Type A acute aortic dissection complicated by preoperative myocardial infarction: Insights from the International Registry of Acute Aortic Dissection

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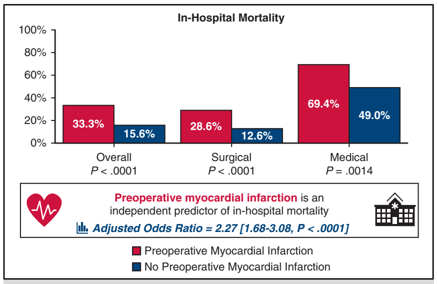
ABSTRACT

Objectives: Type A acute aortic dissection can cause preoperative myocardial infarction due to coronary malperfusion or hemodynamic collapse. This study aims to characterize the presentation, management, and outcomes of patients with concomitant dissection and myocardial infarction.

Methods: A total of 5762 patients with type A dissection were evaluated from the International Registry of Acute Aortic Dissection from 1996 to 2024. Patients with endovascular management, iatrogenic dissection, or insufficient data were excluded. Patients with preoperative myocardial infarction (n = 662, 10.8%) were compared with those without preoperative myocardial infarction (n = 5140, 89.2%).

Results: Age and time to diagnosis and treatment were similar between groups. Patients with preoperative myocardial infarction more frequently presented with chest pain (88.6% vs 82.0%, $P < .0001$) and hemodynamic shock (9.5% vs 6.4%, $P = .009$). Diagnostic imaging revealed more coronary involvement (39.4% vs 14.3%, $P < .0001$). Surgery more often involved coronary artery bypass grafting (25.4% vs 8.3%, $P < .0001$) and root replacement (43.9% vs 35%, $P = .0003$) in the preoperative myocardial infarction group. Patients with preoperative myocardial infarction had higher rates of low output syndrome (9.5% vs 2.2%, $P < .0001$), renal failure (31.2% vs 21.1%, $P < .0001$), coma (5.4% vs 2.8%, $P = .006$), and in-hospital mortality (33.3% vs 15.6%, $P < .0001$). However, 4-year survival was similar ($P = .810$). Preoperative myocardial infarction remained an independent predictor of in-hospital mortality on multivariable analysis (odds ratio, 2.27, $P < .0001$).

Conclusions: Patients with type A acute aortic dissection and preoperative myocardial infarction experienced higher complication and in-hospital mortality rates. Earlier recognition and targeted surgical strategies may improve outcomes. (JTCVS Open 2026;29:101508)



Preoperative MI increases in-hospital mortality in patients with TAAAD.

CENTRAL MESSAGE

Preoperative MI is associated with worse postoperative complications and in-hospital mortality in the setting of TAAAD.

PERSPECTIVE

Acute MI can present alongside acute aortic dissection, complicating diagnosis and management. Patients with preoperative MI have higher cardiac compromise, cerebral and mesenteric malperfusion, and in-hospital mortality. Early recognition and prompt surgical intervention to restore coronary and systemic perfusion may improve outcomes.

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Abbreviations and Acronyms

CABG	= coronary artery bypass grafting
EKG	= electrocardiography
IRAD	= International Registry of Acute Aortic Dissection
MI	= myocardial infarction
TAAAD	= type A acute aortic dissection

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Type A acute aortic dissection (TAAAD) is a rare but life-threatening condition requiring emergency surgery.[1] Concomitant acute myocardial infarction (MI) occurs in 1% to 7% of TAAAD cases.[1-4]

Neri and colleagues[5] describe several mechanisms by which proximal dissection can lead to coronary compromise, including dynamic compression of the coronary ostia by a dissection flap, dissection extension into the coronary arteries, and direct ostial obstruction. Preoperative coronary malperfusion in TAAAD is associated with increased in-hospital mortality, further necessitating urgent surgery to restore perfusion and prevent irreversible myocardial damage.[5,6]

Despite overlapping symptoms, acute MI is 800 times more common than TAAAD.[1] Thus, MI-associated electrocardiography (EKG) changes may obscure the underlying TAAAD and delay diagnosis.[7] As mortality increases up to 1% per hour after TAAAD onset, early concurrent diagnosis of both conditions is critical.[8,9] Case reports suggest standard MI treatments may increase mortality risk in the setting of TAAAD.[10-12] Furthermore, the associated myocardial injury of MI may impair cardiac function and contribute to worse outcomes.

