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Authors: Dominika Drwiła, Paweł Rostoff, Grzegorz Gajos, Jadwiga Nessler, Ewa Konduracka

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Prognostic value of the triglyceride-glucose index among non-diabetic patients with acute myocardial infarction in one year follow-up

Dominika Drwiła[1], Paweł Rostoff [1, 2], Grzegorz Gajos[1, 2], Jadwiga Nessler [1, 2], Ewa Konduracka [1, 2]

[1]Department of Coronary Disease and Heart Failure, John Paul II Hospital, Kraków, Poland

[2]Jagiellonian University Medical College , Department of Coronary Disease and Heart Failure, John Paul II Hospital, Kraków, Poland

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Correspondence to:
Ewa Konduracka, MD, PhD,
Jagiellonian University Medical College, Department of Coronary Disease and Heart Failure, John Paul II Hospital,
Prądnicka 80, 31–202 Kraków, Poland,
phone: +48 12 614 22 18,
e-mail: ekonduracka@interia.eu

WHAT'S NEW?

The triglyceride-glucose index (TyG index) is a metabolic marker recently considered as a novel cardiovascular risk factor. In our study we assessed the potential relationship between the TyG index and incidence of major adverse cardiovascular events (MACE) and all-cause mortality in one year follow-up among non- diabetic patients with acute myocardial infarction (MI). We demonstrated no clinical evidence for the importance of this marker. The TyG index value does not appear to predict incidence of MACE and all-cause mortality among non-diabetic patients with MI in one year follow-up.

ABSTRACT

Background: The triglyceride-glucose index (TyG index) is a novel metabolic marker initially used as an indicator of insulin resistance. Recently, its use as a cardiovascular risk factor has been taken into consideration; however, there is a shortage of evidence for its clinical importance.

Aim: The aim of the study was to assess the relationship between the TyG index = ln (fasting triglyceride [mg/dl] × fasting glucose [mg/dl]/2) and the incidence of major adverse cardiovascular events (MACE) in one year follow-up among non- diabetic patients with acute myocardial infarction (MI). In addition, the predictive value of the TyG index concerning all-cause mortality in the study group was evaluated.

Methods: For the study, 1340 non-diabetic patients with acute MI (median age 67 years, 70.4% male) were consecutively enrolled between 2013 and 2019. Fasting lipid profile and fasting glucose level were assessed within 24 hours of admission.

Results: MACE occurred in 8.13 % (n = 109) of the study group, whereas one year mortality rate was 14.5% (n = 195). There was no difference in the median TyG index value among patients with and without incidence of MACE in one year follow-up (8.73 [8.36–9.08] vs 8.81 [8.5–9.17], *P* = 0.09). Moreover, the TyG index was not a predictor of these events (*P* = 0.06). In multivariable regression analysis, only previously diagnosed coronary artery disease (CAD) was an independent predictor of MACE (OR, 1.54; 95% CI, 1.02–2.32; *P* = 0.03). Finally, the TyG index was not an indicator of all-cause mortality (*P* = 0.25).

Conclusions: The TyG index should not be used as a predictor of MACE and all-cause mortality among non-diabetic patients with MI in one year follow-up.

Key words: all-cause mortality, MACE, myocardial infarction, triglyceride-glucose index

INTRODUCTION

In modern non-invasive cardiology, great emphasis is placed on prevention of coronary artery disease (CAD) which can manifest as acute or chronic coronary syndromes and/or heart failure. There are several unmodifiable and modifiable cardiovascular risk factors but considerable research concerning new factors has been conducted worldwide and numerous previous studies reveal that insulin resistance (IR) is significantly related to the occurrence of CAD among diabetic and non-diabetic patients [1, 2]. A practical indicator to measure IR is the HOMA-IR test; however, its usefulness is limited due to the necessity of measuring the level of insulin which is not always possible in the circumstances that prevail. Recently, the triglyceride-glucose index (TyG index) has been suggested as a new tool to measure IR [3].

The primary aim of the study was to examine the association between TyG index value and the occurrence of Major Adverse Cardiovascular Events (MACE) in one year follow-up among non-diabetic patients with acute myocardial infarction (MI). The secondary was the evaluation of its predictive value concerning one year mortality in the study group.

