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density lipoproteins, insulin resistance, dyslipidaemia, and hyperglycaemia in compromising endothelial integrity. 0RUHRYHU ZH H[DPLQH KRZ LQFUHDVHG H[SUHVVLRQ RI SUR LQÀDPPDWRU\ F\WRNLQHV DQ WR YDVRGLODWRU\ G\VIXQFWLRQ LQ WKH HQGRWKHOLXP $GGLWLRQDOO\ ZH GLVFXVV WKH endothelial dysfunction. By elucidating these mechanisms, this study provides insights into potential therapeutic targets for mitigating endothelial impairment and preventing associated cardiovascular complications.

Keywords: Endothelial impairment; Renin-angiotensin axis; Oxidised low-density lipoproteins; Insulin resistance; Dyslipidaemia; Hyperglycaemia; Pro-in ammatory cytokines; Adhesion molecules

Introduction

Endothelial dysfunction is a pivotal event in the pathogenesis of numerous cardiovascular diseases, serving as a precursor atherosclerosis, hypertension, and thrombosis. e endothelium, a monolayer of cells lining the inner surface of blood vessels, plays a crucial role in regulating vascular homeostasis through its involvement in vascular tone modulation, in ammation, and thrombosis. Perturbations in endothelial function lead to impaired vasodilation, increased vascular permeability, and a pro-thrombotic milieu, ultimately contributing to the development and progression of cardiovascular pathology [1].

Several factors have been implicated in the initiation and progression of endothelial dysfunction. Among these, the renin-angiotensin axis, oxidised low-density lipoproteins (LDL), insulin resistance, dyslipidaemia, and hyperglycaemia have emerged as key players in endothelial impairment. e renin-angiotensin system, traditionally known for its role in blood pressure regulation, also exerts profound e ects on endothelial function through modulation of vascular tone and in ammation. Oxidised LDL, a hallmark of dyslipidaemia, promotes endothelial dysfunction by inducing oxidative stress and in ammation within the vascular wall [2].

Insulin resistance, a central feature of metabolic syndrome, is closely linked to endothelial dysfunction through various mechanisms, including impaired nitric oxide bioavailability and increased oxidative stress. Dyslipidaemia, characterized by elevated levels of triglycerides and LDL cholesterol, contributes to endothelial dysfunction by promoting vascular in ammation and impairing endothelial repair mechanisms. Hyperglycaemia, a hallmark of diabetes mellitus, exacerbates endothelial dysfunction through multiple pathways, including increased production of advanced glycation end products and activation of protein kinase C [3].

Moreover, endothelial dysfunction is perpetuated by the upregulation of pro-in ammatory cytokines and adhesion molecules, which promote leukocyte adhesion and migration into the vascular wall, further exacerbating in ammation and impairing vasodilation. Additionally, autoimmunity has been implicated in the pathogenesis of endothelial dysfunction, as immune-mediated mechanisms can lead to endothelial injury and dysfunction. Understanding the intricate interplay between these factors is essential for elucidating the pathophysiology of endothelial dysfunction and identifying potential therapeutic targets for its prevention and treatment. In this research article, we aim to comprehensively review the role of the renin-angiotensin axis, oxidised LDL, insunivteeom tw -a, wesfulipatiem

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