The risk factors and prognostic implications of concomitant preoperative MI in TAAAD remain poorly defined. Additionally, the impact of MI on management and outcomes has yet to be characterized. This study uses the International Registry of Acute Aortic Dissection (IRAD) to investigate the incidence, associated risk factors, and clinical impact of preoperative MI on TAAAD management and outcomes.

MATERIAL AND METHODS

The IRAD is a multinational registry designed to evaluate patients with acute aortic dissection and intramural hematoma. Cases are entered prospectively at hospital presentation or retrospectively through record review. Data are forwarded to the coordinating center at the University of Michigan and evaluated for face validity and completeness. Further details regarding IRAD's methodology have been described.[1] Ethics board approval was granted at participating centers and the coordinating center (Michigan Medicine HUM00043980, last approved May 2025), with consent obtained according to each board's recommendations. Waivers of consent were granted at select sites on the basis of a retrospective registry with no more than minimal risk to subjects.

Study Design

This analysis evaluated 10,138 patients with TAAAD defined according to the Stanford classification presenting from January 1996 to June 2024.[13] Patients with TAAAD with iatrogenic dissection or endovascular repair and those without sufficient data to determine preoperative MI status were excluded (Figure E1).

Preoperative MI was defined using 2 variables: (1) MI on EKG or (2) a documented preoperative MI complication. EKG evidence of new-onset MI was characterized as development of new Q waves more than 0.03 seconds wide or at least one-third of the QRS complex in 2 or more contiguous leads, or early-phase ST-segment elevation more than 1.0 mm. Preoperative MI diagnosis was based on EKG criteria or echocardiography and laboratory abnormalities (Table E1).

Statistical Analysis

Comparisons were performed using Fisher exact test for categorical variables and Kruskal–Wallis and Mood's median tests for continuous variables. With additions to data collection instruments over 25 years, some variables lack entries. Denominators reflect data available, and missing entries were not defaulted to null. The extent of missingness was 5% to 34% for medical history, 3% to 44% for presenting symptoms, and 4% to 13% for preoperative complications; 6% to 16% had missing data on surgical variables among surgically managed patients. To prepare for multivariable modeling, multiple imputations were performed with 100 imputed datasets for missing data on history, presenting symptoms, and preoperative complications.

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Both in-hospital and 30-day mortality were reported. For 30-day mortality, patients were included if they were admitted for 30 days or longer, they died in the hospital within 30 days after admission, or they were admitted for less than 30 days and had follow-up death status recorded. Kaplan–Meier estimator assessed postdischarge survival among patients discharged alive. Follow-up data were collected in 51% and 49% of patients with MI and no MI, respectively, with comparable median follow-up length (35.5 and 35.6 months, respectively). Log-rank test was used for comparing survival curves between the 2 groups. Survival curves were curtailed at year 4 when at least 10% of the patients discharged remained at risk.

Multivariable analysis was limited to surgically managed patients. Generalized linear modeling was conducted with random intercept to assess factors associated with in-hospital death. Included in the model were demographics, history, presenting signs, preoperative complications, concomitant MI status, and surgical details. Inclusion of surgical variables reduced the cohort size for multivariable modeling due to missing data, because surgical variables were not part of multiple imputation model. To adjust for potential temporal trend, one indicator of time period was created, reflecting 3 periods with the approximately equal number of patients: (1) 1996 to 2010, (2) 2010 to 2018, and (3) 2018 to 2024. Given evidence that in-hospital mortality declined over time,[14] a sensitivity analysis was performed by repeating the multivariable model among surgical patients admitted between 2014 and 2024. Model fit measure as well as c-statistic and Brier score assessed modeling performance when variables with large P values were removed to avoid model overfitting. Modeling results were pooled from the 100 imputed datasets.

RESULTS

Of 5762 patients analyzed, 622 (10.8%) had preoperative MI and 5140 (89.2%) had no evidence of preoperative MI. Patients with preoperative MI were more frequently male (71.1% vs 66.1%, $P = .013$) and less frequently White (67.3% vs 71.4%, $P = .043$, Table 1). Hypertension (79.6% vs 74.8%, $P = .015$), atherosclerosis (21.2% vs 16.4%, $P = .009$), smoking (56.7% vs 48.0%, $P = .002$), and aortic valve disease (14.6% vs 10.6%, $P = .023$) were more prevalent in patients with preoperative MI.

Patients with preoperative MI more frequently presented with chest pain (88.6% vs 82.0%, $P < .0001$, Table E2). Syncope (19.9% vs 15.3%, $P = .008$), hemodynamic shock (9.5% vs 6.4%, $P = .009$), and congestive heart failure (12.1% vs 5.7%, $P < .0001$) were higher in patients with preoperative MI. Patients with preoperative MI were more likely to present with or develop additional preoperative complications (Table 2).

