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THE ROLE OF OXIDATIVE STRESS IN THE DEVELOPMENT AND PROGRESSION OF TYPE 2 DIABETES MELLITUS

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Abstract

Oxidative stress is a key pathogenetic factor in the development and progression of type 2 diabetes mellitus. Excessive production of reactive oxygen species contributes to insulin resistance, pancreatic beta-cell dysfunction, endothelial injury, and chronic diabetic complications. The aim of this study was to analyze the role of oxidative stress in type 2 diabetes and evaluate its clinical significance. The findings indicate that oxidative imbalance interacts with inflammation, hyperglycemia, dyslipidemia, and mitochondrial dysfunction. Understanding these mechanisms may support preventive and therapeutic strategies aimed at reducing diabetic complications.

Keywords:
type 2 diabetes mellitus, oxidative stress, insulin resistance, inflammation, metabolic dysfunction

Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by insulin resistance and impaired insulin secretion. Its prevalence continues to increase worldwide due to obesity, sedentary lifestyle, and aging populations. Chronic hyperglycemia leads to vascular, renal, neurological, and ocular complications. Oxidative stress is one of the central mechanisms linking metabolic dysfunction with tissue injury. Reactive oxygen species damage proteins, lipids, DNA, and cellular organelles, impairing insulin signaling and vascular function.

This topic is relevant because modern medical practice requires evidence-based analysis, early diagnosis, and interdisciplinary approaches. A deeper understanding of pathogenetic and clinical mechanisms allows healthcare professionals to improve prevention, monitoring, and treatment outcomes.

Materials and Methods

An analytical review of scientific literature on oxidative stress and type 2 diabetes mellitus was performed. Studies on reactive oxygen species, mitochondrial dysfunction, beta-cell injury, insulin resistance, inflammation, and diabetic complications were analyzed. The clinical significance of antioxidant defense systems and metabolic control was evaluated.

Results

The analysis showed that hyperglycemia increases production of reactive oxygen species through mitochondrial pathways, advanced glycation end-products, and activation of inflammatory signaling. Oxidative stress impairs pancreatic beta-cell function and worsens insulin resistance. It also contributes to endothelial dysfunction, nephropathy, neuropathy, and retinopathy. Patients with poor glycemic control demonstrate higher oxidative activity and reduced antioxidant capacity.

Discussion

Oxidative stress is both a consequence and amplifier of metabolic dysfunction in diabetes. Lifestyle interventions, adequate glycemic control, weight reduction, and management of dyslipidemia

may reduce oxidative burden. Although antioxidant therapy remains under investigation, prevention of oxidative damage through comprehensive metabolic management is clinically important. Future research should focus on biomarkers of oxidative stress and targeted therapies.

The reviewed evidence indicates that practical implementation depends on clinical context, patient characteristics, available resources, and professional competence. Therefore, future studies should include larger cohorts, standardized protocols, and long-term follow-up to improve reliability of conclusions.

Conclusion

Oxidative stress plays a crucial role in the pathogenesis and progression of type 2 diabetes mellitus. It contributes to insulin resistance, beta-cell dysfunction, and chronic complications. Early control of metabolic risk factors and prevention of oxidative injury may improve outcomes in diabetic patients.

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