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ORIGINAL ARTICLE

What level of hyperglycaemia on admission indicates a poor prognosis in patients with myocardial infarction treated invasively?

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Abstract

Background: Stress hyperglycaemia on admission is a predictor of mortality in patients with acute myocardial infarction (MI).

Aim: To establish what level of hyperglycaemia on admission indicates a significantly poorer long-term prognosis in patients with MI treated invasively.

Methods: Glycaemia on admission was measured in patients with both ST-segment elevation MI (STEMI) and non-ST-segment elevation MI (NSTEMI) treated with percutaneous coronary intervention (PCI). In-hospital and late mortality were evaluated during a 679.3 ± 202 day follow-up.

Results: We enrolled 794 patients (564 men; 71%), mean age 63.8 ± 11.3 years. One per cent of the patients died during initial hospitalisation, and 10% during the two-year follow-up. The mean value of glycaemia in the whole population was 115 ± 36 mg/dL (6.32 ± 1.98 mmol/L). Admission glycaemia in patients who died in hospital was 194 ± 71 mg/dL (10.67 ± ± 3.91 mmol/L), while in the patients discharged home it was 114 ± 35 mg/dL (6.27 ± 1.93 mmol/L) ($p < 0.0001$). In terms of two-year mortality, the patients who died had also significantly higher glycaemia on admission (145 ± 48 mg/dL; 7.98 ± ± 2.64 mmol/L) *vs* 112 ± 31 mg/dL (6.16 ± 1.71 mmol/L, $p < 0.0001$). Apart from admission hyperglycaemia, we found the following risk factors of late mortality in univariate analysis: age, heart rate (HR), left ventricular ejection fraction (LVEF), glomerular filtration rate (GFR), creatinine level, number of significantly narrowed coronary vessels other than the infarct related artery (IRA), and unsuccessful PCI. In multivariate analysis, the following parameters correlated with death in the two-year follow-up: glycaemia on admission, age, HR, LVEF, GFR, creatinine level, total cholesterol, number of significantly narrowed coronary vessels other than the IRA, and unsuccessful PCI. Hyperglycaemia on admission was an independent risk factor of death even after adjustment for confounding variables such as age, sex and LVEF. We compared the areas under ROC curve for in-hospital mortality and the areas under ROC curve for late mortality according to glycaemia on admission. Both were significantly different from those of a random model ($p < 0.001$ and $p < 0.001$, respectively). A glycaemia value of 205 mg/dL (11.28 mmol/L) calculated from ROC curve had the highest sensitivity and specificity for late mortality. Apart from these findings, we observed a linear correlation between glycaemia and mortality.

Conclusions: The best cut-off value for stress hyperglycaemia determined by ROC curve in patients with acute MI treated invasively is 205 mg/dL (11.28 mmol/L). Patients with glucose levels > 205 mg/dL (11.28 mmol/L) on admission have significantly higher late mortality compared to those with glucose levels < 205 mg/dL (11.28 mmol/L). Our results suggest that hyperglycaemia is a reliable marker of poor outcome in acute MI patients with and without previously diagnosed diabetes mellitus. This level of glucose may be used in risk stratification in patients with acute MI.

Key words: myocardial infarction, hyperglycaemia, mortality

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INTRODUCTION

In an era when invasive therapy is the preferred therapy for acute myocardial infarction (AMI), diabetes mellitus (DM) is still associated with considerably increased mortality after an AMI [1]. Also, stress hyperglycaemia on admission is a predictor of mortality and arrhythmias in patients with ST-segment elevation MI (STEMI) [2]. Furthermore, acute hyperglycaemia is a new risk marker for contrast-induced nephropathy in patients with AMI without diabetes and normal renal function [3].

The question is: which is worse in patients undergoing primary angioplasty for AMI? Hyperglycaemia? Diabetes mellitus? Or both? In the study by Ergelen et al. [4], the patients were classified into four groups: non-diabetic/non-hyperglycaemic patients, diabetic/non-hyperglycaemic patients; non-diabetic/hyperglycaemic and diabetic/hyperglycaemic. It turned out that the non-diabetic/hyperglycaemic patients with STEMI represented the highest risk population for in-hospital mortality and major adverse cardiac events (MACE). The worst outcomes for long-term cardiovascular mortality occurred in the diabetic/hyperglycaemic patients. So hyperglycaemia is an independent and powerful prognostic marker of a worse outcome in AMI patients, as well as previously diagnosed diabetes [1].

The aim of our present study was to establish what level of hyperglycaemia on admission indicates a significantly poorer long-term prognosis in patients with MI treated invasively.

