

Age, sex, and contemporary outcomes in surgical repair of type A aortic dissection: Insights from the National Inpatient Sample



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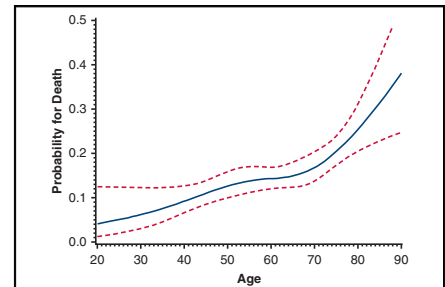
ABSTRACT

Objective: Acute type A aortic dissection (ATAAD) is a surgical emergency with significant morbidity and mortality, as well as significant center-level variation in outcomes. Our study aims to leverage a nationally representative database to assess contemporary in-hospital outcomes in surgical repair of ATAAD, as well as the association of age and sex with outcomes.

Methods: The National Inpatient Sample was queried to identify hospital discharge records of patients aged ≥ 18 years who underwent urgent surgical repair of ATAAD between 2017 and 2018. Patients with a diagnosis of thoracic aortic dissection, who underwent surgical intervention of the ascending aorta, were identified. Patient demographics were assessed, and predictors of in-hospital mortality were identified.

Results: We identified 7805 weighted cases of surgically repaired ATAAD nationally, with an overall mortality of 15.3%. Mean age was 60.0 ± 13.6 years. There was a male predominance, although female subjects made up a larger proportion of older age groups—female subjects up 18.4% of patients younger than 40 years with ATAAD but 53.6% of patients older than 80 years. In multivariable analysis controlling for sex, race, comorbidities, and malperfusion, age was a significant predictor of mortality. Patients aged 71 to 80 years had a 5.3-fold increased risk of mortality compared with patients ≤ 40 years old ($P < .001$), and patients aged > 80 years had a 6.8-fold increased risk of mortality ($P < .001$). Sex was not significantly associated with mortality.

Conclusions: Surgical repair of ATAAD continues to carry high risk of morbidity and mortality, with outcomes impacted significantly by patient age, regardless of patient comorbidity burden. (JTCVS Open 2022;11:23-36)



Contemporary prevalence and outcomes of TAAD repair, by age and sex.

CENTRAL MESSAGE

Surgical repair of ATAAD continues to carry high risk of morbidity and mortality, with outcomes impacted significantly by patient age, regardless of patient comorbidity burden and malperfusion state.

PERSPECTIVE

Much of our understanding on the short-term outcomes of type A aortic dissection repair is limited to single-center experience and databases with selective participation by high-volume aortic centers. Our study aims to leverage a nationally representative database to assess contemporary in-hospital outcomes in surgical repair of ATAAD, as well as the association of age and sex with outcomes.

Acute type A aortic dissection (ATAAD) is a surgical emergency associated with significant risk of morbidity and mortality. Although there is evidence of temporally improving

surgical outcomes of ATAAD repair in recent decades,¹⁻³ short-term mortality in surgical patients remains substantial at approximately 15% to 30%.³⁻¹³

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Abbreviations and Acronyms

ATAAD	= acute type A aortic dissection
GERAADA	= German Registry for Acute Aortic Dissection Type A
ICD-10-CM	= <i>International Classification of Diseases, 10th Revision, Clinical Modification</i>
IRAD	= International Registry of Acute Aortic Dissection
NIS	= National Inpatient Sample
OR	= odds ratio

In addition to hemodynamic compromise, degree of malperfusion, and burden of comorbidities, age has been consistently identified as a risk factor for intra- and postoperative mortality for patients undergoing surgical repair for ATAAD.^{1,2,6,9,11,12,14-25} Given the aging population of the United States and the rising prevalence of thoracic aortic disease in older patients,^{24,26} it is becoming increasingly important to understand contemporary outcomes in this patient population. Furthermore, as women have prolonged life expectancies relative to men and tend to develop aortic disease later in life,^{5,13,27} the relationship between sex, age, and outcomes is of particular interest in ATAAD. In contrast to the well-recognized discrepancy in outcomes for women undergoing other cardiac surgical procedures,^{28,29} the impact of sex on outcomes in ATAAD is limited and conflicting.^{5,13,27,30}

Much of our understanding of current outcomes of patients with ATAAD, as well as the impact of age and sex, is limited to single-center reviews and studies of voluntary registries such as the International Registry of Acute Aortic Dissection (IRAD) and the German Registry for Acute Aortic Dissection Type A (GERAADA). As such, a true perspective of the outcomes of ATAAD is lacking, as there is known center variability in ATAAD outcomes¹ and participation in voluntary registries inevitably presents significant selection bias. The objective of our study was to leverage a nationally representative database with a novel patient identification methodology to assess contemporary in-hospital outcomes in surgical repair of ATAAD, as well as the association of age and sex with outcomes.

METHODS

Database

A retrospective analysis of the National Inpatient Sample (NIS) was performed for the years 2017 to 2018. This database, collected and maintained by the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project, contains data for approximately 20% of inpatient hospital discharge records in the United States. Using discharge weights supplied by the Agency for Healthcare Research and Quality, these data can be

extrapolated as a representative sample of national discharge records. This study was deemed exempt from review by an institutional review board because the NIS is a publicly available, deidentified database.

Discharge records obtained and reported contain patient-specific information including age at admission, sex, race, insurance status, comorbidities, diagnoses reported during the hospitalization, procedures performed during the hospitalization, length of stay, total reported charges, and hospital mortality status. Diagnoses and procedures are reported using the *International Classification of Diseases, Tenth Revision, Clinical Modification* (ICD-10-CM) system.

Patient Selection and Weighting

Patient selection from the NIS database is shown in Figure 1. Discharge records of all adult patients (≥ 18 years old), admitted urgently, with a primary diagnosis of thoracic aortic dissection (I71.00) or thoracoabdominal aortic dissection (I71.03) were identified. The ICD-10-CM diagnosis coding system used in 2017 to 2018 does not distinguish between type A and type B aortic dissection. To identify ATAAD, specifically, we selected only patients who underwent open surgical intervention on the ascending aorta or aortic arch, identified using procedure codes 025X0ZZ, 02BX0ZX, 02BX0ZZ, 02QX0ZZ, 02RX07Z, 02RX08Z, 02RX0JZ, 02RX0KZ, 02UX07Z, 02UX08Z, 02UX0JZ, 02UX0KZ, 02VX0CZ, 02VX0DZ, 02VX0EZ, 02VX0FZ, and 02VX0ZZ. Day of aortic intervention, relative to admission, was also identified for each patient.

