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Impact of Diabetes Mellitus Type 2 in the Activity of Glucose-6-Phosphate Dehydrogenase in Human Erythrocyte

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Abstract - ***Diabetes mellitus is a metabolic affliction saunter that is characterized by a nobler than normal blood glucose poise. Glucose-6-phosphate dehydrogenase (G6PD) enzyme code (E.C.1.1.1.49) is an underlying enzyme in the phosphogluconate pathway. In this study, G6PD vitality in the mortal erythrocyte of male and female patients with type 2 diabetes mellitus was assessed utilizing a spectrophotometer at 340 nm. The activity of the enzyme increased with elevated glycated haemoglobin (HbA1C) levels. G6PD activity was found to be significantly associated with type 2 diabetes mellitus. The association between G6PD and diabetes mellitus was significant (P < 0.001). Moreover, G6PD was positively correlated with HbA1C levels (r = 0.572). The following mean ± standard deviation values were obtained: G6PD activity (IU/g Hb), 3.1103 ± 0.79349; HbA1C (%), 8.6600 ± 1.63120; Hb (g/dL), 13.4933 ± 1.38836; platelet count (103/µl), 283.4667 ± 58.59312; WBC (103/µl), 7.4890 ± 1.49842; HCT (%), 45.0100 ± 2.63430; and BS (mg/dL), 230.2667 ± 75.67760. The results showed that an elevated HbA1C up leads to increased G6PD performance in the human erythrocyte, which is concerning to glucose levels in the special (blood).***

Keywords: G6PD, diabetes mellitus, human erythrocyte.

1. INTRODUCTION

G6PD (E.C.1.1.1.49) is the primary enzyme go is denuded in monosaccharose phosphate pathway. This enzyme is found in the cytosol and mitochondria. Moreover, it converts glucose-6-phosphate accompanied by nicotinamide adenine dinucleotide phosphate $NADP^+$ into 6-phosphogluconolactone and NADPH, NADPH plays a bulky profession in the hexose monophosphate shunt. NADPH participates in the synthesis of biochemical molecules like fatty acids, lipids, and several amino acids. Furthermore, this cofactor plays a concerned in preventing cell damage [1,2,3]. Metabolic processes synthesis reactive oxygen species (ROS), which result in free radicals that combine with proteins, lipids, and several molecules. The harmful species are removed by antioxidants, such as reduced glutathione (GSH), which prevents cell damage [4,5]. The decrease in NADPH leads to a deficiency in reduced GSH in thriving cells, aide installs death [6]. The housekeeping enzymes are G6PD, GSH reductase, and 6-phosphogluconate dehydrogenase, which are found in animal tissues, plants, microorganisms, and blood cells [7]. The different techniques will be used, and they can be isolated from various sources, such as the kidneys and liver of rats [8,9,12], human erythrocytes [10], liver of rainbow trout [11], [12]. The kinetic properties and characteristics of the enzymes have been assessed. If there is an increased production of free radicals, the number of ROS also increases. The species (ROS) are an attraction with component of cells, such as proteins, lipids, and sugars. This link causes the accumulation of ROS, which leads to cell damage, pathogenic disorders, and diabetes [13,14,15]. The pentose phosphate pathway has two main functions: synthesis of RNA, DNA, and ribose-5-phosphate required for nucleotide synthesis in the cell and the synthesis of NADPH, which is a reducing power in reductive biosynthesis. In addition, phosphorylated carbohydrates, such as erythrose-4-phosphate, are required for aromatic amino acid and vitamin synthesis, and sedoheptulose-7-phosphate, which is a component of the bacterial cell wall, is also synthesized in the same manner [16,17,18]. NADPH plays a significant role in the reduction of fatty acid, biosynthesis of cholesterol nitric oxide, reduction of GSH, detoxification of the drug and xenobiotic, and reduction of peroxides in the cell [20]. Reduced GSH and GSH-dependent enzymes keep the cell away from internal and external toxic compounds and ROS [21]. When oxygen depletion and H_2O_2 formation in phagocytes are high, the activity of PPP also increases [6]. Xenobiotics are considered non-toxic when NADPH is used along with cytochrome P450 detoxification systems and GSH peroxidase. Among the important enzymes of the sorbitol pathway, aldose reductase also uses NADPH [19].

