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Original article

Correlation between glycemic gap and short-term adverse outcome in diabetic patients undergoing elective PCI

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ABSTRACT:

Background: Acute hyperglycemia is a common occurrence in individuals who present to the emergency department. Hyperglycemia may have a well-established predictive effect in those with acute coronary syndrome (ACS) in individuals without diabetes; however, this is still a topic of debate in diabetic patients. Our goal was to evaluate the correlation between the glycemic gap and adverse coronary vascular events in diabetic individuals who were undergoing elective percutaneous coronary intervention (PCI).

Methods: Cross-sectional research involving 140 diabetic individuals who underwent elective PCI divided the participants in accordance with the glycemic gab in to: Group (1): cases with glycemic gap ≥42 mg/dl, Group (2): cases with glycemic gap < 42 mg/dl. All participants followed up for one year for possible coronary complications and MACE development.

Results: Our findings revealed no statistically significant variations in the affected coronary arteries between the two groups. Group I had a statistically significant longer hospital stay, higher incidence of major adverse cardiac events, Intensive Care Unit (ICU) admissions, in-stent restenosis, in-stent thrombosis, and mortality rates. Glycemic gap can significantly predict major adverse cardiac events (MACEs) in type 2 diabetes mellitus (T2DM) patients who underwent elective PCI (AUC= 0.615, P value =0.035) at cut off 42 mg/dl, 69.70% sensitivity, 54.21% specificity, 31.9% PPV and 85.3% NPV.

Conclusions: Glycemic gap is a simple method that can be applied easily to all diabetic cases who undergo elective PCI, to predict the short term cardiovascular adverse outcome and so should be tightly controlled before elective PCI.

Keywords: Glycemic Gap, Diabetic Patients, Elective PCI, Short-Term Adverse, Outcomes

INTRODUCTION

Ischemic heart disease (IHD) is expected to cause 9 million fatalities and afflicts 126 million individuals, in accordance with the Global Burden of Disease study. This condition has a substantial impact on the global public health issues and medical expenses [1].

Acute hyperglycemia frequently occurs in individuals with acute coronary syndrome presenting to the emergency room, regardless of their diabetes status. The initial blood glucose level upon admission after an acute myocardial infarction serves as an independent predictive factor for long-term mortality in patients, regardless of their diabetes status [2].

Hyperglycemia is recognized to have a significant predictive impact in non-diabetic people with acute coronary syndrome (ACS), but its applicability to diabetic individuals is still debatable, especially when considering the short-term outcomes [3].

In accordance with the evidence, stress hyperglycemia is characterized by reduced glycogenolysis, enhanced lipolysis, increased

gluconeogenesis, and increased insulin resistance in organs and tissues. These advancements are enabled by elevated levels of the pro-inflammatory cytokines cortisol and glucagon in the bloodstream, as well as increased oxidative stress [4].

Hyperglycemia is the most prevalent symptom in diabetics, irrespective of the presence of stressful events, as a result of a variety of factors, including inadequate glycemic control. The chronic dysfunction, injury, and failure of a variety of organs, such as the heart, nerves, eyes, kidneys, and blood vessels, are among the enduring consequences of hyperglycemia [5].

The negative consequences stem from the patient's blood glucose levels upon admission to the hospital. Research suggests that outcomes are significantly impacted by blood glucose levels that exceed previous levels. This is the consequence of increased levels of cytokines and counter-regulatory hormones that influence glucose metabolism [6].

The prognosis of individuals with acute illness can be predicted using a variety of scores and indications. Glucose levels in the blood and variations in blood sugar are among the few of these. It is hypothesized that measuring the rise in glucose levels beyond the current average will help with the evaluation of stress levels during acute illness (HbA1c) [2].

The glycemic gap serves as a refined prognostic marker by measuring the difference between a patient's admission glucose and their baseline average glucose estimated from HbA1c. This allows for better identification of stress-induced hyperglycemia, which is linked to worse outcomes, especially in diabetic patients undergoing acute coronary events [7]. Many individuals may already have elevated blood glucose levels, which may result in a lack of correlation between stress levels and the measurement of entrance glucose in diabetic patients. The glycemic gap factors in the HbA1c to disprove this mistake. Anemia, hemoglobinopathies, and other conditions may affect HbA1c, which is not affected by stress or infection [7]. The objective of this research is to determine whether diabetic individuals who have elective PCI are more likely to experience unfavorable coronary vascular events and whether there is a correlation between the glycemic gap and these events.

