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Clinical paper

The effect of ethnicity and socioeconomic status on outcomes after resuscitated out-of-hospital cardiac arrest – Findings from a tertiary centre in South London [☆]



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Abstract

Background: Out-of-hospital cardiac arrest is a common cause of morbidity and mortality, and ethnic variation in outcomes is recognised. We investigated ethnic and socioeconomic differences in arrest circumstances, rates of coronary artery disease, treatment, and outcomes in resuscitated OOHCA.

Methods: Patients with resuscitated OOHCA of suspected cardiac aetiology were included in the King's Out-of-Hospital Cardiac Arrest Registry between 1-May-2012 and 31-December-2020.

Results: Of 526 patients (median age 62.0 years, IQR 21.1, 74.1% male), 414 patients (78.7%) were White, 35 (6.7%) were Asian, and 77 (14.6%) were Black. Black patients had more co-existent hypertension ($p = 0.007$) and cardiomyopathy ($p = 0.003$), but less prior coronary revascularisation ($p = 0.026$) compared with White/Asian patients. There were no ethnic differences in location, witnesses, or bystander CPR, but Black patients had more non-shockable rhythms ($p < 0.001$). Black patients received less immediate coronary angiography ($p < 0.001$) and percutaneous coronary intervention ($p < 0.001$) but had lower rates of CAD ($p = 0.004$) than White/Asian patients. All-cause mortality at 12 months was highest amongst Black patients, followed by Asian and then White patients (57.1% vs 48.6% vs 41.3%, $p = 0.032$). In Black patients, excess mortality was driven by higher rates of multi-organ dysfunction but lower cardiac death than White/Asian patients, with cardiac death highest amongst Asian patients ($p = 0.009$). Socioeconomic status had no effect on mortality, and in a multivariable logistic regression, age, location, witnesses, and Black compared to White ethnicity were independent predictors of mortality, whilst social deprivation was not.

Conclusion: In this single-centre study, Black patients had higher mortality after resuscitated OOHCA than White/Asian patients. This may be in part due to differing underlying aetiology rather than differences in arrest circumstances or social deprivation.

Keywords: Out-of-hospital cardiac arrest, Ethnicity, Socioeconomic status

Abbreviations: CAC, Cardiac arrest centres, CAD, Coronary artery disease, CAG, Coronary angiography, CPC, Cerebral Performance Category, DM, Diabetes mellitus, ECG, Electrocardiogram, KCH, King's College Hospital, London, United Kingdom, KOCAR, King's Out of Hospital Cardiac Arrest Registry, LBBB, Left bundle branch block, MODS, Multiple organ dysfunction syndrome, OOHCA, Out-of-hospital cardiac arrest, PCI, Percutaneous coronary intervention, ROSC, Return of spontaneous circulation, SCAI, Society for Cardiovascular Angiography and Interventions, SES, Socioeconomic status, STEMI, ST-elevation myocardial infarction, SYNTAX, Synergy Between Percutaneous Coronary Intervention With Taxus And Cardiac Surgery

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Introduction

Out-of-Hospital Cardiac Arrest (OOHCA) affects nearly 500,000 people annually, and despite improvements in the last decades, outcomes and survival rates remain poor.^{1–3} The incidence and outcomes of OOHCA vary substantially globally² and evidence suggests potential ethnic variation in arrest circumstances and outcomes. There is evidence that Black patients suffer from worse outcomes than other ethnic groups,^{4–8} although this effect is inconsistently seen^{9–11} and the majority of studies have been undertaken in the USA. Furthermore, the underlying causes of these ethnic discrepancies remain unclear. Arrest circumstances (including presenting rhythm, presence of witnesses, and bystander cardiopulmonary resuscitation), socioeconomic status (SES) and systemic factors (including public access defibrillation and specialist care) have all been implicated.¹² However, it is unclear to what extent these factors modulate ethnic differences in OOHCA, and causality is disputed.^{7,11,13} To date, few studies report on the association of ethnicity, social deprivation and co-existent co-morbidities with rates of coronary artery disease (CAD), subsequent treatment and outcome of resuscitated OOHCA patients after conveyance to a cardiac arrest centre (CAC).

We therefore aimed to investigate ethnic and socioeconomic differences in arrest circumstances, management, and outcomes amongst resuscitated OOHCA patients with a primary cardiac aetiology.

Methods

Study population

We performed a retrospective analysis of patients in the King's Out-of-Hospital Cardiac Arrest Registry (KOCAR) which has been described previously.³ Briefly, this is a prospectively collected registry of patients with OOHCA of suspected cardiac aetiology admitted to King's College Hospital (KCH), London, UK. KCH serves over 1 million people and is the main CAC in Southeast London.

Inclusion criteria were patients aged 18 and above suffering from OOHCA with return of spontaneous circulation (ROSC) admitted to KCH between 1st May 2012 and 31st December 2020. Patients were recruited if they had ST-segment elevation on post-arrest 12-lead electrocardiogram (ECG), or absence of a clear non-cardiac aetiology (including patients without ST elevation on post-arrest ECG). We therefore excluded patients with a clear non-cardiac aetiology (e.g. suicide, trauma, drowning, substance overdose), as well as patients with death before or immediately on arrival at KCH, those with suspected or confirmed intra-cerebral bleeding, known neurological disability (Cerebral Performance Category 3 or 4), or any known survival limiting disease to six months preceding the arrest. We also excluded patients if ethnicity or address was not available or recorded in the medical records.