In addition, NADPH allows ribonucleotides to be converted into deoxyribonucleotides for DNA synthesis. NADPH is also required for several water-insoluble compounds [16,17,18]. Diabetes mellitus is characterized by raised aldohexose levels within the blood. There are 2 styles of polygenic disease Mellitus: kind one polygenic disease, that is understood as insulin-dependent polygenic disease, and kind two DM, that is understood as insulin-independent polygenic disease. To decrease the risk of polygenic disease, glucose level

within the blood should be controlled [22]. The sickness is treated by dynamical way habits, and patients should engage in physical activities. Moreover, they should have a nutritious diet and must take glucose-lowering medications [23].

Diabetes is a disease which described by Egyptian from 1500 BCE [24]. Chinese used diabetes for sweet urine, also Japanese and Korean used this term. Sweet urine and blood came from Matthew Dobson in 1776 [25]. In 1990, the diabetes pathogenesis have been understood [26]. Frederick Banting and Charlis Best have improved treatment by using insulin in 1921 and 1922, also discovered drug metformin for type 2 of diabetes. By Novo Nordidesk was improved insulin NPH [27]. The drug sulfonylurea was used since 1942. In 1950, the drug biguainides has been used for diabetes (type 2). Rosalyan and Solomon Berson gained a Nobel Prize for discovering the radioimmunoassay about insulin in1977 [28]. In 1990 discovered the thiazlidinedion which used as sensitivity to insulin. The new medicine discovered for diabetes (Gila monster) [29].

The deficiency of G6PD causes a deficiency of NADPH production, which leads to decreases of detoxification processes. If detoxification system, minimizing cause cell damage and several diseases which link directly with Parkinson's diseases, immune impairment in function syndrome, fibromyolgie and cancer [30,31]. In contrast, recent studies have shown that acute hyperglycaemia will increase G6PD and 6PGD within the heart muscle [32]. Especially, high aldohexose levels have an effect on the G6PD activity in human erythrocytes in men and women.

In recent years, studies have shown that diabetes mellitus DM is related to oxidative stress. Symptom stimulates the assembly of reactive element species [33]. Raised aldohexose levels within the blood cause modifications in NADPH metabolism within the tissues. Moreover, in PPP, there are changes in enzymes adore G6PD and 6PGD, that are essential for manufacturing NADPH. NADPH could be a reducing power in reactive synthesis [34]. Thus, this study aimed to evaluate the effect of type II DM on G6PD activity in human erythrocytes.

2. MATERIALS AND METHODS

One hundred Millimole/Liter Tris-buffer, pH 8.0, 10 Millimole/Liter MgCl2, 0.5 Millimole/Liter EDTA, 310 Millimole/Liter $NADP^+$ (R1), 0.6 Millimole/Liter glucose-6-phosphate (R2), 0.2 g/L digitonin (R3), and 0.9 g/mL NaCl. Data of the blood test results of thirty patients with kind two DM at the diabetic center of Laila Qasim in Erbil, Iraq were collected. Among the patients, twenty were women and ten were men. In total, three milliliters of blood were collected for every patient and placed in EDTA tubes. The G6PD kit was utilized in this study. A photometer (Apel Pd 303) was accustomed find the G6PD activity in patients with kind two polygenic diseases.

2.1. Experiment

The hemoprotein levels (g/dL) of the patients were determined, and 0.2 ml of homogenized blood was washed 3 times with two ml normal saline (0.9 g/dL). The sample was centrifuged at 1500 rpm between every washing, and therefore the supernatant was eliminated (preventing the elimination of erythrocytes). In the end, the erythrocytes were suspended in 0.9 ml of haemolysing solution (vial R3). When material possession it symbolizes quarter-hour at 2–8°C, the suspension was centrifuged once more. The supernatant (haemolysate) was used at intervals one hour.

2.2. Measurement of enzyme activity

The chemical agent 3ml (R1) was additional to fifty µL haemolysate. The sample was mixed and incubated for five minutes at 37°C. Then, 100 µL of R2 was additional, and G6PD activity was measured employing a photometer at 340 nm [35].
Calculate G6PD activity as follows:
IU/g Hb = (Abs/min x 5000) / Hb expressed in g/dL

2.3. Statistical analysis

One-way-ANOVA-using-spss-statistics.

