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ORIGINAL RESEARCH

Cardiovascular Disease Burden and Major Adverse Cardiac Events in Young Black Patients: A National Analysis of 2 Cohorts 10 Years Apart (2017 Versus 2007)

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BACKGROUND: We aim to compare the burden of cardiovascular disease risk factors and major adverse cardiac events and in-hospital outcomes among young Black patients (aged 18–44 years) hospitalized in 2007 and 2017 using data obtained from the National Inpatient Sample database.

METHOD AND RESULTS: Comparison of the sociodemographic characteristics, comorbidities, and inpatient outcomes, including major adverse cardiac events (all-cause mortality, acute myocardial infarction, cardiogenic shock, cardiac arrest, ventricular fibrillation/flutter, pulmonary embolism, and coronary intervention), between 2017 and 2007 was performed. Multivariable analyses were performed, controlling for potential covariates. A total of 2 922 743 (mean age, 31 years; 70.3% women) admissions among young Black individuals were studied (1 341 068 in 2007 and 1 581 675 in 2017). The 2017 cohort had a younger population (mean, 30 versus 31 years; $P<0.001$), more male patients (30.4% versus 28.8%; $P<0.001$), and patients with higher nonelective admissions (76.8% versus 75%; $P<0.001$), and showed an increasing burden of traditional cardiometabolic comorbidities, congestive heart failure, chronic pulmonary disease, coagulopathy, depression, along with notable reductions in alcohol abuse and drug abuse, compared with the 2007 cohort. The adjusted multivariable analysis showed worsening in-hospital outcomes, including major adverse cardiac events (adjusted odds ratio [aOR], 1.21), acute myocardial infarction (aOR, 1.34), cardiogenic shock (aOR, 3.12), atrial fibrillation/flutter (aOR, 1.34), ventricular fibrillation/flutter (aOR, 1.32), cardiac arrest (aOR, 2.55), pulmonary embolism (aOR, 1.89), and stroke (aOR, 1.53). The 2017 cohort showed a decreased rate of percutaneous coronary intervention/coronary artery bypass grafting and all-cause mortality versus the 2007 cohort ($P<0.001$).

CONCLUSIONS: In conclusion, young Black patients have had an increasing burden of cardiovascular disease risk factors and worsened in-hospital outcomes, including major adverse cardiac events and stroke, in the past decade, although with improved survival odds.

Key Words: all-cause mortality ■ Black individuals ■ cardiovascular disease risk factors ■ disparities ■ major adverse cardiac events

In the United States, cardiovascular disease (CVD) is the leading cause of death among men and women, with Black CVD mortality remaining higher than their racial American counterparts.[1] The disparities in access to health care across the United States have been an ever-present issue in the medical community. This primarily socioeconomic and racial disparity has played a significant role in the prevalence of noncommunicable diseases, especially in these (minority) communities. CVD and its associated risk factors continue to plague

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CLINICAL PERSPECTIVE

What Is New?

- Data on cardiovascular risk factors and acute cardiac events in young Black patients are limited.
- In preliminary analysis, we observed a concerning increase in the odds of major cardiac events.
- There was a 200% increased risk of cardiogenic shock and a 150% increased risk of outside-of-hospital cardiac arrest.

What Are the Clinical Implications?

- Can preventive efforts diagnose any abnormalities in health in the earlier stages?
- Can regular primary care visits curtail adverse cardiac events at a later stage of their lives?
- Will improvements in treatment have better outcomes when comparing 2017 and 2027?

Nonstandard Abbreviations and Acronyms

MACE	major adverse cardiac event
NIS	National Inpatient Sample

the Black community and are a major cause of the disparity in life expectancy.[1] Multiple factors contribute to Black patients' vulnerability to CVD, which includes risk factors (ie, hypertension, diabetes, smoking, and obesity), lifestyle, and other social determinants of health. In addition, when considering the prevalence rates of traditional CVD risk factors, it is imperative to understand the individual and combined effects of those factors. Social determinants of health, which incorporate financial, psychosocial, and environmental factors, are significant contributors to the increased incidence and prevalence of CVD and its morbidity and mortality. Given the association of these factors in a much larger capacity with minorities and other ethnic groups, Powell-Wiley et al have posited a possible connection between these lifestyle/social factors and known biological pathways that result in chronic inflammation and CVD.[2] Access to health care, as 1 of these social determinants of health, varies considerably from primary prevention to tertiary intervention, which continues to hamper efforts to improve the health status of this subgroup. One study by Franks et al stated that the lack of health insurance is directly proportional to the increased risk of mortality.[3] Fowler-Brown et al echoed these sentiments in their article that revealed an association between increased adverse cardiovascular outcomes (cerebrovascular accidents) and death and those who lacked health insurance.[4] This is important as the availability of health insurance has a significant impact on access to health care and its quality. This study aims to compare the burden of CVD risk factors and major adverse cardiac events (MACEs) and in-hospital outcomes among young hospitalized Black patients (aged 18–44 years) selecting 2 nationally representative samples of 2007 and 2017.

