ORIGINAL RESEARCH

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Race-Specific Predictors of Mortality in Intracerebral Hemorrhage: Differential Impacts of Intraventricular Hemorrhage and Age Among Blacks and Whites

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***Background*—**Intracerebral hemorrhage (ICH) carries high risk for short-term mortality. We sought to identify race-specific predictors of mortality in ICH patients.

***Methods and Results*—**We used 2 databases, the Johns Hopkins clinical stroke database and the Nationwide Inpatient Sample (NIS). We included 226 patients with the primary diagnosis of spontaneous ICH from our stroke database between 2010 and 2013; in the NIS, 42 077 patients met inclusion criteria. Logistic regression was used to assess differences in predictors of mortality in blacks compared to whites. In our clinical stroke database, Glasgow Coma Scale (GCS; P=0.016), ICH volume (P=0.013), intraventricular haemorrhage (IVH; P=0.023), and diabetes mellitus (P=0.037) were predictors of mortality in blacks, whereas GCS (P=0.007), ICH volume (P=0.005), age (P=0.002), chronic kidney disease (P=0.003), and smoking (P=0.010) predicted mortality in whites. Among patients with IVH, blacks had over 7 times higher odds of mortality compared to whites (odds ratio [OR], 7.27; P value for interaction, 0.017) and were more likely to present with hydrocephalus (OR, 2.76; P=0.026). In the NIS, black ICH patients had higher rates of external ventricular drain (EVD) placement compared to whites (9.7% vs 5.0%; P<0.001) and were more likely to develop hydrocephalus (OR, 1.32; 95% CI, 1.20–1.46). Comparison of a race-specific ICH score to the original ICH score showed that the various ICH score components have differential relevance for ICH score performance by race.

***Conclusions*—**IVH and age differentially predict mortality among blacks and whites. Blacks have higher rates of obstructive hydrocephalus and more frequently require EVD placement compared to their white counterparts. **(*J Am Heart Assoc*. 2016;5: e003540 doi: 10.1161/JAHA.116.003540)**

Key Words: disparities • hydrocephalus • intracerebral hemorrhage • intraventricular hemorrhage • mortality • prediction • race • race and ethnicity • stroke

Spontaneous intracerebral hemorrhage (ICH) accounts for approximately 15% of all strokes annually in the United States and is associated with higher morbidity and mortality than any other stroke subtype.[1,2] ICH incidence rates are especially high among black patients compared to whites.[3–5] In addition, blacks with ICH present at an earlier age than their white counterparts.[6,7]

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Despite advances in general medical and critical care, case fatality and in-hospital mortality rates for ICH remain as high as 30% to 40%.[5,8] Previous prognostic models have established a diversity of clinical and radiographical factors associated with mortality after ICH, including age, ICH volume, level of consciousness, pulse pressure, presence of intraventricular blood, and infratentorial origin.[9–13] The "ICH score," comprising information on age, Glasgow Coma Scale (GCS), ICH volume, intraventricular involvement, and supratentorial versus infratentorial origin, is a commonly used score for predicting short-term mortality among patients with ICH and has subsequently been externally validated.[13–16] However, blacks were under-represented in most of these studies, and comparative prediction models of mortality after ICH between races are lacking; thus, it is unclear whether currently established predictors of mortality in ICH are equally applicable to all races.

The present study sought to identify race-specific predictors of mortality in ICH in a biracial population (blacks and whites) and determine risk factors that are unique to blacks

and whites, respectively. In addition, we sought to evaluate the existing ICH score in blacks versus whites in order to compare its utility in these distinct racial groups and identify ICH score components uniquely relevant to different racial groups.