The NIS provides a weighting methodology to allow data to be extrapolated as a nationally representative sample. Because the database represents a 20% sample of discharged patients, each patient is assigned a discharge weight that is equal to approximately 5. Discharge weights have been used in all presentations of total patient count; however, to avoid overpowering the sample, discharge weights were not used for univariate

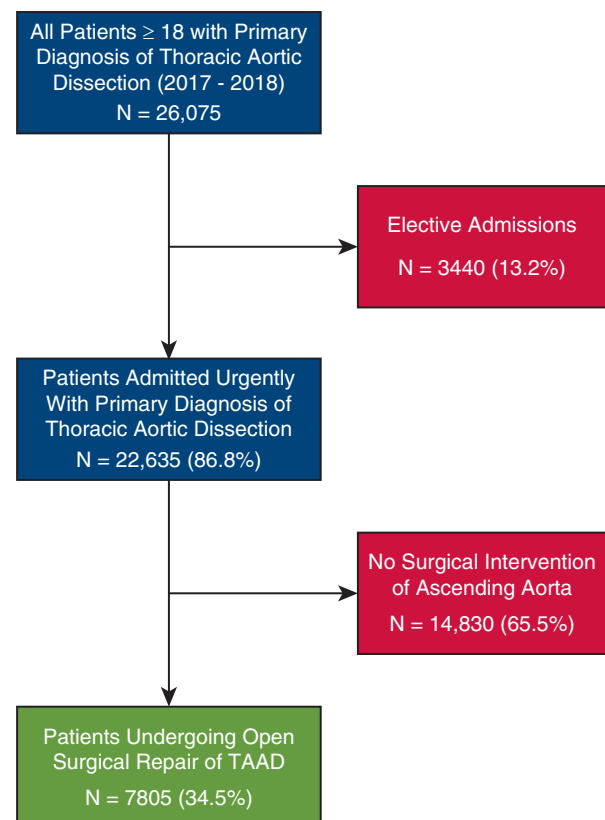


FIGURE 1. Patient selection methodology. TAAD, Type A aortic dissection.

and multivariable analyses. In other words, the N presented is weighted in all cases, but the analyses conducted exclude weighting.

Outcome Variables

The primary outcome of interest was all-cause in-hospital mortality. Length of stay and total hospital costs were also assessed. Length of stay was evaluated as the total number of days from procedure to discharge or death. Hospital charges, representing the total amount billed to patients and insurance companies, were converted to costs, which are the actual economic cost of a hospitalization to the hospital using Healthcare Cost and Utilization Project–provided year- and hospital-specific ratios. When hospital-specific information was unavailable, weighted local group averages were used.

Statistical Analysis

All statistical analyses were performed using SAS, version 9.4 (SAS Institute, Inc). All *P* values were 2-sided. A 95% confidence interval ($P < .05$) was defined as statistical significance for all analyses.

Descriptive analysis was conducted for the selected sample to characterize differences in patient and hospital characteristics, and outcomes based on age group and sex. Patient-specific variables included race (ie, White, Black, Hispanic, other), insurance status (Medicare, Medicaid, private, self-pay, other), and presence of comorbidities (ie, renal failure, obesity, peripheral vascular disease). Additionally, diagnosis codes suggesting neurologic, coronary, mesenteric, and renal malperfusion syndromes were identified. Extremity malperfusion was unable to be identified based on limitations in specific ICD-10-CM codes. The codes used for malperfusion syndromes are identified in Table E1. Other characteristics assessed for each patient included whether they were transferred-in and timing of operation (in days). Hospital-specific variables included region, hospital size (small, medium, large; defined relative to other institutions of the same region and academic status), and hospital teaching-status. The Pearson χ^2 test was used to analyze categorical variables. Student *t* test or one-way analysis of variance was used to evaluate continuous variables.

Univariate and multivariable regression analyses were performed to evaluate predictors of mortality. Age was considered as both a continuous and categorical variable. Given the nonlinearity of the spline plot for the association between age and mortality (Figure 2), age was primarily assessed as a categorical variable by decade. Age group, sex, race as a dichotomous variable (ie, White vs non-White), malperfusion syndromes, patient comorbidities, other patient characteristics, and hospital characteristics were assessed as predictors in univariate analysis. Binary logistic regressions were conducted, and odds ratios (ORs) and *P* values are reported. The variables used in the multivariable model were primarily selected based on significance at $P < .05$ in univariate analysis. Given limitations inherent to ICD-10 coding for comorbidities (ie, it is not possible to determine whether a condition was a pre-existing comorbidity or developed postoperatively), individual comorbidities were replaced in the multivariable model with the Elixhauser Comorbidity Index as an indicator of overall health status and frailty. Additionally, as a primary aim of this study is to assess the impact of both age and sex on surgical outcomes of TAA, sex was included in the multivariable analysis despite insignificance in univariate models.

RESULTS

Over the study period of 2017 to 2018, we identified 7805 weighted cases of surgically repaired ATAAD in the United States, with a mortality rate of 15.3%. Patient demographics, hospital characteristics, and outcomes of the entire sample are described in Table 1. Mean age was noted to be 60.0 ± 13.6 years, with 8.7% of patients ≤ 40 years, 14.7% of patients 41 to 50 years, 23.2% of patients 51 to

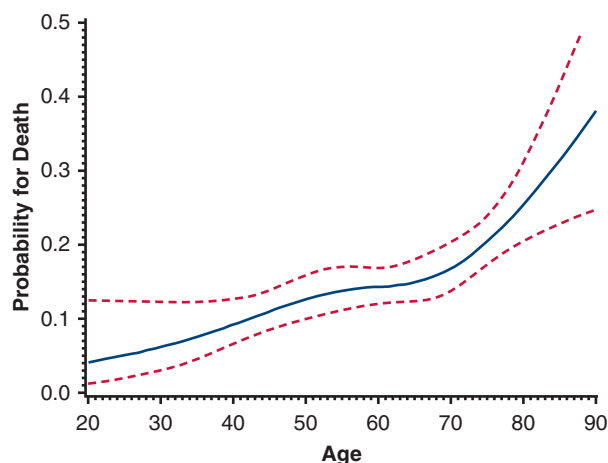


FIGURE 2. Spline plot, age and mortality.

60 years, 25.8% of patients 61 to 70 years, 21.5% of patients 71 to 80 years, and 6.2% of patients >80 years. There was a male predominance, with female patients making up only 34.0% of the sample. White patients made up 63.8% of the total, and the majority of patients used either private insurance (33.9%) or Medicare (41.2%). Common comorbidities included peripheral vascular disease (29.7%), obesity (20.6%), and chronic lung disease (17.0%). Malperfusion was present in a large proportion of patients, with 14.2% neurologic malperfusion, 4.4% coronary malperfusion, 1.9% mesenteric malperfusion, and 48.6% renal malperfusion. About one half of the patients in the sample were transferred from another hospital before surgical intervention (50.6%). The majority of patients were cared for in large hospitals (78.3%) and urban teaching centers (92.4%).

Age was noted to be a statistically significant predictor of mortality in univariate analysis, both as a continuous and categorical variable. Other predictors of mortality identified included insurance status, peripheral vascular disease, and malperfusion syndromes. Coronary malperfusion (mortality 31.9%, $P < .001$), mesenteric malperfusion (mortality 56.7%, $P < .001$), and renal malperfusion (mortality 18.3%, $P = .001$) were associated with increased mortality. Neurologic malperfusion was associated with a trend toward increased mortality (mortality 19.4%, $P = .070$).

