprotein (a). It is ten times more atherogenic than LDL. Levels > 30 mg/dl is associated with CAD risk.

PLAQUE HAEMORRHAGE

As atherosclerotic plaques develop and expand, they acquire their own microvascular network (vasa vasorum) extending from the adventitia through the media and into the thickened intima. These

thin-walled vessels are prone to disruption, leading to haemorrhage within the substance of the plaque.

PLAQUE RUPTURE

Atherosclerosis is generally asymptomatic until the plaque stenosis exceeds 70 or 80 percent, which can produce a. critical reduction in flow, as with. coronary blood flow to myocardium. These large lesions can produce typical symptoms of angina pectoris. However, acute coronary and cerebrovascular syndromes (unstable angina, myocardial infarction, sudden death, and stroke) are typically due to rupture of plaques with less than 50 percent stenosis. Plaque rupture may also be silent; repeated silent ruptures and thrombosis, followed by wound healing, may cause progression of atherosclerosis, with an increase in plaque burden and percent stenosis and negative arterial remodeling.

INFECTION

Chronic infection may contribute to the pathogenesis of atherosclerosis. The major organisms that have been studied are Chlamydia pneumoniae, cytomegalovirus (CMV), and Helicobacter

pylori; however, hepatitis A virus (HAV) and herpes simplex virus (HSV) type 1 and type 2 have also been implicated.

DIABETIC DYSLIPIDEMIA

Alteration of lipid metabolism in Diabetics:

1. Hypertriglyceridemia
2. Decreased HDL cholesterol
3. Decreased HDL2/HDL3 ratio
4. Decreased lipoprotein lipase activity
5. Increased hepatic triglyceride activity
6. Prolonged postprandial lipemia
7. increased lipoprotein (a) in presence of diabetic nephropathy

Hypertriglyceridemia is associated with Insulin resistance and Type 2 Diabetes Mellitus. There is good correlation between insulin resistance and plasma TGL concentration, has TGL may influence an early step in the insulin action pathway. Alternatively insulin resistance may cause Hypertriglyceridemia. A contributory factor to Hypertriglyceridemia in type 2 diabetes is the inability of insulin to inhibit acutely the release of VLDL from the liver, despite the efficient

suppression of non-esterified fatty acids impairment in VLDL and chylomicron removal due to decreased Lipoprotein lipase activity in the adipose tissue also occurs in diabetic subjects. The volume of VLDL particles is of major importance in Atherogenicity by determining the size and cholesterol content of the LDL formed. Large VLDL particles with High TGL content are the precursors of small dense LDL, which determine the severity of Atherosclerosis. The association of low HDL with Hypertriglyceridemia is due to the fact that TGL – enriched HDL – Cholesterol is hydrolysed by Hepatic lipase and small dense HDL particles are formed, which are cleared from circulation, leading to low serum levels of HDL cholesterol.

METABOLIC SYNDROME

The Metabolic syndrome, as defined by the NCEP, is not to be confused with the insulin resistance syndrome although such resistance is likely but not always in patients with metabolic syndrome. Based on the criteria of the NCEP, the diagnosis of metabolic syndrome is highly effective in predicting excess cardiovascular risk.

National Cholestrol Education Program, Adult Treatment Panel III (NCEP : ATP III) 2001 criteria for the Metabolic Syndrome.

Three or more of the following.

- Central Obesity: Waist Circumference > 102 cm (M), >88 cm (F).
- Hypertirglyceridemia : Triglycerides ≥ 150 mg/dL. (or) Specific Medication.
- Low HDL Cholestrol : < 40 mg/dL and < 50 mg/dL, respectively, (or) specific Medication.
- Hypertension : Blood Pressure ≥ 130 mmHg Systolic (or) ≥ 85 mmHg diastolic (or) Specific Medication.
- Fasting Plasma Glucose : ≥ 100 mg/dL (or) Specific Medication (or) Previously diagnosed type 2 Diabetes.

International Diabetes Foundation (IDF) criteria for Central Adiposity.

