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DIABETIC AND ITS PANCREATIC RELATIONSHIP – A MINI REVIEW

R. Senthil[1*]
[1]Department of Bioinformatics, School of Lifesciences, Vel's Institute of Science, Technology and Advanced studies, Pallavaram, Chennai - 600117, Tamil Nadu, India.

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ABSTRACT

In both the United States and Europe, diabetes is currently the major cause of end-stage renal disease (ESRD). People with diabetes live longer lives, type 2 diabetes is becoming more prevalent, and patients with diabetic ESRD are increasingly being admitted for treatment in ESRD programs that had previously rejected them, all of which contribute to this. Nephropathy affects 20–30% of people with type 1 or type 2 diabetes, but only a much smaller proportion of people with type 2 diabetes develop end-stage renal disease. As a result of the much higher prevalence of type 2 diabetes, these patients account for more than half of all diabetic patients who begin dialysis. Numerous therapies are effective, according to recent studies.

Corresponding Author:

Dr. R. Senthil,
Department of Bioinformatics, Vel's Institute of Science, Technology and Advanced studies, Pallavaram, Chennai - 600117, Tamil Nadu, India.
Email: renganathansenthil@gmail.com

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1. INTRODUCTION

Diabetes Mellitus is a set of metabolic illnesses defined by the presence of hyperglycemia for an extended length of time, as well as greater or lesser impairment in carbohydrate, lipid, and protein metabolism. Diabetes mellitus has several causes, but they always include either problems in pancreatic insulin production or cells in the body not reacting appropriately to insulin generated, or both at some time throughout the disease's progression [1]. Type 2 diabetes, often known as non-insulin dependent diabetes mellitus, affects the great majority of diabetics. Type 1 diabetes may be immune-mediated or idiopathic. Hyperglycemia, insulin resistance, and relative insulin insufficiency are the hallmarks of type 2 diabetes mellitus, the most prevalent form of the disease [2]. Between 382 million in 2013 and 422 million in 2016, the predicted worldwide prevalence of diabetes increased, and it is expected to reach around 522 million by 2030. Worldwide, 416 million people were found to have diabetes in 2015, with 90% of those people having type 2 diabetes mellitus. The literature indicates that the rates are almost equal for men and women. However, there is a higher prevalence of type 2 diabetes in populations where men have a higher incidence of diabetes. This could be because men have a different insulin sensitivity due to factors like obesity and localized fat deposition, in addition to other factors like hypertension, smoking, and alcohol intake [3–5].

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2. IMMUNE-MEDIATED DIABETES

Previously known as insulin-dependent diabetes or juvenile-onset diabetes, it is caused by cell-mediated autoimmune destruction of pancreatic -cells, frequently resulting in absolute insulin insufficiency [6]. One or more of the indicators of -cell immune destruction, such as islet cell auto antibodies, insulin auto antibodies, glutamic acid decarboxylase IA-2 and IA-2P, would be present in 85-90 percent of hyperglycemic people [7]. In addition to the DRB genes' influence, this disease has strong HLA ties, including DQA and DQB gene linkage. To what extent do these HLA DR/DQ alleles provide protection or susceptibility [8,9]. Although it may occur

at any age, the eighth and ninth decades of life are particularly vulnerable to immune-mediated diabetes because P-cell loss is rapid in children and babies but slow in adults [10–12]. Ketoacidosis is a frequent complication of this illness. Some types of diabetes have monogenetic abnormalities in -cell function. Hyperglycemia can develop at any age, even before the age of 25 [13,14]. They are inherited in an autosomal dominant manner and are known as maturity-onset diabetes of the young (MODY). They are distinguished by decreased insulin secretion with minimal or no abnormalities in insulin action [15,16]. Mutations in the hepatic transcription factor HNF-1 a, which are located on chromosome 12, are the most common cause of the most common variation. A second kind is brought about by defective glucokinase molecules induced by mutations in the glucokinase gene located on chromosome 7p. The metabolic process of glucose-6-phosphate, which is catalyzed by glucokinase, enhances -cell insulin synthesis [17]. The "glucose sensor" of the -cell is hence glucokinase. Glucokinase gene anomalies cause insulin secretion to be improperly triggered by lower plasma glucose levels. Less frequent variants include mutations in other transcription factors including NeuroDl, insulin promoter factor (IPF)-1, HNF-4a, and HNF-1 [18]. Some diabetes cases are caused by genetically established anomalies in insulin activity. The ranges from hyperinsulinemia through mild hyperglycemia to severe diabetes [1]. Acanthosis nigricans may be seen in affected people. Two childhood diseases characterized by elevated insulin resistance and altered insulin receptor activity include leprechaunism and the Rabson-Mendenhall syndrome, both of which are caused by mutations in the insulin receptor gene [1]. The post-receptor signal transduction pathways are mostly affected, not the insulin receptors' structure and function [1]. Having many hereditary diseases increases the risk of developing diabetes mellitus. Some instances of chromosomal anomalies are Down syndrome, Turner syndrome, and Klinefelter syndrome. Autosomal recessive Wolfram's syndrome is characterized by insulin insufficiency and the absence of -cells at autopsy [1,19].

