



Study of Lipid Profile Variability in Diabetic Patients of Bihar State

KEYWORDS

Diabetics, GOD-POD method, total cholesterol(TC), Triglyceride(TG), low density cholesterol(LDL), CAD- coronary artery disease

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ABSTRACT

The study was conducted in the department of physiology, Patna medical college and hospital, Patna. Forty patients of diabetes aged 40-50 years attending OPD of PMCH and sixty healthy volunteers were recruited in the present study for lipid profile variability (TC, TG, HDL, LDL, VLDL) using GOD-POD method and enzymatic method. TC, TG and LDL showed a partially significant upper range in diabetics with $p(>0.05)$ while HDL and VLDL showed no variations between these two group with $p(>0.1)$. It was concluded that diabetics showed partially significant variation in lipid level as compared to non- diabetics.

Introduction

More cardiovascular diseases occur in patients with either type of diabetes. The link between diabetes and atherosclerosis, is however, not completely understood. Among the metabolic abnormalities that commonly accompany diabetes are disturbances in the production and clearance of plasma lipoproteins. Moreover, development of dyslipidemia may be a harbinger of future diabetes. A characteristic pattern termed diabetic dyslipidemia, consists of increased TG, low HDL and postprandial lipemia. This pattern is most commonly seen type 2 diabetes and may be a treatable risk factor of subsequent cardiovascular disease.

Diabetes and CAD are inextricably intertwined; one tends to lead to the other within 10 to 20 years. Closely linked with diabetes is the entity of metabolic syndrome. Abdominal obesity, high TG, low HDL, prehypertension (BP > 130/85 mm of Hg) and impaired fasting glucose are the five key components of metabolic syndrome. The prevalence of metabolic syndrome using NCEP criteria is higher in Indian (26%) than whites (22%) and double that in Chinese (11%).

Metabolic syndrome is typically associated with atherogenic phenotype B characterised by hypertriglyceridemia, low HDL, high proportion of small dense LDL, high level of apolipoprotein B and low level of low level of apolipoprotein A-1 and this explains the very high risk of CAD associated with metabolic syndrome.

Odimon and colleague (1993) charted the role of hyperglycemia in thrombus formation. Haller(1997) discussed in detail the vascular complications associated with postprandial hyperglycaemia. He concluded that cell adhesion, endothelial permeability, endothelium dependent vaso relaxation and matrix expression participate together in the development of diabetic macrovascular and microvascular changes.

Pathogenesis of Atherosclerosis in Diabetes

The cause of vasculopathic state in diabetes is a confluence of multiple metabolic derangements, which include hyperglycemia, dyslipidemia and disorder of the coagulation system. Moreover the other risk factors such as hypertension, obesity and advanced age are frequently present in addition to these metabolic derangements.

HYPERINSULINEMIA: - insulin excess has been proposed as a contributing factor to premature atherosclerosis.

DYSLIPIDEMIA:- patients with diabetes have high LDL, TG, VLDL and low HDL both of which increases atherogenicity.

ABERRANCE OF COAGULATION SYSTEM:- platelets of patients with diabetes exist in a relatively activated state resulting in increased thromboxane A2 release with resultant vasoreactivity and platelet aggregability.

MATERIALS AND METHODS

Blood samples were obtained after an overnight fast. About 5 ml of blood was collected from the left antecubital vein. Out of which about 2 ml is transferred into an OF vial and mixed well and centrifuged at a speed of 3000 revolutions per minute for 10 min to separate the plasma, which was used for biochemical analysis. Rest 3 ml of blood is transferred to the test tube and this blood was allowed to clot to get serum. This serum was separated in a centrifuge tube at 3000 revolutions per min to get a clear sample of serum. This clear supernatant serum was used for biochemical investigations.

Method of glucose estimation

By GOD-POD method

Sample- fresh centrifuged plasma

Principle- glucose oxidase is an enzyme extracted from the growth medium of *Aspergillus niger*. It catalyses the oxidation of Beta D- glucose present in the plasma to D glucono- 1, 5- lactone with the formation of hydrogen peroxide; lactone is slowly hydrolysed to gluconic acid. The hydrogen peroxide produced is then broken to oxygen and water by a peroxidase enzyme. Oxygen then react with an oxygen acceptor such as ortho toluidine to form a coloured compound, the amount of which can be measured colorimetrically.

Estimation Of Serum Total cholesterol

Method - Enzymatic- (Colorimetric Trinder End point)

The reagents were allowed to attain room temperature prior to use.

