

Stroke After Emergent Surgery for Acute Type a Aortic Dissection

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ABSTRACT

Background: Cerebral ischemia in acute type A aortic dissection (ATAAD) is primarily attributed to hypotension, reduced blood flow to the brain as a result of thromboembolism and real lumen blockage. If we want to improve patient outcomes, we need to know what factors increase the risk of postoperative stroke and how those factors affect neurological recovery and morbidity.

Aim: Finding factors that increase the risk of postoperative stroke and studying how those factors affect neurological recovery and morbidity were the goals of this research following emergent surgical repair of ATAAD.

Patients and Methods: A non-randomized prospective with fifty individuals diagnosed with ATAAD participated in the research.

who underwent emergent surgical repair at Kasr Al-Ainy Hospital, Faculty of Medicine, Cairo University. The study assessed risk factors associated with postoperative stroke, including surgical techniques, perfusion strategies, and patient-specific variables.

Results: The supra-coronary repair method showed a trend toward association with postoperative stroke, yet the result was not noteworthy from a statistical standpoint (p = 0.123). Likewise, there was no statistically significant relationship between circulatory stoppage and stroke after surgery (p = 0.255). Stroke incidence was, however, substantially correlated with retrograde cerebral perfusion (p = 0.001). A significant risk factor for stroke was found to be femoral cannulation. There was a greater frequency of severe carotid stenosis ($p \le 0.007$) and considerably higher blood triglyceride levels (p < 0.001) in patients who had a stroke after surgery. Patients with a stroke had a much higher death rate (75%) than those without a stroke (9.5%; p = 0.01).

Conclusion: Stroke after ATAAD surgery is related with a higher risk of death, particularly in older individuals, those with elevated serum triglyceride levels, and those with significant carotid stenosis. These findings highlight the importance of preoperative risk stratification and tailored surgical strategies to mitigate stroke risk.

Keywords: Stroke; acute type A aortic dissection (ATAAD); surgical repair; retrograde cerebral perfusion; carotid stenosis; serum triglycerides.

1. INTRODUCTION

Acute type A aortic dissection (TAAD) surgical treatment has significantly boosted survival rates over the last 20 years, however cerebral malperfusion is still a difficult condition to overcome. Thrombo-embolism, low blood pressure, and decreased cerebral perfusion from real lumen impairment are the main causes of cerebral ischemia in TAAD [1]. As a result, up to ten percent of patients have a stroke prior to surgery, and twenty to thirty percent show neurological symptoms upon admission [2].

The frequency of postoperative brain damage is still quite varied, ranging from 2.9 percent to 30.4 percent, despite improvements in surgical procedures and cerebral perfusion schemes.[3].

This variability underscores the complexity of predicting and preventing neurological complications in TAAD patients

Although several variables have been shown to increase the likelihood of a stroke, one of them is becoming older, prolonged circulatory arrest, and preoperative malperfusion, the interplay of these factors and their impact on outcomes remains incompletely understood [4, 5].

A deeper understanding of the predictors of postoperative stroke is essential for optimizing surgical and perioperative management, thereby reducing persistent neurological deficits and improving overall outcomes. The purpose of this research is to better understand the factors that put patients receiving emergency surgery to repair TAAD at increased risk of postoperatively stroke, as well as the effects of this risk on their neurological recovery, mid-term survival, as well as morbidity

Patients and methods

Study Design and Population

This prospective, non-randomized study aimed to determine how often and what factors are associated with postoperative stroke in patients having urgent surgery to fix acute type A aortic dissection (ATAAD). The research was carried out at Kasr Al-Ainy Hospital, which is part of the Faculty of Medicine at Cairo University. Fifty patients who fulfilled the inclusion criteria entered the trial in a sequential order.

If a patient had a stroke after surgery, they were divided into two groups:

- **Stroke group (n=8):** People who had a stroke after surgery.
- **No-stroke group (n=42):** Patients who did not develop postoperative stroke.

Inclusion Criteria:

• People who needed emergency surgery to fix a type A aortic dissection.

