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Impact of Tear Location in Acute Type-A Aortic Dissection on Postoperative Neurologic Complications

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ABSTRACT

Objective: The objective of this retrospective study was to establish a relationship between the locations of the primary entry tear in acute type A aortic dissections and the occurrence of postoperative neurological dysfunction

Methods: From January 2018 to December 2019, a total of 316 patients diagnosed with type A acute aortic dissection underwent conventional surgical repair. The patients were divided into two study groups based on the location of the primary tear in the aorta and the presence of postoperative neurological dysfunction. Propensity score-matching analysis was employed to compare outcomes between the groups and reduce selection bias.

Results: The incidence of neurological dysfunction was 10.75% (34 patients). Hypertension demonstrated a high Odd Ratio (OR) of 76.7 (95% Confidence Interval (CI) 15.0-1,385; p<0.001). The cerebral protection strategy showed a low odds ratio of 0.16 (95% CI 0.05-0.47; p=0.001), while the arterial cannulation site had an odds ratio of 0.16 (95% CI 0.05-0.47; p=0.003). Aortic cross-clamping time displayed an odds ratio of 1.03 (95% CI 1.01-1.05; p=0.001) and CPB time had an odds ratio of 1.01 (95% CI 1.00-1.02; p=0.025). Body temperature revealed an odds ratio of 0.20 (95% CI 0.10-0.35; p<0.001). These factors were identified as independent predictors of postoperative neurological dysfunction in a group of studies based on the location of the primary tear.

Conclusion: Hypertension was found to be independently associated with postoperative neurological dysfunction. Furthermore, patients with aortic dissection involving the aortic arch are at a higher risk of developing postoperative neurological dysfunction, with intraoperative factors such as the cerebral protection strategy, arterial cannulation site, aortic cross-clamping time and body temperature acting as risk factors. The location of the primary tear and the involvement of the aortic arch in ATAAD are significant determinants of postoperative neurological outcomes.

Keywords: Type-A aortic dissection; Primary tear; Postoperative; Neurologic dysfunction; Risk factors

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INTRODUCTION

Stanford Type-A Aortic Dissection (AAD) is a life-threatening condition requiring immediate surgical intervention. While surgical repair and the implementation of neuro-protective protocols such as Hypodermic Circulatory Arrest (HCA), selective anterograde cerebral perfusion and careful cannulation techniques have significantly improved patient outcomes, Postoperative Neurological Dysfunction (PND) remains a concerning complication [1]. In addition to the initial neurological symptoms resulting from the dissection, there is a notable risk of developing neurological dysfunction ND following surgical intervention. This study contributes valuable insights into the understanding of postoperative neurological dysfunction in ATAAD patients. By elucidating the relationship between the location of the primary tear, specific risk factors and the occurrence of neurological complications, this research provides a foundation for further research and potential interventions aimed at reducing the incidence of these complications and improving patient care in this high-risk population.

MATERIALS AND METHODS

From January 2018 to December 2019, a total of 316 patients diagnosed with type A acute aortic dissection underwent conventional surgical repair. The patients were divided into two groups based on the presence of postoperative neurological dysfunction and the location of the primary tear of the aortic dissection. Propensity score-matching analysis was employed to compare outcomes between the groups and reduce selection bias.

Statistical Analysis

The mean and standard deviation were used to describe continuous variables. The difference between the two groups was compared using the student's t-test. Categorical variables were presented as percentages or frequency distributions and differences between the groups were assessed using the χ^2 test or Fisher exact test, depending on the circumstances. Propensity score matching was employed to minimize the impact of selection bias between the groups. To determine the variables that served as predictors of neurological complications, a multivariable logistic regression analysis was conducted. A p-value below 0.05 was considered statistically significant and all statistical analyses were performed using R version 4.2.3 (2023-03-15 ucrt).

