Surgery for type A aortic dissection in patients with cerebral malperfusion: Results from the International Registry of Acute Aortic Dissection

Ibrahim Sultan, MD,[a,b] Valentino Bianco, DO, MPH,[a,b] Himanshu J. Patel, MD,[c] George J. Arnaoutakis, MD,[d] Marco Di Eusanio, MD, PhD,[e] Edward P. Chen, MD,[f] Bradley Leshnower, MD,[f] Thoralf M. Sundt, MD,[g] Udo Sechtem, MD,[h] Daniel G. Montgomery, BS,[i] Santi Trimarchi, MD, PhD,[j] Kim A. Eagle, MD,[i] and Thomas G. Gleason, MD, MS[a,b]

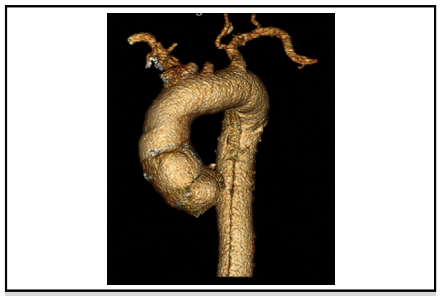
ABSTRACT

Background: The strategy for intervention remains controversial for patients presenting with type A aortic dissection (TAAAD) and cerebral malperfusion with neurologic deficit.

Methods: Surgically managed patients with TAAAD enrolled in the International Registry of Acute Aortic Dissection were evaluated to determine the incidence and prognosis of patients with cerebral malperfusion.

Results: A total of 2402 patients underwent surgical repair of TAAAD. Of these, 362 (15.1%) presented with cerebral malperfusion (CM) and neurologic deficits, and 2040 (84.9%) patients had no neurologic deficits at presentation. Patients with CM were more less likely to present with chest pain (66% vs 86.5%; $P < .001$) and back pain (35.9% vs 44.4%; $P = .008$). Patients with CM were more likely to present with syncope (48.4% vs 10.1%; $P < .001$), peripheral malperfusion (52.7% vs 38.0%; $P < .001$), and shock (16.2% vs 4.1%; $P < .001$). There was no difference in the incidence of Marfan syndrome (2.8% vs 3.0%; $P = .870$) or history of known aortic aneurysm (11.7% vs 13.9%; $P = .296$). Patients with CM were more likely to have a DeBakey I (63.8% vs 47.1%; $P < .001$) and a pericardial effusion (53.8% vs 40.6; $P < .001$) on presentation. There was no difference in total arch replacement (21.3% for CM vs 19.5% for no CM; $P = .473$). Patients with CM had an increased incidence of postoperative cerebrovascular accident (17.5% vs 7.2%; $P < .001$) and acute kidney injury (28.3% vs 18.1%; $P < .001$). In-hospital mortality was greater in patients with CM (25.7% vs 12.0%; $P < .001$).

Conclusions: Fifteen percent of patients with TAAAD presented with CM and neurologic deficits. Despite the fact that this subset of the population was older and more likely to present with peripheral malperfusion, cardiac tamponade, and in shock, in-hospital survival was noted in nearly 75% of the patients. Surgeons may continue to offer lifesaving surgery for TAAAD to this critically ill cohort of patients with acceptable morbidity and mortality. (J Thorac Cardiovasc Surg 2021;161:1713-20)



3D CTA of a patient with acute type A aortic dissection with bilateral occluded carotids.

CENTRAL MESSAGE

It is reasonable to perform surgery on patients with cerebral malperfusion and acute type A aortic dissection, as in-hospital outcomes are acceptable in this critically ill patient population.

PERSPECTIVE

Nearly 15% of patients with acute type A aortic dissection may present with neurocerebral malperfusion. These patients are more likely to present with shock, peripheral malperfusion, and cardiac tamponade. Despite these high-risk features, in-hospital survival was noted in nearly 75% of the cohort. These data support offering immediate surgery for select patients with cerebral malperfusion.

See Commentaries on pages 1721 and 1722.

From the [a]Division of Cardiac Surgery, Department of Cardiothoracic Surgery, University of Pittsburgh, Pittsburgh, Pa; [b]Heart and Vascular Institute, University of Pittsburgh Medical Center, Pittsburgh, Pa; [c]Department of Cardiac Surgery and [i]Cardiovascular Center, University of Michigan, Ann Arbor, Mich; [d]Division of Thoracic and Cardiovascular Surgery, University of Florida, Gainesville, Fla; [e]Cardiac Surgery Department, University of Bologna, Bologna, Italy; [f]Division of Cardiothoracic Surgery, Emory University, Atlanta, Ga; [g]Thoracic Aortic Center, Massachusetts General Hospital, Boston, Mass; [h]Division of Cardiology, Robert-Bosch Krankenhaus, Stuttgart, Germany; and [j]Department of Surgery, Fondazione IRCCS Cà Granda Ospedale Maggiore Policlinico Milan, Milan, Italy.

IRAD research is generously supported by W.L. Gore & Associates, Inc, Medtronic, the Varbedian Aortic Research Fund, the Hewlett Foundation, the Mardigian Foundation, UM Faculty Group Practice, Terumo, and Ann and Bob Aikens.

Received for publication July 1, 2019; revisions received Nov 5, 2019; accepted for publication Nov 6, 2019; available ahead of print Nov 15, 2019.

Address for reprints: Ibrahim Sultan, MD, Division of Cardiac Surgery, 5200 Centre Ave, Suite 715, Pittsburgh, PA 15232 (E-mail: sultani@upmc.edu).

