


ORIGINAL RESEARCH

# Association Between Black Race, Clinical Severity, and Management of Acute Pulmonary Embolism: A Retrospective Cohort Study

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**BACKGROUND:** Existing evidence indicates Black patients have higher incidence of pulmonary embolism (PE) and PE-related mortality compared with other races/ethnicities, yet disparities in presenting severity and treatment remain incompletely understood.

**METHODS AND RESULTS:** We retrospectively queried a multihospital healthcare system for all hospitalizations for acute PE (2012–2019). Of 10 329 hospitalizations, 8743 met inclusion criteria. Black patients (14.3%) were significantly younger ( $54.6 \pm 17.8$  versus  $63.1 \pm 16.6$  years;  $P < 0.001$ ) and more female (56.1% versus 51.6%;  $P = 0.003$ ) compared with White patients. Using ordinal regression, Black race was significantly associated with higher PE severity after matching 1:3 on age and sex (1210:3264; odds ratio [OR], 1.08; 95% CI, 1.03–1.14), adjusting for clinical (OR, 1.13; 95% CI, 1.01–1.27), and socioeconomic (OR, 1.05; 95% CI, 1.05–1.35) characteristics. Among intermediate and high-severity PE, Black race was associated with a decreased risk of intervention controlling for the competing risk of mortality and censoring on hospital discharge. This effect was modified by PE severity ( $P$  value  $< 0.001$ ), with a lower and higher risk of intervention for intermediate and high-severity PE, respectively. Race was not associated with in-hospital mortality (OR, 0.84; 95% CI, 0.69–1.02).

**CONCLUSIONS:** Black patients hospitalized with PE are younger with a higher severity of disease compared with White patients. Although Black patients are less likely to receive an intervention overall, this differed depending on PE severity with higher risk of intervention only for life-threatening PE. This suggests nuanced racial disparities in management of PE and highlights the complexities of healthcare inequalities.

**Key Words:** healthcare disparities ■ outcomes ■ pulmonary embolism ■ racial disparities ■ venous thromboembolism

**R**ace-related disparities in access to and quality of health care are well documented. Black Americans have shorter life expectancy,<sup>1</sup> experience more severe morbidity associated with chronic illness,<sup>2,3</sup> and achieve fewer quality healthcare measures compared with White Americans.<sup>4,5</sup> This disparity is especially evident in the incidence<sup>6</sup> and mortality<sup>7</sup> secondary to venous thromboembolic (VTE) disease,

a serious and common process comprised of both pulmonary embolism (PE) and deep venous thrombosis, affecting more than 1 million Americans annually.<sup>8,9</sup> Although PE is the third most frequent cause of cardiovascular-related death,<sup>10</sup> accounting for 2% of deaths in the United States in 2017,<sup>11</sup> Black patients are disproportionately affected; they are almost twice as likely to be hospitalized for PE<sup>12</sup> and suffer 50% higher

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## CLINICAL PERSPECTIVE

### What Is New?

- Black race was associated with a higher pulmonary embolism severity with a decreased risk of receiving any intervention.
- Among intermediate severity pulmonary embolism hospitalizations, for which current intervention guidelines are in evolution, the reduced risk persisted.
- Yet, among hospitalizations for high-severity pulmonary embolism, Black race was associated with an increased risk of intervention.

### What Are the Clinical Implications?

- Pulmonary embolism hospitalizations exemplified severity and management differences across Black and White races highlighting the complexities of healthcare access, biases, and treatment preferences that shape racial disparities.

age standardized PE-related death compared with White patients.<sup>11</sup>

Contributing factors to healthcare disparities are complex and can be thought of in terms of the National Institute on Minority Health and Health Disparities Research Framework, which includes biological, behavioral, physical, sociocultural, and healthcare system domains over multiple levels of influence including individual, interpersonal, community, and societal.<sup>13,14</sup> Specifically, how race may influence disease severity and management of acute PE is incompletely understood. We hypothesize that Black race is associated with higher acute PE severity on hospital presentation and lower incidence of catheter-based or surgical intervention compared with White race. Our aim is to identify and understand racial inequalities associated with presentation and management of acute PE in order to explain previously noted differences in clinical outcomes related to PE in Black and White patients and inform treatment algorithms and resource allocation. Addressing disparities in every aspect of health care is not only key for advancing racial equality in healthcare delivery and outcomes, which is likely beneficial for a prevalent and morbid condition such as PE, it has also been shown to have a significant economic benefit.<sup>15,16</sup>

## METHODS

The data that support the findings of this study are available from the corresponding author upon reasonable request. UPMC is a large multihospital, single healthcare institution with a mix of private practice and

academic facilities including over 20 hospitals. Our retrospective cohort study used data from 2012 to 2019 on medical care that are captured through structured administrative and clinical databases and an electronic health record (EHR), which are all integrated into the UPMC's Clinical Data Warehouse, to evaluate the association between pulmonary embolism presentation severity, treatment, and outcomes for Black and White races. Patients diagnosed with an acute PE are typically evaluated by the PE response team, formally integrated in 2014, which includes pulmonary medicine, an interventionalist (ie, interventional cardiology or vascular surgery), cardiothoracic surgery, and hematology. This study was approved by the University of Pittsburgh Institutional Review Board (STUDY20120001). All data collection, analysis (Stata 15.1; Stata Corp), and presentation were in compliance with Strengthening the Reporting of Observational Studies in Epidemiology.<sup>17</sup>

## Data Source

At the time of EHR data abstraction (November 30, 2020), our regional healthcare network comprised 22 community and academic hospitals throughout southwestern Pennsylvania and the surrounding states, serving a diverse patient population that is connected digitally. Our data set provides baseline patient-level demographics and comorbidities as well as clinical, procedural, and outcome hospital admission data. Baseline patient demographics (ie, sex, race, and ethnicity), socioeconomic factors, comorbid conditions, and medications were obtained from the outpatient medical record (Epic Systems Corporation) based on data available 90 days before admission. Socioeconomic factors included the Area of Deprivation Index, a measure of neighborhood disadvantage based on Census data incorporating information on education, employment, housing quality, and poverty. It is a validated tool that has linked residing in the top 15% to 20% of the most disadvantaged neighborhoods to limited healthcare access, care, and poor outcomes.<sup>18</sup> Smoking status was quantified by any (ie, former or current) or no (ie, never) prior exposure. Patients were considered to be in the postoperative period if they underwent a surgical intervention in the 90 days before hospital admission within our healthcare system.<sup>9</sup> Baseline comorbid conditions were quantified by the presence of validated *International Classification of Diseases, Ninth Revision, Clinical Modification (ICD-9-CM)* or *International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM)* codes, as previously published.<sup>19</sup> Medications were identified by the presence of an e-prescription or an active, provider-reviewed EHR medication list. These data were linked to the in-patient EHR (CERNER platform, Millennium) with unique patient identifiers. Hospitalization data included age, insurance status, first vital sign (heart rate, systolic blood

pressure), serum laboratory value (troponin-I, BNP [B-type natriuretic peptide], creatinine, hemoglobin, international normalized ratio), and hospitalization events. Hospitalization events including intensive care unit admissions, relevant consultations (ie, pulmonology, cardiology, vascular surgery, and cardiothoracic surgery), echocardiograms, and treatment interventions were identified by pertinent *Current Procedural Terminology* codes or EHR data (Table S1). Treating hospitals were categorized by small, medium, or large in accordance with the National Inpatient Sample, which account for bed size, rurality, and teaching status.<sup>20</sup>

## Study Cohort

We identified all adult ( $\geq 18$  years) acute care hospital admissions between the years 2012 and 2019 with a primary diagnosis of acute PE according to *ICD-9-CM* (415.11, 415.12, 415.13, and 415.19) and *ICD-10-CM* (I26.01, I26.02, I26.09, I26.90, I26.92, I26.94, I26.94, and I26.99) diagnosis codes. We restricted our full cohort to exclude (1) hospitals with fewer than 100 admissions for PE throughout the study duration, (2) patients missing race and/or known pertinent medical history required for classification of presentation severity, (3) patients who did not self-identify as Black or White race, and (4) multiple, separate (ie, discharge and re-admission) admissions in the healthcare network for a single PE. Intra-healthcare network transfers were defined by the presence of 2 admissions for PE occurring within 24 hours at 2 separate hospitals. PE presentation data were combined and only the final treating (ie, tertiary care center) hospitalization was included. Multiple admissions for the same PE event were defined as 2 or more separate admissions within 30 days that were not intra-healthcare network transfers. Missingness of these data, which may in part be representative of healthcare disparities and cannot be considered missing at random, was quantified (Table S2).

## Outcomes

Our primary outcome of interest was PE severity on presentation classified into (1) high (massive), (2) intermediate (submassive), or (3) low severity using recent guidelines published by the European Society of Cardiology (Table S3).<sup>21</sup> Briefly, high-severity PE included evidence of hemodynamic instability, defined by vasopressor support or admission hypotension (systolic blood pressure  $< 90$  mm Hg).<sup>22</sup> Intermediate severity PE included patients without hypotension but with abnormal biomarkers and cardiac workup on presentation (including right heart strain, elevated troponin-I or BNP, vital sign abnormalities, with or without admission to intensive care unit).<sup>23</sup> All others were considered low severity. The accuracy of the EHR PE severity definition was clinically adjudicated via 2 blinded

reviewers on a random subset of included hospitalizations in each severity group with 100% agreement (Table S3).

Secondary in-hospital outcomes included the receipt of PE specific interventions including systemic therapies (ie, systemic thrombolysis administration), targeted therapies (ie, catheter directed therapies or surgical embolectomy), and preventative therapies (ie, inferior vena cava filters), as well as in-hospital mortality.

## Statistical Analysis

Demographic, socioeconomic, and presenting hospitalization data were assessed for White and Black race patients. Continuous variables were expressed as mean ( $\pm$ SD) or median ( $\pm$ interquartile range) for normally or skewed data distributions and compared with Student *t* test or Kruskal–Wallis tests. Categorical variables were expressed as frequency (percentage) and compared with chi-square tests. In the full cohort meeting all inclusion and no exclusion criteria, the percentage of PE hospitalizations per age of presentation by race was depicted with histograms overall and for each level of PE severity.

We compared the PE severity, treatments, and outcomes between the Black and White races. We matched Black and White patients 1:3 without replacement on only preexisting, unmodifiable factors including age (year integer) and sex (binary). To explore the association between outcomes and race, a series of models were used to understand how other existing risk factors may contribute to baseline disparities including (1) the age- and sex-matched cohort, (2) adjusting for comorbid clinical characteristics, and (3) both comorbid clinical and socioeconomic characteristics.<sup>24,25</sup> All models were clustered on hospital size as defined by the Agency for Healthcare Research and Quality, which accounts for bed size, rurality, and teaching status.<sup>20</sup> Grouping in this way allowed similar hospitals (ie, large academic tertiary care hospitals) to be grouped together based on typical functionality rather than clustering by individual hospitals, which introduced an excessive amount of variability.

We evaluated the association between race and PE severity low, intermediate, or high, in the matched cohort using ordinal regression. We evaluated the effect modification on the association between race and outcomes in predefined subgroups (those in the post-operative period, body mass index  $\geq 35$  mg/kg<sup>2</sup>, sex, and a prior history of VTE) with an interaction term, in the matched cohort. A *P* value threshold of 0.05 was used to denote significance including among interaction terms.

