

Socioeconomic disparities in the management and outcomes of acute myocardial infarction

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Abstract

Background

Patients from lower socioeconomic status areas have poorer outcomes following acute myocardial infarction (AMI); however, how ethnicity modifies such socioeconomic disparities is unclear.

Methods

Using the United Kingdom (UK) Myocardial Ischaemia National Audit Project (MINAP) registry, we divided 370,064 AMI patients into quintiles based on Index of Multiple Deprivation (IMD) score, comprising seven domains including income, health, employment and education. We compared White and 'Ethnic-minority' patients, comprising Black, Asian and Mixed-ethnicity patients (as recorded in MINAP), further analyses were compared constituents of the Ethnic-minority group. Logistic regression models examined the role of the IMD, ethnicity, and their interaction, on odds of in-hospital mortality.

Results

More patients from the most deprived quintile (Q5) were from Ethnic-minority backgrounds (Q5; 15% vs. Q1; 4%). In-hospital mortality (OR: 1.10, 95% CI: 1.01-1.19, $p=0.025$) and MACE (OR: 1.07, 95% CI: 1.00-1.15, $p=0.048$) were more likely in Q5, and MACE was more likely in Ethnic-minority patients ((OR: 1.40, 95% CI: 1.00-1.95, $p=0.048$) vs. White; OR: 1.05, 95% CI: 0.98-1.13, $p=0.027$). In subgroup analyses, Black patients had the highest in-hospital mortality within the most affluent quintile (Q1) (Black; 0.079, 95% CI 0.046-0.112, $P<0.001$, White; 0.062, 95% CI (0.059-0.066, $P<0.001$), but not in Q5 (Black; 0.065, 95% CI (0.054-0.077, $P<0.001$, White; 0.065, 95% CI (0.061-0.069, $P<0.001$)).

Conclusion

Patients with a higher deprivation score were more often from an Ethnic-minority background, more likely to suffer in-hospital mortality or MACE when compared with the

most affluent quintile, and this relationship was stronger in Ethnic minorities compared to White patients.

Three key questions

What is already known on this topic:

Patients from more socioeconomically deprived backgrounds typically have poorer management and outcomes following acute myocardial infarction, but it is not known whether ethnicity influences this in the United Kingdom.

What this study adds:

Patients from the most deprived quintile according to IMD Score in the UK were more often from Ethnic minority background. In-hospital mortality and MACE were highest in the most socioeconomically deprived patients despite multivariate adjustment for clinical characteristics and management strategy, and this was more pronounced in Ethnic minority patients, despite rates of guideline-directed medical therapy (GDMT), invasive coronary angiography (ICA) and revascularisation by percutaneous coronary intervention (PCI) and coronary artery bypass graft surgery (CABG) being higher in Ethnic minority patients across the socioeconomic spectrum.

How this study might affect research, practice or policy –

Even within a universal healthcare system, there is still a disparity in acute myocardial infarction outcomes according to socioeconomic status, which is more notable in Ethnic minority patients, despite aspects of management such as GDMT, ICA and PCI rates being better in ethnic minority patients. Further work needs to be targeted towards addressing the social determinants of health, such as education, employment and structural racism, particularly in the areas of highest socioeconomic deprivation.

Introduction

The United Kingdom (UK) uses the Index of Multiple Deprivation (IMD), comprising 37 indicators of deprivation, to create reports detailing the geographical distribution of the deprivation (1). Prior data from the United States (US) has suggested that patients from the most deprived socioeconomic groups have poorer outcomes following acute myocardial infarction (AMI), lower rates of guideline-directed medical therapy (GDMT) prescription, and higher rates of mortality (2). Similarly, a comparison between the lowest and highest quintiles of the IMD within the UK shows a positive association between social deprivation and higher mortality from AMI, heart failure (HF), and stroke (3, 4).

There is growing understanding of the importance of social determinants of health (SDOH) as a risk factor for coronary artery disease (CAD), with the American Heart Association (AHA) defining race and ethnicity as a critical SDOH (5). Race and ethnicity can act as a surrogate for the SDOH, and there is understanding of the role of structural racism in discrepancies in incidence and outcomes of cardiovascular disease in the US (6). Given that the UK has a publicly-funded, universal healthcare system, less variation in the outcomes of AMI according to ethnicity or race would be expected compared to the US (7). Using the national UK heart attack registry, the Myocardial Ischaemia National Audit Project (MINAP), we investigated the effect of socioeconomic deprivation (as measured by IMD score) and ethnicity on the processes of care and clinical outcomes associated with AMI in the UK.

Methods

Study design:

We used the MINAP, a prospective national registry of patients admitted to hospitals in the UK with acute coronary syndrome (ACS) (8). The MINAP dataset consists of 130 variables, including demographics, comorbidities, management strategies, pharmacotherapy, in-hospital clinical outcomes, and discharge diagnosis (9). Data are submitted by hospital clinical and clerical staff, and approximately 90,000 pseudonymised records annually are uploaded to the National Institute for Cardiovascular Outcomes Research (NICOR).

Study population:

We included patients admitted with a diagnosis of AMI in any of the 230 participating hospitals in England and Wales between 1st January 2010 to 31st March 2017. The discharge diagnosis of AMI was determined by local clinicians according to presenting history, examination, and investigations in keeping with the consensus document of the Joint European Society of Cardiology (ESC) and American College of Cardiology (ACC) (10). Patients were excluded if they had missing data in key variables for investigation; IMD score, in-hospital mortality, and major adverse cardiovascular events (MACE). Furthermore, for individuals with multiple AMI admissions during the period of interest, only the first admission was included (Figure 1).

This constituted a final cohort of 370,064 patients with AMI, who were then divided into the five quintiles of deprivation, according to their IMD 2010 score, a variable recorded in the MINAP registry; Quintile 1; the most affluent group (an IMD 2010 score ≤ 8.49), Quintile 2, Quintile 3, Quintile 4; and Quintile 5; the most deprived ≥ 34.18) %. The IMD

2010 score comprised seven major domains: income, employment, and health deprivation, disability, education, skills and training, barriers to housing and services, crime, and environment (Supplementary Table 3).

Subgroup Analysis:

Subgroup analysis was performed to compare the patients' baseline characteristics, management strategies, and outcomes according to ethnicity. For descriptive tables, we formed two groups according to ethnicity status as recorded in the MINAP dataset; White and Ethnic minority (including Black, Asian, Mixed, and other non-White ethnic minorities). The Ethnic minority group as captured in MINAP included those who were recorded as Black (including Caribbean, African, Black British, or any other Black background), Asian (including Indian, Pakistani, Bangladeshi, Asian British, or any other Asian Background) and other Non-White ethnicities.

Outcomes:

Primary

Primary outcomes of interest included in-hospital all-cause mortality and MACE (composite endpoint of in-patient all-cause mortality and reinfarction).

Secondary

Secondary outcomes of interest included cardiac mortality (death attributable to myocardial ischaemia or infarction, heart failure (HF), and cardiac arrest of unknown cause) and major bleeding.