0022-5223/$36.00

https://doi.org/10.1016/j.jtcvs.2019.11.003

Abbreviations and Acronyms

CM = cerebral malperfusion
CVA = cerebrovascular accident
IRAD = International Registry of Acute Aortic Dissection
TAAAD = type A acute aortic dissection

Scanning this QR code will take you to the table of contents to access supplementary information.

Type A acute aortic dissection (TAAAD) is a surgical emergency, with the goal of establishing true lumen flow by resecting the primary tear. However, surgical decision-making is often complicated by the potential futility of operating on patients with irreversible cerebral malperfusion (CM). End-organ malperfusion occurs in 16% to 34% of patients with TAAAD and has been shown to adversely impact outcomes.[1-6] According to previously reported data from the International Registry of Acute Aortic Dissection (IRAD), 6% of patients with TAAAD present with CM.[7,8] Given limited data composed primarily of small, single-center studies, there is still considerable debate regarding the optimal management of patients with TAAAD presenting with CM.[9-14]

Previous surgical data have reported poor outcomes for patients undergoing cardiac or aortic surgery when presenting with acute stroke.[6,15] This includes an increase in both postoperative morbidity and mortality. However, some single-center studies have reported reasonable long-term survival among patients who survive to hospital discharge.[9,13] The aim of the current study is to report management and outcomes, using the interventional cohort of IRAD data, for patients with TAAAD and CM compared with patients without cerebral malperfusion.

METHODS

IRAD is a research consortium established in 1996 composed of 55 academic centers in 12 countries with the primary goal of providing robust data on patient presentation, evaluation, management, and outcomes for aortic dissection. The interventional cohort was founded in 2010 in an effort to obtain granular data on the surgical, endovascular, and hybrid procedures performed during the index hospitalization. Thirty-six of the 55 centers participate in the interventional cohort at this time. The aim of the current study was to elucidate patient outcomes, based on a large multicenter international data sample, comparing patients who presented with TAAAD and CM with patients without CM, to determine the incidence of CM and its impact on patient prognosis.

TABLE 1. Baseline characteristics of patients with TAAAD undergoing surgery

Characteristic	Cerebral malperfusion	No cerebral malperfusion	*P* value
Age	62.1 (52.3-70.1)	61.3 (51.0-71.2)	.475
Sex, female	138 (38.1%)	686 (33.6%)	.096
Race, nonwhite	64 (19.4%)	445 (24.2%)	.059
History of hypertension	255 (76.1%)	1437 (75.2%)	.718
Diabetes	29 (9.0%)	199 (10.8%)	.335
Marfan syndrome	9 (2.8%)	55 (3.0%)	.870
Atherosclerosis	50 (15.6%)	261 (14.4%)	.555
Known aortic aneurysm	38 (11.7%)	256 (13.9%)	.296
Mitral valve disease	6 (2.3%)	44 (2.8%)	.689
Bicuspid aortic valve	16 (5.0%)	65 (3.6%)	.220
Aortic valve disease	33 (10.3%)	174 (9.6%)	.679
Other aortic disease	4 (1.3%)	27 (1.5%)	1.000
Cocaine abuse	4 (1.3%)	48 (2.7%)	.168
Current smoker	70 (33.8%)	450 (32.0%)	.603
Family history aortic disease	23 (10.3%)	116 (8.2%)	.280
Previous cardiac surgery	35 (10.7%)	214 (11.6%)	.650
CABG	9 (2.8%)	79 (4.3%)	.200
Aneurysm/dissection surgery	18 (5.6%)	94 (5.1%)	.756
AVR	15 (4.6%)	70 (3.8%)	.500
MVR	6 (1.9%)	13 (0.7%)	.042

CABG, Coronary artery bypass grafting; *AVR*, aortic valve replacement; *MVR*, mitral valve replacement.

Study Population

The total study population consisted of 2402 patients from whom data were collected through the IRAD consortium from 2010 to 2017. CM was defined by clinical presentation at the time of TAAAD with clear evidence of neurologic deficit on physical examination. All patients with preoperative neurologic deficits were included in the CM group, which consisted of 362 (15.1%) patients. A total of 2040 patients did not have any neurologic deficits at presentation and were therefore considered to not have CM.

Statistical Analysis

Descriptive statistics are presented as median ± standard deviation for continuous and parametric variables and frequency (percentage) for categorical variables. Continuous variables were assessed for normality. If non-normal, data were compared by nonparametric tests. Logistic regression for all factors previously demonstrated to be associated with mortality in TAAAD including CM was used to identify independent predictors of death.[11]

RESULTS

Baseline Characteristics

Patients with CM were more likely to have a history of previous mitral valve surgery (1.9% vs 0.7%; $P = .042$) (Table 1). There was no difference in the incidence of Marfan syndrome (2.8% vs 3.0%; $P = .870$) or history of known aortic aneurysm (11.7% vs 13.9%; $P = .296$),

