

Analysis of the relationship between heart and diabetes

Ruohui Xu

Medical College, Macau University of Science and Technology, Macau, China,
999078

xruohui@163.com

Abstract. The prevalence of diabetes in China and globally is increasing annually due to factors such as enhanced living conditions and the acceleration of the pace of life. One of the primary problems associated with diabetes is the occurrence of cardiac damage. Over the past few decades, the prevalence of diabetes has experienced a significant and rapid increase, leading to its classification as an epidemic. Cardiovascular disease, particularly ischemic heart disease, accounts for around 60% of mortality in individuals with diabetes. Type 2 diabetes has been found to have detrimental effects on both the anatomy and function of the heart. This study employs a literature analysis approach to examine the background of diabetes, the cardiovascular implications of diabetes, and the underlying mechanisms of cardiovascular illness in individuals with diabetes. This paper provides valuable insights into the examination of diabetes and its associated problems, with a particular focus on the investigation of cardiovascular disease in individuals with diabetes.

Keywords: Diabetes, Cardiovascular Disease, Type 2 Diabetes, Insulin Resistance.

1. Introduction

Diabetes is a prevalent endocrine and metabolic disorder, and the occurrence of cardiac problems in individuals with diabetes poses a significant risk to their overall well-being and survival. Recent studies conducted both domestically and internationally have provided evidence to support the assertion that individuals diagnosed with diabetes experience a notably higher prevalence of cardiovascular disease compared to those without diabetes. Approximately 80% of mortality among individuals with diabetes can be attributed to heart disease, with 70% of these cases specifically linked to coronary heart disease in those with diabetes. Hence, it is imperative to administer an appropriate and accurate combination medicine therapy for those afflicted with both diabetes and heart disease.

To date, numerous research have provided fundamental knowledge on the different types of diabetes-related heart disease. However, a comprehensive understanding of its complete etiology and contributing variables remains elusive. Consequently, while there is some comprehension of preventive measures, the development of focused interventions remains a challenge. The study employs a literature analysis research methodology. This scholarly article examines the correlation between diabetes and heart disease, providing a concise overview of the cardiovascular implications of diabetes. Additionally, it delves into the underlying mechanisms of this disease and explores the current therapeutic and preventive strategies.

The research conducted in this publication holds significant importance and has made valuable contributions to the field of diabetic complications, particularly in relation to heart disease. The text has presented a range of references regarding the effects of diabetes on cardiovascular health, including its underlying mechanisms. Additionally, it has explored various treatment modalities and preventive strategies, thereby serving as a valuable reminder for disease prevention. This review will commence by examining the coexistence of diabetes and cardiovascular disease, along with its underlying mechanisms. The objective is to develop more efficacious treatment approaches in order to proactively prevent its onset, safeguard the well-being and longevity of individuals with diabetes, and offer insights to clinicians for accurate diagnosis and treatment.

2. Background causes of diabetes

Diabetes is a collection of metabolic disorders that are distinguished by elevated levels of glucose in the bloodstream. Hyperglycemia arises due to deficiencies in the secretion of insulin, reduced physiologic effects of insulin, or a combination of these factors. Prolonged hyperglycemia in individuals with diabetes results in the chronic impairment and functioning of multiple tissues, particularly the eyes, kidneys, hearts, blood vessels, and nerves. The etiology and pathogenesis of diabetes are intricate and remain incompletely elucidated. According to conventional beliefs, there exists a correlation between the aforementioned factors. The primary determinants are hereditary variables and obesity-related factors, among others. To begin with, it is important to note that diabetes is a genetic disorder that can be passed down through generations. Genetic research has revealed a notable disparity in the prevalence of diabetes between individuals who share blood relations and those who do not. Specifically, the former group exhibits a rate that is five times higher than the latter. The etiology of type I diabetes is influenced by genetic factors to a significant extent, accounting for around 50% of its causation. Conversely, type II diabetes is predominantly influenced by genetic elements, with their contribution exceeding 90%. Consequently, the genetic factors implicated in the development of type II diabetes are considerably more substantial than those associated with type I diabetes. Furthermore, it is currently widely acknowledged that obesity plays a significant role in the development of diabetes. Approximately 60% to 80% of adult individuals diagnosed with diabetes exhibit obesity prior to the development of the condition. There exists a positive correlation between the severity of obesity and the prevalence of diabetes. The available empirical evidence indicates that there exists a correlation between advancing age, declining physical activity, and alterations in the muscle-to-fat ratio inside the human body. Between the ages of 25 and 75, there was a steady decline in muscle tissue, which accounted for 47% of body weight at the age of 25 and reduced to 36% at the age of 75. Concurrently, there was an increase in fat composition, which rose from 20% to 36% over the same time period. The primary factor contributing to the evident rise in diabetes prevalence among the senior population is the presence of obesity, particularly among individuals who are overweight or have excessive adiposity.

