Two-Hour Post-Load Plasma Glucose, a Biomarker to Improve the GRACE Score in Patients without Known Diabetes

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Abstract
Objective: To assess improvement in predictive performance of Global Registry of Acute Coronary Events risk score (GRS) by addition of a glucose matrix. Methods: 1,056 acute coronary syndrome (ACS) survivors without known diabetes had pre-discharge fasting (FPG) and 2-h post-load plasma glucose (2h-PG) measured. GRS was calculated. Major adverse cardiac events (MACE; death and non-fatal myocardial infarction) were recorded during follow-up. Cox proportional hazard regression predicted event-free survival. Likelihood ratio test, Akaike’s information criteria, continuous net reclassification index (NRI > 0), and integrated discrimination improvement (IDI) were used to test the additional prognostic value of glycaemic indices over GRS. Results: During a median follow-up of 36.5 months, 211 MACEs (20.0%), 96 deaths (9.1%), and 115 non-fatal re-infarctions (10.9%), occurred. 2h-PG, but not FPG, independently predicted MACE-free survival at all time points (HR 1.08, 95% CI 1.03–1.13, \( p = 0.002 \), at 3 years). Risk of MACE increased by 8–11% with every 1 mmol/L rise in 2h-PG. 2h-PG significantly improved the prognostic models containing GRS. Models containing GRS and 2h-PG yielded lowest corrected Akaike’s information criteria compared to that with only GRS. 2h-PG, but not FPG, improved NRI > 0 (NRI > 0 0.169, \( p = 0.028 \) at 3 years) and IDI (IDI of 0.66%, \( p = 0.018 \) at 3 years) significantly at all time points during the follow-up. Conclusions: 2h-PG, but not FPG, improves performance of GRS-containing models in predicting post-ACS prognosis in the short to medium term.

Introduction
The Global Registry of Acute Coronary Events (GRACE) risk score (GRS) predicts the risk of death and myocardial infarction (MI) in patients with acute coronary syndrome (ACS) [1–3]. The final GRACE model includes only 8 of the most prognostically significant base-
line variables to make the score clinically usable. The discriminatory performance of the GRACE model leaves room for improvement. Attempts have been made to improve the model by adding multiple blood biomarkers [4–10]. These are mostly emerging biomarkers that are not routinely measured. It is mostly a composite of different rather than a single biomarker that has provided incremental value over and above the GRACE score. Therefore, identifying a single biomarker that can be routinely measured not only to predict prognosis but also to improve the GRS would be useful.

A glycaemic matrix is notably absent in the GRACE score even though post-ACS prognosis is worse in patients with known diabetes mellitus (DM). Addition of fasting plasma glucose (FPG) [11, 12], admission plasma glucose (APG) [13–19], and glycosylated haemoglobin (HbA1c) [20, 21] has been variably successful in improving the GRACE model. The GRACE model predicts post-discharge prognosis up 4 years [3]. We investigate whether FPG and/or 2-h post-load plasma glucose (2h-PG) improves the performance of the GRS in predicting short- to intermediate-term major adverse cardiac events (MACE) in patients with MI but without known DM.

### Materials and Methods

The study methods have been described previously [22]. This analysis includes consecutive post-MI survivors without known DM who underwent a routine pre-discharge oral glucose tolerance test (OGTT) with standard follow-up after discharge. Information on demographics, risk factors for coronary artery disease, past medical history including history of previous MI and revascularisation, prescribed medications, haemodynamic parameters, troponin I, renal function test, Killip class, and presence of ST-segment depression GRs for risk of death or MI from discharge to 6 months were collected from the Myocardial Infarction National Audit Project (MINAP) database. All participants underwent post-discharge OGTT on/after the third day of admission. Venous plasma glucose was measured after ≥8 h of overnight fast and 2 h after administration of 75 g glucose in 200 mL water. Clinically unstable patients were tested later. We excluded patients who were transferred out of our centre before the OGTT or did not tolerate it. Glucose was measured using the glucose oxidase method. The patients diagnosed with impaired glucose tolerance (IGT) and new DM were referred to the endocrinologist for appropriate management.

