

Racial disparities in access to, and outcomes of, acute ischaemic stroke treatments in the USA

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ABSTRACT

Background Racism contributes to higher comorbid risk factors and barriers to preventive measures for black Americans. Advancements in systems of care, tissue plasminogen activator (tPA) availability and endovascular thrombectomy (ET) have impacted practice and outcomes while outpacing contemporary investigation into acute ischaemic stroke (AIS) care disparities. We examined whether recent data suggest ongoing disparity in AIS interventions and outcomes, and if hospital characteristics affect disparities.

Methods We examined 2016–2019 fee-for-service Medicare inpatient data. We ran unadjusted logistic regression models to calculate ORs and 95% CI for two interventions (tPA and ET) and four outcomes (inpatient mortality, 30-day mortality, discharge home and outpatient visit within 30 days), with the main predictor black versus white race, additionally adjusting for demographics, hospital characteristics, stroke severity and comorbidities. **Results** 805 181 AIS admissions were analysed (12.4% black, 87.6% white). Compared with white patients, black patients had reduced odds of receiving tPA (OR 0.71, 95% CI 0.69 to 0.74, $p<0.0001$) and ET (0.69, 95% CI 0.65 to 0.72, $p<0.0001$). After tPA, black patients had reduced odds of 30-day mortality (0.77, 95% CI 0.72 to 0.82, $p<0.0001$), discharge home (0.72, 95% CI 0.68 to 0.77, $p<0.0001$) and outpatient visit within 30 days (0.89, 95% CI 0.84 to 0.95, $p=0.0002$). After ET, black patients had reduced odds of 30-day mortality (0.71, 95% CI 0.63 to 0.79, $p<0.0001$) and discharge home (0.75, 95% CI 0.64 to 0.88, $p=0.0005$). Adjusted models showed little difference in the magnitude, direction or significance of the main effects.

Conclusions Black patients were less likely to receive AIS treatments, and if treated had lower likelihood of 30-day mortality, discharge home and outpatient visits. Despite advancements in practice and therapies, racial disparities remain in the modern era of AIS care and are consistent with inequalities previously identified over the last 20 years. The impact of hospital attributes on AIS care disparities warrants further investigation.

INTRODUCTION

In the USA, racism permeates living conditions and systems of healthcare, leading to adverse health effects among racial minorities. The effects of racism are complex and largely institutionalised, permeating all facets of life along its course. Due to the inequalities

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Racial disparities persist in acute stroke care.

WHAT THIS STUDY ADDS

⇒ Black patients were less likely to receive acute ischaemic stroke treatments, and if treated had lower likelihood of 30-day mortality, discharge home and outpatient visits.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study adds key data to inform the pattern of disparities for US stroke care in the USA.

resulting from racism, black Americans are subject to adverse health conditions, barriers to healthcare, unequal care, unequal representation in healthcare and overall lower life expectancy.¹

Stroke is a common neurological condition with high levels of morbidity and mortality. Acute ischaemic stroke (AIS) has well-studied risk factors, preventative strategies, acute interventions and secondary preventative interventions. Racism contributes to higher comorbid risk factors and barriers to preventive measures for black Americans.² Among black Americans who suffer from an AIS, the likelihood of treatment with tissue plasminogen activator (tPA) has historically been lower compared with white counterparts.³ However, the last decade of AIS management has benefitted from advancements in hospital systems of care,⁴ increased availability of thrombolytics⁵ and standardisation of endovascular thrombectomy (ET).⁶ Despite these changes, there is relatively little contemporary data focused on disparities in AIS care, including differences in tPA and ET rates and related outcomes of treatment. Furthermore, there are relatively little data examining the relationship between hospital attributes and current treatment rates and outcomes, related to disparities by race.

