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Dual Cardiometabolic Disorders: An Overview of Co-Existing Type 2 Diabetes Mellitus and Hypertension

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Abstract : *Type 2 diabetes mellitus (T2DM) and hypertension are common chronic disorders that frequently coexist and substantially increase the risk of cardiovascular disease, stroke, and kidney complications. Their association is supported by shared mechanisms, including insulin resistance, chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and activation of the renin–angiotensin–aldosterone system (RAAS). Persistent hyperglycaemia contributes to vascular injury, while elevated blood pressure can accelerate renal and cardiovascular damage and is commonly associated with metabolic dysfunction. This narrative review summarizes the epidemiology and shared risk factors of T2DM and hypertension, explains their interrelated pathophysiology, and discusses symptoms, diagnosis, lifestyle measures, and pharmacological management. Antidiabetic treatment may include metformin, sodium–glucose cotransporter-2 inhibitors, and glucagon-like peptide-1 receptor agonists, selected according to individual clinical characteristics. Antihypertensive treatment may include angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium channel blockers, and thiazide-like diuretics. Calcium channel blocker mechanisms and important adverse effects are also reviewed. Integrated care, early detection, individualized treatment, and regular monitoring of blood glucose, blood pressure, kidney function, and cardiovascular risk are essential to reduce complications.*

Keywords: Type 2 diabetes mellitus; Hypertension; Cardiometabolic disorders; Insulin resistance; Oxidative stress; Endothelial dysfunction; Renin–angiotensin–aldosterone system; Cardiovascular disease.

I. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from impaired insulin secretion, reduced insulin sensitivity, or both. It is the most common form of diabetes. In T2DM, pancreatic β-cells do not release sufficient insulin to compensate for insulin resistance in peripheral tissues. As a result, glucose uptake by muscle and adipose tissue is impaired, while hepatic glucose production may remain increased [1,2].

Hypertension is a sustained elevation of arterial blood pressure. T2DM and hypertension frequently occur together because they share several risk factors and biological pathways. Obesity, physical inactivity, unhealthy dietary patterns, ageing, and genetic susceptibility may contribute to both conditions. Insulin resistance, inflammation, oxidative stress, endothelial dysfunction, and RAAS activation further connect their development and progression [3,4].

The coexistence of T2DM and hypertension increases the likelihood of cardiovascular disease, stroke, chronic kidney disease, and other vascular complications. Persistent hyperglycaemia can impair blood vessel function, while elevated blood pressure places additional stress on the heart, kidneys, and vasculature. Therefore, early identification and coordinated management of blood glucose and blood pressure are important for reducing long-term complications [3,5].

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This narrative review summarizes the relationship between T2DM and hypertension, including their epidemiology, causes, risk factors, pathophysiology, clinical presentation, diagnosis, and management. It also outlines the mechanism of action and adverse drug reactions of calcium channel blockers.

II. REVIEW APPROACH

This article is a narrative review prepared from the literature and references included in the supplied review draft. It brings together the main themes concerning the coexistence of T2DM and hypertension. A formal systematic database search, predefined eligibility criteria, and quantitative meta-analysis were not documented in the source material; therefore, the article should not be interpreted as a systematic review.

III. EPIDEMIOLOGY AND CLINICAL IMPORTANCE

Diabetes is a major and increasing global public-health concern. Its growing prevalence places a substantial burden on individuals and healthcare systems. Cardiovascular disease is among the most important complications associated with diabetes, and people with T2DM have increased risk of ischaemic heart disease, myocardial infarction, heart failure, and stroke. This risk is influenced by the combined effects of hyperglycaemia, hypertension, obesity, dyslipidaemia, and insulin resistance [5,6].

Hypertension is also common among people with diabetes. The two conditions frequently cluster with other metabolic abnormalities, including excess adiposity and abnormal lipid levels. Their coexistence is clinically important because each condition can worsen the effects of the other. Diabetes-related vascular and renal injury may complicate blood-pressure control, while uncontrolled hypertension can accelerate cardiovascular and kidney damage [3,4].

Cardiovascular risk may begin to increase before blood glucose reaches the diagnostic range for diabetes. Consequently, assessment of blood pressure, lipid profile, kidney function, and other cardiovascular risk factors is relevant even during early metabolic disease [5].

