Effect of Iron Deficiency Anemia on HbA1c Levels in Non-Diabetic Individuals

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ABSTRACT

Background: Glycated hemoglobin (HbA1c) is the parameter of choice to evaluate the long term degree of glycaemic control in patients with diabetes mellitus (DM). Iron Deficiency Anemia is the most common cause of anemia.

Objective: We study the effect of iron deficiency anemia on HbA1c levels in non-diabetic individuals.

Patients and Methods: This was a case-control study in the period from February 2019 to September 2019. Included 90 cases at outpatient Clinic of Internal Medicine Department in Zagazig University Hospitals. The patients were randomly divided (by alternation) into three groups 30 patients, Group1: who are healthy. Group2: who have iron deficiency anemia. Group3: who has any type of anemia other than iron deficiency anemia. A detailed history of clinical examination and biochemical examination was performed including HbA1c.

Results: The study revealed that there was a highly significant difference between the before and after treatment in the iron deficiency group as regard HbA1c and there was no significant difference between the studied groups as regard FBG or PPG. **Conclusion:** There is a significant relation between HbA1c and iron deficiency anemia while there is no relation between iron deficiency anemia and Fasting blood sugar or Postprandial sugar, Iron deficiency anemia elevates HbA1c levels in non-diabetic individuals.

Keywords: Glycated hemoglobin, diabetes mellitus, Iron deficiency anemia.

INTRODUCTION

Glycated hemoglobin (HbA1c) is the parameter of choice to evaluate the long term degree of glycaemic control in patients with diabetes mellitus (DM)[(1)].

It reflects the patient's glycemic status over the previous 3 months. HbA1c is widely used as a screening test for diabetes mellitus, also, HbA1c ≥6.5% (48mmol/mol) is recommended as the cutoff point for diagnosing DM[(2)].

HbA1c is a form of hemoglobin with a glucose residue attached to the terminal NH2 group (valine residue) of one or both HbA beta chains. Red blood cells are freely permeable to the plasma glucose molecules, and hemoglobin is practically exposed to the same glucose concentrations as plasma. Therefore, the levels of HbA1c reflect more specifically the glycaemic control from the past 2 to 3 months, the red blood cells half lifetime, preceding the measurement[(3)].

Clinically, HbA1c is used to determine improvement or worsening in glycaemic control by comparing HbA1c serial results to determine if the patients achieve their HbA1c targets, and recently, it has been also recommended to diagnose DM[(2)].

Depending on the methodology used to measure HbA1c, several factors can affect or interfere in the HbA1c results[(1)].

Traditionally, some diseases and pathological states, such as anemia and hemoglobinopathies, are considered potential factors that can significantly alter HbA1c results[(4)].

Anemia is a public health problem that affects worldwide populations. Its primary cause is iron deficiency (ID). Approximately one-third of the patients with anemia have iron deficiency (IDA)[(5)].

There are several studies on the role of anemia on the HbA1c levels but their results are conflicting[(6)]. Because of the important role of HbA1c on DM diagnosis and the high prevalence of anemia worldwide. So, this study aimed to investigate the effect of IDA on HbA1c levels in patients without DM.

PATIENTS AND METHODS

This was a case-control study in the period from February 2019 to September 2019. included 90 cases at the Outpatient Clinic of Internal Medicine Department in Zagazig University Hospitals. The patients were randomly divided (by alternation) into three groups 30 patients, Group1: healthy control whose mean age was 48.20 ± 8.58 years with a range of (35.0 – 62.0) years, 15 males (50%) and 15 females (50%). Group2: iron deficiency anemia whose mean age was 47.0 ± 7.77 years with a range of (34.0 – 61.0) years, 15 males (50%) and 15 females (50%). Group 2: non-iron deficiency anemia whose mean age was 49.77 ± 8.88 years with a range of (35.0 – 62.0) years, 17 males (56.7%) and 13 females (43.3%).

Ethical approval: **An approval of the study was obtained from Zagazig University academic and ethical committee.** Every patient signed an informed written consent for acceptance of the operation.