MI was defined as per the universal definition [23]. Patients were labelled “known DM” if they were aware of the diagnosis before admission, had it documented in their medical records, or were on anti-DM treatment. Admission HbA1c was not measured as it was not recommended in the guidelines at the time of data collection [24–26]. Patients with pre-existing diabetes were excluded. The glucometabolic states were defined as normal glucose tolerance: FPG <6.1 mmol/L and a 2-h PG <7.8 mmol/L; impaired fasting glucose: FPG 6.1–6.9 mmol/L and 2-h PG <7.8 mmol/L; IGT: FPG <7 mmol/L and 2-h PG 7.8–11 mmol/L, and new DM: FPG ≥7.0 and/or 2-h PG ≥11.1 mmol/L.

All participants were followed up for a median of 36 months for outcomes. Hospital and general practice records were reviewed ensuring complete follow-up. The first occurrence of MACE de-
fined as death or non-fatal re-infarction (the only events predicted by GRS) was obtained from the hospital and general practice databases and confirmed by the office of public health intelligence. As routinely collected anonymised data on standard clinical practice were being retrospectively analysed, the East Yorkshire and North Lincolnshire Research Ethics Committee waived the need for formal ethical approval and patient consent [22].

Statistics
Continuous variables are presented as medians (interquartile range, IQR) and categorical variables as counts and proportions. The baseline characteristics of the patients grouped as above and below the median 2h-PG (8.1 mmol/L) were compared using the Mann-Whitney test for non-parametric continuous variables and the χ² test for categorical variables. Event-free survival was compared between groups using the Kaplan-Meier method. The dataset was censored at 6 months, and 1, 2, and 3 years, and analysed using multivariate Cox proportional hazard regression (MedCalc Statistical Software version 17.0.4, Ostend, Belgium). Gender, smoking status, hypercholesterolaemia, hypertension, history of MI and revascularisation, discharge diagnosis and medications, GRS, FPG, and 2h-PG were “entered” into the model. Multicollinearity was tested (MedCalc Statistical Software version 17.0.4), and variables with variance inflation factor < 4 were included in the same model. Hazard ratios (HRs) and 95% CIs are reported.

The χ² likelihood ratio test was used to compare nested models to determine if the logistic regression models including GRS and FPG or 2h-PG provided a significantly better fit than those with GRS alone. Akaike’s information criterion (AIC) was used to estimate the probability that a given nested or non-nested model including GRS and FPG and/or 2h-PG was the “best”-fitting model of those studied.

FPG and 2h-PG were added, individually and in combination, into logistic regression models containing GRS along with other covariates to calculate the predicted probabilities of MACE at each time point. The incremental predictive value of adding 2h-PG to models with FPG was analysed from these predicted probabilities by comparing the area under the receiver-operating characteristic (ROC) curve (AUC) (MedCalc Statistical Software version 17.0.4), using the category-free continuous net reclassification index (NRI)\(^{(6)}\) and integrated discrimination improvement (IDI). The event and non-event NRI were defined as net percentage of persons with and without the event of interest correctly assigned a higher and lower predicted risk, respectively. The overall NRI is the sum of the event and non-event NRI reported as a number. The IDI was defined as the mean difference in predicted risks between those with and without events.

Results
Baseline characteristics of the patients grouped as below or equal to (group 1) and above (group 2) the median 2h-PG (8.1 mmol/L) are shown in Table 1. OGTT was done on day 3 or later in 97.6% of patients; 61.6% of ST-elevation myocardial infarction (STEMI) and 60.3% of non-STEMI (NSTEMI) patients had glucose measured on day 4 or later after admission. The timing of OGTT was similar in the NSTEMI (median 4.0; IQR 6–3) and STEMI patients (median 4.0; IQR 5–3, \(p = 0.420\)). Patients who had their OGTT on or before day 3 had lower FPG (median 5.0; IQR 5.4–4.7 vs. median 5.1; IQR 5.6–4.8, \(p = 0.002\)) but similar 2h-PG (median 8.1; IQR 10.4–6.4 vs. median 8.0; IQR 10.5–6.5, \(p = 0.762\)) compared to those done later. The patients with STEMI had higher FPG (median 5.1; IQR 5.6–4.8 vs. median 5.05; IQR 5.5–4.7, \(p = 0.020\)) but similar 2h-PG (median 8.2; IQR 10.4–6.6 vs. median 8.0; IQR 10.5–6.4, \(p = 0.323\)) than the NSTEMI patients.

Outcomes
During the median follow-up of 36.5 months, there were 211 MACE (20.0%), 96 deaths (9.1%), and 115 non-fatal re-infarctions (10.9%). Group 2 had higher MACE than group 1 (HR 1.53, 95% CI 1.17–2.01, \(p = 0.002\)). The 2h-PG and GRS, but not FPG, independently predicted MACE at all time points (Table 2). The risk of MACE increased by 8–11% at various time points for each 1 mmol/L rise in 2h-PG.

