

Long-term prognosis is related to mid-term changes of glucometabolic status in patients with acute myocardial infarction treated invasively

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Abstract

Background: Glucometabolic status (GS) in patients with acute myocardial infarction (AMI) has an impact on prognosis, but it may change over time.

Aim: To evaluate the prognosis after AMI treated invasively with respect to changes in GS assessed by oral glucose tolerance test at discharge and at mid-term follow-up visit (FU-visit).

Methods: Glucometabolic status was assessed by two-hour post-load glycaemia and defined as abnormal glucose tolerance (AGT) or normal glucose tolerance (NGT). Out of 454 in-hospital AMI survivors, 368 (81%) patients completed an FU-visit and were divided into four groups with respect to GS at discharge and FU-visit: group 1 — AGT at discharge and FU-visit (n = 101); group 2 — AGT at discharge and NGT at FU-visit (n = 48); group 3 — NGT at discharge and AGT at FU-visit (n = 114); and group 4 — NGT at discharge and FU-visit (n = 105). All-cause mortality was compared between groups with log-rank test.

Results: Median time from AMI to FU-visit was seven months. Median remote follow-up duration after AMI was 31 months. Two-hour post load glycaemia was significantly higher in patients with confirmed AGT at FU-visit than in other groups. Mortality was higher in group 1 (11.9%) than in group 2 (2.1%; p = 0.034) and group 4 (2.9%; p = 0.009). Mortality rates between group 2 and 4 were similar (2.1% vs. 2.9%; p = 0.781). There was no significant difference in mortality between group 1 and group 3 (11.9% vs. 6.1%; p = 0.114). Mortality in group 3 was over two-fold higher than in group 4; however, this difference was statistically non-significant (6.1% vs. 2.9%; p = 0.247).

Conclusions: Prognosis for patients with confirmed AGT was unfavourable; however, patients with AGT at discharge, in whom GS improved, had similar mortality to subjects with persistent NGT. The major clinical implication from this study is the finding that reassessment of GS by repeated oral glucose tolerance test has significant prognostic value and makes initial risk stratification performed at discharge more reliable.

Key words: acute myocardial infarction, abnormal glucose tolerance, glucometabolic status, mortality, oral glucose tolerance test

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INTRODUCTION

Glucometabolic status evaluated by two-hour post-load glycaemia (2h-PG) during oral glucose tolerance test (OGTT) has been a well-established risk factor for worse prognosis in patients with acute myocardial infarction (AMI) [1–4]. Newly detected diabetes mellitus (DM) or impaired glucose tolerance were associated with similar mortality to pre-hospital diabetes [5]. In stable patients after AMI, derangements

in glucose metabolism defined at hospital discharge were shown to be transient, persistent, unchanged, or newly detected [6–10]. However, there are no recommendations on repeated OGTT in patients after AMI. Therefore, the aim of the presented work was to evaluate the prognosis after AMI treated invasively with respect to changes of glucometabolic status assessed by OGTT at discharge and at mid-term follow-up visit (FU-visit).

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METHODS

Study population

The presented analysis was a part of a single-centre, observational study prospectively enrolling AMI patients treated with percutaneous coronary intervention (PCI) between January 2012 and December 2013, who survived the in-hospital period and were discharged to ambulatory care. Patients in whom DM was diagnosed before admission or discovered by elevated fasting glycaemia ≥ 7 mmol/L on at least two occasions during hospitalisation were excluded from the study. The study population encompassed 454 consecutive AMI survivors in whom OGTT was performed at hospital discharge. Repeated OGTT and other laboratory tests (blood count, fasting glycaemia, glycosylated haemoglobin, serum creatinine concentration with glomerular filtration rate [GFR] estimation, lipids) were planned as part of FU-visit on an outpatient basis approximately six months after AMI. OGTT with the use of 75 g of glucose was performed at hospital discharge (but not earlier than four days after AMI onset) and at FU-visit in patients without antidiabetic treatment. Moreover, at FU-visit in all patients an electrocardiogram and echocardiography were performed. Patients were divided into four study groups with respect to glucometabolic status at discharge and at FU-visit. Subjects were prospectively followed, and remote major adverse cardiovascular events (MACE) were recorded after the FU-visit. MACE were defined as the occurrence of death or any of the following events: recurrent myocardial infarction, repeated PCI, coronary artery by-pass grafting, hospitalisation for decompensated heart failure, or stroke.

Definitions of glucometabolic status

Group 1 consisted of patients with 2h-PG ≥ 7.8 mmol/L detected at discharge and 2h-PG ≥ 7.8 mmol/L or antidiabetic pharmacotherapy at FU-visit.