METHODS

We retrospectively studied patients with AMI with ST-segment elevation and without ST-segment elevation consecutively referred to the catheterisation laboratory of our hospital for emergency coronary angioplasty. The study inclusion criteria were: 1) confirmed MI with or without ST-segment elevation; and 2) informed consent from each patient. The study protocol, which conformed to the ethical guidelines of the 1975 Declaration of Helsinki, was approved by the local ethics committee. Exclusion criteria were: 1) cardiogenic shock on admission; and 2) life-limiting non-cardiac disease.

This observational study included consecutive patients with and without known DM.

At the start of the study, medical history was recorded, and all patients underwent physical examination, resting ECG, routine transthoracic echocardiography and coronary angiography.

The patients were divided into two groups according to the presence of hyperglycaemia. We analysed exclusively the level of glycemia independently of the presence of diabetes. We did not focus on the diagnosis of chronic renal disease; we concentrated rather on the degree of kidney disease measured by both creatinine level and estimated glomerular filtration rate (eGFR) according to the K/DOQI guidelines [5] and their influence on mortality.

We concentrated on in-hospital and two-year all-cause mortality. For all patients, mortality data was obtained from the Polish population registry (gained from the Ministry of the Interior and Administration) in Bialystok.

Laboratory analyses

Blood samples for glucose level were drawn on admission from the first blood sample and on the day of admission for the 24 hour glucose profile. Patients were classified as DM according to the American Diabetes Association clinical practice recommendations [6].

Transthoracic echocardiography

All studies were performed using the Philips Ultrasound System Sonos 5500 (Andover, MA, USA) equipped for 3.6 MHz transducer. Basic measurements were taken in every patient. Harmonic imaging was used to evaluate left ventricular ejection fraction (LVEF) according to the recommendations of the European Society of Echocardiography [7]. LVEF was derived using the bi-plane method. All measurements were derived in blinded fashion by two experienced operators.

Coronary angiography

Coronary angiography was performed by injection of contrast medium (low osmolarity, low viscosity) via 6 F catheters after 200 μg of ICGTN, filmed at 12.5 frames/s. The procedure was done via the femoral route using the standard Judkins technique. Luminal stenosis more than 75% by diameter was regarded as significant (visual assessment).

Coronary revascularisation

Percutaneous coronary intervention (PCI). The angioplasty procedure was considered successful when a residual stenosis was < 30%, in the absence of dissection and thrombosis. Contrast flow through the epicardial vessel was graded with the standard TIMI trial flow scale of 0 to 3. All angiograms were analysed by two observers blinded to clinical results.

Statistical analysis

Distribution of every variable was tested using the Kolmogorov-Smirnov test. Afterwards, the Student's t test or the Mann-Whitney U test were used for statistical analysis where applicable. Additional analysis of correlations between non-categorical variables was performed using Pearson or Spearman tests, where applicable. Free of death survival rates were displayed with Kaplan-Meier curves. ROC curves analysis was used to establish the value of hyperglycaemia in the prediction of death. Multivariate logistic regression was used to test associations between variables and outcomes. In univariate analysis, all recognised predictors of mortality in patient acute coronary syndrome (ACS), such as age, creatinine level, and cholesterol level were taken into account. In multivariate analysis, only parameters significant in univariate analysis were calculated.

Data was expressed as means and standard deviations (SD). Relative frequencies were used to present categorical variables. These variables were assessed with χ^2 test. A p value of less than 0.05 was considered as statistically significant. The statistical software NCSS 2010 was used.

RESULTS

We enrolled 794 patients (564 men, 71%), mean age 63.8 ± ± 11.3 years. Total mortality was evaluated during a 679.3 ± ± 202 day follow-up. A diagnosis of DM had been previously established in 19% (n = 151) of the patients, and prediabetic conditions in 6% (n = 48). Hypertension was present in 63% (n = 500) and hypercholesterolaemia in 46% (n = 365) of the patients. LVEF assessed by echocardiography was 46.6 ± 10.4%. Clinical characteristics are set out in Table 1.

Left anterior descending coronary artery (LAD) was the infarct-related artery (IRA) in 41% (n = 326), circumflex coronary artery (Cx) in 15% (n = 119), and right coronary artery (RCA) in 38% (n = 302), of the patients. Stent was implanted in 91% (n = 723) of the patients. Angiographic characteristics are set out in Table 2. The sum of the number of invasive procedures exceeds 100% because 10% of the patients with NSTEMI had multivessel PCI carried out during the same procedure.

One per cent (n = 10) of the patients died during initial hospitalisation and 10% (n = 83) during the two year follow-up. The mean value of glycaemia in the whole investigated population was 115 ± 36 mg/dL (6.32 ± 1.98 mmol/L). Mean glycaemia significantly differed between the group who died during hospitalisation and the rest of the patients. Admission glycaemia in the patients who died was 194 ± 71 mg/dL (10.67 ± 3.91 mmol/L), while in the patients discharged home it was 114 ± 35 mg/dL (6.27 ± 1.93 mmol/L, $p < 0.0001$). All patients who died had STEMI. They were significantly older, had higher creatinine level on admission, higher white blood cell count and CK-MB level. Thirty per cent (n = 3) of them had unsuccessful PCI (Table 3).

