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Outcomes of carotid artery replacement with total arch reconstruction for type A aortic dissection

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Abstract

Background: Cerebral malperfusion and carotid artery dissection in patients with acute type A aortic dissections (TAAD) carry high morbidity and mortality. There are limited data on outcomes of concomitant carotid artery replacement with total arch replacement in the setting of a TAAD.

Methods: All patients with acute TAAD who underwent a total arch replacement (TAR) between 2007 and 2018 were included. Data were retrospectively collected from a prospectively maintained database. Baseline variables were compared, and Kaplan-Meier estimates were used for long term survival. Cox multivariable regression analysis was used to identify predictors of mortality.

Results: A total of 161 patients underwent TAR for acute TAAD. Of these, 111 underwent conventional total arch reconstruction, while 50 had a concomitant carotid artery replacement. Baseline characteristics were similar between both cohorts apart from the carotid replacement cohort having a higher rate of preoperative cerebral malperfusion (48% vs 10.81%; $p < 0.01$) and pre-operative stroke (28% vs 11.71%; $p = 0.02$). There was no difference in (operative) 30-day mortality between the carotid replacement and conventional TAR groups (22% vs 18.9%; $p = 0.81$), 1-year mortality (28% vs 27.9%; $p = 0.99$) or 5-year (32% vs 29.7%; $p = 0.917$) mortality. Postoperative stroke was 0% vs 4.5% ($p = 0.301$) for the carotid vs conventional TAR cohort.

Conclusions: Concomitant carotid artery replacement is a feasible and safe technique to address perioperative cerebral malperfusion, carotid dissection and neurologic dysfunction associated with carotid artery dissection, with no difference in long term survival or postoperative stroke when compared to conventional TAR.

Graphical Abstract

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Keywords

Type A Dissection; malperfusion; carotid artery replacement; total arch replacement; aorta

The incidence of type A aortic dissection has remained consistent at approximately 3.5 per 100,000 person years over the years [1,2]. Malperfusion in acute TAAD has been consistently shown to be a major determinant of morbidity and mortality[3,4]. Cerebral malperfusion, which has been reported in 6-15% of patients with TAAD in the International Registry of Acute Aortic Dissections (IRAD), has been reported to have worse outcomes compared to those without cerebral malperfusion[5–7]. Cerebral malperfusion in TAAD, which may present as stroke or transient ischemic attack, can be a consequence of hypotension, carotid thrombosis or dissection. Moreover, despite imaging findings of carotid dissection or thrombosis in patients with TAAD, only a small percentage of patients will present with cerebral malperfusion or stroke. For this reason, arch replacement strategy has ranged from a simple hemiarch replacement to total arch replacement (TAR) all with reasonable results with respect to morbidity and mortality. Prior work from our group

has demonstrated feasibility and safety of concomitant carotid reconstruction in the short term[8]. However, there is a paucity of long-term data comparing the addition of extra thoracic carotid reconstruction with concomitant TAR in this select patient population. The purpose of this study was to compare the differences in outcomes in patients for patients undergoing extrathoracic concomitant carotid artery reconstruction with TAR versus conventional TAR. This study examines TAR with carotid artery replacement in patients with a carotid artery dissection with cerebral malperfusion or carotid artery thrombosis. Carotid dissection without these features typically does not warrant a replacement.

Patients and Methods

Patient Population and Surgical Technique

All patients who underwent surgery for TAAD after 2007 were prospectively entered into our institutional aortic database. Patients who underwent TAR for acute TAAD from 2007-2018 were included. At our institution, there are four indications for a TAR: 1) primary or secondary tears in the arch, 2) circumferential dissection of the aortic arch, 3) aortic arch aneurysm and 4) dissection extending into the carotid body with cerebral malperfusion or carotid body thrombosis (Figure 1). Patients with a type A aortic dissection that do not meet any one of these criteria typically undergo a hemiarch replacement. Out of these 4 indications, dissection into the carotid body or carotid artery thrombosis is our primary indication for a concomitant carotid artery replacement. It is also important to emphasize that a dissection tear proceeding into the carotid artery is not enough of an indication alone; clinical or radiologic cerebral malperfusion is used as a guide to revascularize the carotid arteries. Cerebral malperfusion was determined via clinical exam with neurologic deficit and/or radiological findings demonstrating dissected or occluded carotid artery(ies).