METHODS

The data that support the findings of this study are available from the coauthor on reasonable request. Because of privacy reasons and data user agreements with Healthcare Cost and Utilization Project, we would not be able to share raw data; however, we will be available to provide additional analyzed data as per requests received.

The National Inpatient Sample (NIS) database was used to perform the retrospective analysis for the years 2007 and 2017. The Healthcare Cost and Utilization Project, which is supported by the Agency for Healthcare Research and Quality in the United States, includes the NIS as a publicly accessible data set. Weighted NIS data, excluding long-term acute care and rehabilitation facilities, consists of ≈35 million annual in-hospital encounters from >1000 nonfederal acute care hospital centers in 45 states. The NIS data represent nearly >95% of the US population, and they comprise demographics, comorbidities, diagnoses, and procedures, with *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*), or *International Classification of Diseases, Tenth Revision* (*ICD-10*), codes (Table 1). No institutional review board approval was required as these data are deidentified and publicly available.

The study included patients from January 1 to December 31 of 2007 and 2017. According to the latest available data from NIS, we selected this snapshot to observe the changes in a time frame of a decade. Hospitalizations among young Black patients between the ages of 18 and 44 years have been included. The demographic and comorbid risk factors controlled in the multivariable logistic regression analyses included age in years at admission, sex, elective versus nonelective admission, primary expected payer, median household income national quartile for patient zip code, admission day being a weekend, bed size of hospital, location/teaching status of hospital, region of hospital, AIDS, alcohol abuse, deficiency anemias, rheumatoid arthritis/collagen vascular diseases, chronic blood loss anemia, congestive heart failure, chronic pulmonary disease, coagulopathy, depression, hyperlipidemia, smoking, diabetes (uncomplicated and with long-term complications), drug abuse, hypertension, hypothyroidism, liver disease, lymphoma, fluid and electrolyte disorders, metastatic

