Comparison of long-term outcomes and risk factors of aortic stenosis treatment in patients undergoing transcatheter aortic valve implantation and surgical aortic valve replacement

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DOI: 10.33963/KP.a2022.0122

Received:
November 24, 2021

Accepted:
May 5, 2022

Early publication date:
May 6, 2022

ABSTRACT

Background: Aortic stenosis (AS) is the most common valvular heart disease and untreated has a bleak prognosis. The only effective method of treatment is valve replacement, surgical (SAVR), or transcatheter (TAVI).

Aims: We decided to analyze outcomes and predictors of long-term mortality in patients undergoing TAVI and SAVR.

Methods: A retrospective analysis of 1229 patients with advanced AS, comprising TAVI (n = 211), SAVR (n = 556), SAVR, and additional procedures (n = 462), operated on from 2014 to 2018, was performed.

Results: No significant differences between SAVR and TAVI were found for 24-month mortality in groups of consecutive patients. Postoperative stroke or transient ischemic attack (TIA), chronic obstructive pulmonary disease (COPD), and transfusion of red blood cells (RBCs) were independent predictors of 1-year mortality after SAVR. The above-mentioned factors regarding the increased estimated surgery risk in the EuroSCORE II (>4%) were predictors of 2-years mortality after SAVR. Risk factors for 6- and 12-month mortality after TAVI were EuroSCORE II, new onset of atrial fibrillation (NOAF), and the increased RBC distribution width (RDW). Postoperative respiratory failure was an independent risk factor for 6-, 12- and 24-month mortality in both groups of patients.

Conclusions: There were no significant differences regarding prognosis after TAVI and SAVR at the 24-month follow-up in the propensity score matching model. Independent predictive factors of late mortality after both procedures were EuroSCORE II and respiratory failure. Independent predictive factors of late mortality specific for TAVI were NOAF, increased RDW, and for SAVR: TIA, stroke, COPD, and RBC transfusion.

Key words: aortic stenosis, transcatheter aortic valve implantation, surgical aortic valve replacement, outcomes, risk factors

INTRODUCTION

For a long time, the only effective method of treatment was surgical aortic valve replacement (SAVR). Since the first transcatheter aortic valve implantation (TAVI), this method has evolved from novel technology to daily therapy. The choice between TAVI and SAVR should be based on the Heart Team evaluation according to the individual patient characteristics. Analysis and comparison of late outcomes and predictors of mortality in patients undergoing TAVI and SAVR in the

WHAT'S NEW?

We performed an analysis of outcomes and predictors of long-term mortality in 1229 patients undergoing transcatheter aortic valve implantation (TAVI) and surgical aortic valve replacement (SAVR) in the National Institute of Cardiology in Warsaw from January 2014 to July 2018. We found no significant differences regarding the prognosis for up to 24 months between SAVR and TAVI in consecutive patient groups. Independent risk factors of late mortality after SAVR and TAVI were elevated EuroScore II and postprocedure respiratory failure. Independent predictive factors of late mortality after SAVR were stroke, chronic obstructive pulmonary disease, and red blood cells (RBC) transfusion, while after TAVI these were new onset of atrial fibrillation and the increased RDW (RBC distribution width).

years 2014–2018 in a single high-volume center in Poland were performed to improve the qualification of patients for that treatment method.

METHODS

A retrospective analysis of 1229 patients with advanced aortic stenosis (AS), comprising TAVI (n = 211), isolated SAVR (n = 556) from January 2014 to July 2018 was performed.

Severe AS was defined according to the 2021 European Society of Cardiology and the European Association for Cardio-Thoracic Surgery (ESC/EACTS) guidelines [1] by transthoracic echocardiography (TTE) as a mean gradient ≥40 mm Hg and effective orifice area below 1 cm^2. The EuroSCORE II was used to estimate the risk of death after surgery. The patients were assigned to TAVI or SAVR according to the evaluation and estimation of the team that consisted of cardiac surgeons, cardiologists, anesthesiologists, and radiologists with experience in heart valve treatment. TAVI was recommended for older patients, patients at high risk or unsuitable for SAVR (STS or EuroSCORE II ≥4%, patients with fragility syndrome, porcelain aorta, or sequelae of chest radiation). Young patients, at low surgical risk or undergoing coronary artery bypass graft (CABG) or intervention on the ascending aorta or another valve were candidates for cardiac surgery. SAVR was performed under general anesthesia using extracorporeal circulation by surgeons with abundant experience.