TABLE 2. Presenting signs and symptoms of patients with TAAAD undergoing surgery

Characteristic	Cerebral malperfusion	No cerebral malperfusion	*P* value
Chest pain	214 (66.0%)	1676 (86.5%)	<.001
Pain in head or neck	67 (23.7%)	465 (27.7%)	.161
Back pain	102 (35.9%)	772 (44.4%)	.008
Abdominal pain	77 (27.5%)	428 (25.4%)	.460
Migrating pain	26 (10.0%)	206 (13.0%)	.174
Radiating pain	71 (27.0%)	672 (40.9%)	<.001
Severe or worst ever pain	187 (90.8%)	1325 (90.9%)	.962
Abrupt onset of pain	235 (79.7%)	1490 (82.2%)	.297
Syncope	153 (48.4%)	175 (10.1%)	<.001
Presenting hemodynamics			
Hypertensive	64 (18.5%)	538 (28.3%)	<.001
Normotensive	144 (41.6%)	966 (50.7%)	.002
Hypotensive	64 (18.5%)	301 (15.8%)	.211
Shock	56 (16.2%)	78 (4.1%)	<.001
Murmur of aortic insufficiency	88 (33.8%)	474 (31.3%)	.416
Pulse deficits present	109 (52.7%)	406 (38.0%)	<.001
CVA/stroke	114 (34.8%)	0 (0.0%)	<.001
Coma/altered consciousness	193 (56.3%)	0 (0.0%)	<.001

CVA, Cerebrovascular accident.

TABLE 3. Imaging characteristics of patients with TAAAD undergoing surgery

Characteristic	Cerebral malperfusion	No cerebral malperfusion	*P* value
True IMH	5 (1.6%)	59 (3.5%)	.085
Aneurysm	163 (61.7%)	881 (64.6%)	.369
Site of most proximal extension			
Root	188 (66.7%)	931 (62.7%)	.200
STJ	31 (11.0%)	192 (12.9%)	.371
Ascending	58 (20.6%)	323 (21.7%)	.662
Arch	5 (1.8%)	30 (2.0%)	.786
Site of most distal extension			
Ascending	18 (7.0%)	157 (11.4%)	.038
Arch	36 (14.1%)	263 (19.1%)	.056
LSC	9 (3.5%)	49 (3.6%)	.974
Descending	51 (19.9%)	243 (17.6%)	.382
Abdominal (any)	82 (32.0%)	397 (28.8%)	.298
False lumen patency			
Patent	133 (71.9%)	565 (68.1%)	.311
Partial thrombosis	30 (16.2%)	179 (21.6%)	.104
Complete thrombosis	22 (11.9%)	86 (10.4%)	.542
Site of intimal tear			
Ascending	71 (46.1%)	414 (53.7%)	.085
Arch	17 (11.0%)	58 (7.5%)	.144
Descending	5 (3.2%)	19 (2.5%)	.577
Arch vessel involvement	146 (63.8%)	525 (47.1%)	<.001
LSC	35 (61.4%)	128 (58.4%)	.686
LCC	33 (58.9%)	122 (56.2%)	.715
Brachiocephalic	41 (77.4%)	173 (79.0%)	.794
Pericardial effusion	133 (53.8%)	529 (40.6%)	<.001
Periaortic hematoma	51 (25.2%)	180 (17.5%)	.009
Maximum diameter			
Annulus	2.5 (2.3-2.8)	2.5 (2.3-2.7)	.722
Root	4.0 (3.6-4.6)	4.2 (3.7-4.9)	.296
STJ	4.0 (3.5-4.6)	3.9 (3.3-4.6)	.407
Ascending	5.0 (4.5-5.5)	5.0 (4.4-5.6)	.635
Arch	3.7 (3.1-4.1)	3.6 (3.1-4.1)	.486
Descending	3.2 (2.8-3.7)	3.2 (2.8-3.6)	.685

IMH, Intramyocardial hemorrhage; *STJ*, sinotubular junction; *LSC*, left subclavian artery; *LCC*, left carotid artery.

hypertension (76.1% vs 75.2%; $P = .718$), diabetes (9.0% vs 10.8%; $P = .335$), atherosclerosis (15.6% vs 14.4%; $P = .555$), bicuspid aortic valve (5.0% vs 3.6%; $P = .220$), current smoker status (33.8% vs 32.0%; $P = .603$), or family history of aortic disease (10.3% vs 8.2%; $P = .280$).

Presenting Signs and Symptoms

Patients with CM were less likely to present with chest pain, back pain, radiating pain, and hypertension (Table 2). Patients with CM were much more likely to present with syncope, with concomitant pulse deficits, and in shock. There was no difference in the presentation of head or neck pain (23.7% vs 27.7%; $P = .161$), abdominal pain (27.5% vs 25.4%; $P = .460$), migrating pain (10% vs 13%; $P = .174$), hypotension (18.5% vs 15.8%; $P = .211$), or aortic insufficiency (33.8% vs 31.3%; $P = .416$).

Imaging and Operative Findings

Patients with CM were more likely to have a DeBakey I dissection (63.8% vs 47.1%; $P < .001$), a periaortic hematoma (25.2% vs 17.5%; $P = .009$), and a pericardial effusion (53.8% vs 40.6; $P < .001$) on presentation (Table 3). There was no difference in total arch replacement (21.3% for CM vs 19.5% for no CM; $P = .473$) (Table 4). There was also no difference in hemiarch replacement (51.5% vs 55.8%; $P = .163$) or partial arch replacement (10.8% vs 9.7%; $P = .590$). A greater proportion of patients without CM had valve-sparing aortic root replacement (15.0% vs 20.6%; $P = .031$). The use of antegrade (64.6% vs 62.3%; $P = .505$) and retrograde (35.4% vs 37.7%; $P = .505$) cerebral perfusion were similar for patients with and without CM. Patients in the CM cohort were on average cooled to a lower mean temperature during circulatory arrest (20.0°C [17.4-25.0] vs 23.0°C [18.0-26.5]; $P < .001$).

