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Original Research Article

Association of blood glucose-6-phosphate dehydrogenase and ferritin levels with glycated hemoglobin in patients with type 2 diabetes mellitus: A cross-sectional study

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Abstract

Introduction: Increased Glycated Hemoglobin (HbA1c) is a crucial indicator for inappropriate or inadequate diabetes care in patients with type 2 diabetes since it indicates inadequate long-term glycemic control. Serum ferritin, an indicator of iron stores, can be elevated due to chronic inflammation or iron overload, conditions that are not uncommon in diabetes. G6PD (Glucose-6-Phosphate Dehydrogenase) deficiency, while less directly related to diabetes control, can influence oxidative stress and redox status, potentially affecting overall diabetic complications.
Aims & Objectives: The study aims to investigate whether there is a significant correlation between Serum Ferritin, G6PD, and HbA1c in cases of type 2 diabetes mellitus and normal healthy controls.
Materials and Methods: This cross-sectional analytical study was conducted in the Department of Biochemistry and Endocrinology at KPC Medical College. 42 patients, aged 18 to 80 years, with type 2 diabetes mellitus, diagnosed according to the American Diabetes Association (ADA) criteria, were selected as cases. All are newly diagnosed patients without any comorbidities. An equal number of age and sex-matched healthy individuals were recruited as controls.
Results: Significant negative correlations have been observed both between G6PD and HbA1c [r = (-0.84), p<0.001] and G6PD and serum ferritin values [r = (-0.76), p<0.001]. A significant positive correlation has been observed between serum ferritin and HbA1c values (r =0.97, p<0.001). In this study, an increase in HbA1c is associated with a decrease in the serum levels of G6PD (negatively correlated) and an increase in serum ferritin (positively correlated).
Conclusion: Elevated ferritin can be used as a marker for glycemic control in diabetic patients, while G6PD status is relevant for managing oxidative stress-related complications.

Keywords: Glycemic control, Oxidative stress, Iron overload.

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1. Introduction

Diabetes mellitus is a spectrum of disorders phenotypically sharing the common feature of hyperglycemia. It is a chronic condition and has multiple micro- and macrovascular complications with a major impact on the well-being of mankind worldwide. It ranks among the top ten attributable causes of death in adults, and is estimated to be the causative factor of four million deaths worldwide.[1] Several proteins are glycated non-enzymatically in our body, like Glycated Hemoglobin (HbA1c), &and monitor glycemic status for the last 6-8 weeks. Many researchers have reported that low levels of G6PD enzyme may be associated with lower levels of HbA1c, and some reported just the reverse, which may ultimately lead to erroneously reporting low or high values of HbA1c in the laboratory. Glucose-6-phosphate dehydrogenase (G6PD) is the rate-limiting enzyme of the Hexose Monophosphate Shunt pathway of glucose metabolism. Serum ferritin levels reflect not only the amount of stored iron in our body but are a proven positive acute

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phase reactant and elevated in all inflammatory processes. In a few studies, it has been shown that the rise in serum ferritin associated with Type 2 Diabetes Mellitus, as well as elevated levels of ferritin, may help identify individuals at high risk of T2DM.

Diabetes mellitus may arise from a defect in insulin secretion, insulin action, or both.[2] Type-2 diabetics usually have a block at the receptor level or the post-receptor level or both. The microvascular complications include retinopathy, nephropathy, and neuropathy, while the macrovascular complications include cardiovascular, cerebrovascular, and peripheral vascular disease.[3] Approximately 5.1 million people aged between 20 to 79 years died from diabetes, accounting for 8.4% of the causes of mortality worldwide in this age group.[4] In India, 65.1 million in the age group of 20 to 79 have diabetes (8.9%) and are expected to rise to 109 million by the year 2035.[5] Glycated hemoglobin (HbA1c) estimated in a whole blood sample provides evidence about an individual's average glycemic control during the previous 6 – 8 weeks.[6] The HbA1c is now recommended as a standard of care (SOC) for testing and monitoring diabetes, specifically the type 2 diabetes.[7] The glucose-6-phosphate dehydrogenase (G6PD) deficiency, X linked chromosomal disorder, affects approximately 400 million persons living mainly in tropical countries, which makes it the commonest enzyme deficiency globally.[8] G6PD deficiency can cause hemolytic anemia, especially when exposed to certain drugs, infections or foods. The relationship between G6PD and HbA1c in type 2 diabetic patients is unclear and controversial. Some studies suggest that G6PD deficiency may lower HbA1c levels and mask the true glycemic status of diabetes type 2 patients.[9] Other studies suggest that G6PD deficiency may increase HbA1c levels and worsen the oxidative stress and inflammation of diabetes type 2 patients.[10]