Table 1. *ICD-9/ICD-10* Codes Used to Identify Outcomes/Complications

Diagnosis	*ICD-9* diagnostic/procedural codes	*ICD-10* diagnostic/procedural codes
Acute myocardial infarction	4100: 41000, 41001 41002; 4101: 41010, 41011, 41012; 4102: 41020, 41021, 41022; 4103: 41030, 41031, 41032; 4104: 41040, 41041, 41042; 4105: 41050, 41051, 41052; 4106: 41060, 41061, 41062; 4107: 41070, 41071, 41072; 4108: 41080, 41081, 41082; 4109: 41090, 41091, 41092	I2101, I2102, I2109, I2111, I2119, I2121, I2129, I213, I214, I219 I21A1, I21A9, I220, I221, I222, I228, and I229
Cardiogenic shock	785.51	R57.0
Atrial fibrillation/flutter	427.31 427.32	I48.0, I48.1, I48.2, and I48.91 I48.3, I48.4, and I48.92
Ventricular fibrillation/flutter	427.42	I49.01 I49.02
Cardiac arrest	427.5	I46.x
Supraventricular tachycardia	427.0	I47.1 I49.2
Pulmonary embolism	415.11, 415.13, and 415.19	I26.02, I26.09, I26.92, I26.93, I26.94, and I26.99
Percutaneous coronary intervention	0066, 1755, 3601, 3602, and 3605	0270346, 027034Z, 0270356, 027035Z, 0270366, 027036Z, 0270376, 027037Z, 02703D6, 02703DZ, 02703E6, 02703EZ, 02703F6, 02703FZ, 02703G6, 02703GZ, 02703T6, 02703TZ, 02703Z6, 02703ZZ, 0270446, 027044Z, 0270456, 027045Z, 0270466, 027046Z, 0270476, 027047Z, 02704D6, 02704DZ, 02704E6, 02704EZ, 02704F6, 02704FZ, 02704G6, 02704GZ, 02704T6, 02704TZ, 02704Z6, 02704ZZ, 0271346, 027134Z, 0271356, 027135Z, 0271366, 027136Z, 0271376, 027137Z, 02713D6, 02713DZ, 02713E6, 02713EZ, 02713F6, 02713FZ, 02713G6, 02713GZ, 02713T6, 02713TZ, 02713Z6, 02713ZZ, 0271446, 027144Z, 0271456, 027145Z, 0271466, 027146Z, 0271476, 027147Z, 02714D6, 02714DZ, 02714E6, 02714EZ, 02714F6, 02714FZ, 02714G6, 02714GZ, 02714T6, 02714TZ, 02714Z6, 02714ZZ, 0272346, 027234Z, 0272356, 027235Z, 0272366, 027236Z, 0272376, 027237Z, 02723D6, 02723DZ, 02723E6, 02723EZ, 02723F6, 02723FZ, 02723G6, 02723GZ, 02723T6, 02723TZ, 02723Z6, 02723ZZ, 0272446, 027244Z, 0272456,027245Z, 0272466, 027246Z, 0272476, 027247Z, 02724D6, 02724DZ, 02724E6, 02724EZ, 02724F6, 02724FZ, 02724G6, 02724GZ, 02724T6, 02724TZ, 02724Z6, 02724ZZ, 0273346, 027334Z, 0273356, 027335Z, 0273366, 027336Z, 0273376, 027337Z, 02733D6, 02733DZ, 02733E6, 02733EZ, 02733F6, 02733FZ, 02733G6, 02733GZ, 02733T6, 02733TZ, 02733Z6, 02733ZZ, 0273446, 027344Z, 0273456, 027345Z, 0273466, 027346Z, 0273476, 027347Z, 02734D6, 02734DZ, 02734E6, 02734EZ, 02734F6, 02734FZ, 02734G6, 02734GZ, 02734T6, 02734TZ, 02734Z6, 02734ZZ, 02C03Z6, 02C03ZZ, 02C04Z6, 02C04ZZ, 02C13Z6, 02C13ZZ, 02C14Z6, 02C14ZZ, 02C23Z6, 02C23ZZ, 02C24Z6, 02C24ZZ, 02C33Z6, 02C33ZZ, 02C34Z6, and 02C34ZZ