Materials and Methods

Data Source

We used 2 different databases: the Johns Hopkins clinical stroke database, including information on all consecutive spontaneous ICH admissions to our 2 academic centers, Johns Hopkins Hospital and Johns Hopkins Bayview Medical Center, between January 2010 and December 2013, and the Nationwide Inpatient Sample (NIS), the largest all-payer inpatient database in the United States, representing a 20% stratified sample of all admissions to nonfederal US hospitals. In NIS, we identified adult cases with primary diagnosis of ICH by using International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM) code 431 between 2007 and 2011.[17,18] Patients with trauma, vascular malformations, intracerebral metastatic disease, and aneurysm clipping or coiling were excluded. Because the unit of observation in NIS is discharge after hospitalization, cases transferred to another hospital were excluded in order to prevent double counting of the same patient. In our clinical database, we excluded interhospital transfers. Use of our clinical database was approved by the Johns Hopkins University School of Medicine Institutional Review Board (IRB). Analysis of NIS data is exempt from IRB approval given that they are publicly available and contain no personal identifying information.

Clinical Data Collection

Clinical stroke database

The Johns Hopkins clinical stroke database contains demographic data comprising age, sex, and self-identified race of patients presenting to the emergency room. Patients who were transferred in were excluded to prevent referral bias. The presence of risk factors, including hypertension, hyperlipidemia, diabetes mellitus, smoking status, chronic kidney disease (CKD), and past history of ICH, as well as the prehospital use of antiplatelet agents, anticoagulation, and statins, were obtained from electronic medical records. The following physiological parameters at presentation were recorded: GCS, blood pressure (BP), international normalized ratio (INR), serum glucose, and glomerular filtration rate (GFR). In addition, we collected data on total length of hospitalization and discharge location. Presence of an external ventricular drain (EVD) was recorded for each patient.

Nationwide Inpatient Sample

In NIS, we calculated the Charlson Comorbidity Index (CCI), a weighted score of 17 different comorbidities validated for outcome adjustment for analyses of administrative data sets using ICD-9-CM codes.[19,20] Case severity was determined using the all-patient refined diagnosis-related groups (APR-DRGs), a 4-point ordinal scale (minor, moderate, major, and extreme risk of mortality) derived from age, primary and secondary diagnoses, and procedures.[21]

Neuroimaging Analysis

In our clinical stroke database, ICH location on admission computed tomography (CT) scan was categorized as deep, lobar, cerebellar, or brainstem, and ICH volume was calculated by the ABC2 method as described previously.[22] All images were reviewed by a board-certified vascular neurologist (R.F.), blinded to the patients' race and the primary outcome. A second investigator (V.C.U.) reviewed randomly selected images for just over 10% of the sample, and an intraclass correlation coefficient (ICC) for a 2-way random effects model was used to assess inter-rater agreement of ICH volume (ICC, 0.89; 95% CI, 0.79–0.94). Presence of intraventricular hemorrhage (IVH) and subarachnoid extension (SAE) was recorded. Every patient underwent follow-up head CT in the first 6 to 24 hours. Hematoma expansion was defined as a proportional increase of more than 33% or an absolute increase greater than 6 cc (if baseline ICH volume $\leq$15 cc) from the initial ICH volume.[23] Hydrocephalus was defined as rounding of the frontal horns with increased radius or decreased ventricular angle, increased width of the temporal horns, rounding and enlargement of the atrium with sulcal effacement, increased width of the third ventricle, or ballooning of the fourth ventricle.[24] ICH score was computed for each patient as previously described.[13] Neuroimaging from the NIS was not available for analysis.

Statistical Analysis

Statistical analysis was performed using Stata software (version 13; Stata Statistical Software: Release 13; StataCorp LP, College Station, TX). A P value of <0.05 was considered statistically significant; 95% CIs are reported.

For analysis of data derived from the Johns Hopkins stroke database, continuous variables were analyzed using Wilcoxon rank-sum tests (Mann–Whitney U test). Categorical variables were analyzed using Pearson's chi-square test and Fisher's exact tests, when appropriate. The primary outcome was in-hospital mortality. All nonwhite, nonblack patients (n=4 in our clinical database; n=9232 in the NIS) were excluded from the analysis. Cases with missing information on race in NIS were excluded as well (n=10 126). Multivariable logistic regression

models were developed for blacks and whites separately using age, sex, and other variables known to be associated with mortality after ICH, specifically GCS, ICH volume, infratentorial origin, and IVH. In addition, statistically significant variables derived from the univariate analysis as well as other variables that may plausibly be associated with mortality were considered; however, final model selection was based on the Akaike information criterion. Data regarding smoking status were missing in 13 patients; thus, the sample size in multivariable models was reduced after including smoking.