Transcatheter heart valve size and approach were selected by using multidetector computed tomography angiography. TAVI was done under local anesthesia; fluoroscopic guidance was used for deployment of the valve.

Early complications and long-term mortality (6, 12 and 24 months after the procedure) were reported. Afterward, to identify risk factors of unfavorable outcomes, the impact of clinical and echocardiographic characteristics, blood test results, as well as early complications on long-term mortality, was investigated.

The protocol of the study was approved by the Regional Bioethical Committee at the National Institute of Cardiology (no. 1836). Upon admission to the hospital, patients consented to use their medical data for scientific research. All data were extracted from the electronic medical record system. Before the assessment, all data were fully anonymized. Follow-up was by using data from the Polish Death Database.

Statistical analysis

Depending on the distribution of continuous variables, tested with the Kolmogorov-Smirnow test, the results were presented using the arithmetic mean value and standard deviation (normal distributions) or the median and interquartile range (skew distributions). The significance of differences between the groups was verified by Student t-test or Mann-Whitney test. The results of qualitative variables were reported in the form of absolute and relative frequency (%) of the distinguished units, and the differences in proportions were tested by the χ^2 test or by Fisher exact test, in the case of a small number (<5) of observations expected in the cells of the multi-way tables.

Propensity score matching (PSM) was used to minimize impact of demographic and clinical characteristics on the long-term prognosis of patients, determined by the multivariable binary logistic regression method (area under the curve [AUC], 0.894, fit: Hosmer and Lemeshow goodness — of fit test: $P = 0.811$), in a 1: 1 ratio in the group of patients. The model included demographic variables: age, sex; comorbidities: arterial hypertension, chronic kidney disease, diabetes, autoimmune diseases, chronic obstructive pulmonary disease (COPD), stroke, chronic coronary syndrome, previous myocardial infarction, CABG and EuroSCORE II risk, aortic valve area, and the left ventricular ejection fraction.

Identification of independent factors of all-cause mortality in both groups of patients was carried out on the basis of the multivariable Cox proportional hazard method after checking the assumptions necessary for its performance. Hazard coefficients were calculated together with 95% confidence intervals (95% confidence interval [CI]). The procedure of backward selection of variables was applied, a significance of 0.05 was required for a variable to stay in the multivariable model. The variables included in the model are EuroSCORE II score, age, sex, arterial hypertension, chronic kidney disease, diabetes, presence of autoimmune diseases, COPD, chronic coronary syndrome, previous CABG, myocardial infarction, stroke, aortic valve area (AVA), and left ventricular ejection fraction (LVEF).

Table 1. Baseline characteristics of patients qualified for TAVI and SAVR — procedural aspects

Procedural aspects	SAVR (n = 556)		TAVI (n = 211)	
Approach	Full sternotomy, n (%)	416 (74.8)	Trans-femoral, n (%)	188 (89)
	Ministernotomy, n (%)	140 (25.1)	Trans-subclavian, n (%)	8 (3.8)
			Trans-apical, n (%)	13 (6.2)
			Trans-aortic, n (%)	2 (1)
Types of valves	Mechanical prostheses, n (%)	161 (28.9)	Self-expanding, n (%)	109 (51.6)
	Biological prostheses, n (%)	395 (71.1)	Balloon-expandable, n (%)	102 (48.4)

Data are given as counts (percentage)

Abbreviations: SAVR, surgical aortic valve replacement; TAVI, transcatheter aortic valve implantation