Exclusion Criteria:

- Patients who underwent combined surgical procedures.
- Re-do surgical cases.
- People who have chronic type A aortic dissection
- History of stroke, admitted to the hospital at the same time.

Sample Size: Fifty individuals who had immediate type A aortic dissection and had surgery to correct it were part of the research.

Methods

Preoperative counseling, imaging scans, laboratory investigations, physical examinations, and extensive medical histories were all part of each patient's comprehensive evaluation.

Intraoperative procedures:

The administration of fentanyl (5-10 μ g/kg) and pancuronium (0.02 mg/kg) was used to produce anesthesia. Standard procedure included inserting two central venous catheters with three lumens and two radial artery cannulas on each side. The use of arterial pressure and transesophageal echocardiography (TEE) for the detection of malperfusion were components of the intraoperative monitoring system. The pericardium was incised during a conventional midline sternotomy. In order to find the cannulation site and evaluate the amount of replacement, the aortic arch was dissected. Successful venous drainage was accomplished by cannulating the right atrium and inserting a left ventricular vent catheter via the right superior pulmonary

The Seldinger method was used to conduct central cannulation and reach the actual lumen. An 8-mm Dacron graft was used for axillary cannulation, and the open Seldinger method was used for femoral cannulation. Activation of the extracorporeal circuit and cooling to a target temperature of 28°C followed cannulation. To examine the roots, the aorta was opened longitudinally using the cross-clamp technique and directed into the non-coronary sinus. It was the coronary ostia that received the blood cardioplegia. It was necessary to do a Bentall surgery in order to replace an aortic valve if it could not be repaired. Cooling to 24-26°C was used in 29 instances of hypothermic circulatory arrest (HCA) for the purpose of arch surgery. In certain instances, a catheter was inserted into the superior vena cava (SVC) to combine retrograde cerebral perfusion (RCP) with hemodynamic compression administration (HCA). Weaning the pump off, reversing anticoagulation, and confirming hemostasis were all done after rewarming.

Postoperative Evaluation:Postoperative evaluation involved close monitoring of ventilation status, hemodynamics, blood loss, transfusions, and the need for re-exploration. Neurological events, total ICU stay, hospital stay, and mortality were also recorded. The primary endpoint was the occurrence of stroke, defined as permanent neurological dysfunction confirmed by

physical examination and/or brain injury detected on CT or MRI. Mortality served as a secondary endpoint. Additional secondary endpoints included postoperative complications and morbidity, such as neurological impairment, duration of mechanical ventilation, renal insufficiency, tracheostomy, and re-exploration due to bleeding.

Statistical analysis

In order to get this data, we referred to the version 24 of the Statistical Package for the Social Sciences (SPSS) for Windows. In order to offer a description of the categorical data, percentages and frequencies were used. When representing quantitative data that was presumed to have a normal distribution, the mean and standard deviation were used as representational tools. The Shapiro-Wilk test was used in order to answer the question of whether or not the distribution was normal. In the event that any assumptions were broken, the fissure exact test was used in the process of determining whether or not there were any correlations associated with categorical variables. An independent sample T test was used in order to investigate the difference in a numerical variable that existed between the two groups. It was determined that a P value that was lower than 0.05 was considered to be statistically significant.

2. RESULTS

Table (1): Describing the sociodemographic variables of the patients(n=50).

| Variable | N (%) | |
|--------------------------|-------------------|--|
| Gender | | |
| Male | 40 (80) | |
| Female | 10 (20) | |
| | | |
| | | |
| Age (years) | $50.04 \pm 14.7*$ | |
| Associated comorbidities | | |
| HTN | 20 (40) | |
| DM | 2 (4) | |
| Smoking | 19 (38) | |
| Renal impairment | 3 (6) | |
| TIA | 6 (12) | |
| Family history of stroke | 8 (16) | |
| | | |
| | | |
| | | |
| | | |
| | | |
| * Mean ± SD | | |

Table (1): Describing the intraoperative data among all participants (n=50).

