Carotid Intima-Media Thickness in Patients with Type II Diabetes Mellitus: A Narrative Review

Original Article

*Abubakar A. Abubakar[1], Daniel C. Ugwuanyi[2], Joseph C. Eze[2], Victor K. Nwodo[2], Emmanuel E. Ezugwu[2], Mansur Ramalan[3], Anas Yau[1], Umar Mansur[1], Hassan Mohammed[1]

[1]Department of Radiography, Bayero University Kano, Nigeria, [2]Department of Radiography, Nnamdi Azikiwe University, Awka, Nigeria, [3]Aminu Kano Teaching Hospital/ Bayero University Kano

Corresponding author: aaabubkar.rad@buk.edu.ng +2348039290734

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ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a major global health challenge and a leading cause of cardiovascular morbidity and mortality owing to accelerated atherosclerosis. Carotid intima-media thickness (CIMT), measured using B-mode ultrasonography, has emerged as a reliable, non-invasive marker of subclinical atherosclerosis and an important predictor of future cardiovascular events.

Aim: This narrative review critically evaluates current evidence on the role of B-mode ultrasonography in assessing CIMT among patients with T2DM, with emphasis on its pathophysiological basis, determinants, clinical applications, and future research directions.

Methods: A comprehensive literature search was conducted using PubMed/MEDLINE, Scopus, Web of Science, Embase, Google Scholar, and the Cochrane Library for studies published between January 2000 and June 2026. Evidence from systematic reviews, meta-analyses, randomized controlled trials, cohort studies, case-control studies, and clinical guidelines was synthesized narratively.

Results: The reviewed evidence consistently demonstrates that patients with T2DM have significantly greater CIMT than non-diabetic individuals, reflecting accelerated vascular ageing and early subclinical atherosclerosis. Increased CIMT is influenced by advancing age, longer diabetes duration, poor glycaemic control, hypertension, dyslipidaemia, obesity, renal dysfunction, and adverse lifestyle factors. Among antidiabetic therapies, metformin has the most consistent evidence of vascular protection, while sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs) show promising additional benefits in slowing CIMT progression.

Conclusion: Although further standardized multicentre longitudinal studies are required to strengthen its routine clinical application, CIMT assessment represents an important tool for the early detection of vascular injury and monitoring of cardiovascular risk in patients with T2DM.

Keywords: Type 2 diabetes mellitus; carotid intima-media thickness; B-mode ultrasonography;

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Introduction

Type 2 diabetes mellitus (T2DM) is the most common form of diabetes mellitus and represents a major global public health challenge because of its rapidly increasing prevalence and associated cardiovascular complications. Characterized by chronic hyperglycaemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction, T2DM is closely associated with obesity, physical inactivity, unhealthy dietary habits, ageing, and genetic susceptibility [1]. According to the International Diabetes Federation (2021), approximately 537 million adults were living with diabetes in 2021, with projections increasing to 643 million by 2030 and 783 million by 2045 [2]. More than 90% of these cases are attributed to T2DM, with the greatest increase expected in low- and middle-income countries, particularly in sub-Saharan Africa. In Nigeria, the prevalence of T2DM continues to rise, while delayed diagnosis and poor glycaemic control contribute substantially to the increasing burden of vascular complications [3].

Cardiovascular disease remains the leading cause of morbidity and mortality among individuals with T2DM, accounting for approximately two-thirds of diabetes-related deaths worldwide [4]. Compared with non-diabetic individuals, patients with T2DM have a two- to four-fold greater risk of myocardial infarction, stroke, and cardiovascular mortality [5]. This excess cardiovascular risk results primarily from accelerated atherosclerosis driven by chronic hyperglycaemia, oxidative stress, endothelial dysfunction, inflammation, advanced glycation end-product formation, dyslipidaemia, and insulin resistance [6-9]. These pathological processes frequently develop years before overt cardiovascular disease becomes clinically apparent, emphasizing the importance of identifying subclinical vascular abnormalities at an early stage.

Carotid intima-media thickness (CIMT) has emerged as one of the most widely accepted non-invasive markers of subclinical atherosclerosis. Measured using high-resolution B-mode ultrasonography, CIMT represents the combined thickness of the intimal and medial layers of the carotid artery and reflects cumulative exposure to cardiovascular risk factors before the development of overt atherosclerotic plaque [10, 11]. Numerous prospective cohort studies and systematic reviews have demonstrated that increased CIMT independently predicts future myocardial infarction, stroke, and cardiovascular mortality, making it an important imaging biomarker for cardiovascular risk assessment [12-14]. Among patients with T2DM, increased CIMT has consistently been associated with poor glycaemic control, longer duration of diabetes, hypertension, dyslipidaemia, obesity, and renal dysfunction [15-17]. B-mode ultrasonography remains the preferred imaging modality for CIMT assessment because it is safe, radiation-free, relatively inexpensive, reproducible, and widely available.