Addition of the 2h-PG, but not FPG, significantly improved the ability of a model including GRS to predict MACE at all time points during follow-up (Table 3). Models containing GRS and 2h-PG yielded the lowest corrected AIC and highest Akaike weight and evidence ratio compared to those with GRS alone and GRS with FPG (Table 3) suggesting that the model with GRS and 2h-PG is the “best”-fitting model compared to the other models tested.

2h-PG, but not FPG, significantly improved the net reclassification of the GRS-containing model alone in predicting events during follow-up (Table 4). 2h-PG significantly improved NRI\(^{(3)}\) by 17–23% at 2 and 3 years but not at 6 months and 1 year. Addition of FPG did not change reclassification. Integrated discrimination at all time points was similarly improved by 2h-PG (Table 4). The IDI was 0.41–0.66% at the various time points.

The c-statistic for the model containing GRS only, 2h-PG only, and GRS and 2h-PG were 0.73 (95% CI 0.71–0.76, \(p < 0.0001\)), 0.70 (95% CI 0.67–0.73, \(p < 0.0001\)), and 0.74 (95% CI 0.71–0.77, \(p < 0.0001\)) respectively. The AUC for the GRS-only model was better than the 2h-PG-only model (ΔAUC 0.0320, \(p = 0.028\)). Addition of 2h-PG to the GRS-only model did not improve the c-static (ΔAUC 0.0053, \(p = 0.295\)) but did so when GRS was added to the 2h-PG-only model (ΔAUC 0.0373, \(p = 0.003\)).
Discussion/Conclusion

We show that 2h-PG, measured $\geq 3$ days after the event, would improve performance of a model including GRS in predicting post-MI prognosis in the short- to intermediate-term in patients without known DM. GRS accurately predicts post-MI events in the short and long term [2, 3]. Multiple blood biomarkers have been added to the model to improve its performance [4–10]. Its performance improves when a composite score created mostly from multiple novel biomarkers that are not routinely measured is added. These approaches are too cumbersome to be used clinically and possibly costly with marginal gains. This study suggests that 2h-PG as single glycaemic matrix added to the GRS would improve its performance.

As the post-MI prognosis in patients with DM is inferior to those without, a glycaemic matrix would be a natural choice as an additional biomarker that could improve predictive performance of models containing GRS. The GRS model does not include DM history or a glycaemic matrix as a variable [2, 3]. In the GRACE registry DM, as a dichotomous categorical variable, did not independently predict the 6-month post-discharge events [2]. However, increasing FPG increased the risk of in-hospital mortality irrespective of a history of DM [11].

| Covariate                          | 6 months HR    | 6 months 95% CI | 6 months p value | 1 year HR    | 1 year 95% CI | 1 year p value |
|------------------------------------|----------------|----------------|-----------------|-------------|---------------|----------------|
| GRACE score                        | 1.01           | 1.00–1.01      | 0.006           | 1.01        | 1.01–1.02     | <0.0001        |
| 2h-PG                              | 1.11           | 1.03–1.20      | 0.006           | 1.10        | 1.03–1.16     | 0.003          |
| Hypercholesterolaemia              | 0.76           | 0.43–1.32      | 0.326           | 0.75        | 0.48–1.16     | 0.200          |
| Previous MI                        | 1.29           | 0.69–2.41      | 0.431           | 1.46        | 0.90–2.35     | 0.121          |
| Discharged without β-blocker      | 1.60           | 0.97–2.63      | 0.066           | 1.42        | 0.95–2.12     | 0.091          |
| FPG                                | 0.76           | 0.57–1.03      | 0.079           | 0.75        | 0.59–0.97     | 0.026          |
| Discharged without clopidogrel     | 1.23           | 0.66–2.29      | 0.512           | 1.30        | 0.79–2.13     | 0.296          |
| Hypertension                       | 1.12           | 0.71–1.77      | 0.629           | 1.09        | 0.76–1.57     | 0.628          |
| Previous revascularisation         | 1.08           | 0.51–2.28      | 0.843           | 0.93        | 0.51–1.69     | 0.804          |
| Discharged without ACEI            | 1.69           | 0.98–2.89      | 0.058           | 1.55        | 1.00–2.42     | 0.050          |
| Discharged without aspirin         | 1.35           | 0.64–2.85      | 0.436           | 1.29        | 0.70–2.40     | 0.418          |
| Gender, female                     | 1.12           | 0.71–1.80      | 0.629           | 1.20        | 0.82–1.74     | 0.351          |
| Discharge diagnosis of STEMI        | 1.49           | 0.95–2.33      | 0.083           | 1.28        | 0.89–1.83     | 0.184          |
| Discharged without statin          | 1.19           | 0.63–2.25      | 0.587           | 1.03        | 0.60–1.76     | 0.918          |
| Current smoker                     | 0.77           | 0.46–1.29      | 0.320           | 0.82        | 0.55–1.22     | 0.328          |

