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CARDIOVASCULAR SURGERY AND INTERVENTIONS

Prognostic value of inflammatory indices and end-organ damage in predicting perioperative mortality following emergent repair of type A aortic dissection

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ABSTRACT

Objectives: This study aimed to evaluate whether preoperative biochemical markers and inflammatory indices, in conjunction with established clinical risk factors, can serve as reliable predictors of early postoperative mortality in patients undergoing surgery for type A aortic dissection (TAAD).

Patients and methods: In this retrospective cohort analysis conducted at a single institution, a total of 156 consecutive adult patients who underwent urgent surgical intervention for TAAD were included. The cohort was stratified into the mortality group (n=42) and the survivor group (n=114). Demographic, clinical, intraoperative, and laboratory variables were analyzed and compared between groups. Independent predictors of mortality were determined using multivariable logistic regression analysis.

Results: The overall perioperative mortality rate was 26.9%. Univariate analysis demonstrated that the mortality group had significantly higher rates of preoperative malperfusion (23.8% *vs.* 1.7%, p<0.001) and aortic rupture (21.4% *vs.* 3.5%, p<0.001). Non-survivors demonstrated significantly higher levels of preoperative inflammatory markers, including C-reactive protein (p<0.001), neutrophil-to-lymphocyte ratio (p=0.039), and red cell distribution width (p=0.001), and higher serum creatinine concentrations (p<0.001). However, multivariable analysis identified malperfusion (odds ratio [OR]: 20.707, p=0.007), aortic rupture (OR: 9.525, p=0.002) and elevated creatinine levels (OR: 1.785, p=0.022) as the only independent predictors of mortality. Inflammatory markers were no longer statistically significant (p>0.05).

Conclusion: In acute TAAD, perioperative mortality is primarily driven by end-organ malperfusion, aortic rupture, and renal dysfunction. Although inflammatory markers reflect disease severity, they do not independently predict early mortality in the presence of organ failure.

Keywords: Creatinine, predictor, mortality, type A aortic dissection.

Type A aortic dissection (TAAD), initially classified by Stanford in 1970, is a catastrophic and life-threatening cardiovascular emergency that necessitates rapid multidisciplinary intervention.[1] Left untreated, TAAD is associated with an exceptionally high mortality rate; historical and contemporary registries indicate that approximately 40% of patients succumb to the condition prior to hospital admission, with early mortality increasing by 1% to 2% per hour following symptom onset.[2] Accordingly, urgent surgical intervention remains the definitive standard of care. However, despite significant advancements in surgical techniques, cardiopulmonary bypass (CPB) technologies, and intensive care management over the past decades, the perioperative mortality rate for patients undergoing emergency TAAD surgery remains alarmingly

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high, ranging between 15% and 30% in major international registries.[3] This persistently high mortality burden underscores the critical need for novel, easily accessible prognostic biomarkers to preoperatively risk-stratify patients and identify high-risk individuals who might require tailored therapeutic and hemodynamic strategies.

The pathogenesis and early progression of TAAD are intrinsically linked to a profound systemic inflammatory response syndrome (SIRS), triggered by the exposure of circulating blood to the deeper layers of the dissected aortic media and subsequent visceral or peripheral tissue ischemia.[4] Identifying factors that predict perioperative morbidity in patients undergoing TAAD surgery is important because it allows for the development of preventive and therapeutic strategies. In addition to known determinants of mortality such as advanced age, malperfusion, and unstable preoperative condition, the prognostic roles of numerous hematological, biochemical, and inflammatory parameters in patients undergoing TAAD surgery have recently been investigated. Therefore, these parameters are guiding factors for identifying potential interventions and prospective treatment targets for high-risk patients.[5-7]

Nevertheless, the existing literature presents inconsistent findings. Therefore, this study sought to assess the predictive significance of various hematological, biochemical, and inflammatory markers for perioperative mortality in patients undergoing surgery for TAAD, while also examining additional clinical variables that may contribute to risk stratification.