METHODS

Ethical approval: The research was approved by the Zagazig University committee for ethical approval (IRB reference number 10005/19-10-2022). Informed written consent was obtained from all participants prior to their inclusion in the study, in accordance with the Declaration of Helsinki.

Study population

A cross-sectional investigation was performed on 140 diabetic individuals who received elective PCI at the Department of Cardiology, Zagazig University Hospitals, and the National Heart Institute in Egypt, throughout the study period from April 2022 to April 2024. All diabetic patients, whether type I or type II, undergoing elective PCI were included in our study. Written consent was obtained from all individuals. We excluded Individuals with arrhythmias or valvular heart disorders. Patients were diagnosed with ACS, and history of terminal renal and hepatic illness. In the present investigation, individuals declined to participate.

The patients were classified consistent with the glycemic gab into 2 groups as follow g**roup (1):** patients with glycemic gap ≥ 42 mg/dd, g**roup (2):** Patients with glycemic gap < 42 mg/dl.

All the study participants were subjected to: complete history, complete general and local cardiac examination, Laboratory investigations (Complete blood picture, serum creatinine, liver enzymes HbA1 and complete Lipid profile), Standard 12-leads ECG, complete standard Echocardiography, Coronary angiography and calculating the glycemic gap and follow up one year for coronary complications and MACE development.

The glycemic gap was calculated as the difference between the admission fasting blood glucose (FBG) level and the estimated average glucose (eAG) derived from HbA1c using the following formula:

Glycemic Gap = Admission FBG (mg/dL) – eAG (mg/dL)

The eAG was calculated from HbA1c based on the formula:

eAG (mg/dL) = (28.7 × HbA1c) – 46.7

Blood samples for fasting glucose were drawn, after an overnight fast of at least 8 hours. HbA1c was measured from the same blood sample or collected within the first 24 hours of admission. All samples were analyzed at the hospital's central laboratory using standardized methods.

Sample size

Assuming the frequency of ICU mortality was 9.5% vs 29.5% in those the glycemic gap <80 vd glycemic gap>80. At 80% power and 95 % CI, the estimated sample will be 140 cases.

$$n = \frac{\left[Z_{1-\alpha/2}\sqrt{2P(1-P)} + Z_{1-\beta}\sqrt{P_1(1-P_1)+P_2(1-P_2)}\right]^2}{(P_1-P_2)^2}$$

Where:

- **P1** = Proportion in group 1 (e.g., mortality rate with glycemic gap <80) = 0.095
- **P2** = Proportion in group 2 (e.g., mortality rate with glycemic gap >80) = 0.295
- **P** = Pooled proportion
- **α** = 0.05 (significance level for 95% confidence)
- **β** = 0.20 (for 80% power)
- **n** = Sample size per group

Statistical Analysis

Statistical Package for the Social Sciences was utilized to review, code, and tabulate the collected data (IBM Corp., 2017, Release). Version 25.0 of IBM SPSS Statistics for Windows was published by IBM Corp. in Armonk, New York. A p-value that is below 0.05 on a 95% confidence interval is considered significant.

Potential confounding factors such as renal impairment, active infection, and concurrent medication use (e.g., corticosteroids, insulin, or antiplatelet agents) were assessed at baseline through clinical examination, laboratory testing, and medical record review. These variables were included in the multivariate logistic regression model to control for their influence on the relationship between glycemic gap and adverse outcomes. Specifically, serum creatinine, white blood cell count, and detailed medication history were analyzed as covariates. The adjusted odds ratios (aORs) with 95% confidence intervals were reported to evaluate the independent effect of glycemic gap on short-term adverse outcomes after accounting for these confounders. ROC curve analysis was performed to evaluate the discriminative ability of the glycemic gap in predicting short-term adverse outcomes. The optimal cutoff value for the glycemic gap was determined using the Youden Index, which identifies the point on the ROC curve that maximizes the sum of sensitivity and specificity. This cutoff was subsequently used in subgroup comparisons and logistic regression modeling.