The development of standardized imaging protocols and consensus recommendations has further improved measurement accuracy and inter-observer reproducibility, facilitating its application in both clinical practice and research [19-20]. Nevertheless, considerable heterogeneity persists among published studies regarding ultrasonographic techniques, measurement protocols, study populations, and determinants of CIMT progression. Furthermore, studies from sub-Saharan Africa remains are limited despite the rapidly increasing burden of T2DM in the region [3,13,21].

This narrative review critically examines studies regarding the ultrasonographic assessment of carotid intima-media thickness in patients with T2DM. It discusses the pathophysiological basis of increased CIMT, principles of ultrasonographic measurement, determinants of CIMT progression, evidence comparing diabetic and non-diabetic populations, the effects of antidiabetic therapies, current clinical applications, limitations, and future research directions. By synthesizing contemporary evidence, this review aims to provide clinicians and researchers with an updated understanding of the role of carotid ultrasonography in the cardiovascular evaluation of patients with T2DMMethodology

Study Design

This study was conducted as a narrative literature review to synthesize current evidence on the use of B-mode ultrasonography for assessing carotid intima-media thickness (CIMT) in patients with type 2 diabetes mellitus (T2DM). The review examined the pathophysiology of diabetic atherosclerosis, the role of carotid ultrasonography in detecting subclinical

vascular disease, determinants of CIMT, differences between diabetic and non-diabetic populations, the effects of antidiabetic therapies on CIMT progression, and the clinical utility of CIMT in cardiovascular risk assessment. A narrative review design was selected because of the broad scope of the topic and the heterogeneity of available evidence.

Literature Search Strategy

A comprehensive literature search was conducted using PubMed/MEDLINE, Scopus, Web of Science, Embase, Google Scholar, and the Cochrane Library. Studies published between January 2000 and June 2026 were searched using Medical Subject Headings (MeSH) and free-text terms, including *type 2* diabetes mellitus, carotid intima-media thickness, common carotid artery, B-mode ultrasonography, subclinical atherosclerosis, cardiovascular disease *and* macrovascular complications. *Boolean* operators were used to combine search terms, and the reference lists of relevant articles, systematic reviews, landmark studies, and international guidelines were manually screened to identify additional eligible publications.

Study Selection and Eligibility Criteria

Titles and abstracts of retrieved studies were screened for relevance, followed by full-text review of potentially eligible articles. Priority was given to high-quality evidence, including systematic reviews, meta-analyses, randomized controlled trials, cohort studies, case-control studies, multicentre observational studies, and evidence-based clinical guidelines. Studies were included if they involved adults with T2DM, assessed CIMT using B-mode ultrasonography, investigated determinants of CIMT, compared diabetic with non-diabetic populations, evaluated the vascular effects of antidiabetic therapies, or examined associations between CIMT and cardiovascular outcomes. Studies involving type 1 diabetes, gestational diabetes, paediatric populations, animal experiments, conference abstracts without full-text publications, editorials, letters, expert opinions, duplicate reports, or those lacking adequate CIMT assessment or methodological detail were excluded.

Data Extraction and Narrative Synthesis

Data extracted from eligible studies included study design, setting, sample size, participant characteristics, ultrasonographic protocol, CIMT measurements, cardiovascular risk factors, therapeutic interventions, and principal findings. Owing to substantial heterogeneity in study designs, participant characteristics, imaging protocols, outcome measures, and follow-up duration, quantitative meta-analysis was not undertaken. Instead, findings were synthesized narratively and organized into thematic sections covering the epidemiology of T2DM, diabetic macrovascular disease, CIMT as a marker of subclinical atherosclerosis, determinants of CIMT, comparisons between diabetic and non-diabetic populations, effects of antidiabetic medications on CIMT progression, current clinical applications, limitations of existing study and future research directions.