3. The relationship between diabetes and heart

3.1. Effect of diabetes on heart

While diabetes itself may not result in immediate mortality, its associated consequences significantly affect human health, particularly in relation to cardiovascular disorders. The historical background of diabetes is well acknowledged as a significant risk factor for the development of heart disease. One factor contributing to the heightened risk of heart disease in individuals with diabetes is the presence of comorbidities, including obesity, hypertension, and elevated levels of low-density lipoprotein (LDL) cholesterol. Hyperglycemia, a characteristic manifestation of diabetes, exerts deleterious effects on the integrity of blood vessel walls through many mechanisms. The potential consequence of their action is the impairment of artery wall tissue, hence facilitating the development of cholesterol plaques. Moreover, it is worth noting that these plaques have a higher susceptibility to rupture, which could potentially result in the occurrence of blockages and subsequent heart attacks. The presence of an excessive amount of sugar in the bloodstream can lead to the accumulation of cells, resulting in increased blood viscosity and

a higher propensity for the formation of blood clots. In conclusion, elevated blood glucose concentrations have the effect of reducing the production of nitric oxide, a biologically synthesized vasodilatory compound. In the event that blood vessels persist in a state of contraction rather than relaxation, it is conceivable that complications related to blood flow may arise.

3.2. Diabetes heart disease and its mechanism factors

Two types of cardiovascular disease in diabetes. The prevalence of cardiovascular disease in individuals with diabetes is notably high. There are two distinct types. Firstly, the topic of discussion pertains to the relationship between coronary artery disease and diabetes. Type 2 diabetes patients commonly experience coronary heart disease, which is a multifaceted condition characterized by the presence of widespread, calcified lesions that affect many blood vessels. The pathophysiology of coronary heart disease involves multiple interrelated processes, several of which are exacerbated by diabetes. Notably, hyperglycemia contributes to the occurrence of oxidative stress, glucose oxidation, and systemic inflammation. The synergistic impact of these factors results in the impairment of endothelial cells within the arterial wall and the facilitation of inflammation in the intima of the coronary artery. This phenomenon results in the accumulation of lipids and oxidized lipoproteins on the cellular membrane, subsequently triggering immunological responses mediated by macrophages and T cells. The outcome entails a relentless sequence of endometrial thickening, localized inflammation, and programmed cell death, resulting in impairment of endothelial function and the development of plaques rich in lipids. This process is characterized by the gradual occurrence of micro damage, along with abnormal platelet reactivity and fibrin deposition, in individuals with diabetes. Consequently, it can result in the progressive narrowing of the blood vessel lumen, platelet rupture, and the formation of blood clots, ultimately leading to severe obstruction of blood flow, such as in the case of myocardial infarction [1].