Although computed tomography angiography was most frequently used, patients with preoperative MI were more likely to undergo echocardiography first (28.4% vs 16.7%, $P < .0001$, Table E3). Median time (hours) from hospital arrival to diagnosis was similar (2.8 vs 2.6, $P = .413$). Among surgical patients, time to treatment was also similar (4.0 vs 3.8, $P = .502$).

On imaging, pericardial effusion (44.0% vs 42.5%, $P = .574$) and proximal extent of dissection to aortic root (60.6% vs 56.4%, $P = .072$) were similar. Coronary artery compromise was observed in 39.4% of patients with preoperative MI and 14.3% in those without ($P < .0001$). Patients with preoperative MI were more likely to receive coronary artery bypass grafting (CABG) (25.4% vs 8.3%, $P < .0001$) and root replacement (43.9% vs 35.0%, $P = .0003$, Table 3).

Patients with preoperative MI experienced more postoperative cardiac complications, including tamponade (9.2% vs 6.0%, $P = .013$) and low output syndrome (9.8% vs 5.5%, $P = .006$, Table 4). Additionally, postoperative acute renal failure, coma (complete unresponsiveness, characterized by the absence of appropriate stimuli response), and respiratory insufficiency were more frequent with preoperative MI. In-hospital mortality was higher with preoperative MI (33.3% vs 15.6%, $P < .0001$), regardless of surgical (28.6% vs 12.6%, $P < .0001$) or medical management (69.4% vs 49.0%, $P = .001$, Figure 1). Similar findings were noted for 30-day mortality (43.8% vs 23.7%, $P < .0001$, Figure E2).

After adjustment, preoperative MI remained an independent predictor for in-hospital mortality in the surgical cohort (odds ratio, 2.27, $P < .0001$, Table 5). Other variables associated with mortality included preoperative cardiac tamponade, mesenteric ischemia, root replacement, concomitant CABG, and presenting in the earliest period. A sensitivity analysis of patients presenting in the latest time period yielded similar results, with preoperative MI remaining a predictor of mortality (Table E4). At 4-year follow-up, postdischarge survival was similar (87.6% with preoperative MI vs 84.6% without, log-rank $P = .805$, Figure 2).

DISCUSSION

TAAAD remains a cardiovascular emergency, and early mortality is high.[15] TAAAD presenting with preoperative MI exacerbates the risk of complications and early mortality, likely due to myocardial malperfusion and systemic hemodynamic instability.[6,16]

Concomitant preoperative MI and TAAAD may occur more frequently than previously believed. A prior IRAD study reported that 5% of patients with TAAAD presented to the hospital with MI; however, this statistic was based on EKG findings and did not consider patients who experienced MI after hospital arrival but before surgery.[17] The current study used broader inclusion criteria that captured EKG, echocardiographic, and cardiac enzyme findings, conferring a higher preoperative MI incidence of 10.8%.

TABLE 1. Demographics and history with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
Age, y (mean ± SD)	60.6 ± 14.1	60.9 ± 14.6	.465
Gender, male	442 (71.1%)	3398 (66.1%)	.013
Race, White	376 (67.3%)	3452 (71.4%)	.043
Hypertension	440 (79.6%)	3696 (74.8%)	.015
Diabetes	58 (11.5%)	490 (10.2%)	.357
Atherosclerosis	104 (21.2%)	778 (16.4%)	.009
Marfan, Ehlers-Danlos, Turner, or Loeys-Dietz Syndrome	15 (3.1%)	157 (3.3%)	.894
Known aortic aneurysm	58 (11.6%)	627 (13.0%)	.401
Prior aortic dissection	21 (4.2%)	185 (3.8%)	.626
Family history of aortic disease	28 (8.0%)	272 (7.7%)	.835
Bicuspid aortic valve	15 (3.1%)	166 (3.6%)	.609
Aortic valve disease*	70 (14.1%)	508 (10.6%)	.023
Cocaine abuse	12 (2.4%)	101 (2.1%)	.626
Ever smoker	217 (56.7%)	1637 (48.0%)	.002
Peripheral arterial disease	19 (4.9%)	116 (3.2%)	.071
COPD	41 (10.4%)	290 (7.8%)	.079
Chronic renal insufficiency	35 (8.8%)	281 (7.5%)	.372
Prior cardiac procedures	66 (13.1%)	502 (10.6%)	.098
CABG	21 (4.2%)	166 (3.5%)	.446
Aortic valve replacement	23 (4.6%)	171 (3.6%)	.265
Mitral valve replacement	7 (1.4%)	52 (1.1%)	.504
Aortic aneurysm/dissection	29 (5.8%)	233 (4.9%)	.389
Catheterization or angiography	112 (23.2%)	372 (8.5%)	<.0001
PTCI	36 (7.6%)	113 (2.6%)	<.0001

