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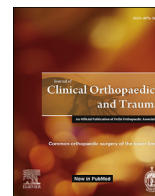
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Racial Disparities in Limb Amputations After Traumatic Vascular Injury



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ABSTRACT

Objectives: The influence of race or ethnicity on limb loss after traumatic vascular injury is unclear. We sought to determine whether there were racial differences in rates of amputation between American Indians, blacks, Asians, and Hispanics compared to white patients following arterial axillosubclavian vessel injury (ASVI), femoral artery injury (FAI), or popliteal artery injury (PAI). As black race has been identified as an independent prognostic factor for postsurgical complication in trauma-associated lower extremity amputation, we further hypothesized that black race would be associated with a higher risk for limb loss after arterial ASVI, FAI, and PAI injury in a large national database.

Methods: The National Trauma Data Bank was queried for patients ≥ 16 -years-old with arterial ASVI, FAI, or PAI to determine the risk of arm, above knee amputation (AKA), and below knee amputation (BKA), respectively. Covariates were included in separate multivariable logistic regression models for analysis. The reference group included white trauma patients.

Results: From 5,683,057 patients, 21,843 were identified with arterial ASVI, FAI, or PAI (<0.4%). For arterial ASVI, American Indian race was associated with higher risk for upper-extremity amputation as compared to white race (OR = 5.10, CI = 1.62–16.06, $p < 0.05$). For FAI, black race was associated with (OR = 0.66, CI = 0.49–0.89, $p < 0.05$) a lower risk of AKA, compared to white race. For PAI, race was not associated with risk for BKA.

Conclusion: Black race is associated with a lower risk of AKA after FAI, compared to whites. Race was not associated with a risk for limb loss after PAI. Future prospective studies examining socioeconomic factors and access to healthcare within this patient population is warranted to identify barriers and areas of improvement.

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1. Introduction

Major extremity amputation following vascular injury is rare. In the upper extremity, radial artery injuries are the most common vascular injury, but rarely lead to amputation.^{1–3} Conversely, axillosubclavian vessel injury (ASVI), though rare, is associated with approximately a 6% rate of amputation.^{1,2} Lower extremity vascular injuries are even more likely to lead to amputation, as common and superficial femoral artery injuries (FAI) carry a 7–13% rate of lower

limb loss.^{1,2} The highest rate of limb loss is seen with popliteal artery injury (PAI) with amputation rates ranging from 9 to 26%.^{1–7} Patients requiring limb amputations after trauma are at increased risk of permanent disability, chronic pain, poor quality of life, anxiety, depression, and lifelong increased healthcare costs.^{8–12}

Recently, the association between race and the risk of limb loss following trauma has received increased attention. Hicks et al. identified a nearly five-fold increased risk of amputation amongst older black patients following vascular trauma as compared to a cohort of white patients.¹³ Additionally, Low et al. identified black race as a significant prognostic factor of postsurgical complications (e.g., infection, thrombosis, acute kidney injury, respiratory failure, and sepsis) following trauma-associated lower extremity amputation.¹⁴ Black patients may be at a higher risk of developing rhabdomyolysis and compartment syndrome, and exhibit lower levels

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Abbreviations

axillosubclavian vessel injury (ASVI)
 femoral artery injury (FAI)
 popliteal artery injury (PAI)
 above knee amputation (AKA)
 below knee amputation (BKA)
 interleukin-6 (IL-6)
 tumor necrosis factor alpha (TNF α)
 interleukin -1 receptor agonist (IL-1RA)
 National Trauma Data Bank (NTDB)
 American College of Surgeons (ACS)
 International Classification of Diseases (ICD)
 Injury Severity Score (ISS)
 Abbreviated Injury Score (AIS)
 odds ratio (OR)
 confidence intervals (CI)
 C-reactive protein (CRP)
 Genetics of Evoked Responses to Niacin and
 Endotoxemia (GENE)
 computed-tomography angiography (CTA)
 chronic obstructive pulmonary disease (COPD)

of systemic inflammatory response (e.g., interleukin-6 (IL-6), tumor necrosis factor alpha (TNF α), and IL-1 receptor agonist (IL-1RA)) after induced endotoxemia as compared to whites, which may contribute to increased susceptibility to reperfusion injury.^{15–18}