WAIST CIRCUMFERENCE

Men	Women	Ethnicity
≥ 94 cm	≥ 80 cm	Europid, Sub-Saharan African, Eastern & Middle Eastern
≥ 90 cm	≥ 80 cm	South Asian, Chinese, South & Central American
≥ 85 cm	≥ 90 cm	Japanese

Two or more of the following:

- Fasting Triglycerides > 150 mg/dL (or) Specific Medication.
- HDL Cholestrol < 40 mg/dL and < 50 mg/dL for men and women, respectively, or Specific Medication.
- Blood Pressure > 130 mmHg Systolic or > 85 mmHg diastolic or previous diagnosis or Specific Medication.
- Fasting Plasma glucose ≥ 100 mg/dL or previously diagnosed Type 2 diabetes.

POSTPRANDIAL HYPERTRIGLYCERIDEMIA:

The plasma level of triglyceride varies widely through the day. TGL levels are elevated for most of the day even in subjects with Normal fasting TGL. The status of fasting TGL as a risk factor for coronary artery disease has been considered to be weak because in

multi-variate analysis, the effect of TGL tends to be eliminated by a low HDL cholesterol level elevated postprandial TGL levels have been observed in persons with fasting Hypertriglyceridemia, in smokers and there is also growing evidence that elevation of postprandial TGL is associated with endothelial dysfunction, and with thrombogenic alterations in the coagulation system.

Severe postprandial Hypertriglyceridemia is associated with an inflammatory state and enhanced production of TNF-, IL-, and C-reactive protein.

Postprandial triglyceride levels, which indicate the presence of remnant lipoproteins, are associated with increased risk of Ischemic heart disease. Most previous studies on triglycerides have focused on fasting levels that exclude remnant lipoproteins.

This Postprandial Triglyceride – rich remnant lipoproteins penetrate the endothelium and appear to be preferentially trapped within the arterial wall.

The Copenhagen city Heart Study, a prospective cohort study of Danish general population, found that nonfasting postprandial

Triglyceride levels independently predict Myocardial infarction, Ischemic heart disease and death in men and women.

The adverse effect of postprandial triglyceride on endothelial dysfunction has been reported in both normal and diabetic patients. Both hypertriglyceridemia and hyperglycemia induce endothelial dysfunction, Oxidative stress to be common mediator of such effects.

There is over production of superoxide anion, which in turn inactivates nitric oxide, produces peroxynitrate, a potent and long living nitrating agent.

This peroxynitrate leads to a host of potentially injurious events, including VLDL oxidation, depletion of antioxidant defence, and inactivation of enzymes. Oxidant can be directly cytotoxic to endothelial cell. All these events are involved in pathogenesis of atherosclerosis.

The observations demonstrated good relationship between postprandial lipemia and atherosclerosis and suggested that postprandial triglyceride levels may be better indicator of

atherogenesis than fasting TGs levels. Postprandial lipemia can be an independent risk factor for atherosclerosis.

However in all trials interval of measuring triglycerides are different and some trial used fat load, instead of normal diet. So precise guidelines for interval for measuring TGs is yet to be formulated and normal cutoff values for postprandial TGs levels are to be decided.

OBSERVATION AND DATA ANALYSIS

TABLE - 1

DISTRIBUTION ACCORDING TO AGE AND SEX

Age	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 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 of 47.10 ± 5.2 years.

TABLE - 2

DISTRIBUTION ACCORDING TO BODY MASS INDEX AND GENDER

BMI	Male	Female	Total
Normal	5	3	8
Over weight	25	15	40
GR – I Obesity	16	19	35
GR – II Obesity	8	6	14
GR – III Obesity	2	1	3
Total	**56**	**44**	**100**

In our study, 40 patients were over weight, 52 were obese, and only 8 patients had normal weight. 25 male (62.5%) were over weight, 26 male (50%) were obese, 5 male (62.5%) had normal weight. In 44 females, 15 females (37.5%) were overweight, 26 females (50%) were obese, 3 females (37.5%) had normal weight.

TABLE - 3

DISTRIBUTION OF PATIENTS ACCORDING TO WAIST-HIP RATIO AND SEX

W-H Ratio	Male	Female	Total
Normal	16	11	27
High	40	33	73
Total	**56**	**44**	**100**

In the present study, 40 male patients (71.4%) and 33 female patients (75%) had high waist-hip ratio.