We sought to assess relationships between race and AIS care in the USA in the modern era. Using contemporary data, we aimed to



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compare utilisation of tPA and ET between black and white patients. Among those receiving interventions, we planned to examine disparities in outcomes and investigate how intervention rates and outcomes among black patients vary at hospitals with differing attributes, including rural–urban location, teaching status, stroke certification status, deprivation index and volume of AIS treated at the hospital.

METHODS

The datasets that we used are publicly available from Medicare. Medicare is federal health insurance that covers approximately 44 million people in the USA 65 years of age or older. We used deidentified, complete fee-for-service Medicare inpatient and outpatient datasets from 1 January 2016 to 31 December 2019 (parts A and B). These datasets included encounter date, demographics, death date and claims information for all Medicare beneficiaries.

The International Classification of Diseases, 10th Revision, Clinical Modification (ICD-10) code of I63.x in the primary diagnosis position was used to identify index AIS admissions were identified, which has been validated with a positive predictive value (PPV) of $\geq 82\%$.⁷ All AIS admissions were included, excluding those with an associated rehabilitation revenue code (xx3025–xx3099) and those with missing age or age < 65 years.

Age, sex and race ethnicity were abstracted from Medicare. Non-white and black categories (eg, Hispanic, Asian, North American Native, unknown and other) were excluded from this analysis as the validity and accuracy of race in Medicare data is best for white and black.⁸ We used Clinical Classification Software codes for ICD-10 to identify medical comorbidities, including diabetes, hypertension, coronary artery disease, congestive heart failure, chronic kidney disease, hyperlipidaemia, atrial fibrillation/flutter and smoking. We identified tPA administration using ICD-10 procedure codes 3E03317 and 3E04317, and endovascular intervention using codes 03CG3ZZ, 03CH3ZZ, 03CJ3ZZ, 03CK3ZZ, 03CL3ZZ, 03CP3ZZ and 03CQ3ZZ. The Charlson Comorbidity Index (CCI) summarises the degree of comorbidity in administrative data analysis and has been previously validated; the CCI was calculated for each admission. In a minority of AIS admissions, National Institutes of Health Stroke Scale (NIHSS) is recorded using ICD-10 codes (under R29.7) and was extracted when available.

We determined teaching hospital status by receipt of Medicare graduate medical education payment. We grouped urban versus non-urban hospital location using National Center for Health Statistics classification definitions: large metropolitan areas with at least 1 million residents, small metropolitan areas with less than 1 million residents, micropolitan areas and not metropolitan or micropolitan (non-urban residual). A 4-year total AIS volume was determined by the total number of AIS admissions at the treating hospital during the study period. We

defined intensive care unit (ICU)-level care as a hospital claim with a revenue centre code including 200, 201, 202, 203, 204, 207, 208, 209, 210, 211, 212, 213 and 219. ICU availability at the hospital was defined as at least one AIS admission with at least 1 ICU-level care code. The Multi-dimensional Deprivation Index (MDI) estimates poverty with data from the US Census American Community Survey, incorporating information about the standard of living, education, health, economic security, housing quality and neighbourhood quality (range 0–1, with higher values denoting higher deprivation). The MDI was used to determine socioeconomic status of the hospital zip code. Hospital stroke certification (certified vs not certified) was based on the Joint Commission and Det Norske Veritas certification programmes.^{9 10} Provider National Provider Identifier code was linked to the Healthcare Provider Taxonomy Code Set and the National Council for Prescription Drug Programme's Mapping of healthcare provider taxonomy codes¹¹ (with missing descriptions retrieved via Google search) to identify Neurology specialty of providers. Neurologist availability at the hospital was defined as at least one index AIS admission with a Neurology provider during the study period.

We examined the dichotomous outcomes of inpatient mortality, 30-day all-cause mortality, discharge home and outpatient visit within 30 days. Discharge disposition was available in Medicare, including inpatient mortality and discharge home. Death within 30 days of index AIS hospitalisation is defined as 30-day all-cause mortality. Outpatient visit within 30 days was defined as any outpatient visit within 30 days of discharge from the AIS hospitalisation.