PATIENTS AND METHODS

Ethical Considerations

The study protocol received approval from the Institutional Clinical Research Ethics Committee of University of Health Sciences Türkiye, Bursa City Hospital (approval no: 2023-19/12; date: November 22, 2023). All procedures were carried out in accordance with the ethical standards set forth in the Declaration of Helsinki. Prior to enrollment, comprehensive information regarding the purpose of the study and surgical interventions was provided to all patients or their legal surrogates; both verbal and written informed consent were obtained.

Study Population and Design

This study was conducted as a retrospective, single-center observational cohort analysis. A total of 156 adult patients who underwent urgent surgical repair for TAAD at our institution from July 2019 to October 2023 were retrospectively evaluated. The cohort was stratified into two groups based on perioperative outcomes: The mortality group (n=42) and the survivor group (n=114).

Baseline demographic data—including age, sex, height, weight, and body mass index (BMI)—and relevant comorbid conditions such as diabetes mellitus, chronic obstructive pulmonary disease (COPD), hypertension, and chronic renal insufficiency were systematically retrieved from the institution's electronic medical record system. Preoperative hematological parameters, biochemical markers (the inflammatory prognostic index [IPI] was calculated using the formula: (C-reactive protein [CRP] × neutrophil-to-lymphocyte ratio [NLR])/albumin) and echocardiographic findings recorded upon hospital admission were also analyzed. Malperfusion was defined by preoperative clinical findings, computed tomography angiography findings, and, when present, evidence of end-organ ischemia or dysfunction. Aortic rupture was defined according to preoperative imaging or intraoperative findings indicating free rupture, pericardial tamponade, mediastinal hematoma, or hemothorax. In addition, perioperative variables—including CPB duration, aortic cross-clamp (ACC) time, lengths of stay in the intensive care unit (ICU) and overall hospitalization, incidence of postoperative cerebrovascular events (CVE), and requirement for re-exploration for bleeding—were systematically recorded. Perioperative mortality was defined as all-cause in-hospital mortality during the index hospitalization. Patients with a history of median sternotomy (redo surgery), documented malignancies, active infectious diseases or sepsis, and underlying autoimmune, hematological, or chronic inflammatory disorders were excluded to prevent confounding of inflammatory biomarkers.

Surgical Approach

All surgical interventions were carried out under general anesthesia through a conventional median sternotomy. Before cannulation, systemic anticoagulation was administered using unfractionated heparin (350 IU/kg) to ensure an activated clotting time (ACT) exceeding 400 seconds. The right subclavian artery was the primary vessel for arterial cannulation to facilitate antegrade cerebral perfusion; femoral artery cannulation was reserved for cases in which the subclavian approach was unsuitable. Venous return was established via a two-stage right atrial cannula to complete the CPB circuit.

Throughout CPB, a non-pulsatile flow of 2.0-2.5 L/min/m² was maintained, with a target mean arterial pressure of 50-70 mmHg and a hematocrit of 20-25%. After placement of the ACC, diastolic cardiac arrest was achieved via antegrade cardioplegia, enabling comprehensive assessment of the aortic root, valve, ascending aorta, and aortic arch.

The subsequent surgical strategy was tailored to the extent of the dissection and valvular pathology. For intimal tears confined to the ascending aorta with preserved aortic valve morphology and intact coaptation, a supracoronary ascending aortic replacement was executed using an appropriately sized Dacron graft. In cases where the dissection involved the aortic arch or its branch vessels, the repair was extended distally. Concomitant aortic valve repair or replacement was undertaken when intrinsic valvular structural abnormalities were detected. Distal anastomoses were routinely performed using the open technique during a brief period of circulatory arrest, with selective antegrade cerebral perfusion applied for neuroprotection under moderate hypothermia (24-28 °C). Upon completion of the anastomoses, patients were systematically rewarmed, weaned from CPB, and decannulated. Heparin was reversed using protamine sulfate (1-1.3 mg per 1 mg of administered heparin) to normalize the ACT. All surgical procedures were performed by a consistent, dedicated cardiovascular surgical team. Following surgery, patients were promptly admitted to the cardiovascular ICU for continuous hemodynamic surveillance.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 23.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using the independent samples t-test. Non-normally distributed continuous variables were reported as medians (minimum-maximum) and compared using the Mann-Whitney U test. Categorical variables were presented as frequencies and percentages,