It remains unclear if race genomics and related inflammatory responses in trauma are responsible for the differences in outcomes after vascular trauma. Alternatively, race may be a surrogate for socioeconomic factors and poorer access to healthcare which may contribute to delayed care and unidentified or poorly controlled comorbidities after vascular trauma and thus, higher risk for limb loss. We sought to determine whether there were racial differences in rates of amputation between American Indians, blacks, Asians, and Hispanics compared to white patients following arterial ASVI, FAI, or PAI. We further hypothesized that black race would be associated with a higher risk for limb loss after arterial ASVI, FAI, and PAI in a large national database.

2. Methods

This is a retrospective analysis of the National Trauma Data Bank (NTDB), the largest aggregation of trauma registry data in the United States. Assembled by the American College of Surgeons (ACS), the database is updated annually with contributions from over 900 registered trauma centers.¹⁹ The NTDB was queried from January 2007 to December 2015 for patients \geq 16-years-old with arterial ASVI, FAI and PAI, defined by International Classification of Diseases (ICD) version-9 diagnosis codes. The primary outcome was limb loss including upper-extremity, above knee amputations (AKA), and below knee amputations (BKA), defined by ICD-9 procedure codes. American Indian, black, Asian, Hispanic race/ethnicities were compared to a reference group of whites.

Demographic variables collected included age, gender, comorbidities, and insurance status. The injury profile included mechanism (blunt, penetrating, or other), injury severity (Injury Severity Score (ISS) and severe Abbreviated Injury Score (AIS)), and associated vascular, nerve and orthopedic injuries, defined by the appropriate ICD-9 diagnosis codes. All variables were coded as present or absent. Descriptive statistics were performed for all variables. Racial subgroups were compared to a reference group of

white patients. A Mann-Whitney-U test or Student's *t*-test or were used to compare continuous variables and chi-square was used to compare categorical variables for bivariate analysis. Categorical data was reported as percentages and continuous data was reported as medians with interquartile range or as means with standard deviation.

We used separate logistic regression models for each analysis of limb loss (upper-extremity, AKA, and BKA) to identify and control for confounding variables. A univariable logistic regression analysis was performed to identify significant covariates. These were chosen based on a review of the literature and availability in the database.^{13–16} These were then entered into a hierarchical logistic regression model to determine the adjusted-risk for limb loss in patients with arterial ASVI, FAI, and PAI stratified by race. The reference group included whites. The risk of limb amputation was reported with an odds ratio (OR) and a 95% confidence intervals (CI). All P-values were two-sided, with a statistical significance level defined as <0.05 . All statistical analyses were performed with IBM SPSS Statistics for Windows (Version 24, IBM Corp, Armonk, NY).

3. Results

3.1. Demographics for arterial ASVI and rate/risk of upper extremity amputation

The incidence of arterial ASVI was 0.1% and comprised 24.8% of the major vascular injuries studied. Of these, 59.7% were white, 30.2% were Hispanic, 9.5% were black, 0.3% were American Indian, and 0.3% were Asian. Compared to white patients, non-white races were significantly younger and had fewer medical co-morbidities. Penetrating trauma accounted for at least 50% of arterial ASVI in all racial subgroups excluding whites, who experienced significantly higher rates of blunt trauma. Compared to whites, Black (32.0% vs. 19.2%, $p < 0.001$) and Hispanic (33.8% vs. 19.2%, $p < 0.001$) patients had the highest rates of no insurance (Table 1). Upper extremity amputations secondary to traumatic arterial ASVI comprised 15.0% of all amputations, with lower rates of amputation identified in black (1.1%) and Hispanic groups (1.5%) as compared to whites (3.1%, $p < 0.001$) (Table 2). American Indians had a significantly increased risk of upper extremity amputation than whites (OR = 5.10, CI = 1.62–16.06, $p < 0.05$), whereas there was no statistically significant risk for upper extremity amputation amongst blacks, Asians, and Hispanics when compared to whites ($p > 0.05$) (Table 3).

