

Lipid Profile and Its Relationship with Diabetic Patient in Metabolic Syndrome

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Abstract – *Dyslipidemia was visit in patients with Met S. High TG was the most widely recognized lipid variation from the norm, and a vast number of patients had more than one strange lipid parameter. In light of their particular blood glucose levels, an A indistinguishable example of dyslipidemia was seen in the investigation populace Insulin protection and sort 2 diabetes are related with a bunching of interrelated plasma lipid and lipoprotein variations from the norm, which incorporate lessened HDL cholesterol, a power of little thick LDL particles, and hoisted triglyceride levels. Each of these dyslipidemia highlights is related with an expanded danger of cardiovascular infection. Expanded hepatic emission of substantial triglyceride-rich VLDL and debilitated leeway of VLDL has all the earmarks of being of focal significance in the pathophysiology of this dyslipidemia. Little thick LDL particles emerge from the intravascular preparing of particular bigger VLDL forerunners. Commonly, lessened plasma HDL levels in sort 2 diabetes are show as diminishments in the HDL2b subspecies and relative or total increments in littler denser HDL3b and HDL3c. Albeit behavioral intercessions, for example, eating regimen and exercise can enhance diabetic dyslipidemia, for most patients, pharmacological treatment is expected to achieve treatment objectives. There are a few classes of solutions that can be utilized to treat lipid and lipoprotein irregularities related with insulin protection and sort 2 diabetes, including statins, fibrates, niacin, and thiazolidinedione's. Clinical trials have demonstrated huge change in coronary course illness after diabetic dyslipidemia treatment.*

Keywords: Lipid Profile, Blood Sugar, Metabolic Syndrome.

INTRODUCTION

Diabetes mellitus is a heterogeneous gathering of disarranges described by industrious hyperglycemia. The two most normal types of diabetes are sort 1 diabetes (T1D, beforehand known as insulin dependent diabetes or IDDM) and sort 2 diabetes (T2D, beforehand known as non-insulin-subordinate diabetes or NIDDM). Both are caused by a blend of hereditary and ecological hazard factors. Notwithstanding, there are other uncommon types of diabetes that are straightforwardly acquired. These incorporate development beginning diabetes in the youthful (MODY), and diabetes because of transformations in mitochondrial DNA.

All types of diabetes have intense impacts on wellbeing. Notwithstanding the results of anomalous digestion of glucose (e.g., hyperlipidemia, glycosylation of proteins, and so on.), there are various long haul difficulties related with the ailment. These incorporate cardiovascular, fringe vascular, visual, neurologic and renal variations from the norm, which are in charge of dreariness, inability and unexpected

passing in youthful grown-ups. Moreover, the illness is related with conceptive intricacies causing issues for the two moms and their kids. Albeit enhanced glycemic control may diminish the danger of building up these intricacies, diabetes remains an exceptionally noteworthy reason for social, mental and monetary weights in populaces around the world.

Type 1 Diabetes

Epidemiology. T1D is caused by the autoimmune destruction of the beta cells of the pancreas, and represents approximately 10% of all cases with diabetes. At present, lifelong insulin therapy is the only treatment for the disease. Without exogenous insulin injections, individuals with T1D will not survive.

The incidence of T1D is increasing worldwide at a rate of about 3% per year (Adiels, et. al., 2008). This trend appears to be most dramatic in the youngest age groups, and is completely unrelated to the current increase in T2D in children. More children with beta cell autoantibodies, a hallmark of T1D, are being diagnosed with the T1D around the world each year.

Although the peak age at onset is at puberty, T1D can also develop in adults. Epidemiologic studies have revealed no significant gender differences in incidence among individuals diagnosed before age 15 (Goodarzi, et. al., 2014). However, after age 25, the male to female incidence ratio is approximately 1.5. There is also a notable seasonal variation in the incidence of T1D in many countries, with lower rates in the warm summer months, and higher rates during the cold winter.

Type 2 Diabetes

Epidemiology. T2D is the most common form of the disease, accounting for approximately 90% of all affected individuals. A diagnosis of T2D is made if a fasting plasma glucose concentration is > 7.0 mmol/L (> 126 mg/dl) or plasma glucose 2 hours after a standard glucose challenge is > 11.1 mmol/L (> 200 mg/dl) (WHO, 1999). T2D is caused by relative impaired insulin secretion and peripheral insulin resistance. Typically, T2D is managed with diet, exercise, oral hypoglycemic agents and sometimes exogenous insulin. However, it is associated with the same long-term complications as T1D.