In the NIS, comparisons of EVD placement (ICD-9-CM procedure codes 02.2/02.21) and obstructive hydrocephalus (ICD-9-CM code 331.4) among black and white patients were made using the chi-square test. Univariate logistic regression was performed to determine the unadjusted association of race with EVD placement or hydrocephalus, respectively. Multivariable models were adjusted for age, sex, hospital characteristics (teaching status, bed size, location, region, and ICH case volume), discharge quarter, weekend admission, modified CCI, APR-DRG subclass, hypertension, diabetes mellitus, dyslipidemia, coronary artery disease, congestive heart failure, atrial fibrillation, valvular disease, anemia, thrombocytopenia, alcohol abuse, drug abuse, CKD, transfusion of blood products, craniotomy/craniectomy, withdrawal of care status, and death. We used a generalized estimation equations approach to account for clustering of patients within hospitals.

Results

Patient Characteristics

A total of 226 black and white patients with spontaneous ICH meeting our inclusion criteria presented to our institutions between January 2010 and December 2013. Median age was 62 years (interquartile range [IQR], 52–78), and 52.2% were male. Baseline characteristics of black (115; 50.9%) and white (111; 49.1%) patients are presented in Table 1.

Race-Specific Predictors of In-Hospital Mortality

In order to identify differential predictors of mortality among blacks and whites, we performed univariate logistic regression analysis of in-hospital mortality stratified by race (Table 2). In blacks, GCS, serum glucose, history of diabetes, ICH volume, and IVH were associated with in-hospital mortality. In whites, GCS, ICH volume, and SAE were associated with mortality. Multivariable logistic regression identified GCS, diabetes mellitus, ICH volume, and IVH as independent predictors of in-hospital mortality in blacks (area under the curve [AUC] for the model, 0.889; 95% confidence interval [CI], 0.809–0.968; Table 3). In whites, age, GCS, CKD, smoking, and ICH volume were independently associated with mortality (AUC for the model, 0.902; 95% CI, 0.828–0.975).

Race as an effect modifier of the association between IVH and mortality was explored further: The unadjusted odds of in-hospital mortality among blacks with IVH were almost 4 times higher compared to whites presenting with IVH (odds ratio [OR], 3.92; 95% CI, 1.10–13.97; *P* value for IVH×race interaction, 0.035). In multivariable analysis, the odds of in-hospital mortality among blacks with IVH were over 7 times higher compared to whites presenting with IVH after adjusting for age, sex, glucose, diabetes, CKD, smoking, ICH volume, infratentorial origin, IVH, and SAE (OR, 7.27; 95% CI, 1.43–36.99; *P* value for IVH×race interaction, 0.017). Given that IVH might be more common in ICH originating from typical hypertensive locations, we additionally adjusted for ICH location; however, this did not alter the differential effect of race on IVH as a predictor of mortality (OR, 7.26; 95% CI, 1.40–37.80; *P* value for interaction, 0.018).

What Drives the Differential Impact of IVH by Race: Interplay Between Hydrocephalus and Age

In order to explore the mechanism by which mortality in the setting of IVH is increased in blacks compared to whites, we compared EVD placement rates by race. Among patients presenting with IVH, blacks were more likely to undergo EVD placement compared to whites (48.1% vs 27.9%; *P*=0.045), suggesting increased frequency of subsequent hydrocephalus necessitating EVD placement in blacks. In logistic regression analysis, the odds of EVD placement among blacks was increased over 2-fold compared to whites (OR, 2.39; 95% CI, 1.01–5.65); this association was confounded by age (OR, 0.98; 95% CI, 0.35–2.79 after including age into the model). Age was negatively associated with EVD placement after adjusting for race, sex and volume (OR, 0.47 per 10 year increase in age; 95% CI, 0.30–0.73). We then compared the rate of hydrocephalus on admission CT by race among the 95 IVH patients. Blacks were more likely to present with hydrocephalus compared to whites (35 of 52; 67.3% vs 21/43; 48.8%). In regression analysis, odds of hydrocephalus was 2.76 times higher among blacks compared to whites after adjusting for sex and ICH volume (95% CI, 1.12–6.73); however, this association was attenuated with loss of statistical significance after adjusting for age (OR, 1.97; 95% CI, 0.75–5.21).