Group 2 consisted of patients with 2h-PG ≥ 7.8 mmol/L detected at discharge and without antidiabetic pharmacotherapy at FU visit with 2h-PG < 7.8 mmol/L.

Group 3 consisted of patients with 2h-PG < 7.8 mmol/L at discharge but with 2h-PG ≥ 7.8 mmol/L or antidiabetic pharmacotherapy at FU-visit.

Group 4 was established as a control group and consisted of patients with 2h-PG < 7.8 mmol/L at discharge and without antidiabetic pharmacotherapy at FU-visit with 2h-PG < 7.8 mmol/L.

Abnormal glucose tolerance (AGT) was defined as 2h-PG ≥ 7.8 mmol/L. Patients using antidiabetic agents at FU-visit were considered as having AGT. Patients without antidiabetic pharmacotherapy and 2h-PG < 7.8 mmol/L were classified as having normal glucose tolerance (NGT). Group 1 was considered as persistent AGT, group 2 as transient AGT, group 3 as newly detected AGT, group 4 as persistent NGT.

Definitions of AMI

Clinical AMI criteria evaluated on admission were: chest pain persisting > 20 min, ST segment elevation of at least 0.1 mV in two or more contiguous electrocardiographic leads, or non-diagnostic electrocardiogram (without persistent ST segment elevation, left bundle branch block, or acute ischaemic changes) with enzymatic confirmation of AMI.

Catheterisation protocol and treatment

All patients before coronary angiography received a single dose of oral aspirin (300 mg), loading dose of P2Y₁₂ inhibitor, just before PCI, and 100 U/kg of intravenous heparin (additional boluses were given as appropriate to achieve activated clotting time > 250 s). In all patients, coronary angiography and PCI of infarct-related artery were performed immediately after admission. After the intervention, all patients received 75–100 mg of aspirin daily indefinitely, a maintenance dose of P2Y₁₂ inhibitor, as well as beta-blockers, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and statins, if these agents were not contraindicated. All patients with disturbances in glucose metabolism were advised on lifestyle modification. Antidiabetic pharmacotherapy for new onset DM detected at hospital discharge was started after consultancy with a diabetology specialist. Patients with newly detected DM during FU-visit on repeated OGTT were referred to a diabetology specialist on an outpatient basis.

Ethics

All clinical data were obtained as a result of the diagnostic procedures and therapy, which were in accordance with the appropriate guidelines. All patients provided informed, written consent for hospitalisation, invasive treatment, and use of their data for research purposes. Follow-up visits were performed on an outpatient basis as a routine follow-up. Remote follow-up was performed by telephone contact with patients or their families as well as during routine ambulatory visits. The study protocol was in line with ethical standards and was approved by the Institutional Review Board.

Statistical analysis

Continuous parameters were expressed as means with standard deviations unless otherwise specified, and categorical variables were presented as numbers and percentages. Comparative analyses between groups were performed using Student's t-test for continuous variables and χ^2 or Fisher's exact test, as appropriate, for dichotomous parameters. Log-rank tests were used to compare Kaplan-Meier curves plotted for cumulative survival. All tests were double-sided. P value < 0.05 was considered statistically significant. All analyses were performed using the software package Statistica (version 6.1, StatSoft Inc., Tulsa, OK, USA).

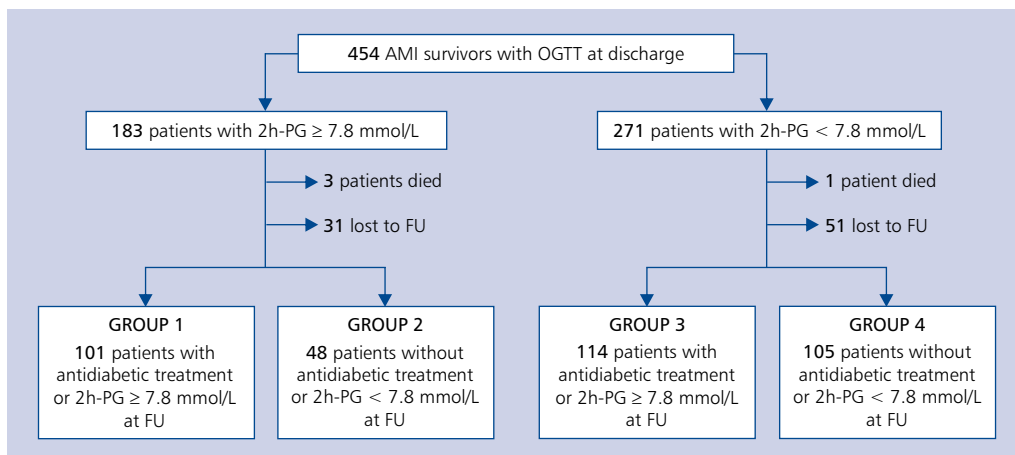


Figure 1. Patient enrolment into the study; 2h-PG — two-hour post load glucose; AMI — acute myocardial infarction; FU — follow-up; OGTT — oral glucose tolerance test

The analysis of receiver-operator characteristic (ROC) curves was performed using MedCalc (version 11.3.0.0, MedCalc, Ostend, Belgium).