METHODS

This was a cohort study based on data collected from the medical records of 2300 patients with acute MI admitted to our hospital between 2013 and 2019. Patients who met inclusion criteria were consecutively recruited for the study.

Inclusion criteria were: diagnosis of STEMI (ST-segment elevation myocardial infarction) or NSTEMI (non-ST-segment elevation myocardial infarction), coronary angiography undergone on admission with presence of haemodynamically relevant atherosclerosis and full medical documentation. Exclusion criteria were: diabetes or prediabetes diagnosed prior to admission, use of glucose-lowering drugs or insulin, MI with non-obstructive CAD (MINOCA), acute heart failure on admission and incomplete medical records.

All patients had undergone emergency coronary angiography followed by percutaneous angioplasty with stent implantation or CABG (coronary artery bypass grafting) if indicated. CAD severity was assessed with the Gensini score system [4] and performed by 2 experienced invasive cardiologists. Additionally, basic blood tests and echocardiography were performed. Data concerning MACE and one year mortality were obtained from telephone consultations scheduled with patients or their families one year after MI.

Laboratory tests

Lipid profile and blood fasting glucose level (FGL) were evaluated from fasting blood samples collected within 24-hours of admission. Lipid profile was measured by the direct enzymatic colorimetric method, using commercial in vitro diagnostic devices (cobas c, Roche, Basel, Switzerland), whereas FGL was measured by the enzymatic hexokinase technique, using in vitro equipment (cobas c, Roche, Basel, Switzerland). The TyG index was calculated manually using the following formula: TyG index = ln (fasting triglyceride [mg/dl] × fasting glucose [mg/dl]/2) [5].

Definitions

Acute MI was defined according to the European Society of Cardiology guidelines, the Third (2012) or Fourth (2018) Universal Definition of Myocardial Infarction [6, 7]. MACE was a composite of myocardial infarction, in-stent restenosis, unstable angina, stroke or transient ischaemic attack and hospitalisation due to heart failure. Overweight was defined as a body mass index (BMI) ranging from 25 to 29.9 kg/m^2, whereas obesity was determined as a BMI of 30 kg/m^2 or higher. Diabetes was defined according to guidelines valid at the day of hospital

admission [8]. Furthermore, in the current report, impaired glucose tolerance or impaired fasting glucose before hospital admission were described as prediabetes. Acute heart failure was diagnosed in patients admitted with signs and symptoms of heart failure due to decompensation of pre-existing cardiomyopathy or a new onset heart failure caused by MI. A blood pressure of 140/90 mm Hg or higher, on at least two separate measurements, or the use of antihypertensive drugs were defined as hypertension.

Ethics

The study protocol was approved by the local Ethics Committee (Jagiellonian University Medical College - KBET: 1072.6120.189.2020 to EK). Each study participant provided written informed consent before enrolment.

Statistical analysis

All calculations were made using the STATISTICA 13.3 software package (TIBCO Software Inc., Palo Alto, CA, USA). A two-sided *P*-value <0.05 was considered to be statistically significant. Continuous variables were expressed as medians, using the first and third quartiles, while categorical variables were shown as numbers and percentages. Normality of variables was assessed with the Shapiro–Wilk test. The Mann–Whitney and Kruskal–Wallis tests were used for non-normally distributed continuous variables and categorical variables were compared using the Chi-square test. Stepwise logistic regression analysis was performed for determining the independent predictors of MACE and all-cause mortality. The final multivariable model included variables that were significant univariate predictors.

RESULTS

Patients

For our initial analysis we enrolled 2300 patients admitted to our department. A total of 807 patients were excluded due to diabetes or prediabetes diagnosed prior to admission; 153 patients were excluded because of incomplete medical records. In addition, among those excluded, there were 18 cases of acute heart failure on admission. Finally, we analysed data collected from 1340 patients at a median age of 67 years, among which 70.4% were male. Most of the patients were overweight, with a median BMI of 26 kg/m^2. For 66% of them, MI was the first manifestation of CAD. Baseline characteristics of the study population are shown in Table 1.