TABLE 4. Intraoperative characteristics of patients with TAAAD undergoing surgery

Characteristic	Cerebral malperfusion	No cerebral malperfusion	*P* value
Crossclamp	171 (52.1%)	1090 (58.2%)	.040
Aortic procedures			
Noncoronary sinus replacement	21 (7.8%)	109 (7.4%)	.815
Valve sparing	41 (15.0%)	316 (20.6%)	.031
Commissural resuspension	92 (33.8%)	599 (38.3%)	.164
Bentall	78 (34.8%)	362 (29.4%)	.103
Ascending replacement	260 (81.8%)	1508 (83.6%)	.419
Hemiarch	158 (51.5%)	973 (55.8%)	.163
Partial arch	30 (10.8%)	152 (9.7%)	.590
Complete arch	63 (21.3%)	323 (19.5%)	.473
Single arterial button	12 (4.5%)	75 (5.1%)	.656
Branched graft	31 (11.3%)	142 (9.4%)	.317
Elephant trunk	11 (4.1%)	91 (5.9%)	.225
Coronary ostium repair	34 (12.4%)	217 (14.2%)	.422
Concomitant CABG	41 (13.9%)	206 (12.4%)	.456
AVR	106 (37.5%)	487 (30.9%)	.028
Extracorporeal circulation			
Arterial line (cooling)			
R. axillary	93 (35.1%)	663 (43.8%)	.008
L. axillary	4 (1.5%)	16 (1.1%)	.524
R. femoral	102 (38.5%)	465 (30.7%)	.012
L. femoral	43 (16.2%)	172 (11.4%)	.025
Apex left ventricle	1 (0.4%)	4 (0.3%)	.554
Aorta	35 (13.2%)	196 (13.0%)	.910
Carotid	3 (1.1%)	11 (0.7%)	.452
Innominate	4 (1.5%)	57 (3.8%)	.067
Graft	2 (0.8%)	14 (0.9%)	1.000
Hypothermic circulatory arrest	280 (89.5%)	1529 (88.2%)	.532
EEG monitoring	64 (22.5%)	407 (25.5%)	.272
Cerebral perfusion	247 (78.9%)	1477 (83.2%)	.068
Antegrade	148 (64.6%)	867 (62.3%)	.505
Retrograde	81 (35.4%)	524 (37.7%)	.505
Cerebral perfusion time, min	35.0 (24.0-50.0)	34.0 (23.0-46.0)	.500
Cerebral ischemia time, min	9.0 (3.0-30.5)	5.0 (0.0-24.0)	.001
Visceral ischemia time, min	45.5 (33.0-58.3)	36.0 (29.0-47.0)	.008
Clamping time, min	117.5 (85.0-152.0)	116.0 (80.8-161.0)	.706
Cardiac arrest time, min	35.0 (26.5-66.0)	38.5 (25.0-95.0)	.233
Total CPB time, min	197.0 (153.0-247.0)	192.0 (150.0-240.0)	.235
Minimum temperature	20.0 (17.4-25.0)	23.0 (18.0-26.5)	<.001

CABG, Coronary artery bypass grafting; *AVR*, aortic valve replacement; *R.*, right; *L.*, left; *EEG*, electroencephalogram; *CPB*, cardiopulmonary bypass.

Postoperative Outcomes

In-hospital mortality was significantly greater for the CM group (25.7% vs 12.0%; $P < .001$) (Table 5). Patients with CM had an increased incidence of postoperative cerebrovascular accidents (CVAs) (17.5% vs 7.2%; $P < .001$), coma (9.3% vs 2.6%; $P < .001$), acute renal failure (28.3% vs 18.1%; $P < .001$), limb ischemia (19.8% vs 8.5%; $P < .001$), hypotension (15.5% vs 10.8%; $P = .014$), cardiac tamponade (9.2% vs 6.2%; $P = .045$), and prolonged ventilation (34.5% vs 22.8%; $P < .001$). Kaplan–Meier estimates at 1 year indicate lower survival in the CM cohort (62.6% vs 81.3%; $P < .001$) (Figure E1).

Multivariable Analysis for Predictors of Mortality

Multivariable logistic regression analysis for patients with CM showed that predictors of death in this cohort included preoperative CM, age >70 years, previous cardiac surgery, perioperative myocardial ischemia, tamponade, pulse deficit (peripheral malperfusion), and extension of dissection into the sinus segment (Figure 1 and Table 6).

DISCUSSION

The outcomes presented herein are based on international data from a number of aortic centers around the world and represent the largest study to date assessing the impact of CM at the time of presentation for patients with TAAAD.

TABLE 5. In-hospital outcomes of patients with TAAAD undergoing surgery

Characteristic	Cerebral malperfusion	No cerebral malperfusion	*P* value
Neurologic deficit			
CVA	50 (17.5%)	131 (7.2%)	<.001
Coma	26 (9.3%)	47 (2.6%)	<.001
SCI	2 (0.7%)	32 (1.8%)	.306
Transient neurologic deficit	38 (45.2%)	139 (57.4%)	.053
Myocardial ischemia	6 (3.8%)	28 (3.7%)	.939
Myocardial infarction	9 (5.6%)	22 (2.8%)	.074
Mesenteric ischemia/ infarction	14 (4.3%)	53 (2.7%)	.115
Acute renal failure requiring dialysis	94 (28.3%)	357 (18.1%)	<.001
Extension of dissection	8 (2.5%)	47 (2.4%)	.932
Hypotension	50 (15.5%)	210 (10.8%)	.014
Cardiac tamponade	30 (9.2%)	122 (6.2%)	.045
Limb ischemia	64 (19.8%)	164 (8.5%)	<.001
Respiratory insufficiency (prolonged ventilation)	59 (34.5%)	268 (22.8%)	.001
Bleeding requiring re-thoracotomy	14 (8.9%)	74 (6.5%)	.264
Mortality	93 (25.7%)	244 (12.0%)	<.001

CVA, Cerebrovascular accident; *SCI*, spinal cord injury.