with inter-group comparisons conducted via the chi-square test. Variables that were statistically significant in univariate analysis were included in a multivariable logistic regression model to identify independent predictors of mortality. Categorical variables were entered into the model using "absence" as the reference category; perioperative mortality was coded as the event (1) and survival as (0). Although CPB duration is an intraoperative variable reflecting surgical complexity, it was included in the multivariable model to provide a more comprehensive assessment of factors associated with perioperative mortality in emergent surgery. Receiver operating characteristic (ROC) curve analysis was employed to establish optimal cut-off values for these predictors. For all statistical tests, a two-sided p-value of <0.05 was considered indicative of significance.

RESULTS

A total of 156 patients were included in the study: 42 in the mortality group and 114 in the survivor group. Patients in the mortality group were significantly older compared to the survivor group (65.6±12.8 years *vs.* 58.8±11.1 years, p=0.002) (Table 1). No significant differences were observed between the groups with respect to sex, height, weight, BMI, or the prevalence of preoperative comorbidities, including hypertension, diabetes mellitus, COPD, and chronic renal failure (all p>0.05) (Table 1).

With respect to preoperative clinical severity, the incidences of malperfusion and aortic rupture were markedly higher in the mortality group. Preoperative malperfusion was present in 23.8% of non-survivors compared with 1.7% of survivors (p<0.001). The incidence of aortic rupture was 21.4% in the non-survivor group, compared with 3.5% in the survivor group (p<0.001) (Table 1).

Intraoperatively, while ACC durations were comparable between the cohorts, the CPB duration was significantly prolonged in the mortality group (227.8±116.2 min *vs.* 166.2±83.8 min, p=0.003) (Table 1). Postoperatively, durations of both ICU stay and total hospital stay were significantly shorter in the mortality group, primarily because of early postoperative mortality (p=0.001 and p<0.001, respectively). The frequency of re-exploration for bleeding and the occurrence of postoperative CVE were comparable between the two groups, with no statistically significant differences observed (p>0.05) (Table 1).

Preoperative laboratory analyses revealed heightened systemic inflammation and tissue stress in the mortality group (Table 2). The red cell distribution width (RDW) (p=0.001), CRP levels (p<0.001), and the NLR (p=0.039) were significantly elevated in patients who did not survive. Furthermore, markers of renal impairment, specifically urea (p=0.040) and creatinine (p<0.001), were significantly higher in the mortality cohort (Table 2). There were no significant differences between the groups in hemoglobin levels, white blood cell counts, platelet parameters, liver function tests (aspartate aminotransferase [AST], alanine aminotransferase [ALT]), or composite inflammatory markers, including the platelet-to-lymphocyte ratio (PLR) and systemic immune-inflammation index (all p>0.05) (Table 2). Extremely elevated

Table 1. Perioperative patient characteristics

Variable	Mortality group (n=42)	Survivor group (n=114)	p-value
Age (year)	65.6±12.8	58.8±11.1	0.002*
Gender (female)	15 (35.7%)	38 (33.3%)	0.781
Height (cm)	168.7±9.7	168.3±8.8	0.794
Weight (kg)	80.2±13.8	81.7±13.9	0.534
BMI (kg/m^2)	28.5±5.8	28.7±4.6	0.846
Hypertension	34 (80.9%)	84 (73.6%)	0.300
Diabetes mellitus	5 (11.9%)	18 (15.7%)	0.405
COPD	3 (7.1%)	8 (7.0%)	0.662
CRF	3 (7.1%)	5 (4.3%)	0.332
Malperfusion	10 (23.8%)	2 (1.7%)	<0.001*
Rupture	9 (21.4%)	4 (3.5%)	<0.001*
ACC duration (min)	101 (41-384)	90.5 (25-244)	0.135
CPB duration (min)	227.8±116.2	166.2±83.8	0.003*
ICU stay duration (day)	2 (0-14)	3 (2-19)	0.001*
Hospital stay duration (day)	2 (0-14)	7 (5-43)	<0.001*
Re-exploration for bleeding	7 (16.6%)	9 (7.8%)	0.098
Postoperative CVE	5 (11.9%)	8 (7.0%)	0.381

ACC: Aortic cross-clamp; BMI: Body mass index; COPD: Chronic obstructive pulmonary disease; CPB: Cardiopulmonary bypass; CRF: Chronic renal failure; CVE: Cerebrovascular event; ICU: Intensive care unit.