| Variable | N (%) |
|---|-----------------|
| Extent of Dissection: | |
| Ascending Aorta | 50 (100) |
| Aortic Arch | 40 (80) |
| Descending Aorta | 40 (80) |
| | |
| Repair type | |
| Bentall | 22 (44) |
| Supracoronary (interpositional graft) | 26(52) |
| Supracoronary + Rt coronary sinus replacement | 1 (2) |
| Elephant trunk | 1 (2) |
| | |
| Circulatory arrest | 29 (58) |
| Circulatory arrest time (minutes) | 22.72 ± 8.5 |
| Cerebral perfusion | |
| Antegrade | 26 (52) |
| Retrograde | 24 (48) |
| | |
| Cannulation type (arterial) | |
| Axillary | 15 (30) |
| Femoral | 26 (52) |
| Central | 9 (18) |
| | |
| Cross clamp time (minutes) | 186.43 ± 39.7* |
| * Mean ± SD. | |

Table (3): describing the postoperative findings among the patients (n=50).

| Variable | N (%) | |
|--------------------------------|-----------------|--|
| Time of extubation (hours) | 28.8 ± 22.7* | |
| Renal insufficiency | 5 (1) | |
| Tracheostomy | 11 (22) | |
| Re-exploration due to bleeding | 7 (14) | |
| Post-operative stroke | 8 (16) | |
| Post-operative NIHSS score | 2.4 ± 0.5 * | |
| * Mean ± SD | | |

Relations:

Table (4): Showing the association between Postoperative stroke and sociodemographic variables of the diseased sample (n=50).

| Variable | Postoperative strok | Postoperative stroke | |
|-------------------------------|---------------------|----------------------|---------|
| | Yes (n=8) | No (n=42) | |
| Gender | | | |
| Male | 6 (75) | 34 (81) | 0.653* |
| Female | 2 (25) | 8 (19) | |
| Age | 59.25 ± 5.47 | 48.29 ± 15.25 | 0.001** |
| DM` | 1 (12.5) | 1 (2.4) | 0.297* |
| HTN | 1 (12.5) | 19 (45.2) | 0.123* |
| Smoking | 4 (50) | 15 (35.7) | 0.459* |
| Renal impairment | 0 | 3 (7.1) | 1.00* |
| TIA | 3 (37.5) | 3 (7.1) | 0.044* |
| F.H of stroke | 4 (50) | 4 (9.5) | 0.016* |
| * Fissure exact test. | | | |
| ** Independent sample T test. | | | |

Table (5): Showing the association between preoperative findings and post-operative stroke occurrence in patients (n=50).

| Variable | Postoperative stroke | | P value |
|--|----------------------|----------------|----------|
| | Yes (n=8) | No (n=42) | |
| ABG | | | |
| Normal | 6 (75) | 33 (78.6) | |
| Acidosis | 2 (25) | 3 (7.1) | 0.506* |
| Decreased Po ² | 0 | 4 (9.5) | |
| Increased PCo ² | 0 | 2 (4.8) | |
| Triglycerides | 196.5 ± 20.47 | 137.55 ± 48.57 | <0.001** |
| Significantly stenosed Rt carotid (≥50%) | 4 (50) | 2 (4.8) | 0.007* |
| Significantly stenosed Lt carotid (≥50%) | 6 (75) | 3 (7.1) | <0.001* |
| * Fissure exact test ** Independent sample T test | | | |

Despite the fact that all stroke patients were male (p = 0.653), there was no significant association between gender and postoperative stroke. The age difference between stroke and non-stroke patients was statistically significant (p = 0.001). Stroke development could not be predicted by co-morbidities including diabetes mellitus, hypertension, smoking, or renal impairment (p > 0.05). On the other hand, postoperative stroke was significantly predicted by a history of transient ischemic

attack (TIA) (p = 0.044) and a family history of stroke (p = 0.016). In addition, there was a substantial increase in the likelihood of severe carotid stenosis (p \leq 0.007) and serum triglyceride levels (p < 0.001) in individuals who had a stroke. Acidosis, ischemic alterations, and the risk of stroke after surgery were not significantly associated (p > 0.05).