3. RESULTS

In the laboratory, the G6PD activity within the human erythrocytes of patients with the polygenic disease was assessed. Table (1) showed the activity of G6PD in both male and female. The activity of G6PD per patient in male was greater than the activity of G6PD in females. Table (2) shows the mean ± SD of G6PD activity, glycated hemoprotein (HbA1C) level, Hb, platelet count, WBC, HCT, and blood sugar. High glycated hemoprotein (HbA1C) level was considerably related to G-6-PD enzyme activity within the human erythrocyte ($P < 0.001$) as seen in Table (3). Table (3) displays the correlation pair of data. G6PD activity raised, as shown in Figures (1) and a (2) of depicts the info that was collected from thirty patients. Figures 3 show the relation among the thirty patients in terms of blood sugar levels in patients with type 2 polygenic diseases. The normal range of G6PD is (5.0 – 14.0) IU/ g Hb [36].

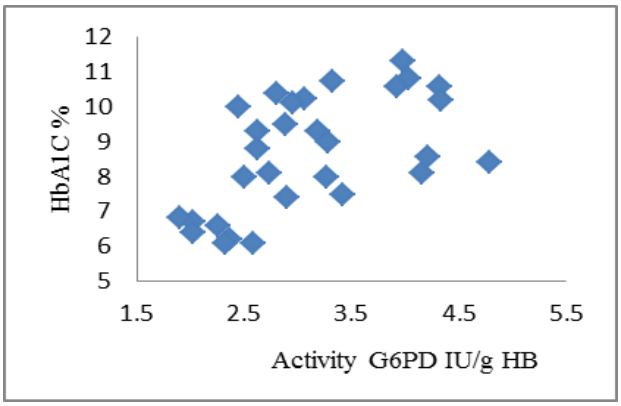

Figure 1: The relationship between HbA1C and the activity of G6PD in human erythrocyte.

Table 1: Total activity G6PD in male and female among type 2 diabetic patients

Parameter	No. of Samples	Total activity	Activity per patient
Total activity G6PD in males	10	31.25	3.12
Total activity G6PD in females	20	59.91	2.99

Table 2: The blood parameters and G6PD activity among type 2 diabetic patients

Parameter	Mean ± SD
G6PD activity IU/g HB	3.1103 ± 0.79349
HbA1C %	8.6600 ± 1.63120
HB (g/dl)	13.4933 ± 1.38836
Platelet (103/µl)	283.466 ± 58.59312
WBC (103/µl)	7.4890 ± 1.49842
HCT %	45.0100 ± 2.63430
B.S mg /dl	230.2667 ± 75.6776

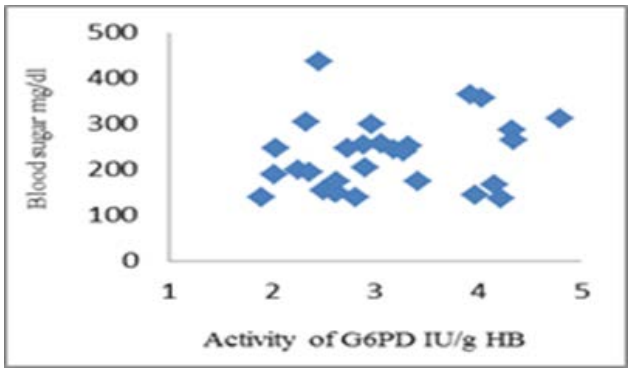

Figure 2: The relationship between the activity of G6PD in human erythrocyte and blood sugar.

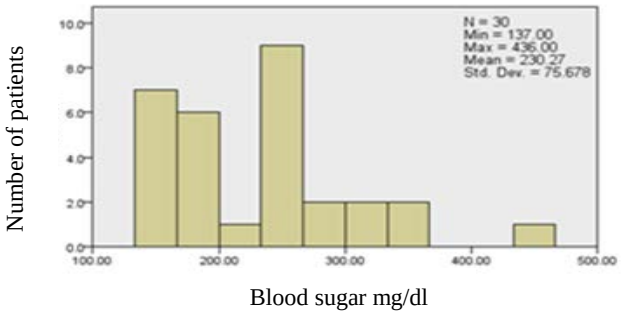

Figure 3: The blood sugar of diabetic patients for thirty samples, minimum values, maximum value, mean and standard deviation

Table 3: Pair sample correlations of activity G6PD, BS, Hb, PLT, WBC and HCT.