Table 2. Baseline characteristics of patients qualified for TAVI and SAVR

Baseline characteristic	TAVI (n = 211)	SAVR (n = 556)	*P*-value
Age, years, mean (SD)	80.1 (7.8)	66.7 (10.5)	<0.01
Female sex, n (%)	126 (59.7)	262 (47.1)	<0.01
Hypertension, n (%)	166 (78.7)	412 (74.1)	0.20
Chronic kidney disease, n (%)	93 (44.1)	50 (9.0)	<0.01
Diabetes mellitus, n (%)	85 (40.3)	139 (25.0)	<0.01
Chronic obstructive pulmonary disease, n (%)	30 (14.2)	41 (7.4)	<0.01
Coronary artery disease, n (%)	131 (62.1)	167 (30.0)	<0.01
Previous myocardial infarction, n (%)	38 (18.1)	32 (5.8)	<0.01
Previous CABG, n (%)	23 (10.9)	7 (1.3)	<0.01
Previous stroke, n (%)	12 (5.7)	20 (3.6)	0.19
EuroSCORE II, %, median (IQR)	4.01 (2.69–7.75)	1.51 (1.03–2.24)	<0.01
Aortic valve area, cm^2, mean (SD)	0.65 (0.17)	0.70 (0.18)	0.04
Mean gradient, mm Hg, mean (SD)	51.2 (14.7)	55.8 (16.9)	0.02
Maximal gradient, mm Hg, mean (SD)	84.9 (24.5)	90.5 (27.5)	0.08
Left ventricular ejection fraction, %, mean (SD)	56.1 (13.2)	61.4 (10.2)	<0.01
Right ventricular systolic pressure, mm Hg, mean (SD)	46.3 (13.7)	38.4 (11.2)	<0.01
Interventricular septum dimension, mm, mean (SD)	14.9 (2.38)	14.8 (2.74)	0.90
Posterior wall dimension, mm, mean (SD)	11.8 (2.2)	12.6 (2.3)	<0.01
Left ventricular end-diastolic dimension, mm, mean (SD)	46.4 (7.6)	48.0 (6.8)	0.61

Data are given as mean and standard deviation (SD), median (IQR), or counts (percentage)

Abbreviations: CABG, coronary artery bypass graft; other — see Table 1

The probability of survival was estimated using the Kaplan-Meier method, and the homogeneity of the curves of different groups was estimated with the log-rank test.

The verification of two-sided hypotheses was carried out at the statistical significance level of $P \leq 0.05$. Calculations were performed with SAS version 9.4 4 (SAS Institute Inc., Cary, NC, US).

RESULTS

Baseline characteristics of patients

Full sternotomy was used in 419 (74.8%) patients and ministernotomy in 140 (25.1%). Mechanical prostheses were implanted in 161 patients (28.9%) and biological valves in 395 cases (71.1%). One hundred and eighty-eight (89%) patients were suitable for transfemoral access. Non-transfemoral TAVI was performed in 23 patients (11%) (Table 1).

Patients who qualified for TAVI were older, had higher EuroSCORE II results, and more comorbidities. Patients assigned to SAVR were characterized by higher LVEF and lower right ventricular systolic pressure (RVSP). The baseline characteristics of each group are summarized in Table 2.

Long term outcomes

A complete 24-month follow-up study was achieved in 89.3% of patients. The distribution of observation time was 0–715 days. The overall 6-month mortality was significantly lower in the surgical group than in TAVI (3.2% SAVR vs. 11% TAVI). This observation was also confirmed in 12-month (4.7% SAVR vs. 14.5% TAVI) and 24-month observation (6.1% SAVR 25.4% TAVI) (Table 3). Interestingly, the propensity score model revealed no difference in survival after TAVI and SAVR. However, there was a trend of higher mortality in the TAVI group compared to the SAVR group (P= 0.053) during the 24-month follow-up (Table 4). The Kaplan-Meier survival curve is illustrated in Figure 1.