Coronary artery bypass grafting	3610, 3611, 3612, 3613, 3614, 3615, 3616, 3617, 3619, 362, 363, 3631, 3632, 3633, 3634, and 3639	0210083, 0210088, 0210089, 021008C, 021008F, 021008W, 0210093, 0210098, 0210099, 021009C, 021009F, 021009W, 02100A3, 02100A8, 02100A9, 02100AC, 02100AF, 02100AW, 02100J3, 02100J8, 02100J9, 02100JC, 02100JF, 02100JW, 02100K3, 02100K8, 02100K9, 02100KC, 02100KF, 02100KW, 02100Z3, 02100Z8, 02100Z9, 02100ZC, 02100ZF, 0210344, 02103D4, 0210444, 0210483, 0210488, 0210489, 021048C, 021048F, 021048W, 0210493, 0210498, 0210499, 021049C, 021049F, 021049W, 02104A3, 02104A8, 02104A9, 02104AC, 02104AF, 02104AW, 02104D4, 02104J3, 02104J8, 02104J9, 02104JC, 02104JF, 02104JW, 02104K3, 02104K8, 02104K9, 02104KC, 02104KF, 02104KW, 02104Z3, 02104Z8, 02104Z9, 02104ZC, 02104ZF, 0211083, 0211088, 0211089, 021108C, 021108F, 021108W, 0211093, 0211098, 0211099, 021109C, 021109F, 021109W, 02110A3, 02110A8, 02110A9, 02110AC, 02110AF, 02110AW, 02110J3, 02110J8, 02110J9, 02110JC, 02110JF, 02110JW, 02110K3, 02110K8, 02110K9, 02110KC, 02110KF, 02110KW, 02110Z3, 02110Z8, 02110Z9, 02110ZC, 02110ZF, 0211344, 02113D4, 0211444, 0211483, 0211488, 0211489, 021148C, 021148F, 021148W, 0211493, 0211498, 0211499, 021149C, 021149F, 021149W, 02114A3, 02114A8, 02114A9, 02114AC, 02114AF, 02114AW, 02114D4, 02114J3, 02114J8, 02114J9, 02114JC, 02114JF, 02114JW, 02114K3, 02114K8, 02114K9, 02114KC, 02114KF, 02114KW, 02114Z3, 02114Z8, 02114Z9, 02114ZC, 02114ZF, 0212083, 0212088, 0212089, 021208C, 021208F, 021208W, 0212093, 0212098, 0212099, 021209C, 021209F, 021209W, 02120A3, 02120A8, 02120A9, 02120AC, 02120AF, 02120AW, 02120J3, 02120J8, 02120J9, 02120JC, 02120JF, 02120JW, 02120K3, 02120K8, 02120K9, 02120KC, 02120KF, 02120KW, 02120Z3, 02120Z8, 02120Z9, 02120ZC, 02120ZF, 0212344, 02123D4, 0212444, 0212483, 0212488, 0212489, 021248C, 021248F, 021248W, 0212493, 0212498, 0212499, 021249C, 021249F, 021249W, 02124A3, 02124A8, 02124A9, 02124AC, 02124AF, 02124AW, 02124D4, 02124J3, 02124J8, 02124J9, 02124JC, 02124JF, 02124JW, 02124K3, 02124K8, 02124K9, 02124KC, 02124KF, 02124KW, 02124Z3, 02124Z8, 02124Z9, 02124ZC, 02124ZF, 0213083, 0213088, 0213089, 021308C, 021308F, 021308W, 0213093, 0213098, 0213099, 021309C, 021309F, 021309W, 02130A3, 02130A8, 02130A9, 02130AC, 02130AF, 02130AW, 02130J3, 02130J8, 02130J9, 02130JC, 02130JF, 02130JW, 02130K3, 02130K8, 02130K9, 02130KC, 02130KF, 02130KW, 02130Z3, 02130Z8, 02130Z9, 02130ZC, 02130ZF, 0213344, 02133D4, 0213444, 0213483, 0213488, 0213489, 021348C, 021348F, 021348W, 0213493, 0213498, 0213499, 021349C, 021349F, 021349W, 02134A3, 02134A8, 02134A9, 02134AC, 02134AF, 02134AW, 02134D4, 02134J3, 02134J8, 02134J9, 02134JC, 02134JF, 02134JW, 02134K3, 02134K8, 02134K9, 02134KC, 02134KF, 02134KW, 02134Z3, 02134Z8, 02134Z9, 02134ZC, and 02134ZF

