Published in final edited form as:

J Crit Care. 2016 April ; 32: 3–8. doi:10.1016/j.jcrc.2015.11.012.

Troponin elevation predicts critical care needs and in-hospital mortality after thrombolysis in white but not black stroke patients

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Abstract

Introduction—Stroke patients undergoing intravenous thrombolysis (IVT) are at increased risk for critical care interventions and mortality. Cardiac troponin elevation is common in stroke patients; however, its prognostic significance is unclear. The present study evaluates troponin elevation as a predictor of critical care needs and mortality in post-IVT patients, and describes racial differences in its predictive accuracy.

Methods—Logistic regression and receiver operating characteristics (ROC) analysis were used to determine racial differences in the predictive accuracy of troponin elevation for critical care needs and mortality in post-IVT patients.

Results—Troponin elevation predicted critical care needs in white (OR 29.40, 95% CI 4.86–177.81) but not black patients (OR 0.50, 95% CI 0.14–1.78; p-value for interaction <0.001). Adding troponin elevation to a prediction model for critical care needs in whites improved the area under the curve (AUC) from 0.670 to 0.844 (p=0.006); however, addition of troponin elevation did not improve the model in blacks (AUC 0.843 vs. 0.851, p=0.54). Troponin elevation was associated with in-hospital mortality in whites (OR 21.94, 95% CI 3.51–137.11) but not blacks (OR 1.10, 95% CI 0.19–6.32; p-value for interaction 0.022).

Conclusion—Troponin is a useful predictor of poor outcome in white but not black post-IVT stroke patients.

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Conflict of interests

The authors declare that they have no conflict of interests.

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Keywords

stroke; IV thrombolysis; IV tPA; troponin; critical care needs; racial differences; racial disparities

Introduction

Intravenous thrombolysis with recombinant tissue plasminogen activator (IVT) is the only approved therapy for acute ischemic stroke and is currently the cornerstone of therapy for patients presenting within 4.5 hours of symptom onset [1]. Stroke patients undergoing IVT are at increased risk for needing critical care interventions due to sequelae of their underlying stroke as well as IVT-related complications. Post-IVT patients are monitored either in an intensive care unit (ICU) or a stroke unit [2]; however, no evidence-based triaging standards exist and triaging criteria for monitoring intensity and environment vary by institution. Since only approximately 30% of post-IVT patients require critical care resources [3], identification of a-priori factors predicting need for critical care interventions are needed.

Markers of myocardial injury, such as cardiac troponin I, are elevated in up to 30% of all stroke patients [4,5]. The etiology of elevated serum troponin in acute stroke patients is unclear, but may be related to increased cardiac strain in the setting of acute hypertension, may be caused by true thrombotic coronary ischemia, may reflect concomitant atrial fibrillation or congestive heart failure as the underlying stroke etiology, or may be related to myocardial damage in the setting of sympathoadrenal activation [6,7]. The prognostic significance of troponin elevation is unclear and whether troponin elevation is associated with poor outcome and mortality remains controversial [4,8]. Aforementioned studies investigating the predictive value of serum troponin in stroke patients were carried out in mainly white European populations, with underrepresentation of black patients. Black stroke patients may differ from their white counterparts with regard to underlying stroke etiology and risk factor profile, i.e. atrial fibrillation is more common in white compared to black stroke patients [9,10], while blacks more commonly present with refractory hypertension [11]; these racial differences might extend to differences in other clinical features on presentation, including troponinemia.

The present study aimed to determine the significance of troponin elevation as a predictor of critical care needs and mortality in stroke patients undergoing IVT, and explore the predictive value of troponin elevation differs by race. We tested the hypothesis that troponin elevation is an important and valuable predictor of need for ICU level of care in whites but not blacks. To our knowledge, this is the first study to explore racial differences in predictors of critical care needs after IVT in order to develop tailored prediction models.

