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Rates of Spinal Cord Infarction after Repair of Aortic Aneurysm or Dissection

Gino Gialdini, MD[1], **Neal S. Parikh, MD**[1,2], **Abhinaba Chatterjee**[1], **Michael P. Lerario, MD**[2,3], **Hooman Kamel, MD**[1,2], **Darren B. Schneider, MD**[4], **Babak B. Navi, MD, MS**[1,2], **Santosh B. Murthy, MD, MPH**[1,2], **Costantino Iadecola, MD**[1,2], and **Alexander E. Merkler, MD**[1,2]

[1]Clinical and Translational Neuroscience Unit, Feil Family Brain and Mind Research Institute, Weill Cornell Medicine, New York, NY

[2]Department of Neurology, Weill Cornell Medicine, New York, NY

[3]Department of Neurology, New York-Presbyterian Queens, Flushing, NY

[4]Department of Vascular and Endovascular Surgery, Weill Cornell Medicine, New York, NY

Abstract

Background and Purpose—The rate of spinal cord infarction (SCI) after surgical or endovascular repair of an aortic aneurysm or dissection is unclear.

Methods—Using administrative claims data, we identified adult patients discharged from nonfederal acute care hospitals in California, New York, and Florida who underwent surgical or endovascular repair of an aortic aneurysm or dissection between 2005 and 2013. Patients with SCI diagnosed prior to the aortic repair were excluded. Our primary outcome was a SCI during the index hospitalization for aortic repair. Descriptive statistics were used to estimate crude rates of SCI. Analyses were stratified by whether the aneurysm or dissection had ruptured and by type of repair (surgical vs. endovascular).

Results—We identified 91,212 patients who had repair of an aortic aneurysm or dissection. SCI occurred in 235 cases (0.26%; 95% CI, 0.22–0.29%). In patients with ruptured aneurysm or dissection, the rate of SCI was 0.74% (95% CI, 0.60–0.88%) compared to 0.16% (95% CI, 0.13–0.19%) with unruptured aneurysm. In secondary analyses, rates of SCI were similar after endovascular (0.91%; 95% CI, 0.62–1.19%) compared to surgical repair (0.68%; 95% CI, 0.53–0.83%, $P = 0.147$) of ruptured aortic aneurysm or dissection; however, rates of SCI were higher after surgical (0.20%; 95% CI, 0.15–0.25%) versus endovascular repair (0.11%; 95% CI, 0.08–0.14%, $P < 0.001$) of unruptured aneurysm.

Conclusions—SCI occurs in approximately 1 in 130 patients undergoing aortic dissection or ruptured aortic aneurysm repair and in 1 in 600 patients undergoing unruptured aortic aneurysm repair.

Correspondence: Alexander E. Merkler, MD, Weill Cornell Medicine, 525 East 68th Street, F610, New York, NY 10065, Tel: 212-746-0225, Fax: 212-746-5509, alm9097@med.cornell.edu.

Conflicts-of-Interest/Disclosures

The authors report no disclosures.

Keywords

Spinal cord infarction; aortic dissection; aortic aneurysm; aorta surgery; endovascular repair

Spinal cord infarction (SCI) accounts for up to 1% of all strokes[1] and can lead to severe disability including paraplegia and tetraplegia. Both surgical and endovascular repair of an aortic aneurysm or dissection can lead to infarction of the spinal cord because the vascular supply of the spinal cord largely originates directly from the aorta.[2] The rate of spinal cord infarction in patients undergoing aortic repair appears to vary depending on the presence and location of dissection,[3, 4] aneurysm rupture,[5] and type of repair (surgical or endovascular).[6, 7] Reported rates of SCI in these patients vary considerably, from 0.2% to 16%, and are largely based on single-center studies.[3, 8–12] Furthermore, since new intraprocedural strategies to prevent SCI have been developed over the last several years, including cerebrospinal fluid drainage,[13] a better understanding of the risk of SCI after aortic repair is required. This is particularly true as the number of patients who undergo repair of unruptured aneurysms in the United States is increasing.[14] We therefore sought to evaluate and compare the incidence of SCI after surgical and endovascular repair of aortic aneurysm and dissection in a large, heterogeneous cohort of patients from across the United States.