Analysis of patient characteristics and outcomes, by age, is presented in Table 2. Female and White patients make up an increasing proportion of patients undergoing surgical repair of ATAAD across increasing age groups ($P < .001$). Female patients make up 18.4% of patients younger than 40 years with surgical ATAAD; meanwhile, 53.6% of patients older than 80 years are female (Figure 3). Similarly, White patients make up 47.6% of patients younger than the age of 40 years, yet they make up 90.6% of patients older than the age of 80 years. Importantly, as shown in Figure 4, there is a significant and substantial association

TABLE 1. Demographics, hospital characteristics, and primary outcomes of patient sample

Variable	Total	Died	Alive	P value
Total	7805	1195 (15.3)	6605 (84.6)	
Age, y	60.0 ± 13.6	64.2 ± 13.0	59.2 ± 13.6	<.001
Age group, y				<.001
≤40	680 (8.7)	50 (7.4)	630 (92.6)	
41-50	1145 (14.7)	135 (11.8)	1010 (88.2)	
51-60	1810 (23.2)	245 (13.5)	1565 (86.5)	
61-70	2010 (25.8)	275 (13.7)	1735 (86.3)	
71-80	1675 (21.5)	360 (21.5)	1315 (78.5)	
>80	485 (6.2)	130 (27.1)	350 (72.9)	
Female	2650 (34.0)	450 (37.7)	2200 (63.3)	.191
Race				.003
White	4705 (63.8)	835 (17.7)	3870 (82.3)	
Black	1650 (22.3)	160 (9.7)	1490 (90.3)	
Hispanic	585 (7.9)	60 (10.3)	525 (89.7)	
Other race	440 (6.0)	50 (11.4)	390 (88.6)	
Unknown race	420 (5.4)	90 (21.4)	330 (78.6)	
Insurance status				<.001
Medicare	3200 (41.2)	635 (19.8)	2560 (80.2)	
Medicaid	1215 (15.6)	155 (12.8)	1060 (87.2)	
Private	2635 (33.9)	310 (11.8)	2325 (88.2)	
Self-pay	460 (5.9)	60 (13.0)	400 (87.0)	
Other	295 (3.8)	35 (8.9)	260 (91.1)	
Elixhauser Comorbidity Index	5.6 ± 2.0	5.8 ± 2.0	5.6 ± 1.9	.131
CHF	185 (2.4)	25 (13.5)	160 (86.5)	.757
PVD	2315 (29.7)	440 (19.0)	1870 (81.0)	.008
CLD	1325 (17.0)	210 (15.8)	1115 (84.2)	.793
DM w/out complications	285 (3.7)	40 (14.0)	245 (86.0)	.783
DM w/complications	655 (8.4)	90 (13.7)	565 (86.3)	.599
Hypothyroidism	620 (7.9)	80 (12.9)	540 (87.1)	.436
Obesity	1610 (20.6)	245 (15.2)	1365 (84.8)	.954
Weight loss	860 (11.0)	135 (15.7)	720 (84.3)	.856
Depression	570 (7.3)	90 (15.8)	480 (84.2)	.885
Marfan syndrome	140 (1.8)	15 (10.7)	125 (89.3)	.444
Malperfusion syndromes				
Neurologic malperfusion	1110 (14.2)	215 (19.4)	895 (80.6)	.070
Coronary malperfusion	345 (4.4)	110 (31.9)	235 (68.1)	<.001
Mesenteric malperfusion	150 (1.9)	85 (56.7)	65 (43.3)	<.001
Renal malperfusion	3790 (48.6)	695 (18.3)	3095 (81.7)	.001
Transfer	3950 (50.6)	590 (14.9)	3360 (85.1)	.669
Repair day				.072
Day 0	5265 (68.3)	885 (16.8)	4375 (83.2)	
Day 1	1460 (18.9)	200 (13.7)	1260 (86.3)	
Day 2+	985 (12.6)	110 (11.2)	875 (88.8)	
Length of stay, d	13.8 ± 12.7	8.2 ± 15.8	14.9 ± 11.8	<.001
Total hospital costs, \$	94,751 ± 75,500	96,331 ± 72,647	94,427 ± 76,023	.719
Hospital region				.540
Northeast	1595 (20.4)	255 (16.0)	1335 (84.0)	
Midwest	1795 (23.0)	245 (13.6)	1550 (86.4)	
South	2770 (35.5)	405 (14.6)	2365 (85.4)	
West	1645 (21.1)	290 (17.6)	1355 (82.4)	

(Continued)

TABLE 1. Continued

Variable	Total	Died	Alive	P value
Hospital size				.347
Small	390 (5.0)	70 (17.9)	320 (82.1)	
Medium	1300 (16.7)	215 (16.5)	1085 (83.5)	
Large	6115 (78.3)	910 (14.9)	5200 (85.1)	
Teaching status				.363
Rural	60 (0.8)	10 (16.7)	50 (83.3)	
Urban, nonteaching	535 (6.9)	100 (18.7)	435 (81.3)	
Urban, teaching	7210 (92.4)	1085 (15.0)	6120 (85.0)	

Values reported are n (%) or mean \pm standard deviation. Percent reported in "Total" column represents percent of all patients; percent reported in "Died" and "Alive" columns represents percent mortality/survival for that subgroup. CHF, Congestive heart failure; PVD, peripheral vascular disease; CLD, chronic lung disease; DM, diabetes mellitus.

between increasing age and mortality, with a 7.4% mortality rate among patients ≤ 40 years compared with 21.5% in patients 71 to 80 years and 27.1% in patients older than 80 years ($P < .001$). Figure 4 also depicts a mortality rate among female patients aged older than 80 years of 34.6%, compared with a mortality rate of 18.2% among male patients aged older than 80 years. Given this finding, as well as the increasing proportion of female patients among older age groups, a subgroup univariate logistic analysis was conducted to assess the interaction between age, sex, and mortality. When selecting for patients aged younger than 70 years, the OR for mortality for female versus male sex was 0.93 (0.63-1.38), $P = .71$. For patients aged ≥ 70 years, the OR for mortality for female versus male sex was 1.24 (0.78-1.95), $P = .36$.

The Elixhauser Comorbidity Index was significantly associated with age, with a greater burden of comorbidities among older patients. On the contrary, malperfusion syndromes were not associated with patient age at presentation. Total hospital costs and length of stay among surviving patients were not significantly associated with age group. Finally, there was no association identified between hospital characteristics and age.

Age remained an independent predictor of survival on multivariable analysis (Table 3). Compared with patients ≤ 40 years old, older patients had significantly increased risk of mortality. Patients aged 71 to 80 years had a 5.3-fold increased risk of mortality compared with patients ≤ 40 years old ($P = .001$), and patients aged > 80 years had a 6.8-fold increased risk of mortality ($P < .001$). While there is a greater burden of comorbidity among older patients (Table 2), multivariable analysis revealed that comorbidity burden is not significantly associated with survival when controlling for age as a risk factor. Coronary (2.59, $P = .001$), mesenteric (OR, 6.17; $P < .001$), and renal malperfusion (OR, 1.40; $P = .036$) were also found to be significant predictors of mortality. Despite the increased prevalence of patients undergoing surgical repair of ATAAD among female patients in older age groups, when controlling for age, sex was not found to be associated

with increased mortality. In contrast White race remained a significant predictor of mortality in multivariable analysis (OR, 1.65; $P = .006$).

DISCUSSION

Our study is the first to leverage the NIS to present contemporary prevalence and outcomes of surgical repair of ATAAD. As such, we present the largest recent assessment of the impact of age and sex on surgical outcomes in ATAAD. We found a significant increase in in-hospital mortality after the age of 70 years, increasing from 12.5% to 22.7%. Although female patients make up a significantly greater proportion of septuagenarians and octogenarians (48.8% of patients ≥ 70 years vs 28.3% of patients < 70 years), female sex was not a significant predictor of mortality in ATAAD when we controlled for age (Figure 5).