We sought to validate these findings in the NIS data set: 8784 black and 33 293 white patients with a primary diagnosis of ICH between 2007 and 2011 were included. Among white ICH patients, 5.2% underwent EVD placement, whereas 10.3% of black patients with ICH received EVD

Table 1. Baseline Characteristics Among Hospitalized Patients With ICH, by Race

Characteristics	All Patients (n=226)	Black (n=115)	White (n=111)	*P* Value
Age, y: median (IQR)	62 (52–78)	56 (49–62)	71 (60–84)	<0.001
Sex, male: n (%)	128 (56.6)	60 (52.2)	68 (61.3)	0.168
GCS, median (IQR)	14 (8–15)	13 (8–15)	14 (8–15)	0.496
BP, mm Hg: median (IQR)				
SBP	191 (160–220)	207 (170–224)	179 (150–219)	<0.001
DBP	102 (87–120)	110 (94–129)	96 (79–110)	<0.001
Glucose, mg/dL: median (IQR)	134 (108–173)	135 (107–174)	132 (109–173)	0.919
GFR, mL/min per 1.72 m^2				0.183
>60	159 (70.4)	80 (69.6)	79 (71.2)	
30 to 60	45 (19.9)	20 (17.4)	25 (22.5)	
<30	22 (9.7)	15 (13.0)	7 (6.3)	
INR, median (IQR)	1.1 (1.0–1.2)	1.1 (1.0–1.1)	1.1 (1.0–1.2)	0.495
Comorbidities, n (%)				
Hypertension	186 (82.3)	99 (86.1)	87 (78.4)	0.129
Hyperlipidemia	84 (37.2)	30 (26.1)	54 (48.7)	<0.001
Diabetes mellitus	47 (20.8)	28 (24.4)	19 (17.1)	0.181
Previous hemorrhagic stroke	12 (5.3)	6 (5.2)	6 (5.4)	0.950
Chronic kidney disease	27 (12.0)	15 (13.0)	12 (10.8)	0.605
Current smoking (n=213)	66 (31.0)	43 (38.4)	23 (22.8)	0.014
Medications, n (%)				
Antiplatelet agent	83 (36.7)	31 (27.0)	52 (46.9)	0.002
Anticoagulation	21 (9.3)	5 (4.4)	16 (14.4)	0.009
Statin	60 (26.6)	20 (17.4)	40 (36.0)	0.002
Imaging				
ICH vol., cc: median (IQR)	15 (5–40)	10 (4–26)	20 (6–54)	0.005
Hematoma expansion, n (%)	26 (11.5)	10 (8.7)	16 (14.4)	0.128
Infratentorial origin, n (%)	40 (17.7)	22 (19.1)	18 (16.2)	0.505
Location				0.211
Lobar, n (%)	62 (27.4)	25 (21.7)	37 (33.3)	
Deep, n (%)	119 (52.7)	64 (55.6)	55 (49.6)	
Brainstem, n (%)	17 (7.5)	11 (9.6)	6 (5.4)	
Cerebellum, n (%)	23 (10.2)	11 (9.6)	12 (10.8)	
Isolated IVH	5 (2.2)	4 (3.5)	1 (0.9)	0.188
Intraventricular blood, n (%)	95 (42.0)	52 (45.2)	43 (38.7)	0.324
SAE, n (%)	45 (19.9)	15 (13.0)	30 (27.0)	0.008
ICH score, median (IQR)	2 (1–3)	1 (0–3)	2 (1–3)	0.032
LOS, days: median (IQR)	9 (4–19)	9 (4–22)	8 (3–16)	0.246
Discharge				0.003
Home, n (%)	35 (15.5)	25 (21.7)	10 (9.0)	
ACIR, n (%)	68 (30.1)	40 (34.8)	28 (25.2)	
SA, n (%)	50 (22.1)	23 (20.0)	27 (24.3)	
In hospital death, n (%)	73 (32.3)	27 (23.5)	46 (41.4)	