RESULTS:

The age, sex, and BMI of the patient categories do not appear to be significantly different. **Table 1**

With regard to the influenced coronary arteries, there are statistically non-significantly differences between the two categories. **Table 2**

Patients in the GLYCEMIC GAP ≥42 group had a statistically significantly longer hospital stay. The major adverse cardiac events (MACEs) prevalence was also statistically significantly elevated in the GLYCEMIC GAP ≥42 group. Additionally, ICU admissions were more frequent in the glycemic gap ≥42 group. The prevalence of in-stent restenosis and in-stent thrombosis was detected to be statistically significantly higher in the GLYCEMIC GAP ≥42 group. Additionally, mortality rates were statistically significantly elevated in this demographic. **Table 3**

The logistic regression analysis for the development of (MACEs) reveals that the GLYCEMIC GAP is a MACEs significant indicator in both univariate and multivariate models, confirming that the GLYCEMIC GAP is an independent and significant predictor of MACEs, Other variables such as age, sex, smoking, hypertension, hemoglobin levels, HbA1c, and glucose on admission were not

significant predictors in the multivariate model. Despite the fact that cholesterol levels were significant in the univariate analysis, they did not maintain their significance in the multivariate model. **Table 4**

Glycemic gap can significantly predict MACEs in type 2 diabetes mellitus (T2DM) patients who underwent elective PCI (AUC= 0.615, P value =0.035) at cut off 42 mg/dl, 69.70% sensitivity, 54.21% specificity, 31.9% PPV and 85.3% NPV. **Table 5; Figure 1**

Table 1: Demographic data of the groups studied

		Glycemic gap <42 (n=68)	**Glycemic gap ≥42 (n=72)**	**P value**
Age (years)	**Mean ± SD**	63.78 ± 8.47	65.14 ± 8.76	0.353
	Range	52 – 78	50 - 79	
Sex	**Male**	42 (61.76%)	47 (65.28%)	0.666
	Female	26 (38.24%)	25 (34.72%)	
BMI (kg/m2)	**Mean ± SD**	30.07 ± 4.78	29.99 ± 5.12	0.924
	Range	19.28 - 40.68	21.13 - 43.15	

Table 2: Coronary angiography findings of the studied groups

		Glycemic gap <42 (n=68)	**Glycemic gap ≥42 (n=72)**	**P value**
LAD		40 (58.82%)	50 (69.44%)	0.190
RCA		50 (73.53%)	46 (63.89%)	0.219
LCx		46 (67.65%)	45 (62.5%)	0.523
Number of diseased vessels	**Single vessel**	11 (16.18%)	15 (20.83%)	0.757
	Double vessel	46 (67.65%)	45 (62.5%)	
	Triple-vessel	11 (16.18%)	12 (16.67%)	
Ectasia		6 (8.82%)	10 (13.89%)	0.346

Table 3: Outcomes of the studied groups

		Glycemic gap <42 (n=68)	Glycemic gap ≥42 (n=72)	P value
Hospital stay (days)	**Mean ± SD**	11.84 ± 4.79	15.61 ± 5.71	**<0.001***
	Range	4 - 20	5 - 25	
MACEs		10 (14.71%)	23 (31.94%)	**0.016***
ICU admission		13 (19.12%)	25 (34.72%)	**0.038***
Acute heart failure		16 (23.53%)	22 (30.56%)	0.350
In-stent restenosis		4 (5.88%)	13 (18.06%)	**0.037***
In-stent thrombosis		2 (2.94%)	10 (13.89%)	**0.032***
MI		2 (2.94%)	5 (6.94%)	0.443
Urgent revascularization (CABG)		2 (2.94%)	4 (5.56%)	0.681
Stroke		0 (0%)	2 (2.78%)	0.497
Mortality		1 (1.47%)	9 (12.5%)	**0.018***