Overall surgical mortality of 15.3% in this study is consistent with the majority of existing literature in ATAAD, which reports operative mortality ranging from 15% to 30%.³⁻¹³ At IRAD-participating institutions from 1996 to 2001, Trimarchi and colleagues²⁵ identified an overall in-hospital mortality rate of 25.1% in 526 patients with ATAAD undergoing surgical repair.²⁵ In contrast, in a more contemporary IRAD analysis, Pape and colleagues³ found that surgical mortality has declined from 25% in 2003 to 18% in 2013. Similarly, Dobarina and colleagues,¹ who used the NIS to assess trends and volume–outcome relationships in ATAAD repair in the United States from 2005 to 2014, found a decrease in-hospital mortality from 23.5% in 2005 to 14.2% in 2014.

Increasing age has consistently been identified as a significant predictor of mortality for patients undergoing ATAAD repair.^{1,2,6,9,11,12,14-25} Using the Taiwan National Health Insurance Research Database, Hsu and colleagues¹⁶ reported significantly greater in-hospital mortality among octogenarians versus nonoctogenarians; in-hospital complications also occurred more frequently in the octogenarian group. Similarly, Trimarchi and colleagues²⁴ identified age ≥ 70 years as an independent

TABLE 2. Demographics, hospital characteristics, and primary outcomes, by age

Variable	≤40 y	41-50 y	51-60 y	61-70 y	71-80 y	>80 y	P value
Total, n (%)	680 (8.7)	1145 (14.7)	1810 (23.2)	2010 (25.8)	1675 (21.5)	485 (6.2)	
Female	125 (18.4)	290 (25.3)	490 (27.1)	690 (34.3)	795 (47.5)	260 (53.6)	<.001
Race							<.001
White	300 (47.6)	495 (45.4)	935 (55.8)	1220 (63.9)	1325 (82.8)	435 (90.6)	
Black	185 (29.4)	415 (38.1)	500 (29.9)	405 (21.2)	140 (8.8)	<10 (<2.1)	
Hispanic	95 (15.1)	125 (11.5)	135 (8.1)	150 (7.9)	60 (3.7)	20 (4.2)	
Other race	50 (8.0)	55 (5.0)	105 (6.3)	135 (7.0)	75 (4.7)	20 (4.2)	
Unknown race	50 (8.0)	55 (5.0)	135 (7.5)	100 (5.0)	75 (4.7)	<10 (<2.1)	
Insurance status							<.001
Medicare	20 (3.0)	100 (8.7)	215 (12.0)	930 (46.4)	1480 (88.4)	455 (93.8)	
Medicaid	215 (32.1)	310 (27.1)	485 (27.2)	190 (9.5)	10 (0.6)	<10 (<2.0)	
Private	300 (44.8)	590 (51.5)	830 (46.5)	750 (37.4)	140 (8.4)	25 (5.2)	
Self-pay	100 (14.9)	100 (8.7)	175 (9.8)	0 (0.0)	15 (0.9)	0 (0.0)	
Other	45 (6.6)	45 (3.9)	105 (5.8)	70 (3.5)	30 (1.8)	0 (0.0)	
Elixhauser Comorbidity Index	5.0 ± 2.0	5.5 ± 2.1	5.6 ± 2.0	5.7 ± 2.0	5.8 ± 1.9	6.0 ± 1.6	<.001
CHF	0 (0.0)	35 (3.1)	45 (2.5)	30 (1.5)	50 (3.0)	25 (5.2)	.098
PVD	135 (19.9)	320 (27.9)	575 (31.8)	605 (30.1)	535 (31.9)	145 (29.9)	.049
CLD	65 (9.6)	175 (15.3)	320 (17.7)	370 (18.4)	330 (19.7)	65 (13.4)	.063
DM w/out complications	5 (0.7)	15 (1.3)	75 (4.1)	90 (4.5)	85 (5.1)	15 (3.1)	.015
DM w/complications	25 (3.7)	55 (4.8)	135 (7.5)	210 (10.4)	185 (11.0)	45 (9.3)	<.001
Hypothyroidism	25 (3.7)	45 (3.9)	85 (4.7)	135 (6.7)	240 (14.3)	90 (18.6)	<.001
Obesity	205 (30.1)	310 (27.1)	405 (22.4)	435 (21.6)	215 (12.8)	40 (8.2)	<.001
Weight loss	45 (6.6)	120 (10.5)	165 (9.1)	190 (9.5)	250 (14.9)	90 (18.6)	.001
Depression	25 (3.7)	70 (6.1)	135 (7.5)	185 (9.2)	135 (8.1)	20 (4.1)	.253
Marfan syndrome	70 (10.3)	25 (2.2)	30 (1.7)	15 (0.7)	0 (0.0)	0 (0.0)	<.001
Malperfusion syndromes							
Neurologic malperfusion	115 (16.9)	170 (14.8)	255 (14.1)	290 (14.4)	200 (11.9)	85 (17.5)	.672
Coronary malperfusion	15 (2.2)	60 (5.2)	100 (5.5)	65 (3.2)	80 (4.8)	25 (5.2)	.472
Mesenteric malperfusion	10 (1.5)	20 (1.7)	40 (2.2)	45 (2.2)	30 (1.8)	5 (1.0)	.989
Renal malperfusion	335 (49.3)	565 (49.3)	870 (48.1)	970 (48.3)	795 (47.5)	260 (53.6)	.936
Transfer	395 (58.1)	600 (52.4)	915 (50.6)	955 (47.5)	850 (50.7)	235 (48.5)	.117
Repair day							.531
Day 0	440 (65.2)	810 (72.3)	1325 (73.4)	1315 (66.2)	1055 (63.9)	320 (67.4)	
Day 1	145 (21.5)	195 (17.4)	220 (18.3)	340 (17.1)	365 (22.1)	85 (17.9)	
Day 2+	95 (14.0)	140 (12.2)	265 (14.6)	355 (17.7)	255 (15.2)	80 (16.5)	
Mortality	50 (7.4)	135 (11.8)	245 (13.5)	275 (13.7)	360 (21.5)	130 (27.1)	<.001
Length of stay, d	13.0 ± 10.1	14.5 ± 14.0	14.0 ± 14.8	14.2 ± 13.2	13.6 ± 10.3	13.3 ± 9.2	.945
Total hospital costs, \$	97,080 ± 77,626	94,168 ± 72,616	93,423 ± 80,164	96,763 ± 81,127	96,305 ± 71,773	84,097 ± 41,233	.308
Hospital region							.399
Northeast	120 (17.6)	200 (17.5)	385 (21.3)	430 (21.4)	345 (20.6)	115 (23.7)	
Midwest	150 (22.1)	295 (25.8)	440 (24.3)	430 (21.4)	385 (23.0)	95 (19.6)	
South	200 (29.4)	475 (41.5)	635 (35.1)	700 (34.8)	570 (34.0)	190 (39.2)	
West	210 (30.9)	175 (15.3)	350 (19.3)	450 (22.4)	375 (22.4)	85 (17.5)	
Hospital size							.242
Small	35 (5.1)	40 (3.5)	75 (4.1)	100 (5.0)	95 (5.7)	45 (9.3)	
Medium	130 (19.1)	175 (15.3)	295 (16.3)	365 (18.2)	245 (14.6)	90 (18.6)	
Large	515 (75.7)	930 (81.2)	1440 (79.6)	1545 (76.9)	1335 (79.7)	350 (72.2)	
Teaching status							.776
Rural	<10 (<1.5)	10 (0.9)	15 (0.8)	15 (0.7)	15 (0.9)	0 (0.0)	
Urban, nonteaching	35 (5.1)	55 (4.8)	170 (9.4)	125 (6.2)	115 (6.9)	35 (7.2)	
Urban, teaching	640 (94.1)	1080 (94.3)	1625 (89.8)	18,370 (93.0)	1545 (92.2)	450 (92.8)	

CHF, Congestive heart failure; PVD, peripheral vascular disease; CLD, chronic lung disease; DM, diabetes mellitus.