An in-depth description of our surgical strategy for TAR has been previously described[9]. Briefly, TARs are performed in zone 3 with under deep hypothermic circulatory arrest and bilateral antegrade cerebral perfusion. In the TAR cohort, brachiocephalic anastomoses were performed in the chest in a stepwise manner. Once the arterial inflow temperature is 28 degrees Celsius or below on CPB, the innominate artery is ligated proximally and clamped distally. One limb of a trifurcate graft is anastomosed to the innominate artery and unilateral antegrade cerebral perfusion (ACP) is established. Next, the second limb is sewn to the left carotid artery and bilateral ACP is established prior to lower body circulatory arrest. After initiation of circulatory arrest, the aortic arch is trimmed to zone 3, the left subclavian is anastomosed to the third limb of the trifurcate graft and the distal anastomosis is performed in a standard fashion. With concomitant carotid replacement, revascularization of the carotid artery is the first step once the patient is placed on CPB. The carotid artery is clamped at the bifurcation of the internal and external carotid artery and a limb of the trifurcate graft is utilized and tunneled via a subfascial plane to the carotid bifurcations for distal anastomoses as described previously[8]. Similar to brachiocephalic anastomoses in the chest, this is performed prior to circulatory arrest to allow for uninterrupted ACP.

For hypothermic circulatory arrest, EEG and SSEP monitoring were utilized in all patients to evaluate the neurocerebral effects of cooling on the patient. Circulatory arrest was initiated once the patient achieved electrical silent for 4 minutes.

Strokes were assessed and adjudicated by a comprehensive stroke team, which included a stroke neurologist, stroke nurse, and a stroke interventionalist. Any concern for a change in mental status or neurologic exam triggered a stroke alert which led to an exam by a stroke nurse and immediate imaging including CTA of the head and neck with brain perfusion. This was reviewed by the operating surgeon, stroke neurologist and interventionalist to evaluate for the possibility of thrombectomy in the setting of a large vessel occlusion.

Statistical Analysis

Baseline characteristics between the groups were compared. Chi-squared analysis and student's t-tests were used to compare categorical and continuous variables respectively. Kaplan-Meier survival estimates were calculated for overall survival of the groups. Cox multivariable regression analysis was used to determine predictors of mortality using variables established in previous literature as predictive of total arch replacement outcomes. These variables included age, gender, presentation of tamponade, rupture, or shock, presentation of any malperfusion, diabetes mellitus, coronary artery disease, previous cardiovascular intervention, carotid replacement, and preoperative stroke. The use and analysis of this data was approved by our institutional review board.

Results

Baseline Characteristics

A total of 469 patients had surgery for acute TAAD. One hundred and sixty-one patients had total TAR for acute TAAD. Of these, 111 patients had conventional zone 3 TAR and 50 patients had a concomitant carotid replacement (Table 1). There was no significant difference between baseline variables including age, sex, race, body mass index (BMI), hypertension, diabetes, chronic obstructive pulmonary disease (COPD), renal failure, atrial fibrillation, coronary artery disease (CAD), hematocrit, creatinine, and ejection fraction. The carotid replacement group had a higher rate of preoperative neurologic malperfusion (48% vs 10.81%; $p < 0.01$) and pre-operative stroke (28% VS 11.71%; $P = 0.02$). The groups were similar with respect to coronary (5.41% vs 8%, $p = 0.781$), gastrointestinal (6.31% vs 6%, $p = 1.0$), renal (9.01% vs 8%, $p = 1.0$), and peripheral (18% vs 22%, $p = 0.706$) malperfusion. The carotid replacement group had significantly ($p = 0.004$) more patients with either one (23.4% vs 32%), two (3.6% vs 18%), or three or more (6.31% vs 8%) malperfused beds. It is important to note that in the carotid replacement cohort, 24 (48%) of patients had clinical cerebral malperfusion, leaving 28 (52%) of patients having carotid thrombosis as their indication for a carotid artery replacement.