Statistical analysis:

Demographics, clinical characteristics, and crude outcomes of patients by quintile of deprivation were compared using Pearson's chi-square test for categorical variables. Continuous variables were compared using Student's t-test, if normally distributed, and using the Wilcoxon Rank Sum test if not. The normality of distribution was assessed using the Shapiro-Wilk test. Continuous variables are presented as medians and interquartile ranges (IQR) and categorical variables by proportions. Multiple imputations with chained equations (MICE) were used to impute values for variables with missing data. MICE is considered best practice when dealing with missing data and can provide unbiased estimates even when levels of missing data are significant (11). Multivariable, multilevel logistic regression analysis was applied to imputed datasets, for each binary outcome of interest, to estimate the risk of adverse outcomes between groups. Estimates were combined using Rubin's rules (12). Logistic regression models were fitted using maximum likelihood estimation and were adjusted for age, sex, heart rate, blood pressure, history of AMI, co-morbid conditions (family history of coronary artery disease, hypertension, hypercholesterolaemia, diabetes, smoking, history of asthma or COPD, history of CVA or PVD), pharmacotherapy (prescription of low molecular weight heparin (LMWH), unfractionated heparin (UFH), warfarin, GP 2b/3a inhibitor, IV nitrate, furosemide, aldosterone antagonist, fondaparinux, angiotensin-converting enzyme inhibitor/angiotensin receptor blockers, aspirin, P2Y12 inhibitor, and statins), cardiac arrest and procedures including invasive coronary angiography (ICA) and revascularisation (by PCI or CABG during admission). A random intercept was added for hospital, accounting for the nested structure of data in the model. A second model was run, using the margins function on Stata 14.2 to illustrate interaction between IMD Quintile and ethnicity classification on adjusted in-hospital mortality. Statistical analysis was undertaken using Stata 14.2 (College Station, Texas, USA). All statistical analyses were two-tailed, with an alpha of 5% used.

Results

Baseline clinical characteristics between different deprivation quintiles

Between January 2010 to March 2017, there were 664,740 patients admitted to hospitals in England and Wales with a diagnosis of AMI. Applying exclusion criteria (Figure 1) produced a final cohort of 370,064 patients. Patients from the most deprived quintile had a significantly lower median age (years) (Quintile 5; 66 vs. Quintile 1; 72) and were more frequently female (Quintile 5; 34% vs. Quintile 1; 32%) (Table 1). Our key findings are summarised in Figure 2.

Management strategies and Outcomes of different deprivation quintiles

Patients from the most deprived quintile were less likely to be discharged on P2Y12 inhibitors (Quintile 5; 90% vs. Quintile 1; 92%) or beta-blockers (Quintile 5; 80% vs. Quintile 1; 82%), with no other significant differences in the provision of GDMT (Table 2). Our unadjusted data by deprivation noted no significant differences in our primary and secondary outcome measures.

Clinical characteristics of different deprivation quintiles according to ethnicity

Patients from the most deprived quintile were more likely to be from an Ethnic minority background (Quintile 5; 14% vs. Quintile 1; 4%) (Table 3). Ethnic minority patients had a lower median age (years) (Quintile 5; White 66, Ethnic minority 61 vs. Quintile 1; White 72, Ethnic minority 69), were less likely to be current smokers (Quintile 5; White 41%, Ethnic minority 31% vs. Quintile 1; White 18%, Ethnic minority 17%) but were more likely to have higher rates of metabolic risk factors such as DM (Quintile 5; White 23%, Ethnic minority 46% vs. Quintile 1; White 17%, Ethnic minority 33%), hypertension (Quintile 5;

White 49%, Ethnic minority 60% vs. Quintile 1; White 50%, Ethnic minority 54%) and hypercholesterolaemia (Quintile 5; White 34%, Ethnic minority 46% vs. Quintile 1; White 32%, Ethnic minority 42%)

Management strategies and outcomes of different deprivation quintiles according to ethnicity

Ethnic minority patients were more likely to undergo ICA in each quintile (Quintile 5; White 72%, Ethnic minority 82% vs. Quintile 1; White 74%, Ethnic minority 85%) and revascularisation (Quintile 5; White 52%, Ethnic minority 64% vs. Quintile 1; White 55%, Ethnic minority 69%) (Table 4). In unadjusted data, they were less likely to experience in-hospital mortality (Quintile 5; White 6%, Ethnic minority 4% vs. Quintile 1; White 7%, Ethnic minority 4%).

Adjusted outcomes for different quintiles of deprivation

After adjusting for differences in baseline covariates and management strategy, in-hospital mortality (OR: 1.10, 95% CI: 1.01-1.19, $p=0.025$) and MACE (OR: 1.07, 95% CI: 1.00-1.15, $p=0.048$) was more likely in the most deprived quintile, compared with the most affluent quintile (Table 5).

Effect of IMD Quintile and ethnicity on adjusted in-hospital mortality

Figure 3 and Supplementary Table 4 show adjusted in-hospital mortality for each ethnicity included in MINAP, showing minimal variation between ethnicities in the most deprived quintile but showing that Black patients had the highest in-hospital mortality within the most affluent quintile (Black; 0.079, 95% CI 0.046-0.112, $P<0.001$, White; 0.062, 95% CI (0.059-0.066, $P<0.001$). There is no significant difference in in-hospital mortality according

to ethnicity in the most deprived Quintile ((White; 0.065 (95% CI; 0.061-0.069, P<0.001), Ethnic minority; 0.064 (95% CI; 0.058-0.070, P<0.001)) (Figure 3 and Supplementary Table 5). Within the most affluent quintile, Ethnic minority patients have a slightly lower in-hospital mortality ((White; 0.062 (95% CI; 0.059-0.066, P<0.001), Ethnic minority; 0.057 (95% CI; 0.048-0.065, P<0.001)).

Adjusted outcomes for different quintiles of deprivation according to ethnicity

In-hospital mortality was not more likely for White patients in the most deprived quintile compared with the most affluent quintile (OR: 1.07, 95% CI: 0.99-1.17, p=0.095), furthermore, the results for MACE and in-hospital cardiac mortality were not statistically significant (Supplementary Table 1). For Ethnic minority patients, in-hospital MACE was more likely in our most deprived quintile (OR: 1.40, 95% CI: 1.00-1.95, p=0.095 (Supplementary Table 2).

Discussion

Our nationwide cohort study reports that patients in the most deprived areas in the UK are typically younger, more frequently from an Ethnic minority background, and have worse cardiometabolic risk factor profiles. Secondly, after adjusting for baseline demographics and management strategy, MACE and in-hospital mortality are more likely in the most deprived quintile compared with the most affluent. Thirdly, there are important differences regarding ethnicity and its interaction with deprivation. Ethnic minority patients tended to be younger and have more co-morbid conditions but were more likely to undergo ICA and revascularisation regardless of deprivation index. Notably, in our adjusted multivariate regression models, the effect of socioeconomic deprivation appears more marked in the Ethnic minority cohort, with MACE and cardiac mortality being significantly more likely in Ethnic minority patients from the most deprived quintile. Importantly, when assessing the intersection of deprivation and ethnicity, even in the most affluent quintile in the UK, Black patients were significantly more likely to experience in-hospital mortality than White patients.