3.2. Demographics for FAI and rate/risk of AKA

The incidence of FAI was 0.2% and accounted for nearly half (43.5%) of the major vascular injuries identified in this study. Of these, 45.8% were white, 41.0% were Hispanic, 12.7% were black, 0.3% were Asian, and 0.2% were American Indian. Compared to white patients, Black patients experienced the highest rates of penetrating trauma (90.7% vs. 45.2%, $p < 0.001$) (Table 4). American Indians had the highest proportion of AKAs compared to whites (15.4% vs. 6.4%, $p < 0.001$) (Table 2). There was a significantly decreased risk of AKA associated with black race (OR = 0.66, CI = 0.49–0.89, $p < 0.05$) (Table 5).

3.3. Basic demographics for PAI and rate/risk of BKA

The incidence of PAI was 0.1% and constituted 31.7% of the major vessel injuries classified in this study. Of these, 58.3% were white, 31.6% were Hispanic, 9.7% were black, 0.3% were Asian, and 0.2% were American Indian. Compared to white patients, Black (32.0% vs. 15.0%, $p < 0.001$) and Hispanic (26.3% vs. 15.0%, $p < 0.001$) patients

Table 1
Basic demographics for arterial ASVI, stratified by race (N = 4595).

Characteristic	White (n = 2745)	American Indian (n = 13)	Black (n = 437)	Asian (n = 12)	Hispanic (n = 1388)	p-value
Age, year, median (IQR)	40 (30)	29 (24)	28 (18)	23.5 (28)	29 (18)	p < 0.001
Male, n (%)	2039 (74.3%) (n = 2677)	9 (75.0%) (n = 12)	383 (87.6%) (n = 422)	11 (91.7%) (n = 10)	1224 (88.2%) (n = 1295)	p < 0.001
ISS, median (IQR)	18 (17)	19.5 (18)	17 (16)	21.5 (22)	17 (16)	0.416
AIS severe, head	312 (11.4%)	1 (7.7%)	37 (8.5%)	1 (8.3%)	89 (6.4%)	p < 0.001
AIS severe, spine	66 (2.4%)	0 (0.0%)	12 (2.7%)	0 (0.0%)	29 (2.1%)	0.853
AIS severe, thorax	561 (20.4%)	6 (46.2%)	92 (21.1%)	0 (0.0%)	226 (16.3%)	p < 0.001
AIS severe, abdomen	112 (4.1%)	0 (0.0%)	10 (2.3%)	0 (0.0%)	42 (3.0%)	0.179
Private pay, n (%)	636 (23.2%)	4 (30.8%)	47 (10.8%)	5 (41.7%)	210 (15.1%)	p < 0.001
No insurance, n (%)	526 (19.2%)	0 (0.0%)	140 (32.0%)	1 (8.3%)	469 (33.8%)	p < 0.001
Medicaid, n (%)	310 (11.6%)	2 (15.4%)	67 (15.3%)	3 (25.0%)	296 (21.3%)	p < 0.001
Medicare, n (%)	341 (12.4%)	0 (0.0%)	19 (4.3%)	0 (0.0%)	52 (3.7%)	p < 0.001
Hypotension	520 (18.9%)	2 (15.4%)	105 (24.0%)	2 (16.7%)	383 (27.6%)	p < 0.001
Comorbidities						
Congestive heart failure	31 (1.1%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	10 (0.7%)	0.346
Chronic renal failure	5 (0.2%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	8 (0.2%)	0.995
Smoker	423 (15.4%)	2 (15.4%)	34 (7.8%)	0 (0.0%)	197 (14.2%)	0.001
Diabetes	158 (5.8%)	0 (0.0%)	18 (4.1%)	0 (0.0%)	42 (3.0%)	0.002
Hypertension	449 (16.4%)	0 (0.0%)	34 (7.8%)	1 (8.3%)	122 (8.8%)	p < 0.001
Peripheral vascular disease	6 (0.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (0.1%)	0.720
COPD	119 (4.3%)	0 (0.0%)	10 (2.3%)	0 (0.0%)	46 (3.3%)	0.150
Mechanism, penetrating	821 (29.9%)	7 (53.8%)	334 (76.4%)	6 (50.0%)	1053 (75.9%)	p < 0.001
Mechanism, blunt	1827 (66.6%)	6 (46.2%)	98 (22.4%)	6 (50.0%)	311 (22.4%)	
Mechanism, other	97 (3.5%)	0 (0.0%)	5 (1.1%)	0 (0.0%)	24 (1.7%)	
Open wound	40 (1.5%)	0 (0.0%)	9 (2.1%)	0 (0.0%)	24 (1.7%)	0.826
Open wound, complicated	41 (1.5%)	0 (0.0%)	4 (0.9%)	0 (0.0%)	13 (0.9%)	0.541
Crush injury	1 (<0.01%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0.954
Axillary nerve injury	20 (0.7%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	10 (0.7%)	0.804
Closed humerus fracture	332 (12.1%)	1 (7.7%)	13 (3.0%)	2 (16.7%)	51 (3.7%)	p < 0.001
Open humerus fracture	154 (5.6%)	0 (0.0%)	19 (4.3%)	1 (8.3%)	76 (5.5%)	0.715
Closed shoulder dislocation	206 (7.5%)	0 (0.0%)	6 (1.4%)	1 (8.3%)	17 (1.2%)	p < 0.001
Open shoulder dislocation	20 (0.7%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	2 (0.1%)	0.127