Table 4: Logistic regression analysis for the MACEs development

	Univariate			**Multivariate**		
	OR	**95% CI**	**P value**	**OR**	**95% CI**	**P value**
Age	0.975	0.931 – 1.021	0.280	0.968	0.921 – 1.017	0.194
Sex	1.004	0.446 – 2.259	0.993	0.911	0.293 – 2.830	0.872
Smoking	1.800	0.817 – 3.963	0.145	2.286	0.957 – 5.460	0.063
HTN	1.369	0.426 – 4.398	0.597	1.585	0.427 – 5.875	0.491
Glycemic gap	2.722	1.182 – 6.270	**0.019***	8.926	1.125 – 70.839	**0.038***
Hb	1.066	0.810 – 1.402	0.649	1.082	0.751 – 1.561	0.672
HbA1c	1.295	0.846 – 1.983	0.234	0.793	0.006 – 106.489	0.926
Glucose on admission	1.009	0.996 – 1.023	0.180	0.980	0.827 – 1.162	0.820
Cholesterol	1.011	1.002 – 1.021	**0.021***	1.008	0.997 – 1.018	0.145

Table 5: ROC curve analysis of glycemic gap in prediction of MACEs

	Cut-off	**AUC**	**Sensitivity**	**Specificity**	**PPV**	**NPV**	**P value**
Glycemic gap (mg/dl)	42	0.615	69.70	54.21	31.9	85.3	**0.035***

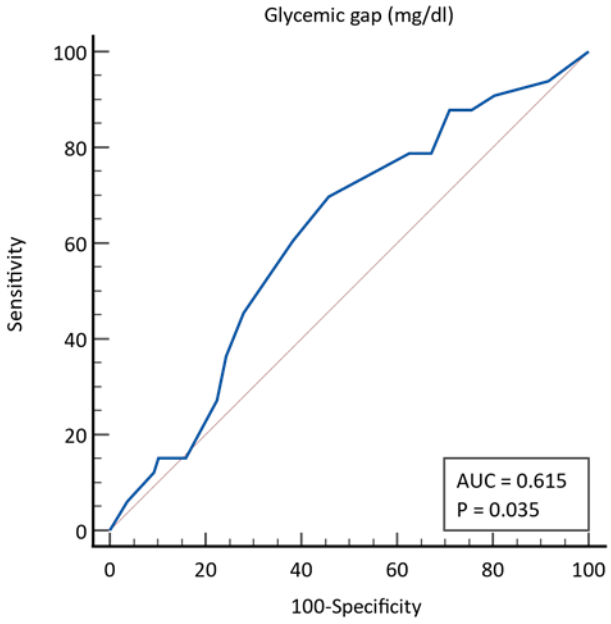

Figure 1: ROC curve of glycemic gap in prediction of MACEs

DISCUSSION

Acute myocardial infarction symptoms may manifest as hyperglycemia in individuals admitted to the emergency room. Regardless of diabetes diagnosis, the admission blood glucose level throughout acute myocardial infarction is a strong predictor of long-term death in individuals [2].

Stress reactions and/or underlying abnormal glucometabolic conditions might be indicated by elevated blood glucose levels. Acute myocardial infarction caused by hyperglycemia is related with an inflammatory and prothrombotic condition, reduced myocardial contractility, and increased short- and long-term mortality rates [9]. This research aimed to elucidate the relationship between the glycemic gap and unfavorable coronary vascular events in patients undergoing elective coronary PCI, thereby facilitating optimal management through stringent control of the glycemic gap.

In our investigation, age, sex, and BMI exhibited no significant differences between the examined groups. This finding aligns with Liao et al. [10], who demonstrated that In diabetic males, a heightened glycemic gap (> 42 mg/dL) was more prevalent than in females; additionally, there was no significant age disparity between the investigated groups [10]. Donagaon and Dharmalingam enlisted 200 patients in their study, of whom 64.5% (129) were male. Additionally, 62% were between the age range of 51 to 70 years. The results showed that there were no significant variations in the distribution of the Glycemic gap depending on gender (P = 0.165), age (P = 0.418), lifestyle (P = 0.465), or primary diagnosis (P = 0.733) [11].