Data collection

Data were collected using a dedicated database, and patients were identified using the Utstein criteria.^{14–15} Patient co-morbidities, cardiac arrest circumstances and management, in-hospital manage-

ment and clinical course, as well as long-term outcomes were collected. Zero-flow time was defined as intra-arrest time with no cardiopulmonary resuscitation (CPR), and low-flow time as intra-arrest time with CPR prior to achieving ROSC. Clinical scores including Society for Cardiovascular Angiography and Interventions (SCAI) classification for cardiogenic shock,¹⁶ the Synergy Between Percutaneous Coronary Intervention With Taxus And Cardiac Surgery (SYNTAX) score for grading the complexity of coronary artery disease,¹⁷ and the BCIS Jeopardy score for estimating the amount of myocardium at risk,¹⁸ were calculated retrospectively by an interventional cardiologist. SCAI shock grades C-E were considered as cardiogenic shock. A description of these scoring systems is available in the [supplementary materials](#).

Patient ethnicity was collected retrospectively from the electronic medical records. At our centre, ethnicity is self-declared during a patients' admission. The ethnicity was then categorised into either White, Asian or Black ethnicity according to the Office for National Statistics list of ethnic groups.¹⁹ All ethnicities extracted from the medical records fit into these categories. Where no ethnicity was recorded, the patient was excluded from the final analysis.

Patients' postcodes were extracted from the electronic medical records and matched to the English Indices of Deprivation 2019.²⁰ This government resource describes relative deprivation of neighbourhoods in the UK, ranking postcodes from least to most deprived in various domains. We extracted the Index of Multiple Deprivation rank (a composite marker encompassing income, employment, education, health, crime, living environment and other measures of deprivation) and Health Deprivation and Disability rank. Patients were subsequently divided into tertiles of deprivation within the registry. Where no postcode was available, the patient was excluded from the final analysis.

Outcomes

The primary endpoint was all-cause mortality 12 months post-OOHCA, and two secondary endpoints were used. Firstly, poor neurological outcome at six month follow-up, defined by the Cerebral Performance Category (CPC), a five-point scale recommended by the Utstein guidelines for use in cardiac arrest patients. CPC 3–5 were considered poor neurological outcomes. Secondly, cause of death was used as a secondary endpoint. The cause was classified as neurological,²¹ cardiac (refractory arrhythmia or refractory cardiogenic shock), multiple organ dysfunction syndrome (MODS)²² or other unrelated cause. These end-points were collected by a cardiologist or research nurse blinded to ethnicity/social deprivation status.

Statistical analysis

Non-normally distributed data are shown as median \pm interquartile range. Normality was assessed using the Shapiro-Wilk test, with all continuous variables displaying non-normal distribution (age, pH, lactate, creatinine, troponin, SYNTAX score, Jeopardy score). Comparison of means was performed using a Kruskal-Wallis test for non-normally distributed variables. Comparison of proportions was performed using a Chi-Square test. Survival curves were constructed using a Kaplan-Meier analysis and compared using a log rank test. To adjust for potential confounding effects of arrest circumstances, SES and ethnicity on outcomes post-OOHCA, we performed a multivariable analysis using a binary logistic regression analysis. Covari-

ates included patient age, gender, location of arrest, presence of a witness, bystander CPR, ethnicity, and social deprivation (Index of Multiple Deprivation) based on current literature.²³ All patients with a complete dataset were included for this analysis ($n = 523/526$). Odds ratios (OR) and 95% confidence intervals (CI) are reported. All statistical tests were two-sided, and a p value < 0.05 was considered statistically significant. All statistical analysis was performed using IBM SPSS Statistics, version 28.

Ethical approval

The study was performed according to the principles of Declaration of Helsinki and received approval by the Research Ethics Committee, Confidential Advisory Group and Health Research Authority (REC number 18/SC/0008).

Results

Baseline characteristics and arrest circumstances

Between the 1st May 2012 and 31st December 2020, 1398 patients suffered from OOHCA in the South London area for which the ambulance services were called. Of these, 613 patients reached KCH with ROSC, and were assessed as having a primary cardiac aetiology of cardiac arrest according to the above-described methodology (Fig. 1). Of the remaining patients, 87 did not have a recorded ethnicity or postcode, and were therefore excluded. The full cohort therefore included 526 patients.

The median age was 62.0 ± 21.1 years, and 74.1% of patients were male (Table 1). The median zero-flow time was 0 ± 5 minutes, and low-flow time was 21 ± 19 minutes. Most cardiac arrests occurred at home ($n = 289/525$, 55.0%) and 411/526 patients (78.1%) had shockable rhythms. A total of 307/526 patients (58.4%) had ST elevation/left bundle branch block (STEMI/LBBB). Four hundred and fourteen patients (78.7%) were White, 35 (6.7%) were Asian, and 77 (14.6%) were Black. One hundred and seventy-five (33.3%) patients were in the low deprivation group, 174 (33.1%) in the intermediate deprivation group, and 177 (33.7%) in the high deprivation group.

Ethnicity

There was no difference in the age of patients, but there were more males amongst White and Asian patients than Black patients ($p = 0.008$; Table 1). The risk factor profile of patients differed according to ethnicity (Supplementary Fig. 1): more Asian and Black patients had diabetes mellitus (DM) than White patients, with higher rates of insulin-dependent DM amongst Black patients and non-insulin dependent DM amongst Asian patients ($p < 0.001$). Black patients were also more likely to have essential hypertension ($p = 0.007$) and previously diagnosed cardiomyopathy ($p = 0.003$), whilst Asian and White patients were more likely to have previously received coronary revascularization (either percutaneous coronary intervention or coronary artery bypass grafting; $p = 0.026$). Asian patients were less likely to be smokers ($p = 0.010$).