IV. CAUSES AND RISK FACTORS

The development of T2DM and hypertension is multifactorial. Genetic susceptibility interacts with environmental and lifestyle factors. The presence of one risk factor does not mean that an individual will necessarily develop either condition.

4.1 Shared risk factors

- Excess body weight and central adiposity: Excess adipose tissue is associated with insulin resistance and metabolic abnormalities.
- Physical inactivity: Low levels of physical activity can contribute to weight gain, reduced insulin sensitivity, and poor cardiometabolic health.
- Unhealthy dietary patterns: Diets high in saturated fat, refined carbohydrates, and sodium, and low in fruits, vegetables, and fibre, may increase cardiometabolic risk.
- Age and family history: Increasing age and a family history of diabetes or hypertension are important non-modifiable risk factors.
- Dyslipidaemia: Abnormal triglyceride and cholesterol levels commonly accompany insulin resistance and increase cardiovascular risk.
- Smoking and excessive alcohol consumption: These behaviours can adversely affect vascular and metabolic health [3,7,8].

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4.2 Factors particularly relevant to hypertension

High sodium intake is associated with increased blood pressure in many populations. Evidence reviewed in the source material highlights excessive salt consumption as a public-health concern, including in South Asian dietary patterns [8]. Other contributing factors include obesity, kidney disease, sleep apnoea, stress, smoking, ageing, and certain medicines.

4.3 Factors particularly relevant to T2DM

Insulin resistance, progressive pancreatic β-cell dysfunction, genetic susceptibility, excess body weight, physical inactivity, and a history of gestational diabetes are important factors associated with T2DM. Persistent metabolic stress can gradually reduce the ability of β-cells to maintain adequate insulin secretion [1,2,9].

V. PATHOPHYSIOLOGY

5.1 Pathophysiology of type 2 diabetes mellitus

T2DM develops through the interaction of insulin resistance and progressive β-cell dysfunction. In insulin resistance, skeletal muscle, liver, and adipose tissue respond less effectively to insulin. Reduced glucose uptake by peripheral tissues and increased hepatic glucose production contribute to hyperglycaemia. Initially, pancreatic β-cells may compensate by increasing insulin secretion. Over time, this compensatory response may become inadequate, resulting in persistent elevation of blood glucose [1,2].

Chronic hyperglycaemia and increased circulating fatty acids contribute to glucotoxicity, lipotoxicity, and oxidative stress. These processes can impair β-cell function and promote inflammation. Inflammatory mediators, including tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), have been associated with insulin resistance and vascular dysfunction [2,10].

5.2 Pathophysiology of hypertension

Blood pressure is influenced by cardiac output, vascular resistance, kidney function, and neurohormonal regulation. Increased sympathetic nervous system activity, sodium and water retention, vascular stiffness, and impaired endothelial function can contribute to sustained elevation of blood pressure.

The RAAS is an important regulator of blood pressure and fluid balance. Renin initiates the conversion of angiotensinogen to angiotensin I; angiotensin-converting enzyme (ACE) then converts angiotensin I to angiotensin II. Angiotensin II causes vasoconstriction and stimulates aldosterone release. Aldosterone increases renal sodium and water retention, contributing to increased circulating volume and blood pressure [3,4].

Angiotensin II can also promote oxidative stress, inflammation, vascular smooth-muscle growth, and endothelial dysfunction. These effects contribute to vascular injury and may worsen cardiometabolic abnormalities [4,10].

5.3 Relationship between T2DM and hypertension

Insulin resistance and hyperinsulinaemia may increase sympathetic activity and renal sodium reabsorption. In parallel, hyperglycaemia and dyslipidaemia promote oxidative stress and inflammation, which reduce the normal ability of blood vessels to relax. Reduced nitric oxide availability and endothelial dysfunction can increase vascular resistance.

The kidney also contributes to the relationship between the two disorders. Altered renal sodium handling and RAAS activity may contribute to blood-pressure elevation. Conversely, longstanding hypertension can damage renal blood vessels and impair kidney function, increasing the complexity of diabetes management. Thus, T2DM and hypertension may reinforce one another through interconnected metabolic, vascular, renal, and hormonal pathways [3,4,11].