MI, Myocardial infarction; *COPD*, chronic obstructive pulmonary disease; *CABG*, coronary artery bypass grafting; *PTCI*, percutaneous transluminal coronary intervention. *Aortic stenosis, insufficiency, or regurgitation grade ≥2.

Coronary artery involvement was observed in 39.4% of patients with preoperative MI, more than double that of patients without preoperative MI (14.3%). The increased prevalence of right coronary involvement specifically aligns with its exposure to elevated wall shear forces at the anterolateral aortic root.[18,19] Survivorship bias also may contribute, because patients with left coronary involvement often experience rapid hemodynamic collapse and are less likely to survive to hospital arrival.[20] However, the majority of preoperative MI cases did not show direct coronary involvement, suggesting that dynamic obstruction, hypotension, dissection-induced shock, or left ventricular

TABLE 2. Preprocedural complications of patients with type A acute aortic dissection with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
Cardiac tamponade	77 (14.6%)	552 (10.8%)	.014
Low output syndrome	36 (9.5%)	77 (2.2%)	<.0001
Acute renal failure	60 (11.3%)	359 (7.1%)	.0009
Limb ischemia	67 (12.6%)	539 (10.6%)	.162
Mesenteric ischemia	32 (6.1%)	130 (2.5%)	<.0001
Spinal cord ischemia	9 (1.7%)	55 (1.1%)	.205
Stroke	38 (7.0%)	183 (3.6%)	.0004
Coma	32 (5.9%)	83 (1.6%)	<.0001
Extension of dissection	44 (8.5%)	210 (4.2%)	<.0001

MI, Myocardial infarction.

TABLE 3. Management of patients with type A acute aortic dissection with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
Management			
Surgical	550 (88.4%)	4706 (91.6%)	.013
Medical	72 (11.6%)	434 (8.4%)	.013
Surgical details			
LV dysfunction at surgery	80 (17.4%)	376 (8.7%)	<.0001
RV dysfunction at surgery	48 (10.5%)	260 (6.0%)	.0006
Cerebral perfusion during circulatory arrest	375 (80.5%)	3344 (77.9%)	.215
Concomitant CABG	117 (25.4%)	356 (8.3%)	<.0001
Root replacement	190 (43.9%)	1393 (35.0%)	.0003
Hypothermic circulatory arrest, median (Q1-Q3), min	33 (24-52)	33 (24-51)	.839
Cardiopulmonary bypass time, median (Q1-Q3), min	212 (168-275)	188 (148-236)	.0005

MI, Myocardial infarction; *LV*, left ventricular; *RV*, right ventricular; *CABG*, coronary artery bypass grafting.

dysfunction may contribute to MI.[21-23] Additionally, given that variables associated with coronary artery disease were higher in the MI cohort, some dissection-related MI cases could be due to underlying coronary stenosis. Signs of peripheral malperfusion were more common, demonstrating that patients with preoperative MI may experience a more profound initial physiologic insult.[24]

TAAAD with preoperative MI often mimics isolated MI, with characteristic symptoms of severe, abrupt-onset chest pain, and ST-segment changes on EKG.[7,10,25,26] Prior studies reported that 4% to 14% of TAAAD cases have new Q waves or ST elevation on EKG.[3] Relying solely on chest pain and EKG to rule out TAAAD may increase risk of inappropriate treatment, including thrombolytic therapy, which is contraindicated in TAAAD.[7,11,12,27-29] However, we found no delay in diagnosis or treatment between groups.