Predictors of mortality

Multivariable analysis identified postprocedural respiratory insufficiency as the independent predictor of 6- and 12-month mortality in both groups of patients. For SAVR, the independent predictors of 12- and 24-month mortality were postoperative bleeding defined as necessity for RBC transfusion (hazard ratio [HR], 4.77; 95% CI, 1.07–21.43; $P = 0.04$), as well as a history of stroke/TIA (HR, 5.74; 95% CI,

Table 3. Long term outcomes of aortic stenosis treatment, overall 6-, 12- and 24- month mortality

Long term outcomes	AVR (n = 556)	TAVI (n = 211)	*P*-value
6-month mortality, n (%)	18/556 (3.2)	23/209 (11.0)	<0.01
12-month mortality, n (%)	26/555 (4.7)	30/207 (14.5)	<0.01
24-month mortality, n (%)	32/525 (6.1)	43/169 (25.4)	<0.01

Abbreviation: see Table 1

Table 4. Long-term outcomes of aortic stenosis treatment, overall 6-, 12- and 24-month mortality. Propensity score model

Long term outcomes	AVR (n = 101)	TAVI (n = 101)	*P*-value
6-month mortality, n (%)	5/101 (4.9)	6/100 (6.0)	0.74
12-month mortality, n (%)	7/101 (6.9)	9/99 (9.1)	0.57
24-month mortality, n (%)	11/91 (12.1)	17/72 (23.6)	0.053

Abbreviation: see Table 1

Table 5. Independent predictors of 6- and 12-month mortality after TAVI. Multivariable analysis of deaths over 6-months was made on the basis of n = 196, number of deaths: 21. Multivariable analysis of deaths over 12 months was made on the basis of n = 194, number of deaths: 28

Risk factor	6-month mortality		12-month mortality	
	Hazard ratio (95% CI)	*P*-value	Hazard ratio (95% CI)	*P*-value
EuroSCORE II	1.20 (1.08–1.32)	<0.01	1.15 (1.07–1.24)	<0.01
RDW, %	1.33 (1.11–1.59)	<0.01	1.24 (1.09–1.42)	<0.01
Respiratory failure	5.74 (1.49–22.10)	0.01	9.22 (3.34–25.45)	<0.01
New onset atrial fibrillation	6.14 (1.69–22.29)	<0.01	3.90 (1.52–10.00)	<0.01

Abbreviations: RDW, red blood cell distribution width

Table 6. Independent predictors of 6-, 12- and 24-month mortality after SAVR. Multivariable analysis of 6-month mortality was made on the basis of n = 511, number of deaths: 15. Multivariable analysis of 12-month mortality was made on the basis of n = 510, number of deaths: 19. Multivariable analysis of 24-month mortality was made on the basis of n = 480, number of deaths: 25

Risk factor	6-month mortality		12-month mortality		24-month mortality	
	Hazard ratio (95% CI)	*P*-value	Hazard ratio (95% CI)	*P*-value	Hazard ratio (95% CI)	*P*-value
Respiratory failure	16.268 (8.194–32.296)	<0.0001	6.478 (2.203–19.026)	0.0007	5.046 (1.899–13.410)	0.001
Stroke/TIA	—	—	5.743 (1.568–21.039)	0.008	5.616 (1.677–18.810)	0.005
RBC transfusion	—	—	4.772 (1.066–21.37)	0.041	3.082 (1.032–9.205)	0.044
COPD	—	—	3.380 (1.049–10.896)	0.041	3.483 (1.232–9.844)	0.019
EuroSCORE II	—	—	—	—	1.155 (1.038–1.284)	0.008

Abbreviations: COPD, chronic obstructive pulmonary disease; RBC, red blood cells; TIA, transient ischemic attack