The Elixhauser Comorbidity Indexes and predefined comorbidities in available databases were used to identify cardiovascular and extracardiac comorbidities. *ICD-9* indicates *International Classification of Diseases, Ninth Revision*; and *ICD-10*, *International Classification of Diseases, Tenth Revision*.

cancer, other neurologic disorders, obesity, peripheral vascular disorders, pulmonary circulation disorders, renal failure, solid tumor without metastasis, valvular disease, and personal history of sudden cardiac arrest; and this list is included in footnotes. MACE was defined as all-cause mortality, acute myocardial infarction and/or coronary intervention (percutaneous coronary intervention/coronary artery bypass grafting), cardiogenic shock, cardiac arrest, ventricular fibrillation/flutter, and pulmonary embolism. We compared sociodemographic characteristics, comorbidities, and inpatient outcomes for MACEs, including coronary intervention, from the 2017 cohort versus 2007 cohort or can use group (Table 2).

Table 2. Baseline Demographics, Comorbidities, and In-Hospital Outcomes of Young (Aged 18–44 Years) Black Patients Hospitalized in 2007 Versus 2017

Variables	2007 (n=1 341 068)	2017 (n=1 581 675)	Total (n=2 922 743)	*P* value*
Age at admission, median (IQR), y	31 (24–38)	30 (25–37)	31 (24–37)	<0.001
Sex				<0.001
Men	28.8	30.4	29.7	
Women	71.2	69.6	70.3	
Nonelective admissions	75.00	76.8	76.0	<0.001
Primary expected payer				<0.001
Medicare	9.7	10.2	10.0	
Medicaid	40.6	50.7	46.0	
Private, including HMO	31.1	27.2	28.9	
Self-pay/no charges/others	12.0	8.2	10.0	
Location/teaching status of hospital				<0.001
Rural	6.7	4.9	5.7	
Urban nonteaching	32.6	16.1	23.6	
Urban teaching	60.7	79.1	70.7	
Comorbidities				
Alcohol abuse	4.1	3.8	3.9	<0.001
Rheumatoid arthritis/collagen vascular diseases	1.4	1.8	1.6	<0.001
Chronic blood loss anemia	6.3	9.3	8.0	<0.001
Congestive heart failure	1.8	2.8	2.4	<0.001
Chronic pulmonary disease	9.1	12.3	10.9	<0.001
Coagulopathy	1.7	3.2	2.5	<0.001
Depression	4.0	6.8	5.5	<0.001
Hypertension (secondary/complicated)	19.6	21.1	20.4	<0.001
Diabetes, uncomplicated	6.5	3.9	5.1	<0.001
Diabetes with chronic complications	1.7	5.4	3.7	<0.001
Hyperlipidemia	4.3	5.5	5.0	<0.001
Smoking	13.3	27.4	21.0	<0.001
Drug abuse	9.5	9.1	9.3	<0.001
Hypothyroidism	1.3	1.8	1.6	<0.001
Liver disease	1.0	1.7	1.4	<0.001
Fluid and electrolyte disorders	11.4	16.3	14.0	<0.001
Other neurologic disorders	3.3	4.5	4.0	<0.001
Obesity	8.1	16.6	12.7	<0.001
Peripheral vascular diseases	0.5	0.7	0.6	<0.001
Pulmonary circulation disorders	0.6	0.5	0.5	<0.001
Chronic kidney disease	4.7	6.0	5.4	<0.001
Valvular heart disease	0.9	0.8	0.8	<0.001

Data are given as percentage unless otherwise indicated. HMO indicates health maintenance organization; and IQR, interquartile range.
*$P<0.05$ indicates statistical significance.

The Pearson χ^2 test and Mann-Whitney *U* test were used to equate the categorical and continuous variables between the 2 cohorts after excluding missing data. Significant variables in univariate analysis were incorporated into multivariate analyses to control confounders while assessing the risk of outcomes in the 2017 cohort compared with the 2007 cohort using an adjusted odds ratio (aOR) with a 95% CI. *P*<0.05 was considered statistically significant. Weighted data and complex sample modules in SPSS v.25 (IBM Corp, Armonk, NY) were used for statistical analysis. In our study, the analysis incorporated self-weighted NIS data using the predefined variable "DISCWT" (discharge weights). In our study, the analysis involved the use of NIS discharge weights, which were determined by the ratio of the number of sampled discharges/the number of universe discharges within each NIS stratum. The estimation of universe discharges was historically based on data obtained from the American Heart Association annual hospital survey, where the total number of discharges in the universe was calculated by summing the births and admissions reported in the American Heart Association annual survey across all hospitals in the universe.

RESULTS

A total of 2 922 743 admissions among young Black patients between the ages of 18 and 44 years (median age, 31 years; 70.3% women; 29.7% men; *P*<0.001) were studied (1 341 068 in the 2007 cohort and 1 581 675 in the 2017 cohort; Table 2). Patients in the 2017 cohort were often younger (age, 30 versus 31 years), were more often men (30.4% versus 28.8%; *P*<0.001), and had higher nonelective admission rates (76.8% versus 75%; *P*<0.001). The 2017 cohort also had a higher burden of traditional cardiometabolic comorbidities, such as hypertension (secondary/complicated), diabetes with chronic complications, hyperlipidemia, smoking, congestive heart failure, chronic pulmonary disease, coagulopathy, and depression (*P*<0.001). In addition, patients in the 2017 cohort had reduced rates of alcohol abuse (4.1% verus 3.8%; *P*<0.001) and drug abuse (9.1% versus 9.5%; *P*<0.001) compared with the 2007 cohort (Table 2).

Adjusted multivariable analysis revealed worsening in-hospital outcomes and MACEs in the 2017 cohort, with an aOR of 1.21 (*P*<0.001). These included acute myocardial infarctions (aOR, 1.34; *P*<0.001), cardiogenic shock (aOR, 3.12; *P*<0.001), atrial fibrillation/flutter (aOR, 1.34; *P*<0.001), ventricular fibrillation/flutter (aOR, 1.32; *P*<0.001), cardiac arrest (aOR, 2.55; *P*<0.001), pulmonary embolism (aOR, 1.89; *P*<0.001), and stroke (aOR 1.53, *P*<0.001). A notable finding was a decreased rate of percutaneous coronary intervention (aOR, 0.86; *P*<0.001), coronary artery bypass grafting (aOR, 0.25; *P*<0.001), and all-cause mortality (0.5% versus 0.6%; *P*<0.001) in the 2017 cohort compared with the 2007 cohort (Table 3).

