



Correlation between Ankle-Brachial Index and LDL/HDL Cholesterol Ratio in Patients with Type 2 Diabetes Mellitus

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Abstract

Objective: To determine the correlation between the LDL/HDL cholesterol ratio and ankle-brachial index (ABI) in patients with type 2 diabetes mellitus (T2DM). **Methods:** This cross-sectional study included 50 patients with T2DM attending the Diabetes outpatient department at Government General Hospital, Mahabubnagar. Patients aged 40-60 years with a duration of diabetes of 1-15 years were included. Patients with hypertension, coronary artery disease, chronic kidney disease, clinically detectable peripheral arterial disease, foot gangrene, foot ulcers, pulselessness, or a history of smoking were excluded. Blood pressure was measured in the upper and lower limbs using a digital oscillometric sphygmomanometer, and ABI was calculated. Serum lipid profile was measured, and the LDL/HDL ratio was calculated. Pearson correlation was used for analysis, with $p < 0.01$ considered statistically significant. **Results:** Mean total cholesterol was 231.9 ± 24.18 mg/dL, triglycerides 171.1 ± 30.08 mg/dL, LDL 147.68 ± 25.33 mg/dL, HDL 50.00 ± 8.27 mg/dL, and LDL/HDL ratio 3.11 ± 1.06 . ABI showed moderate negative correlations with total cholesterol ($r = -0.522$), triglycerides ($r = -0.589$), LDL ($r = -0.533$), and LDL/HDL ratio ($r = -0.571$). ABI showed a moderate positive correlation with HDL ($r = 0.535$). All correlations were statistically significant ($p < 0.01$). **Conclusion:** Higher atherogenic lipid values, particularly a higher LDL/HDL ratio, were associated with lower ABI among patients with T2DM. Combined assessment of ABI and LDL/HDL ratio may support early identification of peripheral vascular risk in diabetic patients. Larger prospective studies are required to confirm these findings.

Keywords: Ankle-Brachial Index; Peripheral Arterial Disease; Type 2 Diabetes Mellitus; LDL/HDL Ratio; Dyslipidemia



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INTRODUCTION

Peripheral arterial disease (PAD) is a clinical condition characterized by arterial occlusion in the limbs. Type 2 diabetes mellitus (T2DM) is a major risk factor for PAD, and people with diabetes have more than a two-fold higher prevalence of PAD compared with the general population (1). Despite its epidemiological and clinical importance, PAD remains largely underdiagnosed and undertreated, possibly because many patients are asymptomatic (2).

The ankle-brachial index (ABI) is a simple, non-invasive, and cost-effective bedside screening tool for PAD (3). ABI is calculated as the ratio of systolic blood pressure at the ankle to systolic blood pressure in the brachial artery (4). ABI values of 1.00-1.40 are generally considered normal, 0.91-0.99 borderline, < 0.90 diagnostic of PAD, < 0.50 suggestive of critical limb ischemia, and > 1.40 suggestive of non-compressible calcified arteries.

Dyslipidemia is recognized as an independent risk factor in the pathogenesis of PAD. In individuals with diabetes mellitus, characteristic alterations in lipid metabolism give rise to diabetic dyslipidemia, which further increases the risk of developing PAD.

The LDL/HDL cholesterol ratio may provide a more comprehensive measure of atherogenic burden than evaluation of LDL and HDL levels individually. An elevated LDL/HDL ratio reflects a predominance of cholesterol in a form conducive to plaque formation, whereas a reduced ratio indicates comparatively more effective reverse cholesterol transport from peripheral arteries. Therefore, correlating this lipoprotein ratio with ABI may provide a useful representation of lipid-mediated atherosclerotic risk in diabetic patients.

The objective of this study was to determine the correlation between the LDL/HDL cholesterol ratio and ABI in patients with T2DM.

Table 1: Interpretation of Ankle-Brachial Index values

ABI value	Interpretation
1.00-1.40	Normal
0.91-0.99	Borderline
<0.90	Diagnostic of PAD
<0.50	Critical limb ischemia
>1.40	Non-compressible calcified arteries

MATERIALS AND METHODS

This cross-sectional study was conducted among patients with T2DM attending the Diabetes outpatient department at Government General Hospital, Mahabubnagar, Telangana, India, during the study period. A total of 50 patients meeting the eligibility criteria were recruited. Convenient sampling was involved.

Patients of both genders aged 40-60 years with a duration of diabetes between 1 and 15 years were included. Patients with hypertension, coronary artery disease, chronic kidney disease, clinically detectable PAD, pulselessness, gangrene, foot ulcers, or a history of smoking were excluded.