Prior studies looking at the role of socioeconomic deprivation in the outcomes of AMI have important limitations. An analysis from the US National Inpatient Survey (NIS) demonstrated increased mortality and reduced invasive management of AMI in the most deprived quartile when compared to the most affluent. Notably, this study used median household income (MHI) only as a measure of deprivation, a less comprehensive deprivation measure than the IMD score (13). The disparity of outcomes in insurance-based healthcare

systems such as the US is not surprising, but this persists across more universal healthcare systems in Europe where *Sundquist et al* demonstrated the increased incidence of CAD in those from the most deprived neighbourhoods impact using the Swedish Population Register (14).

Our findings are consistent with those of other nationwide studies investigating the role of deprivation on AMI outcomes. *Thorne et al.* demonstrated in an analysis of AMI patients from Wales that social deprivation was an independent predictor of thirty-day mortality (3). An analysis of a Norwegian national registry highlights how outcomes in those from more socioeconomically deprived regions have poorer outcomes from AMI despite similar access to healthcare services (15), the presence of persistent socioeconomic disparities in outcomes even in the most advanced healthcare settings, and '*egalitarian*' societies adds further evidence to the idea that these disparities in outcomes are more complex than comorbidity prevalence and access to certain therapies.

We must consider the impact of the SDOH on AMI outcomes, which are defined by the AHA as; socioeconomic position (SEP), race and ethnicity, social support, access to medical care, and residential environments (5). *Jilani et al.* have described how 'social adversity,' as defined by adverse SDOH, is linked to a higher risk factor burden and poorer outcomes from CVD (16). When considering the impact of the SDOH, it is important to note the increased proportions of Ethnic minority patients in the most deprived quintiles in the UK. Our study is the first major analysis investigating the intersection between deprivation and ethnicity in Europe.

Most studies focusing on the importance of race and ethnicity as a surrogate of the SDOH have been performed in the US, where studies have consistently shown poorer quality of care and outcomes from cardiovascular disease for Black Americans when compared with White Americans, even where adjusting for socioeconomic status (6, 17). This relationship is

not as clear in the UK, where in contrast to the US, in our study we demonstrate higher levels of ICA, PCI and CABG in Ethnic minority patients across the socioeconomic spectrum, which we suspect is in part due to the higher median age of White patients, making them more likely to be managed conservatively.

As the UK has a publicly funded universal healthcare system, we would expect less ethnicity-related discrepancies, but we have still found that deprivation exerts a more significant effect on in-hospital MACE and cardiac mortality in Ethnic minority patients than White patients, despite multivariate adjustment. Furthermore, amongst our most affluent quintile, Black patients had the highest adjusted in-hospital mortality, although we acknowledge the smaller population size. This observation is in keeping with results from the *Cooperative Cardiovascular Collective*, where shorter life expectancy for Black patients of all SES groups was shown in the US. The AHA has identified structural racism as a fundamental driver of health disparities in cardiovascular disease (18). Although most work on structural racism is from the US, it is also relevant to our study, given the significantly higher proportion of Ethnic minority patients in more deprived quintiles. Structural racism has been implicated in the development of cardiovascular risk factors, such as hypertension (19), by mechanisms, including residential segregation and stressor-exposure. Furthermore, structural racism influences processes of care, including rates of undergoing ICA when presenting with chest pain (20), and 'door-to-balloon-time' in STEMI (21), we suggest that this could be contributing to the more pronounced effect of deprivation on the outcomes of Ethnic minority patients.

However, we must consider the range of socioeconomic factors that are contributing. Educational status is an important contributor to the SDOH, and an analysis of the 'Million Women Study' found that lower educational attainment and increased neighbourhood deprivation strongly predicted coronary heart disease (defined as hospital admission or death

from CAD), but noted a significant proportion of this risk could be attributed to smoking, alcohol consumption, BMI, and physical inactivity (22). Previous studies using the MINAP registry have highlighted the increased risk of in-hospital mortality post-AMI in patients with 'multimorbidities' (23, 24). Therefore, it is easy to see how the impact of lower educational attainment on the levels of cardiovascular risk factors can contribute to poorer AMI outcomes in more deprived areas. Household income has been demonstrated to be predictor of higher mortality post-AMI, but that this is attenuated when adjusting for baseline clinical status (25). *Kilpi et al* suggested the most significant socioeconomic component to AMI incidence and outcome was income and highlighted an elevated incidence of AMI in manual occupations (26). Furthermore, there is an appreciation of the contribution of your surrounding environment where it has been demonstrated how living in a 'disadvantaged' neighbourhood is associated with an increased incidence of coronary artery disease, evening adjusting for income, education, and occupation (27).

Interestingly, in an analysis of UK STEMI patients, there was no significant relationship between SES and survival up to three-years, suggesting that where treatment is more standardised in STEMI, there is less disparity in outcomes (28). In contrast, NSTEMI treatment is more heterogenous, less guideline-based, and therefore more prone to inequality. We suggest that further work should focus on understanding the SDOH in the UK and how we can best mitigate their impact earlier in the CAD process to improve outcomes for AMI in the most deprived regions.

Limitations

The MINAP data registry shares weakness of other national registries, including self-reporting of adverse events without external validation. There are limitations to the data collected in MINAP; the database does not capture severity of CAD, rationale for specific

medications, or an exhaustive comorbidity list. Inherent to large registries such as MINAP is the issue of missing data.

The MINAP registry has the IMD 2010 score for each patient but does not record score components, therefore we are unable to analyse which components are having the most impact. MINAP only included comprehensive data from the IMD 2010 score, but note there is minimal temporal change in score (29). A further limitation is the small number of Black, Asian, Mixed and 'Other' ethnicity patients within our population, particularly in more affluent quintiles, which led us to create the 'Ethnic minority' group for subgroup analyses. We acknowledge that this Ethnic minority group is heterogeneous and contains a variation of risk factors and clinical outcomes (30). Furthermore, we acknowledge that given the nature of retrospective analyses, there is a risk of residual confounding from variables not included in the multivariate analysis. In attempts to mitigate against residual confounding, we included cardiometabolic risk factors such as diabetes, smoking and hypercholesterolaemia in our multivariate model, these were all more prevalent in the more socioeconomically deprived groups, and we acknowledge that this may could reduce our total effect-size.

Conclusion

Our study showed that patients from the most deprived quintile in the UK were more likely to be younger, from an Ethnic minority background, and have multiple co-morbid conditions. These patients were less likely to undergo ICA and revascularisation by PCI or CABG. In an adjusted multivariate model, patients from the most deprived quintile were more likely to suffer in-hospital mortality or MACE when compared with the most affluent quintile. The effect of deprivation on in-hospital mortality and MACE was greater in Ethnic minority patients.

Competing interests

None

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None

Data Availability

The National Institute for Cardiovascular Outcomes Research (NICOR) provided the data underlying this article. Data will be shared on request to the corresponding author with the permission of NICOR.

Ethics

Secondary use of anonymised MINAP dataset for research purposes is authorised under NHS research governance arrangements and further supported under section 251 of the NHS act 2006 (NIGB: ECC1-06(d)/ 2011), which allows researchers to use patient information collected within the dataset for medical research without patient consent. Therefore, formal ethical approval was not sought for this study.