Table 1. Clinical and laboratory characteristics of the population (n = 794)

	Percent (n) or mean ± SD
Male sex	71% (564)
Age	63.79 ± 11.27
BMI	28.15 ± 5.69
SBP	138.19 ± 48.20
DBP	85.52 ± 16.06
HR on admission	72.64 ± 17.94
LVEF (%)	46.61 ± 10.39
STEMI	73% (580)
NSTEMI	27% (214)
Duration of follow-up	679.33 ± 201.98
Duration of hospitalisation	5.78 ± 3.28
In-hospital mortality	1% (8)
Death during follow-up	10% (79)
Arterial hypertension	63% (500)
DM type 2	19% (151)
Hypercholesterolaemia	46% (365)
Prediabetic conditions	6% (48)
MI in the past	33% (262)
Creatinine	1.02 ± 0.34
GFR [mL/min/1.7 m^2]	88.90 ± 30.83
CK on admission	585.91 ± 1,475.96
CK max	1,854.36 ± 2,376.77
CK-MB on admission	66.22 ± 86.08
CK-MB max	203.06 ± 204.78
Glycaemia on admission	114.94 ± 36.17
LDL-cholesterol	105.80 ± 42.95
HDL-cholesterol	44.45 ± 13.80
Total cholesterol	174.29 ± 56.51
Triglycerides	122.82 ± 92.46

BMI — body mass index; SBP — systolic blood pressure; DBP — diastolic blood pressure; HR — heart rate; STEMI — ST-segment elevation myocardial infarction; NSTEMI — non-ST-segment elevation myocardial infarction; DM — diabetes mellitus; MI — myocardial infarction; GFR — glomerular filtration rate; CK — phosphocreatine kinase; CK-MB — cardiac fraction of phosphocreatine kinase

Table 2. Angiographic characteristics (n = 794)

Angiographic parameters	Percent (n) or mean ± SD
Left main coronary artery as IRA	1% (8)
Left ascending coronary artery as IRA	41% (326)
Diagonal artery as IRA	4% (32)
Circumflex artery as IRA	15% (119)
Marginal artery as IRA	5% (40)
Intermediate artery as IRA	2% (16)
Right coronary artery as IRA	38% (302)
Posterior descending coronary artery as IRA	1% (8)
Postero-lateral artery as IRA	2% (16)
Vein graft as IRA	1% (8)
Arterial graft as IRA	0% (0)
POBA	11% (87)
Stent implantation	91% (723)
Number of stents	1.11 ± 0.56
Unsuccessful PCI	5% (40)
Number of significantly narrowed coronary vessels except IRA	0.74 ± 1.02

IRA — infarct related artery; POBA — percutaneous balloon angioplasty; PCI — percutaneous coronary intervention. The sum of the number of invasive procedures exceeds 100% because 10% of the patients with NSTEMI had multivessel PCI carried out during the same procedure

In terms of two-year mortality, the patients who died had also significantly higher glycaemia on admission (145 ± ± 48 mg/dL, 7.98 ± 2.64 mmol/L *vs* 112 ± 31 mg/dL, 6.16 ± ± 1.71 mmol/L, $p < 0.0001$). Apart from admission hyperglycaemia, we found the following risk factors of late mortality in univariate analysis: age, heart rate (HR) on admission, LVEF, GFR, creatinine level, number of significantly narrowed coronary vessels other than the IRA, and unsuccessful PCI (Table 4).

In multivariate analysis, the following parameters correlated with death in the two-year follow-up: glycaemia on admission, age, HR on admission, LVEF, GFR, creatinine level, total cholesterol level, number of significantly narrowed coronary vessels other than the IRA and unsuccessful PCI (Table 5). Hyperglycaemia on admission was an independent risk factor of death, even after adjustment for confounding variables such as age, sex and LVEF.

We compared the areas under ROC curves for in-hospital mortality (Fig. 1) and the areas under ROC curve for late mortality according to glycaemia on admission. AUC was 0.857 for in-hospital mortality according to glycaemia, while CI–95%–+95%: –0.729–0.985 and SE –0.065. AUC was 0.686