RESULTS

Out of 454 in-hospital AMI survivors with OGTT at discharge, 368 (81.0%) patients completed FU-visit with laboratory re-evaluation of glucometabolic status. For all patients, remote follow-up was obtained. The median time to FU-visit was seven months. The median remote follow-up duration after AMI was 31 months. Four (0.9%) patients died before FU-visit. All survivors to the FU-visit were encouraged to participate in the visit; however, 82 (18.1%) patients refused and thus were considered lost to follow-up. Out of all patients who completed FU-visit, 353 (95.9%) had re-evaluated glucometabolic status with OGTT. Fifteen patients in whom OGTT was not repeated were treated with antidiabetic pharmacotherapy. The enrolment of patients into the study is shown in Figure 1.

Comparative analysis of demographic, clinical, laboratory, and pharmacotherapy data obtained at follow-up visit

Patients with persistent AGT, when compared to the control group, were older, more often had a history of atrial fibrillation/flutter (AF/AFL) and pre-hospital history of arterial hypertension, had lower estimated GFR, higher concentration of triglycerides, and were more often treated with dihydropyridine calcium channel blocker or diuretic.

Patients with transient AGT, when compared to the control group, more often had a history of AF/AFL, had lower estimated GFR, and were more often treated with angiotensin converting enzyme inhibitor or angiotensin II receptor antagonist.

Patients with newly detected AGT, when compared to the control group, more often had a history of AF/AFL and had higher concentration triglycerides.

Triglyceride concentration was higher in groups with AGT at FU-visit than in the control group.

Comparative analysis glucometabolic data and markers of myocardial injury

Obesity was diagnosed during hospitalisation most frequently within the group with persistent AGT at FU-visit, but there were no statistically significant differences in the prevalence of obese patients between study groups. In-hospital admission glycaemia was significantly higher in group with transient or persistent AGT compared to groups with newly detected AGT or persistent NGT. The difference between persistent AGT and transient AGT was not significant ($p = 0.103$). In-hospital fasting glycaemia was significantly higher in group with persistent AGT compared to all other groups. At FU-visit, fasting glycaemia was significantly higher in all study groups when compared to the control group, but there were no significant differences between study groups. Two-hour post load glycaemia during OGTT performed in-hospital and at FU-visit was significantly higher in patients with persistent AGT than in other groups.

Patients with transient AGT had the highest concentrations of highly sensitive troponin T and MB isoenzyme of creatine kinase (CK-MB); however, the only statistically significant difference was observed between groups 2 and 3 with respect to CK-MB value. There was a trend towards significantly higher CK-MB in group 2 than in the control group ($p = 0.056$).

Detailed characteristics of particular study groups are presented in Tables 1 and 2.

The analysis of ROC curves

Receiver-operator characteristic curve analysis was performed to assess the diagnostic accuracy of in-hospital admission glycaemia, fasting glycaemia, and 2h-PG, which were measured during index hospitalisation for the prediction of sustained AGT or newly detected AGT at FU-visit. In patients with AGT

Table 1. Comparative analysis of glucometabolic data and markers of myocardial injury