Table 2. Laboratory data

Variable	Mortality group (n=42)	Survivor group (n=114)	p-value
Hemoglobin (g/dL)	11.5±2.7	11.8±2.6	0.786
RDW (fL)	44.3 (38.1-68.7)	42.0 (34.6-60.3)	0.001*
WBC (10^3/µL)	13.2±4.6	12.2±4.7	0.284
Neutrophil (10^3/µL)	10.4±4.4	9.4±4.5	0.214
Lymphocyte (10^3/µL)	1.09 (0.27-5.53)	1.48 (0.24-36.1)	0.094
Monocyte (10^3/µL)	0.76 (0.18-1.80)	0.75 (0.18-6.8)	0.829
Eosinophil (10^3/µL)	0.03 (0-0.79)	0.04 (0-1.00)	0.394
Platelet (10^3/µL)	166.8±108.2	192.5±83.1	0.118
PDW (fL)	13.5±3.2	12.9±3.2	0.242
MPV (fL)	11.0±1.1	10.5±0.8	0.265
Plateletcrit (%)	0.18±0.10	0.19±0.08	0.438
CRP (mg/L)	12.0 (1.2-178.5)	4.5 (0.5-279.4)	<0.001*
Urea (mg/dL)	41.4 (15-178.5)	37.0 (16.3-171.4)	0.040*
Creatinine (mg/dL)	1.37 (0.45-9.9)	0.95 (0.48-8.0)	<0.001*
AST (IU/L)	30 (15-10075)	28 (9-1237)	0.123
ALT (IU/L)	20.5 (7-5483)	20 (5-485)	0.376
Albumin (g/L)	30.7±8.8	33.3±7.6	0.097
NLR	8.5 (1.0-53.6)	7.0 (0.13-26.9)	0.039*
PLR	101.2 (17.6-915.2)	126.7 (5.5-929.4)	0.541
SII	1184 (93-22578)	1090 (27-8513)	0.689

ALT: Alanine transaminase; AST: Aspartate aminotransferase; CRP: C-reactive protein; MPV: Mean platelet volume; NLR: Neutrophil-to-lymphocyte ratio; PDW: Platelet distribution width; PLR: Platelet-to-lymphocyte ratio; RDW: Red cell distribution width, SII: Systemic immune-inflammation index; WBC: White blood cell.

Table 3. Multivariable logistic regression analysis

Variable	Beta	Standard error	Wald	Expected beta	p-value
Age	0.033	0.022	2.255	1.034	0.133
CPB duration	0.004	0.002	3.158	1.004	0.076
Malperfusion	3.030	1.126	7.238	20.707	0.007*
Rupture	2.254	0.739	9.315	9.525	0.002*
NLR	0.029	0.036	0.656	1.029	0.418
RDW	0.083	0.054	2.339	1.087	0.126
CRP	0.003	0.005	0.328	1.003	0.567
Urea	-0.009	0.010	0.813	0.991	0.367
Creatinine	0.579	0.253	5.249	1.785	0.022*

CPB: Cardiopulmonary bypass; CRP: C-reactive protein; NLR: Neutrophil-to-lymphocyte ratio; RDW: Red cell distribution width.

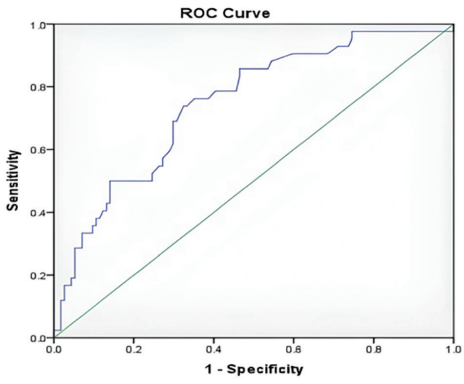
AST and ALT values observed in several patients were rechecked in the institutional database and considered to reflect severe hypoperfusion-related hepatic injury in these critically ill patients.

