Original paper

# Clinical characteristics and in-hospital outcomes in patients treated for Stanford A acute aortic dissection

Marta Mazur[1], Magdalena Synak[1], Olga Wcisłek[2], Witold Gerber[1,3], Piotr Buszman[1], Krzysztof Milewski[1,3], Paweł Kaźmierczak[1], Andrzej Bochenek[1,3], Krzysztof Sanetra[1]

[1]American Heart of Poland, Katowice, Poland
[2]Andrzej Frycz Modrzewski Krakow University, Krakow, Poland
[3]Academy of Silesia, Katowice, Poland

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**Key words:** hypertension, aortic dissection, acute aortic syndrome.

### Abstract

**Introduction:** Stanford A acute aortic dissection is a life-threatening condition. The severity of the disease and delayed diagnosis or surgery contribute to high mortality rates. However, improvements in the diagnostic process and treatment process have been introduced in recent years.
**Aim of the research:** This study aims to evaluate recent results of treatment and clinical characteristics of patients hospitalised due to acute aortic dissection type A, classified according to the Stanford classification.
**Material and methods:** A retrospective analysis of a single-centre registry was performed, involving 55 patients admitted with acute aortic dissection type A as per the Stanford classification. Clinical characteristics of admitted patients and in-hospital outcomes were assessed.
**Results:** Men accounted for 74.5% of the analysed population. Admitted women were older than men (median: 70.5 vs. 58 years; $p = 0.010$). The median ascending aortic diameter was 48 mm. Arterial hypertension (HA) was reported in 30.9% of patients, and 41.2% of them were on pharmacotherapy for HA. Elevated blood pressure values at admission were observed in 27.3% of patients, of whom only 26.7% had a documented history of hypertension. All admitted patients were qualified for emergency cardiac surgery. Supracoronary aortic grafting procedure was performed in 57.4% of patients, and the Bentall procedure was performed in 42.6%. In-hospital mortality was recorded at 21.8% of cases, of which 58.3% were intraoperative deaths.
**Conclusions:** The perioperative mortality for acute aortic dissection type A, as per Stanford classification, remains high despite diagnostic and surgical advancements. Uncontrolled arterial hypertension is associated with dissection risk even in moderately enlarged aortas.

## Introduction

Acute aortic syndromes (AAS) are sudden conditions damaging the inner and middle layers of the aorta, potentially leading to aortic dissection. Acute aortic dissection (AAD) stems from intramural bleeding that separates the aortic wall layers, creating a false lumen (FL) [1]. The Stanford classification categorises dissection by location: Type A involves the ascending aorta, Type B involves the descending aorta, whereas non-A non-B involves the aortic arch and descending aorta without the ascending aorta [2]. The annual incidence of AAD is 3–6 cases per 100,000 people [3]. Mortality can reach 50% and may be associated with delayed disease diagnosis [4]. Risk factors for AAD include a pre-existing aortic disease, a family history of aortic disease, smoking, and hypertension [5].

## Aim of the research

In our study, we analyse recent outcomes of in-hospital treatment of patients admitted to the cardiothoracic department with a diagnosis of Stanford A acute aortic dissection. We also address the correlation between the occurrence of arterial hypertension (AH) with pharmacotherapy and arterial hypertension without pharmacotherapy and the occurrence of acute aortic dissection.

## Material and methods

This is an observational, retrospective registry of 55 patients admitted to the cardiac surgery department due to acute aortic dissection between March 2018 and May 2023. The primary endpoint was in-hospital outcome, including assessment of in-hospital and perioperative mortality. The analysis was performed for all patients and both genders separately. Medical information was collected from the hospital's computerised system. A sex-based analysis was performed due to previously reported gender-related differences in presentation, surgical timing, and outcomes in patients with AAD, as de-

scribed by the International Registry of Acute Aortic Dissection (IRAD) [6].

### Definitions

Acute aortic dissection (AAD): Acute aortic dissection is diagnosed based on clinical symptoms, echocardiography, and conclusively by computed tomography angiography.