Patients' characteristics	Group 1 (n = 101)	Group 2 (n = 48)	Group 3 (n = 114)	Group 4 (n = 105)
In-hospital diagnosis of obesity (BMI ≥ 30 kg/m ²)	32 (31.7%)	12 (25.0%)	26 (22.8%)	23 (21.9%)
In-hospital admission glycaemia [mmol/L]	7.86 \pm 2.05 ^{a, c}	7.28 \pm 1.95 ^{a, d}	6.47 \pm 1.44	6.55 \pm 1.68
In-hospital fasting glycaemia [mmol/L]	6.0 \pm 0.87 ^{a, b, c}	5.6 \pm 0.71	5.43 \pm 0.55	5.4 \pm 0.59
In-hospital 2-hour post-load glycaemia [mmol/L]	10.58 \pm 2.23 ^{a, b, c}	9.27 \pm 1.3 ^{a, d}	6.47 \pm 1.11	6.52 \pm 1.1
Fasting glycaemia at FU-visit [mmol/L]	6.0 \pm 0.85 ^a	5.77 \pm 0.57 ^a	5.84 \pm 0.69 ^a	5.57 \pm 0.62
2-h post-load glycaemia at FU-visit [mmol/L]	9.58 \pm 1.84 ^{a, b, c}	6.02 \pm 2.02 ^d	8.83 \pm 1.59 ^a	5.74 \pm 1.88
Glycosylated haemoglobin at FU-visit [%]	6.16 \pm 0.52 ^{a, b, c}	5.7 \pm 0.35 ^d	6.01 \pm 0.37 ^a	5.7 \pm 0.47
TnT-hs [ng/mL]	1.49 \pm 2.34	1.69 \pm 2.48	1.31 \pm 2.18	1.51 \pm 2.37
CK-MB [ng/mL]	117.17 \pm 147.38	151.09 \pm 150.78 ^{d, e}	94.41 \pm 118.18	107.33 \pm 120.06

Values presented as means \pm standard deviation or number and percentage of subjects; AGT — abnormal glucose tolerance; BMI — body mass index; CK-MB — creatine kinase-MB isoenzyme; FU-visit — follow-up ambulatory visit; NGT — normal glucose tolerance; Group 1 — AGT at discharge and FU-visit; Group 2 — AGT at discharge but NGT at FU-visit; Group 3 — NGT at discharge but AGT at FU-visit; Group 4 — NGT at discharge and FU-visit; NGT — normal glucose tolerance; TnT-hs — troponin T high sensitive assay; ^ap < 0.05 vs. group 4; ^bp < 0.05 — group 1 vs. group 2; ^cp < 0.05 — group 1 vs. group 3; ^dp < 0.05 — group 2 vs. group 3; ^etrend towards significant difference between group 2 and group 4 (p = 0.056); *values obtained during oral glucose tolerance test in patients without antidiabetic treatment

Table 2. Comparative analysis of demographic, clinical, laboratory, and pharmacotherapy data obtained at follow-up visit

Patients' characteristics at FU-visit	Group 1 (n = 101)	Group 2 (n = 48)	Group 3 (n = 114)	Group 4 (n = 105)
Age [years]	64.8 \pm 9.2 ^{a, c}	62.2 \pm 9.2	61.4 \pm 9.3	59.7 \pm 10.6
Female sex	26 (25.7%)	14 (29.2%)	28 (24.6%)	25 (23.8%)
Pre-hospital history of arterial hypertension	77 (76.2%) ^{a, c}	30 (62.5%)	69 (60.5%)	57 (54.3%)
Left ventricle ejection fraction [%]	46.36 \pm 9.44	46.1 \pm 8.3	48 \pm 6.5	48 \pm 7.97
eGFR [mL/min/1.73 m ²]	82.7 \pm 26.9 ^a	83.9 \pm 18.8 ^a	87.89 \pm 18.23	92 \pm 22.5
Haemoglobin [mmol/L]	8.72 \pm 0.81	8.98 \pm 0.77	8.85 \pm 0.86	8.86 \pm 0.92
Atrial fibrillation/flutter	14 (13.9%) ^a	8 (16.6%) ^a	9 (7.9%) ^a	2 (1.9%)
HDL cholesterol [mmol/L]	1.28 \pm 0.39	1.36 \pm 0.37	1.29 \pm 0.46	1.35 \pm 0.38
LDL cholesterol [mmol/L]	2.44 \pm 0.87 ^c	2.58 \pm 0.94	2.73 \pm 1.2	2.53 \pm 0.93
Triglycerides [mmol/L]	1.46 \pm 1 ^a	1.31 \pm 0.6	1.46 \pm 0.94 ^a	1.23 \pm 0.6
Acetylsalicylic acid	94 (93.1%)	44 (91.7%)	104 (91.2%)	102 (97.1%)
P2Y ₁₂ receptor inhibitor	100 (99%) ^c	46 (95.8%)	105 (92.1%)	104 (99%)
Beta-adrenergic blocker	100 (97%)	45 (93.7%)	110 (96.5%)	98 (93.3%)
ACE-I/ARB	95 (94.1%)	48 (100%) ^{a, d}	102 (89.5%)	93 (88.6%)
Statin	95 (94.1%)	46 (95.8%)	109 (95.6%)	99 (94.3%)
Dihydropyridine CCB	28 (27.7%) ^a	12 (25%)	23 (20.2%)	17 (16.2%)
Diuretic	43 (42.6%) ^{a, c}	13 (27.1%)	26 (22.8%)	17 (16.2%)
Aldosterone receptor antagonist	33 (32.7%) ^c	13 (27.1%)	22 (19.3%)	23 (22.1%)
Antidiabetic pharmacotherapy	15 (14.9%)	0 (0%)	0 (0%)	0 (0%)
Oral hypoglycaemic agent	13 (12.9%)	—	—	—
Metformin	9 (8.9%)	—	—	—
Insulin	2 (2%)	—	—	—