Serum ferritin levels reflect the status of iron reserves in healthy individuals and act as a positive acute phase reactant, and several studies have found increased ferritin levels in association with such diabetic complications as retinopathy, nephropathy, and vascular dysfunction in patients with diabetes and with elevated FBG.[11,12] A prospective cohort study conducted in China reported a significant correlation between serum ferritin levels and HbA1c.[13] and epidemiological studies confirmed this correlation.[14] Chen et al. has also reported excessive iron as a cause of metabolic syndrome, while insulin resistance decreased with decreasing serum ferritin levels.[13] The studies conducted by Kunutsor et al. also supported these findings.[14] Many studies in this regard, however, have raised the question of whether serum ferritin levels and other inflammatory markers can serve as a marker in the early diagnosis of type 2 diabetes, although studies on this particular issue are yet to be reported.

2. Aims and Objectives

The study aims to investigate whether there is any significant correlation between Serum Ferritin, G6PD, and HbA1c in patients suffering from Type-2 Diabetes Mellitus when compared with normal healthy controls.

The objectives of the study are:

1. To determine the glycemic state of a person with type 2 diabetes by measuring their blood sugar levels during fasting and after meals (post-prandial). To estimate HbA1c as a glycation index and to estimate G6PD enzyme activity and serum Ferritin.
2. To find out the relationship between Serum Ferritin, G6PD, and glycemic control of type 2 Diabetes Mellitus.

3. Materials and Methods

3.1. Study design

The study was conducted as a cross-sectional analytical study involving individuals diagnosed with Type 2 Diabetes.

3.2. Inclusion criteria

Male and female patients aged 18 to 80 years with type 2 diabetes diagnosed according to the American Diabetes Association criteria (HbA1c ≥ 6.5% or fasting blood glucose ≥ 126 mg/dL or postprandial blood glucose ≥ 200 mg/dL).

1. Patients who are willing to participate and give informed consent voluntarily.

3.3. Exclusion criteria

1. Patients with other types of diabetes (type 1, gestational, secondary, or monogenic). –
2. Patients with hemoglobinopathies or other causes of hemolytic anemia (such as malaria, sickle cell disease, or autoimmune hemolytic anemia).
3. Patients with chronic kidney disease (serum creatinine > 1.5 mg/dL) or liver disease (serum bilirubin > 2 mg/dL).
4. Patients who are pregnant or lactating.
5. Patients who are taking drugs that can affect G6PD activity (such as aspirin, sulfonamides, quinolones, or antimalarials).

3.4. Selection of study subjects

The prevalence of type 2 diabetes mellitus is 9.3 % in India as per the National Non-Communicable Disease Monitoring Survey. Using the formula for sample size calculation, Sample size $=4PQ/L^2$, where P is the prevalence of Type-2 Diabetes Mellitus, Q=100-P, and L is an allowable error of 10 percent, the sample size was calculated initially as 33.74. Adding an allowance of 20% for non-response, 42 Type 2 Diabetes patients were recruited as cases from the outpatient department of the Department of Endocrinology of KPC Medical College. An equal number of age- and sex-matched

healthy individuals were recruited as controls. The controls were selected from the patient's relatives who accompanied the patients included in the study, faculty, and hospital staff.

3.5. Sample collection

Relevant clinical information was collected, including demographics, medical history, and diabetes-specific parameters (e.g., duration of diabetes, current treatment regimen). Venous blood samples were collected from each participant following standard protocols of asepsis, once 6ml after 8-10 hours of calorie deprivation, and then 2ml after two hours of taking lunch. The initial 6ml of blood sample was divided as 2ml in a clot activator polystyrene tube for serum Ferritin, 2ml in fluoride vials for Fasting Plasma Glucose, and 2ml in an EDTA vial for HbA1c and G6PD, while the whole 2ml of the second collection was collected in a fluoride vial for Post Prandial Plasma Glucose.