To evaluate the directionality of the association between Black race and the receipt of in-patient PE intervention in those at risk of a procedure (ie, those alive

and in the hospital), we used Fine-Gray models. Among hospitalization for intermediate and high-severity PE, we evaluated the association between race and any PE intervention, systemic therapies, targeted therapies, and preventative therapies controlling for the competing risk of mortality and censoring for hospital discharge. Subgroup analysis was completed to evaluate the association between the receipt of an intervention and race by PE severity level. Finally, we evaluated the association between race and in-hospital mortality using logistic regression.

### Sensitivity Analysis

To understand the robustness of our results, we completed 3 sensitivity analyses. First, we evaluated our primary outcome among matched patients with 2 alternative definitions of PE severity including (1) high severity (systolic blood pressure <70 mm Hg), intermediate severity (systolic blood pressure >70 and <90 mm Hg), and low severity for all others and (2) alterations in intermediate severity only, which included removing preexisting conditions from the definition as existing and recorded diagnosis may represent either health care and access to care disparities across races. Second, we explored for potential sampling bias in our matching technique and evaluated if our outcomes were sensitive to the matching methodology by evaluating point estimates in the full, unmatched cohort with multivariable regression including (1) age and sex; (2) age, sex, and clinical characteristics; and (3) age, sex, and clinical as well as socioeconomic characteristics as covariates. Third, we considered the effect of the healthcare network-wide implementation of a PE response team that includes multiple consulting services to streamline the management of acute PE, including pulmonary medicine, an interventionalist (ie, interventional cardiology or vascular surgery), cardiothoracic surgery, and hematology. This service has been shown to improve outcomes<sup>26</sup> and was formally implemented at UPMC in 2014. Thus, to explore the impact of a PE response team in our study, we restricted our analysis in the full cohort to only these years.

## RESULTS

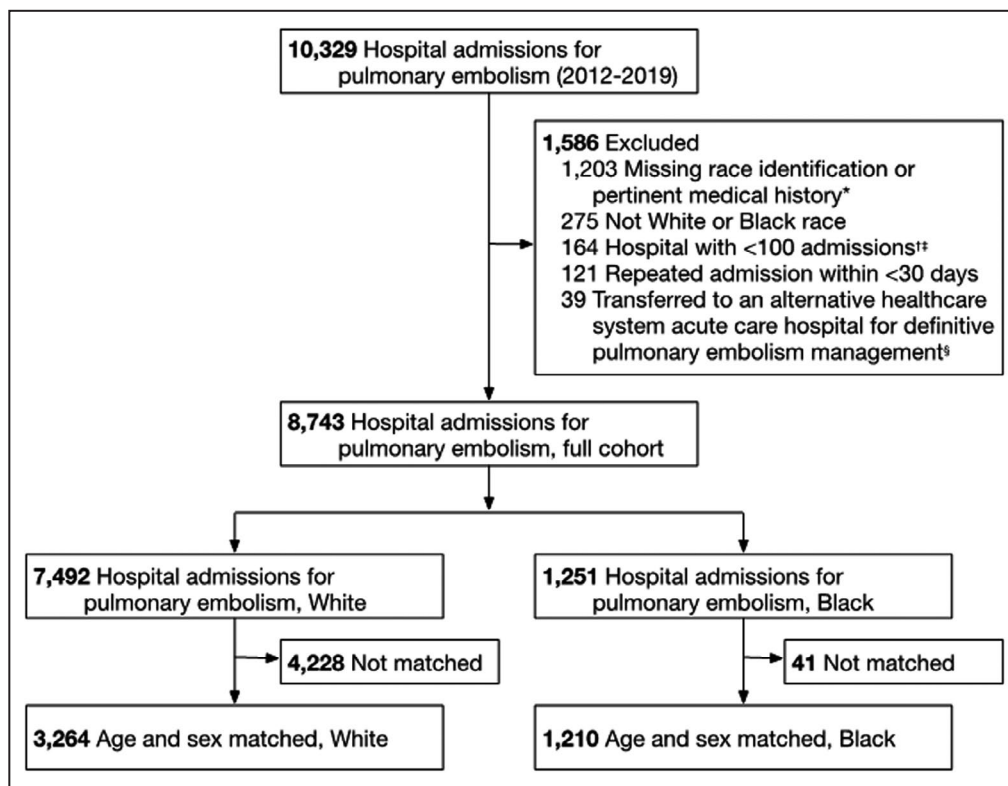
We identified 10 329 hospitalizations with a primary diagnosis of PE, of which 8743 met all inclusion and no exclusion criteria (age, 61.9±17.0 years; 4567 [52.2%] women) including 14.3% (n=1251) who self-identified as Black (Figure 1). Overall, 11.1% hospitalizations were for low-, 84.0% intermediate, and 4.9% high-severity PE presentation for which 8.7% underwent any intervention, and 2.9% suffered in-hospital mortality. Black patients were younger (54.6±18.8 versus 63.1±16.6 years),

more female (56.1% versus 51.6%), resided in the top 20% most disadvantaged neighborhoods (70.3% versus 22.9%), and had less private health insurance (19.5% versus 30.5%) when compared with White patients (Table 1).

Age at hospitalization for Black patients occurred at a younger age when compared with White patients overall (Figure 2A), and among those in the low-, intermediate, and high-severity PE groups (Figure 2B). Matching on age and sex in a 1:3 ratio of Black to White patients resulted 1210 Black and 3264 White patients. In our matched cohort, the age differences decreased from nearly 10 years to 1 year. Statistically significant differences between groups, such as cardiopulmonary comorbidities and the prevalence of cancer, remained (Table 1; Figure S1). Unmatched Black patients (n=41) were younger, more female, with a higher body mass index and more likely to have a history of VTE, whereas unmatched White patients (n=4228) were older, more male, with a lower body mass index, and less likely to have a history of VTE (Table S4).

In the matched cohort, 12.6% of hospitalizations were for low-, 82.9% intermediate, and 4.5% high-severity PE (Table S5). Observed trends were consistent among severity groups. Clinical severity on presentation was associated with race after accounting for matching characteristics (OR, 1.08; 95% CI, 1.03–1.14), and after adjusting for both clinical (OR, 1.13; 95% CI, 1.01–1.27) as well as clinical and socioeconomic variables (OR, 1.05; 95% CI, 1.05–1.35) with Black patients having a higher risk of presenting with a higher severity PE when compared with White patients (Table 2; Table S6).<sup>27</sup> On subgroup analysis, postoperative status, sex, and history of VTE were found to modify the association of race with PE severity (Figure 3), with a higher risk of PE severity appreciated in patients who were in a postoperative state (OR, 1.34; 95% CI, 1.11–1.60) compared with a non-postoperative state (OR, 1.04; 95% CI, 0.99–1.08), female (OR, 1.24; 95% CI, 1.01–1.53) compared with male (OR, 0.93; 95% CI, 0.86–1.00) sex, and those without a history of VTE (OR, 1.15; 95% CI, 1.09–1.22) compared with those with a history of VTE (OR, 0.97; 95% CI, 0.86–1.10).

There was a total of 462 hospitalizations with PE interventions in the matched cohort, and >95% occurred in those with intermediate and high-severity PE hospitalizations. Of these, 49 (1.3%) received systemic, 209 (5.7%) targeted, and 232 (6.3%) preventative interventions. Black patients had a lower risk of receipt of any intervention (including systemic, targeted, and preventative therapies) compared with White patients across the sequential models (Table 2, Figure 4A; Table S7). This association was modified by PE severity with the risk of receipt of interventions lower in intermediate severity and higher in high-severity PE hospitalizations (Table 2; Figure 4B and 4C).



**Figure 1. Study cohort.**

\*Patients with pertinent medical history required for pulmonary embolism severity classification (ie, congestive heart failure, chronic pulmonary obstructive disease, and cancer;  $n=1200$ ) and race categorization ( $n=3$ ) were excluded. †Seven hospitals within the healthcare system had  $<100$  admissions for pulmonary embolism throughout the study duration. ‡3.2% ( $n=6$ ) of hospitalizations for Black patients and 14.1% ( $n=158$ ) of hospitalizations for White patients occurred at a hospital with  $<100$  admissions ( $P<0.001$ ). 3.2% ( $n=6$ ) of hospitalizations for Black patients and 2.9% ( $n=32$ ) of hospitalizations for White patients were transferred to an alternative healthcare system ( $P=0.790$ ). And 10.7% ( $n=20$ ) of hospitalizations for Black patients and 9.0% ( $n=101$ ) of hospitalizations for White patients had a repeat admission within  $<30$  days ( $P=0.461$ ). §For the 39 patients were admitted and diagnosed with a pulmonary embolism at a small, nonteaching hospital and transferred ( $<24$  hours between admissions) within the healthcare network and admitted to a large, urban, tertiary-care teaching hospital for definitive care, only the final treating hospital admission was included in the analysis. Admissions may be excluded for more than 1 indication.

Race did not significantly associate with in-hospital mortality (Table 2).

### Sensitivity Analysis

The robustness of our results was apparent in multiple sensitivity analyses. In the matched cohort, the association between race and the primary outcome was consistent throughout alternative definitions of PE severity with an OR of 1.10 (95% CI, 1.02–1.17) after vital sign adjustment and 1.10 (95% CI, 1.02–1.17) after exclusion of comorbidities from the classification scheme.

In the full cohort ( $n=8743$ ), the association between race and the primary outcome was consistent in our multivariate model (Table S8) after multivariable adjustment for age and sex (OR, 1.10; 95% CI,

1.01–1.19), clinical characteristics (OR, 1.12; 95% CI, 1.01–1.25), and clinical characteristics in combination with socioeconomic status (OR, 1.12; 95% CI, 0.98–1.27).

When restricting the full cohort to include only hospitalizations after implementation of a network-wide PE response team service ( $n=7445$ ), race continued to be associated with PE severity on presentation (OR, 1.14; 95% CI, 1.08–1.20).

## DISCUSSION

Existing data demonstrate race-based disparities in the incidence and clinical sequelae associated with PE.<sup>6,7,9,11,12,28</sup> In this large study including over 20 hospitals caring for patients, we observed that Black race