Furthermore, another condition that warrants attention is diabetic cardiomyopathy. The term "diabetes cardiomyopathy" (DCM) was initially introduced by Rubler in 1972 within the context of medical literature. Diabetes cardiomyopathy has consistently garnered significant attention from researchers due to its status as a prominent consequence of diabetes. Diabetes cardiomyopathy refers to the impairment of ventricular function in individuals with diabetes, regardless of the presence of established factors that often contribute to cardiac failure, such as coronary artery disease or hypertension. The disease progression encompasses a latent subclinical phase, characterized by cellular structural damage and anomalies that initially manifest as diastolic dysfunction, subsequently progressing to systolic dysfunction, and ultimately culminating in heart failure [2]. The genesis and progression of DCM are primarily influenced by several key variables, including left ventricular hypertrophy, metabolic abnormalities, alterations in extracellular matrix, small vessel disease, cardiac autonomic neuropathy, insulin resistance, oxidative stress, and apoptosis [3]. The primary etiological factor contributing to the development of DCM is hyperglycemia. This phenomenon results in elevated concentrations of unbound fatty acids and growth hormones, which subsequently disrupt the usual processes of matrix provision and use, calcium balance, and lipid processing. Furthermore, it induces heightened insulin secretion and the generation of reactive oxygen species, resulting in oxidative stress that triggers aberrant gene regulation, erroneous signal transmission, and death of cardiac cells. The induction of connective tissue growth factor, fibrogenesis, and advanced glycation end products has been shown to enhance the myocardial stiffness in individuals with diabetes. The presence of left ventricular diastolic dysfunction in DCM has the potential to progress to systolic dysfunction, resulting in a reduction in left ventricular ejection fraction (EF) [4]. Hence, the identification of left ventricular diastolic failure in individuals with diabetes holds substantial importance in terms of promptly diagnosing and treating diabetic cardiomyopathy (DCM), as well as preventing the onset of systolic dysfunction.

Two risk mechanisms of cardiovascular disease in diabetes. Subsequently, two risk pathways associated with the development of cardiovascular disease in individuals with diabetes can be identified. Insulin resistance (IR) is a prevalent occurrence among individuals diagnosed with type 2 diabetes.

Ischemic reperfusion (IR) results in the development of microvascular or macrovascular lesions, peripheral arterial dysfunction, restriction of blood flow, hypertension, and impairment of myocardial and endothelial cells. These effects contribute to an increased likelihood of experiencing coronary artery blockage, stroke, and heart failure [5]. This finding suggests a significant association between insulin resistance (IR) and cardiovascular disease (CVD). The potential association between these two pathophysiological states involves the modulation of insulin signaling proteins, including IR-B, IRS-1, PI3K, AKT, GLUT4, and PGC-1. This modulation disrupts insulin-mediated glucose uptake and other insulin-related processes in cardiac and endothelial cells. The pathogenesis of cardiovascular disease in individuals with diabetes involves several factors, including the downregulation of AMP-activated protein kinase (AMPK) and 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase (PFK-2), the upregulation of NADPH oxidase activity induced by M1 macrophages activated by adipose tissue, and the elevation of circulating angiotensin levels [6].

Impaired endothelial function represents the second aspect under consideration. Within the framework of insulin resistance, endothelial dysfunction has the potential to give rise to many cardiovascular ailments, such as hypertension, atherosclerosis, and coronary artery disease. Nitric oxide (NO) is often regarded as the most potent endogenous vasodilator in the human body. A key characteristic of endothelial dysfunction is the reduced availability of NO. Nitric oxide (NO) plays a crucial role in maintaining vascular wall homeostasis by facilitating platelet aggregation, inhibiting leukocyte adhesion, and exerting anti-inflammatory actions. Insulin resistance is characterized by a specific impairment in the synthesis of nitric oxide (NO), which may result in compensatory hyperinsulinemia. This hyperinsulinemia can activate the mitogen-activated protein kinase (MAPK) pathway, hence causing heightened vasoconstriction and triggering inflammatory reactions. Moreover, insulin resistance results in elevated levels of thrombin, pro-inflammatory markers, and reactive oxygen species (ROS), hence causing an upsurge in intracellular levels of adhesion molecule 1 (ICAM1) and vascular cell adhesion molecule 1 (VCAM-1). The correlation between endothelial function and insulin metabolism holds significant importance. The relationship between insulin resistance and endothelial signaling problems results in inflammation, which disrupts the equilibrium between endothelial vasodilators and vasoconstrictors, hence elevating the risk of cardiovascular complications [7].