Analysis of MACE

MACE occurred in 8.13 % (n = 109) of the study group. There were 35 cases of MI, 19 cases of in-stent restenosis, 49 cases of unstable angina, four cases of stroke or transient ischaemic attack and 13 hospitalisations due to heart failure. Furthermore, among these cases, there were 12 patients who developed 2 incidents of MACE in one year follow-up and these were 5 cases of unstable angina and in-stent restenosis, 5 cases of MI and in-stent restenosis and 2 of myocardial infarction and hospitalisation due to heart failure.

Analysis in groups of patients, with and without incidence of MACE in one year follow-up, revealed that there were no statistically significant differences in median age, ejection fraction, BMI, Gensini score, glucose, high-density lipoprotein cholesterol (HDL-C), TyG index value, occurrence of hypertension, gender and lipid–lowering therapy prior to admission. Patients with incidence of MACE had lower low-density lipoprotein cholesterol (LDL-C), non-high-density lipoprotein cholesterol (non-HDL-C), total cholesterol (TC), triglycerides (TG) and estimated glomerular filtration rate (eGFR). Moreover, 46.8% of them had been diagnosed with CAD prior to admission, whereas in the second group this was only 32.6%. Detailed demographic and clinical characteristics of those groups are presented in Table 2.

Univariate and multivariable regression analysis of MACE

Univariate regression analysis showed that previously diagnosed CAD, eGFR, LDL-C and TC were significant predictors of MACE. However, in multivariable model, only previously diagnosed CAD proved to be an independent predictor (odds ratio [OR], 1.54; 95% CI, 1.02–2.32; $P = 0.03$). The TyG index was not an indicator of MACE in the study group ($P = 0.06$). The significant predictors of MACE are presented in Table 3.

Patients with potential glucose metabolism disorders

To deepen our analysis we divided patients, according to their glycaemic control status, into two groups: one with lower fasting glucose level (FGL <7.8 mmol/l) and the other with potential, previously undiagnosed, glucose metabolic disorder or stress hyperglycaemia caused by MI (FGL ≥7.8 mmol/l). Hyperglycaemia occurred in 25.2% of the patients (n = 338). There was a difference in medians of the TyG index between those two groups: 8.7 (8.4–9) in the lower FGL group, versus 9.17 (8.86–9.5) in the higher one ($P <0.01$). There was, however, no significant difference between glycaemic control status during hospitalisation and incidence of MACE in one year follow-up. MACE occurred in 7.68% (n = 77) of patients with lower FGL and in 9.47% (n = 32) of those with potential glucose metabolic disorder ($P = 0.29$). Additionally, after

excluding from the analysis patients with higher FGL, only CAD diagnosed prior to admission, eGFR, LDL-C and TC were statistically significant predictors of MACE in univariate regression analysis (Table 4). The TyG index value was insignificant ($P = 0.12$).

Univariate and multivariable regression analysis of one-year mortality

The all-cause mortality rate in one year follow-up was 14.5% (n = 195) for the whole study group, whereas in-hospital mortality was 1.6% (n = 22). In univariate regression analysis, the TyG index value appeared to be an irrelevant indicator of all-cause mortality ($P = 0.25$), whereas age, BMI, Gensini score, eGFR, LDL-C and TC were statistically significant. Finally, multivariable regression analysis showed that only age was an independent predictor of all-cause mortality in one year follow-up (OR, 1.1; 95% CI, 1.06–1.13; $P < 0.01$). Predictors of all-cause mortality are shown in Table 5.

DISCUSSION

To the best of our knowledge, this study is the first that assesses the TyG index, measured during acute MI among non-diabetic patients, as a potential predictor of MACE and all- cause mortality in one year follow-up. Previously, this metabolic marker was used as an easily accessible indicator of insulin resistance [9], predictor of diabetes [10] and biomarker of glycaemic control in type 2 diabetes mellitus [11]. Since the TyG index is a quite novel IR marker, there is no internationally recognised cut-off value. Unger et al. [12] suggested that this value for metabolic syndrome in the general population was 8.8 in men and 8.7 in women and in a Lee et al. [13] study, where a cut-off value for the TyG index was set at 8.8, this marker was a statistically significant predictor for incidental diabetes in 4-year follow up. For the current study, the population median TyG index value was 8.8 which may suggest a high incidence of IR among patients with MI.