**Table 1. Baseline and Admission Characteristics**

Preoperative variables	Full cohort (n=8743)			Age- and sex-matched cohort (n=4474)		
	White (n=7492)	Black (n=1251)	P Value	White (n=3264)	Black (n=1210)	P Value
Demographics						
Age, y	63.1 (±16.6)	54.6 (±17.8)	<0.001	56.8 (±17.0)	55.1 (±17.5)	0.003
Female sex	3865 (51.6%)	702 (56.1%)	0.003	1769 (54.2%)	672 (55.5%)	0.42
Hispanic ethnicity	36 (0.5%)	4 (0.3%)	0.480	20 (0.7%)	4 (0.4%)	0.29
Area of Deprivation Index	58.0 (±23.5)	80.8 (±21.3)	<0.001	58.8 (±23.5)	80.6 (±21.4)	<0.001
Insurance			<0.001			<0.001
Commercial	2298 (30.7%)	238 (19.0%)		1274 (39.0%)	231 (19.1%)	
Medicaid	849 (11.3%)	414 (33.1%)		504 (15.4%)	395 (32.6%)	
Medicare	4179 (55.8%)	528 (42.2%)		1395 (42.7%)	517 (42.7%)	
Self-pay/other	166 (2.2%)	71 (5.7%)		91 (2.8%)	67 (5.5%)	
Comorbid conditions						
Cerebrovascular event*	574 (7.7%)	115 (9.2%)	0.063	208 (6.4%)	115 (9.5%)	<0.001
Diabetes mellitus	1267 (16.9%)	269 (21.5%)	<0.001	532 (16.3%)	265 (21.9%)	<0.001
Hypertension	3655 (48.8%)	671 (53.6%)	0.001	1389 (42.6%)	659 (54.5%)	<0.001
Heart failure	780 (10.4%)	180 (14.4%)	<0.001	298 (9.1%)	176 (14.5%)	<0.001
Chronic obstructive pulmonary disease	1215 (16.2%)	213 (17.0%)	0.47	476 (14.6%)	209 (17.3%)	0.026
Cancer	2065 (27.6%)	239 (19.1%)	<0.001	814 (24.9%)	236 (19.5%)	<0.001
Coronary artery disease	1105 (14.7%)	110 (8.8%)	<0.001	362 (11.1%)	108 (8.9%)	0.036
End-stage renal disease	55 (0.7%)	22 (1.8%)	<0.001	27 (0.8%)	22 (1.8%)	0.005
Venous thromboembolism	2123 (28.4%)	464 (37.1%)	<0.001	979 (30.0%)	446 (36.9%)	<0.001
Smoking history	40147 (54.4%)	752 (61.0%)	<0.001	1743 (54.2%)	726 (60.9%)	<0.001
Body mass index ≥ 35 kg/m <sup>2</sup>	2302 (30.7%)	471 (37.6%)	<0.001	1136 (34.8%)	454 (37.5%)	0.092
Postoperative period <sup>†</sup>	2079 (27.7%)	313 (25.0%)	0.045	910 (27.9%)	304 (25.1%)	0.066
Medications before hospital admission						
Aspirin	1390 (21.3%)	1279 (16.4%)	0.126	830 (27.0%)	284 (25.0%)	0.19
Anticoagulation <sup>‡</sup>	1700 (26.0%)	1176 (15.0%)	0.275	313 (10.2%)	179 (15.7%)	<0.001
Hospital admission <sup>§</sup>						
Vital signs						
Heart rate, bpm	93.9 (±20.0)	95.6 (±20.5)	0.006	94.8 (±20.1)	95.5 (±20.5)	0.28
Systolic blood pressure, mm Hg	135.5 (±24.6)	137.3 (±25.1)	0.023	134.8 (±23.4)	137.6 (±25.0)	<0.001
Laboratory value						
Troponin-I, ng/mL	0.5 (±8.8)	0.4 (±1.2)	0.72	0.3 (±0.8)	0.3 (±1.1)	0.15
B-type natriuretic peptide, pg/mL	348.9 (±510.8)	360.6 (±616.2)	0.75	277.6 (±465.0)	354.2 (±593.3)	0.044
Creatinine, mg/dL	1.0 (±0.6)	1.2 (±1.2)	<0.001	1.0 (±0.7)	1.2 (±1.2)	<0.001
Hemoglobin, g/dL	12.1 (±2.0)	11.7 (±2.1)	<0.001	12.2 (±2.0)	11.7 (±2.1)	<0.001
International normalized ratio	1.2 (±0.5)	1.3 (±0.6)	0.12	1.2 (±0.6)	1.3 (±0.6)	0.12
Treating hospital characteristics						
Intensive care admission	1961 (26.2%)	322 (25.7%)	0.75	826 (25.3%)	310 (25.6%)	0.83
Vasopressor exposure	270 (3.6%)	52 (4.2%)	0.34	115 (3.5%)	51 (4.2%)	0.28
Relevant consultation	1007 (13.4%)	171 (13.7%)	0.83	539 (16.5%)	167 (13.8%)	0.027
Pulmonology	629 (8.4%)	97 (7.8%)	0.45	356 (10.9%)	96 (7.9%)	0.003
Cardiology	399 (5.3%)	65 (5.2%)	0.85	200 (6.1%)	63 (5.2%)	0.24
Vascular surgery	85 (1.1%)	24 (1.9%)	0.021	41 (1.3%)	23 (1.9%)	0.11

(Continued)

**Table 1. Continued**

Preoperative variables	Full cohort (n=8743)			Age- and sex-matched cohort (n=4474)		
	White (n=7492)	Black (n=1251)	P Value	White (n=3264)	Black (n=1210)	P Value
Cardiothoracic surgery	35 (0.5%)	7 (0.6%)	0.66	539 (16.5%)	167 (13.8%)	0.027
Admission echocardiogram	3930 (52.5%)	595 (47.6%)	0.001	1658 (50.8%)	578 (47.8%)	0.072
Right heart strain	1072 (34.5%)	165 (31.5%)	0.18	443 (33.8%)	156 (30.8%)	0.22
Treating hospital bed size <sup>  </sup>			<0.001			<0.001
Large	5533 (73.9%)	1062 (84.9%)		2444 (74.9%)	1028 (85.0%)	
Medium	1206 (16.1%)	132 (10.6%)		518 (15.9%)	125 (10.3%)	
Small	753 (10.1%)	57 (4.6%)		302 (9.3%)	57 (4.7%)	
Admission year			0.081			0.41
2012	465 (6.2%)	105 (8.4%)		220 (6.7%)	101 (8.3%)	
2013	612 (8.2%)	116 (9.3%)		276 (8.5%)	111 (9.2%)	
2014	895 (11.9%)	144 (11.5%)		406 (12.4%)	138 (11.4%)	
2015	1001 (13.4%)	161 (12.9%)		440 (13.5%)	159 (13.1%)	
2016	1140 (15.2%)	167 (13.3%)		497 (15.2%)	162 (13.4%)	
2017	1181 (15.8%)	200 (16.0%)		500 (15.3%)	194 (16.0%)	
2018	1154 (15.4%)	184 (14.7%)		495 (15.2%)	179 (14.8%)	
2019	1044 (13.9%)	174 (13.9%)		430 (13.2%)	166 (13.7%)	

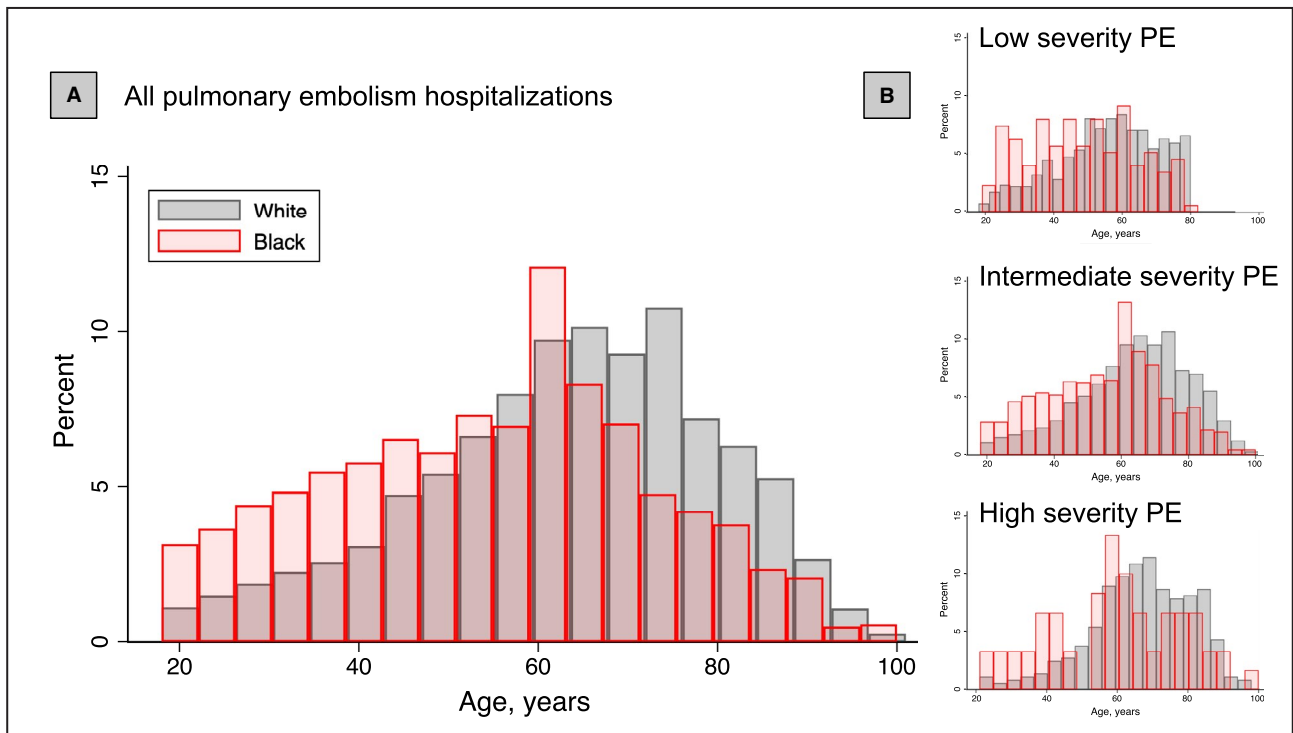
\*Includes a prehospitalization stroke or transient ischemic attack, as defined by *International Classification of Diseases, Clinical Modification of the Ninth or Tenth Revisions*.

<sup>†</sup>Any surgical intervention in the 90 d before pulmonary embolism hospitalization.

<sup>‡</sup>Anticoagulation therapies include the presence of warfarin, dabigatran, rivaroxaban, edoxaban, apixiaban before admission.

<sup>§</sup>Maximal initially recorded vital sign or resulted laboratory value that first resulted upon admission to the transferring or treating hospital.

<sup>||</sup>Hospital bed size is based upon the admission capacity (ie, hospital beds), rural or urban location, and teaching status.<sup>20</sup>



**Figure 2. Age of hospitalization for pulmonary embolism by age, per classification for severity in the full cohort.**

**A**, Overall, Black patients (red) are hospitalized for pulmonary embolism younger than White (gray) patients. **B**, These patterns are consistently observed for hospitalizations for low (top), intermediate (middle), and high (bottom) severity pulmonary embolism. PE indicates pulmonary embolism.

**Table 2. Primary and secondary outcomes**

Primary outcome	Age and sex matched*		Matched and adjusted for clinical characteristics†		Matched and adjusted for clinical and socioeconomic characteristics‡	
	Risk ratio (95% CIs)§	P value	Risk ratio (95% CIs)	P value	Risk ratio (95% CIs)	P value
Pulmonary embolism severity	NA	0.003	1.13 (1.01–1.27)	0.003	1.05 (1.05–1.35)	0.002
Secondary outcome (in-hospital)¶	White (N=3264), No. events (%)	Risk ratio (95% CIs)§	Risk ratio (95% CIs)	P value	Risk ratio (95% CIs)	P value
Any intervention	354 (10.9%)	0.77 (0.66–0.89)	0.73 (0.64–0.84)	<0.001	0.72 (0.63–0.83)	<0.001
Severity subgroup						
Intermediate risk	306 (9.4%)	0.68 (0.57–0.80)	0.65 (0.56–0.75)	<0.001	0.63 (0.54–0.75)	<0.001
High risk	48 (1.5%)	1.51 (1.29–1.77)	1.45 (1.36–1.55)	<0.001	1.45 (1.37–1.55)	<0.001
Systemic therapeutic intervention						
Severity subgroup						
Intermediate risk	28 (1.0%)	0.29 (0.09–1.02)	0.27 (0.09–0.88)	0.030	0.24 (0.13–0.45)	<0.001
High risk	12 (8.5%)	1.14 (0.94–1.38)	1.41 (0.91–2.19)	0.180	1.58 (0.8–3.13)	0.190
Targeted therapeutic interventions*¶						
Severity subgroup						
Intermediate risk	150 (5.6%)	0.52 (0.29–0.93)	0.48 (0.27–0.86)	0.030	0.48 (0.25–0.94)	0.030
High risk	19 (13.4%)	3.03 (2.57–3.56)	3.76 (3.05–4.63)	<0.001	3.51 (2.5–4.94)	<0.001
Preventative intervention						
Severity subgroup						
Intermediate risk	140 (5.2%)	0.81 (0.71–0.92)	0.80 (0.72–0.9)	0.001	0.78 (0.73–0.84)	<0.001
High risk	32 (23.5%)	1.00 (0.63–1.57)	0.82 (0.48–1.39)	0.990	0.8 (0.49–1.32)	0.390
Mortality**						
Severity subgroup						
Intermediate risk	33 (1.2%)	0.65 (0.39–1.08)	0.80 (0.46–1.40)	0.090	0.85 (0.48–1.50)	0.570
High risk	50 (35.5%)	0.81 (0.63–1.03)	0.83 (0.67–1.02)	0.090	0.69 (0.52–0.92)	0.010

\*Matched 1:3 (Black:White) on age and sex without replacement (89% 1:3 pairs; 9% 1:2 pairs; 2% 1:1 pairs). Regression analysis of secondary outcomes include pulmonary embolism severity covariates.

