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Cardiovascular Disease and Premature Diabetes: Prevention and Risk Reduction Interventions based on Pathophysiology

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Abstract

Prediabetes is caused by pathophysiologic defects like insulin resistance, beta-cell dysfunction, and increased lipolysis, as well as an inadequate incretin effect and inflammation. Recent studies have shown that some people with prediabetes experience the long-term complications of diabetes; these confusions incorporate micro vascular and macro vascular messes. Last but not least, we discuss the implications of randomized control trials on the reduction of macro vascular risk and provide an overview of those studies that aim to stop the progression from prediabetes to diabetes.

Keywords: Impaired fasting glucose; Impaired glucose tolerance; Prediabetes complications; Cardiovascular Disease; Macro vascular

Introduction

According to the World Health Organization (WHO), one in ten adults worldwide had type 2 diabetes mellitus, which is one of the main causes of premature morbidity and mortality. Diabetes-related healthcare accounts for one out of every five dollars spent on health care in the United States [1]. Additionally, macro vascular complications account for the majority of the direct and indirect costs associated with diabetes in developing nations, accounting for at least a third of their total expenditures on diabetes-related health care. An interlude of prediabetes, itself a toxic state that is linked to the development of macro vascular complications, occurs during the T2DM progression [2].

Method

The causes and effects of prediabetes

Impaired glucose tolerance (IGT) is defined by the American Diabetes Association as a 2-hour plasma glucose value in the 75-gram oral glucose tolerance test of A fasting plasma glucose level between 100 and 125 mg/dL is considered impaired fasting glucose (IFG).6 Finally, prediabetes can also be characterized by a haemoglobin A1c level between 5.7% and 6.4%. It is important to point out that the ADA criteria define normal glucose tolerance (NGT) as having a fasting glucose level of less than 100 mg/dl and a plasma glucose level of less than 140 mg/dl after two hours [3]. When diagnosing prediabetes using HbA1c, it is important to remember that HbA1c is not always reliable as an integrated measure of mean plasma glucose because of well-known conditions like anemia, chronic kidney disease, and other systemic illnesses and Hematological disorders. HbA1c levels should be used with caution when diagnosing prediabetes because of racial and ethnic differences in their relationship to blood glucose levels. Before beginning treatment, it is always a good idea to get a real blood glucose reading to confirm the diagnosis.8 The Centers for Disease Control and Prevention (CDC) estimated that one million adults in the United States had diabetes and 86 million had prediabetes. Prediabetes affects more than 400 million people worldwide, and by 2030, more than 470 million people will have the condition [4].

Lipolysis, Incretin, alpha cell, and inflammation in prediabetes

Increased lipolysis, decreased endogenous levels of glucagon-like peptide, and impaired postprandial suppression of glucagon secretion by the pancreatic alpha cells are additional prediabetes defects. Additionally, as noted in, prediabetes' toxic environment is exacerbated by the abnormal expression of proinflammatory cytokines. For example, low adiponectin levels have been exhibited to be prescient of movement from NGT to prediabetes, and from prediabetes to [5]. Emerging insights regarding the gut Microbiome and its association with cardio metabolic disorders like obesity, diabetes, dyslipidaemia, etc., have highlighted the recognized pathophysiological defects in T2DM, highlighting those that have also been described in prediabetes. Elevated levels of molecular markers like intercellular adhesion molecule and tumor necrosis factor- have also been reported. Pertain to prediabetes. As of late, an upset stomach macrobiotic communicated as stomach symbiosis has been related with the movement and upkeep of weight, T2DM, cardiovascular illness (CVD) and the metabolic disorder

Transition from normoglycemia to prediabetes

During a six-year follow-up period, the subject's progresses' longitudinal mean weight change occurred. During the ten years of follow-up in the Baltimore Longitudinal Study of Aging (BLSA), 6.2% of the initial NGT participants developed prediabetes, giving the BLSA an annualized rate of 6.2%. The primary outcome of incident prediabetes was tracked longitudinally in the Pathobiology of Prediabetes in a Biracial Cohort study, which included initially normoglycemia African American and European American offspring of T2DM parents. An annualized rate was calculated for participants who developed incident prediabetes over a mean of five years of follow-up [6]. Participants with incident prediabetes were older, more likely to be male, had a higher baseline BMI and fat mass, less physical activity, and had lower measures of insulin sensitivity and disposition index than nonprogressors. HDL

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and adiponectin levels in the blood as well as higher triglyceride levels.

Cardiovascular disease

In the EPIC-Norfolk study, a higher 10-year cardiovascular mortality rate was linked to an increase in within the normal range. The EPIC-Norfolk results are consistent with the Paris Prospective Study cohort data, which showed that IGT subjects had a doubling of CVD mortality compared to NGT subjects [7]. The majority of patients with prediabetes have features of insulin resistance (metabolic) syndrome, such as upper-body obesity, hypertriglyceridemia, decreased HDL cholesterol levels, and hypertension, among other things. This highlights the finding of increased mortality. In prediabetes patients, components of the metabolic syndrome can frequently be identified years before a diagnosis is made. These characteristics result in advanced atherosclerotic vascular changes, which are frequently preceded by impairment of endothelium-dependent vasodilation, dysfunction of vascular smooth muscle, and elevated arterial stiffness [8]. A recent cross-sectional study found a positive correlation between the prevalence of arterial stiffness and prediabetes, indicating that prediabetes control may prevent arterial stiffness with early intervention. According to the Multi-Ethnic Study of Atherosclerosis (MESA) study, the prevalence of unrecognized myocardial infarction is almost three times higher in people with prediabetes than in people with NGT. In a more recent randomized controlled trial involving 6522 people with prediabetes and coronary artery disease, it was discovered that although acarbose did not lower the risk of major adverse cardiovascular events, it did lower the incidence of diabetes.

Conclusion

Patients should be routinely tested for prediabetes by doctors and other healthcare professionals, and those who have the condition should be referred for intensive lifestyle counselling. Similar to the DPP and Kindred studies, the objective is to lose more than 5% of body weight and keep it off through calorie restriction and increased physical activity [9]. In order to increase the availability of structured lifestyle programs that are based on evidence, healthcare providers ought to make an effort to establish strong ties within healthcare systems, communities, and payers. Based on HbA1c results, a recent survey of outpatients found that many had prediabetes. Amazingly, clinicians recognized and diagnosed prediabetes in those patients whose HbA1c tests showed it. Only 23% of those with prediabetes whose status was accurately documented in the clinical records had treatment documentation [10]. During the prediabetes phase, it is possible to intervene in the disease process. Patients, specialists, health systems, and primary care physicians all have a responsibility to prevent missed opportunities for prevention. Although it has long been known that diabetes poses significant risks to the cardiovascular system, it is now becoming clear that people with prediabetes are more at risk for CVD than those with diabetes. The impact on cardiovascular health is staggering when considering the millions of prediabetes individuals worldwide. However, education, increased awareness, care coordination, organization, and novel reimbursement mechanisms at multiple levels are necessary for identifying and intervening in the at-risk populations of prediabetes.

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