Discussion

Pathophysiological Basis of Carotid Intima-Media Thickness in Type 2 Diabetes Mellitus

Type 2 diabetes mellitus (T2DM) is characterized by chronic hyperglycaemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction, which collectively accelerate the development of atherosclerosis and substantially increase the risk of cardiovascular disease [1, 4, 7]. Vascular injury begins years before the onset of clinically overt cardiovascular disease and is initiated by persistent metabolic disturbances that activate several interrelated biochemical pathways, including advanced glycation end-product (AGE) formation, oxidative stress, activation of protein kinase C, increased polyol pathway flux, and the hexosamine biosynthetic pathway [8,9,22]. These mechanisms induce endothelial dysfunction, chronic vascular inflammation, smooth muscle cell proliferation, extracellular matrix remodelling, and progressive thickening of the carotid arterial wall.

Endothelial dysfunction is considered the earliest detectable vascular abnormality in T2DM and plays a pivotal role in the initiation of atherosclerosis. Chronic hyperglycaemia reduces nitric oxide bioavailability while increasing the production of reactive oxygen species, resulting in impaired vasodilatation, enhanced leukocyte adhesion, platelet activation, and increased vascular permeability [6,9,22]. Oxidative stress further amplifies vascular injury through oxidation of low-density lipoprotein cholesterol (LDL-C), promotion of foam-cell formation, and activation of pro-inflammatory signalling pathways that accelerate atherosclerotic lesion development [6,9]. Persistent

hyperglycaemia also promotes the non-enzymatic glycation of proteins and lipids, leading to the accumulation of AGEs within the arterial wall. Binding of AGEs to their cellular receptor (RAGE) activates inflammatory transcription factors, increases oxidative stress, and stimulates vascular smooth muscle cell migration and proliferation, thereby contributing to arterial stiffness and progressive intima-media thickening [8,22]. Simultaneously, insulin resistance promotes hyperinsulinaemia, which enhances smooth muscle cell growth and extracellular matrix deposition, further accelerating vascular remodelling [6,7].

In addition to hyperglycaemia, diabetic dyslipidaemia and chronic low-grade inflammation substantially contribute to carotid atherosclerosis [7]. Elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and increased concentrations of small dense LDL particles facilitate lipid deposition within the arterial wall, whereas inflammatory mediators sustain endothelial injury and plaque progression [7, 23-25]. Coexisting hypertension further aggravates vascular damage through increased haemodynamic stress, endothelial disruption, collagen deposition, and arterial stiffening, thereby promoting progressive increases in carotid intima-media thickness (CIMT) [26-28]. The cumulative interaction of these metabolic, inflammatory, and haemodynamic abnormalities produces structural alterations within the carotid artery that can be detected non-invasively using high-resolution B-mode ultrasonography. Consequently, CIMT has become an established marker of subclinical atherosclerosis and an important surrogate indicator of cardiovascular risk in patients with T2DM [10, 13, 18].

Numerous prospective cohort studies and systematic reviews have demonstrated that increased CIMT independently predicts future myocardial infarction, stroke, and cardiovascular mortality, while serial CIMT assessment provides a valuable means of monitoring disease progression and evaluating the vascular effects of therapeutic interventions [12-15].

Factors Influencing Carotid Intima-Media Thickness in Type 2 Diabetes Mellitus

Age: Age is one of the strongest physiological determinants of carotid intima-media thickness (CIMT). Progressive arterial ageing is characterized by endothelial dysfunction, arterial stiffness, collagen deposition, elastin fragmentation, and vascular smooth muscle hypertrophy, all of which contribute to gradual thickening of the carotid arterial [26,27]. Large epidemiological studies have consistently demonstrated a linear increase in CIMT with advancing age. The Atherosclerosis Risk in Communities (ARIC) study identified age as one of the strongest independent predictors of CIMT, while Engelen et al. [29] established age-specific reference intervals demonstrating progressive increases in CIMT across adulthood [29, 30]. In patients with type 2 diabetes mellitus (T2DM), chronic hyperglycaemia accelerates vascular ageing through oxidative stress, endothelial dysfunction, and chronic inflammation, resulting in greater CIMT than that observed in age-matched non-diabetic individuals [7, 27, 31]. Although age remains an independent predictor of CIMT, its effect is partially mediated by the cumulative influence of hypertension, dyslipidaemia, obesity, and diabetes duration [13,14].

Gender: Gender differences in CIMT have been reported in both diabetic and non-diabetic populations. Men generally exhibit greater CIMT than women during early and middle adulthood because of higher exposure to cardiovascular risk factors and the protective vascular effects of oestrogen before menopause [14,32]. Oestrogen improves endothelial function, suppresses vascular inflammation, and enhances lipid metabolism; however, these protective effects diminish after menopause, resulting in accelerated arterial ageing in women [32]. Among patients with T2DM, diabetes-related endothelial dysfunction largely attenuates this female vascular advantage, and several studies have demonstrated comparable CIMT values between men and women after adjustment for age, blood pressure, obesity, and glycaemic status [15, 31, 32].