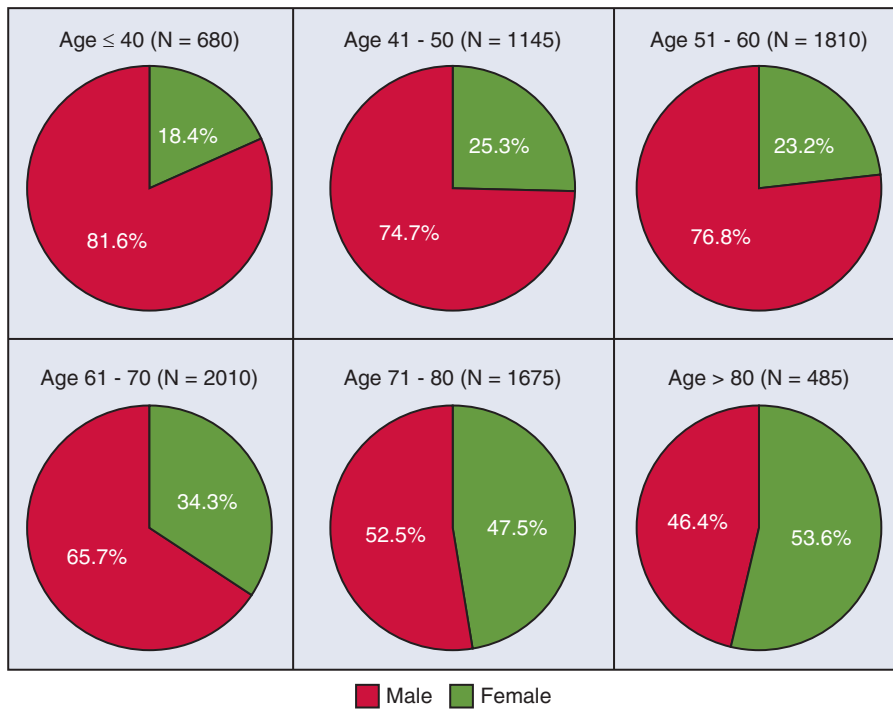


FIGURE 3. Sex of patients undergoing acute type A aortic dissection repair, by age group, 2017-2018.

predictor of mortality in both medically and surgically managed ATAAD, with overall mortality rates significantly greater in patients ≥ 70 years. Age-related discrepancies in outcomes have been shown to persist following stratification by Penn class.¹⁷

Despite consistent identification of age-related differences in ATAAD surgical mortality, the degree varies within the literature, ranging from 25.0% to 61.0% in patients ≥ 70 years old.^{6,14,15,18,20,21,23} The majority of previous studies use data from either single institutions or voluntary

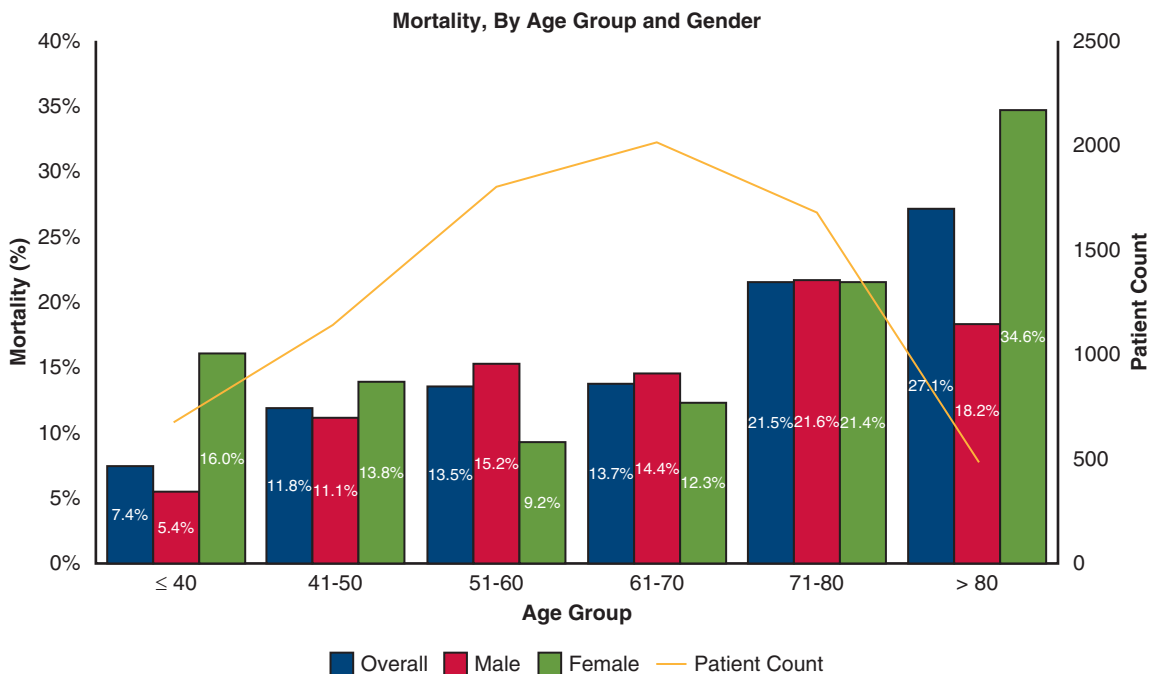


FIGURE 4. Incidence of acute type A aortic dissection repair, and mortality, by age group and sex, 2017-2018.

TABLE 3. Multivariable predictors of mortality

Variable	Odds ratio for mortality (confidence interval)	P value
Age group (vs ≤ 40 y)		
41-50 y	2.86 (1.06-7.73)	.039
51-60 y	3.33 (1.28-8.66)	.014
61-70 y	3.12 (1.21-8.08)	.019
71-80 y	5.31 (2.05-13.73)	.001
>80 y	6.82 (2.44-19.05)	<.001
Female	1.09 (0.80-1.50)	.584
Elixhauser Comorbidity Index	1.01 (0.93-1.10)	.787
White race	1.65 (1.16-2.35)	.006
Coronary malperfusion	2.59 (1.47-4.57)	.001
Mesenteric malperfusion	6.17 (2.68-14.2)	<.0001
Renal malperfusion	1.40 (1.02-1.90)	.036

registries. Single-institution studies are often limited by small sample sizes. Furthermore, as ATAAD repair in lower-volume hospitals is associated with an increased risk of mortality, this may contribute to the heterogeneity in reported outcomes between institutions.¹ As not all institutions participate in registries such as IRAD and GERAADA, studies using such registries are limited by institutional selection bias.^{4,31,32} The majority of ATAAD repaired in the United States are not performed at high-volume aortic centers that participate in these registries. Many of these institutions also lack the level of expertise and resources that high-volume aortic centers have in pre-, intra-, and postoperative care of these patients. Our use of the NIS database, which captures and provides a nationally representative snapshot of hospitalization outcomes, eliminates both sample size and institutional biases.