Table 3. Risk factors of in-hospital death in univariate analysis

	Alive patients (n = 784)		Patients who died (n = 10)		P
	Mean	SD or n	Mean	SD or n	
Glycaemia on admission	113.93	34.45	194.00	70.66	0.000
Creatinine	1.01	0.33	1.81	0.73	0.000
DBP on admission	85.89	15.41	57.00	33.43	0.000
Unsuccessful PCI	4%	31	30%	3	0.000
Total cholesterol	174.76	56.26	87.25	32.35	0.002
White blood cell count on admission	9.85	5.26	14.61	3.32	0.004
LDL-cholesterol	106.15	42.82	47.25	17.88	0.006
HDL-cholesterol	44.54	13.76	27.25	11.09	0.012
SBP on admission	138.66	48.23	102.50	29.37	0.018
CK-MB on admission	65.43	84.64	120.40	153.62	0.045
Age	63.70	11.28	70.80	8.92	0.048
STEMI	72%	564	100%	10	0.051

DBP — diastolic blood pressure; SBP — systolic blood pressure; PCI — percutaneous coronary intervention; STEMI — ST-segment elevation myocardial infarction; CK-MB — cardiac fraction of phosphocreatine kinase

Table 4. Risk factors of late mortality in univariate analysis

	Alive patients (n = 711)		Patients who died (n = 83)		P
	Mean	SD or n	Mean	SD or n	
Glycaemia on admission	111.49	30.87	144.49	58.43	0.000
Creatinine	0.99	0.29	1.29	0.58	0.000
Age	62.78	11.20	72.45	7.68	0.000
GFR	90.54	30.77	67.14	22.47	0.000
LVEF	47.30	10.15	40.09	10.52	0.000
Hypercholesterolaemia	48%	341	21%	17	0.000
HR	71.94	17.73	79.20	18.66	0.001
DBP on admission	86.13	15.22	80.25	21.45	0.002
Total cholesterol	176.33	57.01	154.69	47.50	0.002
Number of significantly narrowed coronary vessels other than the IRA	0.71	0.98	1.06	1.24	0.003
Unsuccessful PCI	4%	28	10%	8	0.023

DBP — diastolic blood pressure; LVEF — left ventricular ejection fraction; HR — heart rate; GFR — glomerular filtration rate; IRA — infarct related artery; PCI — percutaneous coronary intervention

Table 5. Risk factors of late mortality in multivariate analysis

	β	Standard deviation for β	B	Standard deviation for B	t(782)	P
Glycaemia on admission	0.192	0.033	0.002	0.000	5.788	0.000
Creatinine	0.230	0.036	0.205	0.032	6.319	0.000
Age	0.209	0.042	0.006	0.001	4.985	0.000
GFR	0.120	0.044	0.001	0.000	2.693	0.007
LVEF	0.083	0.034	0.003	0.001	2.440	0.015
Hypercholesterolaemia	0.107	0.032	0.067	0.020	3.309	0.001
HR	0.049	0.034	0.001	0.001	1.462	0.144
DBP on admission	0.072	0.032	0.001	0.001	2.226	0.026
Total cholesterol	0.073	0.033	0.000	0.000	2.214	0.027
Number of significantly narrowed coronary vessels other than the IRA	0.026	0.033	0.010	0.013	0.787	0.431
Unsuccessful PCI	0.013	0.033	0.019	0.047	0.394	0.694

DBP — diastolic blood pressure; LVEF — left ventricular ejection fraction; HR — heart rate; GFR — glomerular filtration rate; IRA — infarct related artery; PCI — percutaneous coronary intervention. For model: R = 0.4577; R2 = 0.1984; F(11.782) = 18.849; p < 0.00001

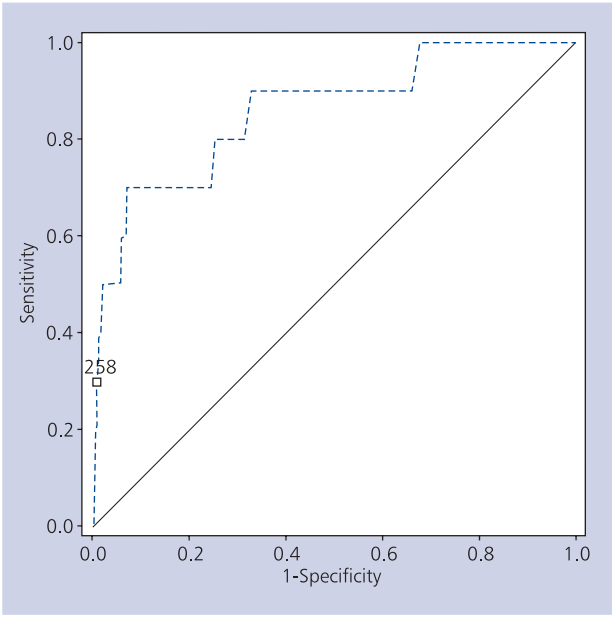

Figure 1. Comparison of areas under ROC curve for in-hospital mortality according to glycaemia on admission (p < 0.001). AUC was 0.857 for in-hospital mortality according to glycaemia, while CI–95%–+95% –0.729–0.985 and SE –0.065