Statistical analysis

In order to assess differences in characteristics of AIS patients by black/white race, we calculated baseline characteristics of the index AIS admissions stratified by race. Frequencies were calculated for categorical variables, and means and SDs for continuous variables. Standardised mean difference scores were calculated to compare the groups because of the large sample sizes, using a score of ≥ 0.1 (or ≤ -0.1) as signifying a meaningful difference.

We calculated the unadjusted risk and 95% CI for black and white patients separately for two interventions (tPA and ET) and four outcomes (inpatient mortality, 30-day mortality, discharge home and outpatient visit within 30 days). The four outcomes were calculated among those receiving tPA and those receiving ET separately. We also calculated the risk difference (as the difference of black–white) and risk ratio (black/white) using the FREQ procedure with options RISKDIFF and RELRISK, and OR (for black vs white patients) using unadjusted logistic regression. We calculated the risk and risk difference in order to estimate absolute risk, and the OR was considered the primary ratio measure of effect for this analysis.

We ran adjusted multilevel logistic regression models for the above outcomes, with the main predictor black versus white race, additionally adjusting for individual-level variables (age, sex, CCI) and hospital-level variables

(AIS case volume of the hospital, teaching hospital status, MDI, urban vs non-urban hospital location, stroke certification status of the hospital, ICU availability at the hospital and neurologist availability at the hospital). These models assessed the effect of race on the outcomes independent of the above confounders. To determine the amount of variance explained by hospital-level variables, we calculated the pseudo- R^2 comparing nested models with and without hospital-level variables.¹²

We calculated the frequency of available NIHSS scores per race, as well as the mean NIHSS for black and white patients separately, using the t-test to test for differences. We ran a second set of adjusted logistic regression models, this time adjusting for the above variables as well as NIHSS, limited to individuals with available NIHSS scores. These models assessed the effect of race on outcomes independent of recorded stroke severity.

We then turned our attention to the percentage of black AIS patients treated at the hospital level and summarised the distribution of this variable among all hospitals with at least one AIS admission during the study period, and secondarily among hospitals with at least the first quartile of AIS admissions. We summarised the distribution of several hospital-level variables, stratified by the quartile of percentage of black AIS patients treated at the hospital: urban versus rural location, MDI, teaching status, stroke certification status, AIS case volume during the study period, and neurologist and ICU-level care availability at the hospital. We calculated significance of differences using the χ^2 test. Finally, we ran separate outcome models, as above, limited to hospitals with the above characteristics with the highest percentage of black patients treated. The purpose of these analyses was to assess characteristics of hospitals that treat higher percentages of black AIS patients and related outcomes. We performed analyses by using SAS V.9.4. We acknowledge the relationship between the methodology and purpose of this paper and three of our prior papers.^{13–15}

Patient and public involvement

Patients were not involved in the planning or execution of this study.

RESULTS

Table 1 shows distributions of demographics and comorbidities of AIS patients, stratified by black versus white race. All variables were significantly different by race except for smoking history and hyperlipidaemia. Black patients were significantly younger (51.4% vs 67.5% with age >75 years) but had a higher prevalence of diabetes (50.5% vs 34.0%), congestive heart failure (22.4% vs 18.1%) and chronic kidney disease (39.1% vs 26.2%), as well as a higher CCI (mean of 4.92 (SD 2.19) vs 4.29 (2.12)), signifying greater comorbidity.

