

Original research article**Serum lipid profile in diabetes mellitus****Dr. Prabhu N Biradar**

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Abstract

The condition known as diabetes mellitus is the metabolic disease that is most prevalent among people all over the world. It is well-established that diabetes mellitus is linked to both lipid problems and the development of cardiovascular diseases. The patients with diabetes mellitus who visit the outpatient department of medicine at HSK Hospital and Research Centre are going to participate in this study so that researchers can evaluate how their lipaemic levels have changed. Researchers looked at the levels of total cholesterol (TC), triglycerides (TG), LDL cholesterol (LDL-C), and HDL cholesterol (HDL-C) in the serum of people with diabetes. This is a case control study in which the cases consisted of 76 diabetic patients and the controls consisted of 50 individuals of the same age range and gender. All of the samples were taken from people who had abstained from food for at least a day and a half prior to having their blood drawn. Manual analysis using a spectrophotometer and liquid chemistry were employed in order to arrive at the values for the parameters. In both IDDM and NIDDM, the levels of triglycerides, total cholesterol, and LDL cholesterol were all greater in patients' bodies compared to those of healthy controls. The HDL-C levels of patients with NIDDM who were taking sulfonylureas or biguanides were dramatically decreased, whereas the HDL-C levels of IDDM patients who were taking insulin did not significantly change. In patients with NIDDM, there was a strong association between FBS and total cholesterol, total fat, LDL-C and HDL-C. In participants with IDDM, there was a substantial association between TC, TG, and LDL-C.

Keywords: Serum, Lipid, Profile, Diabetes, Mellitus**Introduction**

Diabetes mellitus (DM) is a category of metabolic diseases that is characterised by an increase in blood glucose level that results from abnormalities in insulin production, insulin action, or both ^[1]. Diabetes mellitus is a group of metabolic diseases that is characterised by an increase in blood glucose level. Diabetes is related with chronic hyperglycemia, which can cause long-term damage, dysfunction, and disturbance in functioning of numerous organs, including the eyes, kidneys, nerves, heart and blood vessels. Diabetes also increases the risk of cardiovascular disease ^[2]. Patients who have type 2 diabetes have a higher risk of developing cardiovascular disease, which is linked to atherogenic abnormalities and dyslipidemia. The major cause of illness and mortality around the world is coronary artery disease, specifically myocardial infarction ^[3]. In patients with type 2 diabetes, hyperglycemia and atherosclerosis are linked to one another. The glycosylation of all proteins is brought on by persistent hyperglycemia, but collagen cross-linking and the matrix proteins of the artery wall are particularly affected. This, in turn, leads to dysfunction in endothelial cells, which plays a role in the development of atherosclerosis. There is a 95% chance of having dyslipidemia if you have diabetes mellitus ^[5]. When hyperlipidemia is identified and treated in diabetes patients at an earlier stage, the risk of developing cardiovascular and cerebrovascular disorders is decreased. Modifications to one's lifestyle, such as diet and exercise, are extremely valuable in the treatment of diabetic dyslipidemia; however, pharmaceutical treatment is frequently required ^[6]. Lipoprotein metabolism ^[7]. The purpose of this research was to identify the lipid abnormalities that are linked to persistent hyperglycemia caused by either type of diabetes (NIDDM or IDDM).