ASVI = axillosubclavian vessel injury; ISS = Injury Severity Score; AIS = Abbreviated Injury Score; COPD = chronic obstructive pulmonary disease.

Table 2
Amputation rates in trauma patients with associated vascular injury stratified by race/ethnicity.

Amputation type	White	American Indian	Black	Asian	Hispanic	p-value
	(n = 2745)	(n = 13)	(n = 437)	(n = 12)	(n = 1388)	
Upper extremity amputation, n (%)	85 (3.1%)	2 (15.4%)	5 (1.1%)	0 (0.0%)	21 (1.5%)	0.008
	(n = 3686)	(n = 13)	(n = 1020)	(n = 20)	(n = 3298)	
AKA, n (%)	235 (6.4%)	2 (15.4%)	30 (2.9%)	2 (7.7%)	78 (2.4%)	p < 0.001
	(n = 3480)	(n = 9)	(n = 578)	(n = 18)	(n = 1884)	
BKA, n (%)	185 (5.3%)	0 (0.0%)	18 (3.1%)	2 (11.1%)	78 (4.1%)	0.050

AKA = above knee amputation; BKA = below knee amputation.

Table 3
Adjusted^a odds ratio for risk of upper extremity amputation in trauma patients with arterial ASVI stratified by race/ethnicity (compared to reference group of Whites).

Risk factor	OR	CI	p value
American Indian	5.10	1.62–16.06	<0.05
Black	0.72	0.40–1.32	0.29
Asian	0.28	0.03–2.57	0.26
Hispanic or Latino	0.82	0.28–2.38	0.72

ASVI = axillosubclavian vessel injury.