In contrast to the consistent sex-related discrepancies in other aspects of cardiac surgery,^{28,29} the literature regarding sex-related differences in ATAAD is limited and conflicting. In an early study, Nienaber and colleagues²⁷ assessed sex differences in acute type A and type B aortic dissection using IRAD, with female patients making up 32% of the 1078 patients; they found that female sex was an independent predictor of mortality. The authors also noted age-related differences in sex distribution, with female patients comprising a much greater proportion of dissections at age ≥ 75 years compared with ≤ 65 years. They further identified differences in mortality between female and male patients in the 66- to 75-year age group (36% vs 16%; $P < .001$), but no differences within other age brackets.²⁷ In contrast, more recent studies have demonstrated a lack of sex-related difference in outcomes.^{5,13,30} In their review of 251 ATAAD repairs across four academic institutions, Conway and colleagues⁵ identified a 68.5% male predominance and highlighted that female patients presented at

older ages; they identified no difference in mortality between female and male patients. In a single-center assessment of 504 patients who underwent surgical repair of ATAAD, Fukui and colleagues³⁰ also identified that female patients, who made up 48.6% of their sample, presented at older ages. Although differences existed in age and procedure between male and female patients, neither of these variables was found to be independently associated with operative mortality.³⁰ Rylski and colleagues¹³ assessed sex-related differences using GERAADA. While ATAAD was approximately twice as common in male patients, and there were differences in extent of dissection, degree of malperfusion, and complexity of repair between sexes, there was no difference in 30-day mortality between women and men.¹³ Most recently, Huckaby and colleagues³³ assessed sex-associated outcomes in ATAAD using IRAD. They found that, over the study period of 1996 to 2018, in-hospital mortality was greater among female patients. However, when controlling for age and other patient characteristics, a significant sex-related difference in survival no longer existed.

In this study, we identify no overall association between sex and in-hospital mortality, nor do we identify sex-related differences in mortality by age group. This is consistent with the findings of the recent single-center, GERAADA, and IRAD studies.^{13,30,33} Among patients ≥ 70 years old, female patients appeared to have increased risk of mortality than male patients (28.2% vs 19.1%); however, this difference was not significant. There are a number of possible explanations for the observed age–sex interaction. First, the difference in age of presentation may be related to the fact that premenopausal female patients benefit from the cardioprotective effects of estrogen; therefore, women often do not experience cardiovascular disease until the onset of menopause. Additionally, as demonstrated in the study of the Nienaber and colleagues, there is an increased rate of hypertension among older female patients; this supports the notion that hypertensive vasculopathy plays a key role in dissection pathogenesis, particularly in this demographic.²⁷ Further exploration into sex-related differences is warranted to elucidate why mortality in women who are septuagenarians and octogenarians is not a significant contributor to increased rates of mortality in these age groups.

We have also identified a significant difference in race, by age group, in our sample. White patients make up 52.3% of patients < 70 years and 81.5% of patients ≥ 70 years; in contrast, Black patients make up 26.7% in < 70 years and 6.9% in ≥ 70 year age groups. This may be, in part, due to health care disparities contributing to differences in life expectancy among various racial groups.³⁴ This finding is consistent with the study by Bossone and colleagues³⁵ of ATAAD in Black patients. Using 2006-2011 IRAD data, they identified that ATAAD was significantly less prevalent

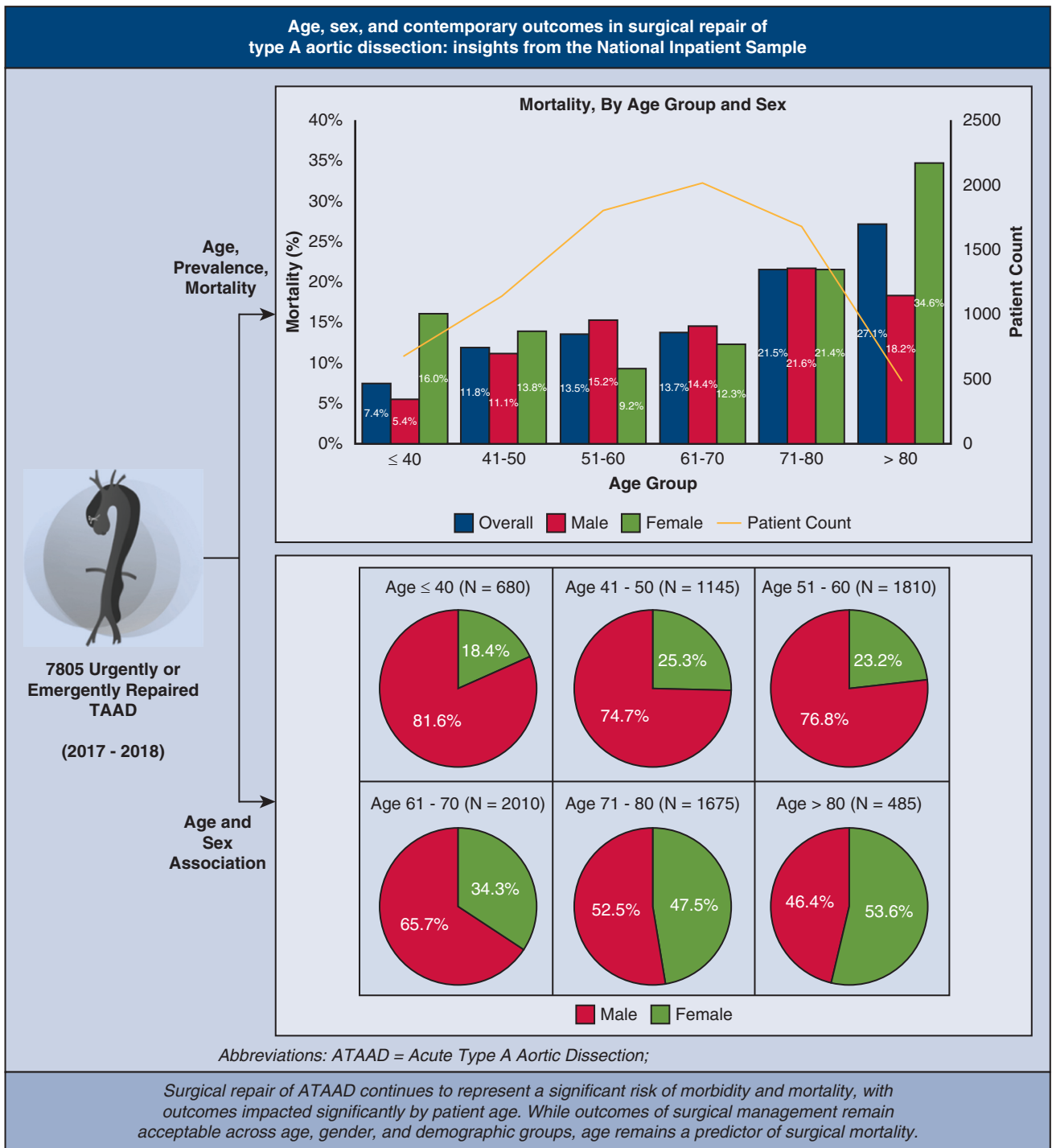


FIGURE 5. Summary of methodology and key findings. TAAD, Type A aortic dissection; ATAAD, acute type A aortic dissection.

in the Black population compared with Whites; further, they found that Black patients were younger and made up a smaller proportion of the cohort of patients aged ≥ 70 years.³⁵ Interestingly, we also found that White race was associated with increased mortality following ATAAD repair. This discrepancy may be related to comorbidity

burden, degree of dissection, or intraoperative factors, and it certainly warrants further exploration.