There were no ethnic differences in location of arrest, witnessed arrests or bystander CPR but Black patients were less likely to have an initial shockable rhythm than Asian and White patients ($p < 0.001$; Table 1). Black patients were also more likely to receive intra-arrest adrenaline than White and Asian patients ($p = 0.002$). There was a trend toward fewer Black patients having STEMI/LBBB on post-ROSC ECG ($p = 0.065$). Black patients had higher serum lactate ($p = 0.002$) and creatinine ($p = 0.007$), indicating worse metabolic disturbance than amongst White and Asian patients. Troponin I levels, however, were lowest amongst Black patients ($p = 0.009$).

Black patients were less likely to receive immediate coronary angiography (within 2 hours; $p < 0.001$) or percutaneous coronary intervention ($p < 0.001$) than White or Asian patients (Table 2). They were also less likely to receive mechanical circulatory support devices including intra-aortic balloon pump, Impella device or receive extracorporeal membrane oxygenation ($p = 0.041$), despite similar rates of cardiogenic shock ($p = 0.409$). Amongst patients undergoing coronary angiography, Black patients were found to have less CAD and trend toward less culprit CAD than White and Asian patients ($p = 0.004$; $p = 0.064$ respectively). Furthermore, median SYNTAX and Jeopardy scores were also considerably lower amongst Black than White and Asian patients, indicating lower complexity of disease ($p = 0.010$; $p = 0.001$ respectively).

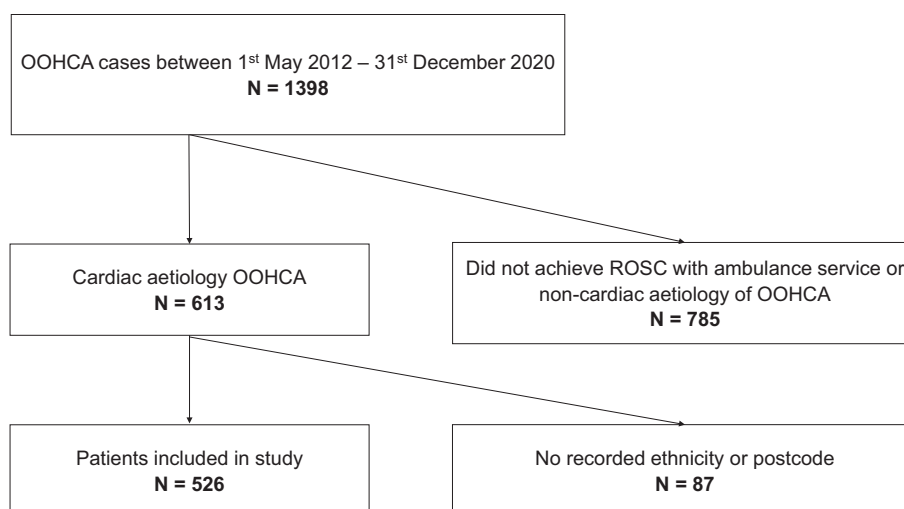


Fig. 1 – Study flow chart OOHCA: Out-of-hospital cardiac arrest; ROSC: Return of spontaneous circulation.

Table 1 – Baseline characteristics and arrest circumstances by ethnicity.