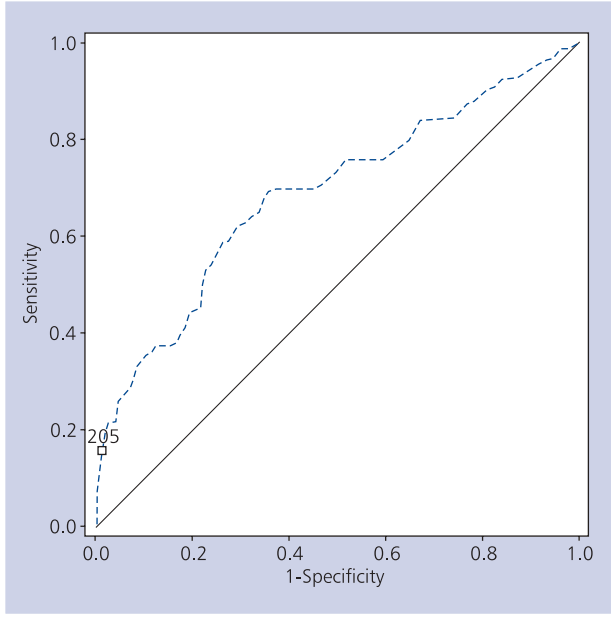

Figure 2. Comparison of areas under ROC curve for late mortality according to glycaemia on admission (p < 0.001). AUC was 0.686 for late mortality according to glycaemia, CI –0.619–0.753 and SE –0.034

for late mortality according to glycaemia, CI: –0.619–0.753 and SE –0.034. Both were significantly different to those of the random model (Fig. 2) (p < 0.001 and p < 0.001, respectively). Kaplan-Meier curve in patients with mean glycaemia ≥ 115 mg/dL (6.32 mmol/L) and < 115 mg/dL in the whole population (p < 0.01) is shown in Figure 3. Kaplan-Meier curve in patients with glycaemia ≥ 205 mg/dL (11.28 mmol/L) *vs* < 205 mg/dL (p < 0.001) is shown in Figure 4. Glycaemia value of 205 mg/dL (11.28 mmol/L) was calculated from ROC curves. This value had the highest sensitivity and specificity for late mortality. Apart from these findings, we observed a linear correlation between glycaemia and mortality. Sensitivity and specificity for glycaemia and the risk of in-hospital and late mortality are displayed in Figures 5 and 6.

We found the following selected parameters which positively and significantly correlated with glycaemia on admis-

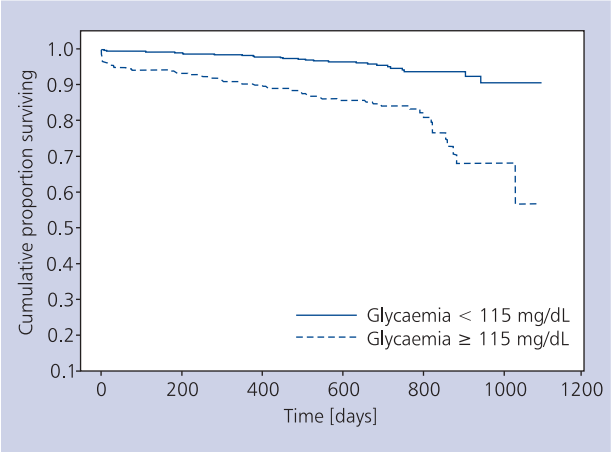

Figure 3. Kaplan-Meier curve in patients with glycaemia ≥ 115 mg/dL *vs* < 115 mg/dL ($p < 0.01$)

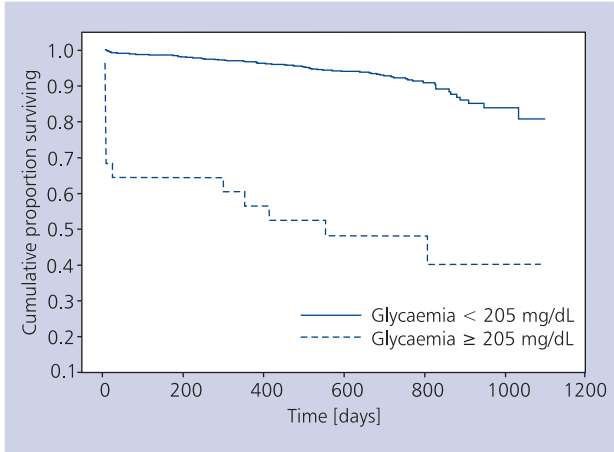

Figure 4. Kaplan-Meier curve in patients with glycaemia ≥ 205 mg/dL *vs* < 205 mg/dL ($p < 0.001$)