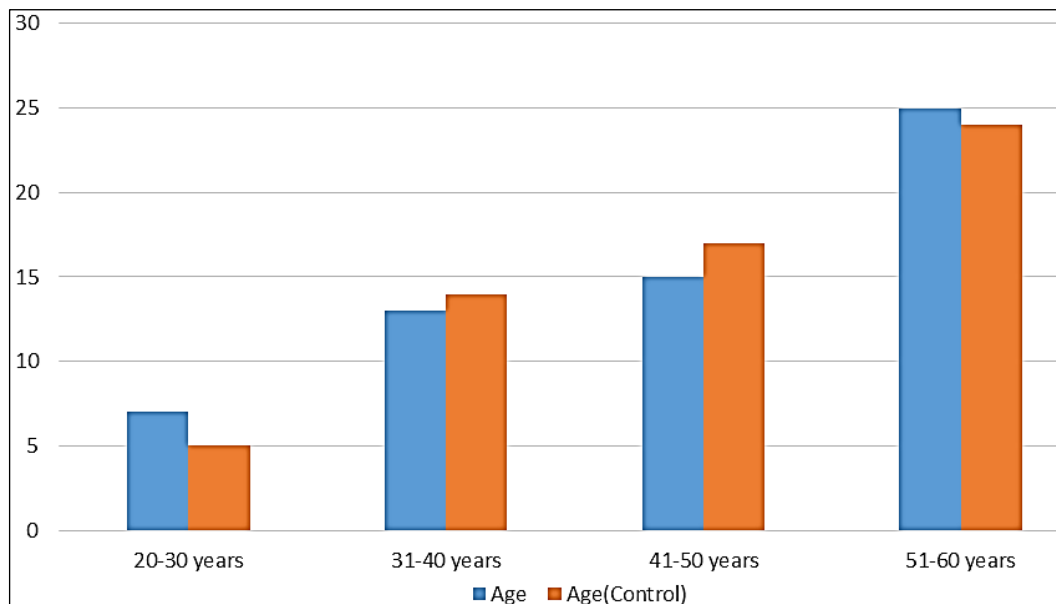
Aims and Objectives

A total number of 60 control who were healthy non-smokers non alcoholics and at the time of study all of them were keeping good health and 60 diabetics who were on treatment were studied. The diabetics were on either sulfonylurea/biguanides or insulin treatment for type 1 or type 2 diabetes. In our study, we excluded diabetic subjects who were smokers, alcoholics and who were hypertensives, familial hyperlipidemia patients and patients with complications.

Results

Table 1: Sex Distribution (Patients and control)

	Male	Female
Patients	38	22
Control	30	30



Graph 1: Age Distribution: (Patients and Control)

Table 2

Groups	FB Glucose Mg/dl	TC mg/dl	TG mg/dl	HDL-c Mg/dl	LDL-c Mg/dl
Control	91.39±09.28	172.60±31.2	121.5±18.7	56.28±15.9	93.3±36.3
IDDM Subjects	216.2±37.9	269.9±30.5	236.4±17.7	39.5±15.4	145.2±32.5
p-value	<.001	<.001	<.001	<.001	<.001
Statistical Significance	H. S	H.S	H.S	N. S	S. S

Discussion

When compared to their respective controls ^[13, 14], the fasting blood glucose levels in all of the diabetics showed a highly significant (p0.001) difference. Both our study in IDDM and our study in NIDDM are consistent with previous research carried out by John D. Bagdade 215 and James M. Falko ^[13]. The presence of diabetes appears to be linked to an increase in the body's production of cholesterol. It has been postulated that hyperphagia, which is a symptom of diabetes, causes an increase in the activity of HMG-CoA reductase in the intestine. This, in turn, causes an increase in the synthesis of cholesterol, which leads to an increase in its concentration in the plasma. The increased absorption of dietary cholesterol contributes to an increase in the body's total cholesterol level. According to the findings of our research, the levels of TG in patients with both IDDM and NIDDM who were treated with insulin, sulfonylurea, or biguanide were elevated, and the increase was statistically and clinically significant. It is possible that greater rates of generation of triglyceride rich VLDL by the liver ^[17] and slower clearance of TG by peripheral tissues, especially adipose tissue and muscle, are to blame for the hypertriglyceridemia. A lack of insulin causes a significant increase in total TG synthesis, which in turn causes a significant increase in VLDL packing.

Multiple investigations that used radioactive substrates to track the metabolism of plasma VLDL support the hypothesis that hypertriglyceridemia in patients with poorly managed IDDM ^[18] is caused by a single etiologic mechanism that involves simultaneous increased synthesis of plasma VLDL and decreased clearance of that protein.

In addition, the structural makeup of the VLDL itself could shift if there is an increase in protein components like apolipoprotein C-III. This component inhibits the lipase enzyme and the uptake of VLDL remains by the liver ^[19].

In NIDDM, greater production rates of triglyceride and VLDL particles have been found to be the most typically observed metabolic abnormalities ^[20] when TG levels are elevated above 200 mg/dl. There is evidence that a significant proportion of hypertriglyceridemic NIDDM patients also have a problem in the clearance of triglyceride with lipoproteins. According to the findings of our research, the mean levels of HDL-C in patients with IDDM who were receiving insulin therapy were not statistically significant

when compared to the controls. This finding is consistent with the research conducted by Kennedy and his colleagues, but it is not at odds with the findings published by Nikkila *et al.* [21].