3.6. Laboratory procedures

The assay of plasma glucose was done in a fully automated biochemistry analyzer by the Glucose Oxidase- Peroxidase method using standardized kits in the NABL-accredited central laboratory of KPC Medical College. The estimation of G6PD activity was done using standardized marketed kits for assay (Accurex autozyme quantitative kit) in a semi-autoanalyzer, and HbA1c was estimated using the HPLC method in Biorad D-10 equipment. Serum ferritin was measured in an ELISA reader using standardized ELISA kits (AccuBind Ferritin Sandwich Assay -96 wells) after the construction of the calibration curve, before the assay of the samples, and values obtained by extrapolation from the standard curve. All the tests were done on the same day of collection of samples, except serum for assay of Ferritin was stored in a -20°C refrigerator and assayed by ELISA once a week. Inter-laboratory checks were performed for validation of G6PD and serum Ferritin assays.

3.7. Study duration

The study was conducted over six months, from January to June 2024, after approval from the Institutional Ethics Committee of KPC Medical College.

3.8. Statistical analysis

Data were tabulated in Microsoft Office XL then data cleansing was performed. Statistical analysis was done by the Microsoft Excel 2007 Analysis Tool Pak for descriptive statistics, independent t-tests, and scatterplots. The online free social statistics calculator was used for normality test (Kolmogorov-Smirnov), Chi-square tests, and the calculation of correlation coefficients. The continuous data were checked for normality using the Kolmogorov-Smirnov Test. Data comparison was conducted using an independent t-test (2-tailed). The comparison of categorical data (gender distribution) was done by the Chi-square test. Pearson's correlation coefficients were calculated to determine the relationship between variables. A p-value of <0.05 was considered statistically significant.

4. Results

The mean age (62.1 years ± 11.96) of cases of Type 2 Diabetes Mellitus was significantly higher (p=0.024) than that of the non- Diabetic control group (54.36 years ± 16.73). Among the 42 cases of Type 2 Diabetes, there are 27 females & 15 males. The mean Fasting & Post prandial blood glucose levels & HbA1c in cases are 119.27 ± 27.65 mg/dL, 200.22 ± 37.95 mg/dL & 10.23% ± 2.57, respectively. In the control group, the mean Fasting, Post-prandial blood glucose levels & HbA1c are 89.61 ± 13.87 mg/dL, 110.61 ± 10.64 mg/dL & 5.41% ± 0.55, respectively. The difference in the mean above parameters is statistically significant (p<0.0001). The blood G6PD levels are significantly higher (p<0.00001) in healthy controls (11.52 ± 1.39 unit/g of Hb) than the Type 2 Diabetes cases (5.59 ± 1.32 unit/g of Hb) whereas the serum ferritin levels in cases (169.58 µg/L ± 34.64) are significantly (p<0.00001) higher than the healthy controls (21.02 µg/L ± 8.90). The BMI in Type 2 Diabetes cases (23.82 ± 3.99 kg/m^2) does not differ significantly (p=0.11) with that of the healthy non-Diabetic controls (24.85 ±2.50 kg/m^2).(**Table 1**) A significant positive correlation has been found between serum ferritin & HbA1c (r=0.97, p<0.001, N=42) (**Figure 1**), whereas a significant negative correlation has been found between serum ferritin and G6PD (r=-0.76, p<0.001, N=42) (**Figure 2**). A significant negative correlation has been observed between G6PD and HbA1c levels (r=-0.84, p<0.001, N=42) (**Figure 3**).

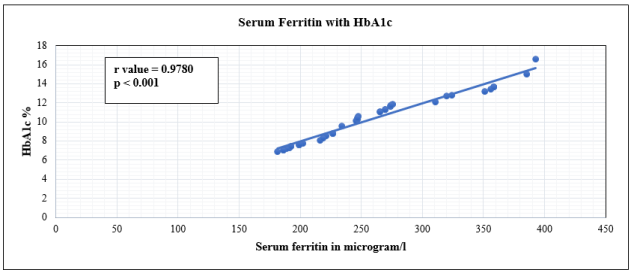

Figure 1: Scatterplot showing correlation between serum ferritin and HbA1c (cases n=42)