	All n = 526	White n = 414	Asian n = 35	Black n = 77	Significance	
Baseline characteristics & risk factors						
Age median ± IQR	62.0 ± 21.1	62.2 ± 20.7	61.5 ± 21.0	61.6 ± 24.2	<i>p</i> = 0.582 ^a	
Male gender (%; <i>n</i>)	74.1% 390/526	76.6% 317/414	77.1% 27/35	59.7% 46/77	<i>p</i> = 0.008 ^b	
Diabetes mellitus (%; <i>n</i>)	NIDDM				<i>p</i> < 0.001 ^b	
		12.7% 67/526	9.9% 41/414	37.1% 13/35		16.9% 13/77
	IDDM					
		4.0% 21/526	2.7% 11/414	5.7% 2/35	10.4% 8/77	
Hypertension (%; <i>n</i>)	48.1% 253/526	44.7% 185/414	54.3% 19/35	63.6% 49/77	<i>p</i> = 0.007 ^b	
Known cardiomyopathy (%; <i>n</i>)	9.1% 48/526	8.2% 34/414	0% 0/35	18.2% 14/77	<i>p</i> = 0.003 ^b	
Previous revascularization (%; <i>n</i>)	13.1% 69/526	13.8% 57/414	22.9% 8/35	5.2% 4/77	<i>p</i> = 0.026 ^b	
Previous cerebral infarction (%; <i>n</i>)	5.9% 31/526	5.6% 23/414	8.6% 3/35	6.5% 5/77	<i>p</i> = 0.745 ^b	
Smoker (%; <i>n</i>)	49.8% 262/526	52.2% 216/414	25.7% 9/35	48.1% 37/77	<i>p</i> = 0.010 ^b	
Arrest & hospital characteristics						
Arrest at residence (%; <i>n</i>)	55.0% 289/525	54.0% 223/413	54.3% 19/35	61.0% 47/77	<i>p</i> = 0.519 ^b	
Witnessed (%; <i>n</i>)	83.6% 439/525	84.5% 349/413	85.7% 30/35	77.9% 60/77	<i>p</i> = 0.337 ^b	
Bystander CPR (%; <i>n</i>)	77.6% 406/523	78.8% 324/411	74.3% 26/35	72.7% 56/77	<i>p</i> = 0.442 ^b	
Shockable initial rhythm (%; <i>n</i>)	78.1% 411/526	81.9% 339/414	77.1% 27/35	58.4% 45/77	<i>p</i> < 0.001 ^b	
Adrenaline (%; <i>n</i>)	59.6% 307/515	56.0% 227/405	62.9% 22/35	77.3% 58/75	<i>p</i> = 0.002 ^b	
Troponin I * median ± IQR	1733 ± 9302	2062 ± 11241	1519 ± 9136	692 ± 3245	<i>p</i> = 0.009 ^a	
Lactate median ± IQR	4.5 ± 5.8	4.0 ± 5.3	4.8 ± 6.9	5.9 ± 6.1	<i>p</i> = 0.002 ^a	
Venous pH median ± IQR	7.23 ± 0.21	7.23 ± 0.20	7.27 ± 0.38	7.20 ± 0.24	<i>p</i> = 0.450 ^a	
Serum creatinine median ± IQR	104 ± 43	103 ± 40	98 ± 49	115 ± 72	<i>p</i> = 0.007 ^a	
STEM / LBBB on ECG (%; <i>n</i>)	58.4% 307/526	59.9% 248/414	65.7% 23/35	46.8% 36/77	<i>p</i> = 0.065 ^b	
LVEF on arrival (%; <i>n</i>)	EF 41-49%				<i>p</i> = 0.171 ^b	
		12.3% 59/481	13.2% 50/379	11.8% 4/34		7.4% 5/68
	EF ≤40%					
		56.8% 273/481	57.3% 217/379	64.7% 22/34	50.0% 34/68	
Shock (SCAI grade C-E) (%; <i>n</i>)	43.5% 227/522	42.0% 173/412	48.6% 17/35	49.3% 37/75	<i>p</i> = 0.409 ^b	
Admission to critical care (%; <i>n</i>)	95.7% 448/468	95.1% 350/368	96.8% 30/31	98.6% 68/69	<i>p</i> = 0.412 ^b	
Targeted temperature management ** (%; <i>n</i>)					<i>p</i> = 0.857 ^b	
		72.0% 309/429	72.5% 242/334	73.3% 22/30		69.2% 20/45

EF: ejection fraction; IDDM: insulin-dependent diabetes mellitus; LVEF: left ventricular ejection fraction; NIDDM: non-insulin dependent diabetes mellitus; SCAI: Society for Cardiovascular Angiography and Interventions.

^a: Kruskal-Wallis test.

^b: Chi-Square test (2-sided).

*: *n* = 356. Our centre changed Troponin assay from Troponin I to Troponin T during the study period. We display Troponin I as more patients had a Troponin I than T level available for analysis.

** : Data displayed as patients undergoing targeted temperature management amongst patients admitted to critical care.

Social deprivation

Stratifying patients by social deprivation (Index of Multiple Deprivation) revealed an overall similar risk factor profile, although the most deprived tertile were more likely to be female (*p* = 0.028), non-insulin dependent diabetics (*p* = 0.043), and smokers (*p* = 0.019) than less deprived patients (Supplementary Table 1). The least deprived tertile was more likely to suffer from shockable rhythms (*p* = 0.011) and

have a witnessed arrest (*p* = 0.036), with other arrest circumstances including location, bystander CPR and adrenaline similar between tertiles. The most deprived patients had more severe metabolic derangement, with higher lactate (*p* = 0.002) and lower pH (*p* = 0.040) on arrival to the centre. Rates of immediate coronary angiography, percutaneous coronary intervention and CAD, however, were similar, although SYNTAX and Jeopardy scores were

Table 2 – Coronary angiography characteristics and outcomes by ethnicity.

	All n = 526	White n = 414	Asian n = 35	Black n = 77	Significance
Coronary angiography					
Early CAG (%; n)	77.9% 410/526	81.2% 336/414	85.7% 30/35	57.1% 44/77	p < 0.001 ^b
PCI (%; n)	52.9% 278/526	56.0% 232/414	68.6% 24/35	28.6% 22/77	p < 0.001 ^b
Mechanical circulatory support* (%; n)	23.6% 124/526	24.9% 103/414	31.4% 11/35	13.0% 10/77	p = 0.041 ^b
Coronary artery disease (%; n)	82.0% 379/462	84.0% 316/376	85.3% 29/34	65.4% 34/52	p = 0.004 ^b
Culprit coronary artery disease (%; n)	67.4% 293/435	68.9% 246/357	71.9% 23/32	52.2% 24/46	<i>p</i> = 0.064 ^b
SYNTAX score median ± IQR	14.0 ± 17.0	15.0 ± 16.0	16.8 ± 14.8	7.0 ± 21.3	p = 0.010 ^a
Jeopardy score median ± IQR	6.0 ± 6.0	6.0 ± 6.0	6.0 ± 4.5	3.0 ± 6.0	p = 0.001 ^a
Outcomes					
Mortality at 12 months (%; n)	44.1% 232/526	41.3% 171/414	48.6% 17/35	57.1% 44/77	p = 0.032 ^b
Cause of death at 12 months** (%; n)					
Neurological	56.5% 131/232	60.2% 103/171	35.3% 6/17	50.0% 22/44	
MODS	24.1% 56/232	20.5% 35/171	23.5% 4/17	38.6% 17/44	p = 0.009 ^b
Cardiac	15.5% 36/232	15.2% 26/171	41.2% 7/17	6.8% 3/44	
Other	3.9% 9/232	4.1% 7/171	0% 0/17	4.5% 2/44	
Poor neurological outcome at 6 months (CPC 3-5) (%; n)	49.4% 260/526	45.2% 187/414	57.1% 20/35	68.8% 53/77	p < 0.001 ^b