Moreover, Jensen et al. disclosed that the mean age of the total patient cohort was 62.6 years, with 29.5% of the patients being female. Individuals with elevated GAP mean values were older, leaner, exhibited a more rapid heart rate, presented with anterior ST elevation or left bundle branch block, had a worse Killip classification, and showed higher transrectal ultrasound assessments (TRS) compared to those with low GAP mean values. Furthermore, they had a higher likelihood of being female and having hypertension, diabetes mellitus, and a prior history of stroke. Blood glucose levels, mean blood glucose level (MGL), coefficient of variation (CV), standard deviation (SD), and GAPadm (the distinction between ABG and A1c-derived average glucose) were significantly increased in individuals with greater GAP mean values [12].

The duration of diabetes mellitus was considerably greater in cases with a glycemic gap of ≥ 42 mg/dl compared to those with a glycemic gap of < 42 mg/dl (P value < 0.001). In our investigation, smoking and hypertension exhibited no significant distinctions between the examined groups. Our results align with those of Lepper et al., who

discovered no statistically significant difference in glucose–ADAG (A1c-derived average glucose) levels between smokers and hypertensive patients [13].

A significant increase in HbA1c, admission blood glucose levels, serum cholesterol, and ADAG was observed in cases where the glycemic gap was greater than or equal to 42 mg/dl, as compared to situations where the glycemic gap was less than 42 mg/dl (P value being less than 0.001). Deckers et al. discovered that individuals who have suffered an acute myocardial infarction (AMI) have higher glucose levels upon admission than individuals who do not have diabetes or who are mixed. It has been found that these levels are significantly associated with an elevated risk of poor outcomes and mortality [9].

In our investigation, showed lower EF in cases with a hyperglycemia gap ≥ 42 mg/dl contrasted with those with a glycemic gap < 42 mg/dl (P value < 0.001). This finding aligns with Liao et al., who demonstrated that patients with elevated glycemic gaps beyond 42 mg/dL exhibited a reduced LVEF (p = 0.04) in contrast to those with lower glycemic gaps below 42 mg/dL [10].

Our analysis revealed that the quantity of affected coronary arteries and the occurrence of ectasia were not substantially different between the examined groups. Ekmekci et al. estimated that among those with Glucose–ADAG < 42 mg/dL, 13 (10.2%) had single, 33 (26.0%) had double, and 81 (63.8%) had triple diseased vessels. In contrast, among those with Glucose–ADAG > 42 mg/dL, 15 (10.6%) had single, 32 (22.7%) had double, and 94 (66.7%) had triple diseased vessels [14].

Our research demonstrated that patients with a glycemic gap of 42 mg/dl or higher experienced significantly higher rates of mortality, in-stent restenosis, in-stent thrombosis, and hospital stay than those with a glycemic gap of 42 mg/dl or lower. There was no significant difference in the incidence of heart failure, myocardial infarction, emergent revascularization (CABG), and stroke among the groups that were examined. This result is consistent with the results of Dorn et al. [15] and Ha et al. [16].

The occurrence of MACEs was not substantially correlated with afflictions of the LAD and RCA. In this study, Osman et al. identified a significant disparity in MACE among the groups, with age, STEMI, osteal conditions, moderate severity, distal location, and all left main (LM) diseases serving as risk factors for MACE in their univariate analysis (P-value 0.05). Nonetheless, osteal, all LM disease, STEMI, and SYNTAX score were significant predictors of MACE (P-value 0.05) in the multivariate analysis. Univariate analysis in a separate study indicated that age was a significant risk factor for MACE in coronary artery disease (CAD) (P-value 0.034) [17].

In our investigation, the glycemic gap emerged as a significant predictor for MACEs in both univariate (OR 2.722, 95 percent CI 1.182 – 6.270, P value 0.019) and multivariate (OR 8.926, 95 percent CI 1.125 – 70.839, P value 0.038) analyses. This result is similar with the findings of Zelihic et al. [18] and Xu et al. [19]. In our study, cholesterol was a significant predictor of MACEs just in univariate analysis (OR 1.011, 95% CI 1.002 – 1.021, P value 0.021). This outcome contradicts the findings of Hoebers et al. [20].