Table (6): showing the association between intraoperative findings and postoperative stroke occurrence in patients (n=50)

| Variable | Postoperative stroke | | P value |
|-----------------------------------|----------------------|-------------------|---------|
| | Yes (n=8) | No (n=42) | |
| Repair type | | | |
| Bentall | 1 (12.5) | 21 (50) | |
| Supracoronary | 7 (87.5) | 19 (38) | 0.123* |
| Supracoronary + Rt coronary sinus | 0 | 1 (2.4) | |
| replacement | 0 | 1 (2.4) | |
| Elephant trunk | | | |
| Circulatory arrest | 3 (37.5) | 26 (61.9) | 0.255* |
| Cerebral perfusion | | | |
| Antegrade | 0 | 26 (61.9) | 0.001* |
| Retrograde | 8 (100) | 16 (38.1) | |
| Cannulation type (arterial) | | | |
| Axillary | 0 | 15 (35.7) | |
| Femoral | 8 (100) | 18 (42.9) | 0.012* |
| Aortic | 0 | 9 (21.4) | |
| Cross clamp time (minutes) | 197.25 ± 11.61 | 184.9 ± 37.97 | 0.094** |
| * Fissure exact test | , | <u> </u> | |

^{**} Independent sample T test

Although supracoronary repair showed the highest association with postoperative stroke, the relationship was statistically insignificant (p = 0.123). Similarly, insignificant association was found between intraoperative cardiac arrest and postoperative stroke (p = 0.255).

Conversely, retrograde cerebral perfusion during surgery was significantly associated with postoperative stroke (p = 0.001). Additionally, femoral cannulation emerged as a notable risk factor for stroke. In contrast, venous cannulation was insignificantly associated with stroke occurrence (p = 0.598).

Table (7): Showing the relation between postoperative findings and postoperative stroke in patients (n=50).

| Variable | Postoperative stroke | | P value |
|--------------------|----------------------|-----------|---------|
| | Yes (n=8) | No (n=42) | |
| Post-operative GCS | | | |
| GCS 15 | 0 | 42 (84) | <0.001* |
| GCS 14-4 | 2 (25) | 0 | |
| Coma (GCS 3) | 6 (75) | 0 | |
| Mortality | 6 (75%) | 4 (9.5) | 0.01* |

* Fissure exact test.

GCS: Glasgow Coma Scale

Three-quarters of patients with postoperative stroke were comatose, with a Glasgow Coma Scale (GCS) score of 3, while the remaining quarter exhibited impaired consciousness (25%, 2 patients). Patients experiencing a stroke after surgery had a much greater death risk than those who did not.

3. DISCUSSION

Very few people ever recover from the potentially fatal illness known as acute aortic dissection (3). Aortic dissection, pericardial tamponade, aortic regurgitation, endorgan malperfusion, and severe heart failure are consequences that patients often die from if surgical repair is not performed promptly (4). Various brain injury mechanisms can occur during aortic dissection. According to multicenter registers like the NORCAAD registry, individuals who have preoperative cerebral malperfusion have a threefold higher risk of stroke compared to patients with other types of malperfusion. However, coma rates do not follow the same pattern, indicating that different injury mechanisms are at work (5).

Consistent with earlier research indicating a 12% postoperative stroke rate and a 15.8% prevalence of permanent postoperative neurologic injuries in patients having surgery for acute type A aortic dissection, our study found a 16% incidence of postoperative stroke (Dumfarth J et al., 6; Most H et al., 7). Also, patients who had a stroke after surgery were older than those who didn't (p=0.001), which is in line with what another research found: that patients who presented with both aortic dissection and a stroke tended to be older (Bossone E et al., 8).

According to research by (Zhou Y et al.) (9), in-hospital mortality was higher for patients with type B acute aortic dissection who had raised triglyceride levels, suggesting that aortic dissection patients with higher triglycerides had worse outcomes. A risk factor for in-hospital mortality was found to be the triglyceride-to-high-density lipoprotein (HDL) ratio.