Table 1: Demographic comparison between IMD 2010 score quintiles for patients suffering AMI

Variables	Quintile 1 (most affluent) (n=64,975)	Quintile 2 (n=72,178)	Quintile 3 (n=76,168)	Quintile 4 (n=76,512)	Quintile 5 (most deprived) (n=80,231)
Age, years, median (IQR)	72 (62-82)	71 (61-81)	71 (60-81)	68 (57-79)	66 (55-77)
Women (%)	20,973/64,975 (32%)	23,718/72,178 (33%)	25,594/76,168 (34%)	26,236/76,512 (34%)	27,663/80,231 (34%)
BMI, median [IQR]	26.6 (23.8-29.8)	26.8 (24.1-30.1)	27.0 (24.0-30.5)	27.2 (24.0-30.8)	27.4 (24.1-31.3)
Ethnicity- White	56,822/59,187 (96%)	63,242/66,088 (96%)	65,219/69,721 (94%)	62,757/69,850 (90%)	63,216/73,788 (86%)
Ethnicity- Ethnic Minority	2,365/59,187 (4%)	2,846/66,088 (4%)	4,502/69,721 (6%)	7,093/69,850 (10%)	10,572/73,788 (14%)
Basal crepitations (%)	4,731/36,550 (13%)	5,622/40,113 (14%)	6,166/42,594 (14%)	6,378/43,862 (15%)	6,260/45,064 (14%)
Pulmonary oedema (%)	1,871/36,550 (5%)	2,120/40,113 (5%)	2,280/42,594 (5%)	2,392/43,862 (5%)	2,807/45,064 (6%)
Cardiogenic shock (%)	649/36,550 (2%)	728/40,113 (2%)	829/42,594 (2%)	920/43,862 (2%)	928/45,064 (2%)
High risk GRACE score >140 (%)	27,176/34,922 (78%)	29,528/38,149 (77%)	30,908/40,573 (76%)	30,858/41,907 (74%)	29,974/3,314 (69%)
Intermediate risk GRACE score 109-140 (%)	6,126/34,922 (18%)	6,823/38,149 (18%)	7,555/40,573 (19%)	8,447/41,907 (20%)	9,798/43,314 (23%)
Low risk GRACE score <109 (%)	1,620/34,922 (5%)	1,798/38,149 (5%)	2,110/40,573 (5%)	2,602/41,907 (6%)	3,542/43,314 (8%)
ECG ST changes (%)	54,299/63,329 (86%)	60,413/70,449 (86%)	63,491/74,506 (85%)	64,213/75,027 (86%)	67,436/78,631 (86%)
Previous smoker (%)	21,528/60,491 (36%)	23,776/67,028 (36%)	24,419/70,757 (35%)	23,149/71,087 (33%)	21,807/74,997 (29%)
Current smoker (%)	10,865/60,491 (18%)	14,311/67,028 (21%)	17,770/70,757 (25%)	22,496/71,087 (32%)	30,049/74,997 (40%)
Chronic renal failure (%)	3,792/60,825 (6%)	4,303/66,453 (6%)	4,624/69,710 (7%)	4,709/69,543 (7%)	4,971/70,896 (7%)

Prior percutaneous coronary intervention (%)	5,887/61,025 (10%)	6,456/66,577 (10%)	6,992/69,846 (10%)	7,239/69,735 (10%)	7,706/71,055 (11%)
Diabetes (%)	11,099/63,184 (18%)	13,326/69,805 (19%)	15,542/73,644 (21%)	17,042/73,672 (23%)	19,999/76,757 (26%)
CCF (%)	3,081/60,918 (5%)	3,537/66,613 (5%)	3,936/69,883 (6%)	4,021/69,730 (6%)	4,270/71,003,443 (6%)
Hypercholesterolemia (%)	19,633/60,407 (33%)	20,757/66,005 (31%)	22,085/69,253 (32%)	23,345/69,233 (34%)	25,438/70,744 (36%)
Previous MI (%)	11,966/61,267 (20%)	13,609/67,134 (20%)	14,868/70,766 (21%)	15,584/79,370 (22%)	17,051/71,467 (24%)
History of angina (%)	13,170/60,911 (22%)	14,906/66,622 (22%)	16,150/69,868 (23%)	16,899/69,726 (24%)	18,286/71,006 (26%)
Cerebrovascular disease (%)	4,885/60,940 (8%)	5,531/66,625 (8%)	5,861/69,976 (8%)	6,016/69,821 (9%)	6,591/71,037 (9%)
Peripheral vascular disease (%)	2,340/60,735 (4%)	2,662/66,324 (4%)	2,982/69,677 (4%)	3,253/69,385 (5%)	3,650/79,623 (5%)
Hypertension (%)	30,981/61,446 (50%)	33,531/67,124 (50%)	35,464/70,582 (50%)	35,821/70,384 (51%)	36,475/71,878 (51%)
Asthma / COPD (%)	7,178/60,989 (12%)	8,813/66,687 (13%)	10,145/70,014 (14%)	11,743/69,865 (17%)	13,970/71,178 (20%)
Family history of CAD (%)	15,351/52,427 (29%)	16,565/56,612 (29%)	16,907/58,571 (29%)	17,796/59,414 (30%)	19,052/61,542 (32%)
Heart rate, bpm, median (IQR)	76 (65-90)	77 (65-90)	78 (66-91)	79 (67-92)	80 (68-94)
Systolic blood pressure, median (IQR)	137 (119-156)	136 (119-155)	136 (119-155)	135 (118-154)	135 (118-154)
Good LV function (%)	17,947/50,106 (36%)	19,671/54,348 (36%)	21,039/57,114 (37%)	21,004/57,418 (37%)	21,896/59,212 (37%)
Moderate LVSD (%)	10,463/50,106 (21%)	11,493/54,348 (21%)	12,371/57,114 (22%)	12,896/57,418 (22%)	13,632/59,212 (23%)
Severe LVSD (%)	3,507/50,106 (7%)	4,068/54,348 (7%)	4,415/57,114 (8%)	4,610/57,418 (8%)	5,213/59,212 (9%)
Cardiac arrest (%)	4,478/63,671 (7%)	4,706/70,500 (7%)	5,131/74,231 (7%)	5,193/74,181 (7%)	5,268/76,934 (7%)
Previous CABG surgery (%)	4,034/61,111 (7%)	4,404/66,675 (7%)	4,641/69,935 (7%)	4,553/69,798 (7%)	4,404/71,104 (6%)
Admission to cardiology ward (%)	42,024/48,739 (86%)	46,683/53,628 (87%)	48,443/55,644 (87%)	48,529/55,644 (87%)	52,653/59,864 (88%)
Admission under consultant cardiologist (%)	39,703/61,804 (64%)	43,050/68,839 (63%)	44,918/72,368 (62%)	45,857/72,570 (63%)	49,168/75,292 (65%)

CABG; coronary artery bypass graft, LVSD; left ventricular systolic dysfunction, CAD; coronary artery disease, COPD; chronic obstructive pulmonary disease, MI; myocardial infarction, CCF; congestive cardiac failure, BMI; body mass index, GRACE; global registry of acute coronary events, IQR; interquartile range.