Dyslipidemia is one of the major cardiovascular disease (CVD) risk factors and plays an important role in the progress of atherosclerosis, the underlying pathology of CVD. The prevalence of dyslipidemia in type 2 diabetes is double with respect to the general population (Chahal and Ginsberg, 2006). These are more complex abnormalities that are caused by the interrelation among obesity, insulin resistance and hyperinsulinism (Assman and Schulte, 1992).

When the overweight subjects were compared with their respective thinner counterparts, they presented 2.4 to 7.1 times higher probability to have an elevated total cholesterol, LDL cholesterol, triglycerides and blood pressure as well as 12.6 times higher probability to have hyperinsulinemia.

It is worth to emphasize that the fatty tissue is exclusively related to risk factors, such as the altered insulin and lipid profile, which can contribute to the development of the insulin resistance syndrome, which comprises several risk factors for the emergence of cardiovascular complications (Jeppesen, et. al., 1997).

In patients with type 2 diabetes, which is equivalent to CHD (Assman and Schulte, 1992), it is most commonly characterized by elevated TG and reduced HDL-C (Kawamoto, et. al., 2011). These abnormalities can be present alone or in combination with other metabolic disorders. The prevalence of dyslipidaemia varies depending on the population studied, geographic location, socioeconomic development and the definition used (Alberti, et. al., 2006). (Haffner, et.

al., 1990). Very few cross-sectional studies have evaluated the relationship between lipid and blood glucose concentrations in type 2 diabetics in Punjabi population. The present study was planned to identify the prevalence of abnormalities in lipid profile among type 2 diabetic Punjabi population.

MATERIAL AND METHODS

This study included 80 male patients with MetS, whereas those with history of CVD, thyroid disorders, or currently on lipid-lowering agents were excluded. Detailed history was noted and clinical examination was carried out. Body mass index (BMI), waist circumference (WC), and systolic and diastolic blood pressure were measured using standard methods.

Laboratory assessment included venous blood samples in a fasted state for the determination of components of the lipid profile [total cholesterol (TC), HDL-C, and TG] and blood glucose levels. The serum glucose was measured using the glucose oxidase/peroxidase method and the lipid profile by the enzymatic colorimetric method. LDL-C was calculated from the formula of (Friedewald, et. al., 1972).

MetS was defined as per JIS (Joint Interim Statement) criteria (Alberti, et al., 2009). Accordingly, MetS was attributed in patients if three or more risk determinants were present: increased WC (>90 cm), elevated TG (>150 mg/dL), low HDL-C (<40 mg/dL), hypertension ($>130/85$ mmHg), and impaired fasting glucose (IFG; >100 mg/dL). Dyslipidemia was defined according to ATP-III guidelines (NCEP, 2002).

Patients were categorized into three groups depending on their fasting blood glucose levels (Alberti and Zimmet, 1998). Group I comprised patients with normal fasting glucose (NFG; <100 mg/dL), Group II had patients with IFG status ($100-125$ mg/dL), and Group III had patients with T2DM (>126 mg/dL).

Statistical Analysis: Intergroup comparisons were done using Pearson's χ^2 -test, and mean values were compared using analysis of variance. Statistically significant differences were reported at $p < 0.05$.

RESULT

The baseline characteristics of 72 patients with MetS show that their mean age (years) was 50.18 ± 9.63 , BMI (kg/m^2) 26.71 ± 3.16 , and WC (cm) 98.61 ± 7.64 . Biochemical analysis showed that mean fasting blood sugar was 121.33 ± 37.39 mg/dL whereas HDL, TG, TC, and LDL-C were 45.36 ± 6.34 , 195.11 ± 68.10 , 194.48 ± 38.44 , and 109.75 ± 34.70 mg/dL, respectively (Table 1).

Table 1: Baseline characteristics of subjects (n = 72)