Values presented as means \pm standard deviation or number and percentage of subjects; eGFR — estimated glomerular filtration rate; FU-visit — follow-up ambulatory visit; HDL — high-density lipoprotein; LDL — low-density lipoprotein; ACE-I — angiotensin converting enzyme inhibitor; ARB — angiotensin II receptor antagonist; CCB — calcium channel blocker; ^ap < 0.05 vs. group 4; ^bp < 0.05 — group 1 vs. group 2; ^cp < 0.05 — group 1 vs. group 3; ^dp < 0.05 — group 2 vs. group 3

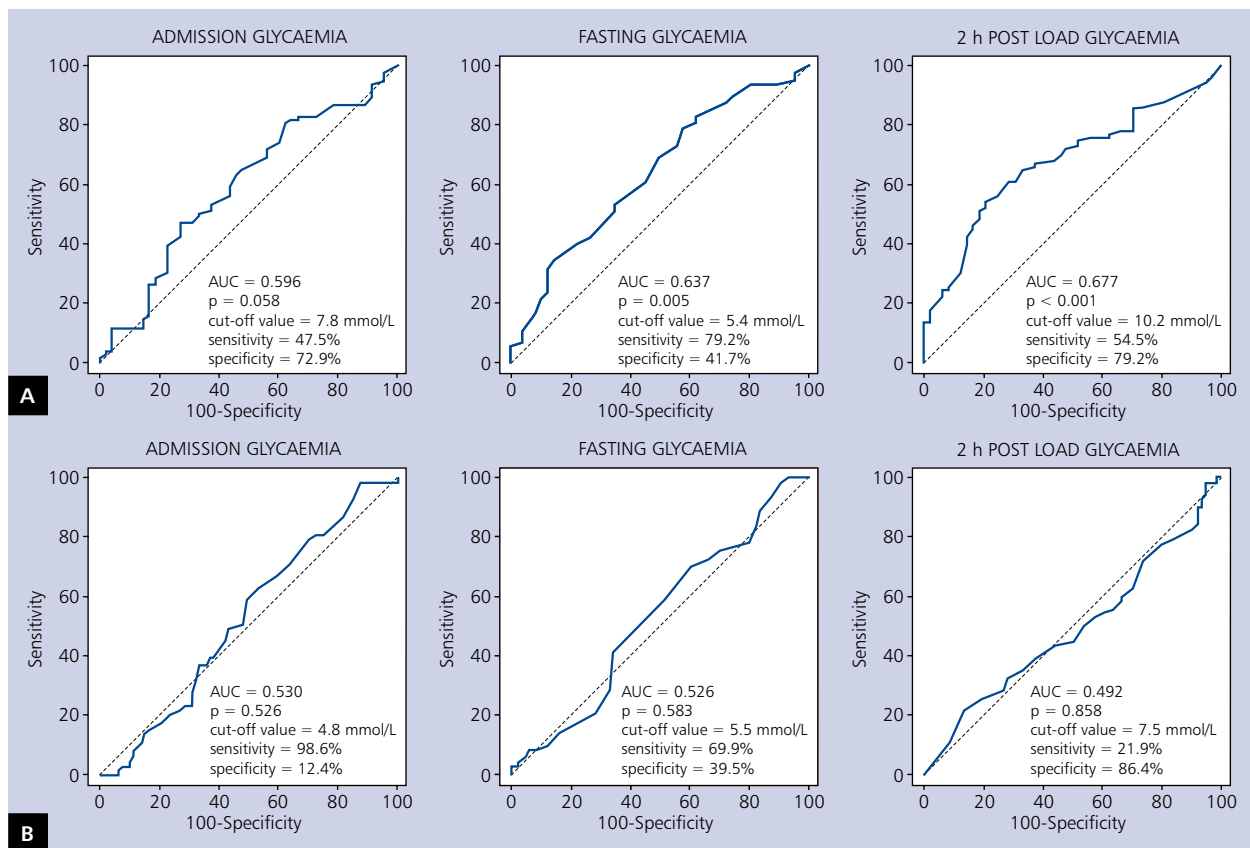


Figure 2. The analysis of receiver-operator characteristic (ROC) curves of in-hospital glycaemic parameters for the prediction of persistent abnormal glucose tolerance (AGT) at follow-up ambulatory visit (FU-visit) among patients with AGT at discharge (A) or newly detected AGT at FU-visit among patients with normal glucose tolerance at discharge (B); 2 h — two hour; AUC — area under the curve

at discharge, in-hospital fasting glycaemia and in-hospital 2h-PG discriminated between AGT and NGT at FU-visit. The highest value of area under the curve (AUC) was observed for 2h-PG (AUC = 0.677). In patients with NGT at discharge none of the analysed glycaemic parameters during hospitalisation had the ability to distinguish between AGT and NGT at FU-visit. The results of ROC analysis are presented in Figure 2.