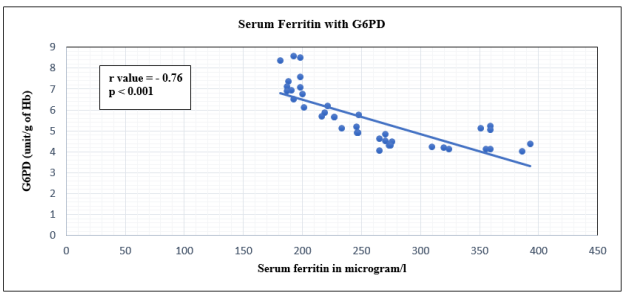

Figure 2: Scatterplot showing correlation between Serum Ferritin and G6PD (cases n=42)

Table 1: The demographic and biochemical parameters of the study:

Variables	Cases (n = 42), Mean±SD p value(K-S)	Controls (n = 42), Mean±SD p value(K-S)	p – value
Age (in completed years)	62.1 ± 11.96, 0.11	54.36 ± 16.73, 0.15	0.024
Gender (Male/Female)	15(M) / 27(F)	22(M) / 20(F)	0.12
Body Mass Index (BMI) (kg/m^2)	23.82 ± 3.99, 0.97	24.85 ± 2.50, 0.48	0.11
Fasting Blood Glucose (FBS) (mg/dl)	119.27 ± 27.65, 0.30	89.61 ± 13.87, 0.52	<0.0001
Postprandial Blood Glucose (PPBS) (mg/dl)	200.22 ± 37.95, 0.84	110.61 ± 10.64, 0.90	<0.0001
Glycated Hemoglobin (HbA1c%)	10.23 ± 2.57, 0.56	5.41 ± 0.55, 0.80	<0.0001
Glucose 6 phosphate dehydrogenase (G6PD) (units/g of Hb)	5.59 ± 1.32, 0.17	11.52 ± 1.39, 0.32	<0.00001
Serum Ferritin (µg/L)	169.58 ± 34.64, 0.26	21.02 ± 8.90, 0.19	<0.00001

Description of clinical and biochemical parameters of cases and controls (N=84). K-S: Kolmogorov Smirnov Test for normality (p>0.05 signifies normal distribution); p-value < 0.05 is considered statistically significant.

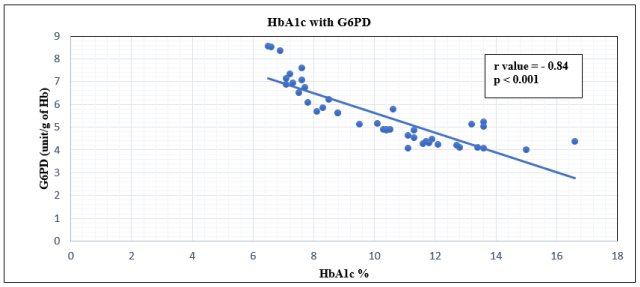

Figure 3: Scatterplot showing correlation between HbA1c and G6PD (cases n=42)

5. Discussion

The present study provides a comprehensive analysis of the biochemical and clinical profiles of individuals with Type 2 Diabetes Mellitus (T2DM) compared to non-diabetic controls. The findings highlight significant differences in various parameters, underscoring the multifaceted nature of T2DM and its systemic implications. The mean age of T2DM cases (62.1 ± 11.96 years) was significantly higher than that of the control group (54.36 ± 16.73 years; p=0.024), aligning with the understanding that T2DM prevalence increases with age. The gender distribution among the 42 T2DM cases showed a higher proportion of females (27) compared to males (15), which may reflect gender-specific risk factors or healthcare-seeking behaviors.

T2DM cases exhibited markedly elevated levels of fasting blood glucose (119.27 ± 27.65 mg/dL), postprandial blood glucose (200.22 ± 37.95 mg/dL), and HbA1c (10.23% ± 2.57) compared to controls (89.61 ± 13.87 mg/dL, 110.61 ± 10.64 mg/dL, and 5.41% ± 0.55, respectively), with all differences being highly significant (p<0.0001). These findings are consistent with the pathophysiological hallmark of T2DM —chronic hyperglycemia due to insulin resistance and/or impaired insulin secretion.

No significant difference was observed in BMI between T2DM cases (23.82 ± 3.99 kg/m²) and controls (24.85 ± 2.50 kg/m²; p=0.11), suggests that factors beyond BMI, such as fat distribution, genetic predisposition, and metabolic factors, may play more pivotal roles in the development and progression of T2DM in this population. The study found significantly lower blood G6PD levels in T2DM cases (5.59 ± 1.32 U/g Hb) compared to controls (11.52 ± 1.39 U/g Hb; p<0.00001). The negative correlation between G6PD activity and HbA1c levels (r=-0.84, p<0.001, N=42) can be explained by the role of G6PD in maintaining cellular redox balance and red blood cell integrity.

