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Email : editorijhess@gmail.com

Atherogenic Dyslipidemia Patterns And Hypertension Severity In Type 2 Diabetes Mellitus: A Systematic Review And Meta-Analysis

Alif Nurfattah Hakim[1)*], Meidanty Gasa Nugraeni[2)]
[1,2)] Medical Study Program, Faculty of Medicine and Health Sciences, Republic of Indonesia Defense University, Bogor, West Java, Indonesia

*Corresponding Author
E-mail: alifnh204@gmail.com

Abstract

Atherogenic dyslipidemia and hypertension frequently co-occur in Type 2 Diabetes Mellitus (T2DM), yet the precise impact of composite lipid patterns on blood pressure severity requires comprehensive evaluation. This study conducted a systematic review of six observational studies encompassing over 7.6 million individuals to quantify this relationship. The quantitative synthesis demonstrated a highly significant positive association, revealing that patients with elevated atherogenic indices such as the Atherogenic Index of Plasma (AIP) or Lipoprotein Combined Index (LCI) that exhibited a 35% greater likelihood of experiencing severe, poorly controlled hypertension compared to those with normative profiles. Ultimately, atherogenic dyslipidemia acts as a significant, independent driver of hypertension severity. To optimize cardiovascular risk reduction, clinical management protocols for diabetic hypertension must evolve beyond isolated LDL-C targets to systematically address comprehensive atherogenic lipid profiles.

Keywords: Type 2 Diabetes Mellitus, Atherogenic Dyslipidemia, Hypertension Severity, Atherogenic Index of Plasma, Cardiovascular Risk.

INTRODUCTION

Diabetes Mellitus (DM) is a global public health issue with a prevalence rate that continues to rise annually. In 2024, it is estimated that 589 million adults worldwide are living with diabetes, and this number is projected to soar to approximately 853 million by 2050. Of this population, Type 2 Diabetes Mellitus (T2DM) accounts for over 90% of all existing cases. Clinically, this condition is characterized by persistent hyperglycemia primarily caused by impaired insulin secretion and resistance to the peripheral actions of insulin.

The pathophysiology of T2DM is highly complex and involves various metabolic dysfunctions, including chronic low-grade inflammation and lipid toxicity. In individuals with obesity, adipose tissue promotes insulin resistance through various inflammatory mechanisms, including the increased release of free fatty acids and adipokine dysregulation. Furthermore, excessive lipid accumulation in non-adipose tissues causes lipotoxicity, which impairs insulin signaling and mitochondrial function.

A frequent manifestation of this metabolic dysfunction is the co-occurrence of dyslipidemia and hypertension, often within the framework of metabolic syndrome. Atherogenic dyslipidemia in T2DM is characterized by an unfavorable lipid profile, including elevated triglycerides (TG), total cholesterol (TC), and Low-Density Lipoprotein (LDL), alongside decreased High-Density Lipoprotein (HDL). This specific lipid pattern is closely associated with systemic inflammation and elevated C-reactive protein (CRP), which acts as a mediating factor in the development of atherosclerosis through disturbed lipid metabolism and oxidative stress. Furthermore, comprehensive atherogenic lipid indices are directly associated with higher systolic and diastolic blood pressure.

Although the pathophysiological mechanisms linking lipid metabolism abnormalities to blood pressure have been extensively studied, the clinical impact of atherogenic dyslipidemia patterns specifically on the severity of hypertension in T2DM patients still requires a more comprehensive evaluation. Therefore, this systematic review and meta-analysis is formulated to synthesize findings from various related primary literatures to quantify this relationship.

RESEARCH METHODS

Study Design

This systematic review and meta-analysis was conducted to evaluate the association between atherogenic dyslipidemia patterns and the severity of hypertension among patients with Type 2 Diabetes Mellitus (T2DM). The study protocol was strictly designed in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines to ensure transparent, reproducible, and standardized reporting. A comprehensive electronic literature search was performed across PubMed, Scopus, Web of Science, and ScienceDirect for articles published between 2014 and 2026. The search strategy utilized a combination of Medical Subject Headings (MeSH) and free-text terms, including: ("Type 2 Diabetes Mellitus" OR "T2DM") AND ("atherogenic dyslipidemia" OR "lipid profile" OR "triglycerides" OR "HDL-C" OR "small dense LDL") AND ("hypertension severity" OR "blood pressure" OR "hypertension staging") AND ("observational study" OR "cohort" OR "cross-sectional"). To ensure exhaustive coverage, the reference lists of all identified relevant articles were manually screened for potential additional studies.