3. DIABETES AND ITS COMORBIDITY

Type 1 diabetes mellitus symptoms appear quickly and can progress considerably over weeks or even days, especially in children and adolescents [20]. Excessive thirst (polydipsia), unusually high amounts of urine (polyuria), increased appetite (polyphagia), feeling fatigued all the time, loss of muscle mass, and unexplained weight loss are all frequent symptoms of type 1 diabetes [21]. The autoimmune death of cells results in a lack of insulin secretion, which leads to the metabolic derangements characteristic with T lDM [22]. In TlDM patients, there is also aberrant pancreatic a-cell activity and excessive glucagon secretion. Hyperglycemia does not reduce glucagon secretion 1 in type 2 diabetic individuals. Skeletal muscle and other peripheral tissues have impaired glucose metabolism due to insulin deficiency-induced uncontrolled lipolysis and elevated free fatty acid levels [23]. It is useful to compare the quantity of albumin in the sample to its creatinine concentration. This is known as the albumin/creatinine ratio (ACR). Because women excrete less creatinine than men, the normal ACR 1 is 2::3.5 mg/mmol for females and 2::2.5 mg/mmol for males. ACR is often stated as mg of albumin per gramme of creatinine or g of albumin per mg of creatinine, with a normal range of 30 to 300 g albumin/mg creatinine [24–26].

4. ANEMIA AND DIABETES

Anemia is a frequent complication of diabetes, particularly in individuals with albuminuria or impaired renal function [27]. The presence of renal interstitium injury, systemic inflammation, and autonomic neuropathy have all been postulated as factors in diabetic nephropathy anemia (DN) [28]. Dysfunction, like many other pathophysiological changes in DN, may be visible prior to measurable changes in the glomerular filtration rate (GFR). Creatinine [29].

Because the majority of diabetes' late sequelae (retinopathy, neuropathy, heart disease, and peripheral artery disease) involve ischemic tissue damage, therapy with human recombinant erythropoietin should be advantageous, although definitive proof for this theory is presently lacking [27]. According to the study, having anemia in addition to diabetes increases the risk of getting diabetic eye disease, developing heart disease, or having a stroke. People who have diabetes and anemia are more likely to die younger than those who only have diabetes [30]. Anemia, fortunately, is treatable, and advantages such as increased energy, activity level, and improved quality of life can be obtained [31].

Renal anemia may go unnoticed or untreated in this group. To make matters worse, no systematic research of the prevalence and determinants of anemia in diabetic patients has been conducted, especially in the absence of overt nephropathy (representing the majority of patients) [32]. Anemia occurs when the body's red blood cells are depleted, resulting in less oxygen being carried to the cells [33].

Anemia sufferers commonly experience fatigue or weakness and may struggle to accomplish daily tasks. Other symptoms include pallor, lack of appetite, dizziness, lightheadedness, a rapid pulse, and shortness of breath [34]. These symptoms are not usually

recognized as signs of anemia since they are also associated with diabetes. Diabetes can cause anemia because the kidneys produce the hormone erythropoietin, which regulates red blood cell formation (EPO) [35]. The blood level of erythropoietin hormone (EPO) should be between 2.6 and 18.5 mIU/mL. Diabetes damages the kidneys on several levels, and one disease typically leads to another. Diabetes-related kidney changes range from diabetic nephropathy to chronic kidney disease [36].

Early detection and therapy are crucial for preventing or delaying disease development. Diabetic nephropathy is usually associated with anemia [37]. It has only recently been recognised that anemia in diabetic patients occurs not only in individuals with advanced renal failure, but also frequently in those with relatively normal renal function [38]. Anemia is more prevalent and severe in diabetics than in nondiabetics at all glomerular filtration rates (GFR) [39]. An abnormal erythropoietin response to anemia is a major cause of anemia. Iatrogenic causes such as iron deficiency ACE inhibitor usage are also factors [35]. When blood creatinine 1 remains normal, erythropoietin concentration predicts faster loss of renal function [40,41].

5. CONCLUSION

The rate of advancement is expected as haemoglobin levels rise. It is now being examined if correcting anemia delays development. Because ischemic tissue damage is involved in the bulk of diabetes' late sequelae (retinopathy, neuropathy, heart disease, and peripheral artery disease), therapy with human recombinant erythropoietin should be beneficial, however convincing confirmation for this notion is still missing. Research shows that the risk of developing diabetic eye disease, cardiovascular disease, and stroke is higher in those with both diabetes and anemia. A shorter life expectancy is associated with diabetes and anemia compared to diabetes alone. There is hope: anemia can be cured, and with it comes the possibility of gains like more energy, more exercise, and an overall better quality of life.

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COMPETING INTEREST

The authors declare no conflict of interest.

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