Research Article

The Journal of Clinical Pathology (ISSN 2995-8598)

Correlation between Estimated Average Glucose Levels Calculated from HbA1C Values and Random Blood Glucose Levels in a Cohort of Subjects.

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Received Date : November 05, 2024
Accepted Date : November 06, 2024
Published Date : December 04, 2024

ABSTRACT

Diabetes mellitus (DM) is a chronic illness, caused due to resistance to insulin or poor production. Diagnostic criteria of Diabetes mellitus (DM) has been devised by World Health Organization (WHO), American Diabetes Association (ADA), based on plasma glucose level. Hemoglobin A1c (HbA1c) level remains the gold standard test for the assessment of glycemic control, and it reflects the mean glucose values in the previous 3 months period. HbA1c is expressed as a percentage, whereas the monitoring and treatment of diabetes are based on blood glucose levels expressed as mg/dL. It is appropriate to make it easy for the patient to understand both random blood sugar (RBS) and estimated average glucose (eAG) expressed with the same units. "Estimated average glucose" or eAG derived from HbA1c has been promoted by the American Diabetes Association (ADA). American Association of Clinical Chemists concludes that the correlation (r =0.92) is strong enough to justify reporting both HbA1c and eAG which indicate the 3 months control of the average sugar of the patient. This article determines the statistical correlation between eAG derived from HbA1C with RBS values both in diabetic and prediabetic subjects.

Methods: The RBS and HbA1c levels of 123 males and 39 females (12 – 70 years) were obtained and the eAG levels were calculated using Nathan's regression equation. The samples were divided into three groups on the basis of HbA1c levels as group 1: HbA1c greater than or equal to 6.5% (diabetic),group 2: HbA1c 5.7 to 6.4% (prediabetic), and group 3: HbA1c less than 5.7% (non diabetic).

Results: A total of 162 (123 males and 39 females) were enrolled for the study. The mean ±SD values of RBS, HbA1c and eAG of the population were 167.44 ± 88.74 mg/dl, 7.23 ±2.14 % and 161.20 ± 61.43 mg/dl respectively. HbA1C values were significantly correlated with RBS ($p < 0.001$) and eAG ($p < 0.001$).

Conclusion: The clinical importance of HbA1C,eAG in diagnosis and management of Diabetes Mellitus (DM) can be re-emphasized by this study. HbA1C along with eAG may be added as a test in the management of Diabetes Mellitus (DM), for the better understanding and maintenance of good glycemic control. However, eAG and RBS values cannot be used interchangeably.

Keywords: *Hemoglobin A1c (HbA1c), Random blood sugar (RBS), Estimated average glucose (eAG).*

INTRODUCTION

Diabetes poses a global health challenge. In 2021, 1 in 10 adults (537 million) had diabetes, and this is projected to increase to 783 million by 2045 (a 46% increase).1 Half of those with diabetes are undiagnosed.1 In the Middle East and North Africa (MENA), 73 million adults had diabetes in 2021, projected to rise to 136 million by 2045 (an 87% increase).[1]

Qatar is part of MENA, with a population of 3.05 million in 2024, of which only 10.8% are Qataris and the remainder are expatriates from over 150 nationalities.[2] The sole DM nationally representative population-based survey (2012 Qatar STEP wise Survey)[3] was conducted only among Qataris aged 18–64 years and estimated that DM, obesity, physical inactivity, and smoking prevalence were 10.4%, 41.5%, 40.0%,

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and 16.3%, respectively.[4]

Qatar faces a significant burden of diabetes, with a projected increase in prevalence from 17.8% (37,179 persons) in 2023 to 29.5% (84,516 persons) by 2050 among adult Qataris aged 20-79 years.[5] Obesity is the main driver of the diabetes epidemic in Qatar, accounting for 57.5% of diabetes cases.[6] Diabetes expenditure is expected to reach nearly one-third of national health expenditure by 2050.[7]

Diabetes prevalence in Qatar is twice the global prevalence.[8] In a recent modeling study, the prevalence of Type 2 DM (T2DM) among Qataris was projected to increase from 17% in 2012 to at least 24% by 2050. National T2DM health expenditure was projected to account for up to 32% of Qatar's total health expenditure by 2050.[9]

Type 2 DM (T2DM), which accounts for 90% of DM cases,[10] is linked to non-modifiable (age, genetics, and sociodemographic factors, etc)[10-13] and modifiable (unhealthy diet, obesity, physical inactivity, tobacco use, and alcohol consumption, etc)[12,14,16, 17,18]

Among the various biochemical markers associated with DM diagnosis and management, glycated hemoglobin A1c (HbA1c) is of utmost importance owing to its utility as a reliable marker to assess timely control over the preceding 2 to 3 months.[19] It is recommended that diabetic patients have their HbA1c levels checked at least two times per year because quantitative and direct relationships have been identified between HbA1c concentration and the risk of diabetic micro vascular complications.[20] Therefore, clinicians use HbA1c test results to guide treatment decisions, and the test has become the cornerstone for assessing diabetes care.[21] The conventional approach for the expression of HbA1c values is percent (%) of total hemoglobin, which is not easily comprehensible for a DM patient with nonmedical background.[22]

A new term in diabetes management, estimated average glucose or eAG has been promoted by the American Diabetes Association (ADA), joining with the European Association for the Study of Diabetes (EASD) and International Diabetes Federation (IDF). American Association of Clinical Chemists has suggested that the correlation (r =0.92) is strong enough to justify reporting both the HbA1c result and an estimated average glucose as eAG is expressed in mg/dl and indicates the three - months control of the average sugar of the patient. This makes it very simple understanding for the patient as both the FBS/PPBS and the eAG are expressed with the same denominator result when a clinician orders the HbA1c test.[23]

In 2008, Nathan et al conducted the International HbA1c-Derived Average Glucose (ADAG) trial, which established a linear dependence between HbA1c and averaged plasma glucose levels, and a simple mathematical equation for the calculation of estimated average glucose (eAG) level using the HbA1c level was introduced.[24] The relationship between HbA1C and eAG is described by the equation 28.7 X A1C – 46.7 = eAG. This equation has been extensively evaluated since then, and citing eAG values with HbA1c laboratory reports has become a common practice. Still most clinical laboratories have not yet started reporting eAG values and a widespread understanding of its utility in the medical fraternity is missing.

OBJECTIVES

1. To determine the statistical correlation between HBA1C with random blood sugar (RBS) values both in diabetic and prediabetic subjects.
2. To determine the statistical correlation between eAG derived from HbA1C using the Nathan's regression equation with RBS both in diabetic and prediabetic subjects.
3. To analyze the significance of eAG as opposed to HbA1C as a marker of long-term glycemic control in DM.