|                  | 2 years HR    | 2 years 95% CI | 2 years p value | 3 years HR  | 3 years 95% CI | 3 years p value |
|------------------|---------------|----------------|----------------|-------------|---------------|----------------|
| GRACE score      | 1.01          | 1.01–1.02      | <0.0001        | 1.01        | 1.01–1.02     | <0.0001        |
| 2h-PG            | 1.08          | 1.02–1.13      | 0.004          | 1.08        | 1.03–1.13     | 0.002          |
| Hypercholesterolaemia | 0.73     | 0.50–1.07      | 0.107          | 0.71        | 0.50–1.01     | 0.059          |
| Previous MI      | 1.47          | 0.97–2.21      | 0.067          | 1.45        | 1.00–2.11     | 0.052          |
| Discharged without β-blocker      | 1.29          | 0.91–1.83      | 0.151          | 1.39        | 1.01–1.91     | 0.044          |
| FPG               | 0.91          | 0.75–1.09      | 0.291          | 0.88        | 0.74–1.05     | 0.159          |
| Discharged without clopidogrel     | 1.39          | 0.92–2.09      | 0.116          | 1.37        | 0.93–2.00     | 0.108          |
| Hypertension      | 1.02          | 0.75–1.39      | 0.907          | 1.11        | 0.83–1.48     | 0.476          |
| Previous revascularisation         | 1.16          | 0.72–1.88      | 0.541          | 1.21        | 0.79–1.88     | 0.384          |
| Discharged without ACEI            | 1.22          | 0.82–1.81      | 0.324          | 1.38        | 0.96–1.98     | 0.079          |
| Discharged without aspirin         | 1.46          | 0.87–2.43      | 0.149          | 1.46        | 0.90–2.36     | 0.125          |
| Gender, female     | 1.22          | 0.89–1.69      | 0.218          | 1.11        | 0.82–1.50     | 0.514          |
| Discharge diagnosis of STEMI        | 1.13          | 0.83–1.53      | 0.454          | 1.12        | 0.84–1.49     | 0.454          |
| Discharged without statin          | 1.18          | 0.75–1.87      | 0.476          | 0.93        | 0.60–1.44     | 0.740          |
| Current smoker     | 0.82          | 0.58–1.16      | 0.264          | 0.97        | 0.71–1.32     | 0.834          |
In the absence of OGTT, it is uncertain whether the raised FPG or undetected raised 2h-PG either to the IGT or DM range affected the outcomes in these patients. The 6-month post-ACS survival was affected by FPG only if it was above the diagnostic threshold for diabetes [11].

FPG, APG, HbA$_1c$, and AGT individually predicts adverse post-ACS prognosis in patients without known DM. The 2h-PG is a better predictor than both APG and FPG [27]. FPG, APG, and HbA$_1c$, after adjusting for GRS, predict post-MI outcomes in some [12, 13, 16, 19, 20] but not other [11, 14, 15, 17, 18, 21] studies. Attempts at using APG [16–18], FPG [12, 28], and HbA$_1c$ [20] to improve the predictive ability of models containing GRS have yielded conflicting results. This study suggests that in patients without known DM, 2h-PG but not FPG improves the performance of GRS-containing models in predicting post-MI prognosis in the short to medium term. Aronson et al. [12] shows that FPG predicts mortality after MI and improves the prognostic models containing GRS. This study is somewhat different from ours. Most (73%) patients in their study had STEMI, FPG was measured in the first 24 h of admission, and post-load glucose was not measured [12]. In contrast, STEMI was diagnosed in 44% of our patients, and glucose was measured a lot later. As a binary dichotomous variable, troponin in the GRS model does not account for the effect of the degree of myocardial infarction on prognosis. FPG is higher when measured within 24 h of MI than later and following STEMI compared to NSTEMI [29–31]. The effect of FPG on the predictive performance of the model in this study [12] may have been exaggerated due to the higher levels of FPG measured early after STEMI combined with GRS unaffected by the higher volume of myocardial infarction in STEMI. It is also unclear whether this effect would persist if 2h-PG was included in the models. The morbidity associated with 2h-PG rather than FPG in our study could be due to the progression of atherosclerosis seen after challenge but not with fasting hyperglycaemia [32].