Duration of Diabetes Mellitus: Duration of diabetes is a major determinant of CIMT because it reflects cumulative exposure to chronic hyperglycaemia and its associated vascular injury. Prolonged diabetes promotes endothelial dysfunction, oxidative stress, inflammation, vascular smooth muscle proliferation, and advanced glycation end-product accumulation, leading to progressive arterial wall thickening [8,9]. Several longitudinal and observational studies have demonstrated that longer diabetes duration is

independently associated with increased CIMT and faster progression of subclinical atherosclerosis [3, 15, 31]. Consequently, diabetes duration is regarded as an indicator of cumulative glycaemic burden rather than simply disease chronology, and patients with longstanding diabetes frequently exhibit increased CIMT despite apparently satisfactory glycaemic control [16,33]**.** Poor glycaemic control is a key determinant of CIMT in patients with T2DM. Glycated haemoglobin (HbA1c), which reflects long-term glycaemic exposure, has consistently demonstrated stronger associations with CIMT than fasting plasma glucose because it better represents cumulative hyperglycaemia [16, 34]. Persistent hyperglycaemia promotes oxidative stress, endothelial dysfunction, inflammation, and advanced glycation end-product accumulation, resulting in progressive thickening of the carotid arterial wall [8,9].

Numerous studies have reported positive associations between HbA1c and CIMT, while glycaemic variability has also been shown to independently contribute to vascular injury and CIMT progression [34, 35]. Although intensive glycaemic control may slow progression of CIMT, reversal of established vascular changes is generally limited, indicating the importance of early intervention [36, 37].

Hypertension: Hypertension is among the most important determinants of CIMT in patients with T2DM because elevated blood pressure accelerates endothelial injury, vascular remodelling, arterial stiffness, and medial hypertrophy [28, 38]**.** Both systolic and diastolic blood pressures correlate positively with CIMT, although systolic blood pressure is generally considered the stronger predictor because it reflects arterial stiffness and cumulative haemodynamic stress [38]. Longitudinal studies have demonstrated that poor blood pressure control significantly accelerates CIMT progression, whereas intensive blood pressure management reduces cardiovascular risk in diabetic populations [1].

Obesity: Obesity contributes substantially to CIMT progression through insulin resistance, chronic inflammation, oxidative stress, endothelial dysfunction, and adipose tissue-derived inflammatory cytokines [39]. Body mass index (BMI) remains the most widely used anthropometric measure and has consistently shown positive associations with CIMT among patients with T2DM (World Health Organization, 2000 [40]. Measures of central obesity generally correlate more strongly with vascular injury than body surface area because visceral adiposity is metabolically more active and contributes to insulin resistance and inflammation [39]. Although several novel obesity indices have been proposed, BMI and waist-related measures remain the most practical predictors of obesity-associated vascular risk [41].

Dyslipidaemia: Diabetic dyslipidaemia is characterized by elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C), and increased concentrations of small dense low-density lipoprotein cholesterol (LDL-C), all of which contribute to accelerated carotid atherosclerosis [23, 24]. LDL-C is the principal atherogenic lipoprotein and demonstrates the strongest positive association with CIMT because of its central role in plaque formation and vascular inflammation [23]. Elevated triglycerides also contribute to endothelial dysfunction, while HDL-C exerts anti-inflammatory, antioxidant, and endothelial protective effects; consequently, reduced HDL-C concentrations are associated with greater CIMT and increased cardiovascular risk [24, 25]. These findings support aggressive lipid-lowering therapy as an essential component of cardiovascular risk reduction in patients with T2DM.

Renal Function: Renal dysfunction independently predicts increased CIMT in patients with T2DM through mechanisms involving endothelial dysfunction, chronic inflammation, oxidative stress, vascular calcification, and arterial stiffening [17]. Reduced estimated glomerular filtration rate (eGFR) and albuminuria have consistently been associated with increased CIMT and higher cardiovascular event rates, even in patients without overt chronic kidney disease [17]. Because vascular injury often precedes clinically apparent nephropathy, CIMT assessment may provide valuable information regarding cardiovascular risk in diabetic patients with early renal impairment.