^a controlled for blunt mechanism, concomitant axillary/subclavian vein injury, injury severity score ≥ 25 , brachial plexus injury, compartment syndrome, humerus fracture, shoulder dislocation, diabetes.

had the highest rates of no insurance (Table 6). BKAs with PAI accounted for 38.2% of limb amputations, with the highest rates of limb loss identified in Asians compared to whites (11.1% vs. 5.3%, $p = 0.050$) (Table 2). Blacks with PAI had a similar risk of BKA as compared to whites with PAI (OR = 1.10, CI = 0.81–1.48, $p = 0.55$) (Table 7).

4. Discussion

This analysis of a nationwide trauma databank spanning nine years identified nearly 22,000 patients with arterial ASVI, FAI, and PAI with a 0.4% rate of major amputation including an upper arm amputation rate of 2.5% for arterial ASVI, an AKA rate of 4.3% for FAI, and a BKA rate of 4.7% for PAI. This study found that black race was associated with a significantly decreased risk of AKA after FAI and similar risk of upper extremity amputation following arterial ASVI and BKA following PAI, compared to white patients.

Genomics and/or socioeconomic factors may be related to limb loss after FAI. Contrary to our hypothesis, our study found that black race was associated with a 33% lower risk of AKA following FAI as compared to white patients. These findings corroborate a 2016 study by Hicks et al. that similarly reported a decreased risk of amputation (OR = 0.54, CI = 0.38–0.77) for black patients as compared to whites. However, when stratified by age, older black patients exhibited an increased risk of amputation (OR = 4.21, CI = 1.28–13.6). The authors implicated a higher rate of penetrating

Table 4
Basic demographics for FAI, stratified by race (N = 8043).

Characteristic	White (n = 3686)	American Indian (n = 13)	Black (n = 1020)	Asian (n = 26)	Hispanic (n = 3298)	p-value
Age, year, median (IQR)	35 (27)	30 (35)	25 (13)	31.5 (24)	26 (14)	p < 0.001
Male, n (%)	2975 (80.7%) (n = 3596)	10 (76.9%) (n = 13)	950 (93.1%) (n = 971)	20 (76.9%) (n = 25)	3006 (91.2%) (n = 3085)	p < 0.001
ISS, median (IQR)	16 (12)	18 (22)	16 (9)	16 (11)	16 (8)	p < 0.001
AIS severe, head	241 (6.5%)	2 (15.4%)	28 (2.7%)	2 (7.7%)	83 (2.5%)	p < 0.001
AIS severe, spine	15 (0.4%)	0 (0.0%)	3 (0.3%)	0 (0.0%)	18 (0.5%)	0.815
AIS severe, thorax	176 (4.8%)	4 (30.8%)	16 (1.6%)	0 (0.0%)	76 (2.3%)	p < 0.001
AIS severe, abdomen	124 (3.4%)	0 (0.0%)	33 (3.2%)	2 (7.7%)	95 (2.9%)	0.473
Private pay, n (%)	876 (23.8%)	2 (15.4%)	90 (8.8%)	2 (7.7%)	459 (13.9%)	p < 0.001
No insurance, n (%)	758 (20.6%)	1 (7.7%)	356 (34.9%)	8 (30.8%)	1139 (34.5%)	p < 0.001
Medicaid, n (%)	496 (13.5%)	1 (7.7%)	195 (19.1%)	4 (15.4%)	832 (25.2%)	p < 0.001
Medicare, n (%)	324 (8.8%)	2 (15.4%)	15 (1.5%)	2 (7.7%)	78 (2.4%)	p < 0.001
Hypotension	706 (19.2%)	2 (15.4%)	262 (25.7%)	3 (11.5%)	753 (22.8%)	p < 0.001
Comorbidities						
Congestive heart failure	33 (0.9%)	0 (0.0%)	2.4 (0.1%)	0 (0.0%)	8 (0.2%)	0.001
Chronic renal failure	9 (0.2%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	9 (0.3%)	0.592
Smoker	632 (17.1%)	0 (0.0%)	92 (9.0%)	1 (3.8%)	595 (18.0%)	p < 0.001
Diabetes	182 (4.9%)	0 (0.0%)	10 (1.0%)	1 (3.8%)	83 (2.5%)	p < 0.001
Hypertension	510 (13.8%)	1 (7.7%)	45 (4.4%)	0 (0.0%)	227 (6.9%)	p < 0.001
Peripheral vascular disease	10 (0.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	5 (0.2%)	0.460
COPD	151 (4.1%)	0 (0.0%)	28 (2.7%)	1 (3.8%)	130 (3.9%)	0.336
Mechanism, penetrating	1666 (45.2%)	5 (38.5%)	925 (90.7%)	19 (73.1%)	2839 (86.1%)	p < 0.001
Mechanism, blunt	1862 (50.5%)	8 (61.5%)	81 (7.9%)	6 (23.1%)	408 (12.4%)	
Mechanism, other	158 (4.3%)	0 (0.0%)	14 (1.4%)	1 (3.8%)	51 (1.5%)	
Closed femur fracture	512 (13.9%)	2 (15.4%)	51 (5.0%)	1 (3.8%)	191 (5.8%)	p < 0.001