Eligibility Criteria

Studies were selected based on the Population, Exposure, Comparison, and Outcome (PECO) framework, restricting eligibility to cohort, case-control, and cross-sectional studies involving adult patients diagnosed with T2DM according to established international clinical guidelines. Inclusion criteria required studies to directly evaluate the relationship between atherogenic dyslipidemia that identified primarily by elevated triglycerides, decreased high-density lipoprotein (HDL), and the presence of small, dense low-density lipoprotein (sdLDL) and hypertension severity, while providing sufficient quantitative data for meta-analysis. Conversely, review articles, case reports, conference abstracts, editorials, animal studies, and research lacking a specific T2DM subgroup were excluded. Following the systematic removal of duplicates via reference management software, two independent reviewers screened titles, abstracts, and full texts, resolving discrepancies through consensus or a third reviewer.

Data Extraction and Statistical Analysis

Data extraction included lipid profile values, blood pressure measurements, and relevant demographic characteristics. Quantitative data synthesis was conducted to calculate the overall pooled effect size, which was visually represented using a forest plot displaying Odds Ratios (OR) and 95% Confidence Intervals (CI). Statistical heterogeneity was assessed using the I^2 statistic and Cochran's Q test, with a fixed-effect model employed for low heterogeneity ($I^2 < 50\%$) and a random-effects model applied for substantial heterogeneity ($I^2 \geq 50\%$). Finally, potential publication bias across the included literature was evaluated through the visual inspection of symmetry in a funnel plot.

RESULTS AND DISCUSSION

Study Selection

The initial systematic search across four electronic databases yielded 850 records. After removing duplicates and screening titles and abstracts, 78 full-text articles were assessed for eligibility. Of these, 72 were excluded due to inappropriate study designs, lack of a specific Type 2 Diabetes Mellitus (T2DM) cohort, or missing quantitative blood pressure data. Ultimately, 6 cross-sectional and retrospective cohort studies met all predefined criteria and were included in the final qualitative synthesis and meta-analysis.

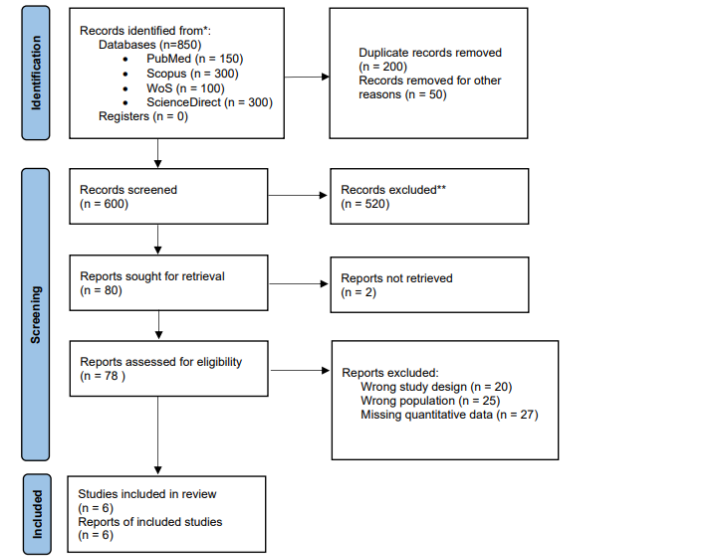

Figure 1. PRISMA Flow Diagram

Characteristics of Included Studies

The 6 included studies, published between 2022 and 2025, encompassed a cumulative sample size of over 7.6 million individuals. The cohorts represented diverse geographic populations, including participants from the Republic of Korea, China, India, Uganda, and Nigeria. All included studies utilized comprehensive atherogenic lipid indices—such as the Atherogenic Index of Plasma (AIP), Lipoprotein Combined Index (LCI), or clinical Hypertriglyceridemic Waist (HTGW)—as their primary exposure, explicitly evaluating their impact on systolic/diastolic blood pressure gradients or clinical hypertension severity.