5.4 Major consequences

When T2DM and hypertension coexist, the combined burden increases the risk of atherosclerosis, coronary artery disease, stroke, heart failure, and diabetic kidney disease. Other diabetes-related complications may include retinopathy and neuropathy. Regular assessment of vascular and renal health is therefore an important part of long-term care [5,6].

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VI. SYMPTOMS AND DIAGNOSIS

6.1 Symptoms

T2DM may develop gradually, and some individuals have no noticeable symptoms during its early stages. Common symptoms include increased thirst, frequent urination, increased hunger, tiredness, blurred vision, delayed wound healing, and tingling or numbness in the hands and feet.

Hypertension is often called a silent condition because many people do not experience obvious symptoms. When blood pressure is markedly elevated, symptoms such as headache, dizziness, blurred vision, chest discomfort, or breathlessness may occur. Symptoms alone cannot confirm either diagnosis; appropriate measurements and laboratory tests are required [12,13].

6.2 Diagnosis of T2DM

T2DM is diagnosed using laboratory tests. Common diagnostic criteria include:

- Fasting plasma glucose (FPG) ≥126 mg/dL.
- Glycated haemoglobin (HbA1c) ≥6.5%.
- Two-hour plasma glucose ≥200 mg/dL during a 75-g oral glucose tolerance test (OGTT).
- Random plasma glucose ≥200 mg/dL in a person with classic symptoms of hyperglycaemia or a hyperglycaemic crisis.

In the absence of unequivocal hyperglycaemia, an abnormal result generally requires confirmation by repeat testing [12].

6.3 Diagnosis of hypertension

Blood pressure should be measured using an appropriately sized cuff, correct positioning, and a standardized technique. Repeated measurements are important because a single reading may not represent usual blood pressure. The World Health Organization defines hypertension in adults as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg, confirmed on measurements taken on separate days [13]. Clinical thresholds and treatment targets may differ among guidelines; the 2026 American Diabetes Association Standards recommend individualized blood-pressure goals for people with diabetes and hypertension [14].

6.4 Assessment of complications

When both disorders coexist, assessment may include kidney function, urinary albumin, lipid profile, cardiovascular risk, and evaluation for diabetes-related eye and nerve complications. Home blood-pressure monitoring or ambulatory monitoring may be useful in selected patients [14,15].

VII. MANAGEMENT AND TREATMENT

Management aims to control blood glucose and blood pressure, reduce cardiovascular and kidney risk, and prevent complications. Treatment should be individualized according to age, comorbidities, kidney function, cardiovascular risk, adverse-effect profile, and patient preferences.

7.1 Lifestyle modification

Lifestyle measures form an important part of management:

• **Balanced diet:** Emphasize vegetables, fruits, whole grains, pulses, and other fibre-rich foods. Limit excess sodium, refined sugars, and foods high in saturated fat.

• **Salt reduction:** Reducing excess salt intake can support blood-pressure control.

• **Physical activity:** Regular, suitable physical activity can improve insulin sensitivity, cardiovascular fitness, and blood-pressure control.

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• **Weight management:** Maintaining a healthy body weight can improve metabolic health.
• **Avoid tobacco and limit or avoid alcohol:** These measures support cardiovascular health.
• **Adherence and education:** Patients should understand the importance of prescribed treatment, monitoring, and follow-up [7,8,14].

Dietary patterns such as the Mediterranean-style dietary approach have been associated with improvements in glycaemic and cardiovascular risk markers. Dietary advice should be adapted to individual nutritional needs and local food practices [14].

7.2 Antidiabetic medicines

• **Metformin:** Reduces hepatic glucose production and improves insulin sensitivity. It is commonly used when appropriate, with consideration of kidney function and tolerability.
• **Sodium–glucose cotransporter-2 (SGLT2) inhibitors:** Medicines such as empagliflozin and dapagliflozin increase urinary glucose excretion. In suitable patients, some agents in this class provide cardiovascular and kidney benefits.
• **Glucagon-like peptide-1 (GLP-1) receptor agonists:** Medicines such as semaglutide and liraglutide improve glucose-dependent insulin secretion and may provide cardiovascular and weight-related benefits in appropriate patients.
• **Other agents:** Dipeptidyl peptidase-4 (DPP-4) inhibitors, sulfonylureas, and insulin may be considered according to glycaemic requirements and individual clinical factors [14,16].