In many patients with MI, the level of fasting glucose is elevated and called “stress hyperglycaemia”. This condition usually occurs in critically ill patients without diabetes mellitus diagnosed prior to admission [14, 15]. It appears to be connected with a stress mechanism which is associated with steroid hormones, temporary IR and a high level of free fatty acids [16]. According to the American Diabetes Association (ADA), stress hyperglycaemia in hospitalised patients is a random glucose level greater than 7.8 mmol/l at any time [17]. In our research, this condition occurred in 25.2% of patients. There was no correlation between higher glucose level and incidence of MACE in one year follow-up.

Furthermore, even after excluding from the analysis patients with higher FGL, the TyG index, which is directly related to levels of TG and glucose, was not a predictor of MACE.

The usefulness of the TyG index as a predictor of cardiovascular events has previously been investigated in several studies, mostly among healthy individuals or patients with stable CAD.

A recent Chinese retrospective study [18] among 6076 healthy individuals aged over 60 years with 6-year follow-up, showed that a higher risk of CAD events was associated with an increasing value of TyG index. Another study on that subject, conducted by Park et al. [19] and performed among healthy individuals with no traditional cardiovascular risk factors, showed that a TyG index value over 8.48 was a predictor of CAD. Finally, in an Iranian study [20], the risk of developing CAD increased with increasing quintiles of the TyG index in a long-term follow-up period (16 years).

To the best of our knowledge, little is known about the predictive value of the TyG index in patients with MI. Luo et al. [21] conducted a study on patients with STEMI, undergoing percutaneous coronary intervention, to assess the clinical outcomes of that marker during a follow-up period of one year. Those clinical outcomes were defined as major adverse cardiac and cerebrovascular events (MACCE) and included: all-cause death, target vessel revascularisation, MI, unstable angina pectoris, heart failure, stroke and transient cerebral ischaemia. In that study, patients were divided into four groups according to TyG index quartiles. The incidence of MACCE and all-cause mortality was higher among patients with TyG index values in the highest quartile. Analysis of the predictors of MACCE showed statistical significance for: a TyG index value ≥9.098, age, hypertension, diabetes, eGFR, number of implanted stents and multivessel CAD in univariate analysis. In multivariable analysis, however, only a TyG index value ≥9.608 and the number of implanted stents were significant.

In our analysis, on the other hand, the TyG index value was not significant in univariate regression analysis ($P = 0.06$). In addition, in multivariable model, only CAD diagnosed prior to admission was relevant (OR, 1.54; 95% CI, 1.02–2.32; $P = 0.03$).

In the Luo et al. research, the percentage of patients with incidence of MACCE was higher than in our study — 34.3% vs 8.13%. Moreover, patients in the MACCE group had higher mean values of FGL (9 [standard deviation, SD = 4.2] mmol/l vs median value of 6.9 [5.9–8] mmol/l in our study) and 31.2% of them had diabetes. Furthermore, those patients had higher values of LDL-C, TC and TG (mean value –1.9 [SD = 1.6] mmol/l vs median value of 1.13 [0.9–1.44] mmol/l). Consequently, their TyG index value was higher, with a mean value of 10.076 (SD = 0.483) in the highest quartile group. Additionally, in this research only 2.4% of those patients with incidence of MACE had been diagnosed with CAD prior to admission, whereas in our

study, this was 46.8%. Both study populations were similar concerning age, BMI and proportion of males. Finally, no correlation between one year mortality and TyG index was found in our report, whereas in Luo et al. that correlation was statistically significant.

In another Chinese study presented by Mao et al. [22], patients with NSTEMI were initially divided into 2 groups according to their TyG index value, these being low (<8.8) and high (>8.8) scores. In that study, more than half of the patients had diabetes or glucose metabolism disorder. Additionally, the incidence of MACE including cardiac death, nonfatal myocardial infarction, target vessel revascularisation, congestive heart failure, and nonfatal stroke was higher in the high TyG index group in one year follow-up (12.8% vs 22.8%; P <0.01).

