Journal of Rare Cardiovascular Diseases
ISSN: 2299-3711 (Print) | e-ISSN: 2300-5505 (Online)
www.jrcd.eu
RESEARCH ARTICLE

THE STUDY OF FASTING LIPID LEVELS AS A CARDIOVASCULAR RISK FACTOR IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

Dr Faisal Jamadar [1],Dr Suhail M Shaikh [2], Dr.Vivekanand Kamat*[3], Dr Vithal Rao [4]

[1]Assistant professor, Jawaharlal Nehru Medical College Belagavi

[2] Consultant Physician, Dr Raghoji multi-speciality Hospital, solapur -maharashtra

[3]Physician District Hospital Dharwad.

[4]Professor Al ameen Medical College and Hospital bijapur.

*Corresponding
Dr.Vivekanand Kamar
Physician District
Hospital Dharwad

Article History
Received:26/09/2025
Revised: 17/10/2025
Accepted: 05/11/2025
Published: 28/11/2025

Abstract: **Background**: Type 2 diabetes mellitus (T2DM) is strongly associated with atherosclerotic cardiovascular disease (CVD), and diabetic dyslipidemia is a major modifiable risk factor. Regional Indian data, especially from North Karnataka, remain limited.
Methods: This hospital-based cross-sectional case–control study included 100 T2DM patients and 100 age- and sex-matched healthy controls attending a tertiary-care teaching hospital in Vijayapur, Karnataka. All subjects underwent clinical evaluation, waist–hip ratio (WHR) measurement, fasting blood sugar estimation and fasting lipid profile (TC, TG, LDL-C, HDL-C, VLDL-C); CVD risk was assessed clinically and using ECG and 2D echocardiography.
Results: Most patients were between 51–60 years of age and sex distribution was similar in both groups. Dyslipidemia was present in 77% of T2DM cases versus 8% of controls, with significantly higher mean fasting TC, TG and LDL-C and significantly lower HDL-C in diabetics ($p < 0.05$ for all). Elevated CVD risk was observed in 76% of T2DM patients compared with 24% of controls ($p < 0.05$).
Conclusions: Fasting dyslipidemia is highly prevalent among T2DM patients in this North Karnataka cohort and shows a strong association with increased CVD risk. Routine fasting lipid profiling and early aggressive lipid management should be integral to diabetes care in similar populations.

Keywords: *Type 2 diabetes mellitus; fasting lipid profile; dyslipidemia; cardiovascular risk; India*

INTRODUCTION

Cardiovascular disease is the leading cause of death in patients with T2DM, accounting for two- to four-fold higher morbidity and mortality than in non-diabetics. This excess risk is mediated in part by diabetic dyslipidemia, characterized by elevated triglycerides, increased small dense LDL and reduced HDL cholesterol.
India faces a rapidly growing burden of T2DM, with earlier onset and high rates of CVD, and South Asian populations are particularly prone to atherogenic dyslipidemia and central obesity. However, there are few data from North Karnataka evaluating fasting lipid abnormalities and their relationship with cardiovascular risk in T2DM. This study aimed (1) to assess fasting lipid levels in T2DM patients and (2) to evaluate the significance of fasting dyslipidemia as a cardiovascular risk factor compared with healthy controls in a North Karnataka tertiary-care setting.

Materials and Methods

This hospital-based cross-sectional case–control study was conducted in the Department of General Medicine, Al Ameen Medical College Hospital, Vijayapur, Karnataka, from December 2017 to September 2019. One hundred patients with T2DM (duration ≥5 years, onset after 30 years) and 100 healthy, non-diabetic controls were enrolled.
Inclusion criteria (cases):
T2DM diagnosed as per WHO/ADA criteria.
Age ≥30 years and duration of T2DM ≥5 years.
Inclusion criteria (controls):
Apparently healthy adults without diabetes, hypertension, known CVD, renal or liver disease, and normal fasting glucose.
Exclusion criteria (both groups):
History or evidence of CAD, stroke, heart disease or hypertension.
Known primary lipid disorders, liver disease, renal dysfunction, hypothyroidism or major GI surgery.
Drugs affecting lipid metabolism or established lipid-lowering therapy.

