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Commentary

Diabetes Mellitus and Ischemic Heart Disease: Understanding the Mechanisms at Work

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DESCRIPTION

Ischemic Heart Disease (IHD) remains a leading cause of morbidity and mortality worldwide, with diabetes mellitus significantly amplifying the risk and severity of this condition. Diabetes mellitus, characterized by chronic hyperglycemia, insulin resistance, and impaired insulin secretion, is intricately linked with the development and progression of cardiovascular diseases, particularly ischemic heart disease. Understanding the mechanisms underlying this association is crucial for effective management and prevention strategies.

Endothelial dysfunction

Endothelial dysfunction plays a pivotal role in the pathogenesis of both diabetes mellitus and ischemic heart disease. In diabetes, chronic hyperglycemia leads to the production of Reactive Oxygen Species (ROS), which damage endothelial cells and impair Nitric Oxide (NO) bioavailability, crucial for vascular tone regulation. This dysfunction results in vasoconstriction, inflammation, and increased permeability, predisposing the arteries to atherosclerosis—a hallmark of ischemic heart disease.

Atherosclerosis

Atherosclerosis, the underlying pathology of most cases of ischemic heart disease, is accelerated in individuals with diabetes mellitus. Hyperglycemia promotes lipid oxidation, enhances Low-Density Lipoprotein (LDL) cholesterol uptake by macrophages, and triggers pro-inflammatory responses, contributing to the formation of atherosclerotic plaques. Moreover, diabetes exacerbates atherogenic dyslipidemia, characterized by elevated triglycerides, reduced High-Density Lipoprotein (HDL) cholesterol, and small, dense LDL particles, further promoting plaque formation and instability.

Platelet dysfunction and thrombosis

Diabetes mellitus induces platelet hyperactivity and impaired thrombus resolution, fostering a pro-thrombotic state. Elevated levels of circulating coagulation factors and impaired fibrinolysis in diabetic individuals increase the risk of thrombus formation, leading to coronary artery occlusion and subsequent myocardial ischemia. Additionally, diabetes-related alterations in platelet membrane glycoproteins and signaling pathways contribute to enhanced platelet aggregation and adhesion, exacerbating the risk of acute coronary events.

Coronary microvascular dysfunction

Beyond macrovascular complications, diabetes mellitus also impairs coronary microvascular function, contributing to ischemic heart disease development. Insulin resistance, hyperglycemia-induced oxidative stress, and inflammation disrupt endothelial and smooth muscle cell function in the coronary microcirculation, leading to microvascular dysfunction. This impairment compromises myocardial perfusion, exacerbating ischemia and increasing the risk of adverse cardiovascular events.

Mitochondrial dysfunction and oxidative stress

Mitochondrial dysfunction and oxidative stress are key pathophysiological mechanisms linking diabetes mellitus to ischemic heart disease. Hyperglycemia-induced mitochondrial ROS production overwhelms antioxidant defenses, leading to cellular damage and apoptosis in cardiac myocytes. Moreover, impaired mitochondrial biogenesis and function compromise myocardial energetics, exacerbating ischemic injury and cardiac dysfunction in diabetic individuals.

Inflammatory pathways

Chronic low-grade inflammation is a common denominator in both diabetes mellitus and ischemic heart disease. Diabetes-driven activation of inflammatory pathways, such as Nuclear Factor-kappa B (NF-κB) and Toll-Like Receptors (TLRs), promotes atherosclerosis, plaque instability, and myocardial injury. Furthermore, adipose tissue dysfunction in obesity-related diabetes contributes to the release of pro-inflammatory adipokines and cytokines, exacerbating systemic inflammation and cardiovascular risk.

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Autonomic dysfunction

Diabetes mellitus disrupts autonomic nervous system regulation, predisposing individuals to Cardiac Autonomic Neuropathy (CAN). CAN impairs heart rate variability, baroreflex sensitivity, and sympathetic-parasympathetic balance, increasing the risk of arrhythmias, silent myocardial ischemia, and sudden cardiac death-a common manifestation of ischemic heart disease in diabetic patients.

In conclusion, the development of ischemic heart disease in diabetes mellitus stems from a complex interplay of endothelial dysfunction, accelerated atherosclerosis, platelet dysfunction, coronary microvascular impairment, mitochondrial dysfunction, inflammation, and autonomic dysfunction. Targeting these interconnected pathways through comprehensive risk factor modification, glycemic control, lifestyle interventions, and pharmacotherapy is paramount for mitigating the burden of ischemic heart disease in diabetic individuals and improving cardiovascular outcomes.