Online supplemental table 1 shows the unadjusted risks of outcomes by race, as well as the unadjusted risk difference, risk ratio and OR comparing black patients to white patients. Black patients were less likely to receive AIS treatments. The likelihood of receiving tPA was 0.096 (95% CI 0.094 to 0.098) among black patients compared with 0.122 (95% CI 0.122 to 0.123) among white patients, with a risk difference of -0.026 (95% CI -0.028 to -0.024), corresponding to almost 30% reduced odds of receiving tPA (OR 0.714, 95% CI 0.694 to 0.735, $p<0.0001$). The likelihood of receiving ET was 0.027 (95% CI 0.026 to 0.028) among black patients compared with 0.033 (95% CI 0.033 to 0.034) among white patients, with a risk difference of -0.006 (95% CI -0.007 to -0.005), corresponding to a greater than 30% reduced odds of receiving ET (OR 0.685, 95% CI 0.651 to 0.721, $p<0.0001$). Among those receiving tPA, black patients had reduced risk and odds of 30-day mortality compared with White patients (OR 0.768, 95% CI 0.717 to 0.824, $p<0.0001$) but also reduced risk and odds of discharge home (OR 0.724, 95% CI 0.681 to 0.771, $p<0.0001$) and outpatient visit within 30 days (0.891, 95% CI 0.839 to 0.946, $p=0.0002$). Among

Table 1 Distribution of variables at time of index stroke admission, stratified by race

	Black patients	White patients	Standardised mean difference score
Total	99 728	705 453	–
Age >75 years	51 226 (51.4)	476 158 (67.5)	–0.333
Male	42 864 (43.0)	320 471 (45.4)	0.2984
Smoking history	33 382 (33.5)	244 456 (34.7)	–0.0249
Diabetes	50 331 (50.5)	239 688 (34.0)	0.3387
Hypertension	54 273 (54.4)	419 691 (59.5)	–0.1026
Hyperlipidaemia	56 923 (57.1)	416 702 (59.1)	–0.0403
Atrial fibrillation/flutter	22 785 (22.9)	251 014 (35.6)	–0.2828
Congestive heart failure	22 289 (22.4)	127 343 (18.1)	0.1072
Chronic kidney disease	39 023 (39.1)	184 867 (26.2)	0.2782
Charlson Comorbidity Index score, mean (SD)	4.92 (2.19)	4.29 (2.12)	0.2885
National Institutes of Health Stroke Scale, mean (SD)	7.9 (7.8) N=30 928	7.1 (7.6) N=231 151	$P<0.0001$

those receiving ET, black patients had reduced risk and odds of 30-day mortality (OR 0.706, 95% CI 0.633 to 0.788, $p < 0.0001$) and discharge home (OR 0.753, 95% CI 0.642 to 0.883, $p = 0.0005$).

Online supplemental file 1 shows unadjusted logistic regression models of outcomes testing for differences between black and white patients, as well as models adjusted for demographics, comorbidities and hospital characteristics. The amount of variance explained by hospital-level variables was 53.2% for the outcome of TPA and 33.2% for ET; among those receiving TPA, the amount of variance explained was: inpatient mortality (10.7%), 30-day mortality (9.0%), discharge home (5.4%), 30-day outpatient visit (17.6%); among those receiving ET, the amount of variance was: inpatient mortality (8.8%), 30-day mortality (23.6%), discharge home (3.3%) and 30-day outpatient visit (13.5%). The adjusted models show little difference from the respective unadjusted models in the magnitude, direction and significance of the main effects. NIHSS was recorded for a minority of AIS admissions, in only 31.0% of black patients and 32.8% of white patients. The mean NIHSS was 7.9 among black patients and 7.1 among white patients ($p < 0.0001$). Online supplemental table 2 also shows models adjusting for NIHSS when available. The NIHSS-adjusted models show only two substantial differences when compared with the unadjusted models: a further ~25% decreased odds of 30-day mortality for black patients compared with white patients after receiving tPA, and a loss of statistical difference in discharge home after ET.