†Adjusted clinical characteristics included in the ordinal regression (severity) and Fine-Gray models (interventions), and logistic regression (in-hospital mortality) include race, age, sex, body mass index  $\geq 35$  mg/kg, recent surgery in the last 90 d, prior venous thromboembolism, and aspirin use.

‡Adjusted clinical and socioeconomic characteristics included in the ordinal regression (severity) and Fine-Gray models (interventions), and logistic regression (in-hospital mortality) are expanded to include the Area of Deprivation Index, and insurance status.

§Ordinal (low-, intermediate, and high-risk pulmonary embolism) logistic regression evaluating the risk of interest, clustered on hospital size (Tables S6 through S7). Risk ratios corresponding to odds ratios for the primary and mortality outcomes, subdistribution hazard ratios for intervention related secondary outcomes. Of note, the reported subdistribution hazard ratios are reported to demonstrate the direction of the effect, their quantification of the magnitude of this effect on the cumulative incidence must be considered an approximation.<sup>27</sup>

¶Interventions as defined by *Current Procedural Terminology* codes (Table S1).

\*\*Only among hospitalizations for intermediate and high-risk pulmonary embolisms

\*\*†The average time to death was 6.42 $\pm$ 7.37 d for Black patients (n=24), and 4.24 $\pm$ 5.91 d for White patients (n=83). This was not different between groups (P=0.150).



was associated with higher severity of PE on presentation and lower risk of receiving catheter-based or surgical intervention including catheter-directed thrombolysis, suction thrombectomy, surgical embolectomy, or inferior vena cava filters. No difference in in-hospital mortality between races was detected.

Although our study was designed to investigate the influence of race on PE severity and mortality, we found interesting patterns related to age and sex. We demonstrate that Black patients present nearly 10 years younger (55 versus 63 years), with women accounting for a higher percentage of Black (56%) compared with White (52%) patients. The observed age differences persisted across severity groups. Differences in age of disease onset among races has been seen previously in PE<sup>12</sup> and is well known in other cardiovascular disease processes such as hypertension, heart failure, peripheral arterial disease, and stroke.<sup>29</sup> Earlier age of disease onset has been shown to increase the likelihood of long-term disability and disease-related dysfunction in other disease processes<sup>30</sup> and is thus likely to be the case with acute PE. The reason for this younger age at PE presentation is unclear. Although the incidence of certain inherited thrombophilias (ie, factor V Leiden and prothrombin gene mutations) has been shown to be lower in people of African descent compared with those of European descent,<sup>31,32</sup> Black patients have been known to have higher levels of a number of hemostatic factors and endothelial markers such as factor VIII, von Willebrand factor, plasmin antiplasmin complex, and D-dimer.<sup>33</sup> The mechanistic differences in thrombophilias among races remains to be investigated; however, varying biologic etiologies may contribute to age differences on presentation. In addition, our findings should heighten the alertness for VTE in younger Black patients and focus resources on preventive measures and more aggressive VTE prophylaxis in this susceptible group.

Our observation that Black patients present with a higher clinical severity PE compared with age- and sex-matched White patients is consistent with data from other clinical events and conditions,<sup>34</sup> and some have suggested this may be related to disparities in access to care.<sup>35</sup> We recognize, however, that difference in the PE severity on presentation is multifactorial and may also be related to a more aggressive disease process with biologic differences between races,<sup>32</sup> hereditary predisposition to hypercoagulability,<sup>36</sup> and underlying conditions such as malignancy.<sup>37</sup> In our cohort, we observed that more White patients had a diagnosis of cancers; however, we have little information regarding genetic mutations, thrombophilias, or family history of PE.

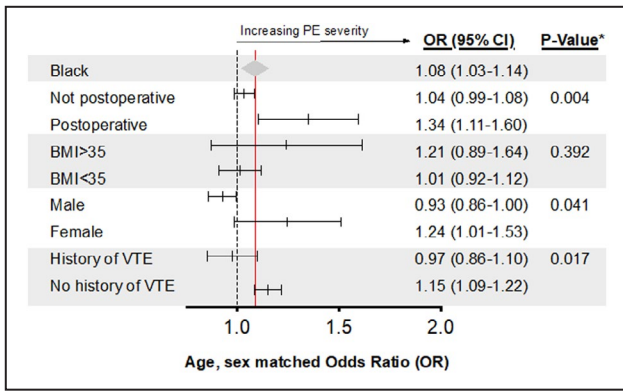
In our analysis, the relative risk increase in presentation severity ranged from 5% to 13% in our sequential adjustment for potential confounders and

was consistent and robust throughout our analysis. Although large measurable racial inequalities are often the target of scientific inquiry, it is likely that the culmination of multiple small differences affect care. These findings highlight the need for targeted interventions to include improved access to care and identification of populations at high risk for VTE.

Our data add to existing evidence that racial disparities exist in cardiovascular health care.<sup>38,39</sup> Across all severity groups, Black patients were less likely to get any intervention compared with age- and sex-matched White patients after controlling for clinical and socioeconomic characteristics. When stratified by severity class and intervention type, Black patients presenting with intermediate severity PE less frequently received a catheter-based or surgical intervention compared with White patients. It is possible that overt and/or implicit bias contributes to differences in intervention. Although explicit racial bias has declined over time, many people still harbor implicit bias and negative attitudes toward Black people,<sup>40</sup> often manifesting in miscommunications and subtle unintentional forms of discrimination. This in addition to overt historical mistreatment of Black patients contributes to racial distrust in the medical community. Therefore, given that the utility of catheter-directed thrombolysis is still controversial in intermediate severity PE, it is possible that Black patients are less likely to consent to a procedure that is still under investigation and is not universally endorsed by the current guidelines.<sup>41,42</sup> It is also possible that there are differences in available resources at different hospitals where Black patients present. However, our analysis took into account variations between hospitals including rurality, teaching status, and size where care was received, and many Black patients were managed at tertiary care facilities that do not experience restrictions in resource allocation or subspecialists that may be encountered at more rural hospitals. Alternatively, as mentioned previously, catheter-directed thrombolysis is still controversial in intermediate severity PE.<sup>21</sup> It is possible that this finding is indicative of unjustified procedures being performed in our control group, and further examination in this area is warranted.

In contrast to what we observed in the intermediate severity group, Black patients with high-severity PE were more likely to receive a surgical or catheter-directed intervention compared with White patients. Although this may be in part due to racial differences in preferences for end-of-life treatment,<sup>43</sup> it may also be due to an exaggerated tendency of physicians to avoid discussing limiting end-of-life care in Black and minority patients.<sup>44</sup>

Despite race-related differences in severity and intervention, we did not observe a difference in mortality. This may have been due, in part, to the low rate (<5%) of in-hospital mortality associated with acute PE.<sup>9,45</sup> Although



**Figure 3. Risk of pulmonary embolism severity risk among subgroups in the matched cohort.**

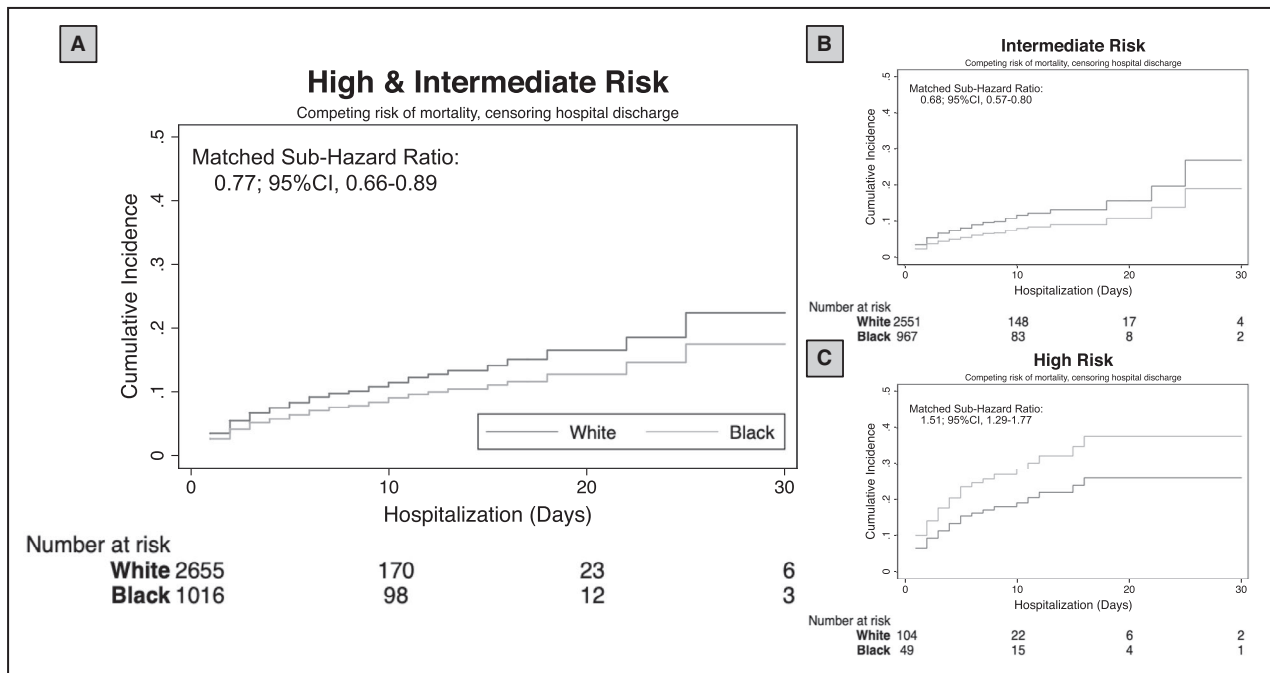
\*P value of the interaction term. The dashed line corresponds to an OR of 1. The red line and the gray triangle corresponding to the overall OR and 95% CI for Black race in the matched cohort. Postoperative within 90 days of admission date. BMI indicates body mass index; OR, odds ratio; and PE, pulmonary embolism.

some retrospective studies have suggested that patients with intermediate severity PE who receive catheter-directed therapy compared with medical therapy alone (systemic anticoagulation or systemic thrombolysis) have a lower 30-day and 1-year mortality<sup>23</sup> and improved ventricular

functional recovery,<sup>46</sup> data from multiple randomized trials have not supported this notion,<sup>47,48</sup> nor has there been evidence that in-hospital mortality is improved. Therefore, thoroughly investigating the association between severity, management, and pertinent clinical outcomes would require analysis of long-term cardiopulmonary function, PE-related disability (ie, chronic thromboembolic pulmonary hypertension), and mortality.