FAI = femoral artery injury; ISS = Injury Severity Score; AIS = Abbreviated Injury Score; COPD = chronic obstructive pulmonary disease.

Table 5
Adjusted^a odds ratio for risk of AKA in trauma patients with FAI stratified by race/ethnicity (compared to reference group of Whites).

Risk factor	OR	CI	p value
American Indian	1.36	0.52–3.51	0.53
Black	0.66	0.49–0.89	<0.05
Asian	0.28	0.07–1.18	0.08
Hispanic or Latino	0.73	0.40–1.36	0.32

AKA = above knee amputation; FAI = femoral artery injury.

^a controlled for age ≥65, blunt mechanism, femur fracture, pelvic fracture, compartment syndrome, diabetes, peripheral arterial disease, injury severity score ≥25.

injury amongst young black patients as an explanation for this lower amputation risk.¹³ This age-dependent relationship between race and amputation risk is further emphasized by Weber et al. in their study demonstrating young black patients to have a lower probability than young white patients to undergo amputation after traumatic injury, whereas older black patients had a higher probability than their matched white counterparts.²⁰ The black patient population in this study, who tended to be younger with higher rates of penetrating trauma (>90%), supports this trend of decreased risk of subsequent amputation after traumatic vascular injury as compared to white patients. Additionally, these racial differences in amputation rates may be explained by variances in genomics and inflammatory states. C-reactive protein (CRP) is a sensitive systemic marker of inflammation and higher baseline levels have been shown to have a prognostic value in the progression to cardiovascular events in healthy individuals.^{21–23} Epidemiological studies have shown that black race is associated with higher baseline levels of inflammatory markers, CRP and IL-6.^{18,24,25} The Genetics of Evoked Responses to Niacin and Endotoxemia (GENE) study by Ferguson et al. confirms higher baseline values of CRP amongst blacks compared to whites, but also demonstrates comparatively lower median peak values of CRP after induced endotoxemia.¹⁸ Elevated CRP levels following serious trauma is strongly correlated with survival and reflects the inflammatory response to subsequent complications.²⁶ This may

explain why black patients, with theoretically lower peak CRP values, are associated with less posttraumatic complications—specifically amputation—following vascular injury. Although genomic factors may also help explain the differences between black and white patients and risk of AKA after FAI, it is unclear why Hispanic patients with FAI who had similarly high rates of penetrating trauma (86.1%) and lack of insurance to black patients did not experience a reduced risk of AKA as compared to whites. This disparity may possibly be explained by additional cultural and language barriers that Hispanic patients face that complicate access to care in the setting of trauma.^{27,28}