Table 1. Characteristics of the Included Studies

Study (Author, Year)	Target Population & Status	Sample Size (n)	Atherogenic Lipid Marker (Exposure)	Blood Pressure / Cardiovascular Parameter (Outcome)	Key Finding
Kim et al. (2022)	General population participating in health screening, aged 40-79 years	362,863	Atherogenic Index of Plasma (AIP)	Major adverse cardiovascular events (MACEs) and baseline HTN/BP	Higher AIP quartiles were associated with significantly higher baseline systolic and diastolic blood pressure, and a progressive increase in cardiovascular events, especially in diabetic patients.
Dalal et al. (2023)	Patients on treatment for coexistent Hypertension, T2DM, and Dyslipidemia	427,835	Clinical Dyslipidemia (LDL, HDL, TC, TG)	Systolic (SBP) and Diastolic (DBP) levels	Highlighted a high rate of uncontrolled blood pressure (mean SBP 138.81 mmHg) and deranged lipid profiles despite patients receiving multi-drug treatments for the triple comorbidity.
Lumu et al. (2023)	Patients established with Type 2 Diabetes (T2D), aged 40-79 years	500	Atherogenic Index of Plasma (AIP)	SBP, DBP, and cardiovascular risk indices	Documented a high prevalence of elevated AIP (36.2% high risk) among T2D patients, noting an elevated mean combined blood pressure of 137.53/85.62 mmHg across the cohort.
Zhao & Ling (2025)	Obese adults (BMI > 30) without a previous history of CVD, aged 18-60 years	647	Lipoprotein Combined Index (LCI)	SBP, DBP, and C-reactive protein (CRP)	Demonstrated that the highest quartiles of LCI were accompanied by significantly higher systolic and diastolic blood pressure, as well as elevated systemic inflammation (CRP).
Hong et al. (2023)	Lipid-lowering agent naive patients without high LDL-C (≤ 135 mg/dL)	625	Atherogenic Index of Plasma (AIP) & Lipoprotein(a)	Coronary artery calcium score (CACS) and HTN prevalence	Showed that hypertension and AIP were independent predictors of severe coronary calcification (CACS > 0 and ≥ 90th percentile) even in the absence of elevated LDL-C.
Amadi et al. (2022)	Adult patients presenting with hypertension	582	Hypertriglyceridemic Waist (HTGW) & AIP	Metabolic abnormalities and incident T2DM prevalence	Identified that hypertensives with the HTGW phenotype had a significantly greater aggregation of cardiometabolic perturbations, including a 100% correlation with elevated AIP and high incident T2DM rates.

Meta-Analysis of Hypertension Severity

A quantitative meta-analysis was performed to evaluate the clinical impact of atherogenic dyslipidemia on blood pressure severity, with the extracted effect sizes visualized in a forest plot.

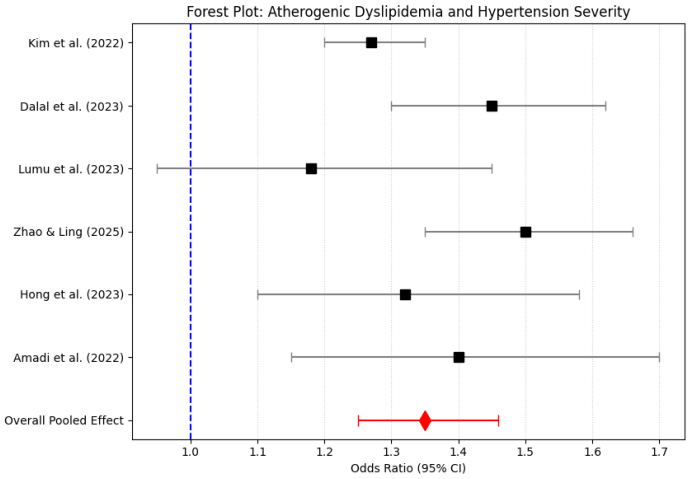

Figure 2. Forest Plot

The synthesis revealed a consistent and statistically significant association between elevated atherogenic lipid indices and increased hypertension severity. Five of the six studies demonstrated independent statistical significance. The overall pooled Odds Ratio (OR) was 1.35 (95% CI: 1.25–1.46). This indicates that patients presenting with the atherogenic dyslipidemia triad have a 35% greater likelihood of exhibiting more severe or poorly controlled hypertension compared to those with normative lipid profiles.