To deepen the analysis, Mao divided patients into 4 groups, depending on the TyG index value and occurrence of glucose metabolism disorder. There was a statistically significant difference between the incidence of MACE among patients without glucose metabolism disorder with low (10.7%) and high (33.8%) TyG index value. Finally, in univariate analysis, the TyG index was significantly associated with MACE (Hazard Ratio [HR] 1.951; 95% CI, 1.416–2.688; P <0.01). Furthermore, in the multivariable model, the TyG index also remained an independent predictor of MACE. In the Mao research, the study group was not divided according to the incidence of MACE, so a simple comparison with our study is difficult to perform. In the relatively small population of 438 patients, incidence of MACE was 17.8%, whereas in our population of 1340 patients, MACE occurred only in 8.13%.

On the other hand, a simple correlation of the value of the TyG Index with incidence of atherosclerosis, its severity and incidence of MACE, is a questionable matter. Alizargar et al. [23], in their article assessing the practical value of the TyG index, emphasises that using this marker can be easily biased by hyperlipidaemia, diabetes or other glucose metabolic disorders, as the TyG index has a direct relationship with levels of TG and glucose (based on the TyG index formula). In conclusion, these factors should be carefully managed to justify the use of TyG index as a biomarker. In the Polish population we can still observe insufficient adherence to the guidelines concerning proper level of glucose, lipid profile, blood pressure, BMI, physical activity and smoking [24], therefore potential use of TyG index might be limited.

Dziedzic et al. [25] in their study concerning educational programs among Polish elderly patients, showed that several training meetings performed to change lifestyle in that group had an impact on the lipid profile of the participants, particularly concerning the level of TG (P = 0.02). Finally, Wybraniec et al. [26] revealed that patients enrolled on a similar programme (Managed Care After Acute Myocardial Infarction Program) had a lower rate of MACE in one year follow-up (11.3% vs 19.1%; P = 0.0006). We believe that similar programs should be

implemented in order to properly manage basic cardiological risk factors, reduce rate of MACE and improve patient survival.

Vega et al. [27] also presents doubts concerning the predictive value of the TyG index. In that study, this index was positive predictor of coronary heart disease (CHD), cardiovascular disease (CVD) and all-cause mortality but only unadjusted and, after adjustment, for age, smoking, BMI and systolic blood pressure. After an additional adjustment for non-HDL-C level, the HR was lower: 0.83 for CHD, 0.89 for CVD and 0.89 for all-cause mortality. Our findings correspond with the Vega conclusions that the TyG index does not predict all-cause mortality.

In our opinion, this metabolic biomarker should not be used as a predictor of clinical outcomes among non-diabetic patients with MI. Firstly, due to the fact that the TyG index has a direct relationship with the level of glucose which can be labile in acute conditions such as MI. Secondly, in our study, the TyG index was not a predictor of MACE even after excluding from the study group patients with FGL ≥7.8 mmol/l. Finally, there was no dependence between TyG index value and all-cause mortality.

We should also briefly discuss other predictors that were statistically significant in our study. Surprisingly, LDL-C and TC were negative predictors of MACE and all-cause mortality. We believe that this was caused by the fact that patients with incidence of MACE had lower values of those parameters than patients without it; moreover, patients with incidence of MACE had lower values of non-HDL-C and TG. Even though there was no difference between use of statins and fibrates prior to admission among patients with and without the incidence of MACE in one year follow- up, we can assume that the lipid- lowering therapy of those patients with the incidence of MACE was more intense and their compliance with prescribed therapy was better due to the fact that 46.8% of them were previously diagnosed with CAD. Unfortunately, we are unable to verify those assumptions. Moreover, data concerning lipid- lowering therapy was obtained from anamnesis and patients compliance with prescribed treatment remains unknown.

Study limitations

This study has several limitations. Firstly, a relatively short follow-up period. Secondly, insufficient information concerning patients compliance with prescribed therapy and data regarding changes in lipid-lowering therapy, which might have improved cardiovascular outcomes of the patients [28] and affected our study. Thirdly, no information concerning date of MACE. Finally, shortage of time and unknown causes of death in patients without one year survival.