Methods

Design

We performed a retrospective cohort study using administrative claims data from all inpatient discharges in California (2005–2011), Florida (2005–2013), and New York (2006–2013). Data from all hospitalizations at non-federal hospitals were collected by the California Office of Statewide Health Planning and Development, the New York Statewide Planning and Research Cooperative System, and the Florida Agency for Health Care Administration. These databases were deidentified by the Agency for Healthcare Research and Quality, which assigned patients a personal linkage number that allows them to be anonymously tracked across multiple hospitalizations. Up to 25 discharge diagnoses and 21 procedures were coded for each encounter using the *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*) system with each diagnosis labeled as having been present before hospital admission or having newly developed during the hospitalization. Due to the de-identified nature of the data, the study was exempt from formal review by the institutional review board at Weill Cornell Medicine.

Patients

We included all patients at the time of their first recorded hospitalization for a surgical or endovascular repair of a thoracic, thoraco-abdominal, or abdominal aortic dissection or aneurysm. Patients undergoing repair for both ruptured and unruptured aneurysms were included. Cases were identified using *International Classification of Diseases, 9th Revision, Clinical Modification* (*ICD-9-CM*) procedure codes for endovascular (39.71 and 39.73) or surgical aortic repair (38.34, 38.44–45, 39.23–26, 39.57–58) in addition to diagnosis codes

for aortic dissection or aneurysm (*ICD-9-CM* codes 441.0–9) in any discharge diagnosis position within the same encounter. This approach has been used previously.[15, 16]

Measurements

Our primary outcome was SCI, defined by the *ICD-9-CM* code 336.1 in any discharge diagnosis position during the index hospitalization for repair of aortic aneurysm or dissection. We excluded cases with present-on-admission codes for SCI and any cases with a prior history of SCI before index hospitalization for aortic repair. In a secondary analysis, we expanded our definition of SCI to include any new-onset motor impairment coded as tetraplegia, paraplegia, and diplegia (*ICD-9-CM* codes 344.0–2).

Using *ICD-9-CM* codes, we identified the following vascular risk factors in our cohort: hypertension, diabetes, coronary artery disease, atrial fibrillation, peripheral vascular disease, congestive heart failure, chronic kidney disease, tobacco use, alcohol abuse, chronic obstructive pulmonary disease. In addition, we included the Elixhauser comorbidity index, a comprehensive set of comorbidity measures commonly used for administrative data, to address disease severity.[17]

Statistical analysis

We compared patients' baseline characteristics using the chi-squared test for categorical variables and the Mann-Whitney U test for continuous variables since the data were not normally distributed. Exact confidence intervals (CIs) were used to report crude rates of SCI in patients who underwent a surgical or endovascular aortic repair. We evaluated the rates of SCI in patients who had an emergency surgery for either a ruptured aneurysm or aortic dissection and then separately in patients who had nonemergent surgery for an unruptured aneurysm. We further stratified our results for these two groups by treatment approach: surgical versus endovascular. As patients who have a ruptured aortic aneurysm or dissection have high mortality,[16] and in particular, intraoperative mortality, we included only patients who survived to the end of the index hospitalization in a sensitivity analysis, using the same statistical tests as described above. All statistical analyses were performed by G.G. and A.E.M. using Stata/MP (version 13, College Station, TX). The threshold of statistical significance was set at $\alpha = 0.05$.

Results

We identified 91,212 patients who underwent aortic aneurysm or dissection repair, 38,432 of whom received surgical intervention and 51,427 of whom received endovascular intervention. Of these, 1,353 patients (1.48%) underwent both surgical and endovascular repair procedures within the same encounter. A ruptured aortic aneurysm or dissection was diagnosed in 15,304 patients, while 74,555 patients presented with unruptured aneurysms. SCI was identified in 235 cases (0.26%; 95% CI, 0.22–0.29%) and was more common among patients who were younger, females, non-white, and in patients with a higher burden of vascular risk factors (Table 1). SCI occurred more often in patients who underwent repair of a ruptured aortic aneurysm or aortic dissection (114 cases, 0.74%; 95% CI, 0.60–0.88%)

as compared to in patients who underwent repair of an unruptured aneurysm (106 cases, 0.16%; 95% CI, 0.13–0.19%).