Inclusion criteria: Patients without anaemia, haemoglobin (Hb) more than 13g/dl (if male) or more than 12g/dl (if female) with serum ferritin above 15 µg/mL and below 150 µg/mL (if female) or below 200 µg/mL (if male),plus complete blood count within the reference values. Patients with IDA- serum ferritin below

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15 µg/mL, haemoglobin (Hb) below 13g/dl (if male) or below 12g/dl (if female) and mean corpuscular volume (MCV) below 80 fl. Patients with any type o anemia other than iron deficiency anemia with, hemoglobin (Hb) below 13g/dl (if male) or below 12g/dl (if female) and mean corpuscular volume (MCV) more than 80 fl.

Exclusion criteria: Patients with overt kidney failure, heart failure, and liver failure. Patients with diabetes as the patients had to have no history of DM and two fasting glycemia ≤ 126 mg/dL, performed close to the date of the complete blood count, and HbA1c b 6.5% (48 mmol/mol).

All patients were subjected to thorough clinical examination, thorough history of present illness and past history of previous hospital admission and any medical disorder, Full general examination and anthropometric measurements include; (pulse examination, blood pressure measurement, temperature, body mass index (BMI)), the laboratory investigations were done in the Medical Biochemistry Department and the Clinical Pathology Department of Zagazig University Hospitals and they include; Complete blood picture (CBC). Liver function tests: serum bilirubin (total and direct), serum albumin, serum alanine transaminase, and aspartate transferase. Renal function tests: serum creatinine & serum urea. Bleeding profile (Prothrombin time (PT), INR, and Partial thromboplastin time (PTT)).

Erythrocyte sedimentation rate (ESR):

The basic principle of the ESR is that when anticoagulated blood is placed in a vertical column the RBCs normally settle quite slowly. This occurs for 2 main reasons: RBCs repel each other due to the negative charges on their surfaces, or zeta potential, and the large surface-area-to-volume ratio of normal RBCs resists settling. The aggregation of RBCs into rouleaux, which happens slowly under normal conditions, markedly accelerates sedimentation by decreasing the surface-area-to-volume ratio. Conditions that promote the formation of rouleaux produce an elevated ESR. The most common promoter of rouleaux is increased plasma fibrinogen. Fibrinogen's positive charge decreases the RBCs' zeta potential, leading to increased rouleaux and an increased ESR. The clinical utility of the ESR is largely attributable to increased fibrinogen in acute-phase reactions and chronic inflammatory conditions. The ESR may be elevated by other conditions that decrease the zeta potential or the RBC surface-area-to-volume ratio. The zeta potential is reduced by other plasma proteins, including immunoglobulins, as well as cholesterol, phospholipids, and some medications. By creating more space between RBCs, anemia reduces the effect of the zeta potential to slow sedimentation. Decreases in the surface-area-to-volume ratio, as in macrocytosis, also increase the ESR. The ESR may be decreased by conditions that interfere with the formation of rouleaux or increase the RBC surface-area-to-volume ratio. Rouleaux formation is hindered by spherocytosis, sickle cell disease, microcytosis, marked variation in RBC size (anisocytosis), and some drugs. Polycythemia decreases the compactness of rouleaux formation. The surface-area-to-volume ratio is increased in some thalassemia's and hemoglobinopathies.

Hemoglobin A1c (HBA1c):

Glycation of proteins is a frequent occurrence, but in the case of hemoglobin, a non-enzymatic reaction occurs between glucose and the N-end of the beta chain. This forms a Schiff base which is itself converted to 1-deoxyfructose. This rearrangement is an example of an Amadori rearrangement. When blood glucose levels are high, glucose molecules attach to the hemoglobin in red blood cells. The longer hyperglycemia occurs in the blood, the more glucose binds to hemoglobin in the red blood cells and the higher the glycated hemoglobin. Once a hemoglobin molecule is glycated, it remains that way. A buildup of glycated hemoglobin within the red cell, therefore, reflects the average level of glucose to which the cell has been exposed during its life-cycle. Measuring glycated hemoglobin assesses the effectiveness of therapy by monitoring long-term serum glucose regulation. A1c is a weighted average of blood glucose levels during the life of the red blood cells (117 days for men and 106 days for women). Therefore, glucose levels on days nearer to the test contribute substantially more to the level of A1c than the levels in days further from the test (NGSP: HbA1c and eAG, 2015).