Methods

Patients and study design

This study was approved by the Johns Hopkins University School of Medicine Institutional Review Board. A waiver of consent was granted based on 45 CFR 46.116. An IRB waiver

of HIPAA privacy authorization was also granted to allow review of medical records to abstract data to de-identify for use in research. Patients who were treated with IVT for acute ischemic stroke in the emergency department at Johns Hopkins Hospital and Johns Hopkins Bayview Medical Center, per standard protocol, between January 2010 and December 2014 were identified from our prospectively collected stroke database.

Demographic data including age, sex, and race were collected for all patients. Other variables of interest, obtained from the medical record, included stroke risk factors: hypertension, hyperlipidemia, diabetes mellitus, smoking status, history of atrial fibrillation, history of coronary artery disease (CAD), prior history of stroke, and the pre-hospital use of antiplatelet agents, anticoagulation, and statins. NIH Stroke Scale (NIHSS) and the following physiologic parameters at presentation were recorded: blood pressure (BP), serum glucose, serum creatinine, glomerular filtration rate (GFR), and elevated troponin upon admission defined as a serum Troponin I >0.06 ng/ml within the first 6 hours of presentation. Decisions about whether troponin should be checked on an individual patient are made by the teams caring for the patient in the emergency department and on the neurology service, but it is standard for most patients undergoing IVT to get a troponin level checked on admission (pre-IVT). Data on total length of stay (LOS), length of ICU stay, discharge destination, withdrawal of care, and in-hospital mortality were collected. The presence of any critical care intervention was recorded while blinded to the troponin status of each patient. A critical care intervention was considered any therapy or intervention that required ICU resources as defined previously [3]. Specifically, ICU admission criteria included: uncontrolled hypertension requiring titration of IV antihypertensives, use of vasopressors either for symptomatic systemic hypotension or blood pressure augmentation, need for invasive hemodynamic monitoring, uncontrolled hyperglycemia requiring IV insulin, respiratory compromise resulting in either initiation of bilevel positive airway pressure (BiPAP) or mechanical ventilation, anaphylaxis, arterial bleeding, management of cerebral edema and increased ICP, neurosurgical intervention such as decompressive craniectomy, or symptomatic intracerebral hemorrhage (sICH) defined as any ICH with neurological deterioration, as indicated by a change in NIHSS ≥ 4 compared to the baseline as described previously [12]. Our definition of an ICU intervention also included ICU monitoring, as for patients with any event or complication that would require monitoring in an ICU setting even if no immediate ICU intervention was performed, such as progressive decrease in mental status with impaired airway protection, increasing oxygen requirement, or detection of potentially life-threatening arrhythmia.

IV thrombolysis protocol

At our institutions, IVT is administered according to the American Heart Association's national guidelines [2]. Post IVT monitoring conforms to the recommendations of the Brain Attack Coalition, which have become the standard of care for most stroke centers. All patients receiving IVT are monitored in the neurointensive care unit for at least 24 hours after initiation of thrombolysis, and undergo neuroimaging with either CT or MRI within 24 hours after treatment before being considered for transfer to the floor.

Statistical Analysis

Statistical analysis was performed using Stata version 13 (*Stata Statistical Software: Release 13*. College Station, TX). A two-sided p-value of <0.05 was considered statistically significant, and 95% confidence intervals are reported. For univariate analyses, continuous variables were analyzed using Student's t-tests for normally distributed variables, and Wilcoxon rank-sum tests (Mann-Whitney U test) for non-normally distributed variables. Categorical variables were analyzed using Pearson's Chi2 analysis, and Fisher's exact tests, when appropriate.

The primary outcome of interest was need for ICU-intervention, and in-hospital mortality was the secondary outcome. Serum troponin elevation on admission was the primary predictor of interest. Simple logistic regression was used to determine univariate associations of troponin elevation and need for critical care interventions by race. Multivariable logistic regression was performed adjusting for basic demographic variables as well as other variables previously published to be associated with ICU needs or felt to be potentially clinically relevant for predicting critical care interventions, such as NIHSS, systolic BP (SBP), and serum glucose. Since in-hospital mortality as our secondary outcome is a relatively rare event, we used logistic regression with Firth's penalized likelihood for analyses with mortality as the outcome of interest [13].