### LIMITATIONS

This study has several limitations. Our database is generated from a large, multihospital institution with a wide catchment area with a racial and ethnic profile similar to other large regional and national databases used in the study of acute PE<sup>49–51</sup> and recently reported national percentages.<sup>52</sup> Although the most recent US census data indicate that 12.5% of the population is Hispanic, less than 1% of our total cohort identified as such.<sup>52</sup> Therefore, the generalizability of our data may be limited in other regions. Next, we observed that more White patients had a baseline diagnosis of cancer and more Black patients were prescribed anticoagulation; however, we have little information regarding genetic mutations, thrombophilias, or family history of



**Figure 4. Cumulative hazard of the risk of in-hospital procedures overall among intermediate and high-severity pulmonary embolisms in the matched cohort together (A) and separately (B and C).**

Cumulative hazard curves demonstrate the risk of any intervention in the combined high- and intermediate severity pulmonary embolisms (A), intermediate severity only (B), and high severity only (C) adjusting for the competing risk of mortality, clustering by hospital size, and censoring for hospital discharge in the matched cohort (risk tables). A, Black (light gray) patients hospitalized with intermediate or high-severity pulmonary embolism have a lower relative risk of undergoing any interventions when compared with White (dark gray) patients. The association between receipt of therapy and race differed between intermediate and high-severity subgroups (P value of interaction, <0.001; B, Risk of intervention for intermediate severity pulmonary embolism. C, Risk of intervention for high-severity pulmonary embolism).

PE. In addition, our data were limited by lack of knowledge of any contraindication to systemic thrombolysis. Our data were also limited by our matching strategy, which left 14.8% (n=179) of Black patients and 9.6% (n=313) of White patients to be matched to less than 1:3 Black to White, thus limiting the variation in White participants for these specific age and sex combinations. And finally, PE severity classification was based on recent guidelines<sup>21</sup> combining preexisting comorbidities and patient characteristics along with laboratory values and diagnostic test results. Although this is an accepted measure of PE classification,<sup>41</sup> our data are limited by absent variables in the data set including arterial oxygenation level. Given this, it was possible that a proportion of our cohort were misclassified on the basis of severity.

## CONCLUSIONS

Despite the limitations, this large database offers enough granularity and clinical details that allow for meaningful observations and potential targets for intervention to reduce racial disparities in the care of patients with PE. We demonstrated that Black patients hospitalized with PE are younger with a higher severity compared with White patients. Although Black patients are less likely to receive an intervention overall, this interestingly differed depending on PE severity with higher risk of intervention only for life-threatening PE. This suggests nuanced racial disparities in being offered or accepting an intervention and highlights the complexities of healthcare inequalities. No difference in the overall low rates of in-hospital mortality was observed between races, and further studies are needed to elucidate racial disparities in mortality and PE-related disability. Investigating the long-term benefit of procedural intervention, regardless of race, and resource allocation to heighten PE prevention in young Black patients is needed and may result in improved outcomes for patients with acute PE and lessen the societal socioeconomic burden of VTE.

## ARTICLE INFORMATION

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### Disclosures

None.

### Supplementary Material

Table S1–S8

Figure S1

## REFERENCES

- National Center for Health Statistics (US). Health, United States, 2018. National Center for Health Statistics (US) 2019.
- Laiteerapong N, Fairchild P, Chou C, Chin M, Huang E. Revisiting disparities in quality of care among US adults with diabetes in the era of individualized care, NHANES 2007–2010. *Med Care*. 2015;53:25–31. DOI: 10.1097/MLR.0000000000000255.
- Casagrande S, Fradkin J, Saydah S, Rust K, Cowie CC. The prevalence of meeting A1C, blood pressure, and LDL goals among people with diabetes, 1988–2010. *Am Diabetes Assoc*. 2013;36:2271–2279. DOI: 10.2337/dc12-2258.
- Fiscella K, Sanders MR. Racial and ethnic disparities in the quality of health care. *Annu Rev Public Health*. 2016;37:375–394. DOI: 10.1146/annurev-publhealth-032315-021439.
- 2018 National Healthcare Quality and Disparities Report. Content last reviewed April 2020. Agency for Healthcare Research and Quality, Rockville, MD. n.d. <https://www.ahrq.gov/research/findings/nhqdr/nhqdr18/Index.html>
- White RH. The epidemiology of venous thromboembolism. *Circulation*. 2003;107:I-4–I-8. DOI: 10.1161/01.CIR.0000078468.11849.66.
- Horlander K, Mannino D, Leeper K. Pulmonary embolism mortality in the United States, 1979–1998: an analysis using multiple-cause mortality data. *Arch Intern Med*. 2003;163:1711–1717. DOI: 10.1001/archinte.163.14.1711.
- Arshad N, Isaksen T, Hansen JB, Brækkan SK. Time trends in incidence rates of venous thromboembolism in a large cohort recruited from the general population. *Eur J Epidemiol*. 2017;32:299–305. DOI: 10.1007/s10654-017-0238-y.
- Virani SS, Alonso A, Benjamin EJ, Bittencourt MS, Callaway CW, Carson AP, Chamberlain A, Chang A, Cheng S, Delling F, et al. Heart disease and stroke statistics—2020 update: a report from the American Heart Association. *Circulation*. 2020;141:e139–e596. DOI: 10.1161/CIR.0000000000000757.
- Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. *Lancet*. 2012;379:1835–1846. DOI: 10.1016/S0140-6736(11)61904-1.
- Barco S, Valerio L, Ageno W, Cohen AT, Goldhaber SZ, Hunt BJ, Iorio A, Jimenez D, Klok FA, Kucher N, et al. Age-sex specific pulmonary embolism-related mortality in the USA and Canada, 2000–18: an analysis of the WHO Mortality Database and of the CDC Multiple Cause of Death database. *Lancet Respir Med*. 2021;9:33–42. DOI: 10.1016/S2213-2600(20)30417-3.
- Martin KA, McCabe ME, Feinglass J, Khan SS. Racial disparities exist across age groups in Illinois for pulmonary embolism hospitalizations. *Arterioscler Thromb Vasc Biol*. 2020;40:2338–2340. DOI: 10.1161/ATVBAHA.120.314573.
- Xu Y, Siegal DM, Anand SS. Ethnoracial variations in venous thrombosis: implications for management, and a call to action. *J Thromb Haemost*. 2020;19:1–11. DOI: 10.1111/jth.15140.
- National Institute on Minority Health and Health Disparities. NIMHD Research Framework. 2017. Available at: <https://nimhd.nih.gov/researchFramework>. Accessed June 7, 2021.
- Laveist T, Gaskin D, Richard P. Estimating the economic burden of racial health inequalities in the United States. *Int J Health Serv*. 2011;41:231–238. DOI: 10.2190/HS.41.2.c.
- Nanney MS, Myers SL, Xu M, Kent K, Durfee T, Allen ML. The economic benefits of reducing racial disparities in health: the case of Minnesota. *Int J Environ Res Public Health*. 2019;16:742. DOI: 10.3390/ijerph16050742.
- van Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The strengthening of reporting of observational studies in epidemiology (STROBE) statement: guidelines for reporting observational studies. *Int J Surg*. 2014;12:1495–1499. DOI: 10.1016/j.ijsu.2014.07.013.