We found that 15.1% of patients with TAAAD presented with clinical signs and symptoms of CM. Those presenting with CM were more likely to present with syncope, rather than chest or back pain, which was more common in patients without CM. This may be because of altered consciousness in the CM cohort. This in turn may pose a diagnostic challenge to emergency department physicians attempting to diagnose patients with TAAAD. These patients may continue to go on with malperfusion and end-organ dysfunction because of a delayed diagnosis Although patients with CM had an increased, albeit acceptable, postoperative mortality when compared with patients without CM, it is important to consider that these patients were more likely to present in shock and with pericardial effusions.

Despite this critically ill presentation, nearly three quarters of the patients who underwent surgical repair survived hospital discharge. Moreover, IRAD data from the previous decade reported a 25.1% overall hospital mortality for all-comers, which is nearly identical to the CM group in this study likely related to advancements in cerebral protection and better selection of operative candidates.[16] However, the decision to surgically intervene in patients with CM remains controversial, as survival to hospital discharge may not translate to long-term survival in this cohort.[2] In our selected group, there appears to be a clear difference in mortality within the first month but no additional ''drop off'' after a month, indicating that survivors at a month are likely going to continue to live without any major mortality risk. However, the quality of life and readmission status are not something to which we have access and could very well be poor in the survivors.

Critical malperfusion of organs or peripheral malperfusion occur in approximately 34% of patients with TAAAD, often complicating the management of this challenging patient population.[3] Both the number of organs involved and the severity of malperfusion play a role in determining patient outcomes.[6] Numerous series have reported worse outcomes for patients with TAAAD who present with visceral or peripheral malperfusion[2,3,17-21] and prognosis is largely determined by the patients overall clinical status at the time of presentation.[1,16]

Dissections of the proximal aorta often involve the supra-aortic branches and consequently lead to CM with neurologic deficits, which has an incidence of 6% to 20%.[7,20,22,23] Previous literature on CM in patients with TAAAD is limited and consists largely of single-center studies and case reports.[9,13,24-29] There are currently 2 previous large international registry series that address the issue of CM in TAAAD.[7,8] These were focused on presenting signs and symptoms, whereas our data have critical information on type of surgical intervention and in postoperative outcomes. Bossone and colleagues[7] found that stroke was present on arrival in 6% of patients presenting with TAAAD. On presentation, syncope was more common and chest pain was less common in stroke patients, which is consistent with our current study. In-hospital mortality was significantly greater for patients with stroke (42.4% vs 24.1%; $P < .001$). Di Eusanio and colleagues[8] reported that 4.7% of patients presented with CVA and 2.9% of patients had coma at presentation for TAAAD. Hospital mortality was significantly greater for patients with CVA (40.2%) and coma (63.0%) compared with patients without CVA or coma (22.7%; $P < .001$). Similar to our results, both studies showed that patients with CM were more likely to present in shock and with arch vessel involvement. Interestingly, both studies showed that patients with CM were significantly less likely than patients without CM to undergo surgical management of TAAAD. Yet, mortality was 3- to 4-fold greater for medical management of stroke, CVA, and coma, and good long-term survival was observed in patients with CM who underwent surgical management.[7,8] In this series of surgically managed patients, mortality was acceptable. However, it is important to note that this is among patients who were deemed eligible for repair, which comes with bias from the surgeon. This report only contains the patients with TAAAD and CM who did undergo surgery. The interventional cohort that these data are derived from do not contain