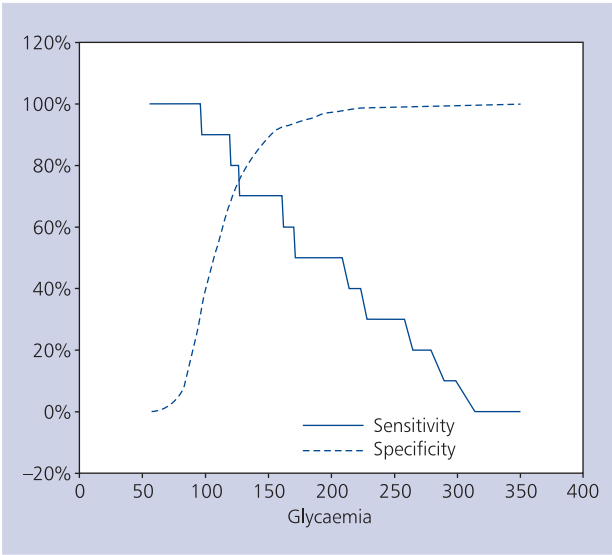

Figure 5. Sensitivity and specificity for predictive value of glycaemia in the assessment of the risk of in-hospital mortality

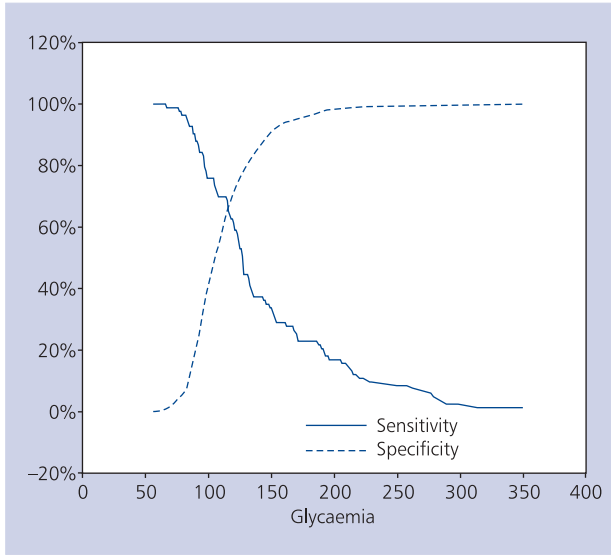

Figure 6. Sensitivity and specificity for predictive value of glycaemia in the assessment of the risk of late mortality

sion: age, body mass index, HR on admission, white blood cell count on admission, LAD as IRA, in-hospital mortality and late mortality. The following parameters correlated negatively with glycaemia on admission: male sex, GFR, and RCA as IRA. Detailed information is shown in Table 6.

DISCUSSION

The stress imposed by AMI leads to the development of insulin resistance, glucose intolerance and hyperglycaemia [8, 9]. Acute hyperglycaemia, both in diabetic and non-diabetic patients with AMI, is associated with adverse outcomes [10] and increased risk of life-threatening complications. This increased risk of complications is one of the possible explanations for the elevated in-hospital mortality in AMI patients presenting with hyperglycaemia [2, 11]. The mechanism of this phenomenon is complex. Hyperglycaemia on admission is associated with the presence and large extent of microvascular obstruction on contrast-enhanced CMR [12], with a larger infarct size determined by SPECT [13], poorer recovery of microvascular integrity and abnormal coronary flow reserve [14]. Admission blood glucose is a predictor of the TIMI frame count (TFC), which reflects coronary blood flow and no-reflow phenomenon [15]. No reflow occurs more frequently during PCI in patients with acute hyperglycaemia, suggesting microvascular dysfunction [16–18].

Table 6. Statistically significant correlations between glycaemia on admission and selected clinical parameters

Male sex	–0.1284	Creatinine	0.1537	White blood cell count on admission	0.0908
	n = 794		n = 789		n = 793
	p = 0.000		p = 0.000		p = 0.010
Age	0.1516	CK-MB on admission	0.0766	In-hospital mortality	0.2470
	n = 793		n = 695		n = 794
	p = 0.000		p = 0.043		p = 0.000
GFR	–0.0879	CK-MB max	0.0882	Late mortality	0.2794
	n = 669		n = 694		n = 794
	p = 0.023		p = 0.020		p = 0.000
BMI	0.1537	DM type 2	0.2613	LAD as IRA	0.0708
	n = 645		n = 775		n = 794
	p = 0.000		p = 0.000		p = 0.046
HR on admission	0.1622	LVEF	–0.2355	RCA as IRA	–0.0798
	n = 765		n = 592		n = 794
	p = 0.000		p = 0.000		p = 0.025

LVEF — left ventricular ejection fraction; HR — heart rate; GFR — glomerular filtration rate; IRA — infarct related artery; BMI — body mass index; DM — diabetes mellitus; CK-MB — cardiac fraction of phosphocreatine kinase; LAD — left anterior descending coronary artery; RCA — right coronary artery

In the light of these findings, it is crucial to establish a cut-off value of glycaemia which would indicate AMI patients with a poor prognosis.

