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CORRELATIVE STUDY OF INSULIN RESISTANCE AND PANCREATIC BETA CELL FUNCTION IN DIABETICS AND NON-DIABETICS

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ABSTRACT

Type 2 Diabetes Mellitus is a metabolic condition characterized by elevated blood sugar levels (hyperglycemia) and disruptions in the metabolism of carbohydrates, fats, and proteins. The etiology of diabetes mellitus can exhibit considerable variability. However, it consistently involves deficiencies in pancreatic insulin secretion or inadequate responsiveness of bodily cells to endogenous insulin, or both, at some stage during the progression of the disease. The study featured a total of 200 individuals who were randomly assigned to four distinct groups. The study evaluated the results of many measurements, including fasting plasma glucose, postprandial plasma glucose, glycated hemoglobin, fasting plasma insulin, serum c-peptide, HOMA-IR, and HOMA-B, among four distinct groups. A significant correlation was seen among the groups for all the metrics, with a threshold of significance of $p<0.05\%$. Offspring without diabetes with a family history of type 2 diabetes were observed to have hyperinsulinemia, elevated serum c-peptide levels, moderate insulin resistance, and enhanced pancreatic beta cell function compared to individuals without a family history of diabetes.

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1. INTRODUCTION

Diabetes mellitus (DM), sometimes known as diabetes, is a collection of metabolic disorders affecting carbohydrate metabolism [1]. It is characterized by elevated blood sugar levels that persist over an extended duration, resulting from impairments in insulin secretion, insulin action, or both [2]. The initial indications of diabetes mellitus encompass polydipsia, polyphagia, polyuria, weight loss, and hazy eyesight [3]. The untreated state of diabetes can lead to severe and even life-threatening outcomes, such as hyperglycemia accompanied by ketoacidosis or non-ketotic hyperosmolar coma [4]. Diabetes is associated with various chronic complications affecting micro and macrovascular systems [5]. These complications include retinopathy, which can result in vision impairment; nephropathy, leading to renal failure; peripheral neuropathy, increasing the likelihood of foot ulcers, amputations, and Charcot's joints; as well as autonomic neuropathy, which manifests as gastrointestinal, genitourinary, cardiovascular symptoms, and sexual dysfunction [6]. Chronic conditions may be accompanied by growth impairment and increased susceptibility to specific infections. Research conducted on individuals with a heightened susceptibility to acquiring diabetes would contribute to the identification of the fundamental etiology of the illness and facilitate the establishment of innovative primary intervention initiatives designed to impede or postpone the onset of diabetes [7]. The hemoglobin A1c level is 6.5% (48 mmol/mol). The experiment should be conducted inside a controlled laboratory environment, employing a methodology that has obtained certification from the

National Glycohemoglobin Standardisation Programme (NGSP) and has been standardized to the Diabetes Control and Complications Trial (DCCT) assay. In a patient presenting with typical signs of hyperglycemia or hyperglycemic crisis, a random plasma glucose level of 200 mg/dl (11.1 mmol/L) was observed [8].

2. MATERIALS & METHODS

The blood samples were obtained from participants using venepuncture, aseptic precautions, and appropriate vacutainers. The blood glucose was measured using a fluoride tube, while the insulin and c-peptide levels were assessed using a plain tube. Additionally, an EDTA sample was utilized to determine the HbAlC levels. Both fasting samples (collected after a 12-hour overnight fasting period) and postprandial samples were obtained. Blood samples were collected from healthy persons and patients diagnosed with type 2 diabetes mellitus. These samples were used to determine serum fasting and postprandial glucose levels using the glucose oxidase/peroxidase (GOD/POD) method. Additionally, the levels of HbAlc were estimated using immunoturbidimetry, fasting plasma insulin was measured using chemiluminescent immunoassay (CLIA), and serum C-peptide was quantified using enzyme-linked immunosorbent assay (ELISA) methods. The basal insulin secretion and sensitivity indices were assessed using the Homeostatic Model Assessment (HOMA) method, which involves the following calculations: HOMA-IR = FPI X FPG/22.5 and HOMA-B = 20 X FPI/(FPG - 3.5). FPI represents the fasting plasma insulin level measured in µIU/ml, while FPG denotes the fasting plasma glucose levels measured in mmol/L. The blood samples underwent processing within 30 minutes. The measurement of blood glucose levels was promptly assessed within the time frame of 8 to 11.