Parameters	Mean ± SD	Parameters	Mean ± SD
Age (year)	50.18 ± 9.63	Age (year)	50.18 ± 9.63
BMI (kg/m ²)	26.71 ± 3.16	BMI (kg/m ²)	26.71 ± 3.16
Waist circumference (cm)	98.61 ± 7.64	Waist circumference (cm)	98.61 ± 7.64
SBP (mmHg)	135.12 ± 15.80	SBP (mmHg)	135.12 ± 15.80
DBP (mmHg)	88.22 ± 10.16	DBP (mmHg)	88.22 ± 10.16
Fasting blood sugar (mg/dL)	121.33 ± 37.39	Fasting blood sugar (mg/dL)	121.33 ± 37.39
Serum HDL-C (mg/dL)	45.36 ± 6.34	Serum HDL-C (mg/dL)	45.36 ± 6.34
Serum triglycerides (mg/dL)	195.11 ± 68.10	Serum triglycerides (mg/dL)	195.11 ± 68.10
Total serum cholesterol (mg/dL)	194.48 ± 38.44	Total serum cholesterol (mg/dL)	194.48 ± 38.44
Serum LDL-C (mg/dL)	109.75 ± 34.70	Serum LDL-C (mg/dL)	109.75 ± 34.70

HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

Table 2: Prevalence of optimal, suboptimal, and high lipid levels in metabolic syndrome (n = 72)

Lipid profile	Suboptimal	High
TG* 19 (26.38%)	21 (29.16%)	32 (44.44%)
LDL-C** 31 (43.05%)	34 (47.22%)	7 (9.72%)
TC*** 43 (59.72%)	19 (26.39%)	10 (13.89%)

TG, serum triglycerides; LDL-C, low-density lipoprotein cholesterol; TC, serum total cholesterol.

* TG: Optimal - <150 mg/dL; Suboptimal -150-199 mg/dL; High: >200 mg/dL.

** LDL-C: Optimal - <100 mg/dL; Suboptimal - 100-159 mg/dL; High: >160 mg/dL.

*** TC: Optimal - <200 mg/dL; Suboptimal - 200-239 mg/dL; High: >240 mg/dL.

Table 3

TG > 200 mg/dL	9 (52.9%)	13 (26.50%)	10 (34.5%)	0.563
LDL-C > 160 mg/dL ⁰	3 (11.5%)	4 (13.8%)		0.584
TC > 240 mg/dL	0	6 (23.1%)	4 (13.8%)	
HDL-C < 40 mg/dL (35.3%)	1 (3.8%)	7 (24.1%)		0.079

TG, serum triglycerides; LDL-C, low-density lipoprotein cholesterol; TC, serum total cholesterol; NFG, normal fasting glucose; IFG, impaired fasting glucose; T2DM, type 2 diabetes mellitus.

Table 4: Mean values of serum lipids (mg/dL) among three groups

.. NFG	(n = 17), IFG (n = 26), T2DM (n = 29),			
Lipid mean ± SD	mean ± SD	mean ± SD	profile (Group 1)	p-Value
(Group 2)	(Group 3)			
TG	211.82 ± 57.92	201.88 ± 64.33	179.24 ± 75.34	0.243
LDL-C	102.2 ± 28.46	113.74 ± 39.56	110.61 ± 33.82	0.565
TC	189.06 ± 27.61	201 ± 43.21	191.83 ± 39.76	0.549
HDL-C	43.35 ± 6.40	46.81 ± 6.01	45.24 ± 6.48	0.217

TG < 150 mg/dL, LDL < 100 mg/dL, TC < 200 mg/dL, and HDL-C > 40mg/dL were observed in 26.38%, 43.05%, 59.72%, and 80.55% patients, respectively. TG > 200 mg/dL, TC > 240 mg/dL, and LDL-C > 160 mg/dL were observed in 44.44%, 13.89%, and 9.72% patients, respectively, suggesting that many patients had more than one lipid abnormality (Tables 2 and 3).

Analysis of distribution of dyslipidemia showed that hypertriglyceridemia (TG > 200 mg/dL) was present in 52.9%, 26.50%, and 34.5% patients with NFG, IFG, and T2DM, respectively. Low HDL-C was observed in

NFG (35.3%), IFG (3.8%) and T2DM (24.1%) of patients. High LDL-C was observed in 11.5% and 13.8% patients with IFG and T2DM, respectively. High TC was observed in 23.1% and 13.8% of patients with IFG and T2DM, respectively. On intergroup comparison, differences were not found to be statistically significant (Table 4).

Comparison of mean values of lipid parameters in MetS patients with NFG, IFG, and T2DM showed no statistical differences.

CONCLUSION

From consequences of this examination, we infer that dyslipidemia is a typical element of MetS, and an extensive number of patients had more than one

singular lipid irregularity. Generally normal dyslipidemia was high TG and slightest was high LDL. Example of the dyslipidemia was comparable in each of the three bunches in light of blood glucose levels.