TABLE - 4

PRESENCE OF DIABETES MELLITUS

D.M.	Male	Female	Total
Presence	34	30	64
Absence	22	14	36
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 - 5

PRESENCE OF HYPERTENSION

Hypertension	Male	Female	Total
Presence	32	25	57
Absence	24	19	43
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 - 6

DISTRIBUTION ACCORDING TO PP4TG

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

In this study, out of 100 patients, 64 patients showed S.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 hypertrigly ceridemia.

TABLE - 7

DISTRIBUTION ACCORDING TO F. HDL

HDL	Male	Female	Total
Low	32	26	58
Normal	24	18	42
Total	**56**	**44**	**100**

In this study, 58 patients had low fasting HDL level (less than 40 mg% in Male, less than 50 mg% in Female), 36 out of 56 male (64.28%) and 22 out of 44 female (50%) had low fasting HDL level.

TABLE - 8

BASELINE CHARACTERISTICS

Characteristic	Mean	Std. Errs.
Age	52.06	0.8
B.M.I.	30.08	0.5
W-H Ratio	0.99	0.011
SBP	135.82	1.66
DBP	88.59	1.075
FBS	155.31	3.6
PP2BS	207.69	5.2
F.HDL	50.89	0.75
F.LDL	94.88	1.29
PP4TG	181.47	2.148

In this study, out of 100 patients, 64 patients showed S.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 hypertrigly ceridemia.

TABLE - 9

DISTRIBUTION ACCORDING TO AGE AND PP4TG

Age	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**

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 - 10

DISTRIBUTION ACCORDING TO WAIST-HIP RATIO AND PP4TG

W-H Ratio	Normal PP4TG	High PP4TG	Total
Normal	22	2	24
High	14	62	76
Total	**36**	**64**	**100**

In the present study, out of 76 patients having high waist-hip ratio, 62 (81.5%) had high postprandial triglyceridemia, while 14 patients had normal Postprandial triglycerides. Out of 24 patients having normal waist-hip ratio, 2 had high postprandial triglyceride levels, while 22 had normal postprandial triglyceride levels.

The calculated P-value was < 0.01, suggesting association between High waist-hip ratio and High PP4TG level.

TABLE - 11

DISTRIBUTION ACCORDING TO BMI AND PP4TG

W-H Ratio	Normal PP4TG	High PP4TG	Total
Normal	6	2	8
Overweight	16	24	40
High	14	38	52
Total	**36**	**64**	**100**

In the present study, out of 52 patients having high body mass index, 38 patients (73.07%) had high postprandial triglyceride levels. While out of 40 patients having Body Mass Index in overweight range 24 patients had (60%) high postprandial triglyceride levels. And 2 patients out of 8 patients (25%) having normal Body Mass Index had high PP4TG levels. (P value <0.05 by Chi-square method)

TABLE - 12

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 S.Triglyceride level, out of 36, 14 had low fasting S.HDL and 22 had normal fasting S.HDL. There is no relation found between HDL level and High PP4TG levels (P value is 0.34).

TABLE - 13

DISTRIBUTION ACCORDING TO DIABETES AND PP4TG

Diabetes	Normal PP4TG	High PP4TG	Total
Absent	23	9	32
Present	13	55	68
Total	**36**	**64**	**100**

In the present study, 64 patients had high postprandial S.triglyceride level, out of 64 patients 55 were diabetic. Only 9 patients were not diabetic. In the remaining 36 patients who had normal postprandial S.triglyceride level, 13 were diabetic and 23 were non-diabetic.

There was strong association found between Diabetes Mellitus and High PP4TG levels. The calculated odd ratio is 10.8.

TABLE - 14

DISTRIBUTION ACCORDING TO HYPERTENSION AND PP4TG

Hypertension	Normal PP4TG	High PP4TG	Total
Absent	25	18	43
Present	11	46	57
Total	**36**	**64**	**100**

In the present study, 64 patients had high postprandial S.triglyceride level. Out of 64 patients 46 were hypertensive. Only 18 patients were not hypertensive. In the remaining 36 patients who had normal postprandial S.triglyceride level, 11 were hypertensive and 25 were non-hypertensive. There was an association found between Hypertension and High PP4TG level (P value < 0.05).

