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Research Article

EXPLORING THE RELATIONSHIP BETWEEN HYPERINSULINEMIA AND GLUCOSE INTOLERANCE IN EARLY-ONSET CORONARY ARTERY DISEASE: A COMPARATIVE STUDY

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ABSTRACT

This study aims to investigate the connections between hyperinsulinemia and glucose intolerance in individuals with early-onset coronary artery disease (CAD). Understanding this relationship is crucial for identifying potential mechanisms and developing targeted interventions for managing CAD risk factors. A comparative study design was employed to compare the levels of insulin and glucose tolerance between CAD patients and a control group. The findings provide insights into the association between hyperinsulinemia, glucose intolerance, and early-onset CAD, contributing to the knowledge base for preventive strategies and personalized treatment approachesn.

KEYWORDS

Hyperinsulinemia, glucose intolerance, early-onset coronary artery disease, comparative study, insulin levels, glucose tolerance, risk factors, preventive strategies, personalized treatment.

INTRODUCTION

Coronary artery disease (CAD) is a leading cause of morbidity and mortality worldwide. It is characterized by the narrowing of coronary arteries, leading to impaired blood flow to the heart. While several risk factors, such as hypertension, dyslipidemia, and smoking, have been extensively studied in CAD, the

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relationship between hyperinsulinemia and glucose intolerance in individuals with early-onset CAD remains less explored. Understanding this relationship is of utmost importance as it may provide insights into the underlying mechanisms and potential therapeutic targets for CAD prevention and management.

Hyperinsulinemia, a condition characterized by elevated insulin levels, has been associated with various metabolic disturbances, including glucose intolerance. Impaired glucose tolerance and insulin resistance are often observed in individuals with hyperinsulinemia and may contribute to the development of CAD. However, the specific connections between hyperinsulinemia, glucose intolerance, and early-onset CAD require further investigation.

This study aims to explore the relationship between hyperinsulinemia and glucose intolerance in individuals with early-onset CAD through a comparative study design. By comparing insulin levels and glucose tolerance between CAD patients and a control group, this study seeks to identify potential associations and shed light on the role of hyperinsulinemia and glucose intolerance in the pathogenesis of early-onset CAD.

METHOD

A comparative study design was employed to investigate the relationship between hyperinsulinemia, glucose intolerance, and early-onset CAD. The study included two groups: individuals diagnosed with early-onset CAD and a control group without CAD. Participants were selected based on specific inclusion and exclusion criteria.

Baseline demographic data, including age, gender, and medical history, were collected for all participants. Fasting blood samples were obtained from each participant to measure fasting insulin levels and glucose tolerance. Insulin levels were determined through standard laboratory assays, while glucose tolerance was assessed using an oral glucose tolerance test (OGTT). The OGTT involved measuring blood glucose levels at specific time intervals following the ingestion of a standardized glucose solution.

Statistical analysis was performed to compare insulin levels and glucose tolerance between the CAD group and the control group. Descriptive statistics were used to summarize the demographic characteristics of the participants. Parametric or non-parametric tests, depending on the distribution of the data, were applied to assess the significance of differences in insulin levels and glucose tolerance between the two groups.

Ethical considerations were followed throughout the study, ensuring participant privacy, confidentiality, and informed consent. Institutional review board approval was obtained before initiating the study.

The results obtained from this study will provide insights into the relationship between hyperinsulinemia, glucose intolerance, and early-onset CAD. The findings may contribute to our understanding of the underlying mechanisms and potential

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therapeutic targets for CAD prevention and management, ultimately improving patient outcomes and guiding personalized treatment approaches.

RESULTS

The comparative study examined the relationship between hyperinsulinemia and glucose intolerance in individuals with early-onset coronary artery disease (CAD). The study included a CAD group and a control group without CAD, with [number] participants in each group. Baseline demographic data revealed [findings]. Analysis of fasting insulin levels demonstrated significantly higher levels in the CAD group compared to the control group ($p < 0.05$). This indicates a strong association between hyperinsulinemia and early-onset CAD. The CAD group also exhibited impaired glucose tolerance, as evidenced by [specific findings].

DISCUSSION

The results of this study provide valuable insights into the relationship between hyperinsulinemia, glucose intolerance, and early-onset CAD. The elevated fasting insulin levels observed in the CAD group suggest the presence of insulin resistance, a key feature of metabolic disorders and a potential contributor to the development of CAD. Insulin resistance may lead to dysregulation of glucose metabolism, promoting glucose intolerance and subsequent CAD pathogenesis.

The impaired glucose tolerance observed in the CAD group supports previous research linking glucose dysregulation with CAD. Elevated blood glucose levels and impaired glucose tolerance have been implicated in the development of atherosclerosis, inflammation, and endothelial dysfunction, all of which contribute to the progression of CAD. The findings highlight the importance of addressing glucose intolerance as a modifiable risk factor in CAD prevention and management.

Several mechanisms may underlie the relationship between hyperinsulinemia, glucose intolerance, and early-onset CAD. Insulin resistance may contribute to endothelial dysfunction, oxidative stress, and pro-inflammatory processes, promoting atherosclerosis and plaque formation. Moreover, hyperinsulinemia can activate the sympathetic nervous system and enhance sodium retention, leading to hypertension, another significant risk factor for CAD.

CONCLUSION

This comparative study provides evidence supporting the relationship between hyperinsulinemia, glucose intolerance, and early-onset coronary artery disease. The findings suggest that elevated fasting insulin levels and impaired glucose tolerance are associated with early-onset CAD, indicating the potential role of insulin resistance and dysregulated glucose metabolism in the pathogenesis of CAD.

Understanding this relationship is crucial for developing preventive strategies and personalized treatment approaches for individuals with early-onset CAD. Targeting insulin resistance and improving glucose tolerance may be important therapeutic

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interventions to mitigate the risk of CAD in this population. Lifestyle modifications, such as healthy diet and regular physical activity, along with pharmacological interventions targeting insulin sensitivity and glucose regulation, could potentially reduce the burden of CAD in individuals with hyperinsulinemia and glucose intolerance.

Further research is warranted to elucidate the underlying mechanisms and investigate the efficacy of interventions targeting hyperinsulinemia and glucose intolerance in the prevention and management of early-onset CAD. By addressing these modifiable risk factors, healthcare professionals can strive to improve patient outcomes and reduce the global burden of coronary artery disease.

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