In this investigation, the connection between glucose levels also, lipid design in patients with MetS was inspected. Hypertriglyceridemia was the most normal lipid variation from the norm saw in these patients. Problematic and high TG levels were seen in 73.6% and 44.4% patients, though low levels of HDL-C were seen in just 19.4%. Comparable perception was made in an examination directed in north Indian populace with MetS wherein TG was the most common lipid abnormality (Bal, et. al., 2011). In our examine, investigation of the gauge qualities of the patients demonstrated that mean WC was expanded (98.61 ± 7.64 cm). WC is a pointer of instinctive fat tissue, which is a wellspring of free unsaturated fats changed over into TG by the liver (Despres and Lemieux, 2006).

With regards to diabetes, addressing the first barrier is most critical at the present time. This barrier pertains to the lack of consistent results across populations with regards to the genetic determinants of the disease. Failure to replicate study results may be due to a variety of factors, the most important of which may be that different gene-environment interactions operate different populations to increase risk of developing diabetes. Thus, considerably more epidemiologic research will be needed before we know the actual risk associated with particular genetic variants. This also likely means that we will not be able to apply a 'one size fits all' model when it comes to the genetic testing for any of the forms of diabetes.

REFERENCES

- Adiels M., Olofsson S.O., Taskinen M.R., Boren J. (2008). Overproduction of very low-density lipoproteins is the hallmark of the dyslipidemia in the metabolic syndrome. *Arterioscler Thromb Vasc Biol.*;28: pp. 1225–36.
- Alberti K.G., Eckel R.H., Grundy S.M., Zimmet P.Z., Cleeman J.I., Donato K.A., et al. (2009). Harmonizing the metabolic syndrome: a Joint Interim Statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. *Circulation.*;120(16): pp. 1640–5.
- Alberti K.G., Zimmet P.Z. (1998). Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med.* 15(7): pp. 539–53.
- Alberti KG, Zimmet P, Shaw J. (2006). Metabolic syndrome—a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med.*; 23(5): pp. 469–80.
- Assmann G., Schulte H. (1992). Relation of high-density lipoprotein cholesterol and triglycerides to incidence of atherosclerotic coronary artery disease (the PROCAM experience). Prospective Cardiovascular Munster study. *Am J Cardiol.*;70: pp. 733–7.
- Bal S.S., Khurana D., Sharma A., Lal V., Bhansali A., Prabhakar S. (2011). Association of metabolic syndrome with carotid atherosclerosis in the young North Indian population. *Diabetes Metab Syndr.*;5(3): pp. 153–7.
- Chahil T.J., Ginsberg H.N. (2006). Diabetic dyslipidemia. *Endocrinol Metab Clin North Am.*;35: pp. 491–510.
- Despres J.P., Lemieux I. (2006). Abdominal obesity and metabolic syndrome. *Nature.*; 444: pp. 881–7.
- Friedewald W.T., Levy R.I., Fredrickson D.S. (1972). Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical Chem.*;18: pp. 499–502.
- Goodarzi M.T., Mohammadian M., Borzouei S., Hassanzadeh T. (2014). Association between plasma cholesteryl ester transfer protein activity and lipid profiles in metabolic syndrome in an Iranian population. *Int Res J Biol Sci.*; 3(4): pp. 87–90.
- Haffner S.M., Stern M.P., Hazuda H.P., Mitchell B.D., Patterson J.K. (1990). Cardiovascular risk factors in confirmed pre-diabetic individuals: does the clock for coronary heart disease start ticking before the onset of clinical diabetes? *JAMA.*; 263: pp. 2893–8.
- Jeppesen J., Hein H.O., Suadicani P., Gyntelberg F. (1997). Relation of high TG-low HDL cholesterol and LDL cholesterol to the incidence of ischemic heart disease. An 8-year follow-up in the Copenhagen Male Study. *Arterioscler Thromb Vasc Biol.*;17: pp. 1114–20.

Kawamoto R., Tabara Y., Kohara K., Miki T., Kusunoki T., Takayama S., et. al. (2011). Relationships between lipid profiles and metabolic syndrome, insulin resistance and serum high molecular adiponectin in Japanese community-dwelling adults. *Lipids Health Dis.* 10: p.79.

Maheux P., Azhar S., Kern P.A., Chen Y.D., Reuven G.M. (1997). Relationship between insulin-mediated glucose disposal and regulation of plasma and adipose tissue lipoprotein lipase. *Diabetologia.* 40: pp. 850–8.

National Cholesterol Education Program (NCEP) (2002). Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) Final Report. *Circulation.* 106: pp. 31-43.

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