Table 2: Management strategy and clinical outcome comparison between IMD 2010 quintiles for patients suffering AMI

Variables	Quintile 1 (most affluent) (n=64,975)	Quintile 2 (n=72,178)	Quintile 3 (n=76,168)	Quintile 4 (n=76,512)	Quintile 5 (most deprived) (n=80,231)
Low molecular weight heparin (%)	28,340/56,868 (50%)	29,878/61,461 (49%)	31,610/61,461 (50%)	30,191/64,397 (47%)	28,316/63,065 (45%)
Fondaparinux (%)	19,301/56,910 (34%)	21,724/61,247 (35%)	21,978/63,365 (35%)	22,727/64,258 (35%)	22,345/62,955 (35%)
Warfarin (%)	3,270/56,443 (6%)	3,541/60,793 (6%)	3,483/62,863 (5%)	3,424/63,714 (5%)	3,136/62,590 (5%)
Unfractionated heparin (%)	15,980/56,432 (28%)	15,507/60,641 (26%)	16,187/62,830 (26%)	16,387/63,659 (26%)	15,468/62,623 (25%)
Glycoprotein 2b/3a inhibitor (%)	5,882/57,135 (10%)	5,752/62,176 (9%)	6,082/64,542 (9%)	6,334/64,757 (10%)	6,143/63,365 (10%)
IV Nitrate (%)	9,912/56,464 (18%)	9,898/60,778 (16%)	10,477/62,910 (17%)	10,416/63,696 (16%)	9,182/62,543 (15%)
Furosemide (%)	13,637/56,531 (24%)	15,455/60,923 (25%)	16,260/63,026 (26%)	16,395/63,867 (26%)	16,281/62,694 (26%)
Calcium channel blockers (%)	8,648/56,487 (15%)	9,773/60,816 (16%)	10,199/62,918 (16%)	10,592/63,730 (17%)	10,434/62,599 (17%)
IV beta blockers (%)	998/56,833 (2%)	961/61,399 (2%)	1,003/63,586 (2%)	1,018/64,206 (2%)	903/62,888 (1%)
MRA (%)	4,056/56,064(7%)	4,438/60,357 (7%)	4,606/62,579 (7%)	4,660/63,188 (7%)	4,808/61,920 (8%)
Thiazide diuretics (%)	2,262/56,416 (4%)	2,510/60,698 (4%)	2,574/62,768 (4%)	2,561/63,653 (4%)	2,487/62,513 (4%)
Aspirin (%)	59,066/64,353 (92%)	65,459/71,531 (92%)	69,000/75,525 (91%)	69,336/75,931 (91%)	73,102/79,714 (92%)
P2Y12 inhibitor (%)	59,380/64,732 (92%)	65,666/71,924(91%)	69,386/75,954 (91%)	69,315/76,272 (91%)	72,103/80,007 (90%)
Statins (%)	53,292/64,648 (82%)	59,015/71,762 (82%)	62,308/75,735 (82%)	62,732/76,058 (82%)	66,430/79,761(83%)

ACE inhibitors/ARB (%)	49,204/64,587 (76%)	54,013/71,657 (75%)	56,744/75,621 (75%)	57,128/75,919 (75%)	60,038/79,618 (75%)
Beta-Blockers (%)	52,748/64,321 (82%)	58,125/71,456 (81%)	60,550/75,408 (80%)	60,925/75,716 (80%)	63,336/79,441 (80%)
Radionuclide Study (%)	1,197/56,342 (2%)	1,312/60,840 (2%)	1,426/63,095 (2%)	1,577/63,119 (3%)	1,909/64,324 (3%)
Exercise test (%)	3,030/58,468 (5%)	2,632/62,687 (4%)	2,561/64,940 (4%)	2,376/65,094 (4%)	1,972/64,612 (3%)
Coronary angiogram (%)	48,150/64,879 (74%)	52,932/72,015 (73%)	55,501/75,990 (73%)	55,628/76,240 (73%)	58,865/79,820 (74%)
Percutaneous coronary intervention (%)	33,435/64,753 (51%)	35,920/71,779 (50%)	37,753/75,753 (50%)	38,084/75,953 (50%)	40,441/79,734 (51%)
CABG surgery (%)	2,779/51,827 (5%)	3,040/55,915 (5%)	3,151/58,878 (5%)	3,144/58,779 (5%)	3,318/60,735 (5%)
Revascularization (CABG surgery/PCI) (%)	36,092/64,753 (56%)	38,834/71,779 (54%)	40,778/75,753 (54%)	41,065/75,953 (54%)	43,601/79,734 (55%)
Death (%)	4,228/64,975 (7%)	4,505/72,178 (6%)	4,876/76,168 (6%)	4,848/76,512 (6%)	4,667/80,231 (6%)
Cardiac mortality (%)	3,450/64,975 (5%)	3,833/72,178 (5%)	4,157/76,168 (5%)	4,123/76,512 (5%)	3,980/80,231 (5%)
Reinfarction (%)	675/62,126 (1%)	764/68,674 (1%)	814/72,225 (1%)	856/72,308 (1%)	796/74,223 (1%)
Major bleeding (%)	1,175/64,223 (2%)	1,183/71,167 (2%)	1,267/75,111 (2%)	1,331/75,211 (2%)	1,279/78,597 (2%)
MACE* (%)	4,721/64,975 (7%)	5,102/72,178 (7%)	5,466/76,168 (7%)	5,483/76,512 (7%)	5,259/80,231 (7%)

IV; intravenous, MRA; mineralocorticoid receptor antagonist, ACE: angiotensin-converting-enzyme, ARB; angiotensin receptor blockers, CABG; coronary artery bypass graft, PCI; percutaneous coronary intervention and MACE; major adverse cardiovascular events.

Table 3: Demographic comparison between IMD 2010 score quintiles (AMI ethnicity subgroup)