For prediction models we used Akaike information criterion (AIC) for model selection. The discriminative ability of the area under the receiver operating characteristics (ROC) curves of the final models with and without troponin were compared by using a non-parametric approach described by DeLong [14]. Model calibration was assessed with the Hosmer-Lemeshow test to determine goodness of fit, and the final prediction models were validated by leave-one-out cross validation.

Troponin values were missing in about 30% of patients. In order to ensure that missingness of troponin values did not influence our findings by introducing selection bias, we compared baseline characteristics of patients with missing and non-missing troponin values. We then used multiple imputation by chained equations (MICE) with 30 iterations to impute missing troponin values for 86 patients. We repeated the primary analysis with the imputed datasets, and the troponin values were averaged across the 30 imputed datasets [15].

Results

Patient selection and characteristics

Three hundred and one patients received IVT for acute ischemic stroke at our institutions between January 2009 and December 2014. Nine non-white, non-black patients were excluded. Of the remaining 292 patients, troponin measurements were available in 206 (70.5%), constituting the primary study population. There were no differences in the frequency of troponin collection by race (50.5% of patients with troponin were black, while 47.7% of patients without troponin were black, $p=0.66$). Compared to patients without measured troponin, patients with troponin were more likely to be male (53.4% vs. 40.7%, $p=0.048$), had higher NIHSS at presentation (median 8 vs. 6, $p=0.003$), and were more likely to have a history of atrial fibrillation (24.8% vs. 11.6%). Baseline characteristic of patients

with missing troponin values not included in the primary analysis can be found as supplemental table 1.

We compared baseline characteristics of blacks (104; 50.5%) to whites (102; 49.5%). Blacks were more likely to present at a younger age (median 60 years vs. 70 years, $p<0.002$), and had higher diastolic blood pressure (DBP) compared to whites (median 95 mm Hg vs. 87 mm Hg, $p=0.001$). White patients more commonly presented with a history of hyperlipidemia (59.8% vs. 43.3%, $p=0.018$) and CAD (35.3% vs. 19.2%, $p=0.010$). Serum troponin levels were elevated in 16.4% of black and 9.8% of white patients ($p=0.164$). Similarly, SBP (median 160 mm Hg in blacks vs. 154 mm Hg in whites) and NIHSS (median 7 in black vs. 8.5 in white patients) did not significantly differ between the 2 groups.

Black patients were more likely to require critical care interventions compared to Whites (40.4% vs. 21.6%, $p=0.004$). Among all patients requiring an ICU level of care, 31.4% of black patients and 28.6% of white patients underwent 2 or more critical care interventions. The most common critical care interventions for either racial group were IV drips for uncontrolled hypertension (19.2% in blacks vs. 9.8% in whites), respiratory compromise (17.3% in blacks vs. 7.8% in whites), and management of cerebral edema (9.6% in blacks vs. 3.9% in whites). Further baseline characteristics by race are presented in table 1.

Elevated troponin predicts need for ICU care in whites but not blacks

Among patients with troponin elevation, 51.9% required critical care interventions, while only 27.9% of patients with normal troponin values needed ICU level of care ($p=0.024$). Among whites, the proportion of patients requiring critical care was significantly higher in patients with compared to without troponin elevation (70% vs. 16.3%, $p=0.001$), while blacks had similar rates of ICU care regardless of their troponin status (41.2% with vs. 40.2% without troponin elevation, $p=1.000$). Supplemental table 2 shows the troponin elevation status of the study population stratified by critical care needs and race.