	B L A N K	S T A N D A R D	S A M P L E
R e a g e n t R	1 0 0 0 μ L	1 0 0 0 μ L	1 0 0 0 μ L
S t a n d a r d	- - - - -	1 0 μ L	- - - - -
S a m p l e	- - - - -	- - - - -	1 0 μ L

They were incubated for 5 min. at 37°C and reading was done against blank at 500nm and calculation was made. The concentration of cholesterol in the sample is directly proportional to the intensity of red complex(red quinone),which was measured at 500nm.

CALCULATION

Cholesterol = absorbance of sample/ absorbance of standard x concentration of standard

REFERENCE VALUES: < 200 mg/dl

ESTIMATION OF SERUM TRIGLYCERIDE METHOD: (Enzymatic-colorimetric method

contents were mixed and incubated for 5 min at 37 degree Celsius. The reading was done against blank at 546 nm.

	B L A N K	STANDARD	S A M P L E
R e a g e n t R	1 0 0 0 μ L	1 0 0 0 μ L	1 0 0 0 μ L
S t a n d a r d	- - - - -	1 0 μ L	- - - - -
S a m p l e	- - - - -	- - - - -	1 0 μ L

CALCULATION

Serum TG = absorbance of Sample / absorbance of Standard x n

n = Standard Concentration

REFERENCE VALUES:> 150 mg/dl.

ESTIMATION OF HDL-CHOLESTEROL

Method – phosphotungstate method

Principle – chylomicrons, LDL and VLDL are precipitated by addition of phosphotungstic acid and magnesium chloride. After centrifugation, the high Density lipoprotein (HDL) fraction remains in the supernatant is determined with CHOD-PAP method.

Reference value- >40mg/dl

Calculation Of LDL & VLDL by Friedwald’s Formula:

LDL = TC – (HDL + VLDL)

VLDL = TG / 5

Reference Value

LDL = Up to 190 mg/dl

VLDL = 14 -31.8 mg/dl

OBSERVATION

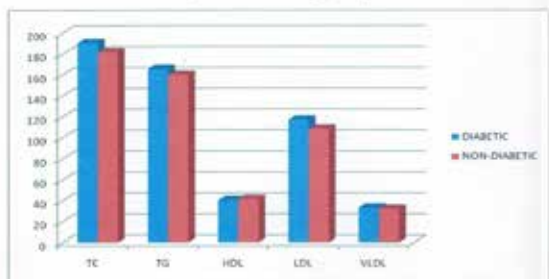
Total cholesterol, triglyceride and LDL showed a partially significant upper range in diabetics while HDL and VLDL showed no variations between these two groups. This may be due to the fact that they were on anti- diabetic drugs or were not the known case of diabetes.

TABLE – I
LIPID LEVEL VARIATIONS AMONG DIABETIC AND NON- DIABETIC (mean ±SD) in mg/dl

	Diabetic n = 40	Non-diabetic n =60	t	p	Significance
Total cholesterol	190.47 ± 20.98	181.83 ± 16.21	1.19	>0.05	PS
Triglyceride	165.70 ± 17.56	160.21 ± 16.62	1.93	>0.05	PS
HDL	40.29 ± 6.31	41.43 ± 7.03	0.05	>0.1	NS
LDL	117.06 ± 21.75	108.36 ± 14.62	1.18	>0.05	PS
VLDL	33.12 ± 3.32	32.04 ± 3.51	0.93	>0.1	NS

Total cholesterol, triglyceride and LDL showed a partially significant upper range in diabetics while HDL and VLDL showed no variations between these two groups.

LIPID LEVEL VARIATIONS AMONG DIABETIC AND NON-DIABETIC (mean in mg/dl)



DISCUSSION

People with diabetes are more prone to having unhealthy high cholesterol levels, which contributes to cardiovascular diseases. The overall prevalence of CAD as assessed by various diagnostic methods is as high as 55% in adult patients with DM as compared with 2-4% for general population. Workers like Bryfogle and Bradley(1957) and Goldenberg et al (1958) have established that diabetes have a greater risk of mortality and morbidity from CAD than non-diabetics. Clinical management of diabetes mellitus requires effective laboratory assessment of lipoprotein abnormalities.diabetes may cause or exacerbate quantitative and/or qualitative changes in lipoproteins. Furthermore, diabetic complications may cause secondary dyslipidemia while important forms of primary dyslipidemia may co- exist with diabetes.

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