P values compare black and white ICH patients by nonparametric tests. ACIR indicates acute inpatient rehabilitation; BP, blood pressure; DBP, diastolic BP; eGFR, estimated glomerular filtration rate; GCS, Glasgow Coma Scale; ICH, intracerebral hemorrhage; INR, international normalized ratio; IVH, intraventricular hemorrhage; SA, subacute rehabilitation; SAE, subarachnoid extension; SBP, systolic BP.

Table 2. Univariate Logistic Regression for Predictors of In-Hospital Mortality by Race

	Black (N=115)			White (N=111)		
Variable	OR	95% CI	*P* Value	OR	95% CI	*P* Value
Age, y	1.00	0.97–1.03	0.897	1.02	0.99–1.05	0.153
Sex, male	0.81	0.34–1.92	0.632	0.71	0.33–1.54	0.389
GCS	0.78	0.70–0.87	<0.001	0.76	0.68–0.86	<0.001
SBP, per 10 mm Hg	0.98	0.89–1.09	0.751	1.06	0.96–1.17	0.218
DBP, mm Hg	0.99	0.84–1.15	0.858	0.98	0.84–1.15	0.812
Glucose, per 50 mg/dL	1.34	1.05–1.17	0.019	1.08	0.87–1.35	0.471
INR	1.77	0.92–3.40	0.086	1.08	0.77–1.51	0.642
GFR, mL/min per 1.72 m^2						
>60	Ref			Ref		
30–60	1.33	0.42–4.21	0.624	0.83	0.33–2.10	0.688
<30	2.67	0.83–8.59	0.100	3.67	0.67–20.10	0.134
Comorbidities						
Hypertension	0.45	0.15–1.38	0.161	1.23	0.49–3.12	0.658
Hyperlipidemia	1.26	0.49–3.29	0.632	0.95	0.44–2.01	0.884
Diabetes mellitus	2.87	1.13–7.29	0.027	1.03	0.38–2.81	0.949
Chronic kidney disease	1.22	0.35–4.19	0.755	2.15	0.64–7.27	0.216
Smoking (n=213)	1.24	0.51–3.02	0.640	1.73	0.68–4.44	0.253
Medications						
Antiplatelet agent	0.93	0.35–2.49	0.890	1.24	0.58–2.65	0.576
Anticoagulation	5.38	0.85–34.03	0.074	1.50	0.52–4.34	0.454
Statin	1.11	0.36–2.39	0.860	0.91	0.41–2.01	0.817
Imaging						
ICH volume, per 10 cc	1.26	1.08–1.47	0.003	1.34	1.16–1.55	<0.001
Infratentorial origin	1.61	0.58–4.49	0.363	1.19	0.43–3.29	0.739
Location						
Deep	Ref			Ref		
Lobar	1.13	0.38–3.36	0.829	2.00	0.85–4.68	0.110
Brainstem	2.04	0.52–7.98	0.305	3.79	0.64–22.61	0.144
Cerebellum	1.34	0.31–5.73	0.694	0.63	0.15–2.61	0.526
Intraventricular blood	6.44	2.35–17.62	<0.001	1.64	0.76–3.56	0.210
SAE	1.77	0.55–5.73	0.339	3.45	1.44–8.28	0.005

DBP indicates diastolic blood pressure; eGFR, estimated glomerular filtration rate; GCS, Glasgow Coma Scale; ICH, intracerebral hemorrhage; INR, international normalized ratio; SAE, subarachnoid extension; SBP, systolic blood pressure.