Definition

Aortic dissection was diagnosed by Computed Tomography Aortography (CTA). Post-Neurologic Dysfunction (PND) was defined as the occurrence of new neurologic dysfunction following surgical intervention, such as stroke, coma, spinal cord ischemia or intracranial hemorrhage, which was confirmed through brain Computed Tomographic (CT) scanning or magnetic resonance imaging. Conditions involving poor consciousness, delirium and convulsions, which can be described as Temporary Neurologic Dysfunction (TND), exhibited complete resolution on CT scans before the patient's discharge.

Operative

All patients were operated on using a midsternal approach. After systemic heparinization, Cardiopulmonary Bypass (CPB) was instituted. For patients who presented with a stable preoperative condition, we opted for a double arterial cannulation approach, utilizing a combination of right axillary and femoral arterial access, coupled with the Antegrade Cerebral Perfusion (ACP) strategy. Conversely, for patients with unstable hemodynamics, we typically employed isolated femoral artery cannulation along with Retrograde Cerebral Perfusion (RCP). In general, our surgical approach involved replacing the dissected aorta with a prosthetic graft, determined by the location of the primary entry tear and the patient's preoperative condition. The ascending aorta was routinely replaced with aortic valve re-suspension. The proximal anastomosis was typically performed first, followed by the open distal anastomosis under circulatory arrest. During the circulatory arrest phase, femoral arterial flow was temporarily suspended. Depending on the previously selected cannulation strategy, we administered selective ACP through the right axillary artery or RCP through the superior vena cava.

In cases where the extent of aortic dissection involved the aortic root and resulted in severe aortic regurgitation that was challenging to repair, we conducted an aortic root replacement using a composite Valsalva graft. For patients with aortic dissection extending to the distal arch and proximal descending aorta, coupled with preoperative malperfusion dilatation, we performed a concurrent frozen elephant trunk procedure using a covered stent graft [2].

Following surgical repair for ATAAD, all patients were transferred to a specialized cardiovascular Intensive Care Unit (ICU) for further treatment and close monitoring.

RESULTS

The present study included a total of 316 patients diagnosed with type A Aortic dissection, of which 228 (72.15%) were males. Among the patients, only 120 (37.97%) had a diagnosis of hypertension and were on medication. Hypertension was the only significant variable identified in the preoperative **Table 1**. There were no other significant differences observed between the two matched groups in terms of preoperative baseline characteristics. Propensity score matching was performed to compare the groups of patients with and without postoperative neurological dysfunction. Initially, 34 pairs of patients were identified for 1:1 matching, followed by a 1:1.72 (25 *vs.* 43) matching based on the location of the primary tear of the aortic dissection.

Table 1: Preoperative characteristics of study groups before and after matching.

Variables	Overall unmatched patients based on the presence of PND or not			Patients after propensity score-matching			Overall unmatched patients based on location and extension of the AD			patients based on location and extension of the AD after propensity score-matching		
	WPND	PND	p-value	WPND	PND	p-value	PAD	DAD	p-value	PAD	DAD	p-value
n	282	34		34	34		133	183		25	43	
Gender (Male)	205 (72.7)	23 (67.6)	0.676	22 (64.7)	23 (67.6)	1	91 (68.4)	137 (74.9)	0.257	15 (60.0)	30 (69.8)	0.579
Age (years)	50.00 (10.87)	50.76 (11.49)	0.7	52.41 (9.90)	50.76 (11.49)	0.529	50.71 (12.11)	49.63 (9.97)	0.387	52.96 (13.06)	50.79 (9.09)	0.423
BMI (kg/m^2)	24.86 (3.70)	24.89 (2.69)	0.959	24.47 (3.10)	24.89 (2.69)	0.552	24.72 (3.56)	24.97 (3.64)	0.54	24.81 (3.20)	24.61 (2.73)	0.789
History of TEVAR	6 (2.1)	0 (0.0)	0.846	0 (0.0)	0 (0.0)	0 (0.0)	2 (1.5)	4 (2.2)	0.983	0 (0.0)	0 (0.0)	
Coronary artery disease	18 (6.4)	1 (2.9)	0.678	1 (2.9)	1 (2.9)	1	4 (3.0)	15 (8.2)	0.094	24/1 (96.0/4.0)	42/1 (97.7/2.3)	1
Hypertension	87 (30.9)	33 (97.1)	<0.001	33 (97.1)	33 (97.1)	1	43 (32.3)	77 (42.1)	0.1	24 (96.0)	42 (97.7)	1
Pregnancy	2 (0.7)	0 (0.0)	1	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.8)	1 (0.5)	1	25/0 (100.0)	43/0 (100.0)	-
Diabetes	3 (1.1)	0 (0.0)	1	0 (0.0)	0 (0.0)	0 (0.0)	2 (1.5)	1 (0.5)	0.78	25/0 (100.0)	43/0 (100.0)	-