MATERIALS AND METHODS

Study Design

This medical center based retrospective analytical cohort study was conducted at the Clinical Laboratory, Department of Pathology, Yasmed Medical Center, Doha, Qatar.

Sample Selection and Sample Size

The study group was selected from patient reporting to the laboratory for HbA1c estimation. The simple random sampling technique was used to obtain laboratory records of both sexes in the age range of 12 to 70 years presenting as outpatients. The total number of sample was 162.

Data Collection

The random blood glucose and HbA1c levels of 162 patient samples (123 male and 39 female) were included in the study. Blood samples were taken on the same day for the determination of both RBS and HbA1c. The eAG levels (mg/dL) were calculated using the following formula: 28.7 X HbA1c – 46.7. The samples were divided into three groups on the basis of HbA1c levels as group 1: HbA1c greater than or equal to 6.5% (diabetic),group 2: HbA1c 5.7 to 6.4% (prediabetic), and group 3: HbA1c less than 5.7% (non diabetic).[25] Glucose levels were determined using the glucose oxidase method in Thermoscientific analyzer with commercially available kits of same company. HbA1c levels were determined using Spectrophotometry method on Siemens DCA Vantage Analyzer.

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Statistical Analysis

Categorical variables were presented as numbers and percentages, while quantitative data were expressed as means with standard deviations and medians with interquartile ranges. Normality of the data was assessed using the Shapiro-Wilk test, along with an inspection of skewness, kurtosis, and the number of modes. Non-parametric tests were applied where the data were not normally distributed. For comparisons between two groups of non-normally distributed data, the Mann–Whitney U test was used, while the Kruskal-Wallis test was employed for comparisons involving more than two groups. The Wilcoxon signed ranks test was utilized to compare RBS (mg/dL) and eAG (mg/dL). Spearman's rank correlation coefficient was used to evaluate the correlation between RBS (mg/dL) with HbA1c (%) and eAG (mg/dL), revealing a strong positive correlation. Data were analyzed using SPSS version 21.0, with statistical significance set at a p-value of less than 0.05.

RESULTS

A total of 162 patients' data (123 males,39 females) were recorded for the study. Age range was between 12 and 70 years with mean age ± SD was 41.60 ± 9.84 years. The mean ± SD values of RBS, HbA1c and eAG of the population were 167.44 ± 88.74 mg/dl, 7.23 ±2.14 % and 161.20 ± 61.43 mg/dl respectively. (Table 1)

Table 1: Distribution of baseline characteristics of study subjects.

Variable	Parameter	Total	Gender		Difference (95%CI)	Significance
			Male	Female		
Age (Years)	Mean ± SD	41.60 ± 9.84	42.09 ± 8.75	40.05 ± 12.70	2.04 (-2.34 to 6.42)	W = 2991.000, p = 0.020[m]
	Median (IQR)	41.00 (36.25 - 47.00)	41.00 (37.00 - 47.00)	38.00 (32.00 - 42.50)		
Gender	Male	123 (75.93%)	123 (100.00%)	0 (0.00%)	100.00% (-100.00% to 100.00%)	χ^2 = 156.575, p = <0.001[f]
	Female	39 (24.07%)	0 (0.00%)	39 (100.00%)	-100.00% (-100.00% to -100.00%)	χ^2 = 156.575, p = <0.001[f]
RBS (mg/dL)	Mean ± SD	167.44 ± 88.74	167.38 ± 89.62	167.62 ± 87.04	-0.23 (-32.40 to 31.94)	W = 2477.500, p = 0.758[m]
	Median (IQR)	133.00 (105.00 -193.50)	134.00 (106.00 -193.50)	127.00 (98.50 - 225.00)		
HbA1c (%)	Mean ± SD	7.23 ± 2.14	7.20 ± 2.01	7.32 ± 2.54	-0.12 (-1.01 to 0.78)	W = 2518.500, p = 0.639[m]
	Median (IQR)	6.30 (5.70 - 8.30)	6.30 (5.75 - 8.30)	6.20 (5.55 - 8.30)		
eAG (mg/dL)	Mean ± SD	161.20 ± 61.43	160.39 ± 57.58	163.75 ± 73.03	-3.36 (-29.01 to 22.30)	
	Median (IQR)	134.61 (117.39 - 192.01)	134.61 (118.82 - 192.01)	131.74 (113.08 - 192.01)		

[t] = t test, [m] = Mann-Whitney U Test, [f] = Fisher's Exact Test

There was a statistically no significant difference found in all the dependent variables (HbA1c/RBS/eAG) in two independent groups, that is, males and females (Table 2A, Table 2B, Table 2C and Table 2D). It showed females have slightly higher values of HbA1c, RBS, and eAG as compared with males (Figure 1A, Figure 1B and Figure 1C). The study sample was divided into three groups on the basis of HbA1C levels as group 1: HbA1c greater than or equal to 6.5% (diabetic),group 2: HbA1c 5.7 to 6.4% (prediabetic), and group 3: HbA1c less than 5.7% (non diabetic). A nonparametric test applied to the three groups showed a statistically significant difference in their RBS and eAG values(Table 3, Figure 2A,Figure 2B, Figure 3C and Figure 2D).

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Table 2A: Association between Gender and Parameters.

Parameters	Gender		p value
	Male (n = 123)	Female (n = 39)	
Age (Years)***	42.09 ± 8.75	40.05 ± 12.70	0.0201
RBS (mg/dL)	167.38 ± 89.62	167.62 ± 87.04	0.7581
HbA1c (%)	7.20 ± 2.01	7.32 ± 2.54	0.6391
eAG(mg/dL)	160.39 ± 57.58	163.75 ± 73.03	0.6391

***Significant at p<0.05, 1: Wilcoxon-Mann-Whitney U Test, 2: Fisher's Exact Test

Table 2B: Association between Gender and RBS (mg/dL)

RBS (mg/dL)	Gender		Wilcoxon-Mann-Whitney U Test	
	Male	Female	W	p value
Mean (SD)	167.38 (89.62)	167.62 (87.04)	2477.500	0.758
Median (IQR)	134 (106-193.5)	127 (98.5-225)		
Min - Max	80 - 577	82 - 415		

The variable RBS (mg/dL) was not normally distributed in the 2 subgroups of the variable Gender. Thus, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons.

There was no significant difference between the groups in terms of RBS (mg/dL) (W = 2477.500, p = 0.758).

Table 2C: Association between Gender and HbA1C (%).