A multivariable logistic regression analysis was conducted to identify independent predictors of perioperative mortality by including variables that were significant in the univariate analysis (age, CPB duration, malperfusion, rupture, NLR, RDW, CRP, urea, and creatinine) (Table 3). Odds ratios (ORs) are presented according to the predefined reference categories used in the logistic regression model. As summarized in Table 3, the model identified the following parameters as independent predictors of mortality:

- Malperfusion (OR: 20.707, p=0.007)
- Rupture (OR: 9.525, p=0.002)
- Elevated creatinine (OR: 1.785, p=0.022).

Conversely, age, CPB duration, inflammatory markers (NLR, RDW, CRP) and urea lost their statistical significance in the multivariable model (p>0.05) (Table 3).

The predictive utility of preoperative serum creatinine for mortality was assessed through ROC curve analysis.



Area under the curve (AUC)	Optimum cut-off value	Sensitivity	Specificity	P value
0.746 (0.660-0.831)	1.14	69.0%	69.3%	<0.001

Figure 1. Receiver operating characteristic (ROC) curve for creatinine.

In the ROC analysis, creatinine had an optimal threshold of 1.14 with 69% sensitivity and 69.3% specificity (AUC=0.746 (0.660-0.831), p<0.001; Figure 1).

DISCUSSION

Despite significant advancements in surgical and anesthetic techniques, acute TAAD remains a catastrophic cardiovascular emergency with substantial perioperative mortality.[8,9] The main finding of this study was that, although preoperative inflammatory markers—including the NLR, RDW, and CRP—were associated with mortality in univariate analysis, they did not retain independent predictive significance in the multivariable model. Instead, the true independent predictors of early surgical mortality were preoperative malperfusion syndrome, aortic rupture, and elevated serum creatinine. These results clearly indicate that the prognosis in TAAD surgery is fundamentally dictated by the severity of gross end-organ damage rather than the isolated magnitude of systemic inflammation.

Extensive data from the literature robustly support our findings. Comprehensive analyses from the German Registry for Acute Aortic Dissection Type A have demonstrated that the presence of preoperative malperfusion exponentially increases surgical mortality and the rate of postoperative complications.[8] The exceptionally high mortality risk observed in patients with malperfusion in our cohort can be attributed to the dissection flap obstructing the arterial supply to vital organs, leading to irreversible ischemic shock.[10] Similarly, aortic rupture is directly associated with massive hemorrhage, cardiac tamponade, and profound cardiogenic shock, establishing it as one of the most formidable independent causes of death across major international centers.[8]

Another potent, independent predictor of perioperative mortality identified in our study was an elevated preoperative creatinine level. In the setting of aortic dissection, acute kidney injury is frequently triggered by either direct involvement of the renal arteries by the dissection flap or global hypoperfusion secondary to shock.[11] Furthermore, indispensable surgical modalities such as prolonged CPB and hypothermic circulatory arrest completely exhaust the already compromised baseline renal reserve. It has been unequivocally established in the literature that renal dysfunction—whether pre-existing or exacerbated by the deleterious effects of extracorporeal circulation—directly amplifies mortality following major aortic arch surgery.[11,12] Because of the retrospective design and the emergency setting, the present study could not determine whether elevated creatinine reflected chronic kidney disease, acute renal malperfusion, or shock-related hypoperfusion. The rationale for investigating the IPI and its components (NLR, CRP, RDW) lies in their ability to reflect the SIRS, which is initiated when subendothelial tissues are exposed to circulating blood following an intimal tear.[13,14]