After explaining the procedure and obtaining written informed consent, participants were asked to rest in the supine position. Blood pressure in both upper limbs and lower limbs was measured using a digital oscillometric sphygmomanometer (OMRON HEM-7156; Omron Healthcare) (5). The Intelli Wrap cuff allowed use across a wide range of arm and calf sizes; therefore, the same cuff was used for all participants. When there was a difference between the blood pressure values of both upper limbs, the higher brachial systolic blood pressure was used for ABI calculation. ABI was measured

separately for both lower limbs. If the ABI values differed between limbs, the lower ABI value was considered for analysis.

Blood samples were collected under aseptic precautions to estimate serum lipid profile, including total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol. The LDL/HDL cholesterol ratio was calculated for each participant.

Ethical approval was obtained from the Institutional Ethics Committee of Government Medical College, Mahabubnagar. All procedures involving human participants were conducted in accordance with institutional ethical standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants before enrolment.

Data were entered and analyzed using Microsoft Excel and SPSS software [insert version]. Quantitative variables were summarized as mean with standard deviation (SD). Pearson correlation test was used to assess the relationship between ABI and lipid profile variables. A p value of <0.01 was considered statistically significant.

RESULTS

A total of 50 patients with T2DM were included in the study. The mean values of serum total cholesterol, triglycerides, LDL cholesterol, HDL cholesterol, and LDL/HDL ratio are presented in Table 2.

The mean total cholesterol was 231.9 ± 24.18 mg/dL, triglycerides 171.1 ± 30.08 mg/dL, LDL cholesterol 147.68 ± 25.33 mg/dL, HDL cholesterol 50.00 ± 8.27 mg/dL, and LDL/HDL ratio 3.11 ± 1.06 .

The correlation of ABI with lipid profile variables is shown in Table 3. ABI had moderate negative correlations with serum total cholesterol ($r=-0.522$, $p<0.01$), triglycerides ($r=-0.589$, $p<0.01$), LDL cholesterol ($r=-0.533$, $p<0.01$), and LDL/HDL ratio ($r=-0.571$, $p<0.01$). ABI showed a moderate positive correlation with HDL cholesterol ($r=0.535$, $p<0.01$).

Table 2

Table 2: Mean lipid profile and LDL/HDL ratio

Lipid profile variable	Mean $\pm$ SD
Total cholesterol	231.9 ± 24.18
Triglycerides	171.1 ± 30.08
LDL cholesterol	147.68 ± 25.33
HDL cholesterol	50.00 ± 8.27
LDL/HDL ratio	3.11 ± 1.06

SD: Standard deviation; LDL: Low-density lipoprotein; HDL: High-density lipoprotein.

Table 3

Table 3: Correlation of ABI with lipid profile variables

Lipid profile variable	r value	p value
Total cholesterol	-0.522	<0.01*
Triglycerides	-0.589	<0.01*
LDL cholesterol	-0.533	<0.01*
HDL cholesterol	0.535	<0.01*
LDL/HDL ratio	-0.571	<0.01*

*Statistically significant at $p < 0.01$. ABI: Ankle-brachial index; LDL: Low-density lipoprotein; HDL: High-density lipoprotein.

DISCUSSION

In this study, the association between the LDL/HDL cholesterol ratio, a marker of atherogenic lipid burden, and ABI, a well-established non-invasive indicator of PAD, was evaluated among patients with T2DM. The findings showed that higher total cholesterol, triglycerides, LDL cholesterol, and LDL/HDL ratio were associated with lower ABI values, whereas higher HDL cholesterol was associated with higher ABI values.

These findings align with the growing recognition that dyslipidemia, particularly diabetic dyslipidemia, is an important contributor to PAD (6,7). Diabetes mellitus alters lipid metabolism and promotes atherogenesis through endothelial dysfunction, oxidative stress, and systemic inflammation, creating a pathological environment conducive to peripheral vascular compromise (8,9). The findings of this study are consistent with earlier studies that demonstrated associations between atherogenic lipid indices and markers of subclinical atherosclerosis, such as carotid intima-media thickness and arterial stiffness (10,11). A higher LDL/HDL ratio reflects a predominance of atherogenic cholesterol particles and may indicate greater atherosclerotic burden, which can manifest clinically as a lower ABI.

ABI is a simple and widely used measure for detecting PAD, with values less than 0.90 indicating significant arterial obstruction (12,13). A low ABI not only indicates PAD but also correlates with systemic atherosclerosis, including coronary artery disease.

A high LDL/HDL ratio implies dominance of LDL-driven lipid deposition and impaired HDL-mediated reverse cholesterol transport. This can promote macrophage foam cell formation, plaque proliferation, and vascular narrowing. HDL facilitates cholesterol efflux and helps remove harmful oxidation products such as oxysterols, which accumulate in atherosclerotic lesions, especially in diabetic states (14). In diabetes, these processes may be amplified by hyperglycemia-induced endothelial dysfunction, insulin resistance, and pro-inflammatory cytokine expression, leading to accelerated atherogenesis (15).