Note: Data are presented as mean SD or as number (%), Statistically significant ($p<0.05$).WPND: Without Post-Neurologic Dysfunction, PND: Post Neurologic Dysfunction, DAD: Distal Aortic Dissection, PAD: Proximal Aortic Dissection.

Intraoperative data, presented in **Table 2**, were compared between the groups of patients with and without postoperative neurological dysfunction after propensity score matching. No significant differences were observed. For the patient's population based on location of the primary tear, several intraoperative variables were found to be significant. These included CPB time (186.20 ± 50.88 min *vs.* 241.52 ± 62.84 min, $p<0.001$), aortic cross-clamping time (98.23 ± 20.77 min *vs.* 121.41 ± 28.75 min, $p<0.001$), arterial cannulation site ($p<0.001$), body temperature ($p<0.001$) and cerebral protection ($p<0.001$). After propensity score matching, the significant variables in **Table 3**, representing the intraoperative data of groups according to the location of the primary tear, were as follows: CPB time (209.84 ± 57.73 min *vs.* 245.40 ± 65.88 min, $p=0.028$), aortic cross-clamping time (100.72 ± 24.46 min *vs.* 122.19 ± 25.67 min, $p=0.001$), arterial cannulation site ($p=0.030$) and body temperature ($p=0.016$). Multivariate analysis presented in **Table 3** revealed the following Odds Ratios (ORs) and Confidence Intervals (CIs): Hypertension OR 76.7, 95% CI 15.0-1,385, $p<0.001$; cerebral protection strategy OR 0.16, 95% CI 0.05-0.47, $p=0.001$; arterial cannulation site OR 0.16, 95% CI 0.05-0.47, $p=0.003$; aortic cross-clamping time OR 1.03, 95% CI 1.01-1.05, $p=0.001$; CPB time (min) OR 1.01, 95% CI 1.00-1.02, $p=0.025$; and body temperature OR 0.20, 95% CI 0.10-0.35, $p<0.001$.

Table 2: Intraoperative characteristic according to the study groups after matching.

Variables	Overall unmatched patients based on the presence of postoperative neurologic dysfunction			Overall patients based on postoperative neurologic dysfunction after propensity score-matching			Overall unmatched patients based on dissection primary entry tear			Patients based on dissection primary entry tear after propensity score-matching		
	WPND	PND	p-value	WPND	PND	p-value	PAD	DAD	p-value	PAD	DAD	p-value
n	282	34		34	34		133	183		25	43	
CPB time (min)	216.75 (66.40)	230.56 (39.40)	0.236	234.09 (83.63)	230.56 (39.40)	0.825	186.20 (50.88)	241.52 (62.84)	<0.001	209.84 (57.73)	245.40 (65.88)	0.028
Arterial cannulation site			0.487			0.7			<0.001			0.03
Aorta	17 (6.0)	1 (2.9)		2 (5.9)	1 (2.9)		18 (13.5)	0 (0.0)		3 (12.0)	0 (0.0)	
Axillary	160 (56.7)	17 (50.0)		20 (58.8)	17 (50.0)		52 (39.1)	125 (68.3)		10 (40.0)	27 (62.8)	
Femoral	38 (13.5)	4 (11.8)		2 (5.9)	4 (11.8)		31 (23.3)	11 (6.0)		4 (16.0)	2 (4.7)	
Femoral + Axillary	67 (23.8)	12 (35.3)		10 (29.4)	12 (35.3)		32 (24.1)	47 (25.7)		8 (32.0)	14 (32.6)	
Aortic cross-clamp time (min)	111.13 (28.65)	115.97 (23.02)	0.344	112.62 (30.96)	115.97 (23.02)	0.614	98.23 (20.77)	121.41 (28.75)	<0.001	100.72 (24.46)	122.19 (25.67)	0.001
Cerebral protection			0.34			0.214			<0.001			0.098
Antegrade	177 (62.8)	18 (52.9)		24 (70.6)	18 (52.9)		69 (51.9)	126 (68.9)		13 (52.0)	29 (67.4)	
Retrograde + antegrade	67 (23.8)	12 (35.3)		9 (26.5)	12 (35.3)		32 (24.1)	47 (25.7)		8 (32.0)	13 (30.2)	
Retrograde	38 (13.5)	4 (11.8)		1 (2.9)	4 (11.8)		32 (24.1)	10 (5.5)		4 (16.0)	1 (2.3)	
Body temperature (°C)			0.78			0.591			<0.001			0.016