CAG: coronary angiography; CPC: cerebral performance category; MODS: Multiple organ dysfunction syndrome; PCI: percutaneous coronary intervention; SYNTAX: Synergy Between Percutaneous Coronary Intervention With Taxus And Cardiac Surgery.

^a: Kruskal-Wallis test.

^b: Chi-Square test (2-sided).

*: Intra-aortic balloon pump, Impella device or extracorporeal membrane oxygenation.

** : Data presented as percentage of All, White, Asian and Black patients dying respectively.

higher amongst the least deprived tertile ($p = 0.034$; $p = 0.007$ respectively), indicating a higher complexity of CAD (Table 3).

More White patients were within the least deprived tertile than amongst Black and Asian patients, who were more likely to be amongst the most deprived tertile ($p < 0.001$; Supplementary Fig. 2).

Outcomes

At 12 months, Black patients suffered from significantly higher all-cause mortality than Asian followed by White patients ($p = 0.032$; Table 2). More Black patients died of MODS, but they suffered from less cardiac aetiology death than White or Asian patients ($p = 0.009$; Fig. 2B). Asian patients were most likely to die of a cardiac aetiology which occurred in 41.2% of deaths. Black patients were also more likely to have poor neurological outcome at six months ($p < 0.001$). However, there was no association between Index of Multiple Deprivation and mortality at 12 months, poor neurological outcome at six months or cause of death, with similar findings for Health and Disability rank and mortality, neurological outcome and cause of death (Table 3). Kaplan-Meier analysis stratified by ethnicity confirmed lower probability of survival amongst Black patients compared to Asian and White patients ($p = 0.015$; Fig. 2A), whilst SES showed no significant effect on probability of survival over one year ($p = 0.260$; Supplementary Fig. 3).

Multivariable analyses

Multivariable analysis (Fig. 3A, 3B, Supplementary Table 2) showed that Black compared to White ethnicity (OR 1.75, 95% CI 1.00–3.04), age (OR 1.05 per year, 95% CI 1.04–1.07), location of OOHCA (arrest at residence OR 1.49, 95% CI 1.01–2.20), and witnessed arrest (OR 0.35, 95% CI 0.21–0.61) were independent predictors of all-cause mortality at 12 months. Similarly, Black compared to White ethnicity (OR 2.57, 95% CI 1.44–4.59), age (OR 1.05 per year, 95% CI 1.04–1.07) and witnessed arrest (OR 0.23, 95% CI 0.13–0.41) were independent predictors of poor neurological outcome at six months.

Discussion

In this registry from a large cardiac arrest centre in London, our primary finding is that poor neurological outcome and death after resuscitated OOHCA is more common amongst Black than Asian and White patients, despite no identified ethnic differences in situational arrest circumstances. This indicates that this disparity might be partly related to biological mechanisms which also led to differing modes of death. Socioeconomic status, however, had no significant effect on outcomes in our study.

Table 3 – Coronary angiography characteristics and outcomes after out-of-hospital cardiac arrest by socioeconomic status.