Death: Death from any cause during the perioperative period.

Major adverse cardiac and cerebrovascular events (MACCE): a composite endpoint that includes mortality, perioperative myocardial infarction, and stroke.

### Statistical analysis

Continuous data were presented as median (interquartile range) while categorical data were presented as numbers and percentages. Fisher's exact test was used for categorical data comparison, while the Mann-Whitney *U* test was used for continuous data comparison. A Kaplan-Meier analysis was performed to visualise cumulative in-hospital mortality risk, with the time scale defined in days from admission to death or discharge. The $p$-value ≤ 0.05 was considered statistically significant. The data were analysed using MedCalc v.18.5 (MedCalc Software, Ostend, Belgium).

## Results

Fifty-five patients (74.5% male) were included in the study. In an area with 671,000 inhabitants, the numbers indicate that the incidence of Stanford A acute aortic dissection was 2.7/100,000 inhabitants/year. Median time from symptom onset to diagnosis was 6.5 hours (IQR: 4.0–9.0), and from symptom onset to surgery 12.5 hours (IQR: 9.0–18.0). These intervals are comparable to IRAD data, which report a median onset-to-diagnosis time of 4.3 hours and an onset-to-surgery time of 8.3–12 hours [7]. Despite comparable timings, in-hospital mortality in our cohort remained at 21.8%, suggesting that other factors, including preoperative instability and undiagnosed hypertension, may be contributing more significantly to final outcomes.

Median age of entire cohort was 60 years. On admission, 9% ($n$ = 5) were intubated before admission, and 27.3% ($n$ = 15) exhibited symptoms of congestive heart failure. Detailed demographic data and information on comorbidities collected during interviews are included in Table 1. The results of laboratory tests

**Table 1.** Demographic and baseline data

| Parameter | All $n$ = 55 | Male $n$ = 41 (74.5%) | Female $n$ = 14 (25.5%) | $P$-value |
|---|---|---|---|---|
| Age [years] | 60.0 (49.75–70.75) | 58.0 (48.0–63.5) | 70.5 (59.0–75.0) | 0.010 |
| BMI [kg/m$^2$] | 26.2 (23.25–28.07) | 26.2 (23.23–28.07) | 25.2 (23.25–31.21) | 0.953 |
| LVEF, % | 55.0 (47.0–55.0) | 55.0 (45.75–55.0) | 50.0 (50.0–55.0) | 0.588 |
| Aortic root diameter [mm] | 36.0 (32.0–39.5) | 36.0 (33.5–44.5) | 31.0 (25.0–36.0) | 0.125 |
| Ascending aorta [mm] | 48.0 (43.0–50.0) | 48.0 (41.5–51.0) | 47.5 (45.0–48.5) | 0.778 |
| Hypertension, $n$ (%) | 17 (30.9) | 13 (31.7) | 4 (28.6) | 1 |
| Hypercholesterolaemia, $n$ (%) | 4 (7.3) | 3 (7.3) | 1 (7.1) | 1 |
| Ischemic heart disease, $n$ (%) | 10 (18.2) | 9 (22) | 1 (7.1) | 0.423 |
| Valve defects, $n$ (%) | 8 (14.5) | 6 (14.6) | 2 (14.3) | 1 |
| Aortic valve regurgitation, $n$ (%) | 7 (12.7) | 6 (14.6) | 1 (7.1) | 0.664 |
| Atrial fibrillation, $n$ (%) | 3 (5.5) | 1 (2.4) | 2 (14.3) | 0.156 |
| Hypothyroidism, $n$ (%) | 3 (5.5) | 0 | 3 (21.4) | 0.014 |
| Kidney failure, $n$ (%) | 2 (3.6) | 2 (4.9) | 0 | 1 |
| COPD, $n$ (%) | 1 (1.8) | 0 | 1 (7.1) | 0.255 |
| Nicotinism, $n$ (%) | 4 (7.3) | 3 (7.3) | 1 (7.1) | 1 |
| Stroke < 2 weeks, $n$ (%) | 1 (1.8) | 1 (2.4) | 0 | 1 |
| Implanted pacemaker, $n$ (%) | 1 (1.8) | 0 | 1 (7.1) | 1 |
| Prior cardiac interventions, $n$ (%) | 7 (12.7) | 5 (12.2) | 2 (14.3) | 1 |
| Prior cardiac surgery, $n$ (%) | 6 (10.9) | 4 (9.8) | 2 (14.3) | 0.638 |