The clinical significance of these findings lies in the potential use of LDL/HDL ratio together with ABI as

combined markers for cardiovascular and peripheral vascular risk assessment. Although lipid profile evaluation is routinely performed in clinical practice, ABI remains underutilized despite its simplicity. Incorporating both parameters may help identify high-risk individuals earlier and guide preventive strategies such as aggressive lipid-lowering therapy and lifestyle modification (16-18).

This study has limitations. The sample size was small, and the cross-sectional design prevents establishing a causal relationship between LDL/HDL ratio and ABI. In addition, conventional lipid parameters were used. Newer markers such as apolipoprotein B, lipoprotein(a), and oxidized LDL may provide additional insight in future studies.

CONCLUSION

This study found a significant moderate negative correlation between ABI and atherogenic lipid parameters, including total cholesterol, triglycerides, LDL cholesterol, and LDL/HDL ratio, in patients with T2DM. HDL cholesterol showed a significant moderate positive correlation with ABI. These findings suggest that LDL/HDL ratio, when assessed along with ABI, may help identify diabetic patients at increased risk of peripheral vascular disease. Larger prospective studies are needed to validate these findings and clarify their predictive value.

REFERENCES

- Jameson JL, Fauci AS, Kasper DL, Hauser SL, Longo DL, Loscalzo J, editors. *Harrison's Principles of Internal Medicine*. 19th ed. New York: McGraw-Hill Education; 2015.
- Soyoye DO, Abiodun OO, Ikem RT, Kolawole BA, Akintomide AO. Diabetes and peripheral artery disease: A review. *World J Diabetes*. 2021;12(6):827-838. doi:10.4239/wjd.v12.i6.827.
- Winsor T. Influence of arterial disease on the systolic blood pressure gradients of the extremity. *Am J Med Sci*. 1950;220:117-126.
- Aboyans V, Criqui MH, Abraham P, et al. Measurement and interpretation of the ankle-brachial index: A scientific statement from the American Heart Association. *Circulation*. 2012;126(24):2890-2909.

5. Richart T, Kuznetsova T, Wizner B, Struijker-Boudier HA, Staessen JA. Validation of automated oscillometric versus manual measurement of the ankle-brachial index. *Hypertens Res.* 2009;32(10):884-888. doi:10.1038/hr.2009.125.
6. Wu L, Parhofer KG. Diabetic dyslipidemia. *Metabolism.* 2014;63(12):1469-1479. doi:10.1016/j.metabol.2014.08.010.
7. Bhowmik B, Siddiquee T, Mujumder A, et al. Serum lipid profile and its association with diabetes and prediabetes in a rural Bangladeshi population. *Int J Environ Res Public Health.* 2018;15(9):1944. doi:10.3390/ijerph15091944.
8. Virani SS, Alonso A, Benjamin EJ, et al. Dyslipidemia and cardiovascular disease: Current insights and future perspectives. *Lancet.* 2020;394(10212):600-612.
9. Goldberg IJ. Diabetic dyslipidemia: Causes and consequences. *J Clin Endocrinol Metab.* 2001;86(3):965-971.
10. Lee YJ, Tsai JC, et al. LDL/HDL ratio and carotid intima-media thickness in type 2 diabetes. *Atherosclerosis.* 2005;181(2):381-388.
11. O'Leary DH, Polak JF, Kronmal RA, Manolio TA, Burke GL, Wolfson SK Jr. Carotid artery intima-media thickness as a risk factor for myocardial infarction and stroke. *N Engl J Med.* 1999;340(1):14-22.
12. Newman AB, Scovick DS, Manolio TA, et al. Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study. *Arterioscler Thromb Vasc Biol.* 1993;13(3):701-707.
13. Hiatt WR, et al. Diagnostic methods for peripheral arterial disease: Clinical and non-invasive approaches. *Circulation.* 2015;131(14):e463-e465.
14. Barter PJ, Nicholls S, Rye KA, Anantharamaiah GM, Navab M, Fogelman AM. The role of HDL in atherosclerosis: Reverse cholesterol transport and beyond. *Atherosclerosis.* 2003;168(2):195-211.
15. Beckman JA, Creager MA, Libby P. Diabetes and atherosclerosis: Epidemiology, pathophysiology, and management. *JAMA.* 2002;287(19):2570-2581.
16. Norgren L, Hiatt WR, Dormandy JA, et al. Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II). *J Vasc Surg.* 2007;45 Suppl S:S5-S67.
17. Criqui MH, Langer RD, Fronek A, Feigelson HS, Klauber MR, McCann TJ, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med.* 1992;326:381-386.
18. Poussa H, Strandberg TE, Tikkanen I, Kauhanen P, Lepantalo M. Diagnosis and treatment of dyslipidemia are neglected in patients with peripheral artery disease. *Scand Cardiovasc J.* 2007;41(3):138-141. doi:10.1080/14017430601187751.