Celvert *et al.* have documented low HDL-C in patients who were using sulfonylurea; however, our investigation reveals low HDL-C in diabetics who were treated with biguanide as well as sulfonylurea [22]. Diabetes may cause a reduction in the activity of lipoprotein lipase, which might lead to lower HDL levels. In patients with IDDM [18], there is an increase in the activity of the cholesterol ester transfer protein. Hepatic TG lipase (HTGL), an enzyme that lines the sinusoids of the liver and is responsible for breaking down TG that has been added to HDL, is elevated in diabetics and has an inverse correlation with HDL. The findings of the study conducted by Sosenko and colleagues, which indicated an increase in LDL-C levels in IDDM23, were supported by the findings of our research. In complete NIDDM patients taking sulfonylurea and patients taking biguanides, the mean LDL-c levels are significantly higher (p 0.001) when compared to the levels seen in the matched controls. It has been shown that individuals with IDDM have higher LDL production rates, however these rates return to normal after insulin infusion [20]. It's possible that this is because of an increase in the synthesis of VLDL or an impairment in the clearance of VLDL residual.

In addition to this, it has been postulated [21] that receptor-mediated clearance of LDL is impaired. There is a change in the lipid composition of the LDL that occurs in NIDDM. As a result of this change, the LDL contains an increased amount of triglycerides. Patients who have hypertriglyceridemia have LDL that has diminished receptor binding to cultured skin fibroblasts. This may be the mechanism that causes an increase in LDL-C in NIDDM [22].

Conclusion

Overall diabetes mellitus is closely associated with dyslipidemia in both IDDM and NIDDM.

References

1. American Diabetes Association. Diagnosis and classification of diabetes Mellitus. *Diabetes Care.* 2005;28(1):537-42.
2. Shera AS, Jawad F, A Maqsood A. Prevalence of diabetes in Pakistan. *Diabetes Res. Clin. Pract.* 2007;76(2):219-22.
3. Roberto T, Dodesini AR, Lepore G. Lipid and Renal disease. *J Am. Soc. Nephrol.* 2006;17:S145-7.
4. Devrajani BR, Shah SZ, Soomro AA, Devrajani T. Type 2 diabetes mellitus: A risk factor for *Helicobacter pylori* infection: A hospital based case-control study. *Int. J Diabetes Dev. Ctries.* 2010;30(1):22-6.
5. Chattanda SP, Mgonda YM. Diabetic dyslipidemia among diabetic patients attending specialized clinics in Dares Salaam. *Tanzania Med. J.* 2008;23(1):08-11.
6. Arjola Z, Klodiana S, Gentian V, *et al.* Lipid profile in diabetes mellitus type 2 patients in Albania and the correlation with BMI, HTN and hepatosteatosis. *J Family Med community health.* 2014;1(4):10-18.
7. Satyanarayana U, Chakrapani U. *Biochemistry.* 2013;(4):319-20.
8. *Clinical chemistry by Carl A. Burtis and Edward A. Ashwood, (4), 965-66.*
9. Watson D. *Clinica Chimica Acta.* 1960; 5:637-47.
10. Lowell B Foster, Ralph T Dunn. *Clinical Chemistry.* 1973;19(3):338-340.
11. Francogillo, *et al.* *Clinical Chemistry.* 1981;27:375-79.
12. Rafi MD. *Textbook of biochemistry for medical students.* 2014;(2):360.
13. James M Falko, *et al.*, *Am J Med. Oct.* 1987;83:641-47.
14. Bhalla Kapil, Shukla R, Gupta VP, *et al.* Glycosylated proteins and serum lipid profile in complicated and uncomplicated NIDDM patients. *Indian J Clin Biochem.* 1995;10(2):57-61.
15. John D Bagdade, *et al.* "Diabetic Lipemia" *NEMJ.* 1966;276(8):427-33.
16. Christopher D Saudek, Nancy L Young. Cholesterol metabolism in diabetes mellitus. *Diabetes.* 1981 Nov;30S(2):76-81.
17. Nikkila and Kekki. Plasma triglyceride transport kinetics in diabetes mellitus. *Metabolism.* 1973;22:1-22.
18. Nikkila, *et al.* *Diabetes.* 1977;26:11-24.
19. Brown and Baginsky. *Biochem Biophys Acta.* 1972;46:325-82.
20. Howard BV. Lipoprotein metabolism in diabetes mellitus. *J Lipid Res.* 1987;28:613-28.
21. Esko, Nikkila A, *et al.* Serum lipids and lipoproteins in insulin treated diabetes. 1978 Nov;27:1078-86.
22. Calvert GD, Graham JJ, Mannik T, Wise PH, Yeates RA. Effects of therapy on Plasma high density lipoprotein cholesterol concentration in diabetes mellitus. *Lance.* 1978;2:66-8.
23. Sosenko, *et al.* *N Engl J Med.* 1980;302:650-54.