HbA1c (%)	Gender		Wilcoxon-Mann-Whitney U Test	
	Male	Female	W	p value
Mean (SD)	7.20 (2.01)	7.32 (2.54)	2518.500	0.639
Median (IQR)	6.3 (5.75-8.3)	6.2 (5.55-8.3)		
Min - Max	5 - 14.1	4.7 - 14		

The variable HbA1c (%) was not normally distributed in the 2 subgroups of the variable Gender. Thus, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons.

There was no significant difference between the groups in terms of HbA1c (%) (W = 2518.500, p = 0.639)

Table 2D: Association between Gender and eAG (mg/dl).

eAG (mg/dL)	Gender		Wilcoxon-Mann-Whitney U Test	
	Male	Female	W	p value
Mean (SD)	160.39 (57.58)	163.75 (73.03)	2518.500	0.639
Median (IQR)	134.61 (118.82-192.01)	131.74 (113.08-192.01)		
Min - Max	97.3 - 358.47	88.69 - 355.6		

The variable eAG (mg/dL) was not normally distributed in the 2 subgroups of the variable Gender. Thus, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were used to make group comparisons.

There was no significant difference between the groups in terms of eAG (mg/dL) (W = 2518.500, p = 0.639).

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Figure 1A: Association between Gender and RBS (mg/dL)

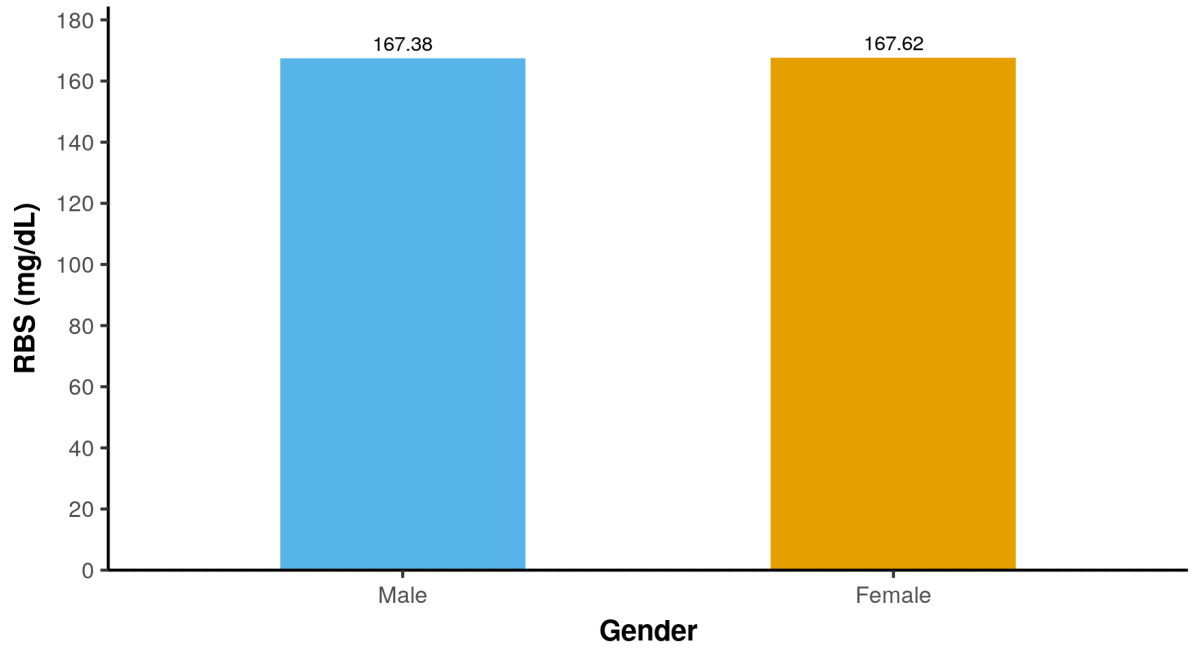

Figure 1B: Association between Gender and HbA1C (%)

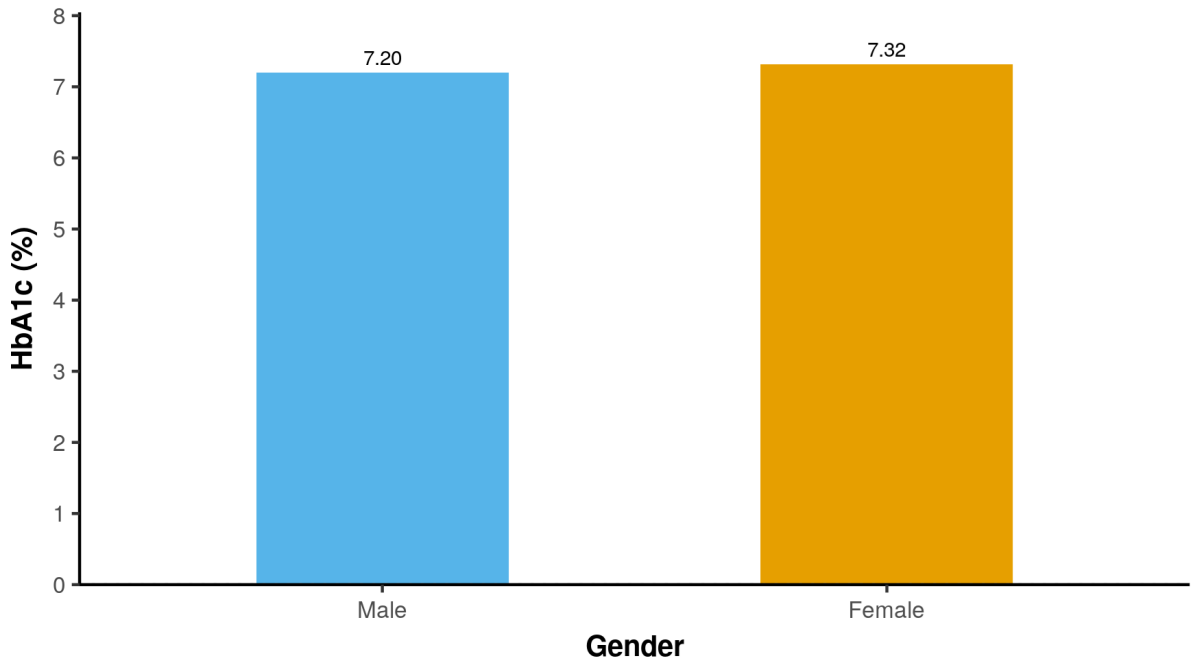

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Figure 1C: Association between Gender and eAG (mg/dl)