Among 4337 hospitals with at least 1 AIS admission during the study period, the mean percentage of black patients treated was 9.5% (SD 17.1, median 1.8%, IQR

0%–11.1%). The lowest quartile of hospitals (1–10 AIS admissions) was removed from the analysis. Among the remaining 3318 hospitals with at least 11 AIS admissions during the study period, the mean percentage of black patients treated was 10.2% (SD 15.9, median 3.6%, IQR 0%–13.0%) (online supplemental figure 1). Table 2 shows the distribution of variables at the hospital level, stratified by the quartile of percentage of black AIS patients treated at the hospital. Hospitals with higher percentages of black AIS patients were more frequently urban, in lower socioeconomic status areas, teaching and high AIS volume. Hospitals within the lowest quartile were less frequent with stroke certification, a neurologist or ICU-level care. When outcome models were run among hospitals with these characteristics, overall measures of effect were similar to the main models (online supplemental table 3).

DISCUSSION

Racism affects basic living conditions and subjects black Americans to barriers to healthcare, unequal care and unequal representation in healthcare.¹ Black Americans begin to experience these inequalities at birth, before the onset of stroke risk factors. Although race is a social construct that does not have a defined biological structure, the inequalities of racism yield biological consequences. Previous studies have demonstrated higher rates of stroke risk factors among black patients, including hypertension, hyperlipidaemia and diabetes.¹⁶

To measure these inequalities, we used race as a surrogate for racism and recognise that comorbidities such as hypertension function in part as mediators of the

Table 2 Distributions of variables at the hospital level, stratified by quartile of percentage of black acute ischaemic stroke patients treated at the hospital

Hospital characteristic	Quartile of percentage of black acute ischaemic stroke patients treated at hospital (range)				P value
	1st quartile (0)	2nd quartile (0.01%–3.6%)	3rd quartile (3.61%–13.0%)	4th quartile (13.01%+)	
Number of hospitals	964	694	830	830	–
Urban, frequency (%)	96 (10.0)	259 (37.3)	419 (50.5)	474 (57.1)	<0.0001
Teaching, frequency (%)	64 (6.6)	280 (40.4)	368 (44.3)	415 (50.0)	<0.0001
Stroke certified, frequency (%)	115 (11.9)	298 (42.9)	337 (40.6)	297 (35.8)	<0.0001
Neurologist at the hospital, frequency (%)	122 (12.7)	482 (69.5)	557 (67.1)	509 (61.4)	<0.0001
ICU at the hospital, frequency (%)	637 (66.1)	687 (99.0)	791 (95.3)	782 (94.2)	<0.0001
Multidimensional Deprivation Index, mean (SD)	0.123 (0.062)	0.126 (0.058)	0.145 (0.068)	0.189 (0.090)	<0.0001
Acute ischaemic stroke cases during study period, mean (SD)	51 (70)	295 (244)	295 (310)	253 (281)	<0.0001

Stroke certification of the hospital was assigned as certified versus not certified based on the Joint Commission and Det Norske Veritas certification programmes. Multidimensional Deprivation Index estimates poverty with data from the United States Census American Community Survey and accounts for standard of living, education, health, economic security, housing quality and neighbourhood quality (range 0–1, with higher values denoting higher deprivation). In this table, only hospitals with at least 11 acute ischaemic stroke admissions during the study period were included. ICU, intensive care unit.

relationship between race and stroke outcome. As such, unadjusted analysis preserves the effect of these mediators. Hence, while we present both unadjusted and adjusted models, we will prioritise the unadjusted results in the following discussion.

Historically, black patients have suffered from disparities in AIS treatment. Large national studies from 1998 to 2012 found significantly lower rates of tPA use within two¹⁷ and four-and-a-half hours¹⁸ of last known well, and lower odds of receiving tPA¹⁹ among black patients compared with white patients. In the years following these studies, AIS care has benefitted from improvements in systems of care and an increased access to thrombolytics.⁵ Smaller studies from 2006 to 2018, often geographically limited or restricted to a particular subgroup of AIS patients, found inconsistent differences in tPA utilisation by race.^{20–24} However, in large national studies from 2016 to 2020, four studies found lower tPA receipt among black patients, similar to our results.^{3 25–27}