In simple logistic regression analysis, troponin elevation was significantly associated with requiring ICU level of care in the entire study population (odds ratio [OR] 2.78, 95% confidence interval [CI] 1.22–6.32). We then developed multivariable models to determine whether troponin elevation was differentially associated with critical care needs in whites vs. blacks independent of basic demographic variables, known cardiovascular comorbidities associated with troponin elevation such as CAD and atrial fibrillation, and other known predictors of ICU needs and poor outcome, such as NIHSS, SBP, and serum glucose. The various multivariable models are summarized in table 2; troponin elevation was not significantly associated with ICU needs in blacks (OR 0.50, 95% CI 0.14–1.78; model 1), after adjusting for age, sex, CAD, atrial fibrillation, NIHSS, SBP, and glucose. However, white patients with elevated troponin had over 29 times higher odds of requiring ICU level of care compared to those with normal troponin level (OR 29.40, 95% CI 4.86–177.81; p -value for interaction <0.001). Similarly, in multivariable models stratified by race, troponin elevation was significantly associated with critical care needs in white but not black patients after adjusting for age, sex, CAD, atrial fibrillation, NIHSS, SBP and serum glucose (OR 28.62, 95% CI 4.47–183.28 in whites vs. OR 0.27, 95% CI 0.06–1.22 in blacks; model 2).

Since our troponin covariate in our original study population (N=292) was missing in about 30% of patients, we used multiple imputation to impute missing troponin values for 86 patients. In order to ensure that our finding of troponin elevation predicting ICU needs in whites but not blacks was independent of any patterns of missingness, we tested the robustness of our multivariable model by performing sensitivity analysis including all 292 patients in our multivariable prediction model derived in the case-wise analysis above. Results obtained after including patients with imputed troponin values in the analysis were similar to the results of the case-wise analysis: troponin elevation was associated with ICU needs in whites (OR 14.13, 95% CI 2.53–78.91; model 3) but not blacks (OR 0.69, 95% CI 0.18–2.61; model 3) after adjusting for age, sex, CAD, atrial fibrillation, NIHSS, SBP and serum glucose. Among white patients troponin elevation was associated with approximately 20 times higher odds of requiring critical care interventions compared to black patients after adjusting for age, sex, CAD, atrial fibrillation, NIHSS, SBP and serum glucose (OR 20.39, 95% CI 2.30–180.39; p-value for interaction 0.007).

Discriminative value of troponin as predictor of ICU care in IVT patients

We then sought to determine whether troponin adds predictive value to established predictors of critical care needs among black and whites. Therefore, we developed race-specific prediction models of ICU needs separately for whites and blacks based on AIC values, and tested their discriminative ability by ROC analysis with and without troponin as a predictor. For whites, the final model comprised of SBP, NIHSS, and troponin achieved an area under the curve of AUC of 0.844 (95% CI 0.746–0.942); goodness of fit was confirmed by Hosmer-Lemeshow test (p=0.55). Omission of troponin from the model significantly reduced the discriminative ability of the model to 0.670 (95% CI 0.529–0.811; p-value for difference of the ROC curves with and without troponin in whites was 0.006; Figure 1). In blacks, the best fitting model comprised sex, NIHSS, SBP, and serum glucose, and achieved an AUC of 0.843 (95% CI 0.756–0.929), and Hosmer-Lemeshow test confirmed goodness of fit (p=0.115); however, adding troponin to the model did not substantially improve the AUC (AUC 0.851, 95% CI 0.767–0.935; p-value for difference of the ROC curves in blacks was 0.538). Figure 1 illustrates the differences in discriminative ability of the prediction models with and without troponin by race.

Elevated troponin predicts in-hospital mortality in whites but not blacks

Mortality rates were higher among patients with an elevated troponin than those with normal troponin (25.9% vs. 5.6%, p=0.002). Troponin elevation was associated with almost 6 times higher odds of in-hospital mortality in all patients (OR 5.91, 95% CI 2.08–16.76). Multivariable analysis identified troponin elevation as an independent predictor of in-hospital mortality in whites but not blacks after adjusting for potential confounders and other known causes of death in stroke patients, such as age, sex, CAD, atrial fibrillation, SBP, NIHSS, and serum glucose levels (OR 21.94, 95% CI 3.51–137.11 in whites vs. OR 1.10, 95% CI 0.19–6.32 in blacks; p-value for interaction 0.022). The direction and strength of this association did not change substantially after including withdrawal of care into the model (OR 19.57, 95% CI 1.15–332.22 in whites vs. OR 0.99, 95% CI 0.10–10.21 in blacks; p-value for interaction 0.045).