Deep Hypothermia	5 (1.8)	1 (2.9)		0 (0.0)	1 (2.9)		2 (1.5)	4 (2.2)		0 (0.0)	1 (2.3)	
Moderate Hypothermia	189 (67.0)	24 (70.6)		24 (70.6)	24 (70.6)		55 (41.4)	158 (86.3)		13 (52.0)	35 (81.4)	
Mild Hypothermia	88 (31.2)	9 (26.5)		10 (29.4)	9 (26.5)		76 (57.1)	21 (11.5)	76 (57.1)	12 (48.0)	7 (16.3)	
Circulatory arrest time (min)	20.55 (6.64)	20.85 (4.47)	0.874	17.67 (5.51)	20.85 (4.47)	0.109	23.27 (5.09)	20.26 (6.52)	0.111	21.00 (1.63)	18.83 (5.56)	0.452

Note: Data are presented as mean SD or as number (%), Statistically significant (p<0.05).WPND: Without Post-Neurologic Dysfunction, PND: Post Neurologic Dysfunction, DAD: Distal Aortic Dissection, PAD: Proximal Aortic Dissection.

Table 3: Multivariate analysis of postoperative neurologic dysfunction.

Variables	Patients based on postoperative neurologic dysfunction			Patients based on dissection primary entry tear		
Variables	**OR**	**95% CI**	**p-value**	**OR**	**95% CI**	**p-value**
Hypertension	76.7	15.8, 1.385	<0.001	1.13	0.61, 2.08	0.7
Cerebral protection	1.68	0.47, 6.88	0.4	0.16	0.05, 0.47	0.001
Arterial cannulation site	0.7	0.18, 2.42	0.6	5.01	1.85, 16.5	0.003
Aortic cross-clamping time	1	0.99, 1.02	0.6	1.03	1.01, 1.05	0.001
CPB time (min)	1	0.99, 1.01	>0.9	1.01	1.00, 1.02	0.025
Body temperature	0.7	0.26, 1.84	0.5	0.2	0.10, 0.35	<0.001

Table 4 presents the presentation of PND according to the location of the primary tear of the aortic dissection. The incidence of neurological dysfunction was 10.75% (34 patients) with 21 patients with primary tear located within the aortic arch.

Table 4: Postoperative neurologic dysfunction PND according to location of the primary tear.

Neurological dysfunction	PAD	DAD	Total (34patients)
Coma	1 (2.95%)	6 (17.65%)	7 (20.60%)
Stroke	4 (11.75%)	9 (26.50%)	13 (38.25%)
POD	1 (2.95%)	1 (2.95%)	2 (5.90%)
Convulsions	1 (2.95%)	0 (0.00%)	1 (2.95%)
ICH	1 (2.95%)	1 (2.95%)	2 (5.90%)
Spinal cord ischemia	2 (5.90%)	1 (2.95%)	3 (8.85%)

Poor consciousness	3 (8.80%)	3 (8.80%)	6 (17.60%)

Note: PAD: Proximal Aortic Dissection, DAD: Distal Aortic Dissection, POD: Postoperative Delirium, ICH: Intracranial Hemorrhage.