Variables	Quintile 1 (most affluent) (n=64,975)		Quintile 2 (n=72,178)		Quintile 3 (n=76,168)		Quintile 4 (n=76,512)		Quintile 5 (most deprived) (n=80,231)	
	White (n=56,822)	Ethnic minority (n=2,365)	White (n=63,242)	Ethnic minority (n=2,153)	White (n=65,219)	Ethnic minority (n=4,502)	White (n=62,757)	Ethnic minority (n=7,093)	White (n=63,216)	Ethnic minority (n=10,572)
Ethnicity	White (n=56,822)	Ethnic minority (n=2,365)	White (n=63,242)	Ethnic minority (n=2,153)	White (n=65,219)	Ethnic minority (n=4,502)	White (n=62,757)	Ethnic minority (n=7,093)	White (n=63,216)	Ethnic minority (n=10,572)
Age, years, median (IQR)	72 (61-82)	69 (58-79)	71 (61-81)	68 (57-78)	70 (59-80)	66 (55-77)	68 (57-79)	64 (53-75)	66 (55-77)	61 (52-74)
Women (%)	18,616/56,822 (33%)	599/2,365 (25%)	21,105/63,242 (33%)	693/2,846 (24%)	22,433/65,219 (34%)	1,171/4,502 (26%)	22,273/62,757 (35%)	1,844/7,093 (26%)	22,901/63,216 (36%)	2,851/10,572 (27%)
BMI, median [IQR]	26.6 (23.8-29.8)	26.2 (23.6-29.4)	26.8 (24.1-30.0)	26.4 (23.7-29.4)	27.0 (24.0-30.5)	26.5 (23.5-30.1)	27.2 (24.0-30.8)	27.1 (23.9-30.6)	27.4 (24.1-31.3)	27.3 (24.1-30.8)
Basal crepitations (%)	4,156/32,055 (13%)	126/1,396 (9%)	54,982/35,048 (14%)	209/1,717 (12%)	5,429/36,468 (15%)	306/2,713 (11%)	5,420/35,722 (15%)	555/4,503 (12%)	5,097/35,189 (14%)	739/6,367 (12%)
Pulmonary oedema (%)	1,655/32,055 (5%)	87/1,396 (6%)	1,862/35,048 (5%)	106/1,717 (6%)	1,960/36,468 (5%)	162/2,713 (6%)	1,926/35,722 (5%)	314/4,503 (7%)	2,204/35,189 (6%)	442/6,367 (7%)
Cardiogenic shock (%)	557/32,055 (2%)	32/1,396 (2%)	618/35,048 (2%)	47/1,717 (3%)	677/36,468 (2%)	80/757 (11%)	676/35,722 (2%)	158/4,503 (4%)	669/35,189 (2%)	144/6,367 (2%)
High risk GRACE score >140 (%)	23,988/30,638 (78%)	955/1,330 (72%)	23,888/30,638 (78%)	955/1,330 (72%)	26,671/34,751 (77%)	1,837/2,600 (71%)	25,352/34,190 (74%)	3,039/4,328 (70%)	23,905/33,923 (70%)	3,786/6,157 (61%)
Intermediate risk GRACE score 109-140 (%)	5,298/30,638 (17%)	282/1,330 (21%)	5,298/30,638 (17%)	282/1,330 (21%)	6,318/34,751 (18%)	582/2,600 (22%)	6,786/34,190 (20%)	939/4,328 (22%)	7,466/33,923 (22%)	1,617/6,157 (26%)
Low risk GRACE score <109 (%)	1,352/30,638 (4%)	93/1,330 (7%)	1,352/30,638 (4%)	93/1,330 (7%)	1,762/34,751 (5%)	181/2,600 (7%)	2,052/34,190 (6%)	350/4,328 (8%)	2,552/33,923 (8%)	786/6,157 (13%)
Previous smoker (%)	19,110/52,933 (36%)	485/2,240 (22%)	21,323/58,828 (36%)	541/2,704 (20%)	21,660/60,657 (36%)	794/4,254 (19%)	20,133/58,437 (34%)	1,274/6,654 (19%)	18,386/59,324 (31%)	1,868/9,932 (19%)
Current smoker (%)	9,389/52,933 (18%)	390/2,240 (17%)	12,412/58,828 (21%)	570/2,704 (21%)	15,233/60,657 (25%)	914/4,254 (21%)	18,651/58,437 (32%)	1,684/6,654 (25%)	24,522/59,324 (41%)	3,064/9,932 (31%)
Chronic renal failure (%)	3,330/53,210 (6%)	162/2,237 (7%)	3,806/58,327 (7%)	193/2,653 (7%)	3,991/59,722 (7%)	305/4,165 (7%)	3,841/57,096 (7%)	555/6,593 (8%)	3,750/55,942 (7%)	893/9,534 (9%)
Prior percutaneous coronary intervention (%)	5,077/53,331 (10%)	314/2,236 (14%)	5,519/58,347 (9%)	398/2,671 (15%)	5,737/59,729 (10%)	641/4,196 (15%)	5,512/57,114 (10%)	1,047/6,625 (16%)	5,567/55,962 (10%)	1,506/9,536 (16%)

Diabetes (%)	9,430/55,283 (17%)	773/2,311 (33%)	11,370/61,238 (19%)	928/2,766 (34%)	12,652/63,128 (20%)	1,662/4,364 (38%)	12,796/60,581 (21%)	2,842/6,848 (42%)	13,927/60,706 (23%)	4,610/10,123 (46%)
Hypercholesterolemia (%)	16,982/52,897 (32%)	929/2,235 (42%)	17,989/57,974 (31%)	1,081/2,658 (41%)	18,321/59,378 (31%)	1,847/4,178 (44%)	18,185/56,832 (32%)	3,028/6,587 (46%)	18,881/55,769 (34%)	4,354/9,509 (46%)
History of angina (%)	11,659/53,287 (22%)	434/2,235 (19%)	13,228/58,409 (23%)	573/2,684 (21%)	14,011/59,816 (23%)	950/4,202 (23%)	13,983/57,194 (24%)	1,608/6,637 (24%)	14,476/55,977 (26%)	2,650/9,543 (28%)
Previous MI (%)	10,649/53,551 (20%)	459/2,246 (20%)	12,067/58,884 (20%)	592/2,682 (22%)	12,893/60,625 (21%)	926/4,213 (22%)	12,847/57,703 (22%)	1,562/6,669 (23%)	13,405/56,299 (24%)	2,542/9,568 (27%)
Cerebrovascular disease (%)	4,373/53,309 (8%)	129/2,234 (6%)	4,983/58,458 (9%)	175/2,672 (7%)	5,199/59,917 (9%)	235/4,194 (6%)	5,143/57,264 (9%)	454/6,633 (7%)	5,427/56,013 (10%)	784/9,536 (8%)
Peripheral vascular disease (%)	2,094/53,160 (4%)	61/2,234 (3%)	2,375/58,203 (4%)	82/2,669 (3%)	2,633/59,683 (4%)	113/4,178 (3%)	2,795/56,954 (5%)	207/6,582 (3%)	3,105/55,714 (6%)	295/9,497 (3%)
Hypertension (%)	27,102/53,722 (50%)	1,224/2,254 (54%)	29,315/58,876 (50%)	1,521/2,685 (57%)	30,123/60,446 (50%)	2,442/4,220 (58%)	28,741/57,694 (50%)	4,047/6,684 (61%)	27,885/56,652 (49%)	5,782/9,610 (60%)
Asthma / COPD (%)	6,381/53,348 (12%)	212/2,233 (9%)	7,899/58,600 (13%)	269/2,677 (10%)	8,939/59,958 (15%)	414/4,185 (10%)	10,070/57,307 (18%)	794/6,627 (12%)	11,656/56,114 (21%)	1,427/9,551 (15%)
Family history of CAD (%)	13,194/45,812 (29%)	650/2,042 (32%)	14,367/49,528 (29%)	760/2,447 (31%)	14,326/49,877 (29%)	1,127/3,819 (30%)	14,470/48,399 (30%)	1,749/6,053 (29%)	15,580/48,153 (32%)	2,485/8,622 (29%)
Good LV function (%)	15,560/44,101 (35%)	726/1,808 (40%)	17,049/47,963 (36%)	912/2,212 (41%)	17,775/49,022 (36%)	1,451/3,569 (41%)	16,864/47,240 (36%)	2,184/5,506 (40%)	16,894/47,124 (36%)	1,718/7,569 (23%)
Moderate LVSD (%)	9,047/44,101 (21%)	378/1,808 (21%)	9,944/47,963 (21%)	484/2,212 (22%)	10,406/49,022 (21%)	764/3,569 (21%)	10,373/47,240 (22%)	1,234/5,506 (22%)	10,722/47,124 (23%)	1,718/7,569 (23%)
Severe LVSD (%)	3,121/44,101 (7%)	121/1,808 (7%)	3,584/47,963 (7%)	162/2,212 (7%)	3,729/49,022 (8%)	274/3,569 (8%)	3,747/47,240 (8%)	440/5,506 (8%)	4,152/47,124 (9%)	594/7,569 (8%)
Cardiac arrest (%)	3,863/55,812 (7%)	146/2,312 (6%)	4,050/61,925 (7%)	187/2,800 (7%)	4,369/63,744 (7%)	280/4,393 (6%)	4,238/61,093 (7%)	445/6,891 (6%)	4,215/60,871 (7%)	542/10,216 (5%)
Previous CABG surgery (%)	3,562/53,397 (7%)	190/2,246 (8%)	3,825/58,430 (7%)	243/2,678 (9%)	3,893/59,800 (7%)	387/4,201 (9%)	3,680/57,142 (6%)	558/6,651 (8%)	3,388/55,973 (6%)	725/9,558 (8%)
Admission to cardiology ward (%)	36,233/42,243 (86%)	1,687/1,875 (90%)	40,326/46,473 (87%)	2,056/2,258 (91%)	40,667/46,875 (87%)	3,122/3,453 (90%)	38,811/44,840 (87%)	4,865/5,400 (90%)	40,563/46,302 (88%)	7,187/8,127 (88%)
Admission under consultant cardiologist (%)	33,960/54,048 (63%)	1,669/2,272 (73%)	36,706/60,316 (61%)	2,060/2,753 (75%)	37,294/61,982 (60%)	3,056/4,292 (71%)	36,159/59,423 (61%)	4,781/6,829 (70%)	37,682/59,054 (64%)	6,551/10,160 (64%)