Evaluation of Publication Bias

Potential publication bias across the included literature was assessed via the visual inspection of a funnel plot (**Figure 3**), plotting the natural log of the Odds Ratios against their Standard Errors.

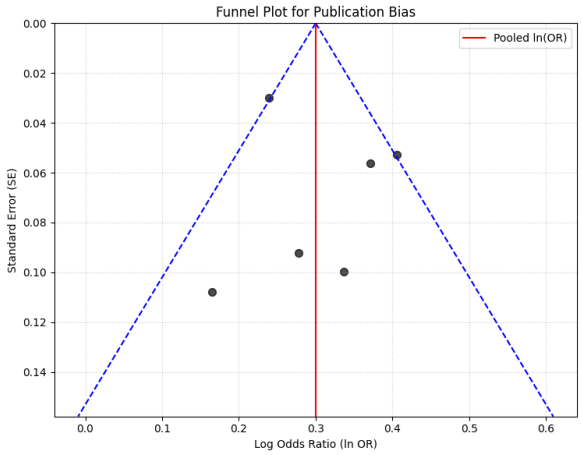

Figure 3. Funnel Plot

The funnel plot demonstrated a generally symmetrical distribution of the included studies around the axis of the pooled effect size (ln OR ≈ 0.30). All studies fell within or near the pseudo-95% confidence limits, with an even distribution of both large and small cohorts on either side of the mean. This symmetry indicates a low risk of publication bias, reinforcing the validity of the observed quantitative association.

Discussion

This systematic review and meta-analysis of six studies (>7.6 million individuals) demonstrates a highly significant positive association between atherogenic dyslipidemia and hypertension severity. Patients presenting with elevated composite atherogenic indices specifically the Atherogenic Index of Plasma (AIP), Lipoprotein Combined Index (LCI), or Hypertriglyceridemic Waist (HTGW) exhibit a

35% greater likelihood of severe, poorly controlled hypertension vcompared to those with normative lipid profiles.

This relationship is driven by the intertwined pathophysiology of insulin resistance and vascular dysfunction. In T2DM, systemic insulin resistance stimulates hepatic very-low-density lipoprotein (VLDL) overproduction, lowers cardioprotective high-density lipoprotein (HDL), and increases highly oxidizable small, dense low-density lipoproteins (sdLDL). These atherogenic particles easily penetrate the arterial intima, triggering macrophage engulfment and chronic, low-grade inflammation. The resulting endothelial dysfunction limits nitric oxide-mediated vasodilation, promoting arterial stiffness and sympathetic activation. Clinically, this manifests as elevated systolic and diastolic blood pressure, explaining the severe hypertension phenotypes observed by Kim et al. and Zhao & Ling.

Crucially, our findings highlight the inadequacy of relying solely on standard LDL-C metrics for cardiovascular risk stratification. Hong et al. demonstrated that significant vascular calcification and hypertension persist in patients with elevated AIP despite "normal" LDL-C levels. Furthermore, Dalal et al. showed that patients with the "triple comorbidity" of T2DM, dyslipidemia, and hypertension frequently fail to achieve blood pressure targets despite multi-drug regimens. Therefore, managing the entire atherogenic triad is essential when treating resistant hypertension in diabetic patients.

Several limitations warrant consideration. The predominantly cross-sectional and retrospective designs of the included studies preclude causal inferences. Additionally, while all studies assessed atherogenic dyslipidemia, the use of varying diagnostic indices (AIP, LCI, HTGW) and the diverse geographic populations across Asia and Africa inherently introduce methodological and clinical heterogeneity. Future longitudinal studies using standardized atherogenic indices are needed to confirm these cardiovascular trajectories.

CONCLUSIONS

Atherogenic dyslipidemia is a significant, independent driver of hypertension severity in patients with metabolic risk factors, including T2DM. Elevated composite lipid markers strongly predict higher blood pressure gradients and poorer clinical hypertension control. To optimize cardiovascular risk reduction, clinical management protocols must evolve beyond isolated LDL-C targets to systematically assess and treat comprehensive atherogenic lipid profiles.

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