Reference values: 4.0-5.6% ; <18 years: Hemoglobin A1c criteria for diagnosing diabetes have not been established for patients who are <18 years of age. > or =18 years: Increased risk for diabetes (prediabetes): 5.7-6.4% . Diabetes: > or =6.5

Based on hemoglobin levels, patients were categorized as having mild, moderate, or severe anemia: mild anemia (male patients, 1212.9 g/dL and female patients, 11-11.9 g/dL); moderate anemia (male patients, 9-11.9 g/dL and female patients, 8-10.9); and severe anemia (male patients, less than 9 g/dL and female patients, less than 8 g/dL). Those with predominantly microcytic indices (MCV<80 fL) and hypochromic indices (MCH<26 pg/cell) were considered to have iron deficiency anemia, which was then also confirmed by low serum ferritin levels (<20 ng/mL in females and <29 ng/mL in male patients).

RESULT

This study showed that mild anemia was present in 8.3% of cases, moderate in 66.7% of cases, and severe anemia in 25% of cases Figure (1).

There was no significant relationship between disease severity and HbA1c **Table (1)**.

There was a highly significant difference between before and after treatment in the iron-deficiency anemia group as regard Hemoglobin, HbA1c, TIBC, Serum ferritin, and hematocrit value, while there is no significant difference between before and after treatment in the iron-deficiency anemia group as regard FBG or PPG **Table (2)**.

There was a highly significant difference between the studied groups as regard reticulocyte count and ferritin **Table (3)**.

This study showed that there was no significant difference between the studied groups as regard FBG while there was a highly significant difference between the studied groups as regard HbA1c **Table (4)**.

There was a highly significant difference between the before and after treatment in the iron deficiency group as regard HbA1c **Table (5)**.

There was a highly significant difference between the studied groups as regard HbA1c after treatment **Table (6)**.

There was a highly significant difference between the studied groups as regard serum iron, TIBC, and Transferrin saturation **Table (7).**

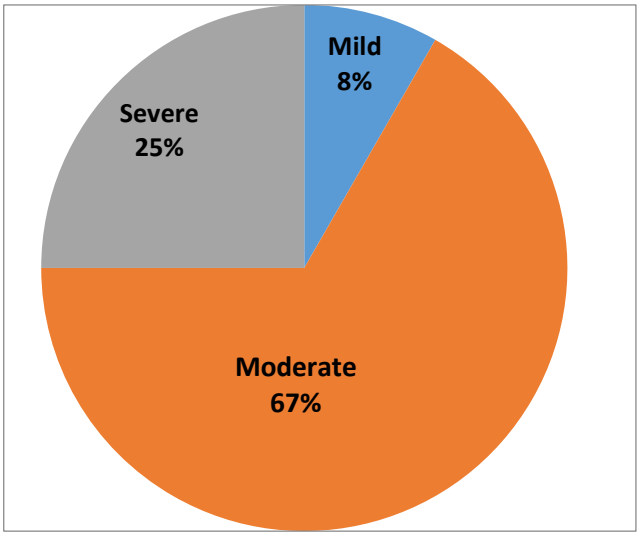

Fig. (1): Distribution of anemia cases as regard severity.

Table (1): Relation between disease severity and HbA1c

	Mild (n = 5)	Moderate (n = 30)	Severe (n = 15)	Test of sig.	p-value
HbA1c:					
Min. – Max.	5.0-6.0	5.1-6.2	5.3-6.3	F= 0.931	0.911
Mean ± SD.	5.5 ± 0.4	5.6 ± 0.41	5.7 ± 0.3		

Table (2): Comparison between the two periods according to CBC in the iron-deficiency anemia group (n = 30)