A key strength of our study relative to previous literature is the use of contemporary NIS data to accurately and comprehensively identify patients with ATAAD undergoing surgical intervention on a national scale. Previous publications have

used the NIS to study ATAAD-related mortality; however, unlike our study, patient selection criteria used *International Classification of Diseases, Ninth Revision, Clinical Modification* coding.^{1,2} The *International Classification of Diseases, Ninth Revision, Clinical Modification* procedural coding system lacks codes specifying ascending aortic surgical intervention. Thus, there is a greater likelihood that such studies capture type B aortic dissections. ICD-10-CM, in contrast, adopted codes more specific to ATAAD and its subsequent surgical management. We are the first study to our knowledge that uses ICD-10-CM coding with the NIS to evaluate surgical outcomes of ATAAD thus ensuring that we are only capturing type A, not type B dissections. We further excluded patients who were admitted electively; this was done to exclude patients with incidentally discovered chronic dissections and/or iatrogenic aortic dissections.

There are certain limitations of the database and study that must be acknowledged. First, as an administrative database, data selection and analysis are reliant on ICD-10 diagnosis and procedure codes extracted from discharge records. There are nuances in specific diagnoses and procedure approaches that cannot be distinguished using these codes. Different hospital systems may also have varied methods of reporting, and data entry errors are possible. Further, we rely on specifics within the procedure code to differentiate between ATAAD and nontype A aortic dissections; thus, we do not capture patients presenting with ATAAD who did not undergo surgical repair or did not survive until hospital admission, and cannot assess prevalence or outcomes of medical management. Second, the coding in the database does not allow us to differentiate between certain comorbidities and consequences of malperfusion that were present upon hospital arrival. This affects the validity of both the comorbidity and malperfusion variables selected. For instance, a diagnosis of renal failure may reference pre-existing chronic kidney disease, or it may reference acute kidney injury secondary to ATAAD. Third, the NIS does not provide detail that is important in the assessment and management of patients with ATAAD. For example, we are unable to fully assess extent of dissection, timing of symptoms, time to operating room, procedural approaches and extent of repair, circulatory arrest time, and cerebral perfusion technique. Fourth, we are unable to identify details regarding the institutions from which patients were transferred, including whether or not they had cardiac surgical capabilities. Finally, the NIS reports independent hospitalizations rather than patients. Therefore, outcomes can only be tracked over the course of a single hospitalization, and survival after discharge is not captured.

CONCLUSIONS

Surgical repair of ATAAD continues to represent a significant risk of morbidity and mortality, with outcomes impacted significantly by patient age. While outcomes of

surgical management remain acceptable across age, sex, and demographic groups, age remains a significant predictor of surgical mortality.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: type A aortic dissection, aortic surgery, demographics

TABLE E1. ICD-10-CM diagnosis codes used to identify neurologic, coronary, mesenteric, and renal malperfusion

Neurologic malperfusion
I6300 Cerebral infarction due to thrombosis of unspecified precerebral artery
I63011 Cerebral infarction due to thrombosis of right vertebral artery
I63012 Cerebral infarction due to thrombosis of left vertebral artery
I63013 Cerebral infarction due to thrombosis of bilateral vertebral arteries
I63019 Cerebral infarction due to thrombosis of unspecified vertebral artery
I6302 Cerebral infarction due to thrombosis of basilar artery
I63031 Cerebral infarction due to thrombosis of right carotid artery
I63032 Cerebral infarction due to thrombosis of left carotid artery
I63033 Cerebral infarction due to thrombosis of bilateral carotid arteries
I63039 Cerebral infarction due to thrombosis of unspecified carotid artery
I6309 Cerebral infarction due to thrombosis of other precerebral artery
I6310 Cerebral infarction due to embolism of unspecified precerebral artery
I63111 Cerebral infarction due to embolism of right vertebral artery
I63112 Cerebral infarction due to embolism of left vertebral artery
I63113 Cerebral infarction due to embolism of bilateral vertebral arteries
I63119 Cerebral infarction due to embolism of unspecified vertebral artery
I6312 Cerebral infarction due to embolism of basilar artery
I63131 Cerebral infarction due to embolism of right carotid artery
I63132 Cerebral infarction due to embolism of left carotid artery
I63133 Cerebral infarction due to embolism of bilateral carotid arteries
I63139 Cerebral infarction due to embolism of unspecified carotid artery
I6319 Cerebral infarction due to embolism of other precerebral artery
I6320 Cerebral infarction due to unspecified occlusion or stenosis of unspecified precerebral arteries
I63211 Cerebral infarction due to unspecified occlusion or stenosis of right vertebral artery
I63212 Cerebral infarction due to unspecified occlusion or stenosis of left vertebral artery
I63213 Cerebral infarction due to unspecified occlusion or stenosis of bilateral vertebral arteries
I63219 Cerebral infarction due to unspecified occlusion or stenosis of unspecified vertebral artery
I6322 Cerebral infarction due to unspecified occlusion or stenosis of basilar artery
I63231 Cerebral infarction due to unspecified occlusion or stenosis of right carotid arteries
I63232 Cerebral infarction due to unspecified occlusion or stenosis of left carotid arteries
I63233 Cerebral infarction due to unspecified occlusion or stenosis of bilateral carotid arteries
I63239 Cerebral infarction due to unspecified occlusion or stenosis of unspecified carotid artery
I6329 Cerebral infarction due to unspecified occlusion or stenosis of other precerebral arteries
I6330 Cerebral infarction due to thrombosis of unspecified cerebral artery
I63311 Cerebral infarction due to thrombosis of right middle cerebral artery
I63312 Cerebral infarction due to thrombosis of left middle cerebral artery
I63313 Cerebral infarction due to thrombosis of bilateral middle cerebral arteries
I63319 Cerebral infarction due to thrombosis of unspecified middle cerebral artery
I63321 Cerebral infarction due to thrombosis of right anterior cerebral artery
I63322 Cerebral infarction due to thrombosis of left anterior cerebral artery
I63323 Cerebral infarction due to thrombosis of bilateral anterior cerebral arteries
I63329 Cerebral infarction due to thrombosis of unspecified anterior cerebral artery
I63331 Cerebral infarction due to thrombosis of right posterior cerebral artery
I63332 Cerebral infarction due to thrombosis of left posterior cerebral artery
I63333 Cerebral infarction due to thrombosis of bilateral posterior cerebral arteries
I63339 Cerebral infarction due to thrombosis of unspecified posterior cerebral artery
I63341 Cerebral infarction due to thrombosis of right cerebellar artery
I63342 Cerebral infarction due to thrombosis of left cerebellar artery
I63343 Cerebral infarction due to thrombosis of bilateral cerebellar arteries
I63349 Cerebral infarction due to thrombosis of unspecified cerebellar artery
I6339 Cerebral infarction due to thrombosis of other cerebral artery
I6340 Cerebral infarction due to embolism of unspecified cerebral artery
I63411 Cerebral infarction due to embolism of right middle cerebral artery
I63412 Cerebral infarction due to embolism of left middle cerebral artery
I63413 Cerebral infarction due to embolism of bilateral middle cerebral arteries

(Continued)