Operative Characteristics and Outcomes

There was no statistical difference in primary tear location between the conventional TAR and carotid replacement groups (Table 2). It is important to note that the tear location is based on an intimal tear site and not the dissection flap extension. Both cohorts had an equal incidence of a secondary arch tear (34.2% vs 34%, $p = 0.99$). In the carotid replacement group, the patients either had a left carotid replacement (28%), a right carotid replacement (36%), or both carotids replaced (36%). There were similar cannulation approaches between the groups, with central being the most frequent (83.78% vs 82%, p

= 0.13). Cardiopulmonary bypass time (min) (245.35 vs 273.96 vs; $p=0.026$) and cross clamp time (163.73 vs 198.07; $p=0.002$) were shorter in the conventional TAR cohort. There was no statistical difference in deep hypothermic circulatory arrest time (min) (34.47 vs 32.36; $p=0.182$) between groups. There was no difference in 30-day mortality between the conventional TAR and carotid replacement groups (18.9% vs 22%; $p=0.81$) (Table 3). There was no significant difference with respect to new postoperative stroke, new onset dialysis requirement, pneumonia, vocal cord paralysis, reoperation for bleeding, new onset atrial fibrillation, new permanent pacemaker, tracheostomy, or PEG tube placement. There was no significant difference in the amount of RBCs (1.65 vs 1.7, $p=0.92$), platelets (0.83 vs 1.22, $p=0.1$), or factor VII (45.95% vs 48%, $p=0.943$) administered perioperatively between conventional TAR and carotid placement groups. No patients had any temporary neurologic deficit or postoperative encephalopathy on discharge. There was no statistical difference in stroke after discharge between cohorts (4.5% vs 5%, $p=0.667$). Discharge destinations were similar between cohorts ($p=0.299$) with the most common destination being home/home health (51.4% vs 40.0%).

Survival Analysis

There was no statistically significant difference in Kaplan-Meier survival estimates between the carotid replacement and conventional TAR groups ($p=0.75$) (Figure 2). Mortality was similar between the conventional TAR and carotid replacement groups at 1-year (27.9% vs 28%; $p=1.0$) and 5-year (29.7% vs 32%; $p=0.917$) follow up. On cox multivariable regression, undergoing a concomitant carotid replacement was not a predictor of mortality (HR 0.82 [0.41, 1.6]; $p=0.558$) (Figure 3). The only significant independent predictors of mortality were age (HR 1.03 [1.00, 1.1]; $p=0.005$), renal failure (HR 3.78 [1.20, 11.9], $p=0.02$) and a pre-operative stroke (HR 2.22 [1.08, 4.6]; $p=0.03$).

Comment

This study on patients with acute TAAD revealed the following findings: First, concomitant carotid artery replacement with TAR is technically feasible and has comparable 30-day mortality when compared to conventional TAR. Second, concomitant carotid artery replacement was not an independent factor for mortality after risk adjustment. Third, there was no difference in long-term survival between the two cohorts.

Acute TAAD is a surgical emergency and the primary goal of the operating surgeon is to always ensure a safe outcome for the patient. Most commonly, this is accomplished with ascending aorta and hemiarch reconstruction [10]. Despite wide adoption of circulatory arrest adjuncts, in hospital mortality for TAAD continues to be around 20% based on national STS data with a stroke rate of 13% [11]. However, these data include patients who have clamped distal aortic anastomoses, hemiarch replacements and TAR. Our high mortality rate is higher than what would be expected of a high volume center because these data only focus on patients who underwent total arch reconstructions. Our prior publication that included all comers demonstrated an operative mortality (STS definition) of 9.1% [8]. It is also important to note that our rate of TAR for TAAD (34%) is significantly higher than most reports from North America. While outcomes of TAR are not significantly different than hemiarch replacement in some centers which may be because of selection bias, others