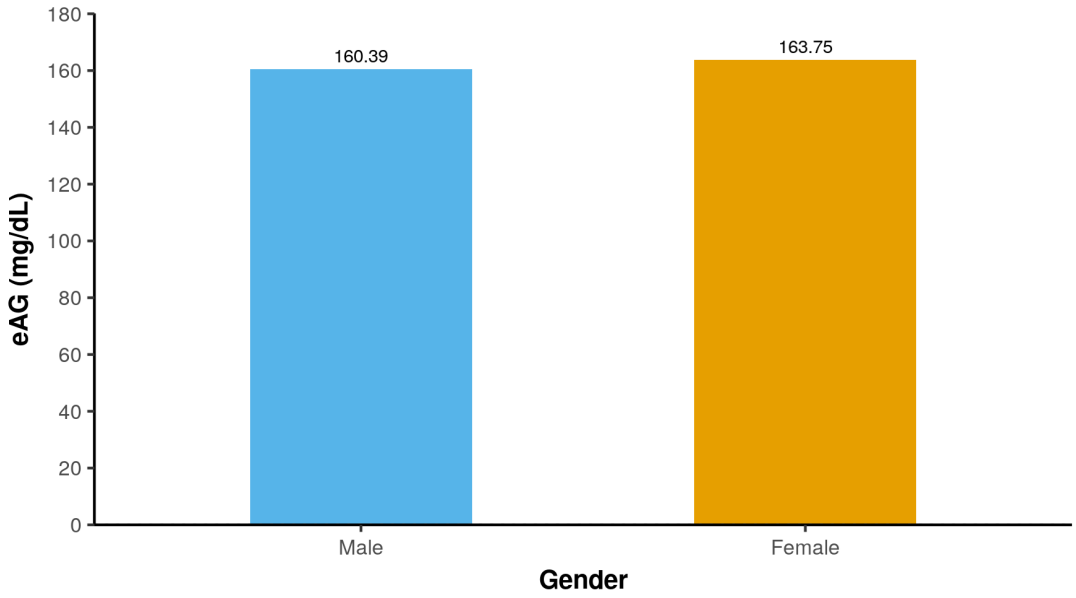

Table 3: Association between HbA1C(%) and Parameters.

Parameter	Metric	Group			Significance	Difference (95% CI), Pairwise Adjusted p Value		
		<5.7%	5.7-6.4%	≥6.5%		<5.7% vs 5.7-6.4%	<5.7% vs ≥6.5%	5.7-6.4% vs ≥6.5%
Age (Years)	Mean ± SD	34.62 ± 8.24	44.74 ± 9.8	43.26 ± 8.98	$\chi2$ = 30.056, p = <0.001[k]	10.12 (5.46 to 14.79), p(adj.) = <0.001	8.64 (4.44 to 12.85), p(adj.)= <0.001	-1.48 (-5.47 to 2.51), p(adj.) = <0.001
	Median (IQR)	36 (29.5 - 38)	42.5 (37.25 - 49.75)	42 (38 - 47)				
Gender	Male	27 (69.23%)	36 (78.26%)	60 (77.92%)	χ^2 = 1.261, p = 0.539[f]	-9.03% (-27.79% to 9.73%)	-8.69% (-25.89% to 8.50%)	0.34% (-14.76% to 15.44%)
	Female	12 (30.77%)	10 (21.74%)	17 (22.08%)		9.03% (-9.73% to 27.79%)	8.69% (-8.50% to 25.89%)	-0.34% (-15.44% to 14.76%)
RBS (mg/dL)	Mean ± SD	100.33 ± 14.96	123.09 ± 27.14	227.92 ± 94.47	$\chi2$ = 101.333, p = <0.001[k]	22.75(-11.9 to 57.41), p(adj.) = 0.269	127.59 (96.3 to 158.88), p(adj.) = 0.000	104.84(75.17 to 134.5), p(adj.) = 0.000
	Median (IQR)	97 (88.5 - 106.5)	113 (105 - 132.75)	203 (155 - 290)				
HbA1c (%)	Mean ± SD	5.31 ± 0.25	5.98 ± 0.19	8.94 ± 1.96	$\chi2$ = 137.967, p = <0.001[k]	0.67 (-0.03 to 1.38), p(adj.) = 0.064	3.63 (2.99 to 4.26), p(adj.) = 0.000	2.95 (2.35 to 3.56), p(adj.) = 0.000
	Median (IQR)	5.3 (5.2 - 5.5)	6 (5.8 - 6.1)	8.4 (7.3 - 10)				
HbA1C	<5.7%	39 (100.00%)	0(0.00%)	0(0.00%)	χ^2 = 162.000, p = <0.001[f]	100.00% (100.00%to 100.00%)	100.00% (100.00%to 100.00%)	0.00% (0.00% to 0.00%)
	5.7-6.4%	0(0.00%)	46 (100.00%)	0(0.00%)		-100.00% (-100.00% to100.00%)	0.00% (0.00% to 0.00%)	100.00% (100.00% to 100.00%)
	≥6.5%	0(0.00%)	0(0.00%)	77 (100.00%)		0.00% (0.00% to 0.00%)	-100.00% (-100.00% to -100.00%)	-100.00% (-100.00% to -100.00%)
eAG (mg/dL)	Mean ± SD	106.2 ± 7.3	125.56 ± 5.54	210.35 ± 56.39	$\chi2$ = 137.967, p = <0.001[k]	19.36 (-0.86 to 39.58), p(adj.) = 0.064	104.14 (85.89 to 122.4), p(adj.) = 0.000	84.78 (67.47 to 102.1), p(adj.) = 0.000
	Median (IQR)	105.91 (103.04 - 111.65)	126 (120.26 - 128.87)	194.88 (163.31 - 240.8)				

[á] = One-Way ANOVA with Tukey's HSD test, [k] = Kruskal-Wallis Test with Dunn Test, [f] = Fisher's Exact Test

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Figure 2A: Association between HbA1c and Age (Years).