DISCUSSION

Our findings revealed an incidence of neurological dysfunction in 10.75% of patients. This is relatively low compared to numerous studies that investigated the occurrence of postoperative neurologic dysfunction in patients with type-A aortic dissection [2]. For instance, Zdravkovic et al., study presented a total of 87 (39.5%) out of 240 patients with neurological damage after surgery and Yuping, Tianhui and Ling last year in a comprehensive analysis, a total of 20 studies encompassing 11382 cases were examined. Among these cases, 1321 patients experienced PND and a total of 34 predictive risk factors were identified across the studies [3].

Notably, hypertension emerged as a significant predictor, demonstrating a high odds ratio of 76.7. Over the past three decades, there has been a substantial rise in the prevalence of hypertension in China. Currently, approximately one-quarter of all Chinese adults are affected by hypertension. This increase can primarily be attributed to the improved life expectancy observed among the general population. The findings of this study indicate that individuals with hypertension exhibited higher rates of PND when compared to those without hypertension. Consistent with previous research, the study conducted by Jiang et al. also identified hypertension as a significant risk factor for Postoperative Neurological Dysfunction (PND) [4].

Acknowledging the pivotal role of the point of entry in aortic dissection and its profound implications for the management of AAD and its outcomes, we undertook a synthesis of two studies. These studies included patients both with and without PND and we categorized them based on tear location and aortic arch involvement. This methodology was adopted to attain a more holistic comprehension of the risk factors associated with PND. If the entry tear surpasses the size of the re-entry tears, it elevates the vulnerability to cracking within the false lumen, proximal descending aorta and the vicinity of the re-entry tear. Furthermore, the precise location of the entry tear can substantially alter the hemodynamics of aortic dissection. When the entry tear is in closer proximity to the proximal ascending aorta, it consistently enlarges the false lumen and exerts pressure on the true lumen, ultimately causing a reduction in the dimensions of the true lumen. In cases involving the proximal ascending aorta, heightened pressure within the false lumen acts as a predictive factor for an increased risk of reverse tearing [5].

Several studies have found the location of the primary tear of ATAAD as a risk factor for PND and it is one of the parameters for GERAADA (German Registry for Acute Aortic Dissection type A) score preoperative prediction for 30-day mortality after ATAAD [6,7]. In their study, Haldenwang, et al., concluded that a dissection "entry" situated in the aortic arch or the descending aorta could elevate the risk of postoperative stroke [8]. This conclusion aligns with our findings. Notably, our study, which was based on the location of the primary tear, discovered intraoperative factors that function as independent predictors of postoperative neurological dysfunction. These factors encompass the cerebral protection strategy, the site of arterial cannulation, the duration of aortic cross-clamping, CPB duration and body temperature. These factors have sparked considerable debate regarding the optimal strategy to decrease mortality and achieve diverse outcomes.

Due to the escalating prevalence of hypertension within its extensive population, China has observed a marked increase in cases of aortic dissection, particularly during colder periods when physical activity tends to decrease. To address this, cardiac centers in China have enhanced their operating rooms and ICUs, aiming to lower the mortality associated with ATAAD. Consequently, surgeons are exposed to a substantial caseload, affording them invaluable experience that translates into significantly early management of ATAAD patients, shorter surgery and CPB durations. Consistent with our study Lin et al in a recent study found experiencing hemorrhagic stroke patients had aortic arch procedures and additionally, prolonged durations of CPB and aortic cross-clamping.

In our study, the occurrence of postoperative stroke as a form of neurological dysfunction was the highest but relatively low, with only 13 patients experiencing this complication. This incidence is lower compared to the findings of Dumfarth, et al., who reported a postoperative stroke rate of 15.8% (n=48) in their study [9]. Similarly, Ghoreishi, et al., using the society of thoracic surgeon's adult cardiac surgery database, reported an incidence of postoperative stroke at 13% [10]. Other PND in our study are resumed in **Table 4**.