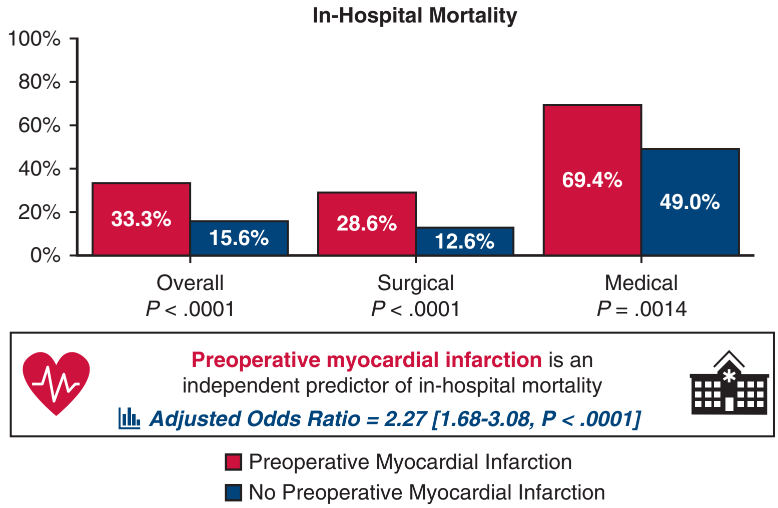
Patients with preoperative MI were more likely to undergo echocardiography as the initial imaging modality. Echocardiography can confirm dissection and measure cardiac function that can guide management in the setting of hemodynamic compromise. Whether echocardiography was performed first due to suspicion of MI or for preoperative cardiac assessment is not captured in IRAD and merits further investigation. D-dimer or other dissection-specific biomarkers are highly sensitive for detecting dissection and may complement definitive imaging.[30,31] Additionally, efforts to refine risk stratification calculators for emergency department use are currently underway.[26]

Most patients with TAAAD with and without preoperative MI underwent aortic surgery (88.4% vs 91.6%), reflecting current guidelines.[9] Patients with preoperative MI more often required CABG and root replacement, and both procedures were independent predictors of in-

TABLE 4. Postoperative outcomes of patients with type A acute aortic dissection with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
In-hospital mortality			
Overall	207 (33.3%)	803 (15.6%)	<.0001
Surgically managed	157 (28.6%)	591 (12.6%)	<.0001
Medically managed	50 (69.4%)	212 (49.0%)	.001
30-d mortality from hospital admission (overall)	201 (43.8%)	762 (23.7%)	<.0001
Postoperative complications*			
Cardiac tamponade	40 (9.2%)	265 (6.0%)	.013
Low output syndrome	29 (9.8%)	172 (5.5%)	.006
Respiratory insufficiency	98 (31.3%)	775 (25.0%)	.017
Acute renal failure	141 (31.2%)	935 (21.1%)	<.0001
Limb ischemia	28 (6.4%)	221 (5.1%)	.213
Mesenteric ischemia	12 (2.8%)	139 (3.2%)	.773
Spinal cord ischemia	11 (2.5%)	63 (1.4%)	.100
Stroke	50 (11.2%)	424 (9.6%)	.275
Coma	24 (5.4%)	125 (2.8%)	.006
Extension of dissection	11 (2.5%)	82 (1.9%)	.358
Bleeding requiring rethoracotomy	30 (10.1%)	233 (7.5%)	.139

MI, Myocardial infarction. *Includes surgically managed patients with TAAAD.



hospital mortality. Not all patients with preoperative MI required CABG, however, suggesting that myocardial ischemia may resolve with aortic repair alone depending on etiology. A prior study reported CABG as a predictor of mortality but did not adjust for preoperative MI.[20] In contrast to our findings, prior IRAD analyses found that root replacement was not associated with increased in-hospital mortality.[32] Additional investigation is needed to clarify whether CABG and root replacement contribute directly to poor outcomes or merely reflect underlying disease severity.

Patients with preoperative MI experienced more complications that likely reflect systemic malperfusion and multiorgan dysfunction triggered by the combined effects of dissection and reduced cardiac output.[6,33] Notably, preoperative MI remained an independent risk factor for in-hospital mortality after adjusting for concomitant high-risk factors and year of dissection. These findings

TABLE 5. Multivariable analysis of in-hospital mortality after type A acute aortic dissection repair

Variable	OR	95% CI for OR	*P* value
Preoperative MI	2.27	1.68-3.08	<.0001
Age	1.02	1.02-1.03	<.0001
Female	1.07	0.85-1.34	.594
Race Black (vs White)	0.61	0.39-0.95	.030
Race other (vs White)	0.78	0.52-1.19	.251
Period 1 (vs 2)	2.13	1.62-2.80	<.0001
Period 3 (vs 2)	1.27	0.94-1.73	.123
Atherosclerosis	1.19	0.88-1.60	.255
Prior PTCI	1.13	0.59-2.17	.710
Preoperative cardiac tamponade	2.13	1.61-2.83	<.0001
Preoperative acute renal failure	1.14	0.75-1.72	.551
Preoperative mesenteric ischemia	3.69	2.10-6.49	<.0001
Preoperative coma	1.75	0.87-3.49	.114
Preoperative stroke	1.59	0.96-2.61	.070
Presenting congestive heart failure	1.33	0.86-2.05	.202
Presenting hemodynamic shock	1.35	0.89-2.07	.160
Hypothermic circulatory arrest time (min)	1.00	1.00-1.01	.001
Root replacement	1.27	1.01-1.60	.039
Concomitant CABG	2.34	1.74-3.15	<.0001

Includes surgical patients with data for all included covariates, N = 3439. *OR*, Odds ratio; *PTCI*, percutaneous transluminal coronary intervention; *CABG*, coronary artery bypass grafting.