TABLE E1. Continued

I63419 Cerebral infarction due to embolism of unspecified middle cerebral artery
I63421 Cerebral infarction due to embolism of right anterior cerebral artery
I63422 Cerebral infarction due to embolism of left anterior cerebral artery
I63423 Cerebral infarction due to embolism of bilateral anterior cerebral arteries
I63429 Cerebral infarction due to embolism of unspecified anterior cerebral artery
I63431 Cerebral infarction due to embolism of right posterior cerebral artery
I63432 Cerebral infarction due to embolism of left posterior cerebral artery
I63433 Cerebral infarction due to embolism of bilateral posterior cerebral arteries
I63439 Cerebral infarction due to embolism of unspecified posterior cerebral artery
I63441 Cerebral infarction due to embolism of right cerebellar artery
I63442 Cerebral infarction due to embolism of left cerebellar artery
I63443 Cerebral infarction due to embolism of bilateral cerebellar arteries
I63449 Cerebral infarction due to embolism of unspecified cerebellar artery
I6349 Cerebral infarction due to embolism of other cerebral artery
I6350 Cerebral infarction due to unspecified occlusion or stenosis of unspecified cerebral artery
I63511 Cerebral infarction due to unspecified occlusion or stenosis of right middle cerebral artery
I63512 Cerebral infarction due to unspecified occlusion or stenosis of left middle cerebral artery
I63513 Cerebral infarction due to unspecified occlusion or stenosis of bilateral middle cerebral arteries
I63519 Cerebral infarction due to unspecified occlusion or stenosis of unspecified middle cerebral artery
I63521 Cerebral infarction due to unspecified occlusion or stenosis of right anterior cerebral artery
I63522 Cerebral infarction due to unspecified occlusion or stenosis of left anterior cerebral artery
I63523 Cerebral infarction due to unspecified occlusion or stenosis of bilateral anterior cerebral arteries
I63529 Cerebral infarction due to unspecified occlusion or stenosis of unspecified anterior cerebral artery
I63531 Cerebral infarction due to unspecified occlusion or stenosis of right posterior cerebral artery
I63532 Cerebral infarction due to unspecified occlusion or stenosis of left posterior cerebral artery
I63533 Cerebral infarction due to unspecified occlusion or stenosis of bilateral posterior cerebral arteries
I63539 Cerebral infarction due to unspecified occlusion or stenosis of unspecified posterior cerebral artery
I63541 Cerebral infarction due to unspecified occlusion or stenosis of right cerebellar artery
I63542 Cerebral infarction due to unspecified occlusion or stenosis of left cerebellar artery
I63543 Cerebral infarction due to unspecified occlusion or stenosis of bilateral cerebellar arteries
I63549 Cerebral infarction due to unspecified occlusion or stenosis of unspecified cerebellar artery
I6359 Cerebral infarction due to unspecified occlusion or stenosis of other cerebral artery
I6381 Other cerebral infarction due to occlusion or stenosis of small artery
I6389 Other cerebral infarction
I639 Cerebral infarction, unspecified
I6501 Occlusion and stenosis of right vertebral artery
I6502 Occlusion and stenosis of left vertebral artery
I6503 Occlusion and stenosis of bilateral vertebral arteries
I6509 Occlusion and stenosis of unspecified vertebral artery
I651 Occlusion and stenosis of basilar artery
I6521 Occlusion and stenosis of right carotid artery
I6522 Occlusion and stenosis of left carotid artery
I6523 Occlusion and stenosis of bilateral carotid arteries
I6529 Occlusion and stenosis of unspecified carotid artery
I658 Occlusion and stenosis of other precerebral arteries
I659 Occlusion and stenosis of unspecified precerebral artery
I6601 Occlusion and stenosis of right middle cerebral artery
I6602 Occlusion and stenosis of left middle cerebral artery
I6603 Occlusion and stenosis of bilateral middle cerebral arteries
I6609 Occlusion and stenosis of unspecified middle cerebral artery
I6611 Occlusion and stenosis of right anterior cerebral artery
I6612 Occlusion and stenosis of left anterior cerebral artery
I6613 Occlusion and stenosis of bilateral anterior cerebral arteries
I6619 Occlusion and stenosis of unspecified anterior cerebral artery
I6621 Occlusion and stenosis of right posterior cerebral artery
I6622 Occlusion and stenosis of left posterior cerebral artery

(Continued)

TABLE E1. Continued

I6623 Occlusion and stenosis of bilateral posterior cerebral arteries
I6629 Occlusion and stenosis of unspecified posterior cerebral artery
I663 Occlusion and stenosis of cerebellar arteries
I668 Occlusion and stenosis of other cerebral arteries
I669 Occlusion and stenosis of unspecified cerebral artery
I670 Dissection of cerebral arteries, nonruptured
Coronary malperfusion
I2101 ST elevation (STEMI) myocardial infarction involving left main coronary artery
I2102 ST elevation (STEMI) myocardial infarction involving left anterior descending coronary artery
I2109 ST elevation (STEMI) myocardial infarction involving other coronary artery of anterior wall
I2111 ST elevation (STEMI) myocardial infarction involving right coronary artery
I2119 ST elevation (STEMI) myocardial infarction involving other coronary artery of inferior wall
I2121 ST elevation (STEMI) myocardial infarction involving left circumflex coronary artery
I2129 ST elevation (STEMI) myocardial infarction involving other sites
I213 ST elevation (STEMI) myocardial infarction of unspecified site
I214 Non-ST elevation (NSTEMI) myocardial infarction
I219 Acute myocardial infarction, unspecified
I21A1 Myocardial infarction type 2
I21A9 Other myocardial infarction type
I220 Subsequent ST elevation (STEMI) myocardial infarction of anterior wall
I221 Subsequent ST elevation (STEMI) myocardial infarction of inferior wall
I222 Subsequent non-ST elevation (NSTEMI) myocardial infarction
I228 Subsequent ST elevation (STEMI) myocardial infarction of other sites
I229 Subsequent ST elevation (STEMI) myocardial infarction of unspecified site
Mesenteric malperfusion
K55011 Focal (segmental) acute (reversible) ischemia of small intestine
K55012 Diffuse acute (reversible) ischemia of small intestine
K55019 Acute (reversible) ischemia of small intestine, extent unspecified
K55021 Focal (segmental) acute infarction of small intestine
K55022 Diffuse acute infarction of small intestine
K55029 Acute infarction of small intestine, extent unspecified
K55031 Focal (segmental) acute (reversible) ischemia of large intestine
K55032 Diffuse acute (reversible) ischemia of large intestine
K55039 Acute (reversible) ischemia of large intestine, extent unspecified
K55041 Focal (segmental) acute infarction of large intestine
K55042 Diffuse acute infarction of large intestine
K55049 Acute infarction of large intestine, extent unspecified
K55051 Focal (segmental) acute (reversible) ischemia of intestine, part unspecified
K55052 Diffuse acute (reversible) ischemia of intestine, part unspecified
K55059 Acute (reversible) ischemia of intestine, part and extent unspecified
K55061 Focal (segmental) acute infarction of intestine, part unspecified
K55062 Diffuse acute infarction of intestine, part unspecified
K55069 Acute infarction of intestine, part and extent unspecified
Renal malperfusion
N170 Acute kidney failure with tubular necrosis
N171 Acute kidney failure with acute cortical necrosis
N172 Acute kidney failure with medullary necrosis
N178 Other acute kidney failure
N179 Acute kidney failure, unspecified
N280 Ischemia and infarction of kidney

ICD-10-CM, International Classification of Diseases, 10th Revision, Clinical Modification.