	Before (n = 30)	After (n = 30)	t	p-value
Hb				
Min. – Max.	8.10 – 12.30	12.10 – 16.90	11.938*	<0.001*
Mean ± SD.	10.41 ± 1.11	14.09 ± 1.33		
Median (IQR)	10.25 (9.60 – 11.40)	14.05 (12.90 – 14.80)		
HbA1c				
Min. – Max.	5.50 – 6.20	4.90 – 5.60	18.311*	<0.001*
Mean ± SD.	5.90 ± 0.19	5.33 ± 0.18		
Median (IQR)	5.90 (5.70 – 6.10)	5.35 (5.20 – 5.50)		
HT. (%)				
Min. – Max.	31.0 – 45.0	43.0 – 52.0	11.162*	<0.001*
Mean ± SD.	36.23 ± 4.45	47.73 ± 2.82		
Median (IQR)	35.0 (32.0 – 40.0)	47.0 (46.0 – 50.0)		
TIBC				
Min. – Max.	461.0 – 600.0	349.0 – 540.0	17.5	**<0.001 (HS)**
Mean ± SD.	534.1 ± 47.23	394.3 ± 39.61		
Median (IQR)	529.5 (496.0 – 586.0)	399.1 (399.0 – 510.0)		
Serum ferritin				
Min. – Max.	4.0 – 45.0	201-304	26.9	**<0.001 (HS)**
Mean ± SD.	26.17 ± 4.36	270.4±23.6		
Median (IQR)	29.0 (11.0 – 9.0)	277(220.0 -290.0)		
FBG				
Min. – Max.	70.0 – 97.0	70.0 – 96.0	0.932	0.812
Mean ± SD.	83.40 ± 8.20	82.90 ± 8.40		
Median (IQR)	82.0 (76.0 – 90.0)	81.0 (75.0 – 89.0)		
PPG				
Min. – Max.	111.0 – 133.0	111.0 – 130.0	1.02	0.712
Mean ± SD.	122.3 ± 7.59	121.9 ± 6.9		
Median (IQR)	122.0 (115.0 – 130.0)	121.0 (114.0 – 128.0)		

t: Paired t-test, p: p-value for comparing between **before** and **after,** *: Statistically significant at $p \leq 0.05$

Table (3): Comparison between the three studied groups according to retics and ferritin.

	Group I (n = 30)	Group II (n = 30)	Group III (n = 30)	H	p-value
Retics					
Min. – Max.	0.56 – 2.47	0.02 – 0.64	0.03 – 0.80	58.536*	<0.001*
Mean ± SD.	1.54 ± 0.47	0.27 ± 0.19	0.26 ± 0.25		
Median (IQR)	1.46 (1.19 – 1.92)	0.22 (0.10 – 0.41)	0.15 (0.08 – 0.53)		
Sig. bet. groups	$p_1<0.001^*, p_2<0.001^*, p_3=0.649$				
Ferritin					
				33.692*	<0.001*
Mean ± SD.	111.7 ± 7.37	26.17 ± 4.36	114.4 ± 7.40		
Median (IQR)	111.0 (43.0 – 160.0)	29.0 (11.0 – 39.0)	109.5 (62.0 – 172.0)		
Sig. bet. groups	$p_1<0.001^*, p_2=0.976, p_3<0.001^*$				

H: H for Kruskal Wallis test, Pairwise comparison bet. each 2 groups was done using Post Hoc Test (Dunn's for multiple comparisons test)

p: p-value for comparing between the studied groups

p_1: p-value for comparing between **normal individuals** and **iron deficiency anemia**

p_2: for comparing between **normal individuals** and **other types of anemia**

p_3: p-value for comparing between **iron deficiency anemia** and **other types of anemia**

*: Statistically significant at $p \leq 0.05$

Table (4): Comparison between the three studied groups according to blood glucose and HbA1c.