Clinical and laboratory assessment
Detailed history and physical examination were performed for all participants. Waist and hip circumferences were measured with standard technique and WHR calculated to assess abdominal obesity. Cardiovascular evaluation included clinical examination, 12-lead ECG and 2D echocardiography to look for ischemic changes or structural heart disease.

After an overnight fast (≥8 hours), blood samples were collected for fasting blood sugar and fasting lipid profile (TC, TG, HDL-C, LDL-C, VLDL-C) using standard enzymatic methods in the hospital laboratory. LDL-C and VLDL-C were calculated as per standard formulae where applicable.

Definitions

Dyslipidemia was defined by any one of the following: TC >200 mg/dL, TG >150 mg/dL, LDL-C >130 mg/dL, HDL-C <40 mg/dL in men or <50 mg/dL in women, or VLDL-C >30 mg/dL. CVD risk was considered increased when there was clinical, ECG or echocardiographic evidence of ischemic heart disease or heart failure, or when the overall risk profile indicated moderate–high cardiovascular risk.

Sample size and statistics

The final sample included 100 T2DM cases and 100 controls, as per departmental protocol and feasibility. Continuous variables were expressed as mean ± SD and categorical variables as frequencies and percentages. Group comparisons used unpaired t-tests for continuous variables and chi-square tests for categorical variables; $p < 0.05$ was taken as statistically significant.

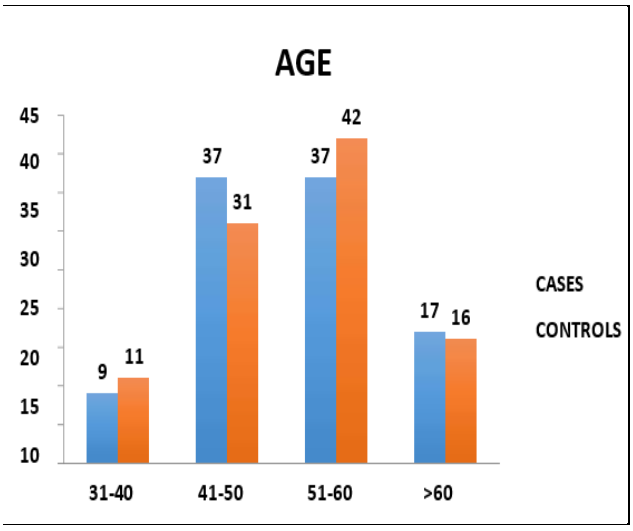
Ethics

Institutional Ethics Committee approval was obtained, and written informed consent was taken from all participants in accordance with the Declaration of Helsinki

Results

Baseline characteristics

Age ranged from 36–68 years among cases and 32–68 years among controls, with most subjects in the 51–60-year age group (37% of cases). Mean age was similar between cases and controls (51.9 ± 8.0 vs 51.3 ± 8.5 years, $p > 0.05$), and sex distribution was identical (53 males and 47 females in each group). Mean WHR was slightly higher in diabetics (0.91 ± 0.1) than controls (0.88 ± 0.1), but this difference was not statistically significant ($p > 0.05$).



CASES (BLUE) CONTROL(ORANGE)

Glycemic status

Mean fasting blood sugar was significantly higher in T2DM patients than in controls (169.3 ± 29.3 mg/dL vs 88.8 ± 13.8 mg/dL, $p < 0.001$), confirming poor glycemic control in the diabetic group.