*Data are presented as number (percentage) and median (interquartile range). BMI – body mass index, COPD – chronic obstructive pulmonary disease.*

performed upon admission and discharge are presented in Table 2. Blood pressure upon admission was 125 mm Hg (96.0–140.0) for systolic and 65 mm Hg (57.75–78.75) for diastolic blood pressure. History of hypertension was reported in 30.9% ($n$ = 17) of patients, and only 41.2% ($n$ = 7) of them were undergoing pharmacotherapy. Upon admission, 27.3% ($n$ = 4) of patients exhibited elevated blood pressure levels (> 140 systolic and/or > 90 diastolic). Notably, only 26.7% ($n$ = 4) of these patients had a prior diagnosis of hypertension, indicating that 73.3% ($n$ = 11) had undiagnosed hypertension (Table 3). Surgery was performed in 98.2% ($n$ = 54) of cases, as one patient passed away just before the procedure started. All patients underwent surgical procedures in deep hypothermia. Aortic replacement procedure using a supra coronary interposition graft was performed in 57.4% ($n$ = 31) of patients. The most frequent prosthesis diameter used was 30 mm. The Bentall procedure was performed in 42.6% ($n$ = 23) of cases, with 26 mm mechanical conduits being the most used. Aortic arch surgery was performed in 20% cases. Simultaneous aorto-coronary bypass surgery (CABG) was performed on 9.3% ($n$ = 5) of operated patients. The strategy of postponed chest closure was used in 37% ($n$ = 20) of cases. Sudden cardiac arrest occurred in 7.4% ($n$ = 4) of operated patients. Median length of hospitalisation for surviving cases was 7 days. In-hospital mortality was 21.8% ($n$ = 12), of which 58.3% ($n$ = 7) were intraoperative deaths. Incidence of MACCE was 27.3% (Figure 1, Table 4). Pleural effusion was observed in 16.7% ($n$ = 9) patients, delirium in 11% ($n$ = 6), atrial fibrillation in 7.4% ($n$ = 4), and stroke in 5.6% ($n$ = 3). There was one case each of perioperative MI, cardiac tamponade, pulmonary oedema, multiorgan failure, acute abdomen, gastrointestinal bleeding, and liver failure. After hospitalisation, 38.2% ($n$ = 21) of patients were directly transferred to a rehabilitation department, 40% ($n$ = 22) were transferred to another hospital department, and 7.3% ($n$ = 4) were discharged home.

## Discussion

A pivotal source of information on acute aortic dissection (AAD) is the International Registry of Acute Aortic Dissection (IRAD), which in January 2021 celebrated its 25th anniversary. The registry offers valuable insights and practical guidelines for optimal