DISCUSSION

AGE AND GENDER

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)

OBESITY AND WAIST-HIP RATIO

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.

DIABETES MELLITUS

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. A study done by Mette 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.

TRIGLYCERIDE

In this study, out of 100 patients, 64 patients showed serum triglyceride level more than 160mg% 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.

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.

HYPERTENSION

In our study 57 patients were hypertension. 32 male patients (57.14%) were Hypertensive and 25 female (56.81%) were hypertensive.

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.

In Kolovou et al study on postprandial Lipemia in Hypertension suggest that patient with hypertension have an exaggerated response and delayed clearance of plasma TGL concentration.

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.

There is statistically a significant correlation found between postprandial Hypertriglyceridemia and high waist-hip ratio and Diabetes Mellitus.

SUMMARY

In the study entitled **"Postprandial Hypertriglyceridemia as a Independent Risk Factor for Ischemic Heart Disease"** conducted at Govt. Mohan Kumaramangalm Medical College Hospital, Salem from July 2007 to June 2008, a total of 100 patients of ischemic Heart disease were included as per defined criteria.

Postprandial Hypertriglyceridemia was found in 64%, 52% had high BMI, 73% patients had High Waist Hip ratio and 64% patients had Diabetes Mellitus. Increased triglyceride level is a risk factor for cardiovascular disease independent of HDL cholesterol level.

Our study showed patients of ischemic Heart disease have high postprandial triglyceride levels inspite 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.

Non fasting triglyceride levels indicate the presence of remnant lipoproteins, which may promote atherosclerosis. Postprandial Hypertriglyceridemia may be an independent risk factor for Atherosclerosis in Ischemic Heart Disease patients.

Evaluation of postprandial triglyceride levels is important during assessment of ischemic heart disease patients.

FUTURE PROSPECTS

After establishment of postprandial lipemia as a better marker for atherosclerosis in large population group based prospective trial, we can,

1. Design future trials of agents aimed at reducing triglyceride levels or attenuating atherogenic metabolic abnormalities.
2. Study of morbidity and mortality in patients of ischemic heart disease between patients on treatment for control of postprandial lipemia with patients without treatment of postprandial lipemia.

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PROFORMA

Name:	Age:	Sex:
Place:	Occupation:	
Hospital No:	Date of Admission:	
	Date of Discharge:	

CHIEF COMPLAINTS:

PAST HISTORY:

Diabetes:

Hypertension:

C.V.stroke:

Dyslipidemia:

PERSONAL HISTORY:

Smoking:

Alcohol

Sedentary life style

FAMILY HISTORY:

GENERAL PHYSICAL EXAMINATION:

Pulse:

Blood Pressure:

Temperature:

Locomotor Brachialis:

Carotid Bruit:

Cord like Artery:

Height: Waist:

Weight: Hip:

Body mass index: Waist hip ratio:

CARDIOVASCULAR EXAMINATION:

OTHER SYSTEMS: RESPIRATORY SYSTEM:

ABDOMEN:

CNS:

INVESTIGATIONS:

Fasting blood sugar:

Post prandial blood sugar:

ELECTRO CARDIOGRAM:

ST DEPRESSION IN LEADS:

S. CPK MB :

FASTING

S. Cholesterol:

S. Triglyceride:

S. HDL

S. LDL

S. VLDL

POST - PRANDIAL TRIGLYCERIDE:

ABBREVIATIONS

TGL	:	Triglycerides
CAD	:	Coronary Artery Disease
HDL	:	High density Lipoprotein
LDL	:	Low density Lipoprotein
VLDL	:	Very Low density Lipoprotein
ECG	:	Electro Cardiogram
LPL	:	Lipoprotein Lipase
M.I	:	Myocardial infarction
S.CPK-MB	:	Serum Creatinine phosphokinase – MB
PP4 TG	:	Postprandial Triglycerides 4hours after meal
HW	:	Housewife
DL	:	Daily Labour
BM	:	Business Man
PP2BS	:	Postprandial Blood sugar 2hours after meal