DISCUSSION

As the leading cause of death among men and women, CVD mortality in the Black community remains higher than in their racial American counterparts.[1,5] The increased incidence of sedentary lifestyles (increased physical inactivity, coupled with high-risk lifestyle choices of smoking, alcoholism, and obesity) in young Black individuals (teenagers and adults) is a major contributor to the increased CVD burden.[6] There are ≈47 million Americans who have metabolic syndrome, a condition marked by obesity, hypertension, dyslipidemia, and insulin resistance that causes diabetes.[3] Numerous risk factors within the constellation of metabolic syndrome either individually or in combination were found to be major contributors to the CVD burden in young adults aged 18 to 44 years, albeit at a lower prevalence in Black individuals. Although the incidence of metabolic syndrome is lower in the Black population in comparison with other major racial groups in the United States,[6,7] Black individuals have a higher prevalence of obesity or adiposity and hypertension, which explains more CVD burden in this group. These untreated risk factors coupled with subpar primary prevention methods (later diagnosis and management) are major contributors to the CVD burden.[8,9]

Of the total 2 922 743 admissions among young Black patients reviewed in the study (1 341 068 in the 2007 cohort and 1 581 675 in the 2017 cohort), there was an increasing burden of traditional cardiometabolic comorbidities, congestive heart failure, chronic pulmonary disease, coagulopathy, and depression. Subclinical risk factors for CVD, such as obesity and hypertension, wreak havoc on the cardiovascular system and were among the highest numbers of comorbidities in the 2007 and 2017 cohorts. The prevalence of obesity was 8.1% in the 2007 cohort versus 16.6% in the 2017 cohort, and the prevalence of hypertension was 19.6% in the 2007 cohort versus 21.1% in the 2017 cohort. These risk factors with high vascular impact (obesity, diabetes, and hypertension) may have led to subclinical changes in vascular function and structure, which lead to vascular dysfunction and clinical vascular diseases. These changes are compounded with tissue injury/wall stress, resulting in pathologic remodeling and progression to target organ dysfunction or end-stage heart failure, and ultimately death. Gibbons elucidates the process by which this vascular dysfunction is propagated.[10] The usual suspects or mediators, norepinephrine, angiotensin II, aldosterone, endothelin, tumor necrosis factor, and interleukin-6, are causative factors in the cause of CVD. These mediators that play

Table 3. In-Hospital Outcomes of Young (Aged 18–44 Years) Black Patients Hospitalized in 2007 Versus 2017

In-hospital outcomes	2007 (n=1,341,068)	2017 (n=1,581,675)	Total (n=2,922,743)	*P**	aOR†	95% CI	Adjusted *P* value*
All-cause mortality	0.6	0.5	0.6	<0.001			
MACEs	1.9	2.4	2.2	<0.001	1.21	1.19–1.23	<0.001
Acute myocardial infarction	0.5	0.7	0.6	<0.001	1.34	1.26–1.35	<0.001
Cardiogenic shock	0.1	0.2	0.1	<0.001	3.12	2.83–3.44	<0.001
Percutaneous coronary intervention	0.3	0.3	0.3	0.641	0.86	0.82–0.91	<0.001
Coronary artery bypass grafting	0.1	0.0	0.1	<0.001	0.25	0.23–0.29	<0.001
Atrial fibrillation/flutter	0.8	1.2	1.0	<0.001	1.34	1.30–1.37	<0.001
Ventricular fibrillation/flutter	0.1	0.1	0.1	<0.001	1.32	1.20–1.44	<0.001
Out-of-hospital cardiac arrest	0.0	0.1	0.0	<0.001	2.55	2.15–3.02	<0.001
Pulmonary embolism	0.6	0.9	0.8	<0.001	1.89	1.82–1.96	<0.001
Stroke	0.6	0.9	0.8	<0.001	1.53	1.49–1.58	<0.001
Disposition of patient							
Routine	88.2	87.3	87.7	<0.001			
Transfers to short-term hospitals	1.1	1.1	1.1				
Other transfers, including SNF and ICF	3.7	3.3	3.5				
Home health care	3.7	4.7	4.2				
Against medical advice	2.5	3.0	2.8				
Length of stay, median (IQR), d	3 (2–4)	3 (2–4)	3 (2–4)	<0.001			
Total charges, median, USD	12285	21298	16742	<0.001			

Data are given as percentage unless otherwise indicated. MACE was defined as all-cause mortality, acute myocardial infarction, cardiogenic shock, cardiac arrest, ventricular fibrillation/flutter, pulmonary embolism, and coronary intervention (percutaneous coronary intervention/coronary artery bypass grafting). aOR indicates adjusted odds ratio; ICF, intermediate-care facility; IQR, interquartile range; MACE, major adverse cardiac event; and SNF, skilled nursing facility.