Lifestyle: Lifestyle behaviours substantially influence CIMT and cardiovascular risk among individuals with T2DM. Cigarette smoking accelerates endothelial dysfunction, oxidative stress, inflammation, and arterial stiffening, thereby promoting increased CIMT

[42]. Conversely, regular physical activity improves endothelial function, insulin sensitivity, blood pressure, and lipid metabolism while slowing progression of subclinical atherosclerosis [43]. Healthy dietary patterns, particularly the Mediterranean diet, have also demonstrated beneficial effects on cardiovascular risk factors and CIMT progression through improvements in glycaemic control, blood pressure, lipid profile, and systemic inflammation [44]

Antidiabetic Medications and Carotid Intima-Media Thickness: Antidiabetic medications differ considerably in their effects on vascular health beyond glucose lowering. Metformin improves endothelial function, reduces oxidative stress and inflammation, and has consistently been associated with slower CIMT progression and reduced cardiovascular risk [17]. Sulfonylureas effectively improve glycaemic control but have shown less consistent effects on CIMT because of their association with weight gain and hyperinsulinaemia [45]. More recently, sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have demonstrated significant cardiovascular benefits by improving endothelial function, reducing arterial stiffness and inflammation, and potentially slowing CIMT progression independent of glucose lowering [46]. These observations suggest that selection of antidiabetic therapy should consider both glycaemic efficacy and cardiovascular protection [46].

Carotid Intima-Media Thickness in Patients with Type 2 Diabetes Mellitus

Several studies consistently demonstrates that individuals with type 2 diabetes mellitus (T2DM) exhibit significantly greater carotid intima-media thickness (CIMT) than non-diabetic individuals, reflecting accelerated subclinical atherosclerosis and premature vascular ageing [12, 13, 14]. Diabetes promotes persistent endothelial injury through chronic hyperglycaemia, oxidative stress, inflammation, advanced glycation end-product accumulation, and vascular smooth muscle proliferation, all of which contribute to progressive thickening of the carotid arterial wall [6,8, 19].

Large population-based studies have established diabetes as an independent determinant of increased CIMT after adjustment for conventional cardiovascular risk factors. A systematic review and meta-analysis by [14] further confirmed that increased CIMT independently predicts myocardial infarction and stroke, reinforcing its value as a surrogate marker of cardiovascular risk.

Among patients with T2DM, CIMT has been shown to increase progressively with advancing age, longer diabetes duration, poor glycaemic control, hypertension, obesity, dyslipidaemia, and declining renal function [15,16,31]. Consequently, diabetic patients generally exhibit higher CIMT values than healthy age- and sex-matched controls, even in the absence of clinically overt cardiovascular disease [13,15]. These findings support the concept that atherosclerosis develops silently for many years before clinical manifestations become evident.

Evidence from different geographical regions consistently supports these observations. Studies conducted in Nigeria reported significantly greater CIMT among patients with T2DM compared with non-diabetic controls and demonstrated positive associations between CIMT, diabetes duration, blood pressure, and lipid abnormalities [3]. Similar findings have been reported in Egypt, where diabetic patients exhibited significantly increased carotid wall thickness and a higher prevalence of subclinical atherosclerosis than healthy controls [21]. Likewise, research from Iran demonstrated that diabetic patients, particularly those with established coronary artery disease, had significantly greater CIMT than non-diabetic individuals [47]. Collectively, these studies indicate that the association between T2DM and increased CIMT is consistent across diverse ethnic and geographical populations.

Longitudinal investigations further demonstrate that individuals with diabetes experience faster annual CIMT progression than non-diabetic populations, reflecting the cumulative vascular effects of chronic hyperglycaemia and associated metabolic abnormalities [14]. Serial Carotid ultrasonographic assessment has therefore become valuable for monitoring vascular disease progression and evaluating the effectiveness of cardiovascular risk-reduction strategies in patients with diabetes [15, 41].

Despite consistent studies supporting increased CIMT in T2DM, absolute CIMT values vary across studies

because of differences in age distribution, ethnicity, cardiovascular risk profiles, ultrasound equipment, image acquisition techniques, and measurement protocols. Adoption of standardized imaging recommendations, particularly the Mannheim Carotid Intima-Media Thickness Consensus, has substantially improved measurement reproducibility and comparability across clinical and research settings [19, 20].