PAI has a high rate of limb loss due to poor collateral circulation around the knee. Prior studies have identified popliteal vessel injuries as one of the most limb threatening peripheral vascular injuries, due to its ligamentous fixation and anatomical orientation to the nearby bony structures.^{29–31} In 1942, Debakey et al. studied the incidence of arterial injury in World War II and showed a 50% rate of amputation with any vascular damage and a staggering 73% amputation rate in association with PAI.³² With the technical advancements of vascular repair in the proceeding Korean and Vietnam Wars, amputation rates fell to 13%.³³ In more recent studies, amputations occurred twice as frequently in PAI/tibial artery injury compared to common/superficial FAI.⁵ We found that AKA secondary to FAI was the most common extremity amputation followed by BKA secondary to PAI. Our study found only a one-tenth higher amputation rate with PAI than FAI. Studies have found that PAI in the setting of blunt trauma, as compared to penetrating trauma, is associated with a significantly increased risk of amputation.^{34–36} Grigorian et al. additionally reported high ISS, compartment syndrome, and concomitant orthopedic injuries to significantly elevate the risk of BKA after PAI.³⁴ Controlling for these known contributors, our analysis found that race is not an independent risk factor for BKA in the setting of PAI. Given the improvement in revascularization techniques and computed-tomography angiography (CTA) imaging coupled with the expedient evaluation and treatment of patients with PAI at skilled trauma centers, race may not play a significant role in determining amputation risk with PAI.^{34,37,38}

Table 6
Basic demographics for PAI, stratified by race (N = 5969).

Characteristic	White (n = 3480)	American Indian (n = 9)	Black (n = 578)	Asian (n = 18)	Hispanic (n = 1884)	p-value
Age, year, median (IQR)	38 (26)	30 (28)	27 (17)	33.5 (16)	29 (19)	p < 0.001
Male, n (%)	2697 (77.5%) (n = 3410)	7 (77.8%) (n = 9)	491 (84.9%) (n = 561)	11 (61.1%) (n = 18)	1597 (84.8%) (n = 1779)	p < 0.001
ISS, median (IQR)	9 (8)	10 (5)	9 (1)	13 (9)	9 (4)	p < 0.001
AIS severe, head	166 (4.8%)	0 (0.0%)	12 (2.1%)	3 (16.7%)	55 (2.9%)	p < 0.001
AIS severe, spine	18 (0.5%)	0 (0.0%)	3 (0.5%)	0 (0.0%)	9 (0.5%)	0.996
AIS severe, thorax	150 (4.3%)	0 (0.0%)	13 (2.2%)	0 (0.0%)	42 (2.2%)	0.001
AIS severe, abdomen	51 (1.5%)	0 (0.0%)	9 (1.6%)	1 (5.6%)	22 (1.2%)	0.502
Private pay, n (%)	946 (27.2%)	2 (22.2%)	55 (9.5%)	5 (27.8%)	329 (17.5%)	p < 0.001
No insurance, n (%)	521 (15.0%)	0 (0.0%)	185 (32.0%)	3 (16.7%)	496 (26.3%)	p < 0.001
Medicaid, n (%)	384 (11.0%)	0 (0.0%)	122 (21.2%)	0 (0.0%)	445 (23.6%)	p < 0.001
Medicare, n (%)	273 (7.8%)	0 (0.0%)	21 (3.6%)	2 (11.1%)	89 (4.7%)	p < 0.001
Hypotension	333 (9.6%)	1 (11.1%)	75 (13.0%)	3 (16.7%)	219 (11.6%)	0.038
Comorbidities						
Congestive heart failure	26 (0.7%)	0 (0.0%)	4 (0.7%)	0 (0.0%)	14 (0.7%)	0.994
Chronic renal failure	9 (0.3%)	0 (0.0%)	1 (0.2%)	0 (0.0%)	4 (0.2%)	0.991
Smoker	633 (18.2%)	3 (33.3%)	55 (9.5%)	1 (5.6%)	446 (23.7%)	p < 0.001
Diabetes	227 (6.5%)	0 (0.0%)	24 (4.2%)	0 (0.0%)	85 (4.5%)	0.009
Hypertension	565 (16.2%)	1 (11.1%)	61 (10.6%)	2 (11.1%)	226 (12.0%)	p < 0.001
Peripheral vascular disease	16 (0.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	4 (0.2%)	0.342
COPD	135 (3.9%)	0 (0.0%)	19 (3.3%)	0 (0.0%)	106 (5.6%)	0.017
Mechanism, penetrating	685 (19.7%)	3 (33.3%)	391 (67.6%)	6 (33.3%)	1129 (59.95%)	p < 0.001
Mechanism, blunt	2644 (76.0%)	6 (66.7%)	176 (30.4%)	12 (66.7%)	702 (37.3%)	
Mechanism, other	151 (4.3%)	0 (0.0%)	11 (1.9%)	0 (0.0%)	53 (2.8%)	
Closed femur fracture	438 (12.6%)	2 (22.2%)	47 (8.1%)	3 (16.7%)	165 (8.8%)	p < 0.001
Open femur fracture	572 (16.4%)	1 (11.1%)	129 (22.3%)	1 (5.6%)	395 (21.0%)	p < 0.001
Closed patella fracture	990 (28.4%)	2 (22.2%)	92 (15.9%)	5 (27.8%)	341 (18.1%)	p < 0.001
Open patella fracture	102 (2.9%)	0 (0.0%)	9 (1.6%)	0 (0.0%)	43 (2.3%)	0.240
Closed tibia/fibula fracture	971 (27.9%)	3 (33.3%)	61 (10.6%)	5 (27.8%)	274 (14.5%)	p < 0.001
Open tibia/fibula fracture	805 (23.1%)	1 (11.1%)	96 (16.6%)	2 (11.1%)	392 (20.8%)	0.003