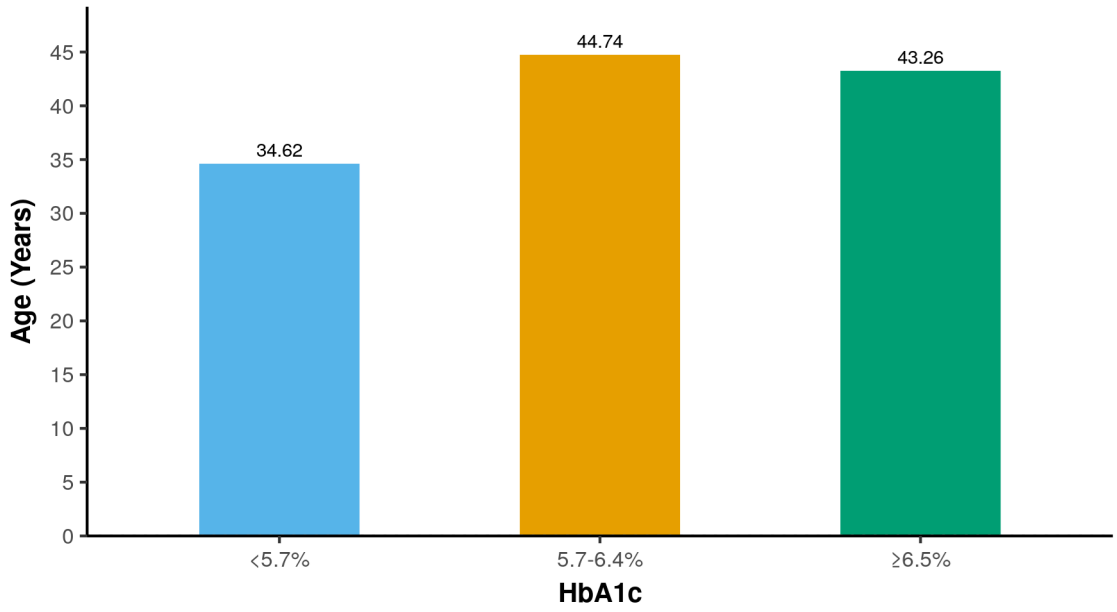

Figure 2B: Association between HbA1c and Gender.

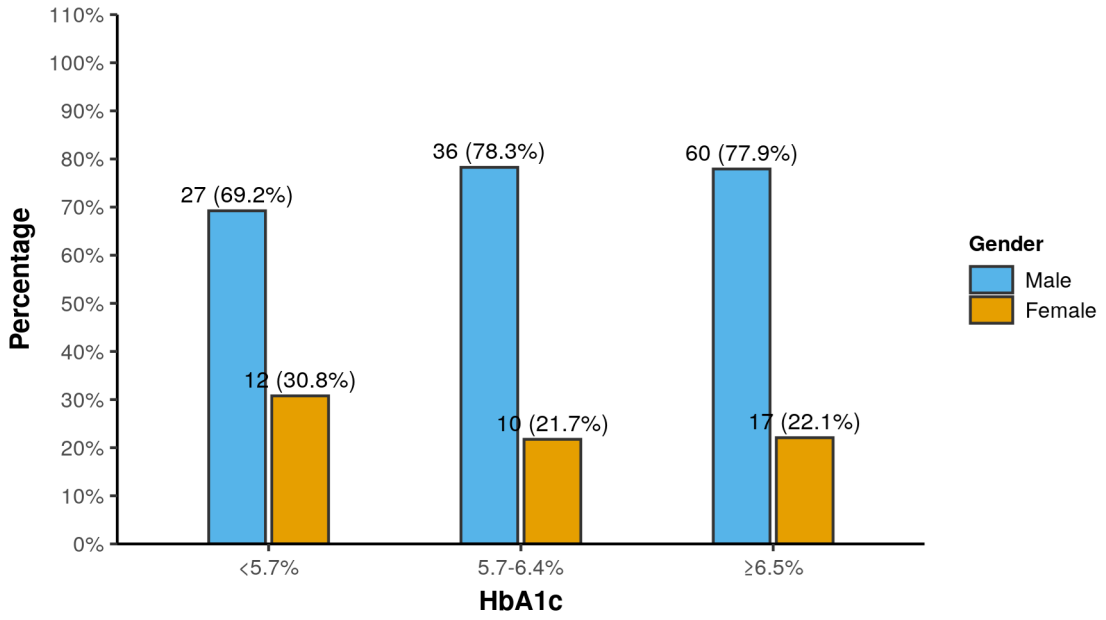

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Figure 2C: Association between HbA1C(%) and RBS(mg/dl)

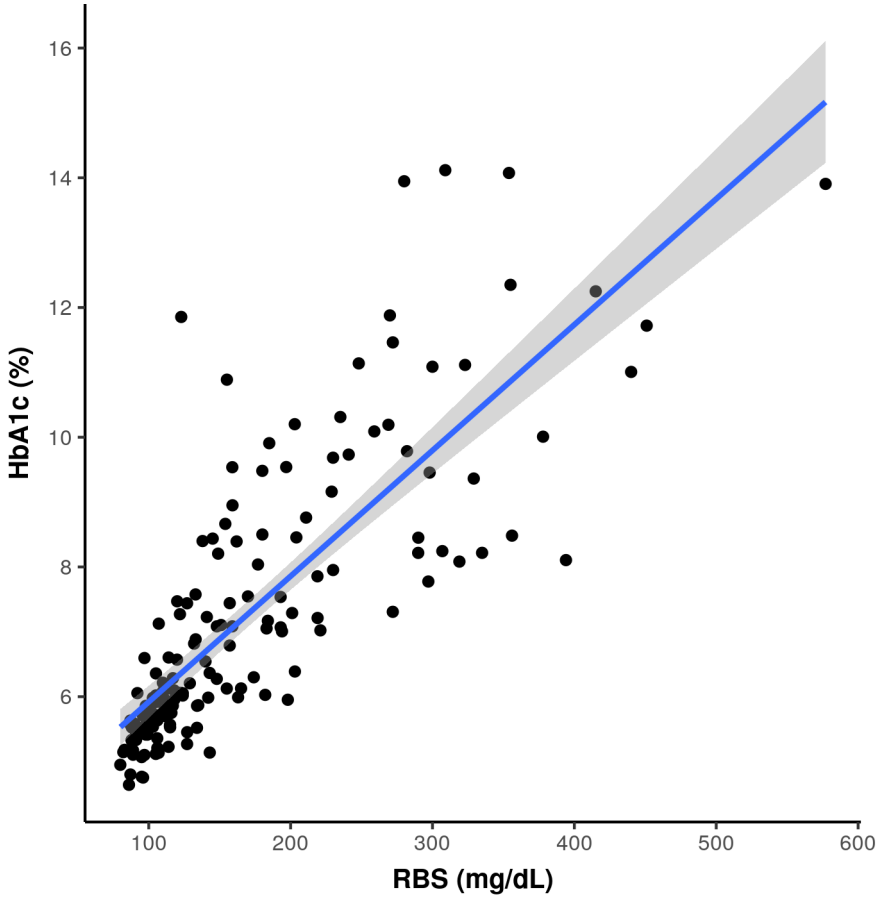
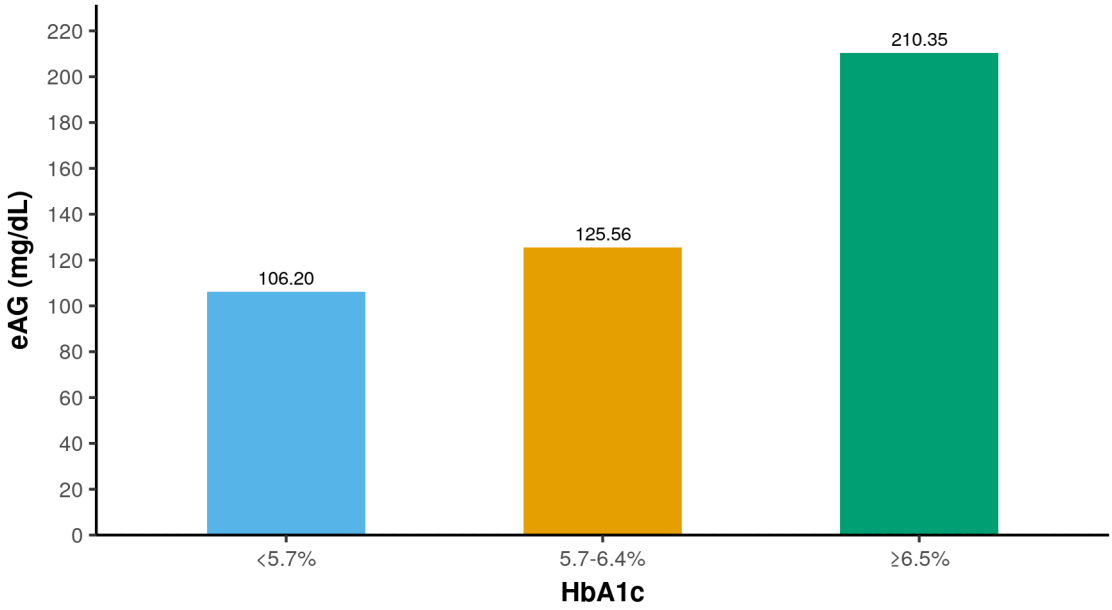