Effect of Antidiabetic Medications on Carotid Intima-Media Thickness in Type 2 Diabetes Mellitus

Carotid intima-media thickness (CIMT) has increasingly been used as a surrogate marker for evaluating the vascular effects of antidiabetic therapies beyond glycaemic control [15]. Although lowering blood glucose remains the primary objective of diabetes management, accumulating evidence indicates that different classes of glucose-lowering agents exert variable effects on endothelial function, oxidative stress, inflammation, arterial stiffness, and atherosclerosis progression. Consequently, serial CIMT assessment has become an important tool for evaluating the cardiovascular benefits of antidiabetic medications [15, 41].

Metformin remains the first-line pharmacological treatment for T2DM because of its favourable metabolic and cardiovascular effects. Beyond reducing hepatic glucose production and improving insulin sensitivity, metformin enhances endothelial function, suppresses oxidative stress and inflammation, and improves vascular nitric oxide bioavailability. Clinical studies have demonstrated slower CIMT progression among metformin-treated patients, supporting its protective effect against subclinical atherosclerosis [15,16]. The United Kingdom Prospective Diabetes Study (UKPDS) further demonstrated that intensive glucose control with metformin significantly reduced diabetes-related cardiovascular events, particularly among overweight patients [37].

Sulfonylureas effectively reduce hyperglycaemia by stimulating pancreatic insulin secretion but appear to provide less consistent vascular protection than metformin. Although improved glycaemic control may indirectly slow CIMT progression, the associated risks of weight gain, hyperinsulinaemia, and hypoglycaemia may offset some of their cardiovascular benefits [45]. Consequently, evidence regarding the direct influence of sulfonylureas on CIMT remains inconclusive.

Recent advances in diabetes management have highlighted the cardiovascular benefits of sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs). Large cardiovascular outcome trials demonstrated that empagliflozin significantly reduced cardiovascular mortality and hospitalization for heart failure, whereas liraglutide reduced major adverse cardiovascular events and cardiovascular mortality among patients with T2DM [46]. These benefits appear to extend beyond glycaemic control and are attributed to improvements in endothelial function, reductions in oxidative stress and inflammation, decreased arterial stiffness, favourable effects on body weight and blood pressure, and attenuation of vascular remodelling.

Clinical Implications

Carotid ultrasonography has become an important tool for cardiovascular risk assessment in patients with type 2 diabetes mellitus (T2DM) by enabling early detection of subclinical atherosclerosis before the onset of overt cardiovascular disease. Measurement of carotid intima-media thickness (CIMT) helps identify patients with accelerated vascular ageing, thereby supporting individualized decisions regarding lipid-lowering therapy, blood pressure management, antiplatelet treatment, and lifestyle modification. In resource-limited settings, carotid ultrasonography is particularly valuable because it is non-invasive, radiation-free, relatively inexpensive, reproducible, and widely available, making it a practical alternative where advanced cardiovascular imaging is inaccessible. Although routine CIMT screening is not recommended for the general population, evidence suggests that selected high-risk groups, including patients with T2DM, chronic kidney disease, familial hypercholesterolaemia, and multiple cardiovascular risk factors, may benefit from carotid ultrasound assessment for improved cardiovascular risk stratification.

Future Research Directions

Future research should prioritize large, multicentre longitudinal studies using standardized ultrasound

protocols to establish population-specific carotid intima-media thickness (CIMT) reference values and improve inter-study comparability. Well-designed randomized controlled trials are also needed to compare the long-term vascular effects of different antidiabetic therapies on CIMT progression. Furthermore, integrating novel biomarkers with advanced ultrasound technologies, including artificial intelligence and automated image analysis, may enhance the accuracy of cardiovascular risk assessment in patients with type 2 diabetes mellitus.

Conclusion

Type 2 diabetes mellitus (T2DM) accelerates subclinical atherosclerosis, increasing the risk of cardiovascular disease. Carotid intima-media thickness (CIMT), measured by B-mode ultrasonography, is a reliable, non-invasive marker of early vascular injury and consistently demonstrates greater carotid wall thickness in patients with T2DM than in non-diabetic individuals. CIMT is influenced by several factors, including age, duration of diabetes, glycaemic control, hypertension, dyslipidaemia, obesity, renal dysfunction, and lifestyle behaviours. Among antidiabetic therapies, metformin has the most consistent evidence of vascular protection, while newer agents such as sodium-glucose cotransporter-2 (SGLT2) inhibitors and glucagon-like peptide-1 receptor agonists (GLP-1 RAs) show promising cardiovascular benefits.

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33. Below is Batch 3 (References 33–47) formatted in Vancouver style with official NLM journal abbreviations.

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