In the absence of HbA$_1c$, we are unable to compare the effect of all the glycaemic matrices on prognosis. None of the patients had HbA$_1c$ measured as routine screening for undiagnosed diabetes as HbA$_1c$ was not recommended in the EASD Guidelines 2007 [24]. On the contrary, screening for diabetes using a non-invasive risk score

### Table 3. Akaike’s information criteria and likelihood ratio test to determine the best-fitting model for predicting MACE at different time points during the follow-up (see text for abbreviations)

| Model          | Akaike’s information criteria | Likelihood ratio test |
|----------------|-------------------------------|-----------------------|
|                | $\Delta$AICc | wi | $\chi^2$ | df | $p$ value |
| 6 months       |                |     |         |   |          |
| GRS            | 581.46         | 0.29 | 2.16 | 0.003 |
| GRS + 2h-PG    | 579.00         | 0.70 | 7.40 | 0.005 |
| GRS + FPG      | 583.00         | 1.00 | 1.04 | 0.308 |
| 1 year         |                |     |         |   |          |
| GRS            | 768.83         | 0.23 | 1.16 | 0.061 |
| GRS + 2h-PG    | 766.99         | 0.57 | 2.91 | <0.001 |
| GRS + FPG      | 769.13         | 0.20 | 1.00 | 0.603 |
| 2 years        |                |     |         |   |          |
| GRS            | 902.83         | 0.07 | 2.62 | <0.001 |
| GRS + 2h-PG    | 897.71         | 0.90 | 33.89 | <0.001 |
| GRS + FPG      | 904.75         | 0.03 | 1.00 | 0.820 |
| 3 years        |                |     |         |   |          |
| GRS            | 966.37         | 0.07 | 2.76 | <0.001 |
| GRS + 2h-PG    | 961.31         | 0.90 | 34.55 | <0.001 |
| GRS + FPG      | 968.40         | 0.03 | 1.00 | 0.05  |

AICc, corrected Akaike’s information criteria; $\Delta$AICc, delta AICc is a measure of each model relative to the best model; wi, Akaike weights, the ratio of $\Delta$AICc values for each model relative to the whole set; $w_j/w_i$, evidence ratios compare the wi of the “best” model and competing models to test the extent to which it is better than another.
and OGTT was recommended. Whether HbA1c would be a useful biomarker to predict post-MI prognosis is debated. HbA1c has been shown to predict post-ACS prognosis in some [20, 33–35] but not all studies [36–40]. The effect of APG, FPG, 2h-PG, and HbA1c on post-MI prognosis in patients without known diabetes has been compared in a few studies [36, 39, 41, 42]. The 2h-PG, but neither FPG nor HbA1c, predicted outcome in EURO- ASPIRE IV [39]. HbA1c ≥6.5%, when included in the same model as known DM, did not increase mortality, and new DM diagnosed by OGTT did, even when the HbA1c was <6.5% in another study [36]. Kowalczyk et al. [41] suggests the usefulness of HbA1c in patients with IGT and new DM, but they do not report the effect of HbA1c on prognosis of patients without. The Emerging Risk Factors Collaboration [43] published a large epidemiological dataset including individuals without DM or cardiovascular disease at baseline suggesting that additional assessment of HbA1c provided little incremental benefit for predicting the risk of cardiovascular disease over and above other glycaemic matrices.

The 2h-PG did not increase the c-statistic of the model containing GRS. Improving c-statistics of models containing powerful variables as GRS may be difficult as ΔAUC heavily depends on the strength of performance of the underlying clinical model. NRI >0 and IDI, tests de-