Further exploratory and unadjusted analysis revealed that in white but not black patients, troponin elevation was associated with need for cerebral edema treatment ($p=0.047$ vs. $p=1.000$), respiratory compromise ($p=0.030$ vs. $p=1.000$), and need for heart rate control ($p=0.047$ vs. $p=1.000$), but troponin elevation was not associated with need for aggressive blood pressure control in either race ($p=1.000$ in whites vs. $p=1.000$ in blacks). Mortality was associated with need for critical care intervention for respiratory compromise ($p<0.001$) and cerebral edema ($p<0.001$), but not with need for aggressive blood pressure control in our patient population ($p=0.194$). This suggests that troponin elevation in whites but not blacks may specifically predict ICU interventions conceivably associated with poor outcome and mortality, such as management of airway compromise, arrhythmias, and cerebral edema.

Discussion

Racial differences with regard to stroke incidence, location, and underlying etiology have been described; however, it is unclear whether the importance of clinical outcome predictors differs by race. Clinical parameters, such as NIHSS and SBP, have previously been used to predict critical care needs in post-IVT patients [3]. Previous studies aiming to address the role of troponin in the setting of acute stroke have been contradictory, and were conducted in predominantly white populations. In the present study, we show that serum troponin elevation on admission is associated with increased mortality and need for critical care resources in white but not black patients undergoing IVT.

In stroke patients, troponin is commonly measured upon admission, but its rate of routine measurement may vary across institutions, and may be driven by clinical decision-making rather than being part of routine work-up. In our sample, patients with measured troponin were more likely to be male, had higher NIHSS at presentation, and were more likely to have a history of atrial fibrillation, suggesting that clinical decision making may have resulted in selection of a sample at higher risk for cardiac events and poor outcome. Nonetheless, troponin elevation was associated with critical care needs and death in whites but not blacks, even after accounting for potential bias by using multiple imputation to impute the missing values, indicating that our results are generalizable to the population of IVT patients.

To be widely applicable in clinical practice, prediction models should be accurate, and reliably predict outcomes in the entire target population with readily available variables. Although troponin elevation was associated with need for critical care needs and mortality in our entire study population, this effect was exclusively driven by the strong association of troponin elevation with need for ICU care and death in whites but not blacks. It is noteworthy that our model predicting ICU needs in blacks has excellent discriminative ability even in the absence of troponin (AUC 0.843), while the discriminative ability of the model without troponin in whites is poor to fair (AUC 0.670). This suggests that need for ICU care is sufficiently explained by other parameters in black patients resulting in a ceiling effect, while white patients may benefit from evaluation of serum troponin for prediction of ICU needs. This illustrates that race-specific considerations may improve and refine existing prediction models to improve predictive accuracy.

Only two patients in our study population were diagnosed with acute coronary syndrome, while all other patients had only mildly increased serum troponin (median 0.2 ng/ml), suggesting that even a mild troponin elevation may be relevant in IVT patients. This is consistent with other reports indicating that troponin elevation may be a useful predictor of poor health outcomes, even if only mildly elevated and below thresholds typically seen in ACS [16,17]. In our sample, black patients had higher troponin levels compared to whites (median 0.77 ng/ml vs. 0.39 ng/ml). This difference was not statistically significant, but it refutes the possibility that the difference in predictive value of troponin elevation between whites and blacks is due to higher troponin levels in whites.

Reference limits for laboratory values may vary by race [18–20]. A recent study, analyzing normative values for the high-sensitivity Troponin T assay from 3 large observational cohorts stratified by age, sex and race found a trend towards higher values in black compared to non-black individuals [21]. To our knowledge, there are presently no reports on racial variations of normative values for Troponin I, but it is possible that the limited predictive value of troponin among black post-IVT patients is in part explained by racial variation of biologically relevant upper limits of normal for Troponin I.

Increased values of cardiac troponin are common in the setting of impaired renal function, at least in part due to reduced renal clearance [22,23]. Therefore, we repeated our analysis including serum creatinine as a covariate in all our models; however, our results remained unchanged, suggesting that differential renal impairment does not explain our observed differences of troponin elevation as a differential predictor of ICU needs and mortality among blacks and whites.