Comparative analyses of mortality and adverse cardiovascular events

Among patients who completed FU-visit, there was no statistically significant difference in mortality between patients with AGT and subjects with NGT determined at hospital discharge (8.7% vs. 4.6%; $p = 0.111$). There was a trend towards more MACE after FU-visit in patients with AGT at discharge (24.2% vs. 16%; $p = 0.051$). However, there were statistically significant differences in mortality rates between study groups with respect to glucometabolic status re-evaluated at FU-visit. Mortality was higher in group with persistent AGT (11.9%) than in group with transient AGT (2.1%; $p = 0.034$) and control group (2.9%; $p = 0.009$). Mortality rates between group 2

and 4 were similar (2.1% vs. 2.9%; $p = 0.781$). There was no significant difference in mortality between group 1 and group 3 although it was almost two-fold higher (11.9% vs. 6.1%; $p = 0.114$). Mortality in group with newly detected AGT was over two-fold higher than in group with persistent NGT; however, this difference was non-significant (6.1% vs. 2.9%; $p = 0.247$). Comparative analysis of mortality rates between groups are presented in Figure 3.

Analysis of MACE showed higher incidence of events in group 1 (24.8%) and group 2 (22.9%) than in group 3 (16.7%) and group 4 (15.2%); however, those differences were not statistically significant. Comparative analysis of prognosis between study groups is shown in Table 2.

Among 15 patients who were treated with antidiabetic drugs (all subjects in group 1) one major adverse event occurred, and there were no deaths during remote follow-up.

DISCUSSION

Initial data had indicated that the prognosis in patients with AGT was significantly determined in the early post-infarction period, but more recent studies have shown that AGT is also

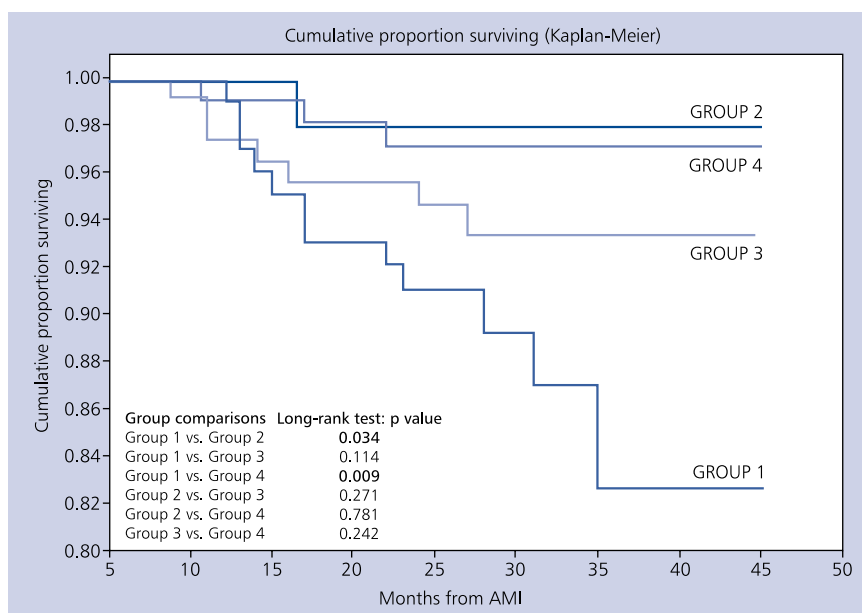


Figure 3. Kaplan-Meier curves for cumulative survival in patients with acute myocardial infarction (AMI) with respect to glucometabolic status at follow-up visit; Group 1 — AGT at discharge and FU-visit; Group 2 — AGT at discharge but NGT at FU-visit; Group 3 — NGT at discharge but AGT at FU-visit; Group 4 — NGT at discharge and FU-visit; AGT — abnormal glucose tolerance; NGT — normal glucose tolerance; FU-visit — follow-up ambulatory visit