3. RESULTS

The sample consisted of 100 individuals diagnosed with type 2 diabetes mellitus (T2D) and 100 persons without T2D who were in good health. The individuals were partitioned into four distinct groups. The age range of the study participants encompassed individuals between the ages of 20 and 60. The average age of the participants was 35±9.6 years. The average weight was 48±3.0 kg, and the average BMI was 22±0.5 kg/m2. The user's text does not contain any information to rewrite academically. The average fasting plasma glucose (FPG), postprandial plasma glucose (PPG), and glycated hemoglobin (HbA1c) levels exhibited greater values in group IV when compared to the remaining groups. The average fasting plasma insulin levels in group II were greater than those in group I, but group III exhibited higher fasting plasma insulin levels than group IV (Table 1). The average serum c-peptide level in group II was found to be higher than that in group I. However, in group III, it was shown to be higher compared to group IV. The Homeostatic Model Assessment of Insulin Resistance (HOMA IR) exhibited greater values in groups II and III compared to groups I and IV. The Homeostatic Model Assessment of Insulin Resistance (HOMA-B) exhibited greater values in groups II and III compared to groups I and IV. The presence of familial connections to type 2 diabetes mellitus (T2DM) increases the average fasting insulin level, c-peptide level, and insulin resistance. This progression aligns with the inherent course of the disease. The occurrence of insulin resistance and significant impairment of pancreatic beta cell function leads to unregulated diabetes.

Table 1: Characteristics of study participants

	Group I		**Group** II		**Group** III		**Group IV**	
N	**50**		**50**		**50**		**50**	
Parameters	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**	**Mean**	**SD**
Age (years)	35.4	9.6	32.8	7.9	35.4	9.6	32.8	7.9
Weight (kg)	48.7	3.0	49.3	2.7	48.8	3.2	50.2	2.8

Height (cm)	148	0.05	148	0.03	148	0.04	150	0.04
BMI (kg/m^2)	22.03	0.61	22.21	0.53	22.17	0.44	22.1	1.56
Systolic BP (mmHg)	116.2	19.9	117.6	9.8	118.4	9.9	119.2	9.9
Diastolic BP (mmHg)	76.2	6.4	77.8	7.1	76.9	7.2	77.4	6.0
FPG (mg/dl)	90.3	7.1	90.8	6.9	103.8	10.5	176.8	47.7
PPPG (mg/dl)	112.3	13.7	115.5	13.1	134.4	22.1	275.5	75.1
HbAlc (%)	5.4	0.4	5.4	0.4	6.2	0.3	9.0	1.3
Fasting plasma Insulin(μIU/ml)	14.2	5.2	21.6	10.0	26.8	14.7	17.0	6.3
C- peptide (ng/ml)	1.7	0.8	2.4	1.2	2.8	1.6	1.8	0.5
HOMA IR	3.2	1.3	4.9	2.5	6.9	4.0	7.6	3.4
HOMAB	195.8	74.6	283.5	116.2	245.1	143.6	61.5	29.5

Glycation refers to the non-enzymatic process through which glucose reacts with proteins, leading to structural modifications and alterations in their biological characteristics. This study demonstrates no significant difference in glycated hemoglobin levels between non-diabetic individuals with and without a family history of diabetes. In their respective studies, the authors have observed an elevation in HbA1C levels among the first-degree relatives of individuals with type 2 diabetes [9,10]. This increase has been attributed to lipid peroxidation processes and decreased glutathione, which leads to protein glycation. This factor is the most robust independent predictor of type 2 diabetes. Additionally, individuals with diabetes who have uncontrolled plasma glucose exhibit elevated levels of glycated hemoglobin, heightened insulin resistance, and diminished pancreatic beta cell function, resulting in decreased levels of plasma insulin and serum c-peptide compared to people with diabetes who maintain good blood glucose control. As mentioned earlier, the data indicate that insulin resistance is the predominant factor contributing to the development of Type 2 Diabetes Mellitus (T2DM). However, once diabetes mellitus is established, there is also a concurrent occurrence of pancreatic beta-cell dysfunction and insulin resistance [11]. As insulin resistance increases, there is a corresponding decline in the function of pancreatic beta cells. As mentioned earlier, the findings indicate that while insulin resistance is the fundamental anomaly in type 2 Diabetes Mellitus, the presence of pancreatic beta cell dysfunction is also required once the diabetes has developed, in addition to insulin resistance. As the duration of diabetes grows, there is a concomitant increase in insulin resistance and a decrease in pancreatic beta cell activity.

4. CONCLUSION

The study findings demonstrate a striking association between those without diabetes but with a familial predisposition to diabetes mellitus, indicating an increased susceptibility to developing the condition. Notable changes in insulin resistance and insulin release by pancreatic beta cells accompany this susceptibility. While fasting plasma glucose, postprandial plasma glucose, and glycated hemoglobin may not be highly suggestive of impaired glucose tolerance status, fasting insulin and insulin resistance levels demonstrate a trend towards altered glycemic status. This observed pattern suggests the underlying development of type 2 diabetes mellitus, characterized by the initial onset of insulin resistance and subsequent disruption of glycemic regulation. Additionally, this study demonstrates a pronounced presence of insulin resistance and impaired functionality of pancreatic beta cells in individuals with uncontrolled diabetes mellitus compared to those with well-regulated plasma glucose levels.

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ETHICAL APPROVAL

Nil

COMPETING INTEREST

The authors declare no conflict of interest.

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