Our study has several limitations. This is a retrospective analysis of a relatively small number of patients. While ICU interventions, procedures, and medication administration were well documented in the vast majority of cases, relying on accuracy of medical records has the potential to result in missing or inaccurate information by virtue of the retrospective nature of this study. Our study population was derived from 2 single stroke centers over the course of 5 years. Therefore, extrapolating our results to community hospitals must be done with caution. In addition, our analysis was limited to black and white post-IVT patients, and our results cannot be extrapolated beyond these two racial groups. Since our study population was restricted to post-IVT patients, no valid inference can be made about stroke patients who are not eligible for IVT. Finally, indications for ICU admission and interventions may differ among institutions across the United States, and the model described in this study might therefore not be valid in institutions where ICU admission criteria differ significantly from ours. Further prospective studies are needed to determine whether troponin elevation may be used as a triaging tool for ICU care in white post-IVT patients.

Triaging decisions in stroke patients are multifactorial and complex. Clinicians have to take a variety of parameters into account when determining the appropriate monitoring environment for each patient, including demographics, physiological parameters, and disease trajectory. Accurate forecasting of mortality and optimal monitoring environment in post-IVT stroke patients may require future race-specific prediction models in order to

account for discrepancies in predictive ability of clinical, physiological, and laboratory factors, such as troponin elevation. We propose that troponin elevation on admission, if used in conjunction with other predictors of critical care needs, may improve the diagnostic accuracy of prediction models of critical care needs in white post-IVT stroke patients.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This publication was made possible by an institutional KL2 grant to Dr. Faigle from the Johns Hopkins Institute for Clinical and Translational Research (ICTR), which is funded in part by Grant Number KL2TR001077 from the National Center for Advancing Translational Sciences (NCATS) a component of the National Institutes of Health (NIH), and the NIH Roadmap for Medical Research.

List of abbreviations

AIC	Akaike information criterion
AUC	area under the curve
BiPAP	bilevel positive airway pressure
BP	blood pressure
CAD	coronary artery disease
CI	confidence interval
DBP	diastolic blood pressure
GFR	glomerular filtration rate
ICU	intensive care unit
IQR	interquartile range
IVT	intravenous thrombolysis
LOS	length of stay
MICE	multiple imputation by chained equations
NIHSS	NIH Stroke Scale
NNCI	neuroimaging negative ischemia
OR	odds ratio
ROC	receiver operating characteristics
SBP	systolic blood pressure
sICH	symptomatic intracerebral hemorrhage

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Figure 1.

The ROC curves for prediction models of critical care needs in whites (A) and blacks (B) are shown. A. ROC curve for the final model in whites including NIHSS, and SBP, with or without troponin. B. ROC curve for the final model in blacks including NIHSS, and SBP, sex, and serum glucose, with or without troponin.

Table 1

Baseline characteristics of all IVT patients with measured troponin stratified by race (N=206).