**P*<0.05 indicates statistical significance.

†Multivariable analyses were adjusted for baseline sociodemographic variables, hospital-level characteristics, and preexisting cardiovascular and extracardiac comorbidities.

a major role in CVD development are further worsened by an individual's genetic makeup. Genetics has a major influence and impact not only on the relative ease of incidence of the risk factors of CVD and its presentation, but also on response to treatment and prognosis, and in this case hospital outcomes.[11]

The increasing incidence of CVD in the United States, particularly in young individuals, is attributable to many factors, but the highest comorbidities are obesity and tobacco use/smoking. As demonstrated in our study, obesity and smoking have a high comorbidity rate. An exponential increase in smoking rates has been identified in the 2017 cohort (27.4% versus 13.3% in 2007). There is a vast body of evidence showing a directly proportional relationship between obesity and left ventricular hypertrophy and left ventricular dilation, which are both recognized precursors of heart failure.[12,13] Obesity is also a significant risk factor for hypertension, diabetes, and dyslipidemia: conditions that all increase the risk of heart failure. The risk of CVD is not exclusive to those who are extremely obese. In fact, it is also associated with those who are moderately obese. As a precursor to the development of other cardiovascular risk factors, obesity as a standalone variable is linked to 11% of cases of heart failure in men and 14% of cases of heart failure in women.[13,14] Kenchaiah et al demonstrated that each 1-unit increment in body mass index increases the risk of heart failure in men and women by 5% and 7%, respectively. They also determined that this risk doubled in patients with obesity versus their "normal body mass index" counterparts.[14]

It is known that CVD disproportionately impacts Black individuals. As aforementioned, not only are significant risk factors, such as obesity and hypertension, more prevalent but also mortality associated with CVD is significantly higher compared with White counterparts.[15,16] According to the study analysis, all-cause mortality decreased by 0.1% in the 2017 cohort compared with the 2007 cohort. Two opposing mediators of the cardiovascular system, angiotensin II and peroxisome proliferator-activated receptor γ, regulate the risks of obesity, cardiovascular dysfunction, and diabetes. These mediators are antagonistic in nature and function. Angiotensin II is proinflammatory, growth stimulating, profibrotic, and proatherogenic; and it reduces endothelial function, increases blood pressure, and decreases peroxisome proliferator-activated receptor γ. Alternatively, peroxisome proliferator-activated receptor γ is anti-inflammatory, growth inhibiting, antifibrotic, and antiatherogenic. It lowers blood pressure, improves endothelial function, and decreases the

function of angiotensin type 1 receptor.[1,11,17] Genotypic variants or correlations affect the activity of the aforementioned factors, and their expression and frequency of expression differ among the races. Carnethon et al examined the significant disparities in the age of onset of CVD and its risk factors in Black individuals, especially in young adults, compared with White Americans. Their findings touched on a genetic component in addition to environmental influences on the incidence of CVD. Cardiovascular biomarkers were compared against certain gene expressions/loci for inflammation, thrombosis, vascular structure, arrhythmia risk hypertension, and lipid disorders. CRP (C-reactive protein), a heritable biomarker of systemic inflammation and a reasonable prognosticator of CVD in the general population, was elevated in Black populations and in those with *APOE* ε2 rs7214, among others.[1,11] Another marker of note was plasma fibrinogen levels, which were associated with an increased risk of CVD. As a major element in thrombus formation, the heritability of plasma fibrinogen was also a predictor of CVD, with predictability as high as 50%. Fibrinogen levels were noticeably higher in individuals of African descent compared with other ethnic groups.[1] This genetic component between individuals of African and European descent may explain racial/ethnic differences in CVD susceptibility and response to treatment in these groups.