Table 4. Net reclassification improvement (NRI) for model improvement with the addition of 2h-PG or FPG to the Global Registry of Acute Coronary Events risk score (GRS)

|                  | GRS vs. GRS and 2h-PG | GRS vs. GRS and FPG |
|------------------|-----------------------|---------------------|
|                  | NRIe/NRIe total p value | NRIe/NRIe total p value |
| **6 months**     |                       |                      |
| ↑                | 37/371 408 0.043 0.043 | 50/558 608 0.043 0.043 |
| ↓                | 48/600 648 0.043 0.043 | 35/413 448 0.043 0.043 |
| Total            | 85/971 1,056 0.043 0.043 | 85/971 1,056 0.043 0.043 |
| NRI >0           | –0.129/0.236 0.347 0.043 | 0.176/–0.149 0.027 0.043 |
| **1 year**       |                       |                      |
| ↑                | 58/364 422 0.043 0.043 | 81/522 603 0.043 0.043 |
| ↓                | 75/559 634 0.043 0.043 | 52/401 453 0.043 0.043 |
| Total            | 133/923 1,056 0.043 0.043 | 133/923 1,056 0.043 0.043 |
| NRI >0           | –0.128/0.211 0.368 0.043 | 0.218/–0.131 0.087 0.043 |
| **2 years**      |                       |                      |
| ↑                | 90/332 422 0.043 0.043 | 71/350 421 0.043 0.043 |
| ↓                | 92/542 634 0.043 0.043 | 111/524 635 0.043 0.043 |
| Total            | 182/874 1,056 0.043 0.043 | 182/874 1,056 0.043 0.043 |
| NRI >0           | –0.011/0.240 0.029 0.005 | –0.220/0.199 0.021 0.799 |
| **3 years**      |                       |                      |
| ↑                | 99/325 424 0.043 0.043 | 128/495 623 0.043 0.043 |
| ↓                | 112/520 632 0.043 0.043 | 83/350 433 0.043 0.043 |
| Total            | 211/845 1,056 0.043 0.043 | 211/845 1,056 0.043 0.043 |
| NRI >0           | –0.062/0.231 0.028 0.005 | 0.213/–0.172 0.042 0.588 |

FPG, fasting plasma glucose; 2h-PG, 2h post-load glucose; NRIe/NRIe, event/non-event NRI; IDIe/IDIe, event/non-event integrated discrimination index; ↑/↓, number of subjects where the predicted probability of the adverse events as predicted by the model B vs. model A increased (↑) or decreased (↓).
vised to deal with this anomaly, improved when 2h-PG is added to GRS. The NRI > 0 did not change for the 6-month and 1-year time points, but the IDI did. The NRI > 0 counts the individuals with and without events whose calculated risk changes on addition of a variable into a model. IDI measures the amount of change in calculated risk for each individual with and without events incorporating both the direction and the extent of change in calculated risk, making it more meaningful than NRI > 0.

The study is limited by its retrospective observational nature. Deaths were recorded from the general practice database linked to the national death register. Although local records are regularly updated, some re-infarctions admitted to other hospitals may have been missed. Information not available had to be excluded from statistical models. Inclusion of a mainly Caucasian population could affect its generalizability. Stress hyperglycaemia is less likely to have affected our result as the OGTT was done ≥3 days after the event. The stress response to an acute event subsides within 2–5 days with no further decrease thereafter [31]. The effect of random fluctuation in glycaemia, however, cannot be excluded. Although reproducibility of the OGTT results and its relation to a long-term glucometabolic state would be important for the diagnosis of DM, its relevance to assessing post-MI prognostic risk is less.

This study concludes that in patients without known diabetes, 2h-PG but not FPG, improves the performance of GRS-containing models in predicting post-MI prognosis in the short to medium term. Thus 2h-PG can be used as an additional prognostic biomarker in addition to GRS in these patients. There is an ongoing debate as to the choice of a glucose matrix for the detection of hyperglycaemia in this high-risk population. It may be reasonable to choose 2h-PG, the one that minimises the risk of missing the diagnosis and is additionally capable of providing prognostic information despite it being cumbersome to measure, in favour of HbA1c that is simpler and feasible to measure, and deemed sufficient for use in the low-risk general population for epidemiological purposes especially since there is no evidence of superiority of HbA1c over 2h-PG in predicting prognosis. Thus, an appropriately timed pre-discharge OGTT may be recommended for all patients without known diabetes admitted with MI.

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Statement of Ethics

As routinely collected anonymised data on standard clinical practice were being retrospectively analysed, the East Yorkshire and North Lincolnshire Research Ethics Committee waived the need for formal ethical approval and patient consent.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contribution

J.J. and S.C. conceived and designed the study. A.G. and S.C. contributed to acquisition of data and drafted the manuscript. S.C. analysed and interpreted the data. J.J., S.C., and T.S. critically revised the manuscript. A.G., J.J., S.C., and T.S. gave final approval and agree to be accountable for all aspects of work ensuring integrity and accuracy.

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