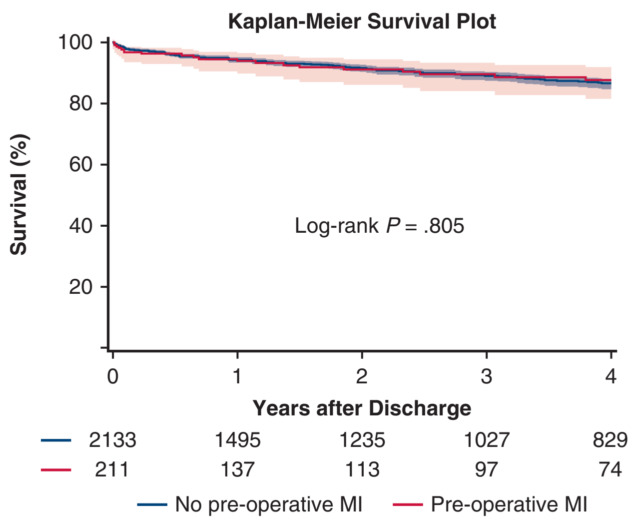


FIGURE 2. The 4-year survival postdischarge for TAAAD. 95% CI. *MI*, Myocardial infarction.

underscore the multifactorial nature of TAAAD mortality, with coronary malperfusion and systemic compromise identified as key contributors to poor outcomes.[6,8,15,20] Because IRAD spans approximately 3 decades of enrollment, time of dissection was included in the multivariable regression model, demonstrating that mortality was higher in period 1 compared with period 2, with MI associated with death after this adjustment.[14]

Despite elevated perioperative mortality, Kaplan–Meier analysis revealed similar 4-year survival, suggesting that myocardial reperfusion may restore midterm prognosis. In contrast, Waterford and colleagues[34] reported a significant reduction in intermediate-term survival after postoperative MI (P = .007), underscoring the importance of managing preoperative ischemia effectively during the index surgery.

Limitations

This study is limited by its retrospective design, which is subject to reporting bias. However, randomized trials in TAAAD are rare, highlighting the value of well-designed observational studies. Patients who did not receive EKG or had missing data for 1 or both variables were excluded (n = 5762; 40.6%), which may introduce selection bias. Also, MI was predefined and excludes more recent guidelines incorporating left bundle branch block in the diagnosis. Initial anticoagulants and antithrombotics are not recorded in the IRAD. Furthermore, we cannot ascertain the reasons a specific intervention was elected for a patient, including why CABG was performed in those with MI. Finally, one-half of patients lack follow-up, limiting assessment of intermediate-term risk. Despite these limitations, this study provides valuable insights from a large multicenter registry.

CONCLUSIONS

Preoperative MI occurs in 10.8% of patients with TAAAD and is associated with higher in-hospital mortality. Despite similar times to diagnosis, patients with preoperative MI had more coronary compromise and postoperative end-organ complications. Preoperative MI is independently associated with a 2-fold increase in hospital mortality. However, this elevated hazard appears confined to the acute phase. This study highlights the importance of early recognition and prompt surgery in patients with TAAAD with preoperative MI to restore coronary and systemic perfusion, and improve early outcomes.

Webcast

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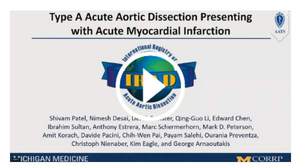

Conflict of Interest Statement

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The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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Key Words: acute aortic dissection, myocardial infarction, type A dissection