Characteristics	All patients (n=206)	White (n=102)	Black (n=104)	p-value
Age – years: median (IQR)	65.5 (52–78)	70 (58–80)	60 (49.5–73)	0.002
Sex – male: n (%)	110 (53.4)	61 (59.8)	49 (47.1)	0.068
NIHSS – median (IQR)	8 (5–13)	8.5 (5–14)	7 (5–12)	0.192
BP – mm Hg: median (IQR)				
SBP	157 (140–181)	154 (142–180)	160 (140–186)	0.306
DBP	90 (80–103)	87 (78–98)	95 (81–110)	0.001
IVT window <3hrs – n (%)	145 (70.4)	74 (72.6)	71 (68.3)	0.501
Endovascular therapy – n (%)	8 (3.9)	1 (1.0)	7 (6.7)	0.065
Risk factors for stroke – n (%)				
Hypertension	171 (83.0)	82 (80.4)	89 (85.6)	0.322
Hyperlipidemia	106 (51.5)	61 (59.8)	45 (43.3)	0.018
Diabetes mellitus	51 (24.8)	24 (23.5)	27 (26.0)	0.686
Coronary artery disease	56 (27.2)	36 (35.3)	20 (19.2)	0.010
Reduced ejection fraction	23 (11.2)	7 (6.9)	16 (15.4)	0.075
Atrial fibrillation	51 (24.8)	31 (30.4)	20 (19.2)	0.063
Prior ischemic stroke	61 (29.6)	24 (23.5)	37 (35.6)	0.058
Current smoking	63 (30.6)	31 (30.4)	32 (30.8)	0.953
Medications – n (%)				
Antiplatelet agent	89 (43.2)	48 (47.1)	41 (39.4)	0.269
Anticoagulation	17 (8.3)	12 (11.8)	5 (4.8)	0.080
Statin	80 (38.8)	46 (45.1)	34 (32.7)	0.068
Troponin elevation – n (%)	27 (13.1)	10 (9.8)	17 (16.4)	0.164
Glucose – mg/dl: median (IQR)	118 (104–156)	118 (104–159)	119 (104–155)	0.718
Creatinine – mg/dl: median (IQR)	1.0 (0.9–1.3)	1.0 (0.9–1.3)	1.0 (0.8–1.3)	0.915
GFR < 60 ml/min – n (%)	69 (33.5)	38 (37.3)	31 (29.8)	0.258
ICU indications – n (%)				
Blood pressure control	30 (14.6)	10 (9.8)	20 (19.2)	0.055
Respiratory	26 (12.6)	8 (7.8)	18 (17.3)	0.058
Cerebral edema	14 (6.8)	4 (3.9)	10 (9.6)	0.165
Symptomatic hemorrhage	5 (2.4)	2 (2.0)	3 (2.9)	1.000
Heart rate control	4 (1.9)	3 (2.9)	1 (1.0)	0.366
Vasopressors	7 (3.4)	2 (2.0)	5 (4.8)	0.445
Angioedema	2 (1.0)	0 (0)	2 (1.9)	0.948
Any ICU indication – n (%)	64 (31.1)	22 (21.6)	42 (40.4)	0.004
LOS – days: median (IQR)	5 (3–7)	5 (3–7)	4.5 (3–7)	0.963
ICU stay – days: median (IQR)	2 (1–2)	2 (1–2)	2 (1–2)	0.652

Characteristics	All patients (n=206)	White (n=102)	Black (n=104)	p-value
Discharge diagnosis – n (%)				0.432
Stroke (neuroimaging positive)	146 (70.9)	73 (71.6)	73 (70.2)	
NNCI	28 (13.6)	16 (15.7)	12 (11.5)	
Stroke mimic	32 (15.5)	13 (12.8)	19 (18.3)	
Discharge to home – n (%)	106 (51.5)	48 (47.1)	58 (55.8)	0.664
Care withdrawal/hospice – n (%)	11 (5.3)	7 (6.9)	4 (4.9)	0.541
Mortality – n (%)	17 (8.3)	9 (8.8)	8 (7.7)	0.805

DBP: diastolic blood pressure; GFR: glomerular filtration rate; ICU: intensive care unit; IQR: interquartile range; IVT: intravenous thrombolysis; LOS: length of stay; NIHSS: NIH Stroke Scale; NNCI: neuroimaging negative ischemia; SBP: systolic blood pressure. P-values compare black to white patients.

Table 2

Multivariable models for troponin as a predictor of critical care needs by race.

Model	Black			White			p for interaction
	OR	95% CI	p	OR	95% CI	p	
Multivariable with interaction (model 1)	0.50	0.14–1.78	0.282	29.40	4.86–177.81	<0.001	<0.001
Multivariable stratified by race (model 2)	0.27	0.06–1.22	0.089	28.61	4.47–183.28	<0.001	N/A
Multivariable with interaction after MICE (model 3)	0.69	0.18–2.61	0.587	14.13	2.53–78.91	0.003	0.007

All models were adjusted for age, sex, coronary artery disease, atrial fibrillation, NIH Stroke Scale, systolic blood pressure, and glucose. MICE: Multiple Imputation by Chained Equations; OR: odds ratio; CI: confidence interval.