In our investigation, univariate analysis revealed that age, sex, smoking, hypertension, hemoglobin, HbA1c, and admission glucose were insignificant predictors of MACEs. Conversely, multivariate analysis indicated that age, sex, smoking, hypertension, hemoglobin, HbA1c, admission glucose, and cholesterol also served as insignificant predictors for MACEs. This conclusion aligns with Liao et al., who discovered that in univariate analysis, age, sex, smoking, hypertension, and dyslipidemia are significant factors [10].

Our research indicates that the glycemic gap is a significant predictor of major MACEs in those with T2DM who have undergone elective PCI. At a threshold of > 41 mg/dl, the glycemic gap has a P value of 0.035 and an area under the curve (AUC) of 0.615. The sensitivity is 69.70%, the specificity is 54.21 percent, the positive predictive value (PPV) is 31.9 percent, and the negative predictive value (NPV) is 85.3 percent. This finding is in direct opposition to the findings of Ujueta et al., who established an optimal cutoff value of 42 mg/dL for major adverse cardiovascular events by employing the maximal Youden's index. Their methodology yielded a sensitivity of 68.9 percent, specificity of 50.7 percent, negative predictive value of 50.4 percent, and positive predictive value of 23.9 percent [21].

It also appears that the glycemic gap is an important entity in potentiating systemic inflammation and oxidative stress, especially in diabetic type 2 PCI patients. Acute hyperglycemia stimulates the release of proinflammatory cytokines including interleukin-

6 (IL-6) and tumor necrosis factor alpha (TNF-α), which leads to endothelial dysfunction and instability of the plaque. In addition, glucose fluctuations-triggered oxidative stress can also lead to reactive oxygen species (ROS) production, reduced nitric oxide bioavailability, and microvascular injury that will ultimately, increase the tendency for adverse cardiac outcomes. These patho-physiological pathways contribute to why we observed the robust relationship between the increased glycemic gap and the incidence of MACEs in the present study [22,23].

Clinical relevance of the glycemic gap is that it allows to distinguish stress hyperglycemia from chronic exposure to hyperglycemia, and therefore represents a risk stratification support. In contrast to HbA1c, which represents long-term glycemic status, the glycemic gap reflects acute metabolic deteriorations, allowing clinicians to quickly recognize patients at greater risk of cardiovascular disease after PCI. Routine consideration of glycemic gap analysis in pre-intervention and post-intervention work-up may help in therapeutic decision making (eg, aggressive glycemic control and/or use of adjunctive pharmacologic strategies) aiming to reduce the risk of in-hospital complications and long-term sequelae [24,25].

There are several limitations of this study, though it presents good results. The study was of a single-center design, with a relatively small sample size; therefore, generalizing these findings to other populations or health care systems should proceed cautiously. In addition, the absence of continuous glucose monitoring limited a more refined analysis of real time on dynamic changes in glucose concentration under stress, which could provide more insight on the dynamicity of the response to stress hyperglycemia. The observed associations could also have been confounded by unmeasured residual variables, such as undernutrition, inflammatory markers, and concurrent drugs.

To maximize the clinical value of the glycemic gap in PCI practice, subsequent studies need to be conducted using a multi-center design and a larger sample size to validate these results in multiple patient groups. Furthermore, integration of continuous glucose monitoring modalities may allow real-time measuring of glucose fluctuation in hospital, which can provide a better perspective of the relationship between the dynamic of glycemia and cardiovascular events. Trials exploring therapeutic interventions aimed at modifying the glycemic gap (e.g., intensive insulin therapy, metabolic manipulation) may provide further information on the potential benefits of modifying this variable with regard to the clinical outcome.

Conclusion:

Glycemic gap can significantly predict MACEs in diabetic cases who underwent elective PCI with a cut off >41 mg/dl, 69.70% sensitivity, 54.21% specificity, 31.9% PPV and 85.3% NPV. Glycemic gap is a simple method that can be applied easily to all diabetic patients who underwent elective PCI, to predict the short term cardiovascular adverse outcome. We recommend future studies on many patients. A limited number of studies have concentrated on the glycemic gap prognostic impact in cardiovascular diseases.

Conflict of Interest Statement: No conflicts of interest to be declared.

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