		Most deprived	Middle tertile	Least deprived	Significance
Index of Multiple Deprivation		n = 177	n = 174	n = 175	
Early CAG (%; n)		77.4% 137/177	75.9% 132/174	80.6% 141/175	<i>p</i> = 0.557 ^b
PCI (%; n)		52.5% 93/177	50.0% 87/174	56.0% 98/175	<i>p</i> = 0.530 ^b
Mechanical circulatory support* (%; n)		24.9% 44/177	24.7% 43/174	21.1% 37/175	<i>p</i> = 0.650 ^b
Coronary artery disease (%; n)		82.4% 126/153	78.9% 120/152	84.7% 133/157	<i>p</i> = 0.415 ^b
Culprit coronary artery disease (%; n)		70.0% 98/140	64.1% 93/145	68.0% 102/150	<i>p</i> = 0.561 ^b
SYNTAX score median ± IQR		12.0 ± 16.5	10.0 ± 17.5	16.5 ± 15.8	<i>p</i> = 0.034^a
Jeopardy score median ± IQR		6.0 ± 6.0	6.0 ± 4.0	6.0 ± 6.0	<i>p</i> = 0.007^a
Mortality at 12 months (%)		46.3% 82/177	46.0% 80/174	40.0% 70/175	<i>p</i> = 0.407 ^a
Cause of death at 12 months* (%)	Neurological	56.1% 46/82	55.0% 44/80	58.6% 41/70	
	MODS	22.0% 18/82	31.3% 25/80	18.6% 13/70	<i>p</i> = 0.315 ^a
	Cardiac	18.3% 15/82	12.5% 10/80	15.7% 11/70	
	Other	3.7% 3/82	1.3% 1/80	7.1% 5/70	
Poor neurological outcome at 6 months (CPC 3-5) (%)		53.7% 95/177	51.1% 89/174	43.4% 76/175	<i>p</i> = 0.135 ^a
Health and Disability Rank		n = 176	n = 175	n = 175	
Early CAG (%; n)		75.6% 133/176	78.3% 137/175	80.0% 140/175	<i>p</i> = 0.600 ^b
PCI (%; n)		52.8% 93/176	49.1% 86/175	56.6% 99/175	<i>p</i> = 0.380 ^b
Mechanical circulatory support** (%; n)		22.2% 39/176	27.4% 48/175	21.1% 37/175	<i>p</i> = 0.331 ^b
Coronary artery disease (%; n)		82.1% 124/151	78.4% 120/153	85.4% 135/158	<i>p</i> = 0.273 ^b
Culprit coronary artery disease (%; n)		71.0% 98/138	63.0% 92/146	68.2% 103/151	<i>p</i> = 0.343 ^b
SYNTAX score median ± IQR		11.0 ± 16.5	11.0 ± 19.3	16.5 ± 14.5	<i>p</i> = 0.026^a
Jeopardy score median ± IQR		6.0 ± 6.0	6.0 ± 6.0	6.0 ± 6.0	<i>p</i> = 0.003^a
Mortality at 12 months (%)		46.0% 81/176	45.7% 80/175	40.6% 71/175	<i>p</i> = 0.514 ^a
Cause of death at 12 months* (%)	Neurological	54.3% 44/81	56.3% 45/80	59.2% 42/71	
	MODS	25.9% 21/81	26.3% 21/80	19.7% 14/71	<i>p</i> = 0.691 ^a
	Cardiac	17.3% 14/81	15.0% 12/80	14.1% 10/71	
	Other	2.5% 2/81	2.5% 2/80	7.0% 5/71	
Poor neurological outcome at 6 months (CPC 3-5) (%)		55.1% 97/176	49.7% 87/175	43.4% 76/175	<i>p</i> = 0.091 ^a

CAG: coronary angiography; CPC: cerebral performance category; MODS: multi-organ dysfunction syndrome; PCI: percutaneous coronary intervention; SYNTAX: Synergy Between Percutaneous Coronary Intervention With Taxus And Cardiac Surgery.

^a: Chi-Square test (2-sided).

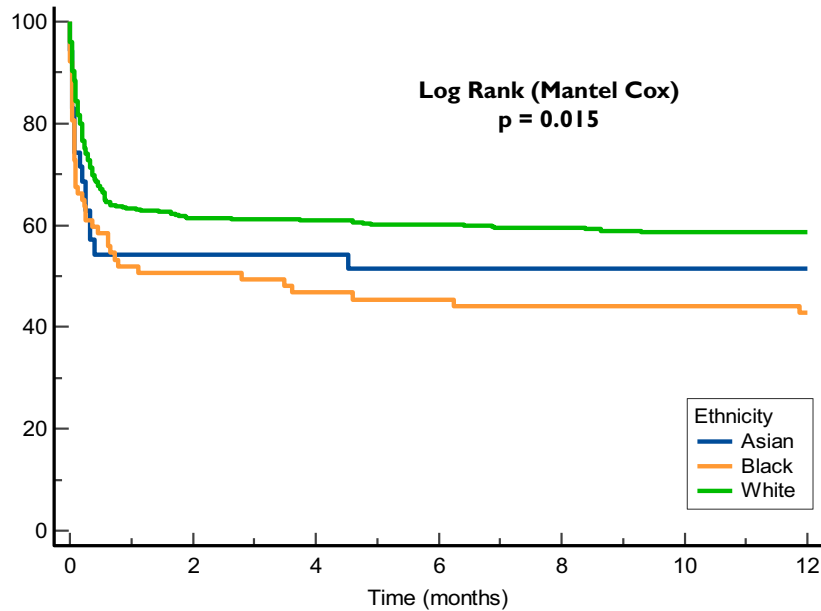
*: Data presented as percentage of tertile 1, 2 and 3 patients dying respectively.

** : Intra-aortic balloon pump, Impella device or extracorporeal membrane oxygenation.

Ethnic and SES discrepancies following OOHCA are well documented, with Black patients known to suffer from higher all-cause mortality than White patients.^{4–8} There are several potential causes for this apparent disparity, including pre-existing chronic health

inequalities,²⁴ OOHCA circumstances, access to public access defibrillation,²⁵ and also to specialist CAC care.²⁶ Limiting unwarranted variation in care based on ethnicity and SES is a priority of many healthcare systems across all conditions, including the