In patients with aortic dissection or ruptured aortic aneurysm, there was no difference in the rate of SCI after surgical (76 cases, 0.68%; 95% CI, 0.53–0.83%) as compared to endovascular repair (38 cases, 0.91%; 95% CI, 0.62–1.19%, $P = 0.147$). However, in patients undergoing repair of an unruptured aneurysm, the rate of SCI appeared higher after surgical (55 cases, 0.20%; 95% CI, 0.15–0.25%) than after endovascular therapy (51 cases, 0.11%; 95% CI, 0.08–0.14%, $P < 0.001$). Characteristics of patients undergoing surgical or endovascular repair are summarized in Tables 2 and 3. Finally, of the 1,353 patients who had codes for both surgical and endovascular aortic repair during the index hospitalization, 15 developed a spinal cord infarction (1.11%; 95% CI, 0.55–1.67%). In a sensitivity analysis after excluding patients who died during the hospitalization for aortic repair, the overall SCI rate was similar (0.21%; 95% CI, 0.18–0.24%) as were the rates of SCI stratified by treatment approach. When we included new paraplegia, diplegia, and tetraplegia within the definition of SCI, the overall rate of SCI was 0.56% (95% CI, 0.51–0.61%) and again, the outcomes were unchanged stratified by treatment approach.

Discussion

In a large, heterogeneous cohort of patients who underwent surgical or endovascular repair of an aortic dissection or aneurysm, we found the overall rate of SCI was 0.26%. The rate of SCI was significantly higher (one in 130 cases) in patients who were treated for a ruptured aortic aneurysm or dissection as compared to an unruptured aneurysm (1 in 600). For patients undergoing treatment of ruptured aortic aneurysm or dissection, the rate of SCI was similar after endovascular compared to surgical repair. On the other hand, for patients with unruptured aneurysm, surgical therapy was associated with a higher risk of SCI compared to endovascular repair.

Our findings are consistent with and build upon prior analyses. The overall rate of SCI in our data appears similar to what was reported by Qureshi et al in a large, population-based study of administrative claims data, in which the overall rate of SCI after endovascular stent graft placement for aortic aneurysm or dissection repair was 0.2% in patients admitted between 2002 to 2011.[18] However, these authors included only patients who underwent endovascular aortic repair and did not separately examine patients undergoing repair of ruptured versus unruptured aneurysms. In the only prospective trial evaluating complications after surgical or endovascular treatment of ruptured abdominal aneurysms, the rate of SCI was 0.9%[19]; however, the number of patients enrolled in the trial was low (116) and there was only one outcome. Furthermore, this trial was performed in academic hospitals in the Netherlands and therefore may not be generalizable to patients undergoing aortic repair in the United States.

In contrast to prior studies, we were able to compare rates of in-hospital SCI in patients undergoing repair of ruptured aortic aneurysms and dissection versus those undergoing repair of unruptured aneurysms. The relatively low rate of SCI seen after repair of unruptured aortic aneurysm is reassuring as these patients are presumably neurologically normal prior to surgery. Additionally, our findings are consistent with prior reports of low

rates of SCI after elective open thoracic aneurysm repair.[20] We did not find any differences according to treatment approach (surgery versus endovascular repair) in patients with ruptured aortic aneurysm or aortic dissection. On the other hand, we found a higher rate of SCI after surgical repair in patients with unruptured aneurysm or dissection. Our findings, if confirmed, may be relevant to treatment approach selection for elective repair. Irrespective of treatment approach, as the number of patients undergoing repair of unruptured aortic aneurysms is increasing in frequency in the United States,[14] further research to prevent ischemic complications after aortic aneurysm repair is warranted.[21]