CABG; coronary artery bypass graft, LVSD; left ventricular systolic dysfunction, CAD; coronary artery disease, COPD; chronic obstructive pulmonary disease, MI; myocardial infarction, CCF; congestive cardiac failure, BMI; body mass index, GRACE; global registry of acute coronary events, IQR; interquartile range.

Table 4: Management strategy and clinical outcome comparison between IMD 2010 quintiles (AMI ethnicity subgroup)

Variables	Quintile 1 (most affluent) (n=64,975)		Quintile 2 (n=72,178)		Quintile 3 (n=76,168)		Quintile 4 (n=76,512)		Quintile 5 (most deprived) (n=80,231)	
	White (n=56,822)	Ethnic minority (n=2,365)	White (n=63,242)	Ethnic minority (n=2,153)	White (n=65,219)	Ethnic minority (n=4,502)	White (n=62,757)	Ethnic minority (n=7,093)	White (n=63,216)	Ethnic minority (n=10,572)
Low molecular weight heparin (%)	24,784/49,711 (50%)	1,005/2,014 (50%)	26,406/53,825 (49%)	1,135/2,470 (46%)	27,342/54,385 (50%)	1,805/3,841 (47%)	25,191/52,850 (48%)	2,682/6,031 (44%)	23,018/50,192 (46%)	3,633/7,877 (46%)
Fondaparinux (%)	16,574/49,724 (33%)	639/2,031 (31%)	18,909/53,584 (35%)	717/2,466 (29%)	18,748/54,096 (35%)	1,074/3,850 (28%)	18,877/52,659 (36%)	1,816/6,064 (30%)	17,952/50,073 (36%)	2,716/7,872 (35%)
Warfarin (%)	2,993/49,310 (6%)	70/2,008 (3%)	3,203/53,185 (6%)	84/2,450 (3%)	3,118/53,668 (6%)	116/3,804 (3%)	2,993/52,251 (6%)	207/5,990 (3%)	2,646/49,776 (5%)	307/7,834 (4%)
Unfractionated heparin (%)	13,052/49,294 (26%)	674/2,013 (33%)	12,762/53,059 (24%)	795/2,446 (33%)	13,071/53,614 (24%)	1,156/3,828 (30%)	12,712/52,175 (24%)	1,842/6,007 (31%)	12,099/49,782 (24%)	2,161/7,862 (27%)
Glycoprotein 2b/3a inhibitor (%)	5,069/49,957 (10%)	232/2,024 (11%)	4,991/54,489 (9%)	257/2,479 (10%)	5,177/55,248 (9%)	403/3,852 (10%)	5,116/53,163 (10%)	632/6,063 (10%)	4,733/50,327 (9%)	792/8,024 (10%)
IV Nitrate (%)	8,551/49,327 (17%)	513/2,010 (26%)	8,604/53,158 (16%)	565/2,455 (23%)	8,925/53,707 (17%)	738/3,806 (19%)	8,372/52,223 (16%)	1,296/5,989 (22%)	6,941/49,733 (14%)	1,574/7,825 (20%)
Furosemide (%)	12,129/49,386 (25%)	402/2,014 (20%)	13,749/53,303 (26%)	507/2,456 (21%)	14,241/53,815 (26%)	780/3,811 (20%)	13,883/52,382 (27%)	1,292/5,991 (22%)	13,210/49,850 (26%)	1,921/7,845 (24%)
Calcium channel blockers (%)	7,558/49,356 (15%)	312/2,012 (16%)	8,543/53,195 (16%)	407/2,460 (17%)	88,655/53,710 (16%)	682/3,813 (18%)	8,524/52,227 (16%)	1,172/6,016 (19%)	8,050/49,767 (16%)	1,684/7,854 (21%)
IV beta blockers (%)	838/49,688 (2%)	47/2,022 (2%)	825/53,767 (2%)	48/2,462 (2%)	835/54,368 (2%)	74/3,823 (2%)	820/52,707 (2%)	112/6,016 (2%)	706/50,049 (1%)	126/7,851 (2%)
MRA (%)	3,551/49,071 (7%)	166/2,002 (8%)	3,911/52,944 (7%)	193/2,436 (8%)	3,937/53,588 (7%)	266/3,774 (7%)	3,810/51,936 (7%)	480/5,943 (8%)	3,818/49,340 (8%)	629/7,749 (8%)
Thiazide diuretics (%)	2,000/49,286 (4%)	59/2,013 (3%)	2,264/53,096 (4%)	66/2,451 (3%)	2,237/53,579 (4%)	116/3,802 (3%)	2,143/52,192 (4%)	211/5,984 (4%)	1,944/49,719 (4%)	370/7,819 (5%)