have shown a distinct survival advantage with hemiarch replacement in the short term. However, data on reducing stroke burden after TAAD has been controversial, particularly in those patients who present with preoperative neurologic deficit. For this reason, some centers may not offer surgery to such patients because of increase in postoperative morbidity and mortality, we have continued to do so with a specific emphasis on carotid revascularization in patients who present with carotid dissection. While conventional TAR may help resect primary and secondary tears in the arch, carotid replacement has a distinct ability to eliminate carotid artery dissection which may play a role in postoperative stroke or continued malperfusion. In an effort to minimize stroke burden in patients who have radiologic or clinical cerebral malperfusion in the setting of common carotid artery dissection, we have continued a carotid replacement strategy in select group of patients. By utilizing this technique, we have not noted any increase in new postoperative strokes in this patient population. It is important to note that while other groups have reported no difference in stroke rates in patients with carotid dissection with or without carotid replacement, our replacement cohort only has patients with carotid dissection with cerebral malperfusion or carotid body thrombosis, which can bias these results[12]. In addition, the carotid replacement technique allows us to mitigate any cerebral malperfusion once the patient is on cardiopulmonary bypass by revascularizing the carotid arteries expeditiously and sequentially as the first step in the operative procedure prior to circulatory arrest or aortic root reconstruction [8,9]. While this may be accomplished with central or axillary artery cannulation in other surgeons' hands, any thrombus in the carotid arteries may potentially propagate to the brain thus increasing stroke burden.

Concomitant carotid replacement does come with additional ischemic and cardiopulmonary bypass time. However, this did not appear to impact postoperative outcomes in our series. Recent data have demonstrated at patients with carotid artery dissection are at increased risk for postoperative stroke because of persistent malperfusion. However, this did not influence survival in their study[13].

Selecting patients for carotid artery replacement has been primarily dependent on burden of common carotid artery dissection and thrombus. Dynamic obstruction may be alleviated with true lumen cannulation and antegrade perfusion, but static obstruction in the common carotid artery may be more challenging to treat. Lu et al. reported a comparatively high incidence of postoperative permanent neurological dysfunction of 10.9% in their cohort of 267 patients with acute type A dissections[14] that had lower rates of preoperative neurologic dysfunction as well as a mixed surgical approach (including both hemi and total arch procedures). With advances in surgical techniques as well as adequate use of cerebral protection, reasonable outcomes with regards to stroke incidence are attainable, with a carotid artery replacement reducing postoperative stroke risk to that of an isolated total arch replacement[7,15]. Reported literature for arch replacement is encouraging, with up to 80% of patient with preoperative CVA or stroke in the setting of a type A aortic dissection having resolution of symptoms after surgery[7,16–18].

The importance of cerebral protection cannot be understated during extended arch reconstruction. We have routinely utilized bilateral ACP for TAR to allow for adequate cerebral perfusion during an extended period of time. In addition, deep hypothermia in

our opinion allows for additional protection for the lower body during arch reconstruction. An arch first technique is utilized in a stepwise manner, debranching and perfusing the innominate, left carotid and (when possible) left subclavian artery in a sequential manner to minimize cerebral ischemic time prior to circulatory arrest. When carotid artery replacement is performed, the carotid arteries are similarly revascularized first prior to distal arch reconstruction.