FIGURE: COMPARISON OF FBS BETWEEN CASES AND CONTROLS

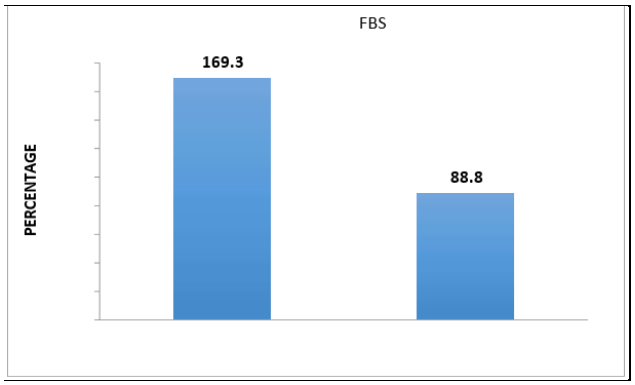
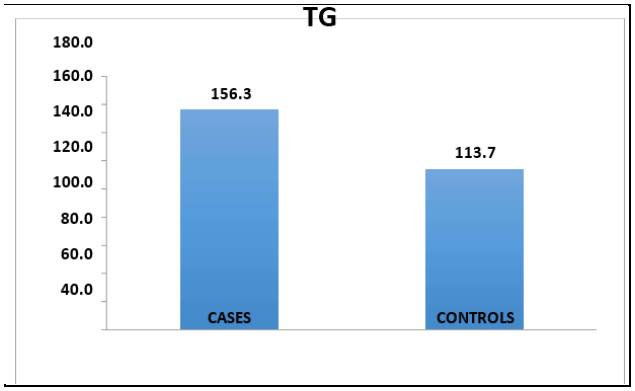


FIGURE: COMPARISON OF FASTING TG BETWEEN CASES AND CONTROLS



Fasting lipid profile

Diabetic patients had significantly more atherogenic fasting lipid profiles.

Mean TC: 212.2 ± 41.5 mg/dL (cases) vs 154.0 ± 16.6 mg/dL (controls), $p < 0.001$.

Mean TG: 156.3 ± 45.1 mg/dL vs 113.7 ± 30.1 mg/dL, $p < 0.001$.

Mean LDL-C: 132.3 ± 34.3 mg/dL vs 84.7 ± 15.1 mg/dL, $p < 0.001$.

Mean HDL-C: 43.6 ± 9.3 mg/dL vs 47.6 ± 6.5 mg/dL, $p < 0.001$.

Overall, dyslipidemia was present in 77% of cases compared with only 8% of controls ($p < 0.001$). Elevated TG, TC and LDL-C with reduced HDL-C formed the predominant pattern, consistent with classical diabetic dyslipidemia.

Cardiovascular risk

Elevated CVD risk was observed in 76% of T2DM patients against 24% of controls ($p < 0.001$). Many diabetic patients showed ECG or echocardiographic evidence of ischemic heart disease, indicating a substantial burden of clinically overt or silent CVD. The strong association between dyslipidemia and CVD risk was statistically significant when comparing cases and controls.

Discussion

This study shows a very high prevalence of fasting dyslipidemia (77%) among T2DM patients in a North Karnataka cohort, with a clear and significant association with increased CVD risk compared with non-diabetic controls. The pattern of higher TC, TG and LDL-C and lower HDL-C in diabetics matches the characteristic "diabetic dyslipidemia" described in large Indian and international studies.

Multiple studies have reported that 70–90% of T2DM patients have at least one lipid abnormality and that most fall into high or very high CVD risk categories. The finding that 76% of diabetics in this study had elevated CVD risk, versus 24% of controls, aligns with these observations and highlights the magnitude of cardiovascular burden in this region. Central obesity, reflected by higher WHR, though not statistically significant here, is biologically plausible as a contributor through insulin resistance and pro-inflammatory adipokines.

Pathophysiologically, insulin resistance promotes increased free fatty acid flux to the liver, leading to VLDL overproduction and hypertriglyceridemia, with subsequent formation of small dense LDL and HDL depletion via CETP-mediated exchange. Glycation and oxidation of lipoproteins, together with advanced glycation end-products and endothelial dysfunction, further accelerate atherosclerosis in T2DM. These mechanisms explain the close link between dyslipidemia and early-onset CVD found in this and other studies.