18. Kind AJH, Buckingham WR. Making neighborhood-disadvantage metrics accessible — the neighborhood atlas. *N Engl J Med*. 2018;378:2456–2458. DOI: 10.1056/NEJMp1802313.
19. Reitz KM, Marroquin OC, Zenati MS, Kennedy J, Korytkowski M, Tzeng E, Koscum S, Newhouse D, Garcia R, Vates J, et al. Association between preoperative metformin exposure and postoperative outcomes in adults with type 2 diabetes. *JAMA Surg*. 2020;155:e200416. DOI: 10.1001/jamasurg.2020.0416.
20. Healthcare Cost and Utilization Project (HCUP) NIS Notes. n.d. Available at: [https://www.hcup-us.ahrq.gov/db/vars/hosp\\_bedsiz/nisnote.jsp](https://www.hcup-us.ahrq.gov/db/vars/hosp_bedsiz/nisnote.jsp). Accessed January 1, 2021.
21. Konstantinides SV, Meyer G, Galiè N, Simon R, Gibbs J, Aboyans V, Ageno W, Agewall S, Almeida A, Andreotti F, Barbato E, et al. 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *Eur Heart J*. 2020;41:543–603.
22. Wiske CP, Shen C, Amoroso N, Brosnahan SB, Goldenberg R, Horowitz J, Jamin C, Sista A, Smith D, Maldonado T. Evaluating time to treatment and in-hospital outcomes of pulmonary embolism response teams. *J Vasc Surg*. 2020;8:717–724. DOI: 10.1016/j.jvsv.2019.12.077.
23. D'Auria S, Sezer A, Thoma F, Sharbaugh M, McKibben J, Maholic R, Avgerinos E, Rivera-Lebron B, Toma C. Outcomes of catheter-directed thrombolysis vs. standard medical therapy in patients with acute submassive pulmonary embolism. *Pulm Circ*. 2020;10:1–8. DOI: 10.1177/2045894019898368.
24. Smedley BD, Stith AY, Nelson AR. *Unequal Treatment: Confronting Racial and Ethnic Disparities in Health Care (with CD)*. National Academies Press; 2003.
25. VanderWeele TJ, Robinson WR. On the causal interpretation of race in regressions adjusting for confounding and mediating variables. *Epidemiology*. 2014;25:473–484. DOI: 10.1097/EDE.000000000000105.
26. Chaudhury P, Gadre S, Schneider E, Renapurkar R, Gomes M, Haddadin I, Heresi G, Tong M, Bartholomew J. Impact of multidisciplinary pulmonary embolism response team availability on management and outcomes. *Am J Cardiol*. 2019;124:1465–1469. DOI: 10.1016/j.amjcard.2019.07.043.
27. Austin PC, Fine JP. Practical recommendations for reporting Fine-Gray model analyses for competing risk data. *Stat Med*. 2017;36. DOI: 10.1002/sim.7501.
28. Ibrahim SA, Stone RA, Obrosky S, Sartorius J, Fine MJ, Aujesky D. Racial differences in 30-day mortality for pulmonary embolism. *Am J Public Health*. 2006;96:2161–2164. DOI: 10.2105/AJPH.2005.078618.
29. Carnethon MR, Pu J, Howard G, Albert MA, Anderson CAM, Bertoni AG, Mujahid MS, Palaniappan L, Taylor HA, Willis M, et al. Cardiovascular health in African Americans: a scientific statement from the American Heart Association. *Circulation*. 2017;136:e393–e423. DOI: 10.1161/CIR.0000000000000534.
30. Ellis C. Stroke in young adults. *Disabil Health J*. 2010;3:222–224. DOI: 10.1016/j.dhjo.2010.01.001.
31. Kujovich JL. Factor v Leiden thrombophilia. *Genet Med*. 2011;13:1–6. DOI: 10.1097/GIM.0b013e3181faa0f2
32. Morange PE, Suchon P, Trégouët DA. Genetics of venous thrombosis: update in 2015. *Thromb Haemost*. 2015;114:910–919. DOI: 10.1160/TH15-05-0410.
33. Lutsey PL, Cushman M, Steffen LM, Green D, Barr RG, Herrington D, Ouyang P, Folsom AR, et al. Plasma hemostatic factors and endothelial markers in four racial/ethnic groups: the MESA study. *J Thromb Haemost*. 2006;4:2629–2635. DOI: 10.1111/j.1538-7836.2006.02237.x.
34. Mielck A, Reitmeir P, Wjst M. Severity of childhood asthma by socioeconomic status. *Int J Epidemiol*. 1996;25:388–393. DOI: 10.1093/ije/25.2.388.
35. Duerson W, Lafer M, Ahmed O, Bandler I, Wang B, Lieberman S, Lebowitz R. Health care disparities in patients undergoing endoscopic sinus surgery for chronic rhinosinusitis: differences in disease presentation and access to care. *Ann Otol Rhinol Laryngol*. 2019;128:608–613. DOI: 10.1177/0003489419834947.
36. Zöller B, Li X, Sundquist J, Sundquist K. A nationwide family study of pulmonary embolism: identification of high risk families with increased risk of hospitalized and fatal pulmonary embolism. *Thromb Res*. 2012;130:178–182. DOI: 10.1016/j.thromres.2012.02.002.
37. Tagalakis V, Patenaude V, Kahn SR, Suissa S. Incidence of and mortality from venous thromboembolism in a real-world population: the Q-VTE study cohort. *Am J Med*. 2013;126:832.e13–832.e21. DOI: 10.1016/j.amjmed.2013.02.024.
38. Schwamm LH, Reeves MJ, Pan W, Smith EE, Frankel MR, Olson D, Zhao X, Peterson E, Fonarow G. Race/ethnicity, quality of care, and outcomes in ischemic stroke. *Circulation*. 2010;121:1492–1501. DOI: 10.1161/CIRCULATIONAHA.109.881490.
39. Dong L, Fakeye OA, Graham G, Gaskin DJ. Racial/ethnic disparities in quality of care for cardiovascular disease in ambulatory settings: a review. *Med Care Res Rev*. 2018;75:263–291. DOI: 10.1177/1077558717725884.
40. Dovidio JF, Penner LA, Albrecht TL, Norton WE, Gaertner SL, Shelton JN. Disparities and distrust: the implications of psychological processes for understanding racial disparities in health and health care. *Soc Sci Med*. 2008;67:478–486. DOI: 10.1016/j.socscimed.2008.03.019.
41. Giri J, Sista AK, Weinberg I, Kearon C, Kumbhani DJ, Desai ND, Piazza G, Gladwin M, Chatterjee S, Kobayashi T, et al. Interventional therapies for acute pulmonary embolism: current status and principles for the development of novel evidence. *Circulation*. 2019;140:E774–E801.
42. Kearon C, Akl EA, Ornelas J, Blaivas A, Jimenez D, Bounameaux H, Huisman M, King CS, Morris TA, Sood N, et al. Antithrombotic therapy for VTE disease: CHEST guideline and expert panel report. *Chest*. 2016;149:315–352. DOI: 10.1016/j.chest.2015.11.026.
43. Barnato AE, Anthony DL, Skinner J, Gallagher PM, Fisher ES. Racial and ethnic differences in preferences for end-of-life treatment. *J Gen Intern Med*. 2009;24:695–701. DOI: 10.1007/s11606-009-0952-6.
44. de Souza J, Gillett K, Froggatt K, Walshe C. Perspectives of elders and their adult children of Black and minority ethnic heritage on end-of-life conversations: a meta-ethnography. *Palliat Med*. 2020;34:195–208. DOI: 10.1177/0269216319887070.
45. Agarwal S, Menon V, Jaber WA. Residential zip code influences outcomes following hospitalization for acute pulmonary embolism in the United States. *Vasc Med*. 2015;20:439–446. DOI: 10.1177/1358863X15592486.
46. Avgerinos ED, Abou Ali AN, Liang NL, Rivera-Lebron B, Toma C, Maholic R, Makaroun M, Chaer R. Catheter-directed interventions compared with systemic thrombolysis achieve improved ventricular function recovery at a potentially lower complication rate for acute pulmonary embolism. *J Vasc Surg*. 2018;6:425–432. DOI: 10.1016/j.jvsv.2017.12.058.
47. Konstantinides SV, Vicaut E, Danays T, Becattini C, Bertolotti L, Beyer-Westendorf J, Bouvaist H, Couturaud F, Dellas C, Duerschmied D, et al. Impact of thrombolytic therapy on the long-term outcome of intermediate-risk pulmonary embolism. *J Am Coll Cardiol*. 2017;69:1536–1544. DOI: 10.1016/j.jacc.2016.12.039.
48. Avgerinos ED, Saadeddin Z, Abou Ali AN, Fish L, Toma C, Chaer M, Rivera-Lebron B, Chaer R. A meta-analysis of outcomes of catheter-directed thrombolysis for high- and intermediate-risk pulmonary embolism. *J Vasc Surg*. 2018;6:530–540. DOI: 10.1016/j.jvsv.2018.03.010.
49. The ARIC investigators. The Atherosclerosis Risk in Communities (ARIC) Study: design and objectives. *Am J Epidemiol*. 1989;129:687–702.
50. Fried LP, Borhani NO, Enright P, Furberg CD, Gardin JM, Kronmal RA, Kuller LH, Manolio TA, Mittelmark MB, Newman A, et al. The cardiovascular health study: design and rationale. *Ann Epidemiol*. 1991;1:263–276. DOI: 10.1016/1047-2797(91)90005-W.
51. Agarwal S, Clark D, Sud K, Jaber WA, Cho L, Menon V. Gender disparities in outcomes and resource utilization for acute pulmonary embolism hospitalizations in the United States. *Am J Cardiol*. 2015;116:1270–1276. DOI: 10.1016/j.amjcard.2015.07.048.
52. 2019 US census data. <https://www.census.gov/quickfacts/Fact/Table/US/1PE120219>. n.d.

# **SUPPLEMENTAL MATERIAL**

**Table S1. Current Procedural Terminology codes, descriptions, and categorization for interventions.**

Intervention type	Current Procedural Terminology code	Description
<b>Systemic treatment</b>		
Systemic thrombolysis		Inpatient pharmacy administration record for thrombolytic therapy‡
<b>Local treatment</b>		
Catheter directed intervention (CDI)	36013*†; 36014*†; 36015*†	Introduction of catheter, right heart or main pulmonary artery; left or right pulmonary artery; segmental or subsegmental pulmonary artery
	37212† or 37201*; 37213†; 37214†	Transcatheter therapy, venous infusion for thrombolysis, any method, including radiological supervision and interpretation, initial treatment day; subsequent day during course of thrombolytic therapy; cessation of thrombolysis including removal of catheter and vessel closure by any method
	37187*†; 37188†;	Percutaneous transluminal venous mechanical thrombectomy, including intraprocedural pharmacological thrombolytic injections and fluoroscopic guidance; subsequent day during course of therapy
Surgical embolectomy	33910*†; 33915*†	Pulmonary artery embolectomy with cardiopulmonary bypass; without cardiopulmonary bypass
<b>Preventative</b>		
Inferior vena cava filter placement	37191† or 75940*	Insertion of intravascular vena cava filter, endovascular approach including vascular access, vessel selection, and radiological supervision and interpretation

\* *Current Procedural Terminology* 2010 edition, Category I codes

† *Current Procedural Terminology* 2013, 2015, and 2016 edition, Category I codes

‡ Patients were identified as receiving any pharmacologic dose of any thrombolytic medication (including alteplase, reteplase, and tenecteplase). Charts for patients who were not known to receive a catheter directed intervention (catheter directed thrombolysis) were reviewed for dosing and indication of thrombolytic administration.

**Table S2. Data missingness in the matched cohort.**

<b>Preoperative Variables*</b>	<b>White (n=3264)</b>	<b>Black (n=1210)</b>	<b>P</b>
<b>Demographics, No. (%)</b>			
Area of deprivation index	132 (4.0%)	62 (5.1%)	.120
<b>Comorbid conditions</b>			
Venous thromboembolism	3 (0.1%)	2 (0.2%)	.510
Smoking history	49 (1.5%)	18 (1.5%)	.970
Body mass index $\geq$ 35 kg/m <sup>2</sup>	64 (2.0%)	28 (2.3%)	.460
<b>Medications prior to hospital admission</b>			
Aspirin	187 (5.7%)	72 (6.0%)	.780
Anticoagulation†	187 (5.7%)	72 (6.0%)	.780
<b>Hospital admission ‡</b>			
Laboratory value			
Troponin-I§	1645 (50.4%)	575 (47.5%)	.087
B-type Natriuretic Peptide§	2561 (78.5%)	980 (81.0%)	.064
Creatinine	253 (7.8%)	81 (6.7%)	.230
Hemoglobin	114 (3.5%)	36 (3.0%)	.390
International normalized ratio	1101 (33.7%)	337 (27.9%)	<.001

\*All data not included within the table has no missingness within the matched cohort.

† Anti-coagulation therapies include the presence of warfarin, dabigatran, rivaroxaban, edoxaban, or apixaban prior to admission.

‡ Maximum initially recorded vital sign or resulted laboratory value which first resulted upon admission to the transferring or treating hospital.

§ Missing values for Troponin-I and B-type Natriuretic Peptide were not clinically indicated as determined by the treatment team.

**Table S3. Definitions and clinical adjudication of pulmonary embolism severity on presentation.**

Pulmonary embolism severity	Indicators			
	Hemodynamic instability*	Clinical parameters†	Right heart strain‡	Elevated biomarkers§
<b>High</b>	+	+	+	+
<b>Intermediate</b>	-	<i>At least one +</i>		
		+/-	+/-	+/-
<b>Low</b>	-	-	-	-
Reviewer 2				
Reviewer 1	High	Intermediate	Low	Total
<b>High</b>	15	0	0	15
<b>Intermediate</b>	0	15	0	15
<b>Low</b>	0	0	15	15

\* Cardiac arrest, vasopressor requirement, or systolic blood pressure <90 mm Hg<sup>20-21</sup>

† Simplified pulmonary embolism severity index (sPESI > 0 or intensive care unit admission<sup>22</sup>)

‡ Right ventricular dysfunction on echocardiogram

§ Troponin-I (ng/mL) or B-type natriuretic peptide (pg/mL)

|| On initial review, one patient was found to be mis-classified by our coding algorithm to intermediate severity when clinically they had a low severity PE. This led to recognition and correction of the coding error and the results shown are upon secondary review.

Percent agreement between two reviewers of PE severity on clinical adjudication was 100% in each PE severity.