(*P*<0.001; we were unable to evaluate EVD placement rates among only those patients with IVH because there is no separate ICD-9-CM code for IVH). Similarly, blacks were more likely to develop hydrocephalus compared to whites (14.5% vs 8.4%; *P*<0.001). Black patients had almost 2-fold increased odds of hydrocephalus compared to whites in the unadjusted model (OR, 1.84; 95% CI, 1.71–1.97); addition of age diminished this association (OR, 1.45; 95% CI, 1.35–1.56). In a model fully adjusted for demographic covariates, hospital characteristics, comorbidities, surrogates for case severity, and withdrawal of care status, black race remained associated with developing hydrocephalus (OR, 1.32; 95% CI, 1.20–1.46). Taken together, these data suggest that black patients more commonly develop hydrocephalus necessitating increased EVD placement rate, in part attributed to their younger age.

Table 3. Multivariable Analysis for Predictors of In-Hospital Mortality by Race

	Black			White		
Variable	OR	95% CI	*P* Value	OR	95% CI	*P* Value
Age, y	1.01	0.96–1.06	0.624	1.10	1.03–1.17	0.002
Sex, male	0.38	0.10–1.37	0.139	3.92	0.76–20.18	0.102
GCS	0.81	0.68–0.96	0.016	0.77	0.64–0.93	0.007
Glucose, per 50 mg/dL	0.87	0.55–1.37	0.546			
Diabetes mellitus	5.03	1.11–22.87	0.037			
Chronic kidney disease				23.35	3.03–180.09	0.003
Smoking	3.31	0.89–12.36	0.074	7.42	1.62–33.99	0.010
ICH volume, per 10 cc	1.27	1.05–1.53	0.013	1.48	1.12–1.94	0.005
Infratentorial origin	2.61	0.63–10.79	0.186	1.83	0.33–10.16	0.487
Intraventricular blood	4.89	1.24–19.20	0.023	1.65	0.41–6.66	0.482
SAE	0.16	0.02–1.31	0.087	1.58	0.34–7.41	0.562

GCS indicates Glasgow Coma Scale; ICH, intracerebral hemorrhage; SAH, subarachnoid extension.

Race-Specific Relevance of Predictors for ICH Score Performance

In our population, ICH score predicted in-hospital mortality well (AUC, 0.773; 95% CI, 0.707–0.840). Race-specific prediction was better among blacks (AUC, 0.823; 95% CI, 0.739–0.907; Table 4) than in whites (AUC, 0.727; 95% CI, 0.627–0.828), but this difference was not statistically significant (*P*=0.154).

We next sought to determine whether the relevance of the individual score components of the ICH score differs by race. Thus, we compared only those score components identified as relevant among blacks and whites by our model to the original ICH score, respectively. In blacks, the ICH score components, GCS, ICH volume, and IVH, as identified by our model, predicted in-hospital mortality well (AUC, 0.803). Addition of the remaining ICH score components, age and infratentorial origin, to the model did not significantly improve the AUC (Table 4). In whites, a partial ICH score only consisting of the components, age, GCS, and ICH volume, as identified by our model, predicted in-hospital mortality moderately well (AUC, 0.727); however, addition of IVH and infratentorial origin to the model did not change the AUC (Table 4).

Discussion

Most previous studies establishing predictors of mortality in ICH were limited in their ability to address between-race differences in their models, mainly because of relatively small sample size, relative under-representation of minorities, or greater racial variation. Here, we identified race-specific predictors of in-hospital mortality in ICH patients and determined the relevance of IVH and hydrocephalus for mortality among blacks and whites.

Table 4. Discriminative Ability of the Complete ICH Score Compared to a Race-Specific Partial ICH Score for Blacks and Whites

	Black		White	
ICH Score Components	ICH Score Complete	ICH Score Partial	ICH Score Complete	ICH Score Partial
Age	+	–	+	+
GCS	+	+	+	+
ICH volume	+	+	+	+
Infratentorial origin	+	–	+	–
IVH	+	+	+	–
AUC (95% CI)	0.823 (0.739–0.907)	0.803 (0.704–0.901)	0.727 (0.627–0.828)	0.727 (0.626–0.829)

AUC indicates area under the curve; GCS, Glasgow Coma Scale; ICH, intracerebral hemorrhage; IVH, intraventricular hemorrhage.