Continued investigation in this area is warranted to fully assess the benefit of carotid artery replacement in this setting. This is challenging given that a type A aortic dissection constitutes a surgical emergency and surgeons endeavor to secure the best outcome for their patients, which may be by reducing ischemic or circulatory arrest times in elderly patients with comorbidities using a hemi arch replacement. Moreover, only a small percentage of patients present with carotid artery dissection which makes this a relatively uncommon occurrence. Lastly, by showing similar outcomes between these cohorts, the data supports the notion that aggressive carotid replacement in patients with carotid dissection with cerebral malperfusion or thrombosis may reduce the long term risk of having a persistent carotid dissection with concerning features. This notion, however, is based on the idea that patients with TAAD with a carotid dissection and cerebral malperfusion or carotid artery thrombosis who only undergo a TAR may potentially have worse outcomes, which cannot be demonstrated in this study. Nevertheless, the risk of embolic events in patients with cerebral malperfusion or high grade stenosis in those with partial carotid artery thrombosis is likely real. Moreover, acute total carotid occlusion with cerebral malperfusion in the setting of a TAAD is an emergency necessitating immediate revascularization. While some groups may not offer surgery in these patients presenting with stroke, our practice has been to aggressively revascularize the carotid artery and perform surgery for TAAD. Finally, it is encouraging that both cohorts had similar discharge destinations despite the increased disease severity in disease presentation, suggesting an equalization of perioperative outcomes.

Limitations

This study has all the usual limitations of a retrospective analysis. Due to large volume of patients undergoing total arch replacement at this institution, overall outcomes may be biased and not representative of the total arch experience across all centers. Furthermore, despite the large time period of this study and a large experience with carotid artery reconstruction, only a relatively small number of total arch reconstructions with carotid replacement patients existed, which can bias results and increase type II error rates. Moreover, our TAR continues to be more than a third of our TAAD which is significantly higher than the typical center in North America. Despite a substantial majority of patients returning to an in-hospital network, there is non-trivial chance that patients are lost to follow up.

Conclusion

Despite the longer perfusion times, operative times, and higher rates of preoperative neurologic dysfunction in TAAD with associated carotid dissection, concomitant carotid

replacement is a feasible and safe surgical option for this patient population with outcomes that are similar to those undergoing TAR for TAAD. Further large cohort studies are warranted to determine the optimal approach for carotid artery dissection in this patient population.

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Figure 1:
Flowchart for Operative Decision Making

Figure 2:
Kaplan-Meier Survival Estimates comparing patients with carotid artery replacement to conventional Total arch reconstruction

Figure 3:
Cox Multivariable Regression Analysis for Mortality

Table 1:

Baseline Characteristics

Variables*	No carotid Replacement (N = 111)	Carotid Replacement (N = 50)	P Value
Age	58.63 ± 12.37	59.66 ± 11.53	0.61
Sex			0.176
Female	35(31.53%)	22(44%)	
Male	76(68.47%)	28 (56%)	
Race			0.403
Caucasian	88(79.28%)	44(88%)	
BMI	29.78 ± 7.08	30.83 ± 6.71	0.371
Tamponade, Rupture, or Shock	44(39.64%)	17(34%)	0.612
Malperfusion	37(33.33%)	29(58%)	0.006
Neurologic	12(10.81%)	24(48%)	<0.001
Coronary	6(5.41%)	4(8%)	0.781
Gastrointestinal	7(6.31%)	3(6%)	1
Renal	10(9.01%)	4(8%)	1
Lower Extremity	20(18.02%)	11(22%)	0.706
Number of Malperfused Beds			0.004
0	74(66.67%)	21(42%)	
1	26(23.42%)	16(32%)	
2	4(3.6%)	9(18%)	
3+	7(6.31%)	4(8%)	
Hypertension	91(81.98%)	37(74%)	0.342
Diabetes Mellitus	8(7.21%)	3(6%)	1
COPD			0.87
None	92(82.88%)	40(80%)	
Mild	7(6.31%)	5(10%)	
Moderate	10(9.01%)	4(8%)	
Severe	2(1.8%)	1(2%)	
Chronic kidney disease	6(5.41%)	1(2%)	0.574
Prior Heart Surgery	11(9.91%)	5(10%)	1
Bicuspid Aortic Valve	6(5.41%)	2(4%)	1
Preoperative CVA	13(11.71%)	14(28%)	0.02
Aortic Insufficiency			0.073
None	22(19.82%)	8(16%)	
Trivial	17(15.32%)	6(12%)	