**Table 2.** Preoperative laboratory parameters

| Parameter | All $n$ = 55 | Male $n$ = 41 (74.5%) | Female $n$ = 14 (25.5%) | $P$-value |
|---|---|---|---|---|
| INR | 1.22 (1.11–1.32) | 1.22 (1.09–1.29) | 1.30 (1.17–1.42) | 0.089 |
| APTT [s] | 37.1 (32.2–39.7) | 37 (31.6–39.7) | 38.5 (32.95–41.0) | 0.548 |
| Na [mmol/l] | 140 (136–143) | 141 (137.0–143.0) | 136 (133.5–142.0) | 0.155 |
| K [mmol/l] | 4.42 (4.01–4.82) | 4.24 (4.0–4.79) | 4.62 (4.23–5.17) | 0.263 |
| RBC [× $10^{12}$] | 3.76 (3.09–4.19) | 3.99 (3.07–4.24) | 3.35 (3.15–3.71) | 0.118 |
| WBC [× $10^9$] | 12.55 (8.9–14.7) | 12.6 (8.95–15.70) | 12.5 (8.7–13.3) | 0.554 |
| HGB [g/dl] | 11.5 (9.78–13.2) | 11.90 (10.05–13.6) | 10.05 (9.50–11.60) | 0.039 |
| HCT (%) | 34.4 (28.25–39.55) | 34.70 (29.77–40.15) | 29.40 (28.45–35.62) | 0.166 |
| PLT [× $10^9$] | 167 (137–216.5) | 167.0 (140.0–221.25) | 138 (118.0–198.25) | 0.163 |
| Creatinine [mg/dl] | 1.3 (1.02–1.69) | 1.39 (1.15–1.87) | 1.05 (0.89–1.60) | 0.073 |
| GFR [ml/$m^2$/min] | 53.15 (48.58–60) | 59.0 (52.40–60.0) | 51.8 (36.75–58.50) | 0.187 |
| Glucose [mg/dl] | 125 (109.75–151.25) | 119.5(105.50–148.50) | 138 (122.0–181.25) | 0.262 |

*Data are presented as median (interquartile range). APTT – activated partial thromboplastin time, CK-MB – creatine kinase-myoglobin binding, GFR – glomerular filtration rate, HCT – haematocrit, HGB – haemoglobin, INR – international normalised ratio, PLT – platelet count test, RBC – red blood cell, WBC – white blood cell.*

**Table 3.** Hypertension analyses and drug adherence ($n$ = 55)

| Variable | Result |
|---|---|
| Hypertension previously diagnosed, $n$ (%) | 17 (30.9) |
| Hypertension previously diagnosed – controlled with pharmacotherapy, $n$ (%) | 7 (41.2) |
| SBP ≥ 140 and/or DBP ≥ 90 at admission, $n$ (%) | 15 (27.3) |
| SBP ≥ 140 and/or DBP ≥ 90 at admission in patients with previously diagnosed hypertension, $n$ (%) | 4 (36.7) |

*Data are presented as numbers (percentages). DBP – diastolic blood pressure, SBP – systolic blood pressure.*