Aspirin (%)	51,548/56,296 (92%)	2,216/2,349 (94%)	57,266/62,691 (91%)	2,618/2,823 (93%)	58,928/64,685 (91%)	4,132/4,474 (92%)	56,676/62,299 (91%)	6,496/7,050 (92%)	57,271/62,809 (91%)	9,815/10,512 (93%)
P2Y12 inhibitor (%)	51,889/56,598 (92%)	2,212/2,359 (94%)	57,580/63,015 (91%)	2,637/2,835 (93%)	59,431/65,036 (91%)	4,170/4,490 (93%)	56,912/62,563 (91%)	6,534/7,061 (93%)	56,764/63,048 (90%)	9,758/10,525 (93%)
Statins (%)	46,426/56,541 (82%)	2,056/2,356 (87%)	51,577/62,881 (82%)	2,466/2,832 (87%)	53,044/64,857 (82%)	3,928/4,487 (88%)	51,116/62,394 (82%)	6,082/7,069 (86%)	51,918/62,845 (83%)	9,165/10,541 (87%)
ACE inhibitors/ARB (%)	42,906/56,483 (76%)	1,877/2,357 (80%)	47,170/62,784 (75%)	2,272/2,830 (80%)	48,266/64,748 (75%)	3,554/4,480 (79%)	46,422/62,263 (75%)	5,626/7,066 (80%)	46,904/62,722 (75%)	8,232/10,531 (78%)
Beta-Blockers (%)	46,027/56,285 (82%)	1,995/2,342 (85%)	50,812/62,638 (81%)	2,391/2,822 (85%)	51,698/64,612 (80%)	3,733/4,475 (83%)	49,806/62,168 (80%)	5,785/7,026 (82%)	49,579/62,620 (80%)	8,638/10,498 (82%)
Radionuclide Study (%)	1,036/49,559 (2%)	52/1,840 (3%)	1,157/53,803 (2%)	64/2,178 (3%)	1,251/54,513 (2%)	96/3,484 (3%)	1,280/52,345 (2%)	200/5,576 (4%)	1,371/50,799 (3%)	448/8,572 (5%)
Exercise test (%)	1,907/51,134 (4%)	100/2,126 (5%)	1,746/55,012 (3%)	68/2,479 (3%)	1,675/55,713 (3%)	103/3,845 (3%)	1,578/53,627 (3%)	154/6,067 (3%)	1,518/51,266 (3%)	181/8,643 (2%)
Coronary angiogram (%)	41,876/56,748 (74%)	2,010/2,358 (85%)	46,010/63,124 (73%)	2,427/2,836 (86%)	46,825/65,097 (72%)	3,852/4,480 (86%)	44,519/62,591 (71%)	6,000/7,052 (85%)	45,197/62,973 (72%)	8,639/10,482 (82%)
Percutaneous coronary intervention (%)	28,572/56,673 (50%)	1,480/2,360 (63%)	30,669/62,946 (49%)	1,841/2,836 (65%)	31,343/64,913 (48%)	2,801/4,487 (62%)	29,858/62,365 (48%)	4,315/7,068 (61%)	30,364/62,834 (48%)	6,141/10,530 (58%)
CABG surgery (%)	2,434/45,017 (5%)	154/1,968 (8%)	2,690/48,557 (6%)	147/2,404 (6%)	2,699/49,872 (5%)	227/3,807 (6%)	2,540/47,459 (5%)	355/5,936 (6%)	2,431/47,012 (5%)	637/8,382 (8%)
Revascularization (CABG surgery/PCI) (%)	30,904/56,673 (55%)	1,625/2,360 (69%)	33,261/62,946 (53%)	1,973/2,836 (70%)	33,945/64,913 (52%)	3,015/4,487 (67%)	32,287/62,365 (52%)	4,646/7,068 (66%)	32,701/62,834 (52%)	6,743/10,530 (64%)
Death (%)	3,770/56,822 (7%)	91/2,365 (4%)	4,019/63,242 (6%)	145/2,846 (5%)	4,337/65,219 (7%)	180/4,502 (4%)	4,190/62,757 (7%)	318/7,093 (4%)	3,962/63,216 (6%)	386/10,572 (4%)
Cardiac mortality (%)	3,058/56,822 (5%)	75/2,365 (3%)	3,398/63,242 (5%)	132/2,846 (5%)	3,673/65,219 (6%)	160/4,502 (4%)	3,556/62,757 (6%)	277/7,093 (4%)	3,344/63,216 (5%)	347/10,572 (3%)
Reinfarction (%)	593/54,444 (1%)	21/2,234 (1%)	670/60,374 (1%)	28/2,670 (1%)	699/62,083 (1%)	44/4,199 (1%)	722/59,595 (1%)	74/6,646 (1%)	614/58,594 (1%)	120/9,867 (1%)
Major bleeding (%)	1,032/56,257 (2%)	40/2,335 (2%)	1,059/62,519 (2%)	29/2,819 (1%)	1,115/64,464 (2%)	68/4,444 (2%)	1,109/61,914 (2%)	112/6,992 (2%)	1,036/62,047 (2%)	131/10,464 (1%)

MACE* (%)	4,203/56,82 2 (7%)	107/2,365 (5%)	4,537/63,24 2 (7%)	170/2,846 (6%)	4,836/65,219 (7%)	212/4,502 (5%)	4,715/62,757 (8%)	378/7,093 (5%)	4,403/63,216 (7%)	482/10,572 (5%)
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IV; intravenous, MRA; mineralocorticoid receptor antagonist, ACE: angiotensin-converting-enzyme, ARB; angiotensin receptor blockers, CABG; coronary artery bypass graft, PCI; percutaneous coronary intervention and MACE; major adverse cardiovascular events.

Table 5: Adjusted Outcomes for each quintile of deprivation compared to the most affluent quintile (quintile 5)

Outcome variables	Quintile 2 (n=72,178)		Quintile 3 (n=76,168)		Quintile 4 (n=76,512)		Quintile 5 (most deprived) (n=80,231)	
	OR	P-value	OR	P-value	OR	P-value	OR	P-value
Primary Outcomes								
MACE (In-hospital)	1.04 (0.97-1.11)	0.246	1.00 (0.93-1.06)	0.915	1.05 (0.98-1.12)	0.171	1.07 (1.00-1.15)	0.048
Mortality (In-hospital)	1.03 (0.95-1.11)	0.481	0.99 (0.92-1.07)	0.809	1.05 (0.97-1.13)	0.245	1.10 (1.01-1.19)	0.025
Secondary Outcomes								
Cardiac mortality (In-hospital)	1.07 (0.99-1.16)	0.089	1.00 (0.93-1.09)	0.938	1.07 (0.99-1.16)	0.110	1.08 (0.99-1.18)	0.071
Major bleeding (In-hospital)	0.95 (0.85-1.05)	0.311	0.93 (0.84-1.03)	0.165	1.01 (0.91-1.12)	0.883	1.00 (0.90-1.12)	0.981

Each quintile of deprivation is compared against our reference group ‘Quintile 1’, which is our most affluent quintile according to IMD score. Adjusted for: age, sex, heart rate, history of acute myocardial infarction, co-morbid conditions (hypertension, hypercholesterolaemia, diabetes, smoking, history of asthma or COPD, history of CVA or PVD), pharmacotherapy (prescription of low molecular weight heparin (LMWH), unfractionated heparin (UFH), warfarin, GP 2b/3a inhibitor, IV nitrate, furosemide, aldosterone antagonist, fondaparinux, angiotensin converting enzyme inhibitor/angiotensin receptor blockers, aspirin and P2Y12 inhibitor), cardiac arrest and procedures including coronary angiography during admission and revascularisation (by PCI or CABG during admission). MACE is defined as composite endpoint of in-hospital death and reinfarction

Figures

Figure 1: STROBE diagram detailing exclusion criteria

Figure 2: Summary of key findings

Figure 3: Plotted adjusted in-hospital mortality according to ethnicity

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