PAI = popliteal artery injury; ISS = Injury Severity Score; AIS = Abbreviated Injury Score; COPD = chronic obstructive pulmonary disease.

Table 7
Adjusted^a odds ratio for risk of BKA in trauma patients with PAI stratified by race/ethnicity (compared to reference group of Whites).

Risk factor	OR	CI	p value
American Indian	0.69	0.17–2.92	0.62
Black	1.10	0.81–1.48	0.55
Asian	0.96	0.34–2.72	0.95
Hispanic or Latino	0.83	0.45–1.54	0.55

BKA = below knee amputation; PAI = popliteal artery injury.

^a controlled for blunt mechanism, injury severity score ≥ 25 , compartment syndrome, distal femur fracture, proximal tibia/fibula fracture.

American Indian race was associated with a five-fold increased risk of upper extremity amputation with arterial ASVI which likely correlates with the high rate of severe AIS of the thorax (46.2% vs. 20.4%) in this group compared to whites. However, given the small population of American Indians captured, it is difficult to ascertain whether American Indian race is truly an independent prognostic factor for upper extremity limb loss with arterial ASVI. Larger population studies are necessary to elucidate the relationship between American Indian race and upper extremity limb loss with arterial ASVI.

The major limitations of the present study are inherent to a retrospective database study including collection bias and input error. Severity of injury is confined to ISS and AIS measures that do not account for patient physiology (e.g., vital sign instability and progressive neurological decline) that may contribute to rates of amputation. Our focus on three major groups of amputations—upper extremity, AKA, and BKA—does not include minor and more common amputations, including digit loss. This study is limited to index hospitalizations and does not account for secondary amputations in subsequent hospitalizations or in the outpatient setting.

5. Conclusion

Black race is associated with a lower risk of AKA after FAI, compared to whites. Race was not associated with a risk for limb loss after PAI. Future prospective studies examining socioeconomic factors and access to healthcare within this patient population is warranted to identify barriers and areas of improvement.

Conflicts of interest

The authors declare no conflict of interest.

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