The results of our study should be considered in the context of several limitations. First, We defined SCI through the use of *International Classification of Diseases, 9th Revision, Clinical Modification* (*ICD-9-CM*) code 336.1 in any diagnostic position. This diagnosis algorithm has been previously used in a study analyzing claims-based data,[18] but its sensitivity and specificity have not been conclusively established. However, *ICD-9-CM* hospital discharge codes have been shown to be generally reliable for major diseases.[22] In order to try to capture all cases of SCI, in a sensitivity analysis we also included patients with diagnoses of tetraplegia, diplegia, or paraplegia. Second, as a limited number of SCI occurred in the non-white population, our results may not be generalizable to non-white patients undergoing aortic repair. Third, we lacked data regarding spinal cord imaging which is the most accurate, albeit limited test, to confirm SCI.[23] Fourth, due to the nature of the data, we could not differentiate whether SCI occurred as direct sequela of a ruptured aneurysm or aortic dissection, or due to complications of vessel repair; however, we excluded cases where SCI was present on admission. Fifth, we may have missed cases of SCI in patients who died perioperatively and therefore were not diagnosed with SCI. However, our results were similar in a sensitivity analysis excluding patients who died during the index hospitalization for aortic repair. Sixth, due to the administrative nature of the data, we lacked important granular data which may have affected the risk of perioperative SCI including aneurysm size at the time of repair, aneurysm growth over time, duration of procedure, and length of aorta replaced or repaired. Furthermore, we were unable to account for collateral blood supply to the spinal cord or use of cerebrospinal fluid drainage, which may affect the risk of SCI after aortic repair.[13,24]

Conclusion

In a large, population-based cohort of patients, we found that SCI occurs in approximately 1 in 130 patients who undergo a surgical or endovascular repair of an aortic dissection or ruptured aneurysm repair and 1 in 600 patients who undergo a surgical or endovascular repair of an unruptured aortic aneurysm. Given the relative frequency of this devastating complication of aortic repair, further study into the optimal method to prevent perioperative SCI is recommended.

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Table 1

Characteristics of Patients, Stratified by Spinal Cord Infarction

Characteristic*	SCI (N = 235)	No SCI (N = 90,977)	P
Age, mean (SD), y	69.1 (11.7)	71.1 (11.3)	0.008
Female	87 (37.0)	21,871 (24.0)	<0.001
Race†			
White	172 (74.8)	72,545 (81.0)	0.06
Black	21 (9.1)	4,656 (5.2)	
Hispanic	21 (9.1)	6,560 (7.3)	
Asian/Other	16 (7.0)	5,835 (6.5)	
Payment source‡			
Medicare	165 (69.8)	65,684 (72.2)	0.002
Medicaid	16 (7.2)	3,482 (3.8)	
Private	41 (17.4)	18,901 (20.8)	
Self-pay/other	13 (5.6)	2,906 (3.2)	
Hypertension	179 (76.2)	68,031 (74.8)	0.60
Diabetes	49 (20.9)	18,252 (20.1)	0.78
Coronary heart disease	80 (34.0)	40,478 (44.5)	0.001
Congestive heart failure	34 (14.5)	11,357 (12.5)	0.36
Peripheral vascular disease	94 (40.0)	22,058 (26.8)	<0.001
Chronic obstructive pulmonary disease	75 (31.9)	24,388 (26.8)	0.08
Chronic kidney disease	61 (26.0)	11,054 (12.2)	<0.001
Atrial fibrillation	65 (27.7)	18,920 (20.8)	0.01
Tobacco use	32 (13.6)	21,759 (23.9)	<0.001
Alcohol use	45 (19.2)	18,307 (20.1)	0.71
Elixhauser comorbidities§, mean (SD)	4.3 (1.9)	2.7 (1.6)	<0.001

Abbreviations: SCI, spinal cord infarction; SD, standard deviation

*Data are presented as number (%) unless otherwise specified.

†Self-reported by patients or their surrogates. Numbers do not sum to group totals because of missing race/ethnicity data in 2.2% of patients.

‡Numbers do not sum to group totals because of missing payment-source data in <0.01% of patients.

§Numbers represent the number of Elixhauser comorbid conditions, which comprise a comprehensive set of 28 comorbidity measures for use with large administrative datasets.

Table 2

Characteristics of Patients with Ruptured Aortic Aneurysm or Dissection, Stratified by Type of Aortic Repair*.