The adjusted multivariable analysis in our study revealed worsening in-hospital outcomes for young Black individuals in the past decade. These outcomes included MACEs, acute myocardial infarctions, cardiogenic shock, atrial fibrillation and flutter, ventricular fibrillation and flutter, cardiac arrest, pulmonary embolism, and stroke. These outcomes were statistically significant, with $P<0.0001$ and aORs >1.0 conclusively. The increasing burden, not only of disease but of these negative outcomes, after 10 years in this 18- to 44-year-old Black demographic is significant. However, there has been a significantly decreased rate of percutaneous coronary intervention/coronary artery bypass grafting and all-cause mortality in the 2017 cohort versus the 2007 cohort ($P<0.001$; Table 3). The negative outcomes in the Black demographic have not improved but worsened, which is concerning. This can in part be attributed to the increase in nonelective admissions because of increased primary care and prevention initiatives, but the increased comorbidities/risk factors cited speak to an area of focus that needs to be addressed.

Improved mortality despite worsening all cardiovascular risks and MACEs may be attributable to better access to health care, more centers, and more coverage. Also, early diagnosis, early hospitalization, and timely management indicate better quality of care in the current era (2017) compared with a decade prior (2007).[18,19] Possible factors responsible for the increased comorbidities, risk factors, and cardiovascular morbidities can potentially be attributed to the disparity in the social determinants of health in this population. Minorities and specifically Black individuals, as the focus group, have relatively less frequent annual wellness visits, absent/insufficient screening measures at a younger age, genetics, stress, inappropriate and/or unhealthy eating habits, lack of awareness or lack of insight into a healthy lifestyle, or financial constraints.[1,2] In addition, access to health care is usually impaired at the primary and tertiary levels because of several factors. These include, but are not limited to, economic factors (financial constraints), with 26% of Black individuals living in poverty relative to 15% of the overall population, access to comprehensive and affordable insurance coverage, as well as stress.[18] This stress is a product of economic factors to some degree, whereby lifestyle (job and neighborhood security) is further compounded by perceived racial discrimination that culminates in a myriad of social risk factors that drive their baseline genetic predisposition of CVD higher.[19] More emphasis could have been given to regular primary care visits, annual wellness care visits in young Black adults, and diagnosing any abnormalities in health in the earlier stages.[20,21]

Sims et al offered a possible solution to this issue by way of promoting positive psychological well-being, which may promote optimal cardiovascular health among Black individuals, thereby potentially reducing racial and ethnic disparities in CVD.[20] In their study, Black individuals who were optimistic were more likely to engage in physical activity, were more likely to make healthier dietary choices, and had lower smoking rates.[20,21] As research indicates and previously stated, Black individuals are at an increased risk for physical inactivity, obesity, and the negative effects of smoking relative to other racial and ethnic groups.[21,22] As such, tailored interventions incorporating the previously mentioned observations/findings would offer greater benefits to improve both positive well-being and risk factors for CVD. These interventions previously stated among others were highlighted by Crook et al and can be further facilitated by approaches through governmental legislative change.[23] Income and health care disparities and the requisite health care reforms have to be put in place to address these disparities. Complementing these efforts would be facilitation of easier access to these health care resources and improvement of community awareness and initiatives that can bridge the gap in knowledge of health and its associated risk factors in this vulnerable group.[5,23] Taylor et al proposed a novel approach in their article in addressing and possibly eliminating these CVD health disparities through community partnerships. These partnerships among the communities, academic and vocational institutions, and industry are a viable method of increasing awareness and aiding in the primary prevention of these CVD risk factors and diseases.[5]

There are several limitations to our study: it is a cross-sectional study without data on follow-up, medication history, or laboratory findings. NIS data do not provide information on outpatient and postdischarge follow-up. We compared the variables that are a decade apart. We could not include data on the burden and impact of electronic cigarettes or vaping and recreational cannabis use on cardiovascular health. Future researchers could include the cross-sectional study with follow-up data and matched studies to reduce the selection bias; they should study the impact of substance abuse, sedentary lifestyle, and uncontrolled cardiac risk factors on cardiovascular outcomes of young Black adults, including all age groups starting from teenagers, especially in these times of pandemic, where more recent studies report the worse impact of a pandemic on behavioral risk factors and effects on CVD.

CONCLUSIONS

To conclude, young Black patients have an increased burden of cardiovascular risk factors, like hypertension, diabetes with chronic complications, kidney disease, heart failure, pulmonary disease, and depression. In-hospital outcomes have also worsened, including MACEs, stroke, and cardiogenic shock; however, all-cause mortality did improve. More research is needed to evaluate, manage, and prevent the factors that can lead to increased cardiovascular risks, mainly smoking, depression, and obesity, to ultimately curtail adverse cardiac events in this population at a later stage of their lives.

ARTICLE INFORMATION

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