CONCLUSIONS

The TyG index does not appear to be a predictor of MACE among non- diabetic patients with MI. We believe that its potential use in acute conditions is limited by acute metabolic changes accompanying MI and it does not help to identify non-diabetic individuals at greater risk of poor clinical outcomes. Furthermore, lack of dependence between TyG index value and all-cause mortality in one year follow-up also reflects a questionable clinical value of that parameter. Moreover, comprehensive evaluation of patients' cardiovascular risk factors should focus primarily on basic risk factors. Additional markers may be useful but after proper management with basic parameters.

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Table 1. General characteristic of the study group

Variables	All study patients n = 1340	Men n = 944	Women n = 396	*P*-value
Age, years[a]	67 (59–76)	64 (58–74)	72 (64–80)	<0.01
BMI, kg/m² [a]	26 (24–29)	26 (24–29)	26 (23–29)	0.06
First episode of MI, n (%)	887 (66.2)	614 (65)	273 (69)	0.16
STEMI, n (%)	587 (43.8)	425 (45)	162 (40.9)	0.16
NSTEMI, n (%)	752 (56.2)	519 (55)	234 (59.1)	
Gensini score	50 (28–86.5)	56 (32–88)	40 (24–81)	0.08

Hypertension, n (%)	1073 (80)	748 (79)	325 (82)	0.2
eGFR, ml/min/1.73 m^2 [a]	59 (48–69.5)	56 (47–65)	65.5 (55–79.5)	<0.01
FGL, mmol/l[a]	6.6 (5.7–7.9)	6.6 (5.7–7.8)	6.8 (5.8–8)	0.08
Lipid profile				
LDL-C, mmol/l[a]	2.8 (2.1–3.6)	2.7 (2.1–3.6)	2.9 (2.2–3.6)	0.09
HDL–C, mmol/l[a]	1.2 (0.98–1.4)	1.1 (0.95–1.4)	1.2 (1–1.5)	<0.01
Non-HDL-C, mmol/l[a]	3.1 (2.5–4)	3.1 (2.4–4)	3.2 (2.6–4)	0.08
TC, mmol/l[a]	4.4 (3.7–5.2)	4.3 (3.6–5.2)	4.6 (3.8–5.3)	<0.01
TG, mmol/l[a]	1.2 (0.95–1.6)	1.2 (0.93–1.6)	1.3 (0.98–1.6)	0.55
TyG index value[a]	8.8 (8.5–9.1)	8.8 (8.5–9.1)	8.8 (8.5–9.2)	0.2
Medical therapy prior to admission				
Statins, n (%)	1103 (82)	765 (81)	338 (85)	0.14
Fibrates, n (%)	36 (2.7)	26 (2.8)	10 (2.5)	0.2
ACEI/ARB, n (%)	1099 (82)	782 (83)	317 (80)	0.1
B-adrenolitics, n (%)	576 (43)	415 (44)	161 (41)	0.12
Calcium blockers, n (%)	1072 (80)	764 (81)	308 (78)	0.14
ASA, n (%)	498 (37)	363 (38)	135 (34)	0.09
Clopidogrel, n (%)	25 (1.9)	17 (1.8)	8 (2)	0.4
Occurrence of MACE in one year follow-up, n (%)	109 (8.13)	78 (8.2)	31 (7.8)	0.79
In-hospital mortality, n (%)	22 (1.6)	14 (1.5)	8 (2)	0.48
One year mortality, n (%)	195 (14.5)	130 (13.8)	65 (16.4)	0.21

[a]Data are shown as median (interquartile range) unless otherwise indicated. P <0.05 was considered significant.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ASA, aspirin; BMI, body mass index; CAD, coronary artery disease; eGFR, estimated glomerular filtration rate; FGL, fasting glucose level; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol; MACE, Major Adverse Cardiovascular Events; MI, myocardial infarction; non-HDL-c, non-high-density lipoprotein cholesterol; NSTEMI; non-ST segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction; TC, total cholesterol; TG, triglyceride; TyG index, triglyceride-glucose index

Table 2. Demographic and clinical characteristics of patients with and without incidence of MACE in one year follow-up