G6PD is an important enzyme that helps the body to produce NADPH, which in turn keeps glutathione active—a key substance that protects cells from damage caused by harmful molecules (ROS). When G6PD levels are low, the body becomes less effective at protecting cells from oxidative damage, which will increase cellular oxidative stress. This increased stress can interfere with insulin function, leading to elevated blood sugar and poor management of diabetes, which is typically reflected by higher HbA1c levels. However, low G6PD also causes red blood cells to break down faster. Since HbA1c measures blood sugar levels based on how long red blood cells live, a shorter lifespan means there's less time for sugar to remain attached to hemoglobin (glycation). This can make HbA1c look lower than it is, even if blood sugar is high, so it may not give an accurate picture of diabetes control in people with low G6PD.[15-18]

A significant elevation in serum ferritin levels was observed in T2DM cases (169.58 ± 34.64 µg/L) compared to controls (21.02 ± 8.90 µg/L; p<0.00001). Multiple studies have reported a positive correlation between serum ferritin and HbA1c levels, similar to our study (r=0.97, p<0.001, n=42), suggesting that higher iron stores may impair glycemic control. The positive correlation between serum ferritin and HbA1c levels in individuals with T2DM reflects the interplay between iron metabolism, oxidative stress, and glucose regulation. Ferritin, primarily an iron-storage protein, also functions as an acute-phase reactant and is elevated during inflammation and oxidative stress, both of which are common in diabetes. Excess iron stored in ferritin

can catalyze the formation of reactive oxygen species (ROS) through the Fenton reaction, generating hydroxyl radicals that can damage cellular structures and impair insulin signaling. This oxidative stress promotes insulin resistance, reduces glucose uptake by cells, and results in sustained hyperglycemia, which is reflected in elevated HbA1c levels.

Higher ferritin levels may directly interfere with pancreatic β-cell function, further reducing insulin secretion. Therefore, as ferritin levels rise—due to increased iron stores or inflammation—glycemic control worsens, leading to higher HbA1c values.[19] Negative correlation was observed between G6PD and serum ferritin levels ($r=-0.76$, $p<0.001$, $N=42$) occurs because low G6PD activity reduces antioxidant defense, leading to increased oxidative stress. This oxidative stress stimulates ferritin production as a protective response to sequester free iron that could otherwise catalyze the formation of harmful reactive oxygen species through the Fenton reaction. However, there are articles on low serum ferritin levels and G6PD deficiency with increased risk of anemia,[20] but more targeted research is needed to elucidate the direct relationship and potential negative correlation between serum ferritin and G6PD activity in diabetic populations. The study highlights the complex interrelationships between iron metabolism, oxidative stress, and glycemic control in patients suffering from T2DM. Elevated serum ferritin levels and reduced G6PD activity are significantly associated with poor glycemic control, as evidenced by higher HbA1c levels. These findings suggest that assessing serum ferritin and G6PD levels could provide additional insights into the metabolic status of T2DM patients and may inform more tailored therapeutic strategies. Further research is warranted to explore the potential benefits of interventions targeting iron overload and oxidative stress in improving glycemic outcomes in T2DM.

6. Conclusion

In this study, an increase in HbA_{1c} is associated with a decrease in G6PD levels (negatively correlated) and an increase in serum ferritin levels (positively correlated). The above findings indicate that poor glycaemic control in Type 2 Diabetes Mellitus is associated with elevated ferritin and decreased G6PD levels. Elevated serum ferritin levels indicate iron overload & chronic inflammation, which can contribute to insulin resistance, oxidative stress, and worsening of diabetes. Lower G6PD levels may increase the risk of oxidative damage and hemolysis in Diabetes cases.

Further studies with a higher sample size are necessary to conclude whether increased serum ferritin and lower activity of G6PD in blood can be used as a marker of poor glycaemic control. Prospective cohort studies may be conducted to understand whether increased ferritin and decreased G6PD are contributory factors in the pathophysiology of Type 2 Diabetes or are the effects of chronic hyperglycaemia.

7. Conflict of Interest

None.

8. Source of Interest

None.

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