A. Kaplan Meier estimates of survival over 12-month follow-up by ethnicity



B. Cause of death by ethnicity

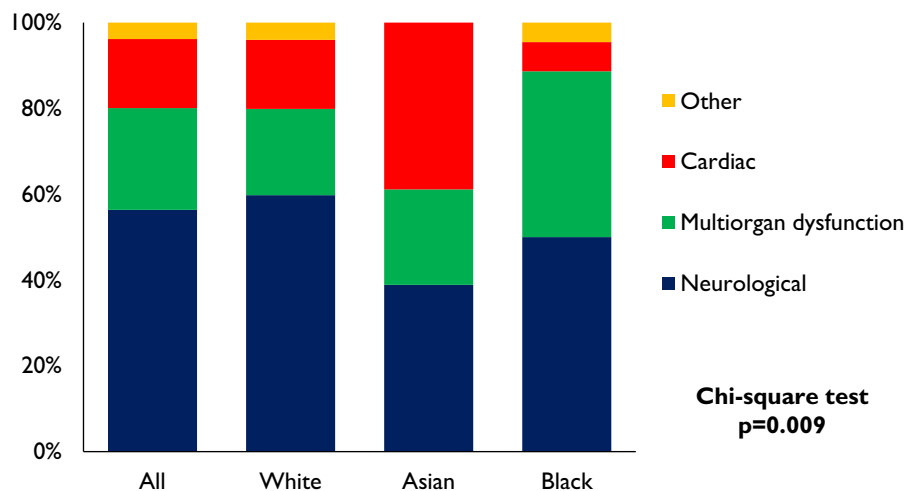


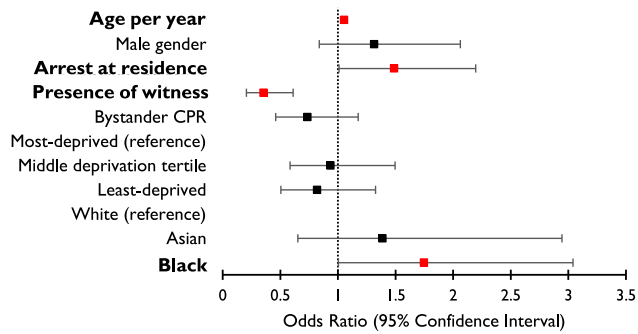
Fig. 2 – (A) Kaplan Meier estimates of survival over 12-month follow-up by ethnicity; (B) Cause of death by ethnicity.

NHS.²⁷ Indeed, in 2021, the NHS Race & Health Observatory advocated for people from minority ethnic backgrounds to be trained in CPR,²⁸ underlining the significant public health challenge and importance of investigating ethnic discrepancies in OOHCA care and outcomes.

The majority of previous studies have focused on the pre-hospital aspects of the association of ethnicity and SES with outcomes. In these large registry studies, it is known that those with Black ethnicity are less likely to have a witnessed arrest, bystander CPR or public access defibrillation.^{4–7,11–12,29–30} Furthermore, previous research has identified “high-risk neighbourhoods” in the UK with a higher incidence of OOHCA, greater levels of deprivation, and lower bystander CPR rates.³¹ These neighbourhoods have higher population density

and a greater degree of urbanisation, with many neighbourhoods in South London defined as “high-risk”.³¹ It is also well established that on arrival to a CAC, Black patients are less likely to undergo invasive coronary angiography.^{11–12,32} A recent study in Denmark showed an association between higher income and higher rates of CAG and 30-day survival in Denmark,³³ and higher income with more implantable cardioverter defibrillator implantation amongst patients with OOHCA not caused by acute myocardial infarction.³⁴ We recognise that at single-centre level, ethnic and socioeconomic differences in pre-hospital factors and in-hospital management are likely to be attenuated, as the ambulance services and hospital serve patients within the same catchment area, providing comparable clinical services and clinician expertise to the population. However, a more nuanced

A. Multivariable analysis of mortality at 12 months



B. Multivariable analysis of poor neurological outcome at 6 months

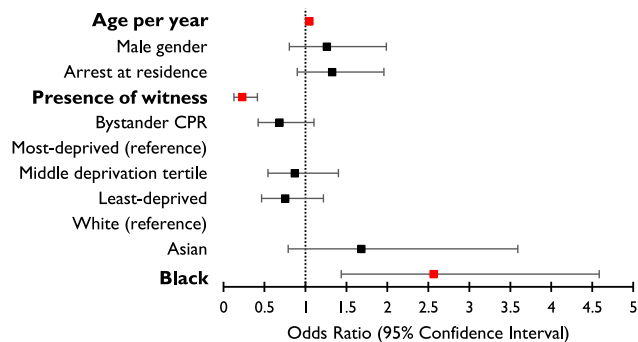


Fig. 3 – (A) Multivariable analysis of mortality at 12 months; (B) Multivariable analysis of poor neurological outcome at six months. *: $p < 0.05$. (A): $n = 523$; Chi Square = 94.5, $p < 0.001$. (B): $n = 523$; Chi Square = 106.9, $p < 0.001$. Red squares denote significance ($p < 0.05$).

understanding of underlying co-morbidities, rates of CAD, cardiovascular treatments, and effect on outcomes in resuscitated OOHCA survivors to a CAC based on ethnicity and SES currently remains unclear.

Our study suggests that in the cohort of patients with resuscitated OOHCA that survive to a CAC, ethnic variations in outcomes persist and are potentially related to biological mechanisms impacting on the aetiology of OOHCA rather than arrest circumstances or SES in themselves. We found that situational circumstances (such as witnessed arrests/bystander CPR) did not differ in this cohort of resuscitated OOHCA patients other than Black patients being more likely to present with non-shockable rhythms in accordance with previous literature.^{6–7,30} Importantly, we instead observed significant differences in pre-existing co-morbidities with Black patients more likely to have hypertension, insulin dependent DM and known cardiomyopathy, White patients more likely to have known CAD with prior revascularisation and Asian patients having non-insulin dependent DM and the highest rates of revascularisation. Our data largely accords with prior data where Black OOHCA patients are more likely than White patients to have hypertension,^{8,29–30,35–36} diabetes,^{8,30,35–36} kidney disease,^{8,30,35–36} and congestive heart failure.^{8,35–36}

We observed that Black patients had a trend towards lower STEMI/LBBB rates on arrival and had lower rates of provision of

immediate coronary angiography (within two hours) and percutaneous coronary intervention. Amongst those patients undergoing angiography, rates of CAD as well as SYNTAX and Jeopardy scores were significantly higher in White and Asian patients than Black patients. This is supported by previous literature where Black patients have lower rates of CAD in both post-mortem³⁶ and in vivo studies,³⁵ and an indication that revascularisation was more beneficial in White patients.³⁵ Despite this, rates of haemodynamic instability were similar between the ethnic groups, suggesting that this might be driven by more systemic ischaemic reperfusion in Black patients, which may reflect differing rates of provision of advanced mechanical circulatory support in this cohort.