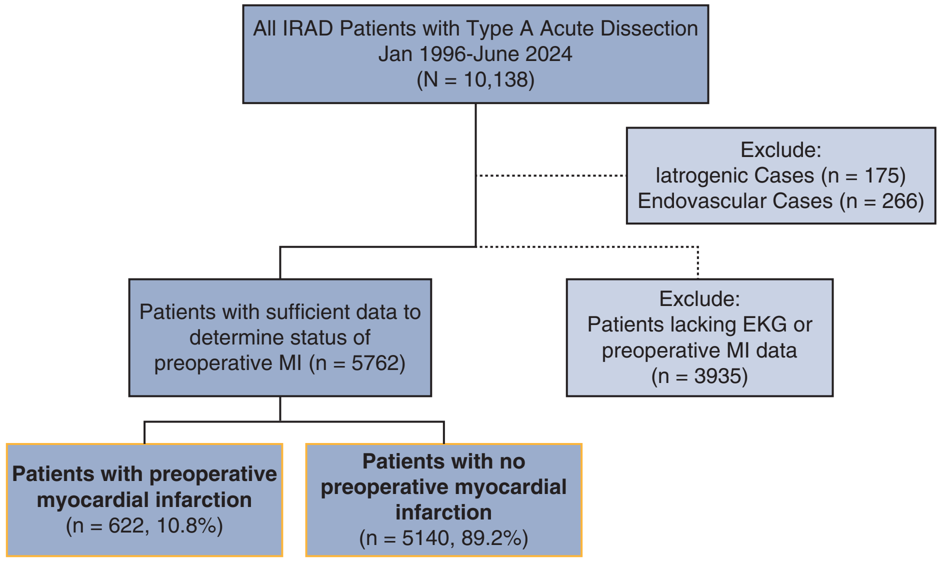

FIGURE E1. Flowchart of study cohort inclusion and exclusion. *MI*, Myocardial infarction; *EKG*, electrocardiography.

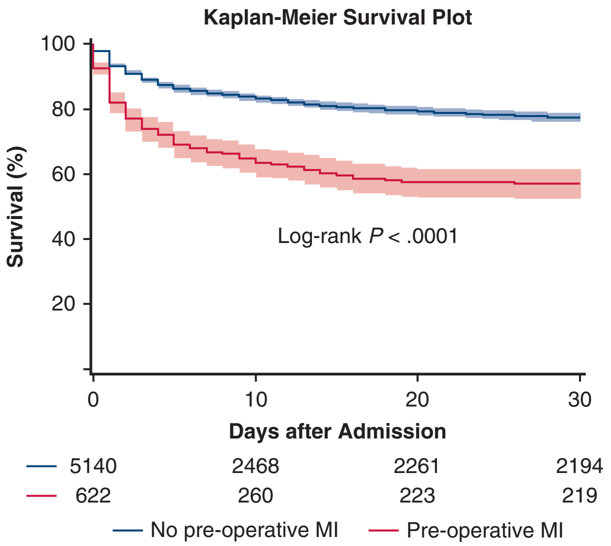

FIGURE E2. The 30-day mortality of TAAAD from hospital admission. 95% CI. *MI*, Myocardial infarction.

TABLE E1. Clinical criteria for preoperative myocardial infarction diagnosis

IRAD form variable	Diagnostic tool	Criteria
(1) New Q waves/ST elevation on EKG	EKG	- New Q waves 0.03 sec in width - New-onset ST-segment elevation > 1.0 mm
(2) Preoperative MI complication	EKG	- New Q waves 0.03 sec in width - New-onset ST-segment elevation > 1.0 mm
	Echocardiography	- New wall motion abnormalities
	Enzyme level elevation	- CK-MB >5% of total CK - LDH subtype 1 > LDH subtype 2 - Troponin > 2 times the normal limit

IRAD, International Registry of Acute Aortic Dissection; *EKG*, electrocardiography; *MI*, myocardial infarction; *CK-MB*, creatine kinase MB; *CK*, creatine kinase; *LDH*, lactate dehydrogenase.