Current guidelines recommend that virtually all adults with T2DM, especially those over 40 years or with additional risk factors, should receive statin therapy, alongside lifestyle modification and optimal blood pressure and glucose control. The present results support routine fasting lipid profiling and early, aggressive lipid-lowering therapy in T2DM patients in North Karnataka to reduce CVD events.

Limitations

Limitations include the single-centre, hospital-based design, modest sample size and cross-sectional nature, which preclude causal inference or long-term event analysis. Potential confounders such as diet, physical activity and smoking were not fully quantified, and advanced lipid parameters (small dense LDL, HDL functionality) were not measured. Nevertheless, the strong and consistent differences between diabetics and controls provide robust clinical signals for everyday practice.

Conclusion

In this North Karnataka tertiary-care cohort, fasting dyslipidemia was highly prevalent in patients with T2DM and was significantly associated with increased cardiovascular risk compared with healthy controls. Incorporating routine fasting lipid profiling, early statin-based therapy and intensive lifestyle modification into diabetes care may substantially reduce the burden of premature cardiovascular disease in similar Indian populations.

Declarations

Ethics approval and consent to participate: Approved by the Institutional Ethics Committee of Al Ameen Medical College, Vijayapur, Karnataka, India; written informed consent obtained from all participants.

Funding: No external funding was received for this study.

Conflicts of interest: The authors declare no conflicts of interest

REFERENCES

1. American Diabetes Association. Screening for type 2 diabetes. Diabetes Care.2004 Jan;27 Suppl 1:S11-14.
2. Tushuizen ME, Diamant M, Heine RJ. Postprandial dysmetabolism and cardiovascular disease in type 2 diabetes. Postgrad Med J. 2005 Jan;81(951):1–6.
3. Tentolouris N, Stylianou A, Lourida E, Perrea D, Kyriaki D, Papavasiliou EC, et al. High postprandial triglyceridemia in patients with type 2 diabetes and microalbuminuria. J Lipid Res. 2007 Jan;48(1):218–25.
4. Postprandial hypertriglyceridemia and insulin resistance in normoglycemic first- degree relatives of patients with type 2 diabetes. - PubMed - NCBI [Internet]. [cited 2019 Oct 12]. Available from: https://www.ncbi.nlm.nih.gov/pubmed/10391812
5. [PDF] DYSLIPIDAEMIA IN TYPE 2 DIABETES MELLITUS PATIENTS -Semantic Scholar [Internet]. [cited 2019 Oct 12]. Available from: https://www.semanticscholar.org/paper/DYSLIPIDAEMIA-IN-TYPE-2- DIABETES-MELLITUS-PATIENTS-Bande-Baligar/9bcd40156b1bcc7d6cdb726886d91a969b842607
6. Haffner SM, Valdez RA, Hazuda HP, Mitchell BD, Morales PA, Stern MP. Prospective analysis of the insulin-resistance syndrome (syndrome X). Diabetes. 1992 Jun;41(6):715–22.
7. Association AD. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes—2019. Diabetes Care. 2019 Jan 1;42(Supplement 1):S13–28.
8. Pathogenesis of Type 2 Diabetes Mellitus - Endotext - NCBI Bookshelf [Internet]. [cited 2019 Oct 12]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK279115/
9. Shaw JE, Zimmet PZ, de Courten M, Dowse GK, Chitson P, Gareeboo H, et al.Impaired fasting glucose or impaired glucose tolerance. What best predicts future diabetes in Mauritius? Diabetes Care. 1999 Mar;22(3):399–402.
10. Zhang X, Gregg EW, Williamson DF, Barker LE, Thomas W, Bullard KM, et al.A1C level and future risk of diabetes: a systematic review. Diabetes Care. 2010 Jul;33(7):1665–73.
11. Dewan S, Wilding JPH. Obesity and type-2 diabetes in the elderly. Gerontology.2003 Jun;49(3):137–45.
12. Chawla A, Chawla R, Jaggi S. Microvasular and macrovascular complications in diabetes mellitus: Distinct or continuum? Indian J Endocrinol Metab. 2016;20(4):546–51.

13. Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham study. Diabetes Care. 1979 Apr;2(2):120–6.
14. Vlassara H, Uribarri J. Advanced Glycation End Products (AGE) and Diabetes: Cause, Effect, or Both? Curr Diab Rep. 2014 Jan;14(1):453.
15. Soro-Paavonen A, Watson AMD, Li J, Paavonen K, Koitka A, Calkin AC, et al.Receptor for Advanced Glycation End Products (RAGE) Deficiency Attenuates the Development of Atherosclerosis in Diabetes. Diabetes. 2008 Sep;57(9):2461– 9.
16. Haffner SM, Agostino RD, Saad MF, O'Leary DH, Savage PJ, Rewers M, et al.Carotid artery atherosclerosis in type-2 diabetic and nondiabetic subjects with and without symptomatic coronary artery disease (The Insulin Resistance Atherosclerosis Study). Am J Cardiol. 2000 Jun 15;85(12):1395–400.
17. Chronic leptin infusion increases arterial pressure. - PubMed - NCBI [Internet]. [cited 2019 Oct 12]. Available from: https://www.ncbi.nlm.nih.gov/pubmed/9453337
18. Pathophysiology of hypertension in diabetes mellitus--implications for management. - PubMed - NCBI [Internet]. [cited 2019 Oct 12]. Available from: https://www.ncbi.nlm.nih.gov/pubmed/1869594
19. UK Prospective Diabetes Study (UKPDS). XI: Biochemical risk factors in type 2 diabetic patients at diagnosis compared with age-matched normal subjects. Diabet Med. 1994 Jul;11(6):534–44.
20. Aronson D. Hyperglycemia and the pathobiology of diabetic complications. Adv Cardiol. 2008;45:1–16.
21. de Graaf J, Hak-Lemmers HL, Hectors MP, Demacker PN, Hendriks JC, Stalenhoef AF. Enhanced susceptibility to in vitro oxidation of the dense low density lipoprotein subfraction in healthy subjects. Arterioscler Thromb. 1991 Apr;11(2):298–306.
22. Wilson PW, Kannel WB, Anderson KM. Lipids, glucose intolerance and vascular disease: the Framingham Study. Monogr Atheroscler. 1985;13:1–11.
23. Harris MI. Hypercholesterolemia in diabetes and glucose intolerance in the U.S. population. Diabetes Care. 1991 May;14(5):366–74.
24. Stewart MW, Laker MF, Dyer RG, Game F, Mitcheson J, Winocour PH, et al.Lipoprotein compositional abnormalities and insulin resistance in type II diabetic patients with mild hyperlipidemia. Arterioscler Thromb. 1993 Jul;13(7):1046–52.
25. Wiggins D, Gibbons GF. The lipolysis/esterification cycle of hepatic triacylglycerol. Its role in the secretion of very-low-density lipoprotein and its response to hormones and sulphonylureas. Biochem J. 1992 Jun 1;284 (Pt 2):457–62.
26. Sosenko JM, Breslow JL, Miettinen OS, Gabbay KH. Hyperglycemia and plasma lipid levels: a prospective study of young insulin-dependent diabetic patients. N Engl J Med. 1980 Mar 20;302(12):650–4.
27. Garg A, Bonanome A, Grundy SM, Zhang ZJ, Unger RH. Comparison of a high- carbohydrate diet with a high-monounsaturated-fat diet in patients with non-insulin-dependent diabetes mellitus. N Engl J Med. 1988 Sep 29;319(13):829–34.
28. Taylor AJ, Villines TC, Stanek EJ, Devine PJ, Griffen L, Miller M, et al.Extended-Release Niacin or Ezetimibe and Carotid Intima–Media Thickness. New England Journal of Medicine. 2009 Nov 26;361(22):2113–22.
29. Kastelein JJP, Bots ML. Statin Therapy with Ezetimibe or Niacin in High-Risk Patients. New England Journal of Medicine. 2009 Nov 26;361(22):2180–3.
30. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al.Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. Circulation. 2005 Oct 25;112(17):2735–5