Consequently, our univariate analysis revealed significantly elevated levels of these markers in the mortality group. However, their loss of independent predictive value when organ damage parameters (malperfusion and creatinine) were introduced into the multivariable model highlights a critical pathophysiological reality: Once tissue necrosis, shock, and major organ ischemia are established, the cellular-level inflammatory response becomes a secondary reaction.[15] Moreover, the profound systemic inflammation induced by CPB itself often overrides the preoperative baseline inflammatory status.[16] During the acute crisis of TAAD, the ultimate determinant of patient survival is whether vital organs are adequately perfused, rather than the mere presence of inflammation. Therefore, while inflammatory parameters serve as excellent prognostic tools in chronic cardiovascular or oncological diseases, they lag behind end-organ perfusion defects in catastrophic emergencies such as TAAD, where every second counts.[17-20] Recent studies have also shown that inflammatory markers may be associated with adverse postoperative outcomes, including atrial fibrillation, among patients undergoing cardiovascular surgery. However, similar to our findings, these markers do not always remain independent predictors after adjustment for major clinical variables.[7] Conversely, other reports evaluating perioperative inflammatory indices in cardiac surgery populations have demonstrated that certain inflammatory parameters may independently predict mortality and morbidity under specific clinical conditions.[21] These findings suggest that the prognostic significance of inflammatory markers may vary according to the patient population, timing of biomarker assessment, and the severity of end-organ injury.

This study has several limitations. First, the retrospective single-center design and the relatively modest sample size (n=156) may restrict the generalizability of our findings and limit the statistical power of multivariable analyses. The relatively low number of outcome events compared with the number of variables included in the multivariable model may pose a risk of overfitting and should be considered when interpreting the results. Second, inflammatory markers were evaluated from a single preoperative blood sample collected at admission. Because the systemic inflammatory response in TAAD is a highly dynamic process, this single "snapshot" precludes the assessment of longitudinal biomarker kinetics and their postoperative variations. Biomarkers in acute aortic syndromes exhibit significant temporal variations.[22] In addition, specific malperfusion territories (cerebral, coronary, mesenteric, renal, or extremity) and individual rupture subtypes were not analyzed separately, which may have influenced the prognostic impact of these variables. The inclusion of intraoperative variables such as CPB duration in the multivariable model may limit strict preoperative risk stratification, although it provides additional

insight into overall perioperative risk. Third, detailed operative variables such as extent of aortic repair, circulatory arrest time, cerebral perfusion strategy, nadir temperature, and cannulation site were not included in the analysis; this omission may have influenced perioperative outcomes. Finally, due to the emergent nature of the disease and the retrospective study design, the exact time intervals from symptom onset to hospital admission and to surgical intervention could not be reliably standardized or analyzed. This factor may have influenced the degree of end-organ dysfunction and inflammatory marker elevation observed at presentation. The prognostic impact of different malperfusion territories and rupture subtypes may not be uniform, and this issue warrants further investigation in larger prospective studies. Future multicenter, prospective studies evaluating longitudinal changes in these markers during the postoperative period are warranted to draw more definitive conclusions.

Malperfusion syndrome, aortic rupture, and elevated preoperative creatinine levels are the most robust independent risk factors for early surgical mortality in patients undergoing emergent repair for TAAD. Although inflammatory markers such as the IPI, NLR, and CRP reflect the severity of the dissection, they lose their independent prognostic utility in clinical scenarios dominated by major ischemic insult and organ failure, particularly renal dysfunction. When stratifying preoperative risk in TAAD patients, clinicians must prioritize assessment of gross organ malperfusion and baseline renal reserve.

Ethics

Ethics Committee Approval: The study protocol received approval from the Institutional Clinical Research Ethics Committee of University of Health Sciences Türkiye, Bursa City Hospital (approval no: 2023-19/12; date: November 22, 2023).

Informed Consent: Both verbal and written informed consent were obtained.

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For transparency, the authors note that an artificial intelligence-assisted language model (ChatGPT, OpenAI) was utilized to support text editing. This assistance was limited to linguistic refinement; all scientific content, critical analysis, and final editorial decisions were made exclusively by the authors.

Footnotes

Authorship Contributions

Surgical and Medical Practices: D.Ç., H.G., A.Y., Y.V., S.H., A.Mü.; Concept: D.Ç., A.Y., G.K., T.T., A.M., A.Mü.; Design: D.Ç., A.Y., G.K., T.T., A.M.; Data Collection or Processing: A.Y., Y.V., S.H.; Analysis or Interpretation: H.G., A.Y., S.H.; Literature Search: H.G., G.K., A.Mü.; Writing: D.Ç., H.G., A.Y., G.K., A.Mü.

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