Figure 2D: Association between HbA1c and eAG (mg/dL)



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As show in (Table 4B and Figure 2C) the variable RBS (mg/dL) was not normally distributed in the 3 subgroups of the variable HbA1c. Thus, non-parametric tests (Kruskal Wallis Test) were used to make group comparisons.There was a significant difference between the 3 groups in terms of RBS (mg/dL) ($\chi 2 = 101.333$, p = <0.001), with the median RBS (mg/dL) being highest in the HbA1c: ≥6.5% group.

As show in (Table 4C and Figure 3) there was a strong positive correlation between eAG (mg/dL) and RBS (mg/dL), and this correlation was statistically significant (rho = 0.86, p = <0.001). For every 1 unit increase in eAG (mg/dL), the RBS (mg/dL) increases by 1.16 units. RBS (mg/dL) = 19.76 + 1.16*eAG (mg/dL).Conversely, for every 1 unit increase in RBS (mg/dL), the eAG (mg/dL) increases by 0.56 units. eAG (mg/dL) = 68.03 + 0.56*RBS (mg/dL).

Table 4A : Association between RBS(mg/dl) and Parameters.

Parameters	RBS (mg/dL)	p value
Age (Years)***	Correlation Coefficient (rho) = 0.24	0.0021
Gender		0.7582
Male	167.38 ± 89.62	
Female	167.62 ± 87.04	
HbA1c (%)***	Correlation Coefficient (rho) = 0.86	<0.0011
HbA1c***		<0.0013
<5.7%	100.33 ± 14.96	
5.7-6.4%	123.09 ± 27.14	
≥6.5%	227.92 ± 94.47	
eAG (mg/dL)***	Correlation Coefficient (rho) = 0.86	<0.0011

***Significant at p<0.05, 1: Spearman Correlation, 2: Wilcoxon-Mann-Whitney U Test, 3: Kruskal Wallis Test

Table 4B: Association between RBS(mg/dl) and HbA1C(%).

RBS (mg/dL)	HbA1c			Kruskal Wallis Test	
	<5.7%	5.7-6.4%	≥6.5%	χ2	p value
Mean (SD)	100.33 (14.96)	123.09 (27.14)	227.92 (94.47)	101.333	<0.001
Median (IQR)	97 (88.5-106.5)	113 (105-132.75)	203 (155-290)		
Min - Max	80 - 143	92 - 203	97 - 577		

Pairwise Comparison of Subcategories of HbA1c	Adjusted P Value
<5.7% - ≥6.5%	<0.001
<5.7% - 5.7-6.4%	0.007
≥6.5% - 5.7-6.4%	<0.001

Post-Hoc pairwise tests for Kruskal-Wallis test performed using Dunn Test method with Sidak correction.

Table 4C : Association between RBS(mg/dl) and eAG(mg/dl).

Correlation	Spearman Correlation Coefficient	P Value
eAG (mg/dL) vs RBS (mg/dL)	0.86 (95%CI: 0.81 to 0.89)	<0.001

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Figure 3: Association between RBS(mg/dl) and eAG (mg/dl)