TABLE E2. Presentation and pain symptoms of patients with type A acute aortic dissection with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
Location of pain			
Chest pain	526 (88.6%)	4115 (82.0%)	<.0001
Anterior chest pain	364 (79.1%)	3189 (72.3%)	.002
Posterior chest pain	136 (29.6%)	1183 (26.8%)	.205
Head or neck pain	117 (23.9%)	1135 (24.7%)	.700
Abdominal pain	120 (23.9%)	1129 (24.2%)	.913
Back pain	217 (42.1%)	2014 (43.0%)	.743
Leg pain	42 (8.8%)	516 (11.4%)	.092
Patient description of pain			
Severe or worst ever pain	418 (86.4%)	3618 (83.8%)	.169
Abrupt onset of pain	426 (82.6%)	3777 (80.8%)	.345
Radiating pain	192 (39.8%)	1792 (39.8%)	1.00
Migrating pain	38 (8.5%)	391 (8.9%)	.861
Sharp pain	139 (39.5%)	1306 (39.8%)	.954
Tearing or ripping pain	80 (25.2%)	914 (28.9%)	.172
Pressure	124 (37.3%)	1095 (35.2%)	.433
Burning	23 (7.4%)	277 (9.3%)	.301
Hemodynamics at presentation			
Normotensive	238 (42.7%)	2262 (46.4%)	.097
Hypotensive	100 (17.9%)	711 (14.6%)	.039
Hypertensive	157 (28.1%)	1484 (30.5%)	.263
Shock	53 (9.5%)	313 (6.4%)	.009
Other presenting characteristics			
Pulse deficit(s)	92 (33.6%)	920 (30.9%)	.376
Syncope	103 (19.9%)	727 (15.3%)	.008
Congestive heart failure	61 (12.1%)	267 (5.7%)	<.0001
Pericardial friction rub	4 (0.9%)	64 (1.5%)	.406

MI, Myocardial infarction.

TABLE E3. Diagnosis and imaging findings of patients with type A acute aortic dissection with and without preoperative myocardial infarction

Variable	Preoperative MI (n = 622)	No preoperative MI (n = 5140)	*P* value
Diagnosis timeline, median h (Q1-Q3)			
Initial hospital arrival to diagnosis, all patients	2.8 (1.0-15.1)	2.6 (1.2-6.3)	.413
Initial hospital arrival to diagnosis, surgical only	2.9 (1.0-15.2)	2.6 (1.2-6.1)	.317
Diagnosis to treatment, surgical only	4.0 (2.1-10.0)	3.8 (2.3-9.0)	.502
Initial imaging modality used			
Computed tomography	285 (63.8%)	3337 (81.4%)	<.0001
Transthoracic/transesophageal echocardiography	127 (28.4%)	683 (16.7%)	<.0001
Chest x-ray findings			
Widening mediastinum	100 (31.8%)	1161 (34.0%)	.455
Pleural effusion	40 (12.6%)	279 (8.1%)	.011
Imaging findings			
Pericardial effusion	190 (44.0%)	1787 (42.5%)	.574
Proximal extent of dissection to aortic root	306 (60.6%)	2641 (56.4%)	.072
Coronary involvement	150 (39.4%)	510 (14.3%)	<.0001
Left coronary involvement	30 (9.7%)	116 (3.4%)	<.0001
Right coronary involvement	65 (21.0%)	299 (8.7%)	<.0001
Maximum aortic diameter, cm (mean ± SD)			
Root	4.3 ± 1.0	4.3 ± 1.0	.532
Sinotubular junction	4.1 ± 1.0	4.1 ± 1.0	.650
Ascending	5.0 ± 1.1	5.0 ± 1.1	.270
Arch	3.9 ± 0.9	3.8 ± 0.8	.080
Descending	3.5 ± 1.0	3.4 ± 0.8	.434

MI, Myocardial infarction.

TABLE E4. Multivariable analysis of in-hospital mortality after type A acute aortic dissection repair among patients treated in the past decade (January 2014 to June 2024)

Variable	OR	95% CI for OR	*P* value
Preoperative MI	3.13	2.03-4.81	<.0001
Age	1.02	1.01-1.03	.002
Female	1.14	0.81-1.59	.458
Race Black (vs White)	0.50	0.28-0.89	.019
Race other (vs White)	0.84	0.51-1.40	.506
History of atherosclerosis	1.11	0.68-1.82	.668
Prior PTCI	1.16	0.52-2.60	.772
Preoperative cardiac tamponade	1.23	0.75-2.02	.405
Preoperative acute renal failure	1.69	0.94-3.06	.081
Preoperative mesenteric ischemia	2.26	1.00-5.12	.051
Preoperative coma	2.19	0.80-6.02	.129
Preoperative cerebral vascular accident	2.22	1.12-4.39	.022
Presenting congestive heart failure	1.63	0.84-3.18	.149
Presenting hemodynamic shock	1.83	0.93-3.61	.080
Hypothermic circulatory arrest time (min)	1.00	1.00-1.01	.033
Root replacement	1.08	0.77-1.51	.663
Concomitant CABG	2.19	1.42-3.38	.0004

Patient cohort only includes surgical management patients with available data for all included covariates during the most recent decade, N = 1989. *OR*, Odds ratio; *MI*, myocardial infarction; *PTCI*, percutaneous transluminal coronary intervention; *CABG*, coronary artery bypass grafting.