Stroke patients had considerably higher blood triglyceride levels than the non-stroke group (p<0.001), according to the present research. Liu P et al. (10), in a similar vein, found that patients whose outcomes were poor had much higher blood triglyceride levels than the control group.

A recent research conducted by Carter et al. (2) shown that having the supra-aortic arteries partially or completely blocked is a strong indicator of cerebral malperfusion after surgery. Stroke was more likely to occur in individuals with carotid substantial stenosis (\geq 50%) in the present research (p \leq 0.007). Smith et al. (6) found that patients who had a stroke after surgery were more likely to have the carotid artery partially or completely blocked before the procedure, which is in agreement with our results.

Patient survival and neurological prognosis may be influenced by surgical treatment decisions (White et al., 11). The present analysis found no statistically significant connection between postoperative stroke and supra-coronary repair procedures (p=0.045), despite the fact that these treatments are most often used to treat stroke after surgery. The frequency of transient neurological impairment after surgery was not correlated with the kind of procedure, according to research by Green et al. (1).

Circulatory arrest was not shown to be a risk factor for postoperative stroke in our investigation. Okita Y et al. (12) found no association between increased mortality or stroke in patients having surgery for aortic arch aneurysms and prolonged (>60 minutes) severe hypothermic circulatory arrest with retrograde cerebral perfusion; our results are in line with theirs. Nevertheless, more research is necessary due to the high incidence of temporary delirium. Alternatively, Reich DL et al., (13) discovered that memory and fine motor impairments, as well as an extended hospital stay, were linked to profound hypothermic circulatory arrests that lasted 25 minutes or more, in addition to older age.

Despite the widespread use of antegrade cerebral perfusion, the optimal location for arterial cannulation is still up for discussion. Our study found a strong correlation between retrograde cerebral perfusion and postoperative stroke (p=0.001), which is in line with Etz CD et al. (14), who also looked at the effects of antegrade and retrograde perfusion in AAD patients but found no differences in early postoperative outcomes depending on perfusion strategy. Both the antegrade and retrograde cerebral perfusion groups did not show any significant differences in neurological consequences, such as stroke, according to AuthorE et al., (15), Tokuda Y et al., and Stamou SC et al. (16).

The incidence of postoperative stroke was significantly higher in patients who had femoral arterial cannulation (p=0.012*). Patients who had a stroke after surgery were more likely to have femoral artery access, which is in line with the results of a research by AuthorG et al., (6). Patients who are in a more critical state when admitted may need arterial access via the groin vessels more quickly and easily, which might explain this link.

This research did not find an ideal arterial cannulation location to reduce the incidence of postoperative stroke in ATAAD

patients, hence the data cannot be used to make a definitive determination.

The most common complication after ATAAD surgery is stroke. Stroke patients had a much higher death rate (75% vs. 16.67%; p=0.04) than stroke-free individuals in our research. Research by Dumfarth J et al., (6), Most H et al., (7), and Bossone E et al., (8) found similar patterns, indicating that patients with postoperative stroke had increased rates of death while hospitalized. New neurological impairment after surgery was also associated with an increased risk of death within 30 days, according to the GERAADA registry (Conzelmann LO et al., 1).

Our best guess is that postoperative stroke isn't the only factor that reduces hospital survival rates; rather, it's a mix of preoperative and postoperative variables.

4. LIMITATIONS

This research represents the results of a single center's experience with a limited sample of patients. Another issue that has to be mentioned is the relatively short period of the study's follow-up.

Strengths

One of the study's strengths is its prospective design.

5. CONCLUSION

A higher mortality rate was associated with postoperative stroke in patients with Acute Type A Aortic Dissection (ATAAD). The serum triglyceride levels of patients who experienced postoperative stroke were substantially higher than those who did not suffer a stroke, and they were elderly. In addition, patients who experienced substantial stenosis (≥50%) in either the left or right carotid arteries were at an increased risk of developing a postoperative stroke. These patients also experienced a higher incidence of postoperative stroke as a result of the use of femoral arterial cannulation and retrograde cerebral perfusion

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