

Comparison of Lipid Profile in Type-2 Obese Diabetics and Obese Non-diabetic Individuals. A hospital Based Study from Western Nepal

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ABSTRACT

Background

Type-2 diabetes mellitus is an independent risk factor for coronary artery disease and risk of coronary disease is three to four fold increased in patients with diabetes compared with non-diabetic population and 60-80% Of type-2 diabetics are obese.

Methods

This study was conducted in Nepalgunj Teaching Hospital, Kohalpur, Banke, Nepal, between 1st March, 2011 and 28th February, 2012. A total of 150 samples were taken to assess the lipid profile in type-2 diabetic patients associated with obesity and 25 obese controls for their lipid profile. Venous blood samples were taken from all the subjects in the morning after fasting overnight. Exclusion criteria included pregnancy, chronic infectious disease, heart failure; renal failure and drug allergy were confirmed from the subject's personal physician report and a detailed history. The data was analyzed using Excel 2003, R 2.8.0 Statistical Package for the Social Sciences (SPSS) for Windows Version 16.0 (SPSS Inc; Chicago, IL, USA) and the EPI Info 3.5.1 Windows Version.

Results

The mean \pm SD age of diabetic patients with obesity was 53.76 ± 6.23 while the mean \pm SD age of control was 49.61 ± 4.8 . Out of 150 patients 105 (70%) were males and 45 (30%) were females. Among control subjects 16 (64%) were males and 9 (36%) were females. Obese type-2 diabetic patients when compared to obese control subjects showed statistically significant increase in the levels of serum total cholesterol ($p < 0.001$), serum triglycerides ($p < 0.001$), serum LDL-cholesterol ($p < 0.001$) while serum HDL-cholesterol levels did not show statistically significant difference in the two group ($p > 0.05$).

Conclusion

This study showed obese diabetic individuals have dyslipidemia and more prone to develop cardiovascular diseases.

KEY WORDS

Lipid profile, Type-2 diabetes mellitus, Obesity

INTRODUCTION

Diabetes mellitus (DM) is a serious health problem being the third greatest cause of death all over the world, and if not treated, it is responsible for many complications affecting various organs in the body. The prevalence of diabetes for all age-groups worldwide was estimated to be 2.8% in 2000 and 4.4% in 2030. The total number of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030.¹ Type-2 diabetes mellitus is an

independent risk factor for coronary artery disease and risk of coronary disease is three to four fold increased in patients with diabetes compared with non-diabetic population.²⁻⁴ Interestingly, in developed countries lower socioeconomic groups are most affected while in developing countries reverse applies.⁵ Diabetes is significant and related public health problem in those aged 40 or more in urban Nepal.⁶ It is calculated 60-80% Of type-2 diabetics are obese.⁷

This study was conducted to examine the lipid profile in obese type-2 diabetic patients and obese control group to correlate coronary heart disease with dyslipidemia.

METHODS

This study was conducted in Nepalgunj Teaching Hospital, Kohalpur, Banke, Nepal, between 1st March, 2011 and 28th February, 2012. A total of 150 samples were taken to assess the lipid profile in type-2 diabetic patients associated with obesity and 25 obese controls for their lipid profile. Venous blood samples were taken from all the subjects in the morning after fasting overnight. Plasma levels of total cholesterol, triglycerides, High Density Lipoprotein-Cholesterol (HDL-C), Low Density Lipoprotein-Cholesterol (LDL-C) were analyzed. Total cholesterol and triglycerides concentration were determined with semi-automated enzymatic analyzer. Serum HDL-Cholesterol level was measured by using phospho-tungstate precipitation method. Serum LDL-C was calculated according to computational procedures of Friedewald et al.⁸ The internal quality control was included in each batch of tests performed. Inclusion criteria: The patients having confirmed diabetes mellitus with BMI more than 30 kg/m² were included in this study. Exclusion criteria included pregnancy, chronic infectious disease, heart failure; renal failure and drug allergy were confirmed from the subject's personal physician report and a detailed. Ethical approval for the study was taken from the institutional research ethical committee. The data was analyzed using Excel 2003, R 2.8.0 Statistical Package for the Social Sciences (SPSS) for Windows Version 16.0 (SPSS Inc; Chicago, IL, USA) and the EPI Info 3.5.1 Windows Version. The t-test was used to observe the relationship between different variables. $p < 0.05$ is considered as statistically significant.