**Table S4. Baseline and hospitalization characteristics for included and excluded patients after matching.**

Preoperative Variables	White		Black	
	Included (n=3264)	Excluded (n=4228)	Included (n=1210)	Excluded (n=41)
<b>Demographics</b>				
Age, years	56.8 ( $\pm$ 17.0)	68.0 ( $\pm$ 14.5)	55.1 ( $\pm$ 17.5)	38.6 ( $\pm$ 17.9)
Female sex	1769 (54.2%)	2096 (49.6%)	672 (55.5%)	30 (73.2%)
Hispanic ethnicity	20 (0.6%)	16 (0.4%)	4 (0.3%)	0 (0.0%)
Area of deprivation index	58.8 ( $\pm$ 23.5)	57.4 ( $\pm$ 23.5)	80.6 ( $\pm$ 21.4)	86.5 ( $\pm$ 15.2)
<b>Insurance</b>				
Commercial	1274 (39.0%)	1024 (24.2%)	231 (19.1%)	7 (17.1%)
Medicaid	504 (15.4%)	345 (8.2%)	395 (32.6%)	19 (46.3%)
Medicare	1395 (42.7%)	2784 (65.8%)	517 (42.7%)	11 (26.8%)
Self-Pay/ Other	91 (2.8%)	75 (1.8%)	67 (5.5%)	4 (9.8%)
<b>Comorbid conditions</b>				
Cerebrovascular event*	208 (6.4%)	366 (8.7%)	115 (9.5%)	0 (0.0%)
Diabetes mellitus	532 (16.3%)	735 (17.4%)	265 (21.9%)	4 (9.8%)
Hypertension	1389 (42.6%)	2266 (53.6%)	659 (54.5%)	12 (29.3%)
Heart Failure	298 (9.1%)	482 (11.4%)	176 (14.5%)	4 (9.8%)
COPD	476 (14.6%)	739 (17.5%)	209 (17.3%)	4 (9.8%)
Cancer	814 (24.9%)	1314 (29.2%)	236 (19.5%)	6 (8.1%)
End stage renal disease	27 (0.8%)	28 (0.7%)	22 (1.8%)	0 (0.0%)
Venous thromboembolism	979 (30.0%)	1144 (27.1%)	446 (36.9%)	18 (43.9%)
Coronary artery disease	362 (11.1%)	743 (17.6%)	108 (8.9%)	2 (4.9%)
Smoking history	1743 (53.4%)	2274 (53.8%)	726 (60.0%)	26 (63.4%)
Body mass index $\geq$ 35 kg/m <sup>2</sup>	1136 (34.8%)	1166 (27.6%)	454 (37.5%)	17 (41.5%)
Postoperative period†	910 (27.9%)	1169 (27.6%)	304 (25.1%)	9 (22.0%)
<b>Medications prior to hospital admission</b>				
Aspirin	830 (25.4%)	1420 (33.6%)	284 (23.5%)	3 (7.3%)
Anticoagulation‡	313 (9.6%)	445 (10.5%)	179 (14.8%)	4 (9.8%)
<b>Hospital admission §</b>				
<b>Vital signs</b>				
Heart rate, beats per minute	94.8 ( $\pm$ 20.1)	93.2 ( $\pm$ 19.9)	95.5 ( $\pm$ 20.5)	97.4 ( $\pm$ 21.1)
Systolic blood pressure, mm Hg	134.8 ( $\pm$ 23.4)	136.1 ( $\pm$ 25.4)	137.6 ( $\pm$ 25.0)	127.1 ( $\pm$ 26.1)
Laboratory value				

Troponin-I, ng/mL	0.3 (±0.8)	0.6 (±11.5)	0.3 (±1.1)	1.0 (±2.6)
B-type Natriuretic Peptide, pg/mL	277.6 (±465.0)	403.4 (±537.1)	354.2 (±593.3)	570.4 (±1202.3)
Creatinine, mg/dL	1.0 (±0.7)	1.0 (±0.6)	1.2 (±1.2)	0.9 (±0.3)
Hemoglobin, g/dL	12.2 (±2.0)	12.1 (±2.0)	11.7 (±2.1)	11.9 (±1.7)
International normalized ratio	1.2 (±0.6)	1.3 (±0.5)	1.3 (±0.6)	1.2 (±0.3)
<b>Treating hospital characteristics</b>				
Intensive care admission	826 (25.3%)	1135 (26.8%)	310 (25.6%)	12 (29.3%)
Vasopressor exposure	115 (3.5%)	155 (3.7%)	51 (4.2%)	1 (2.4%)
Relevant consultation	539 (16.5%)	468 (11.1%)	167 (13.8%)	4 (9.8%)
Pulmonology	356 (10.9%)	273 (6.5%)	96 (7.9%)	1 (2.4%)
Cardiology	200 (6.1%)	199 (4.7%)	63 (5.2%)	2 (4.9%)
Vascular surgery	41 (1.3%)	44 (1.0%)	23 (1.9%)	1 (2.4%)
Cardiothoracic surgery	18 (0.6%)	17 (0.4%)	7 (0.6%)	0 (0.0%)
Admission echocardiogram	1658 (50.8%)	2272 (53.7%)	578 (47.8%)	17 (41.5%)
Right heart strain	443 (26.7%)	629 (27.7%)	156 (27.0%)	9 (52.9%)
Treating hospital bed size				
Large	2444 (74.9%)	3089 (73.1%)	1028 (85.0%)	34 (82.9%)
Medium	518 (15.9%)	688 (16.3%)	125 (10.3%)	7 (17.1%)
Small	302 (9.3%)	451 (10.7%)	57 (4.7%)	0 (0.0%)
Admission year				
2012	220 (6.7%)	245 (5.8%)	101 (8.3%)	4 (9.8%)
2013	276 (8.5%)	336 (7.9%)	111 (9.2%)	5 (12.2%)
2014	406 (12.4%)	489 (11.6%)	138 (11.4%)	6 (14.6%)
2015	440 (13.5%)	561 (13.3%)	159 (13.1%)	2 (4.9%)
2016	497 (15.2%)	643 (15.2%)	162 (13.4%)	5 (12.2%)
2017	500 (15.3%)	681 (16.1%)	194 (16.0%)	6 (14.6%)
2018	495 (15.2%)	659 (15.6%)	179 (14.8%)	5 (12.2%)
2019	430 (13.2%)	614 (14.5%)	166 (13.7%)	8 (19.5%)

\* Includes a pre-hospitalization stroke or transient ischemic attack, as defined by *International Classification of Diseases – Clinical Management* of the 9<sup>th</sup> or 10<sup>th</sup> editions.

† Any surgical intervention in the 90 days prior to pulmonary embolism hospitalization

‡ Anti-coagulation therapies include the presence of warfarin, dabigatran, rivaroxaban, edoxaban, apixiaban prior to admission.

§ Maximum initially recorded vital sign or resulted laboratory value which first resulted upon admission to the transferring or treating hospital. || Hospital bed size is based upon the admission capacity (i.e., hospital beds), rural or urban location, and teaching status.<sup>19</sup> COPD indicates, chronic obstructive pulmonary disease.

**Table S5. Baseline and hospitalization data for each pulmonary embolism presenting severity category, by race in the matched cohort.**

Preoperative Variables	Low		Intermediate		High	
	White (n=417)	Black (n=146)	White (n=2705)	Black (n=1005)	White (n=142)	Black (n=59)
<b>Demographics</b>						
Age, years	51.1 ( $\pm$ 14.7)	48.5 ( $\pm$ 16.2)	57.5 ( $\pm$ 17.2)	55.9 ( $\pm$ 17.4)	61.8 ( $\pm$ 16.0)	58.7 ( $\pm$ 18.4)
Female sex	195 (46.8%)	61 (41.8%)	1489 (55.0%)	571 (56.8%)	85 (59.9%)	40 (67.8%)
Hispanic ethnicity	4 (1.0%)	0 (0.0%)	15 (0.6%)	4 (0.4%)	1 (0.7%)	0 (0.0%)
Area of deprivation index	60.7 ( $\pm$ 23.3)	77.9 ( $\pm$ 24.6)	58.4 ( $\pm$ 23.5)	81.3 ( $\pm$ 20.7)	60.1 ( $\pm$ 25.4)	76.5 ( $\pm$ 24.0)
<b>Insurance</b>						
Commercial	202 (48.4%)	26 (17.8%)	1035 (38.3%)	190 (18.9%)	37 (26.1%)	15 (25.4%)
Medicaid	80 (19.2%)	58 (39.7%)	403 (14.9%)	323 (32.1%)	21 (14.8%)	14 (23.7%)
Medicare	120 (28.8%)	46 (31.5%)	1194 (44.1%)	442 (44.0%)	81 (57.0%)	29 (49.2%)
Self-Pay/ Other	15 (3.6%)	16 (11.0%)	73 (2.7%)	50 (5.0%)	3 (2.1%)	1 (1.7%)
<b>Comorbid conditions</b>						
Cerebrovascular event*	18 (4.3%)	11 (7.5%)	177 (6.5%)	97 (9.7%)	13 (9.2%)	7 (11.9%)
Diabetes mellitus	43 (10.3%)	22 (15.1%)	458 (16.9%)	230 (22.9%)	31 (21.8%)	13 (22.0%)
Hypertension	145 (34.8%)	57 (39.0%)	1167 (43.1%)	569 (56.6%)	77 (54.2%)	33 (55.9%)
Heart Failure	NA	NA	272 (10.1%)	164 (16.3%)	26 (18.3%)	12 (20.3%)
COPD	NA	NA	451 (16.7%)	197 (19.6%)	25 (17.6%)	12 (20.3%)
Cancer	NA	NA	783 (28.9%)	225 (22.4%)	31 (21.8%)	11 (18.6%)
End stage renal disease	1 (0.2%)	4 (2.7%)	24 (0.9%)	16 (1.6%)	2 (1.4%)	2 (3.4%)
Venous thromboembolism	134 (32.1%)	60 (41.1%)	805 (29.8%)	371 (36.9%)	40 (28.2%)	15 (26.3%)
Coronary artery disease	37 (8.9%)	3 (2.1%)	304 (11.2%)	101 (10.0%)	21 (14.8%)	4 (6.8%)
Smoking history	218 (52.3%)	88 (60.3%)	1463 (54.1%)	608 (60.5%)	62 (43.7%)	30 (52.6%)
Body mass index $\geq$ 35 kg/m <sup>2</sup>	143 (34.3%)	44 (30.1%)	936 (34.6%)	388 (38.6%)	57 (40.1%)	22 (37.3%)
Postoperative period†	84 (20.1%)	25 (17.1%)	779 (28.8%)	254 (25.3%)	47 (33.1%)	25 (42.4%)
<b>Medications prior to hospital admission</b>						
Aspirin	86 (20.6%)	14 (9.6%)	699 (25.8%)	255 (22.4%)	45 (31.7%)	15 (28.8%)
Anticoagulation‡	42 (10.1%)	22 (15.1%)	253 (9.4%)	149 (14.8%)	18 (12.7%)	8 (15.4%)
<b>Hospital admission §</b>						
Vital signs						