Multiple prior studies found lower utilisation of ET among black patients compared with white patients.^{26 28–35} In terms of outcomes after AIS interventions, prior studies demonstrated lower^{18 36} or equal rates¹⁷ of inpatient mortality after receiving tPA in black patients, but 30-day mortality has not been frequently examined. Contemporary studies of post-ET mortality are mostly of small sample and restricted populations and often found no significant differences in either inpatient or follow-up mortality.^{21 37 38}

In terms of hospital characteristics related to race disparities in AIS treatment, prior studies showed that higher percentages of black patients are treated at large, urban, teaching and high-volume centres, similar to our findings.^{17 18 37} In three studies, black patients were more often seen at stroke-certified or ET-capable centres, which is compatible with our findings of higher stroke certification in the top three quartiles as compared with the bottom quartile.^{17 32 39}

Black patients had lower tPA utilisation rates¹⁸ and an increased risk of discharge home (in mild stroke presentations)³ at urban hospitals. Patients at teaching hospitals with internal carotid artery (ICA) or MCA strokes had a higher risk of post-ET mortality.²⁹ Finally by hospital size, one study found lower tPA utilisation rates among black patients at small, medium and large hospitals.¹⁸ At large hospitals, ICA and MCA stroke patients had a higher risk of post-ET mortality,²⁹ and patients with mild stroke presentations had increased odds of discharge home.³

Much of the prior research reviewed above has been limited by time periods prior to the contemporary endovascular era, limited geographical scope and a focus on either the individual or hospital level but not both. In our contemporary, national study of AIS treatments and outcomes by race, we found that black AIS patients had a higher burden of comorbidities than white patients but were younger on average. Black patients were ~30%–40% less likely to receive AIS treatments (tPA and ET) compared with white patients. Among those receiving

AIS treatments, despite lower 30-day mortality compared with white patients, black patients had lower likelihood of being discharged home (suggesting a higher likelihood of poorer outcomes such as discharge to rehabilitation or subacute nursing care) and of outpatient follow-up within 30 days. The lower mortality among black patients receiving tPA and ET may be partially accounted for by the lower age distribution of black AIS patients, but other contributing factors could include differences in stroke severity and poststroke complications, although further study is needed. Furthermore, hospitals that care for higher proportions of Black patients are more often high-volume, urban, teaching hospitals with stroke certification and both neurologist and ICU availability. However, even among such hospitals, black patients still had lower odds of tPA and ET utilisation compared with white patients. Black patients also had similar odds of in-hospital mortality, but lower odds of 30-day mortality and discharge home compared with white patients at these particular hospitals after receiving either tPA or ET. The reasons for these stark disparities are not entirely known and require further research, but there is evidence that they are downstream manifestations of structural racism that disproportionately influences access to healthcare, behavioural risk factors, risk factor development and control, access to stroke treatments, and quality of care.¹

Our study was subject to several limitations. As a retrospective study, we were able to demonstrate correlations between race (as a proxy of racism) and outcomes including utilisation of AIS interventions but cannot definitely suggest causality. We used ICD billing codes to capture primary diagnosis, comorbidities, procedural interventions and NIHSS scores, which are subject to inaccuracy, although the primary codes to identify AIS and related procedures have been previously validated. Since we used the Medicare database, results are most applicable to those aged ≥65 years. With administrative data, we did not have access to granular data such as why an intervention was withheld, including contraindications to therapy, radiographic findings precluding ET or prehospitalisation disability status. Since the validity and accuracy of race in Medicare data is best for white and black race,⁸ we restricted our analysis to these two recorded race categories. We did not include stroke certification programmes besides the ones listed above, which could have resulted in an underestimation of certified hospitals.

Using contemporary, national data with extensive geographical coverage and inclusion of multiple hospital types, we demonstrated persistent race disparities in receipt of AIS interventions and outcomes. More research is needed to investigate the causes of these disparities, as well as to develop interventions and protocols in order to achieve more equitable care for AIS in the USA.

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