According to our study, the best cut-off value for stress hyperglycaemia in patients with AMI treated invasively for assessing risk of death is 205 mg/dL (11.28 mmol/L). The value was determined by ROC curve. Patients with glucose levels < 205 mg/dL (11.28 mmol/L) on admission had significantly lower mortality compared to those with glucose levels > 205 mg/dL (11.28 mmol/L). This value had the highest sensitivity and specificity for a poor prognosis in our study. We had such a high cut-off value because ten patients who died had a level of glycaemia over 300 mg/dL. A linear correlation between glycaemia level and late mortality was found, which confirms the predictive significance of mean glycaemia value (115 mg/dL). This correlation is displayed on the Kaplan-Meier curves (Figs. 3, 4).

In the study by Ergelen et al. [4], hyperglycaemia was defined as a venous plasma glucose level ≥ 200 mg/dL (11 mmol/L) on admission. According to this value, the authors selected patients who represented the highest risk population for in-hospital mortality and MACE. After adjustment for potentially confounding factors, both non-diabetes/hyperglycaemia and diabetes/hyperglycaemia status remained independent predictors of long-term cardiovascular mortality.

In STEMI patients treated with primary PCI, multivariate linear regression analysis showed that hyperglycaemia on admission, defined as a value > 200 mg/dL (11 mmol/L), was an independent predictor of infarct size determined by SPECT five days after AMI [13].

Nevertheless, in most studies, hyperglycaemia on admission is defined as blood glucose above 140 mg/dL (7.7 mmol/L) according to the American Diabetes Association and the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus [19]. Jensen et al. [12] proved the presence of microvascular obstruction on contrast-enhanced CMR that relates to admission hyperglycaemia based on this value.

An optimal threshold glycaemia level of 140 mg/dL (7.7 mmol/L) on admission to predict mortality was obtained by Sanjuán et al. [2] by ROC curve. Those who presented glucose ≥ 140 mg/dL (7.7 mmol/L) showed higher rates of malignant ventricular tachyarrhythmias, complicative bundle branch block, new atrio-ventricular block, and in-hospital mortality. Multivariate analysis showed that those with glycaemia ≥ 140 mg/dL (7.8 mmol/L) exhibited a two-fold increase of in-hospital mortality risk, irrespective of DM status.

In a population with MI complicated by cardiogenic shock treated with PCI, hyperglycaemia was also defined as 140 mg/dL (7.7 mmol/L). Patients with hyperglycaemia on admission had higher in-hospital, one-year and five-year mortality compared to patients with blood glucose < 140 mg/dL (< 7.7 mmol/L) [20].

In the study by Kosiborod et al. [21], differences in glucose-associated mortality risks between patients with and without known diabetes persisted when analyses were repeated with admission glucose modelled as a continuous variable (in 10 mg/dL increments). Although in the normal glucose range patients without diabetes had lower 30-day mortality than patients with diabetes, their risk increased more steeply at higher glucose levels, surpassing the risk of patients with diabetes at 140 mg/dL. The results were similar for one-year mortality, with the risk in nondiabetic patients surpassing that of the diabetic group at a glucose level of 170 mg/dL (9.35 mmol/L).

Yang et al. [22] found a striking U-shaped relationship between admission glucose levels and short- and long-term mortality. An initial admission glucose level ≥ 5.1 mmol/L (92.7 mg/dL) to ≤ 7.0 mmol/L (127.3 mg/dL) may be desirable because it was associated with better clinical outcomes.

There is another approach to hyperglycaemia in AMI patients. Peak glycaemia greater than 180 mg/dL according to Lazzeri et al. [23] was associated with the highest mortality, whereas patients whose peak glycaemia was between 140 mg/dL (7.7 mmol/L) and 180 mg/dL (9.9 mmol/L) exhibited intermediate mortality rates.

Limitations of the study

Our study is a retrospective analysis. We included consecutive patients, with few exclusion criteria, resulting in a heterogeneous population.

CONCLUSIONS

The best cut-off value for stress hyperglycaemia determined by ROC curve in patients with AMI treated invasively is 205 mg/dL (11.28 mmol/L). Patients with glucose levels > 205 mg/dL (11.28 mmol/L) on admission have significantly higher late mortality compared to those with glucose levels < 205 mg/dL (11.28 mmol/L). Our results suggest that hyperglycaemia is a reliable and independent marker of poor outcome in AMI patients with and without previously diagnosed DM. This level of glucose may be used in risk stratification in patients with ACS.

Conflict of interest: *none declared*

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Jaka hiperglikemia przy przyjęciu wskazuje na złe rokowanie u chorych z zawałem serca leczonych inwazyjnie?