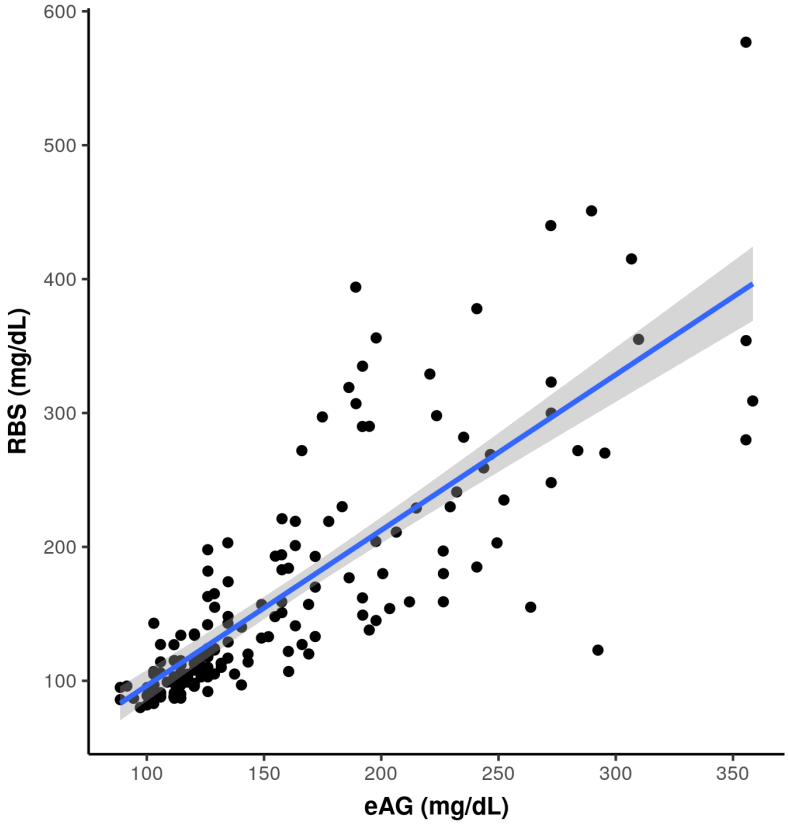
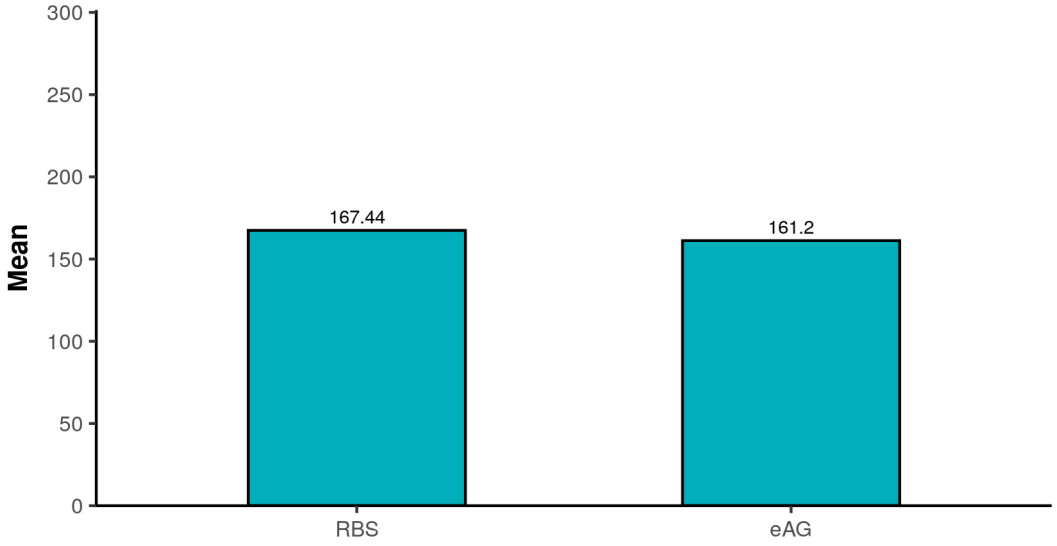


Table 5 and Figure 4 depicts these mean values of RBS and eAG show statistically significant difference ($p < 0.001$)

Table 5: Descriptive statistics of glycemic parameter of study subjects.

Glycemic parameter	Mean (SD)	Median (IQR)	Range	p-value
Random Blood Sugar(RBS)	167.44 (88.74)	133 (105-200.25)	80 - 577	< 0.001
Estimated average glucose(eAG)	161.20 (61.43)	134.61 (117.39-192.01)	88.69 - 358.47	< 0.001

Figure 4: Descriptive statistics of random blood sugar (mg/dL) and estimated average glucose (mg/dL) of study subject.



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DISCUSSION

One limitation often associated with HbA1c is the reporting units of mmol/mol and %, which differs from the usual units of blood glucose monitoring, that is, mg/dL, often creating a confusing situation for the patients as well as clinicians for comprehension.[26]

To overcome these limitations, international bodies including the American Diabetes Association and the International Diabetes Federation proposed a mathematical expression termed eAG, which facilitates comprehension of HbA1c values in units parallel to self-monitoring.[27] Various guidelines recommend reporting eAG with every HbA1c report; however, it is not widely practiced by the majority of laboratories, and advocacy is required regarding its use based on evaluation in the local population.[28] With this perspective in mind, we planned to study the association between RBS and eAG in a cohort of subjects.

This is the study done in Qatari and Non-Qatari for both diabetic and non diabetic groups. In this study, we found statistically significant correlation of RBS with eAG in total study subjects and diabetics (poorly controlled and fairly controlled groups) but no significant correlation was found between eAG and RBS in nondiabetic and prediabetic groups, which is similar to Kim et al findings.[29] We also found in our study that RBS values cannot be used interchangeably with eAG values. Most of the below mentioned studies highlighted an association between eAG/HbA1c and RBS/fasting blood sugar(FBS)) /postprandial blood sugar (PPBS)/self-monitored mean blood glucose (MBG) in diabetics, this association had not been checked in diabetic, prediabetic, and nondiabetic subgroups separately, possibly due to the study design which only included diabetics.

A study by Kariyawasan found a significant statistical correlation in both fasting blood sugar (FBS) and post prandial blood sugar (PPBS) with eAG in the groups of patients with moderately poor control. In those with markedly poor control the both fasting blood sugar (FBS) did not show a statistical correlation with eAG, as opposed to the PPBS.[30] Bozkaya et al have found that a strong positive correlation exists between fasting blood sugar (FBS) levels and estimated average blood glucose levels (r ¼ 0.757, p ¼ 0.05)[26].Rosediani et al revealed that both post prandial blood sugar (PPBS) and fasting blood sugar (FBS) correlated significantly with HbA1c but post prandial blood sugar (PPBS) showed better correlation with HbA1c than fasting blood sugar (FBS) (r ¼ 0.604 vs. 0.575)[31]. Mahato et al found statistically significant correlation of eAG with FBS (r ¼ 0.61, p < 0.001) and post prandial blood sugar (PPBS) levels (r ¼ 0.65, p < 0.001)[32]. Kim et al found that fasting blood sugar (FBS)showed a moderate correlation with eAG (r ¼ 0.672, p < 0.001) in all subjects but when diabetic and nondiabetic subjects were divided into subgroups according to the fasting blood sugar (FBS) level, the correlation between eAG and fasting blood sugar (FBS) decreased in both subgroups as the fasting blood sugar (FBS) level decreased.[29] Guan et al found the relationship between HbA1c and fasting blood sugar (FBS) changed according to the different fasting blood sugar (FBS) ranges.[33] Azim et al found direct correlation between HbA1c and RBS in diabetics.[34] Nkoana and Khine found a positive correlation between self-monitored MBG and HbA1c in all participants (R2 ¼ 0.69, p < 0.0001) but clinically significant differences between mean blood glucose(MBG) and eAG values.[35]

CONCLUSION

In conclusion,in poorly controlled diabetic care-sensitive group, eAG can serve as easily comprehensible way to determine average glucose levels with the same reporting units for self-blood glucose monitoring. This will supplement clinicians to facilitate care and counsel patients in a more convincing way. Moreover, it can serve as a useful measure for clinical laboratories of government hospitals in developing countries to enhance the quality of reporting at no added substantial cost.