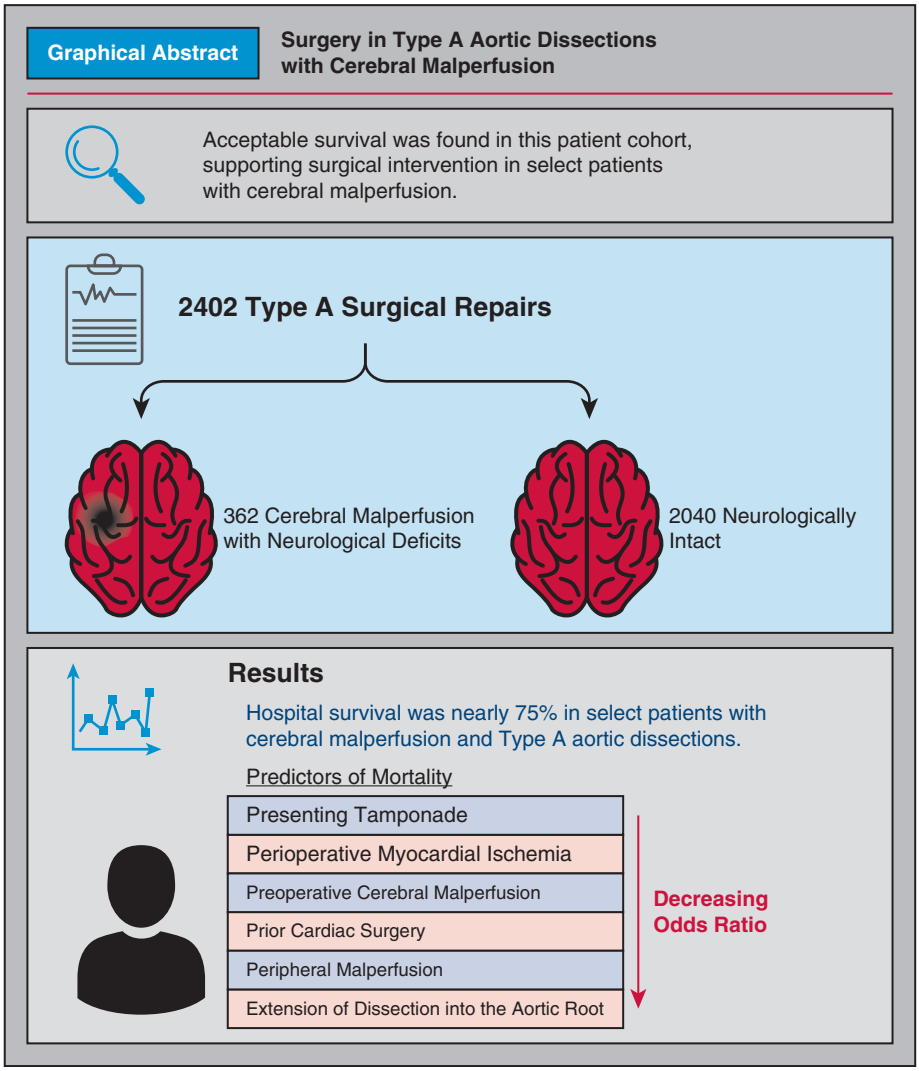


FIGURE 1. Predictors of mortality (presenting tamponade, perioperative myocardial ischemia and cerebral malperfusion, previous cardiac surgery, peripheral malperfusion extension of dissection into the aortic root) and acceptable rate of survival (75%) in patients with cerebral malperfusion.

granular data on the nonoperative cohort. It is certainly likely that these patients who presented with CM were the ''sickest'' of the cohort and may not have been offered surgery.

Early organ reperfusion is critical in patients with TAAAD, and immediate surgical repair has been shown to improve outcomes in patients with malperfusion.[13,30-32] Time from presentation of stroke to surgical intervention is directly associated with outcomes, and intervention within 10 hours is pivotal.[13] The gold standard of care for TAAAD is immediate surgery for central aortic repair. However, recent evidence has supported the potential efficacy of rapid reperfusion strategies in carefully selected patients followed by central repair for select cases of severe visceral malperfusion.[32] However, this is unlikely to be used in the setting of CM, which may involve fenestrating the aortic arch, which may carry with it more risk than benefit in this population. Multiple centers have adopted novel methods of limiting CM time, including techniques for direct carotid perfusion, or aggressive revascularization of the carotid arteries,[33,34] percutaneous endovascular right carotid and brachiocephalic artery stenting followed by central repair,[35] and direct surgical fenestration of the carotid and/or brachiocephalic arteries.[31] The right axillary approach can be quite effective to use in most patients because it will allow for uninterrupted ACP during arch reconstruction. This was underused in the CM cohort and may have had some distinct advantages if used appropriately. Some IRAD centers have been aggressive about carotid reconstruction not only for CM but in the setting of

TABLE 6. Factors associated with death in the study cohort

Characteristic	OR	95% CI lower	95% CI upper	*P* value
Preoperative cerebral malperfusion	2.626	1.234	5.587	.012
Age >70 y	1.704	0.938	3.098	.080
Previous cardiac surgery	2.228	1.069	4.645	.033
Pre- or postoperative myocardial ischemia/infarction	3.250	1.798	5.874	<.001
Presenting tamponade	5.291	1.635	17.124	.005
Pulse deficits	2.105	1.213	3.652	.008
Site of most proximal extension in the aortic root	1.932	1.111	3.361	.020

OR, Odds ratio; *CI*, confidence interval.

radiologic evidence of malperfusion of the carotid arteries with the goal of restoring normal flow to the brain.[34] This may explain the greater rates of total arch replacement in patients who do not have CM or a hard clinical indication for total arch replacement. All of these approaches share the common goal of reducing cerebral ischemic time, which is one of the most critical determinants of recovery for patients with TAAAD and CM.

Limitations

IRAD data are based on the retrospective collection of outcomes from numerous aortic centers internationally and patients are carefully searched for and data are carefully sought using standardized definitions. However, there is a possibility that not all cases are entered from all centers. As such, this study is potentially limited by human error in data collection. Loss to follow-up for long-term outcomes is also a concern, as a certain percentage of patients will present to different centers on readmission. It is important to note that the data collected for IRAD are based on patients who presented to one of the tertiary-level participating IRAD centers. Therefore, a subset of critically ill patients who either were not transferred or died of TAAAD in transit to a tertiary center were not included in the data, so the generalizability of our findings to all hospitals is not possible. Finally, there is significant heterogeneity in the operative and cerebral protection strategy between institutions for patients with or without CM. Despite our cumulative data suggesting no difference between CM and non-CM cohorts, this is unlikely to be the case at the institutional level.

CONCLUSIONS

CM occurred in 15% of patients who presented with TAAAD. Although this subset of the TAAAD population was more likely to present with peripheral malperfusion, cardiac tamponade, and in shock, in-hospital survival was seen in nearly 75% of these patients. It is reasonable to offer such lifesaving surgery for selected patients who present with TAAAD complicated by CM, as outcomes indicate acceptable morbidity and mortality.