Table 3. Comparative analysis between groups with respect to all-cause mortality and major adverse cardiovascular events (MACE) during remote follow-up after follow-up visit

Adverse events	Group 1 (n = 101)	Group 2 (n = 48)	Group 3 (n = 114)	Group 4 (n = 105)
All-cause mortality	12 (11.9%) ^{a, b}	1 (2.1%)	7 (6.1%)	3 (2.9%)
MACE*	25 (24.8%)	11 (22.9%)	19 (16.7%)	16 (15.2%)

Values presented as number and percentage of subjects; *Each event was counted only once; ^ap < 0.05 vs. group 4; ^bp < 0.05 — group 1 vs. group 2

associated with worse long-term outcome [1–4]. In the presented work, among patients without pre-hospital DM, who completed FU-visit, risk stratification with respect to glucometabolic status when performed at hospital discharge revealed no significant differences in remote mortality between AGT and NGT. However, patients with persistent AGT at FU-visit had significantly higher all-cause mortality than patients with AGT at discharge, who improved glucometabolic profile. Transient AGT was associated with similar mortality to persistent NGT. Those observations may be partially explained in relation to conclusions from the study published by Perreault et al. [11], who showed that in patients with prediabetes, conversion to normal glucose regulation, even if transient, was associated with lower long-term cardiovascular risk. The presented study showed that patients with newly detected AGT at FU-visit had over two-fold higher mortality than controls and almost two-fold lower than patients with confirmed AGT; however, the observed differences were statistically non-significant. Henareh et al. [6] showed, that in stable patients who were evaluated

for new onset glucose abnormalities several months after AMI, there were no differences in long-term cardiovascular outcome between patients with AGT and NGT, although increase in two-hour post load glucose had an adverse impact on prognosis [6]. Conversely, in patients with stable coronary artery disease during a median observation of 4.3 years, new onset AGT was associated with worse prognosis, including mortality [12]. In light of published data and the observed divergence in survival curves presented in Figure 2, it is likely that higher mortality in patients with newly detected AGT would become significant during a longer follow-up period.

An important finding from this study was that in patients with AGT at discharge in-hospital fasting glycaemia and particularly 2h-PG were useful parameters for the prediction of AGT at FU-visit. In the long term observation, admission glycaemia was not a reliable risk factor of mortality in relation to glucometabolic status; however, patients with transient AGT appeared to have an increased risk of non-fatal MACE when compared to newly detected AGT and controls with persis-

tent NGT. This observation could be interpreted according to the results of the study by Timmer et al. [13], who showed that in patients without pre-hospital DM, after exclusion of early mortality (within 30 days), admission glycaemia lost its significant impact on long-term mortality, although this parameter was associated with larger infarct size. The association of acute hyperglycaemia with infarct size has been shown recently, especially in patients without evidence of chronic hyperglycaemia [14]. Those data correspond with observed differences in admission glycaemia and CK-MB levels between study groups. Patients with a transient pattern of AGT had significantly higher admission glycaemia and enzymatic evidence of larger infarct size than patients with new onset AGT or persistent NGT. Considering the cited studies, it is reasonable to assume that a transient pattern of AGT is related to a larger infarct size, haemodynamic stress, and acute hyperglycaemia. Whether patients who recovered to NGT at FU-visit would eventually develop glucose abnormalities is not known.

Clinical implications

The major clinical implication from this study is the finding that reassessment of glucometabolic status by repeated OGTT has significant prognostic value and makes initial risk stratification performed at discharge more reliable. However, the optimal secondary prevention strategy in patients with new onset glucose abnormalities and AMI still has not been thoroughly established. Although nonpharmacological treatment is considered the first-line therapy for new onset glucose abnormalities, published studies have shown unsatisfactory adherence to such management in patients with coronary artery disease, especially with concomitant DM [12, 15–17]. Recently, it has been shown that among patients with ST elevation myocardial infarction and without DM, but including patients with prediabetes, subjects who were treated with metformin presented modest improvement of cardiovascular risk profile at four-month follow-up [18]. Anselmino et al. [19] showed that there was a pronounced decrease in cardiovascular events in patients with newly detected DM, who were prescribed glucose lowering drugs, compared with those not receiving such treatment.

Published data indicated also that pharmacological and nonpharmacological management of patients with impaired glucose tolerance had favourable cardiovascular effects [20–22].