RESULTS

The study was conducted on 150 type-2 diabetic patients associated with obesity and 25 obese age and sex matched controls. The mean \pm SD age of diabetic patients with obesity was 53.76 ± 6.23 while the mean \pm SD age of control was 49.61 ± 4.8 . Out of 150 patients 105 (70%) were males and 45 (30%) were females. Among control subjects 16 (64%) were males and 9 (36%) were females (table 1).

Table 1. Age and sex distribution of study subjects.

	Obese type-2 diabetic patients	Obese control
Age in years (range)	30-65	30-62
Mean \pm SD	53.76 ± 6.23	49.61 ± 4.8
Sex distribution		
Males	105 (70%)	16 (64%)
Females	45 (30%)	9 (36%)

Obese type-2 diabetic patients when compared to obese control subjects showed statistically significant increase in the levels of serum total cholesterol ($p < 0.001$), serum triglycerides ($p < 0.001$), serum LDL-cholesterol ($p < 0.001$) while serum HDL-cholesterol levels did not show statistically significant difference in the two group ($p > 0.05$) (table 2). A comparison of lipid profile of obese type-2 diabetic patients and obese control group were represented in fig 1.

Table 2. Lipid profile of patient and control subjects.

	Obese type-2 diabetic patients	Obese control	p value
Total Cholesterol			
Mean \pm SD	221.45 ± 54.38	150.81 ± 33.89	<0.001
Triglycerides			
Mean \pm SD	199.45 ± 54.38	101.3 ± 23.96	<0.001
HDL cholesterol			
Mean \pm SD	35.45 ± 11.09	40.32 ± 7.08	>0.05
LDL cholesterol			
Mean \pm SD	144.55 ± 29.28	82.53 ± 33.07	<0.001

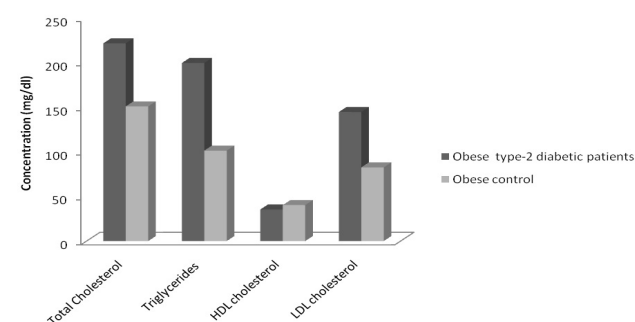


Figure 1. A comparison of lipid profile of obese type-2 diabetic patients and obese control group.

DISCUSSION

Dyslipidemia is very common in type 2 diabetes and it is characterized by hypertriglyceridemia and low levels of HDL-C.⁹ Hypertriglyceridemia predisposes the patients to life threatening complications like diabetic ketoacidosis, coronary artery disease and lipaemia retanalis.¹⁰ It is more common in diabetics as compared to non-diabetics due to four fold increase in VLDL triglyceride.¹¹ Hypercholesterolemia and high level of LDL-C mainly oxidized LDL-C in blood causes development of plaque formation and finally atherosclerosis as well as other cardiac diseases.

In our study, obese diabetics when compared to obese control subjects showed statistically significant increase in the levels of serum total cholesterol ($P < 0.001$), serum triglycerides ($P < 0.001$) and serum LDL –cholesterol ($P < 0.001$). Serum HDL – cholesterol levels did not differ significantly ($P > 0.05$) in the two groups but level were low in obese diabetic compare to obese controls. The studies of Santen et al (1972) and Peret et al (1974) observed

mean serum triglyceride levels higher in obese diabetics in comparison to obese control subject.^{12,13} Sharma (1970) and Jain (1980) observed increase in the levels of serum total lipids, total cholesterol, serum triglycerides and serum phospholipids in diabetic subjects as compared to normal controls.^{14,15} Several other studies have shown similar results as were obtained in our study (16, 17, 18).