Sugar picture	Group I (n = 30)	Group II (n = 30)	Group III (n = 30)	F	p-value
FBG					
Min. – Max.	72.0 – 97.0	70.0 – 97.0	72.0 – 98.0	0.043	0.958
Mean ± SD.	83.27 ± 6.64	83.40 ± 8.20	83.80 ± 7.16		
Median (IQR)	83.0 (81.0 – 88.0)	82.0 (76.0 – 90.0)	83.0 (77.0 – 86.0)		
HbA1c		**Before**			
Min. – Max.	3.90 – 5.40	5.50 – 6.20	4.0 – 5.40	81.230*	<0.001*
Mean ± SD.	4.58 ± 0.51	5.90 ± 0.19	4.72 ± 0.53		
Median (IQR)	4.40 (4.10 – 5.10)	5.90 (5.70 – 6.10)	4.80 (4.20 – 5.30)		
Sig. bet. groups	$p_1<0.001^*, p_2=0.456, p_3<0.001^*$				

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using the Post Hoc Test (Tukey)

p: p-value for comparing between the studied groups

p_1: p-value for comparing between **normal individuals** and **iron deficiency anemia**

p_2: p-value for comparing between **normal individuals** and **other types of anemia**

p_3: p-value for comparing between **iron deficiency anemia** and **other types of anemia**

*: Statistically significant at $p \leq 0.05$

Table (5): Comparison between the two periods according to HbA1c in Iron deficiency anemia (n = 30).

HbA1c	Before (n = 30)	After (n = 30)	t	p-value
Min. – Max.	5.50 – 6.20	4.90 – 5.60	18.311*	<0.001*
Mean ± SD.	5.90 ± 0.19	5.33 ± 0.18		
Median (IQR)	5.90 (5.70 – 6.10)	5.35 (5.20 – 5.50)		

t: Paired t-test

p: p-value for comparing between **before** and **after**

*: Statistically significant at $p \leq 0.05$

Table (6): Comparison between the three studied groups according to HbA1c.

Sugar picture	Group I (n = 30)	Group II (n = 30)	Group III (n = 30)	F	p-value
HbA1c		**After**			
Min. – Max.	3.90 – 5.40	4.90 – 5.60	4.0 – 5.40	24.779	<0.001*
Mean ± SD.	4.58 ± 0.51	5.33 ± 0.18	4.72 ± 0.53		
Median (IQR)	4.40 (4.10 – 5.10)	5.35 (5.20 – 5.50)	4.80 (4.20 – 5.30)		
Sig. bet. groups	p_1<0.001*,p_2=0.454,p_3<0.001*				

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using the Post Hoc Test (Tukey)
p: p-value for comparing between the studied groups
p_1: p-value for comparing between **normal individuals** and **iron deficiency anemia**
p_2: p-value for comparing between **normal individuals** and **other types of anemia**
p_3: p-value for comparing between **iron deficiency anemia** and **other types of anemia**
*: Statistically significant at $p \leq 0.05$

Table (7): Comparison between the three studied groups according to iron profile.

	Group I (n = 30)	Group II (n = 30)	Group III (n = 30)	F	p-value
TIBC					
Min. – Max.	243.0 – 444.0	461.0 – 600.0	257.0 – 446.0	103.448*	<0.001*
Mean ± SD.	357.7 ± 56.46	534.1 ± 47.23	369.4 ± 55.17		
Median (IQR)	357.0 (311.0 – 412.0)	529.5 (496.0 – 586.0)	371.5 (320.0 – 416.0)		
Sig. bet. groups	p_1<0.001*,p_2= 0.673,p_3<0.001*				
Transferrin saturation (%)					
				72.665*	<0.001*
Mean ± SD.	34.70 ± 4.67	11.40 ± 2.91	32.63 ± 3.05		
Median (IQR)	36.0 (25.0 – 42.0)	10.0 (6.0 – 16.0)	31.50 (27.0 – 39.0)		
Sig. bet. groups	p_1<0.001*,p_2= 0.600,p_3<0.001*				

F: F for ANOVA test, Pairwise comparison bet. each 2 groups was done using the Post Hoc Test (Tukey)
p: p-value for comparing between the studied groups
p_1: p-value for comparing between **normal individuals** and **iron deficiency anemia**
p_2: p-value for comparing between **normal individuals** and **other types of anemia**
p_3: p-value for comparing between **iron deficiency anemia** and **other types of anemia**
*: Statistically significant at $p \leq 0.05$

DISCUSSION

The present study showed that there was a highly significant difference between healthy control, iron deficiency anemia, and non-iron deficiency anemia as regard HbA1c, with the highest level of HbA1c in the iron-deficiency anemia group.