4. Preventive measure

To mitigate cardiovascular issues associated with diabetes, it is imperative to implement a series of efficacious interventions. These interventions encompass enhancing one's lifestyle, managing blood glucose levels, regulating blood pressure, controlling cholesterol levels, and administering antithrombotic treatment. The fundamental approach to preventing or delaying the transition from diabetes to diabetes is through lifestyle intervention, which has the potential to mitigate the risk of both microvascular and macrovascular illnesses. More precisely, it pertains to modifying the nutritional composition (reducing salt, fat, saturated fat, and trans fatty acids, while increasing dietary fiber), engaging in consistent physical activity, achieving weight reduction, quitting smoking, and limiting alcohol consumption. The Daqing study conducted in China involved the random selection of 577 individuals with impaired glucose tolerance. These participants were divided into two groups: the control group and the lifestyle intervention group. The study spanned a period of 6 years, during which the intervention was implemented. Subsequently, a follow-up period of 14 years was conducted to assess the long-term effects of the intervention. The prevalence of diabetes in the intervention group shown a reduction of 43% in comparison to the control group. The intervention group exhibited a notable decline in cardiovascular mortality rate, while the statistical analysis did not yield a meaningful difference due to the limited number of cases [8].

Appropriate exercise can lead to a considerable reduction in the likelihood of cardiovascular problems among those diagnosed with type 2 diabetes. Hence, it is imperative for nursing personnel to establish a standardized approach towards patients' exercise behavior and exercise volume. This can be achieved by initially providing patients and their families with a comprehensive explanation of exercise treatment concepts, including the principles of individualization, moderation, and regularity. The notion

of exercise individualization entails prescribing exercise regimens that necessitate patients to independently complete the prescribed number of exercises. The notion of moderation entails patients engaging in cardiovascular exercises that are very efficient, such as jogging, cycling, stair climbing, swimming, and skipping rope, among others. Young and middle-aged individuals with diabetes may consider incorporating long-distance running into their exercise regimen. The principle of regularity emphasizes the importance of maintaining a consistent exercise routine without sporadic interruptions. Additionally, it is crucial to carefully and systematically schedule exercise sessions for diabetic patients. The optimal exercise timing for those with type 2 diabetes, which yields high exercise efficacy, is 30 minutes following breakfast and dinner. Assessing a patient's post-exercise heart rate provides a reliable means of determining the appropriateness of their exercise amount.

5. Conclusion

This study primarily examines the correlation between diabetes and cardiovascular and cerebrovascular disorders. This paper provides an overview of the background of diabetes, followed by an examination of two specific types of cardiovascular and cerebrovascular disorders associated with diabetes. Additionally, preventive treatments for these diseases are discussed. It is determined that while diabetes is not a lethal condition, its complications pose significant harm, with cardiovascular and cerebrovascular disorders exhibiting the highest incidence rate among the many complications. Furthermore, it is important to acknowledge that this study has certain limitations, including a lack of complete coverage and depth in its research. Subsequent modifications and investigations will be conducted in this domain in the forthcoming period. This article lacks a comprehensive examination of the cardiovascular implications of diabetes, instead providing simply a concise overview. In subsequent analysis, a comprehensive examination will be conducted on several specific dimensions and determinants of impact. The global prevalence of diabetes is steadily rising, leading to an escalating level of anxiety among individuals over the potential problems associated with this condition. Regarding the cardiovascular complications associated with diabetes, there remain some unresolved issues that need further exploration and development within this domain. Despite significant advancements in this subject in recent years, further efforts are still required to sustain progress.

References

- [1] Stratton, I.M., Alder, A.I., Neil, H.A., et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS35): Prospective observational study [J]. *BMJ*, 2000, 321, 405-412.
- [2] Giacco F, Brownlee M. Oxidative stress and diabetic complications [J]. *Circ Res*, 2010, 107(9):1058-70
- [3] Ceriello A. The emerging challenge in diabetes: the "metabolic memory" [J]. *Vasc Pharmacol*, 2012, 57(5-6):133-8.
- [4] Mooradian AD. Dyslipidemia in type 2 diabetes mellitus [J]. *Nat Clin Pract Endocrinol Metab*, 2009, 5(3):150-159
- [5] Ginsberg HN. Insulin resistance and cardiovascular disease [J]. *J Clin Invest*, 2000, 106(4):453-458.
- [6] Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis [J]. *Circulation*, 2002, 105(9):1135-43
- [7] Kershaw EE, Flier JS. Adipose tissue as an endocrine organ [J]. *The Journal of Clinical Endocrinology and Metabolism*, 2004, 89:2548e56
- [8] Milan G, Granzotto M, Scarda A, et al. Resistin and adiponectin expression in visceral fat of obese rats: effect of weight loss [J]. *Obesity Research*, 2002, 10:1095e103