Heart rate, beats per minute	85.9 ( $\pm$ 13.4)	85.7 ( $\pm$ 13.7)	95.7 ( $\pm$ 20.3)	96.5 ( $\pm$ 20.4)	102.5 ( $\pm$ 24.7)	103.4 ( $\pm$ 27.5)
Systolic blood pressure, mm Hg	138.5 ( $\pm$ 20.8)	138.7 ( $\pm$ 20.7)	135.4 ( $\pm$ 23.0)	138.7 ( $\pm$ 24.5)	112.9 ( $\pm$ 26.4)	115.5 ( $\pm$ 32.3)
Laboratory value						
Troponin-I, ng/mL	0.0 ( $\pm$ 0.0)	0.1 ( $\pm$ 0.0)	0.3 ( $\pm$ 0.8)	0.3 ( $\pm$ 0.9)	0.9 ( $\pm$ 1.5)	1.4 ( $\pm$ 3.2)
B-type Natriuretic Peptide, pg/mL	34.2 ( $\pm$ 24.7)	33.1 ( $\pm$ 29.4)	294.9 ( $\pm$ 443.0)	360.5 ( $\pm$ 547.8)	585.3 ( $\pm$ 824.8)	695.3 ( $\pm$ 977.7)
Creatinine, mg/dL	0.9 ( $\pm$ 0.3)	1.2 ( $\pm$ 1.5)	1.0 ( $\pm$ 0.7)	1.1 ( $\pm$ 1.2)	1.5 ( $\pm$ 1.0)	1.7 ( $\pm$ 1.2)
Hemoglobin, g/dL	12.8 ( $\pm$ 1.7)	12.3 ( $\pm$ 2.1)	12.1 ( $\pm$ 2.0)	11.7 ( $\pm$ 2.0)	11.6 ( $\pm$ 2.3)	11.4 ( $\pm$ 2.9)
International normalized ratio	1.2 ( $\pm$ 0.2)	1.2 ( $\pm$ 0.2)	1.2 ( $\pm$ 0.4)	1.2 ( $\pm$ 0.4)	2.0 ( $\pm$ 2.0)	1.9 ( $\pm$ 1.6)
<b>Treating hospital characteristics</b>						
Intensive care admission	NA	NA	1783 (65.9%)	826 (82.2%)	118 (83.1%)	52 (88.1%)
Vasopressor exposure	NA	NA	NA	NA	115 (81.0%)	51 (86.4%)
Relevant consultation	86 (20.6%)	18 (12.3%)	1376 (50.9%)	479 (47.7%)	23 (16.2%)	13 (22.0%)
Pulmonology	57 (13.7%)	13 (8.9%)	410 (15.2%)	139 (13.8%)	9 (6.3%)	6 (10.2%)
Cardiology	31 (7.4%)	5 (3.4%)	1376 (50.9%)	479 (47.7%)	13 (9.2%)	6 (10.2%)
Vascular surgery	3 (0.7%)	1 (0.7%)	410 (15.2%)	139 (13.8%)	2 (1.4%)	3 (5.1%)
Cardiothoracic surgery	3 (0.7%)	0 (0.0%)	1376 (50.9%)	479 (47.7%)	6 (4.2%)	0 (0.0%)
Admission echocardiogram	213 (51.1%)	62 (42.5%)	1376 (50.9%)	479 (47.7%)	69 (48.6%)	37 (62.7%)
Right heart strain	NA	NA	410 (29.8%)	139 (29.0%)	33 (47.8%)	17 (45.9%)
Treating hospital bed size						
Large	302 (72.4%)	121 (82.9%)	2026 (74.9%)	855 (85.1%)	116 (81.7%)	52 (88.1%)
Medium	78 (18.7%)	18 (12.3%)	423 (15.6%)	101 (10.0%)	17 (12.0%)	6 (10.2%)
Small	37 (8.9%)	7 (4.8%)	256 (9.5%)	49 (4.9%)	9 (6.3%)	1 (1.7%)
Admission year						
2012	23 (5.5%)	10 (6.8%)	190 (7.0%)	86 (8.6%)	7 (4.9%)	5 (8.5%)
2013	33 (7.9%)	20 (13.7%)	227 (8.4%)	83 (8.3%)	16 (11.3%)	8 (13.6%)
2014	62 (14.9%)	18 (12.3%)	327 (12.1%)	110 (10.9%)	17 (12.0%)	10 (16.9%)
2015	54 (12.9%)	16 (11.0%)	367 (13.6%)	134 (13.3%)	19 (13.4%)	9 (15.3%)
2016	53 (12.7%)	27 (18.5%)	423 (15.6%)	129 (12.8%)	21 (14.8%)	6 (10.2%)
2017	69 (16.5%)	15 (10.3%)	416 (15.4%)	170 (16.9%)	15 (10.6%)	9 (15.3%)
2018	72 (17.3%)	25 (17.1%)	396 (14.6%)	146 (14.5%)	27 (19.0%)	8 (13.6%)
2019	51 (12.2%)	15 (10.3%)	359 (13.3%)	147 (14.6%)	20 (14.1%)	4 (6.8%)

\* Includes a pre-hospitalization stroke or transient ischemic attack, as defined by *International Classification of Diseases – Clinical Management* of the 9<sup>th</sup> or 10<sup>th</sup> editions.

† Any surgical intervention in the 90 days prior to pulmonary embolism hospitalization

‡ Anti-coagulation therapies include the presence of warfarin, dabigatran, rivaroxaban, edoxaban, apixaban prior to admission.

§ Maximal initially recorded vital sign or resulted laboratory value which first resulted upon admission to the transferring or treating hospital.

|| Hospital bed size is based upon the admission capacity (i.e., hospital beds), rural or urban location, and teaching status.<sup>19</sup>

NA indicates, not applicable; COPD, chronic obstructive pulmonary disease.

**Table S6. Multivariable model for primary outcome in the matched cohort.**

Covariates	Matched on age and sex			Matched and adjusted for clinical characteristics			Matched and adjusted for clinical and socioeconomic characteristics		
	OR	95%CI	P	OR	95%CI	P	OR	95%CI	P
Black (White)	1.08	(1.03-1.14)	.003	1.13	(1.01-1.27)	.003	1.05	(1.05-1.35)	.002
Age				1.02	(1.02-1.02)	<.001	1.02	(1.02-1.02)	<.001
Female (Male)				1.45	(1.28-1.63)	<.001	1.22	(1.22-1.63)	<.001
Postoperative 90 days				1.61	(1.40-1.85)	<.001	1.36	(1.36-1.78)	<.001
BMI>35mg/kg <sup>2</sup>				1.22	(1.10-1.36)	<.001	1.07	(1.07-1.30)	.001
Prior venous thromboembolism				0.99	(0.92-1.06)	.715	0.93	(0.93-1.09)	.887
Aspirin				1.18	(1.08-1.30)	<.001	1.12	(1.12-1.25)	<.001
Area of Deprivation Index							0.99	(0.99-1.01)	.252
Insurance (Private)									
Medicaid							0.87	(0.87-1.37)	.446
Medicare							1.07	(1.07-1.31)	.001
Self-pay/other							0.50	(0.50 -1.22)	.275
Constant, cut 1	-1.92	(-2.03- -1.81)		-0.50	(-0.95- -0.04)		-0.37	(-0.58- -0.15)	
Constant, cut 2	3.08	(2.89-3.27)		4.81	(4.30-5.33)		4.91	(4.63-5.19)	

All models are clustered on hospital size, as quantified by the National Inpatient Sample, accounting for bed size, rurality, and teaching status of each hospital.<sup>19</sup> OR indicates, odds ratio; CI, confidence Interval; BMI, body mass index.

**Table S7. Multivariable model for any intervention\* in the matched cohort.**

Covariates	Matched on age and sex			Matched and adjusted for clinical characteristics			Matched and adjusted for clinical and socioeconomic characteristics		
	SHR <sup>†</sup>	95%CI	P	SHR	95%CI	P	SHR	95%CI	P
Black (White)	0.77	(0.66-0.89)	<.001	0.73	(0.64-0.84)	<.001	0.77	(0.60-0.98)	.03
Age				1.00	(0.99-1.01)	.51	1.00	(0.99-1.01)	.61
Female (Male)				0.71	(0.62-0.80)	<.001	0.69	(0.61-0.77)	<.001
Postoperative 90 days				1.19	(1.02-1.38)	.03	1.19	(1.03-1.38)	.02
BMI>35mg/kg <sup>2</sup>				1.26	(1.08-1.47)	.003	1.23	(1.12-1.34)	<.001
Prior venous thromboembolism				1.34	(1.05-1.71)	.02	1.31	(1.00-1.73)	.05
Aspirin				0.86	(0.76-0.97)	.01	0.86	(0.77-0.96)	.008
Area of Deprivation Index							1.00	(0.99-1.01)	.92
Insurance (Private)									
Medicaid							1.05	(0.99-1.12)	.11
Medicare							1.06	(0.85-1.33)	.61
Self-pay/other							0.96	(0.58-1.58)	.87

All models are clustered on hospital size, as quantified by the National Inpatient Sample, accounting for bed size, rurality, and teaching status of each hospital.<sup>19</sup>

SHR indicates, subdistribution hazard ratio; CI, confidence Interval; BMI, body mass index.

\*All procedures including systemic thrombolysis, inferior vena cava filters, surgical embolectomy, and catheter directed therapy (CTD).

†Of note, the reported subdistribution hazard ratios are reported to demonstrate the direction of the effect, their quantification of the magnitude of this effect on the cumulative incidence is only approximately correct.

**Table S8. Multivariable model for primary outcome in the full (non-matched) cohort.**

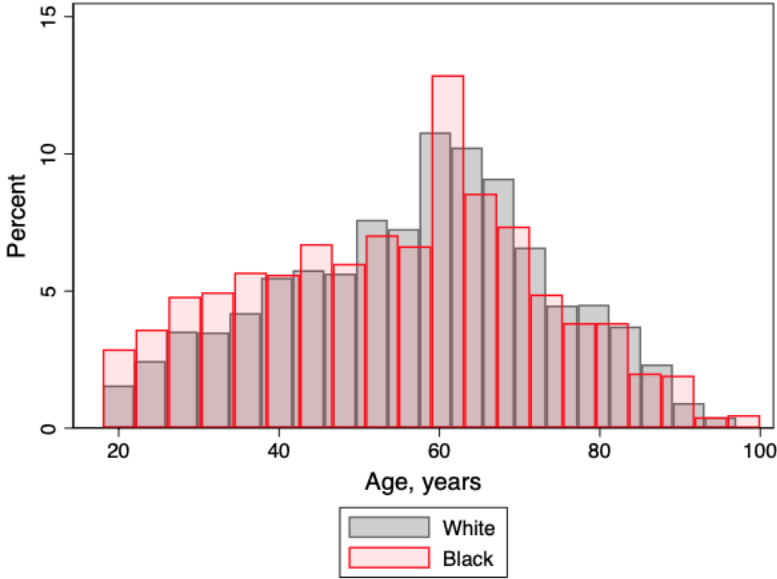
Covariates	Unadjusted		Adjusted for age and sex		Adjusted for clinical characteristics		Adjusted for clinical and socioeconomic characteristics	
	OR	95%CI	OR	95%CI	OR	95%CI	OR	95%CI
Black (White)	0.91	(0.86-0.96)	1.10	(1.01-1.19)	1.12	(1.01-1.25)	1.12	(0.98-1.27)
Age			1.02	(1.02-1.02)	1.02	(1.02-1.02)	1.02	(1.02-1.02)
Female (Male)			1.29	(1.24-1.34)	1.30	(1.24-1.36)	1.27	(1.22-1.32)
Postoperative 90 days					1.59	(1.38-1.83)	1.6	(1.38-1.85)
BMI>35mg/kg <sup>2</sup>					1.12	(1.07-1.17)	1.09	(1.02-1.16)
Prior venous thromboembolism					0.92	(0.82-1.03)	0.95	(0.84-1.08)
Aspirin					1.08	(1.06-1.10)	1.06	(1.00-1.12)
Area of Deprivation Index							1.00	(1.00-1.01)
Insurance (Private)								
Medicaid							1.23	(0.94-1.62)
Medicare							1.16	(1.02-1.32)
Self-pay/other							0.96	(0.65-1.42)
Constant, cut 1	-2.09	(-2.2--1.98)	-0.53	(-0.66--0.39)	-0.43	(-0.6--0.25)	-0.34	(-0.60--0.07)
Constant, cut 2	2.95	(2.73-3.17)	4.67	(4.41-4.94)	4.9	(4.58-5.21)	5.03	(4.62-5.43)

All models are clustered on hospital size, as quantified by the National Inpatient Sample, accounting for bed size, rurality, and teaching status of each hospital.<sup>19</sup>

OR indicates, odds ratio; CI, confidence Interval; BMI, body mass index.



**Figure S1. Distribution of patients by age after matching.**



After matching, Black patients (red) continue to be hospitalized for pulmonary embolism at a younger age than White (gray) patients, although the difference is less pronounced than with the unmatched cohort.