Obesity and type 2 diabetes mellitus are associated with increased deposition of triglycerides in nonadipose tissue, such as the heart, liver, pancreas, and skeletal muscle.¹⁹ Obesity, i.e. fat accumulation in the subcutaneous abdominal and visceral depots, is most strongly associated with the risk of metabolic and cardiovascular complications.²⁰⁻²² Visceral obesity plays an important role in the development of diabetes by mobilizing free fatty acids and certain inflammatory cytokines promoting insulin resistance.²³ In obesity, the low plasma HDL-C levels have been attributed to increased fractional clearance of HDL secondary to depletion of its cholesterol.^{24,25} Many key enzymes involved in HDL metabolism are altered in obese people with insulin resistance. Some of these changes are further developed in type 2 diabetes where in addition to insulin resistance, relative or absolute insulin deficiency. An important metabolic trigger for reduced HDL-C levels in obesity and insulin resistance is the increased VLDL production, at least partly because of increased fatty acid flux to the liver.²⁶ Gambhir et al found that low HDL were

independent risk factor for premature coronary artery disease.²⁷ HDL facilitates reverse cholesterol transport to the liver from peripheral tissues, and thus probably prevents vascular atherosclerotic lesions from developing.²⁸ The cardioprotective properties of HDL include antioxidant activity, anti-inflammatory activity and scavenging toxic by-products of LDL oxidation such as lysophosphatidylcholine, antithrombotic and fibrinolytic activity through promotion of protein C, and inhibition of LDL retention through apo E-related effects.²⁹⁻³¹ Schmitt et al reported LDL uptake by fibroblasts may be impaired in type 2 diabetes and this leads to increase in LDL: HDL ratio in type 2 diabetics.³² Patients with type-2 diabetes have increased risk of cardiovascular disease associated with atherogenic dyslipidemia and coronary artery disease, especially myocardial infarction is the leading cause of morbidity and mortality worldwide.³³ This signifies individuals having diabetes associated obesity are more prone to develop cardiovascular disease than obese non-diabetic individuals.

CONCLUSION

This study showed obese diabetic individuals have dyslipidemia and more prone to develop cardiovascular diseases. The most probable reason, people are more aware of their physical well-being, changing their dietary habits and life style modification.

REFERENCES

1. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. *Diabetes Care* 2004; 27(5):1047-53.
2. Garcia MJ, McNamara PM, Gordon T, Kannel WB. Morbidity and mortality in diabetics in the Framingham population, sixteen year follow-up study. *Diabetes* 1974; 23(2):105-11.
3. Kannel WB, McGee DL. Diabetes and cardiovascular risk factors: the Framingham study. *Circulation* 1979; 59(1):8-13.
4. Ruiz J, Thillet J, Huby T, James RW, Erlich D, Flandre P, et al. Association of elevated lipoprotein(a) levels and coronary heart disease in NIDDM patients. Relationship with apolipoprotein(a) phenotypes. *Diabetologia* 1994; 37(6):585-91.
5. Mohan V, Shanthirani S, Deepa R, Premalatha G, Sastry NG, Saroja R. Intra urban differences in the prevalence of the metabolic syndrome in southern India, the Chennai urban population study. *Diabet Med* 2001; 18(4):280-7.
6. Khattri JB, Nepal MK. Study of depression among geriatric population in Nepal. *Nepal Med Coll J* 2006; 8(4):220-3.
7. Sheth JJ. Diabetes, microalbuminuria and hypertension. *Clin Exp Hypertens* 1999; 21(1-2):61-8.
8. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972; 18(6): 499-502.
9. Betteridge DJ. Diabetic dyslipidemia. *Diabetes Care* 2000; 2(Suppl 1): 31-6.
10. Oh RC, Lanier JB. Management of Hypertriglyceridemia. *Am Fam Physician* 2007; 75(9): 1365-71.
11. Arbeeny CM, Nordin C, Edelstein D, Stran N, Gibbons N, Eder HA. Hyperlipoproteinemia in spontaneously diabetic guinea pigs. *Metabolism* 1989; 38: 895-900.
12. Santen RJ, Willis PW, Fajans SS. Atherosclerosis in diabetes mellitus. Correlations with serum lipid levels, adiposity, and serum insulin level. *Arch Intern Med* 1972; 130(6): 833-43.
13. Perrett AD, Rowe AS, Shahmanesh M, Allison SP, Hartog M. Blood lipids in treated diabetics. *Diabetologia* 1974; 10(2): 115-8.
14. Sharma D, Bansal BC, Prakash C. Serum lipid studies in thin insulin dependent diabetics below the age of 30 years. *J Indian Med Assoc* 1970; 54(9):416-20.
15. Jain AP and Gupta DP. Study of blood lipid in Diabetics without any manifest vascular complications. *J.Dia. Asso. Ind* 1980; 199:29-34.
16. Cohen AM, Fidel J, Cohen B, Furst A, Eisenberg S. Diabetes, blood lipids, lipoproteins, and change of environment: restudy of the "new immigrant Yemenites" in Israel. *Metabolism* 1979; 28(7):716-28.
17. Bijlani PK, Shah K, Raheja BS, Krishnaswamy PR. High density lipoprotein cholesterol in diabetes. *J Assoc Physicians India* 1984; 32(4):309-11.
18. Zargar AH, Wandroo FA, Wadhwa MB, Laway BA, Masoodi SR, Shah NA. Serum lipid profile in non-insulin-dependent diabetes mellitus associated with obesity. *Int. j. diab. Dev. Countries* 1995; 15:9-13.
19. Tushuizen ME, Bunck MC, Pouwels PJ, Bontemps S, van Waesberghe JH, Schindhelm RK, et al. Pancreatic fat content and beta-cell function in men with and without type 2 diabetes. *Diabetes Care* 2007; 30: 2916-21.
20. Kissebah AH & Krakower GR. Regional adiposity and morbidity. *Physiol Rev* 1994; 74: 761-811.