Conflict of Interest Statement

Dr Patel: W. L. Gore & Associates, Inc, Medtronic. Dr Leshnower: Medtronic, Inc. Dr Eagle: W. L. Gore & Associates, Inc, Medtronic, Terumo. Dr Gleason: Medical Advisory Board, Abbott and Medtronic Consultant, Principal investigator clinical trial for Cytosorbants. Institutional research support: Medtronic, Boston Scientific. All authors have nothing to disclose with regard to commercial support.

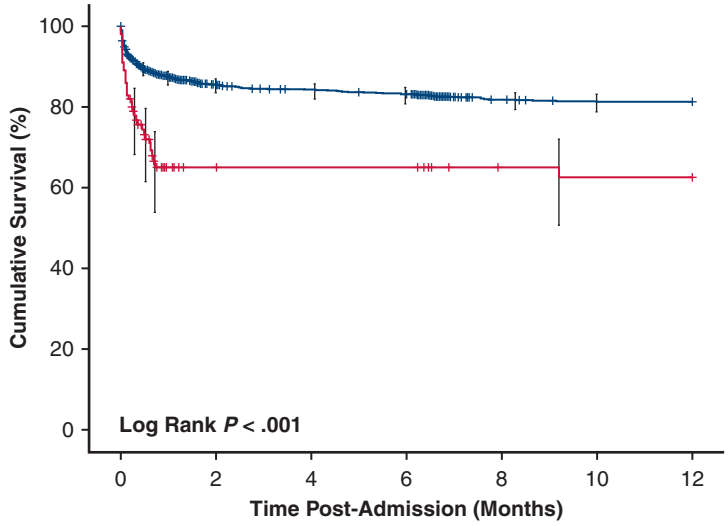
The authors thank Elise Woznicki for her invaluable support with the study.

References

1. Berretta P, Trimarchi S, Patel HJ, Gleason TG, Eagle KA, Di Eusanio M. Malperfusion syndromes in type A aortic dissection: what we have learned from IRAD. *J Vis Surg*. 2018;4:65.
2. Geirsson A, Szeto WY, Pochettino A, McGarvey ML, Keane MG, Woo YJ, et al. Significance of malperfusion syndromes prior to contemporary surgical repair for acute type A dissection: outcomes and need for additional revascularizations. *Eur J Cardiothorac Surg*. 2007;32:255-62.
3. Czerny M, Schoenhoff F, Etz C, Englberger L, Khaladj N, Zierer A, et al. The impact of pre-operative malperfusion on outcome in acute type A aortic dissection: results from the GERAADA registry. *J Am Coll Cardiol*. 2015;65:2628-35.
4. Narayan P, Rogers CA, Benedetto U, Caputo M, Angelini GD, Bryan AJ. Malperfusion rather than merely timing of operative repair determines early and late outcome in type A aortic dissection. *J Thorac Cardiovasc Surg*. 2017;154:81-6.
5. Sultan I, Wallen TJ, Habertheuer A, Siki M, Arnaoutakis GJ, Bavaria J, et al. Concomitant antegrade stent grafting of the descending thoracic aorta during transverse hemiarch reconstruction for acute DeBakey I aortic dissection repair improves aortic remodeling. *J Card Surg*. 2017;32:581-92.
6. Vallabhajosyula P, Gottret JP, Menon R, Sultan I, Abbas Z, Siki M, et al. Central repair with antegrade TEVAR for malperfusion syndromes in acute DeBakey I aortic dissection. *Ann Thorac Surg*. 2017;103:748-55.
7. Bossone E, Corteville DC, Harris KM, Suzuki T, Fattori R, Hutchison S, et al. Stroke and outcomes in patients with acute type A aortic dissection. *Circulation*. 2013;128:S175-9.
8. Di Eusanio M, Patel HJ, Nienaber CA, Montgomery DM, Korach A, Sundt TM, et al. Patients with type A acute aortic dissection presenting with major brain injury: should we operate on them? *J Thorac Cardiovasc Surg*. 2013;145:213-21.e1.
9. Tanaka H, Okada K, Yamashita T, Morimoto Y, Kawanishi Y, Okita Y. Surgical results of acute aortic dissection complicated with cerebral malperfusion. *Ann Thorac Surg*. 2005;80:72-6.
10. Piccione W Jr, Hamilton IN, Najafi H. Intentional delayed repair of acute dissection of the ascending aorta complicated by stroke. *J Thorac Cardiovasc Surg*. 1995;109:807-8.
11. Pocar M, Passolunghi D, Moneta A, Mattioli R, Donatelli F. Coma might not preclude emergency operation in acute aortic dissection. *Ann Thorac Surg*. 2006;81:1348-51.