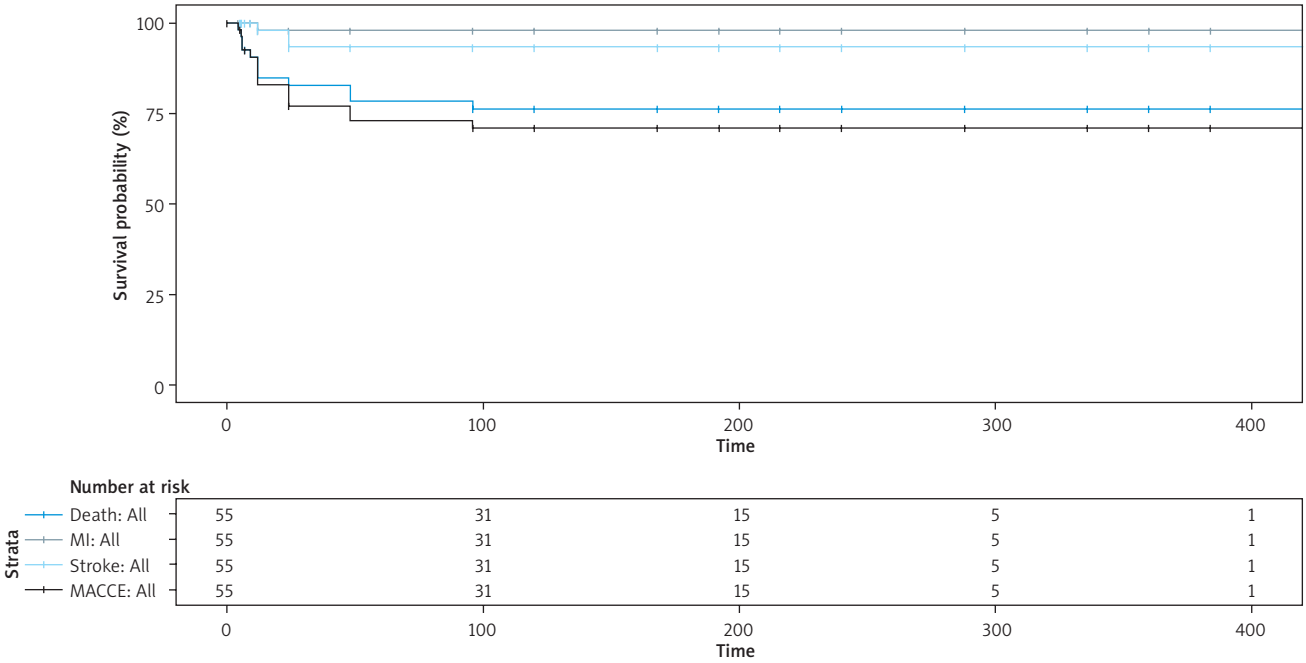


**Figure 1.** Kaplan–Meier analysis of in-hospital clinical outcomes

**Table 4.** In-hospital outcomes

| Parameter | All *n* = 55 | Male *n* = 41 (74.5%) | Female *n* = 14 (25.5%) | *P*-value |
|---|---|---|---|---|
| Mortality, *n* (%) | 12 (21.8) | 9 (22) | 3 (21.4) | 1 |
| Myocardial infarction, *n* (%) | 1 (1.8) | 1 (2.4) | 0 (0) | 1 |
| Stroke (%) | 3 (5.5) | 2 (4.8) | 1 (7.1) | 1 |
| MACCE (%) | 15 (27.3) | 11 (26.8) | 4 (28.6) | 1 |
| Creatinine > 1.5 mg/dl, *n* (%) | 33 (68.8) | 25 (61) | 8 (57) | 1 |
| Max creatinine [mg/dl] | 2.36 (1.75–3.35) | 2.36 (1.79–3.30) | 2.33 (1.27–3.41) | 0.533 |
| HF/HDF, *n* (%) | 5 (9.1) | 4 (9.7) | 1 (7.1) | 1 |
| Creatinine at discharge [mg/dl] | 1.15 (0.8–1.8) | 1.16 (0.85–1.90) | 1.15 (0.7–1.24) | 0.252 |

*Data are presented as number (percentage) and median (interquartile range). HDF – haemodiafiltration, MACCE – major adverse cardiac and cerebrovascular events (mortality, perioperative myocardial infarction, stroke).*

management and treatment outcomes [8]. However, it is worth noting that no centres from Poland or Eastern Europe are currently included in IRAD.

Thoracic aortic dissection is a complex and life-threatening condition associated with high incidence and mortality rates [9]. Type A dissection accounts for approximately 67% of all AAD cases [7], with incidence increasing with age and higher prevalence among men [10, 11]. In our analysis, 80% of patients were male, with a median age of 60 years. While the annual incidence of AAD is estimated at 3–6 cases per 100,000 population, our data showed 2.7 cases per 100,000 inhabitants per year. This period included the COVID-19 pandemic, which significantly reduced access to emergency healthcare and may have contributed to delayed diagnoses or increased rates of unrecognised out-of-hospital deaths. Although AAD symptoms are typically pronounced, fear of hospitalisation and limited emergency medical services during the pandemic could have led to diagnostic delays.

IRAD data indicate a clear circadian pattern, with an increased incidence of AAD between 6:00 am and 12:00 noon, particularly between 8:00 and 9:00 am ($p < 0.001$). Interestingly, surgeries are more frequently performed at night (56.5%), which has been associated with a higher incidence of postoperative tamponade. However, surgical timing does not appear to significantly affect overall mortality. Postponed chest closure, despite increasing the risk of sternal infection, was often necessary due to perioperative coagu-

lation disturbances commonly observed in aortic dissection. Seasonal variability has also been noted, with winter months showing significantly higher rates of AAD occurrence ($p < 0.008$) [7].