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Streszczenie

Wstęp: Hiperglikemia przy przyjęciu do szpitala jest czynnikiem ryzyka zgonu u chorych z ostrym zawałem serca (MI).

Cel: Celem badania było ustalenie, jaka glikemia przy przyjęciu do szpitala wskazuje na istotnie gorsze długoterminowe rokowanie u chorych z MI leczonych inwazyjnie.

Metody: Do badania włączono kolejnych pacjentów zarówno z zawałem serca z uniesieniem odcinka ST (STEMI), jak i bez uniesienia odcinka ST (NSTEMI) leczonych za pomocą angioplastyki wieńcowej (PCI). Badanie objęło pacjentów z cukrzycą i bez cukrzycy. Chorzy byli podzieleni na 2 grupy w zależności od wartości glikemii wyznaczonej za pomocą krzywej ROC. Punktami końcowymi były zgon szpitalny i śmiertelność 2-letnia.

Wyniki: Do badania włączono 794 pacjentów (564 mężczyzn; 71%), w wieku 63,8 ± 11,3 roku. Śmiertelność oceniano w ciągu 679,3 ± 202 dni. Cukrzycę rozpoznano u 19,0%, a stany przedcukrzycowe u 6% osób. Śmiertelność wewnątrzszpitalna wynosiła 1%, natomiast 2-letnia — 10%. Średnia wartość glikemii w całej badanej populacji wynosiła 115 ± 36 mg/dl (6,32 ± 1,98 mmol/l) i różniła się ona istotnie między grupą chorych, która zmarła w czasie hospitalizacji, a resztą populacji. Glikemia przy przyjęciu u osób, które zmarły, wynosiła 194 ± 71 mg/dl (10,67 ± 3,91 mmol/l), podczas gdy u pacjentów wypisanych do domu — 114 ± ± 35 mg/dl (6,27 ± 1,93 mmol/l) ($p < 0,0001$). Pacjenci, którzy zmarli, byli starsi, mieli wyższe stężenia kreatyniny, CK-MB i wyższą leukocytozę; u 30% z nich PCI było nieskuteczne. Jeśli chodzi o śmiertelność 2-letnią, pacjenci, którzy zmarli, mieli istotnie wyższą glikemię przy przyjęciu: 145 ± 48 mg/dl (7,98 ± 2,64 mmol/l) *v.* 112 ± 31 mg/dl (6,16 ± 1,71 mmol/l), $p < 0,0001$. W analizie wieloczynnikowej następujące parametry korelowały ze zgonem w 2-letniej obserwacji: glikemia przy przyjęciu, wiek, HR, LVEF, GFR, stężenie kreatyniny, hipercholesterolemia, stężenie cholesterolu, liczba istotnie zwężonych tętnic wieńcowych, oprócz tętnicy odpowiedzialnej za MI oraz nieskuteczna PCI. Podwyższona glikemia przy przyjęciu była predykatorem zgonu niezależnym od takich czynników, jak wiek, płeć czy LVEF. Po porównaniu pól pod krzywymi ROC dla śmiertelności wewnątrzszpitalnej i późnej w odniesieniu do glikemii przy przyjęciu do szpitala okazało się, że różnią się one istotnie od przypadkowego modelu ($p < 0,001$ i $p < 0,001$, odpowiednio). Krzywe Kaplana-Meiera pokazały różnice przeżycia, gdy wartością glikemii dzielącą na 2 grupy była wartość średnia glikemii w populacji 115 mg/dl (6,32 mmol/l), $p < 0,01$. Ale dopiero gwałtowny spadek przeżycia wykazały krzywe Kaplana-Meiera, gdy punktem podziału na 2 grupy była wartość glikemii 205 mg/dl (11,28 mmol/l) wyznaczona przez krzywą ROC ($p < 0,001$). Ta wartość glikemii miała najwyższą czułość i specyficzność w przewidywaniu późnej śmiertelności. Ponadto zaobserwowano liniową korelację między glikemią i śmiertelnością.

Wnioski: Najbardziej właściwa wartość odcięcia dla hiperglikemii przy przyjęciu do szpitala wyznaczona przez krzywą ROC to 205 mg/dl (11,28 mmol/l). Pacjenci z glikemią > 205 mg/dl (11,28 mmol/l) charakteryzują się istotnie większą śmiertelnością w porównaniu z chorymi z glikemią < 205 mg/dl (11,28 mmol/l). Nasze wyniki wskazują, że hiperglikemia > 205 mg/dl (11,28 mmol/l) jest wiarygodnym i niezależnym markerem niepomyślnego rokowania u chorych z MI, zarówno z cukrzycą, jak i bez wcześniej rozpoznanej cukrzycy. Wyliczone z krzywej ROC stężenie glukozy może być użyte w stratyfikacji ryzyka chorych z MI.

Słowa kluczowe: zawał serca, hiperglikemia, śmiertelność

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