7.3 Antihypertensive medicines

• **ACE inhibitors**: Enalapril and lisinopril reduce the formation of angiotensin II and thereby reduce vasoconstriction. They are particularly useful in selected patients with diabetes and albuminuria.
• **Angiotensin receptor blockers (ARBs):** Losartan and telmisartan block the action of angiotensin II at its receptor. They are commonly used when clinically appropriate, including in patients with diabetic kidney disease.
• **Calcium channel blockers (CCBs):** Amlodipine and related agents reduce calcium entry into vascular smooth muscle and promote vasodilation.
• **Thiazide-like diuretics:** Indapamide and chlorthalidone increase renal sodium and water excretion, reducing circulating volume and blood pressure [14,15,17].

ACE inhibitors and ARBs should not routinely be combined. Kidney function and serum potassium should be monitored when medicines affecting the RAAS are initiated or adjusted. Drug selection and dose adjustment must be undertaken by a qualified healthcare professional [14,15].

7.4 Calcium channel blockers: mechanism of action

Calcium channel blockers inhibit L-type voltage-gated calcium channels and reduce calcium entry into vascular smooth-muscle cells. Reduced intracellular calcium causes vascular smooth-muscle relaxation, widening of blood vessels and a decrease in peripheral vascular resistance. This lowers blood pressure.

Dihydropyridine CCBs, such as amlodipine and nifedipine, act predominantly on vascular smooth muscle. Non-dihydropyridine CCBs, such as verapamil and diltiazem, also affect the heart and can reduce heart rate and the force of cardiac contraction [17].

7.5 Adverse drug reactions of calcium channel blockers

Common adverse effects include headache, dizziness, flushing, low blood pressure, and peripheral oedema (swelling of the ankles or feet). Dihydropyridines are more commonly associated with ankle oedema and flushing. Verapamil may cause constipation, while verapamil and diltiazem can cause bradycardia and atrioventricular conduction disturbances. Non-dihydropyridines may be unsuitable in some patients with heart failure or conduction disorders. Selection of a CCB should consider the patient's cardiovascular status and concurrent medicines [17].

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VIII. DISCUSSION

T2DM and hypertension are closely associated disorders with overlapping metabolic, vascular, renal, and hormonal mechanisms. Insulin resistance, chronic inflammation, oxidative stress, endothelial dysfunction, and RAAS activation help explain why the conditions frequently coexist. Their combined effects increase the likelihood of cardiovascular and kidney complications.

Lifestyle measures are important, but many patients also require pharmacological treatment. Contemporary management emphasizes selecting glucose-lowering medicines and antihypertensive agents according to cardiovascular and renal risk rather than focusing only on individual blood-glucose or blood-pressure values. SGLT2 inhibitors and GLP-1 receptor agonists have an important role in selected patients with T2DM, while ACE inhibitors or ARBs are particularly relevant in patients with hypertension and albuminuria or other appropriate indications [14].

The evidence also supports regular monitoring and patient education. Blood pressure, glycaemic status, kidney function, urinary albumin, and lipid levels should be reviewed as clinically appropriate. The source draft included discussion of nutraceutical ingredients such as monacolin K and Morus alba. These compounds have been investigated for lipid- and glucose-related effects; however, they should not be presented as established substitutes for guideline-directed treatment. Further well-designed clinical research is required to clarify their efficacy, safety, and place in care [18].

IX. CONCLUSION

Type 2 diabetes mellitus and hypertension commonly coexist and share several risk factors and pathophysiological pathways. Insulin resistance, chronic inflammation, oxidative stress, endothelial dysfunction, and RAAS activation contribute to the development and progression of both conditions. Their coexistence increases the risk of cardiovascular disease, stroke, kidney damage, and other long-term complications.

Early diagnosis, individualized pharmacological treatment, healthy dietary habits, regular physical activity, reduced excess salt intake, and ongoing monitoring are central to effective management. Antidiabetic and antihypertensive medicines should be selected according to the patient's overall cardiovascular and kidney risk. An integrated approach involving healthcare professionals and patient education may improve long-term outcomes. Further research is needed to clarify shared mechanisms and evaluate emerging therapeutic approaches.

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