Limitations of the study

The study was nonrandomised and observational. It encompassed consecutive patients who were hospitalised due to AMI; however, not all subjects who were discharged participated in FU-visits. Thus, it is likely that patients who were evaluated at the visit had higher levels of compliance and more favourable risk profiles than the remaining subjects. It suggests that glucometabolic status after AMI was not reflected

adequately. There were no data on cross-over between study groups or the initiation of glucose-lowering medication during remote follow-up after the FU-visit. The authors were unable to receive data on the mechanism of all deaths. It is well known that, in addition to vascular disease, DM and prediabetes are associated with mortality also due to other causes [23, 24]. However, the comparisons between study groups and other studies with respect to cardiac deaths are missing.

CONCLUSIONS

In patients who completed FU-visit at the mid-term period after AMI, reassessment of glucometabolic status by OGTT improved long-term risk stratification. Persistent AGT at FU-visit was associated with significantly higher all-cause mortality than transient AGT or sustained NGT. Patients with AGT at discharge in whom glucometabolic profile improved had similar mortality to subjects with persistent NGT.

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Rokowanie długoterminowe jest zależne od zmian stanu glukometabolicznego w obserwacji średnioterminowej u pacjentów z zawałem serca leczonych inwazyjnie

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Streszczenie

Wstęp: Metabolizm glukozy u pacjentów z zawałem serca (MI) ma znaczenie rokownicze, jednak może zmieniać się po ostrej fazie choroby.

Cel: Celem pracy było określenie znaczenia rokowniczego zmian metabolizmu glukozy wykrytych na podstawie doustnego testu obciążenia glukozą wykonanego przy wypisywaniu ze szpitala oraz podczas wizyty kontrolnej u chorych leczonych inwazyjnie w ostrej fazie MI.

Metody: Stan metaboliczny określono na podstawie glikemii po 2 godzinach od obciążenia glukozą jako nieprawidłowa tolerancja glukozy lub prawidłowa tolerancja glukozy. Spośród 454 pacjentów, którzy przeżyli okres wewnątrzszpitalny, w wizytach kontrolnych uczestniczyło 368 (81%) chorych, którzy zostali podzieleni na cztery grupy w zależności od stanu metabolicznego określonego przy wypisywaniu ze szpitala i podczas wizyty kontrolnej: grupa 1 — nieprawidłowa tolerancja glukozy przy wypisywaniu ze szpitala i podczas wizyty kontrolnej (n = 101); grupa 2 — nieprawidłowa tolerancja glukozy przy wypisywaniu ze szpitala i prawidłowa podczas wizyty kontrolnej (n = 48); grupa 3 — prawidłowa tolerancja glukozy przy wypisywaniu ze szpitala i nieprawidłowa podczas wizyty kontrolnej (n = 114); grupa 4 — prawidłowa tolerancja glukozy przy wypisywaniu ze szpitala oraz podczas wizyty kontrolnej (n = 105). Śmiertelność całkowita została porównana między grupami przy użyciu testu *log-rank*.

Wyniki: Mediana czasu do wizyty kontrolnej wyniosła 7 miesięcy. Mediana czasu całkowitej obserwacji po MI — 31 miesięcy. Podczas wizyty kontrolnej glikemia po 2 godzinach od obciążenia glukozą była znamienne wyższa w grupie pacjentów z potwierdzoną nieprawidłową tolerancją glukozy niż w pozostałych grupach badanych. Śmiertelność była wyższa w grupie 1 (11,9%) niż w grupie 2 (2,1%; p = 0,034), jak również w grupie 1 niż w grupie 4 (2,9%; p = 0,009). Rokowanie pacjentów w grupie 2 i w grupie 4 było podobne (2,1% vs. 2,9%; p = 0,781). Śmiertelność w grupie 1 była prawie 2-krotnie wyższa niż w grupie 3 (11,9% vs. 6,1%; p = 0,114), a w grupie 3 ponad 2-krotnie wyższa niż w grupie 4 (6,1% vs. 2,9%; p = 0,247), jednak różnice te nie były istotnie statystycznie.

Wnioski: Rokowanie pacjentów, u których podczas wizyty kontrolnej potwierdzono nieprawidłową tolerancję glukozy, było gorsze niż w pozostałych grupach badanych. Pacjenci, u których przy wypisywaniu ze szpitala stwierdzono nieprawidłową tolerancję glukozy, jeżeli poprawili profil metaboliczny, mieli podobne rokowanie do osób z potwierdzoną prawidłową tolerancją glukozy. Otrzymane wyniki mają znaczenie kliniczne, ponieważ wskazują, że ponowna ocena stanu metabolicznego na podstawie doustnego testu obciążenia glukozą u chorych po MI poprawia i wzmacnia stratyfikację ryzyka wykonaną przy wypisywaniu ze szpitala.

Słowa kluczowe: doustny test obciążenia glukozą, nieprawidłowa tolerancja glukozy, śmiertelność, zawał serca

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