



THE RELATIONSHIP BETWEEN TYPE 2 DIABETES MELLITUS AND ARTERIAL HYPERTENSION

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Abstract: Type 2 diabetes mellitus (T2DM) and arterial hypertension (AH) are among the most prevalent chronic diseases worldwide and frequently coexist in the same patient. Their combination significantly increases the risk of cardiovascular morbidity and mortality. This article explores the pathophysiological mechanisms linking T2DM and hypertension, highlighting the roles of insulin resistance, endothelial dysfunction, renal impairment, oxidative stress, and sympathetic nervous system activation. Understanding these mechanisms is essential for optimizing management strategies in clinical practice.

Keywords: Type 2 diabetes, arterial hypertension, insulin resistance, endothelial dysfunction, metabolic syndrome.

Introduction

Type 2 diabetes mellitus (T2DM) is a metabolic disorder characterized by chronic hyperglycemia resulting from insulin resistance and β -cell dysfunction. Arterial hypertension (AH), defined as persistently elevated blood pressure, is one of the most common comorbidities in T2DM patients. According to the American Diabetes Association (ADA), approximately **70–80% of individuals with T2DM also have hypertension**, and their coexistence markedly increases the risk of cardiovascular complications such as coronary artery disease, stroke, heart failure, and chronic kidney disease.

The relationship between T2DM and hypertension is not coincidental; rather, both diseases share several common pathogenic processes, including insulin resistance, endothelial dysfunction, activation of the renin–angiotensin–aldosterone system (RAAS), chronic inflammation, and obesity. This paper provides an overview of these mechanisms and explains why the coexistence of T2DM and AH forms a central component of metabolic syndrome.

1. Insulin Resistance as a Shared Pathophysiological Mechanism

Insulin resistance is a hallmark of T2DM and plays a central role in the development of hypertension. Under normal conditions, insulin promotes vasodilation by stimulating nitric oxide (NO) production in endothelial cells. However, when insulin resistance occurs:

- insulin-mediated vasodilation is impaired,



- sodium retention in the kidneys increases,
- sympathetic nervous system activity is enhanced,
- RAAS becomes more active.

These alterations contribute directly to blood pressure elevation. Reaven (1988) first highlighted insulin resistance as a key factor linking T2DM, hypertension, dyslipidemia, and visceral obesity, collectively known as metabolic syndrome.

2. Endothelial Dysfunction

Chronic hyperglycemia leads to the formation of advanced glycation end-products (AGEs), oxidative stress, and inflammation. These mechanisms damage the vascular endothelium, decrease NO synthesis, and increase vasoconstrictor factors such as endothelin-1 and angiotensin II.

As a result:

- vascular tone increases,
- arterial stiffness worsens,
- systemic vascular resistance rises,
- blood pressure becomes persistently elevated.

Endothelial dysfunction is therefore a key mechanism that links T2DM with arterial hypertension and accelerates the development of atherosclerosis.

3. Renal Mechanisms

The kidneys are essential regulators of blood pressure. In T2DM, diabetic nephropathy develops due to glomerular hyperfiltration, basement membrane thickening, and microvascular damage. As renal function declines:

- sodium and water retention increases,
- RAAS becomes hyperactive,
- renal vasoconstriction intensifies.

These changes significantly raise blood pressure. According to KDIGO guidelines, even mild albuminuria in diabetics is strongly associated with the early development of hypertension.

4. Sympathetic Nervous System Activation

Patients with T2DM often exhibit elevated levels of catecholamines due to:

- hyperinsulinemia,
- visceral obesity,



- leptin resistance,
- chronic inflammatory states.

Sympathetic overactivity leads to:

- increased heart rate,
- peripheral vasoconstriction,
- increased cardiac output.

This contributes directly to arterial hypertension and worsens cardiovascular risk.

5. Obesity and Adipose Tissue Dysfunction

Most individuals with T2DM present with central (abdominal) obesity. Visceral adipose tissue acts as an endocrine organ producing leptin, resistin, TNF- α , IL-6, and other pro-inflammatory cytokines.

These substances:

- worsen insulin resistance,
- activate RAAS,
- increase vascular inflammation,
- impair endothelial function.

Thus, obesity magnifies the bidirectional relationship between T2DM and hypertension.

6. Oxidative Stress and Chronic Inflammation

In diabetes, increased production of reactive oxygen species (ROS) and systemic inflammation alter vascular homeostasis. Oxidative stress:

- reduces NO bioavailability,
- increases vascular smooth muscle proliferation,
- promotes arterial stiffness.

Chronic inflammation additionally contributes to sodium retention and worsened vascular reactivity, both of which elevate blood pressure.

Clinical Importance

The coexistence of T2DM and arterial hypertension increases the likelihood of:

- coronary artery disease,
- myocardial infarction,



- stroke,
- heart failure,
- diabetic nephropathy,
- peripheral arterial disease.

Therefore, clinical guidelines recommend maintaining blood pressure **below 130/80 mmHg** in T2DM patients, using lifestyle modification and pharmacological therapies such as ACE inhibitors, ARBs, calcium channel blockers, and thiazide-like diuretics.

Conclusion

Type 2 diabetes mellitus and arterial hypertension share multiple interconnected pathophysiological mechanisms. Insulin resistance, endothelial dysfunction, RAAS activation, oxidative stress, sympathetic overactivity, and obesity represent the major links between these two conditions. Their coexistence significantly accelerates vascular damage, making aggressive management essential for preventing cardiovascular complications. Understanding the biological relationship between T2DM and hypertension enables clinicians to develop more effective treatment strategies and improve long-term outcomes for patients.

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