21. Abate N & Garg A. Heterogeneity in adipose tissue metabolism: causes, implications and management of regional adiposity. *Prog Lipid Res* 1995; 34: 53-70.
22. Arner P. Not all fat is alike. *Lancet* 1998; 351: 1301-1302.
23. Kadowaki T & Yamauchi T. Adiponectin and adiponectin receptors. *Endocrine Rev* 2005; 26:439-451.
24. Borggreve SE, De Vries R, Dullaart RP. Alterations in high-density lipoprotein metabolism and reverse cholesterol transport in insulin resistance and type 2 diabetes mellitus: role of lipolytic enzymes, lecithin: cholesterol acyltransferase and lipid transfer proteins. *Eur J Clin Invest* 2003; 33: 1051-1069.
25. Vajo Z, Terry JG, Brinton EA. Increased intra-abdominal fat may lower HDL levels by increasing the fractional catabolic rate of Lp A-I in postmenopausal women. *Atherosclerosis* 2002; 160: 495-501.
26. Semenkovich CF. Insulin resistance and atherosclerosis. *J Clin Invest* 2006; 116: 1813-1822.
27. Gambhir JK, Kaur H, Gambhir DS, Prabhu KM. Lipoprotein (a) as an independent risk factor for coronary artery disease in patients below 40 years of age. *Indian Heart J* 2002; 52: 411-5.
28. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. *Am J Med* 1977; 62: 707-714.
29. Mooradian AD, Haas MJ, Wong NCW. The effect of select nutrients on serum high density lipoprotein cholesterol and apolipoprotein A-I levels. *Endocr Rev* 2006; 27: 2-16.
30. Mooradian AD, Haas MJ, Wong NCW. Transcriptional control of apolipoprotein A-I gene expression in diabetes mellitus. *Diabetes* 2004; 53: 513-520.
31. Hachem S, Mooradian AD. Familial dyslipidemias: an overview of pathophysiology and management. *Drugs* 2006; 66: 1949-1969.
32. Schmitt JK, Poole JR, Lewis SB. Hemoglobin A1 correlates with the ratio of low to high density lipoprotein in normal weight type 2 diabetics. *Metabolism* 1982; 31: 1084-9.
33. Toth PP. Effective management of the type 2 diabetes patient with cardiovascular and renal disease: secondary prevention strategies after a myocardial infarction. *Curr Diabetes Rev* 2012; 8(3): 219-28.