Hypertension remains the most important risk factor for AAD and was present in 76.6% of IRAD patients [5]. It contributes to aortic wall degeneration and reduced elasticity by impairing blood flow to the outer layers of the vessel wall. Studies have reported hypertension in 45–100% of patients with AAD [8, 11], and a 20-year follow-up by Landenhed *et al.* showed a prevalence of 86%. Notably, hypertensive individuals were found to have a fourfold higher risk of dissection compared to normotensive individuals, and patients later diagnosed with Type A AAD had significantly elevated systolic blood pressure up to five years prior to the event [2]. These findings suggest that not only the presence of hypertension, but also its insufficient control and treatment, play a critical role in AAD development.

However, our findings – consistent with IRAD data – suggest that even moderately enlarged aortas may dissect, particularly in the setting of poorly controlled hypertension. These findings do not suggest a definitive causal relationship, as our study was not powered to detect differences in outcomes based on aortic diameter. However, they support the need for individualised surgical thresholds, especially in hypertensive patients [12].

The outcomes of elective aneurysm surgery and acute dissection repair highlight the need to revisit current guidelines, ideally through larger multicentre studies. Importantly, regional differences in healthcare access, especially in managing hypertension and cardiovascular diseases, may affect the baseline characteristics of patients with Type A AAD. In our study, a supracoronary interposition graft was performed in 57.4% of cases, while 42.6% underwent the Bentall procedure. In all patients, standard surgical techniques were used to repair the ascending aorta and aortic arch. When dissection extended to the descending aorta, patients were referred for vascular surgery consultation and planned stent-graft implantation based on postoperative CT imaging.

Computed tomography remains the primary diagnostic tool for confirming AAD. Transthoracic echocardiography (TTE) offers 77–80% sensitivity and 93–96% specificity, while MRI remains the most sensitive and specific method, particularly in evaluating the ascending aorta and aortic arch [1, 2].

Surgical management of Type A AAD involves preoperative evaluation of aortic valve function and neurological status [1]. Procedures typically include repair of the primary entry tear, open distal anastomosis, and selective cerebral perfusion. Significant root involvement may require replacement via Bentall or David procedures. In extensive disease, involving the arch or descending aorta, the "frozen elephant trunk" (FET) prosthesis can be used to close the primary tear and stabilise the distal aorta [13].

AAD continues to be associated with high mortality, particularly without surgical intervention. Operative treatment reduces mortality to approximately 30%, despite a perioperative mortality rate of around 25%. According to IRAD, in-hospital mortality for Type A AAD has decreased from 31% to 22%, primarily due to improved surgical outcomes (25% to 18%). Mortality remains significantly higher among unstable patients (31% vs. 17%, $p < 0.001$) [4, 8, 9].

Current guidelines recommend surgical intervention when the aortic size index (ASI), adjusted for body surface area (BSA), exceeds 2.75 cm/m$^2$. However, Zafar *et al.* proposed that the aortic height index (AHI), based on patient height, may be a more precise predictor of dissection risk. Incorporating AHI into routine clinical assessment could improve identification of high-risk individuals, optimise timing of surgery, and reduce unexpected complications [14].

## Conclusions

Perioperative mortality in Stanford Type A aortic dissection remains considerable despite advancements in diagnostics and surgical techniques. Our findings, consistent with IRAD data, underscore that dissection may occur even in moderately enlarged aortas – particularly when hypertension is undiagnosed or poorly controlled. This highlights the need for more individualised risk assessment and suggests that current surgical thresholds, based solely on aortic diameter, may not adequately capture dissection risk in real-world populations.

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## Ethical approval

The approval of the research ethics board was not mandatory for the analysis as the report was fully retrospective and contained datasets with anonymized information. No additional intervention was administered to any of the study patients. Following the National Code on Clinical Research, research Ethics Board consent is not obligatory for real retrospective studies.

## Conflict of interest

The authors declare no conflict of interest.

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**Address for correspondence**

**Marta Mazur**
American Heart of Poland
Katowice, Poland
E-mail: marta.mazur@ahop.pl

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