Intra *et al.* [7] reported that HbA1c values were statistically higher (5.71%, (38.64 mmol/mol), 5.59% (37.37 mmol/mol)) compared to those measured in the individuals without anemia (5.33% (34.78 mmol/mol), 5.34% (34.81 mmol/mol); P<0.0001).

This was on the same side as what stated by **Solomon *et al.*** [8] who illustrated that the mean RBC, MCV, MCH, MCHC, RDW were 3.45 ± 0.8, 88.57±8.56, 29.89±4.04, 32.97±2.19, 3.45 ± 0.80 respectively. Pearson correlation test was used to determine the association between HbA1C and hematological parameters of the IDA patients. HbA1C was statistically non-significant with RBC, MCV, MCH, MCHC.

This was in agreement with **Manish *et al.*** [9] who revealed that Among the hematological parameters; RBC, Hgb, MCV, MCH showed a statistically significant mean difference between the iron deficiency and control groups

Grossman *et al.* [10] found that the exact mechanism underlying the IDA effects on HbA1c values is still unclear. A state of iron deficiency affects the lifespan of red blood cells, and the erythrocytes count is decreased, leading to an older population of red blood cells that are in contact with plasma glucose longer, causing falsely higher HbA1c measurements

In the current study, there was a highly significant difference between the before and after treatment in the iron deficiency group as regard HbA1c.

Kumar and Nutakki [11] verified that the mean HbA1c levels were significantly lower in patients after 2 months of treatment than in the controls (P0.05).

Silva, *et al.* [12] conducted a study on the effect of iron deficiency anemia on HbA1c levels is dependent on the degree of anemia with 122 patients. They concluded that IDA affects HbA1c results and this effect is dependent on anemia degree. These upward changes are statistically significant but they may not

clinically relevant when the overall variability of the HbA1c test is considered.

Also, **Bhardwaj *et al.* [13]** illustrated that the mean baseline HbA1c level in anemic patients (6.60) was significantly higher than that of controls (5.48). However, after 3 months of treatment, a significant decline from 6.60 to 5.74 was found in HbA1c levels.

In the present study, there was a highly significant difference between healthy control, iron deficiency anemia, and non-iron deficiency anemia as regard serum iron, TIBC, and Transferrin saturation.

This was in concordant with **Intra *et al.*[7]** who stated that there were differences in the mean levels of hematological and biochemical parameters between subjects affected by IDA and controls, hemoglobin, hematocrit, red blood cell count, MCV, MCH, MCHC, and ferritin values were lower in the iron-deficient subject.

This was in agreement with **Solomon *et al.* [8]** who reported that the mean RBC, Hgb, HCT, MCV, MCH, MCHC, HbA1c were significantly lower in the IDA group compared to the control group

This was in concordant with **Kumar and Nutakki[11]** who noticed that The mean baseline serum iron levels were significantly lower in patients than in controls ($p<0.01$)

In this work, we found there was a highly significant difference between the studied groups as regard reticulocyte count and ferritin.

This was in agreement with **Kumar and Nutakki[11]** who found that the mean baseline serum ferritin levels were significantly lower in patients than in controls. This was in concordant with **Sinha *et al.* [14]** who demonstrated that The mean baseline serum ferritin levels were significantly lower in patients than in control, they also stated that the mean hemoglobin and mean serum ferritin levels increased in anemia patients over 2 months of iron treatment.

In this study, we demonstrated that there was no significant difference between the studied groups as regard FBG or PPG.

Christy *et al.* [15] showed that conditions such as iron deficiency anemia can spuriously elevate A1C levels; consequently, care should be taken before altering the treatment regimen. Our observation also showed significantly higher A1C levels in anemic patients who had FPG between 100-126 mg/dl. As a result, anemia may exaggerate the picture of glycemic status in this group of patients.

CONCLUSION

In conclusion, there was a significant relation between HbA1c and iron deficiency anemia while there is no relation between iron deficiency anemia and Fasting blood sugar or postprandial sugar, Iron deficiency anemia elevates HbA1c levels in non-diabetic individuals.

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