Black patients had higher mortality, driven by a higher rate of MODS, whereas Asian patients suffered from the highest rates of cardiac-aetiology death, but were less likely to have neurological death. This indicates that the modes of death observed are likely to represent underlying co-morbidities and rates of CAD, which then have a critical influence on the course of the disease after admission to a CAC. Importantly, in our study, SES had no effect on mortality, neurological outcome, and cause of death, implying that ethnic differences in outcome cannot be accounted for entirely by SES as has previously been suggested.

At present, the American Heart Association and European Society of Cardiology recommend conveyance of all OOHCA patients to dedicated CACs for consideration of early coronary angiography.^{37–39} Our analysis suggests that the mechanisms of cardiac arrest may differ between ethnicities, with coronary causes being potentially less common amongst Black patients. However, these ethnic variations in OOHCA aetiology are not adequately reflected in our current pathways. Our findings suggest that patients being conveyed to hospital following resuscitation should have access to a complete array of complex interventions, including coronary angiography but also specialist 24/7 imaging, access to renal replacement therapy and other advanced critical care therapies. Future pathways of care which reflect these inherent ethnic variations in OOHCA with provision of appropriate therapies are likely to reduce unwarranted variations in outcome and reduce inequalities in the provision of services post-OOHCA.

Limitations

This was a single-centre observational study carried out in South London, UK, with high rates of ethnic variation which may limit generalisability of the findings to other healthcare settings. The observational study design means certain decisions, such as proceeding to angiography and/or percutaneous coronary intervention, were taken by the clinician responsible for the patient and are therefore the findings are at risk of selection bias. Furthermore, although statistical significance was achieved, the number of patients was small, in particular Asian and Black patients. Our findings should therefore be validated in larger cohorts to ensure that lack of observed effects (e.g. regarding SES) was not due to a type two error. Patients included in this study were resuscitated primary cardiac aetiology OOHCA patients surviving to a CAC, which is reflected in high rates of witnessed arrests, bystander CPR and initial shockable rhythm. The findings may not be representative of a more undifferentiated population.

Our analysis excluded patients with no available ethnicity or post-code on record. Analysis of this cohort with unknown ethnicity/post-

code (n = 87) showed that these patients had a worse 12-month survival than patients included in the analysis. There are several possible reasons for lack of ethnicity/postcode documentation, including immediate death on arrival to a centre, language barriers or lack of permanent residence, which all may have implications for the validity of our findings. In addition, concerns have been raised regarding the accuracy of ethnicity coding in the UK, with a 2021 report by the Nuffield Trust highlighting incomplete and inconsistent coding, as well as an overrepresentation of 'other' ethnic categories and underrepresentation of mixed ethnic groups.⁴⁰ This inherent limitation to our study design should be borne in mind when considering the conclusions of our data analysis.

Furthermore, the use of postcodes as a proxy of SES could be considered a limitation of our study. However, it is not feasible to gather individual markers of SES for a study of this size, and doing so may result in a simplified assessment of SES. The English Index of Multiple Deprivation combines information pertaining to income, employment, education, health, crime, living environment and more, and is therefore a holistic marker of socioeconomic deprivation.²⁰ Additionally, UK postcodes are shared by only 15–20 households, thereby increasing the spatial resolution of this marker.⁴¹

Conclusion

In this single-centre retrospective analysis, Black patients suffering from resuscitated suspected cardiac aetiology OOHCA had worse outcomes than Asian and White patients. This may be partly due to underlying co-morbidities and pathophysiological mechanisms rather than cardiac arrest circumstances or socioeconomic status.

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Conflicts of interest

MP reports grants from Sanofi; grants and personal fees from Gilead Sciences; and personal fees from QIAGEN, outside the submitted work.

CRedit authorship contribution statement

Roman Roy: Conceptualization, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Ritesh Kanyal:** Data curation, Investigation, Writing – review & editing. **Muhamad Abd Razak:** Investigation, Writing – review & editing. **Brian To-Dang:** Investigation, Writing – review & editing. **Shayna Chotai:** Investigation, Writing – review & editing. **Huda Abu-Own:** Investigation, Writing – review & editing. **Antonio Cannata:** Investi-

gation, Writing – review & editing. **Rafal Dworakowski:** Investigation, Writing – review & editing. **Ian Webb:** Investigation, Writing – review & editing. **Manish Pareek:** Supervision, Investigation, Writing – review & editing. **Ajay M Shah:** Supervision, Investigation, Writing – review & editing. **Philip MacCarthy:** Supervision, Investigation, Writing – review & editing. **Jonathon Byrne:** Supervision, Investigation, Writing – review & editing. **Narbeh Melikian:** Conceptualization, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Supervision. **Nilesh Pareek:** Conceptualization, Data curation, Formal analysis, Investigation, Writing – original draft, Writing – review & editing, Supervision.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.resplu.2023.100388>.

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