	N	Correlation	P value Significant
ActivityG6PD & HbA1C	30	0.572	0.001
Activity G6PD & HB	30	-0.029	0.878
Activity G6PD & PLT	30	0.345	0.062
Activity G6PD & WBC	30	0.151	0.425
Activity G6PD & HCT	30	0.110	0.562
Activity G6PD & BS	30	0.221	0.241
BS & Activity G6PD	30	0.221	0.241
BS & HbA1C	30	0.362	0.050
BS & HB	30	-0.033	0.861
BS & PLT	30	-0.216	0.251
BS & WBC	30	-0.283	0.130
BS & HCT	30	0.184	0.331
HbA1C & Activity G6PD	30	0.572	0.001
HbA1C & HB	30	0.074	0.696
HbA1C & PLT	30	0.366	0.046
HbA1C & WBC	30	0.151	0.427
HbA1C & HCT	30	0.364	0.048
HbA1C & BS	30	0.362	0.050

4. DISCUSSION

Glucose-6-phosphate dehydrogenase (E.C.1.1.1.49) is the primary enzyme that is essential in PPP, and it produces 1mol of NADPH. NADPH is important for the removal of ROS and prevention of cell damage [37,38]. ROS is stimulated by high glucose levels, which damages the nucleus by linking with DNA and RNA. By controlling the glucose level in the blood, the number of ROS in cells is decreased [39]. High glucose level in the blood leads to hyperglycaemia, which results in micro- and macrovascular disorders [40]. High glucose level enhances the superoxide anion levels, which leads to increased G6PD, 6PGD, and NADPH oxidase activities [41]. These effects may be avoided by the treatment or control of glucose levels among patients with diabetes and HbA1C. In hyperglycaemia, a large amount of glucose pyruvate is suitable for the TCA cycle to be oxidized. The electron transfer chain increases the electron granter of NADH and $FADH_2$. Hyperglycaemia leads to enhanced voltage inclination in the mitochondria membrane, which is important for preventing the transfer of electron in the complex (III) to return into coenzyme Q, and this leads to an increase in superoxide [42]. This study first evaluated the G6PD activity using kits for G6PD and also assessed the correlation between G6PD activity and HbA1C in both men and women. The activity increased with the increase in HbA1C. Therefore, the glucose level in the blood must be controlled. The inhibition or deficiency in the G6PD among patients with diabetes causes several health problems. In my studies the enzyme activity of G6PD is increasing. G6PD activity is significantly associated with HbA1C based on other tests ($p < 0.001$). In diabetes patients sort (II) the G6PD activity is lower value than the normal range but the value of the activity is

becoming near the normal range with increasing HbA1C value. Several studies have shown that there is increased G6PD activity in the human erythrocyte of patients with type 2 DM. This result is not in accordance with that of previous studies by Gök et al. 2016, and Peiró et al. 2016 [43,44], which explain that the high aldohexose levels enhance the enzyme level in PPP but in different sources. In the present study of the relationship between diabetic diseases and G6PD activity, the result contrasts with that of other previous studies, such as those conducted by Khanam et al. 2018 [45], which indicated the G6PD activity decreased in men with diabetes. The study of Khanam focused only on male patients. However, in the present study, both men and women were assessed. G6PD activity increased in male patients with kind two diabetes disease. Furthermore, G6PD is a key enzyme of PPP. Therefore, it balances the redox processes [46].

5. CONCLUSION

The second type of diabetes disease is influenced by the activity of G6PD enzyme in human erythrocyte. In addition, the DM type 2 found that the activity of G6PD enzyme is increasing in male human erythrocyte than the activity of G6PD enzyme in females, which leads to enhancing the free radicals and reactive oxygen species. Furthermore, the patients are suffering from many problems and inflammation in case of inhibition or deficiency of the enzyme. Controlling hyperglycemia avoids the patients from many risks and inflammation.

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