We recommend that the use of eAG should be validated prior to implementation in clinical practice. It would be ideal to evaluate the relationship between average glucose and HbA1c in each individual patient in order to provide more personalised diabetes care.

REFERENCES

1. IDF Diabetes Atlas. Tenth edition 2021. https://diabetesatlas.org/idfawp/resource-files/2021/07/IDF_Atlas_10th_Edition_2021.pdf. Accessed on December 6, 2021.

2. https://www.psa.gov.qa/en/statistics1/StatisticsSite/pages/population.aspx

3. Supreme Council of Health. Qatar stepwise report 2012: chronic disease risk factor surveillance, 2013. Available: http://www.who.int/ chp/steps/qatar/en/

4. Al-Thani MH, Al-Mutawa KA, Alyafei SA. Characterizing epidemiology of prediabetes, diabetes, and hypertension in Qataris: a cross-sectional study, 2021.

5. Awad SF, Toumi A, Al-Mutawaa KA, et al. Type 2 diabetes epidemic and key risk factors in Qatar: a mathematical modeling analysis. BMJ Open Diabetes Res Care 2022;10.

The Journal of Clinical Pathology (ISSN 2995-8598)

6. Awad SF, O'Flaherty M, Critchley J, Abu-Raddad LJ. Forecasting the burden of type 2 diabetes mellitus in Qatar to 2050: A novel modeling approach. Diabetes Research and Clinical Practice 2018;137:100-8.

7. Awad SF, Dargham SR, Toumi AA, et al. A diabetes risk score for Qatar utilizing a novel mathematical modeling approach to identify individuals at high risk for diabetes. Scientific Reports 2021;11:1-10.

8. International Diabetes Federation: IDF Diabetes Atlas. Eighth edition. Brussels, Belgium (Available at: http://www.diabetesatlas.org; accessed on December 2017). 2017.

9. International Diabetes Federation. IDF diabetes atlas. ninth edition 2019, 2019. Available: https://www.diabetesatlas.org/upload/ resources/2019/IDF_Atlas_9th_Edition_2019.pdf [Accessed 24 Nov 2019].

10. Santosa A, Wall S, Fottrell E, et al. The development and experience of epidemiological transition theory over four decades: a systematic review. Glob Health Action 2014;7:23574.

11. Hu FB. Globalization of diabetes: the role of diet, lifestyle, and genes. Diabetes Care 2011;34:1249–57.

12. World Health Organization. The global burden of disease: 2004 update. Switzerland: World Health Organization, 2008. http://www. who.int/healthinfo/global_burden_disease/GBD_report_2004update_ full.pdfwww.who.int/publications/i/item/9789241563710

13. Pan A, Wang Y, Talaei M, et al. Relation of active, passive, and quitting smoking with incident type 2 diabetes: a systematic review and meta- analysis. Lancet Diabetes Endocrinol 2015;3:958–67.

14. Abdullah A, Peeters A, de Courten M, et al. The magnitude of association between overweight and obesity and the risk of diabetes: a meta-analysis of prospective cohort studies. Diabetes Res Clin Pract 2010;89:309–19.

15. Tuso P. Prediabetes and lifestyle modification: time to prevent a preventable disease. Perm J 2014;18:88–93.

16. Joshi RD, Dhakal CK. Predicting type 2 diabetes using logistic regression and machine learning approaches. Int J Environ Res Public Health 2021;18:7346.

17. Gupta S, Jain U, Chauhan N. Laboratory diagnosis of HbA1c: a review. J Nanomed Res 2017;5:00120

18. UK Prospective Diabetes Study (UKPDS) Group. Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet 1998;352(9131):837–853.

19. American Diabetes Association. Standards of medical care in diabetes–2010. Diabetes Care 2010;33(Suppl 1):S11–S61

20. Wang D, Chen Y. Usefulness of estimated average glucose (eAG) in glycemic control and cardiovascular risk reduction. Clin Biochem 2020;84:45–50

21. Statements P. Standards of medical care in diabetes - 2012. Diabetes Care. 2012;35(SUPPL. 1).

22. Nathan DM, Kuenen J, Borg R, Zheng H, Schoenfeld D, Heine RJA1c- Derived Average Glucose Study Group. Translating the A1C assay into estimated average glucose values. Diabetes Care 2008;31 (08):1473–1478.

23. https://diabetesjournals.org/care/article/46/10/e151/153425/Guidelines-and-Recommendations-for-Laboratory#4675764.

24. Bozkaya G, Ozgu E, Karaca B. The association between estimated average glucose levels and fasting plasma glucose levels. Clinics (São Paulo) 2010;65(11):1077–1080.

25. Law GR, Gilthorpe MS, Secher AL, et al. Translating HbA 1c measurements into estimated average glucose values in pregnant women with diabetes. Diabetologia 2017;60(04):618–624.

26. Young IS. Counterpoint: The reporting of estimated glucose with hemoglobin A1c. Clin Chem 2010;56(04):547–549.

27. Kim HY, Lee SY, Suh S, Kim JH, Lee MK, Park HD. The relationship between estimated average glucose and fasting plasma glucose. Clin Chem Lab Med 2013;51(11):2195–2200.

28. Kariyawasan CC. The association between Hba1c-derived estimated average glucose (eAG) with fasting blood sugar (FBS) and post prandial blood sugar (PPBS) in patients with type 2 diabetes in a cohort of patients in a tertiary care hospital in Sri Lanka. Clinics (São Paulo) 2021;3(02):x

The Journal of Clinical Pathology (ISSN 2995-8598)

29. Rosediani M, Azidah AK, Mafauzy M. Correlation between fasting plasma glucose, post prandial glucose and glycated haemoglobin and fructosamine. Med J Malaysia 2006;61(01):67–71.

30. Mahato RV, Nepal AK, Gelal B, et al. Association of estimated average glucose (eAG) with fasting, postprandial and average of fasting and PP serum glucose levels. Journal of Nepal Association for Medical Laboratory Sciences. 2012;11(01):14–18.

31. Guan X, Zheng L, Sun G, et al. The changing relationship between HbA1c and FPG according to different FPG ranges. J Endocrinol Invest 2016;39(05):523–528.

32. Azim W, Omair M, Khan MQA, Shaheen N, Azim S. Correlation between glycated haemoglobin and random plasma glucose levels for the screening of diabetes mellitus. Int J Pathol 2010;8(02):59–62.

33. Nkoana MK, Khine AA. Correlation between self-monitored mean blood glucose and average plasma glucose estimated from glycated haemoglobin in patients attending the diabetes clinic at Dr.George Mukhari Academic Hospital, Pretoria, South Africa. S Afr Med J 2020;110(05):416–421