It is important to note that none of the patients in our study exhibited neurological dysfunction before undergoing surgery. All instances of PND were newly observed occurrences following the surgical intervention. This distinction underscores the impact of the surgical procedures and associated factors on the development of neurological complications in these patients [11].

Arterial Cannulation

The debate over the impact of cannulation strategy on neurological outcomes continues, highlighted by studies such as Kreibich, et al. While some research shows comparable complication and mortality rates between femoral and axillary artery cannulation, other studies suggest that axillary and subclavian artery cannulation may offer better neurological outcomes and reduced mortality for acute type A aortic dissection repair, as indicated by Klotz et al., Ren et al., Lee, et al. and Moizumi, et al., in their various studies. Similarly, our

study predominantly employed axillary cannulation with antegrade cerebral protection, resulting in a relatively lower incidence of PND compared to Kreibich, et al.'s findings, which did not differentiate between cannulation strategies. Despite studies favoring axillary cannulation, we maintain that the choice of method should be tailored to the surgeon's preference, patient's presentation and dissection extent [12,13].

Temperature Influence on Neurological Outcomes

Gong et al.in their literature concluded the temperature in the periphery during cardiac surgeries plays a role in affecting neurological outcomes. Elevated temperatures are associated with increased metabolic activity, whereas lower temperatures reduce peripheral metabolic activities [14]. Nevertheless, maintaining a reduced body temperature during surgery lowers oxygen demand due to decreased metabolic activity. Browne identifies the technique employed in this process as HCA, which spans from mild (>28℃) to moderate (20℃-28℃) and deep (<20℃) hypothermia. The specific temperature chosen within the hypothermia spectrum is subject to debate, contingent on the hypothermia technique applied during the surgery. For example, Ko, et al. suggest that most cardiac surgeons lean towards using moderate HCA with ACP or deep HCA with RCP [15,16]. Both techniques achieve good cerebral protection without significant difference between their performances, as incidences, morbidity and mortality from stroke.

CPB Time

Cardiac bypass time is limited to under 180 minutes (<3 hours) to mitigate the risk of neurological complications. Prolonged CPB time significantly increases the likelihood of adverse outcomes, including strokes and organ dysfunction. Madhavan, et al., found that a mere 10-minute increase in CPB time raises the risk of stroke by 25%, with longer durations posing even greater dangers, including hypertension and a 32% higher probability of mortality. While the recommended optimal CPB time is three hours, it's important to note that other patient and surgical factors also play a role in influencing outcomes, indicating that maintaining CPB time within three hours alone may not provide a substantial advantage in preventing adverse neurological effects [17].

CONCLUSION

In conclusion, our study has shed light on the complex interplay between various factors influencing postoperative neurological outcomes in patients with aortic dissection. Hypertension emerged as a significant predictor of postoperative neurological dysfunction, mirroring the escalating prevalence of this condition within China's population. The significance of the primary tear's location and aortic arch involvement was underscored in our investigation, aligning with findings from other studies that highlight their roles as risk factors for postoperative neurological dysfunction.

Despite the high stakes involved in postoperative neurological outcomes, our study indicates the potential for substantial advancements in mitigating risks through tailored surgical approaches. Further studies and collaborative efforts are necessary to enhance our understanding and refine strategies for minimizing neurological complications in patients undergoing aortic dissection repair.

LIMITATIONS

The primary limitation of the present study lies in its retrospective study design. While it is important to acknowledge that this study had a retrospective, single-center design, it is noteworthy as it represents a unique endeavor, combining two separate studies to fulfill our objective of identifying risk factors for postoperative neurologic dysfunction following surgical repair for aortic dissection. Preoperative PND was present in some patients this could have effect on the outcomes. Our retrospective database analyses may have limitations in reporting events. We were unable to furnish details regarding late complications, the quality of life and the cause of death after the patient's discharge.

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