12. Tsukube T, Hayashi T, Kawahira T, Haraguchi T, Matsukawa R, Kozawa S, et al. Neurological outcomes after immediate aortic repair for acute type A aortic dissection complicated by coma. *Circulation*. 2011;124:S163-7.
13. Estrera AL, Garami Z, Miller CC, Porat EE, Achouh PE, Dhareshwar J, et al. Acute type A aortic dissection complicated by stroke: can immediate repair be performed safely? *J Thorac Cardiovasc Surg*. 2006;132:1404-8.
14. Sultan I, Szeto WY. Decision making in acute DeBakey I aortic dissection: balancing extensive arch reconstruction versus mortality. *J Thorac Cardiovasc Surg*. 2016;151:349-50.
15. Kilic A, Sultan IS, Arnaoutakis GJ, Higgins RS, Kilic A. Assessment of thoracic endografting operative mortality risk score: development and validation in 2,000 patients. *Ann Thorac Surg*. 2015;100:860-7.
16. Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Mehta RH, et al. Contemporary results of surgery in acute type A aortic dissection: the international registry of acute aortic dissection experience. *J Thorac Cardiovasc Surg*. 2005;129:112-22.
17. Pacini D, Leone A, Belotti LM, Fortuna D, Gabbieri D, Zussa C, et al. Acute type A aortic dissection: significance of multiorgan malperfusion. *Eur J Cardiothorac Surg*. 2013;43:820-6.
18. Girardi LN, Krieger KH, Lee LY, Mack CA, Tortolani AJ, Isom OW. Management strategies for type A dissection complicated by peripheral vascular malperfusion. *Ann Thorac Surg*. 2004;77:1309-14; discussion 14.
19. Kawahito K, Kimura N, Yamaguchi A, Aizawa K. Malperfusion in type A aortic dissection: results of emergency central aortic repair. *Gen Thorac Cardiovasc Surg*. 2019;67:594-601.
20. Girdauskas E, Kuntze T, Borger MA, Falk V, Mohr FW. Surgical risk of preoperative malperfusion in acute type A aortic dissection. *J Thorac Cardiovasc Surg*. 2009;138:1363-9.
21. Sultan I, McGarvey J, Vallabhajosyula P, Desai ND, Bavaria JE, Szeto WY. Routine use of hemiarch during acute type A aortic dissection repair. *Ann Cardiothorac Surg*. 2016;5:245-7.
22. Rylski B, Urbanski PP, Siepe M, Beyersdorf F, Bachet J, Gleason TG, et al. Operative techniques in patients with type A dissection complicated by cerebral malperfusion. *Eur J Cardiothorac Surg*. 2014;46:156-66.
23. Kruger T, Weigang E, Hoffmann I, Blettner M, Aebert H. Cerebral protection during surgery for acute aortic dissection type A: results of the German registry for acute aortic dissection type A (GERAADA). *Circulation*. 2011;124:434-43.
24. Flemming KD, Brown RD Jr. Acute cerebral infarction caused by aortic dissection: caution in the thrombolytic era. *Stroke*. 1999;30:477-8.
25. Shimazaki Y, Minowa T, Watanabe T, Koshika M, Toyama H, Inui K. Acute aortic dissection with new massive cerebral infarction—a successful repair with ligature of the right common carotid artery. *Ann Thorac Cardiovasc Surg*. 2004;10:64-6.
26. Iliescu VA, Dorobantu LF, Stiru O, Bubenek S, Miclea I, Rugina M, et al. Combined cardiac-neurosurgical treatment of acute aortic dissection, stroke, and coma. *Tex Heart Inst J*. 2008;35:200-2.
27. Veyssier-Belot C, Cohen A, Rougemont D, Levy C, Amarenco P, Bousser MG. Cerebral infarction due to painless thoracic aortic and common carotid artery dissections. *Stroke*. 1993;24:2111-3.
28. Roseborough GS, Murphy KP, Barker PB, Sussman M. Correction of symptomatic cerebral malperfusion due to acute type I aortic dissection by transcarotid stenting of the innominate and carotid arteries. *J Vasc Surg*. 2006;44:1091-6.
29. Morimoto N, Okada K, Okita Y. Lack of neurologic improvement after aortic repair for acute type A aortic dissection complicated by cerebral malperfusion: predictors and association with survival. *J Thorac Cardiovasc Surg*. 2011;142:1540-4.
30. Chiu P, Tsou S, Goldstone AB, Louie M, Woo YJ, Fischbein MP. Immediate operation for acute type A aortic dissection complicated by visceral or peripheral malperfusion. *J Thorac Cardiovasc Surg*. 2018;156:18-24.e3.
31. Uchida K, Karube N, Kasama K, Minami T, Yasuda S, Goda M, et al. Early reperfusion strategy improves the outcomes of surgery for type A acute aortic dissection with malperfusion. *J Thorac Cardiovasc Surg*. 2018;156:483-9.
32. Goldberg JB, Lansman SL, Kai M, Tang GHL, Malekan R, Spielvogel D. Malperfusion in type A dissection: consider reperfusion first. *Semin Thorac Cardiovasc Surg*. 2017;29:181-5.
33. Urbanski PP, Wagner M. Perfusion and repair technique in acute aortic dissection with cerebral malperfusion and damage of the innominate artery. *J Thorac Cardiovasc Surg*. 2012;144:982-4.
34. Trivedi D, Navid F, Balzer JR, Joshi R, Lacomis JM, Jovin TG, et al. Aggressive aortic arch and carotid replacement strategy for type A aortic dissection improves neurologic outcomes. *Ann Thorac Surg*. 2016;101:896-903; discussion 903-5.
35. Heran MKS, Balaji N, Cook RC. Novel percutaneous treatment of cerebral malperfusion prior to surgery for acute type A dissection. *Ann Thorac Surg*. 2019;108:e15-7.

Key Words: aortic dissection, type A, cerebral malperfusion, International Registry for Acute Aortic Dissection



N at risk							
No pre-operative CVA or coma	1962	865	837	823	604	596	595
Pre-operative CVA or coma	100	34	33	33	26	25	24

FIGURE E1. Kaplan–Meier curves demonstrating reduced survival in patients undergoing surgery for type A acute aortic dissection after presenting with cerebral malperfusion. *CVA*, Cerebrovascular accident.