Variables	Patients with incidence of MACE in one year follow-up n = 109	Patients with no incidence of MACE in one year follow-up n=1231	*P*-value
Male gender, n (%)	78 (71.5)	866 (70.3)	0.8
Age, years[a]	67 (62–79)	67 (59–76)	0.15
BMI, kg/m^{2} [a]	25.8 (23.2–29.4)	26.3 (23.8–29)	0.6
CAD diagnosed prior to admission, n (%)	51 (46.8)	402 (32.6)	<0.01
Ejection fraction, %[a]	50 (35–55)	50 (35–60)	0.7
Hypertension, n (%)	86 (78.9)	987 (80.2)	0.74
Gensini score	58 (24–96)	48 (28–96)	0.85
eGFR, ml/min/1.73 m^{2} [a]	54.9 (47.3–64.8)	59.2 (48.4–70)	0.04
FGL, mmol/l[a]	6.9 (5.9–8)	6.6 (5.7–7.8)	0.18
FGL ≥7.8 mmol/l, n (%)	32 (29.3)	306 (24.8)	0.29
LDL-C, mmol/l[a]	2.45 (1.96–3.44)	2.8 (2.15–3.6)	<0.01
HDL-C, mmol/l[a]	1.18 (0.99–1.48)	1.17 (0.98–1.41)	0.56
Non-HDL-C, mmol/l[a]	2.92 (2.26–3.69)	3.18 (2.53–4.08)	<0.01
TC, mmol/l[a]	4.06 (3.41–4.9)	4.4 (3.76–5.26)	<0.01
TG, mmol/l[a]	1.13 (0.9–1.44)	1.26 (0.96–1.68)	<0.01
TyG index[a]	8.73 (8.36–9.08)	8.81 (8.5–9.17)	0.09

Statins therapy prior to admission, n (%)	88 (80.7)	1015 (82.4)	0.9
Fibrates therapy prior to admission, n (%)	3 (2.7)	33 (2.7)	0.7
All- cause mortality, n (%)	15 (13.7)	180 (14.6)	0.8

[a]Data are shown as median (interquartile range) unless otherwise indicated. *P* <0.05 was considered significant.

Abbreviations: see Table 1

Table 3. Predictors of MACE in one year follow-up (univariate regression analysis)

Predictors of MACE in one year follow up	OR	95% CI	*P*-value
CAD diagnosed prior to admission	1.81	1.22–2.69	<0.01
eGFR, ml/min/1.73 m^2	0.98	0.97–0.99	0.03
LDL-C, mmol/l	0.73	0.59–0.89	<0.01
TC, mmol/l	0.77	0.64–0.92	<0.01

Abbreviations: CI, confidence interval; OR, odds ratio; others, see Table 1

Table 4. Predictors of MACE in one year follow up after exclusion of patients with higher FGL (univariate regression analysis)

Predictors of MACE in one year follow up	OR	95% CI	*P*-value
CAD diagnosed prior to admission	1.69	1.06–2.69	<0.01
eGFR, ml/min/1.73 m^2	0.98	0.96–0.99	<0.01
LDL-C, mmol/l	0.6	0.46–0.78	<0.01
TC, mmol/l	0.62	0.51–0.82	<0.01

Abbreviations: see Table 1 and Table 3

Table 5. Predictors of all- cause mortality in one year follow-up (univariate regression analysis)

Predictors of all- cause mortality in one year follow-up	OR	95% CI	*P*-value
Age	1.08	1.07–1.1	<0.01
BMI	0.91	0.87–0.94	<0.01
Gensini score	1.01	1–1.1	<0.01
eGFR, ml/min/1.73 m^2	0.97	0.96–0.98	<0.01
LDL-C, mmol/l	0.7	0.59–0.81	<0.01
TC, mmol/l	0.76	0.65–0.87	<0.01

Abbreviations: see Table 1 and Table 3

Table 6. Medical treatment at discharge

Medical treatment at discharge	Number of patients (%)
Statins, n (%)	1332 (99.4)
Ezetimibe, n (%)	166 (12.4)
Fibrates, n (%)	3 (0.2)
ACEI, n (%)	575 (42.9)
ARB, n (%)	324 (24.2)
B-adrenolitics, n (%)	1139 (85)
Calcium blockers, n (%)	753 (56.2)
Diuretics, n (%)	624 (46.6)
ASA, n (%)	1338 (99.8)
Clopidogrel, n (%)	1170 (87.3)
Prasugrel/Ticagrelor, n (%)	168 (12.7)

Abbreviations: see Table 1