Characteristic†	Endovascular (N = 4,179)	Surgery (N = 11,125)	P
SCI	38 (0.9)	76 (0.7)	0.15
Age, mean (SD), y	70.7 (13.2)	65.8 (13.9)	0.02
Female	1,220 (29.2)	3,332 (29.9)	0.35
Race‡			
White	2,883 (70.6)	7,635 (70.6)	0.06
Black	480 (11.8)	1,180 10.9)	
Hispanic	330 (8.0)	1,026 (9.5)	
Asian	194(4.8)	477 (4.4)	
Other	194 (4.8)	492 (4.6)	
Payment source§			
Medicare	2,822 (67.5)	6,202 (55.7)	<0.001
Medicaid	286 (6.8)	1,034 (9.3)	
Private	793 (19.0)	3,000 (27.0)	
Self-pay	125 (3.0)	461(4.2)	
Other	153 (3.7)	427 (3.8)	
Hypertension	3,201 (76.6)	7,949 (71.5)	<0.001
Diabetes	908 (21.7)	2,135 (19.2)	<0.001
Coronary heart disease	1,514 (36.2)	3,581 (32.2)	<0.001
Congestive heart failure	611 (14.6)	2,094 (18.8)	<0.001
Peripheral vascular disease	994 (23.8)	2,163 (19.4)	<0.001
Chronic obstructive pulmonary disease	1,008 (24.1)	2,377 (21.4)	<0.001
Chronic kidney disease	731 (17.5)	1,577 (14.2)	<0.001
Atrial fibrillation	862 (20.6)	3,349 (30.1)	<0.001
Tobacco use	738 (17.7)	1,329 (11.9)	<0.001
Alcohol use	856 (20.5)	2,512 (22.6)	0.005
Elixhauser comorbidities,//, mean (SD)	3.1 (1.7)	3.1 (1.8)	0.02

Abbreviations: SD, standard deviation

*Data do not include cases who underwent combined surgical plus endovascular procedures.

†Data are presented as number (%) unless otherwise specified.

‡Self-reported by patients or their surrogates. Numbers do not sum to group totals because of missing race/ethnicity data in 2.8% of patients.

§Numbers do not sum to group totals because of missing payment-source data in <0.01% of patients.

//Numbers represent the number of Elixhauser comorbid conditions, which comprise a comprehensive set of 28 comorbidity measures for use with large administrative datasets.

Table 3

Characteristics of Patients with Unruptured Aneurysm, stratified by Type of Aortic Repair.*

Characteristic†	Endovascular (N = 47,248)	Surgery (N = 27,307)	P
SCI	51 (0.1)	55 (0.2)	<0.001
Age, mean (SD), y	74.4 (8.7)	67.5 11.9)	<0.001
Female	8,867 (18.8)	8,041 (29.5)	<0.001
Race‡			
White	39,345 (84.5)	21,669 80.8)	<0.001
Black	1,786 (3.8)	1,119 (4.2)	
Hispanic	2,967 (6.4)	2,140 (8.0)	
Asian	1,171 (2.5)	817 (3.0)	
Other	1,322 (2.8)	1,088 (4.0)	
Payment source§			
Medicare	38,719 (82.0)	17,056 (62.5)	<0.001
Medicaid	923 (2.0)	1,197 (4.4)	
Private	6,802 (14.3)	8,134 (29.8)	
Self-pay	229 (0.5)	294 (1.1)	
Other	574 (1.2)	624 (2.2)	
Hypertension	36,014 (76.2)	20,028 (73.3)	<0.001
Diabetes	9,441 (20.0)	5,530 (20.3)	0.40
Coronary heart disease	22,849 (48.4)	12,013 (44.0)	<0.001
Congestive heart failure	4,039 (8.6)	4,477 (16.4)	<0.001
Peripheral vascular disease	11,427 (24.2)	6,900 (25.3)	0.001
Chronic obstructive pulmonary disease	13,024 (27.6)	7,577 (27.8)	0.60
Chronic kidney disease	5,671 (12.0)	2,939 (10.8)	<0.001
Atrial fibrillation	6,850 (14.5)	7,638 (28.0)	<0.001
Tobacco use	13,240 (28.0)	6,164 (22.6)	<0.001
Alcohol use	9,031 (19.1)	5,652 (20.7)	<0.001
Elixhauser comorbidities,// mean (SD)	2.4 (1.5)	3.0 (1.7)	<0.001

Abbreviations: SD, standard deviation

*Data do not include cases who underwent combined surgical plus endovascular procedures.

†Data are presented as number (%) unless otherwise specified.

‡Self-reported by patients or their surrogates. Numbers do not sum to group totals because of missing race/ethnicity data in 1.8% of patients.

§Numbers do not sum to group totals because of missing payment-source data in <0.01% of patients.

//Numbers represent the number of Elixhauser comorbid conditions, which comprise a comprehensive set of 28 comorbidity measures for use with large administrative datasets.