In our study, IVH was a strong predictor of short-term mortality among blacks, but not whites. The observed differential impact on mortality persisted after adjusting for ICH location, suggesting that it was unlikely to be attributed to an increased rate in blacks of ICH in typical hypertensive locations close to the cerebrospinal fluid (CSF) space. Impaired CSF flow and resultant hydrocephalus is one of the most important mechanisms by which IVH may contribute to morbidity and mortality in ICH. Blacks with ICH/IVH presented at a younger age in our study and in others.[3,6,7] Similarly, hydrocephalus after ICH with IVH occurs more commonly in younger patients.[24] Although it is not entirely clear why young IVH patients develop hydrocephalus at a higher and faster rate compared to older patients, the presence of pre-existing cerebral atrophy in older patients may delay hydrocephalus formation by allowing for some degree of compensation for additional intracranial volume. Thus, younger age at presentation may, at least in part, explain the higher rate of hydrocephalus among blacks with IVH compared to white IVH patients, necessitating more EVDs.

One of the most established predictive models of early mortality in ICH is the ICH score. Although the ICH score has been validated subsequently in Hispanic,[14] Asian,[15] and British cohorts,[16] ICH score performance and individual predictors have not been compared between different racial groups. The underlying population establishing the ICH score was predominantly Asian American and white, and blacks were underrepresented in the original cohort as well as subsequent validation cohorts. In addition, most of the validation studies have focused on score performance for an entire sample, and comparisons of predictors of mortality across races are lacking. In our study, the ICH score performed well overall in both blacks and whites, but only ICH volume and GCS were robust predictors among both racial groups. Age, as a component of the ICH score, was an important predictor of mortality in whites, but not blacks. The ICH score incorporates age as a predictor of mortality with a cutoff of 80 years. In our population only 10 of 115 (8.7%) black patients were 80 years or older at the time of presentation (3 of whom died), compared to 44 of 111 (39.6%) white patients (19 of whom died). This highlights the difference in age at presentation across racial groups and suggests that dichotomizing age at above versus below 80 is unlikely to contribute as an important predictor of early death among blacks. Because the ICH score encompasses both IVH as a good predictor of mortality in blacks but not whites, and includes age as a robust predictor of mortality in whites but not blacks, the overall performance of the ICH score in our study population was still moderate to good.

Our race-specific ICH score for blacks including only GCS, volume, and IVH performed as well as the original ICH score. Similarly, a score taking only GCS, volume, and age into account predicted mortality in whites as well as the original ICH score. The original ICH score performed well in either racial group in our population, but the main discriminative ability of the ICH score stems from different score components across races—GCS, ICH volume, and age in whites, and GCS, ICH volume, and IVH in blacks. We acknowledge that the original ICH score was developed to predict 30-day, and not in-hospital, mortality; however, in-hospital and 30-day mortality rates after stroke are highly correlated.[25]

Our study has several limitations. Analysis of our clinical stroke database included a relatively small number of patients derived from 2 single stroke centers, limiting generalizability to larger populations. In NIS, limitations include the potential for miscoding given that it may occur in large administrative data sets reliant on ICD-9-CM coding. In addition, NIS does not contain clinical and physiological data on ICH volume or location, intraventricular extension, or level of consciousness. Racial differences in the association of age and IVH with mortality in our institutional data set were corroborated by our analysis of hydrocephalus and EVD placement in NIS; however, the validity of other race-specific predictors of mortality identified by our institutional multivariable models is limited by the small sample size, and further validation in an external data set is needed. Our analysis was limited to black and white ICH patients, and our results cannot be extrapolated beyond these 2 racial groups. Though aggressiveness of care was not captured in our clinical data set, care withdrawal status was accounted for in the NIS, and all multivariable models using the NIS data set were adjusted for withdrawal of care status. Though aggressiveness of care may differ between whites and blacks, this is unlikely to influence the within-race predictive value of the variables identified in our study; that is, the presence of IVH is unlikely to differentially drive decision making regarding early withdrawal of care in one, but not the other, racial group. Despite these limitations, combining information from individual-level clinical data and large administrative data sets at the population level allows for identification of differences in predictors of mortality in blacks and whites.

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Disclosures

None.

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