Variables*	No carotid Replacement (N = 111)	Carotid Replacement (N = 50)	P Value
Mild	28(25.23%)	7(14%)	0.935
Moderate	11(9.91%)	13(26%)	
Severe	33(29.73%)	16(32%)	
Ejection Fraction	56.24 ± 9.23	56.1 ± 10.32	

* Variables are reported as count (percentage) or mean ± standard deviation for categorical and continuous variables respectively

BSA – Body Surface Area, BMI – Body Mass Index, COPD – Chronic Obstructive Pulmonary Disease, PVD – Peripheral Vascular Disease, CAD – Coronary Artery Disease, CV – Cardiovascular, CVA – Cerebral Vascular Accident

Table 2:**Operative Details**

Variables*	No carotid Replacement (N = 111)	Carotid Replacement (N = 50)	P Value
Primary Tear Location			0.064
STJ	12(10.81%)	11(22%)	
Non-Coronary Sinus	13(11.71%)	10(20%)	
Left Coronary Sinus	8(7.21%)	5(10%)	
Right Coronary Sinus	4(3.6%)	2(4%)	
Ascending Aorta	60(54.05%)	14(28%)	
Aortic Arch	14(12.61%)	8(16%)	
Secondary Arch Tear	38(34.23%)	17(34%)	1
Carotid Replacement			
Left	-	18(36%)	-
Right	-	18(36%)	-
Dual	-	14(28%)	-
Cannulation Strategy			0.13
Central	93(83.78%)	41(82%)	
Subclavian	16(14.41%)	5(10%)	
Femoral	2(1.8%)	4(8%)	
CBP Time (min)	245.35 ± 81.42	273.96 ± 70.98	0.026
Ischemic Time (min)	163.73 ± 62.66	198.07 ± 62.05	0.002
ACP Time	30.95 ± 22.77	36.21 ± 18.69	0.126
Valve Resuspension	60(54.05%)	29(58%)	0.768
Root Repair / Valve Replacement	4(3.6%)	3(6%)	0.785
VSRR	27(24.32%)	9(18%)	0.492
Root Replacement	16(14.41%)	13(26%)	0.122
Conventional Elephant Trunk	22(19.82%)	7(14%)	0.504
Frozen Elephant Trunk	25(22.52%)	8(16%)	0.461

* Variables are reported as count (percentage) or mean ± standard deviation for categorical and continuous variables respectively

STJ – Sinuotubular junction, RCP – Retrograde Cerebral Perfusion, ACP – Antegrade Cerebral Perfusion, CBP – Cardiopulmonary Bypass, VSRR – Valve Sparing Root Replacement, CABG – Coronary Artery Bypass Grafting, RBC – Red Blood Cells, EEG – Electroencephalogram, EP – Electrophysiology

Table 3:**Operative Outcomes**

Variables	No Replacement (N = 111)	Replacement (N = 50)	P Value
In hospital / 30-day Mortality (STS definition)	20(18.02%)	11(22%)	0.706
Pneumonia	15(13.51%)	5(10%)	0.713
Left Vocal Cord Paralysis	6(5.41%)	3(6%)	1
Reoperation for Bleeding	11(9.91%)	3(6%)	0.608
Postoperative stroke	5(4.5%)	0(0%)	0.301
New Atrial Fibrillation	45(40.54%)	22(44%)	0.811
Permanent Pacemaker Requirement	0(0%)	1(2%)	0.681
Stroke Past Discharge	5 (4.5%)	1 (5%)	0.66
Discharge Type			0.299
Dead	20(18.02%)	12(24%)	
Acute Care	11(9.91%)	5(10%)	
Rehab	3(2.7%)	4(8%)	
Long Term Care	20(18.02%)	8(16%)	
Home/Home Health	57(51.35%)	20(40%)	
Other	0(0%)	1(2%)	

* Variables are reported as count (percentage) or mean $\pm$ standard deviation for categorical and continuous variables respectively

ACP – Antegrade Cerebral Protection , CVA – cerebral Vascular Accident, ICU – Intensive Care Unit, LOS – Length of stay, OR -Operating Room