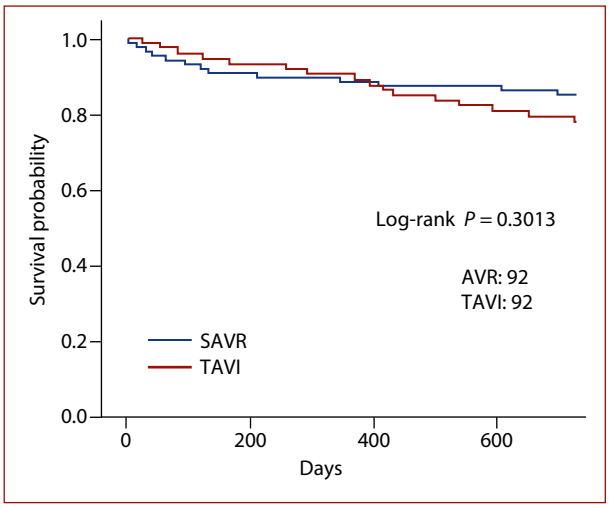

Figure 1. Kaplan-Maier analysis of 24-month survival after TAVI and SAVR

Abbreviations: see Table 1

1.57–21.0; *P* <0.01) and COPD (HR, 3.38; 95% CI, 1.05–10.90; *P* = 0.04). The elevated EuroSCORE II was associated with 24-month mortality in this group of patients (HR, 1.16; 95% CI, 1.04–1.28; *P* <0.01). For TAVI, the predictors of 6- and 12-month mortality were elevated EuroSCORE II risk score (HR, 1.19; 95% CI, 1.08–1.32; *P* <0.01), RDW (HR, 1.33; 95% CI 1.11–1.59; *P* <0.01), and new onset of atrial fibrillation (NOAF) (HR, 6.14; 95% CI, 1.69–22.29; *P* <0.01) (Tables 5 and 6).

DISCUSSION

The only effective treatment for severe AS is the aortic valve replacement both transcatheter and surgical [2–6]. With increased life expectancy and aging of the population, more elderly people require invasive treatment. Diagnosis and qualification in elderly people are decreased by concomitant diseases, which makes the range of treatment efficacy very narrow. Therefore, a greater knowledge of predictors

of mortality is needed and remains a crucial aspect of maintaining safety when choosing a more adequate AS treatment option: TAVI or SAVR.

Our study revealed no difference in all-cause mortality with 24-month follow-up between SAVR and TAVI in the propensity-score matched groups. The favorable results of TAVI have been described previously in multiple large nationwide registries and randomized controlled trials [2, 3]. On the contrary, Makkar et al. [4] revealed statistically significant increased mortality with TAVI relative to SAVR in propensity-score matched groups during a 5-year follow-up. These differences may result from the analysis of different groups of patients.

The main risk factor of mortality at each stage of observation (6-, 12- and 24-month) for 24-month follow-up after TAVI and SAVR was respiratory failure after the procedure.

The elevated surgery risk score (EuroSCORE II >4%), the presence of NOAF, and the increase of RDW were associated with a worse 6- and 12- month survival rate after TAVI. While stroke/TIA, COPD, and RBC transfusion increased the risk of 12-month mortality after SAVR. EuroSCORE II is an approved scale to predict early postoperative mortality. However, clinicians could take advantage of the EuroSCORE II scale to anticipate long-term TAVI and SAVR outcomes. So far, only the logistic EuroSCORE scale is recognized as a predictive risk factor in large, nationwide TAVI registries [5, 6]. Our study showed a predictive value of Euroscore II for long-term mortality after TAVI at each stage of observation, but just after 24-month follow-up in SAVR patients. Dębiński et al. [7] confirmed a worse prognosis in TAVI patients with EuroSCORE II greater than 6%.

COPD increased long-term mortality risk after SAVR. Coexisting COPD and AS increases the risk of pulmonary hypertension, which in this group might be associated with not only elevated left ventricular filling pressure secondary to heart defect but with pulmonary disease (hypoxia causes vasoconstriction, remodeling of small pulmonary arteries, and loss of pulmonary capillaries due to emphysema). Elevated RVSP leads to secondary tricuspid regurgitation, which had a deleterious effect on long-term mortality. Patients suffering from COPD are more prone to postoperative respiratory failure. COPD increased the risk of ventilator dependence, reintubation, atelectasis, acute respiratory distress syndrome, pneumonia, mediastinitis, and wound infections after cardiac surgery [8]. Therapy with glucocorticosteroids impairs wound healing and is a bleeding factor. Ando et al. [9] compared outcomes of 1200 COPD patients qualified for SAVR and TAVI. Tracheostomy, acute respiratory failure, reintubation, and pneumonia occur more frequently after SAVR than after TAVI. Pain caused by classical cardiac surgery increased consumption of analgetic, which suppresses the respiratory drive and may result in respiratory failure, causing atelectasis or aspiration pneumonia [9]. The pulmonary evaluation seems to be obligatory before qualification for the AS treatment because COPD is a risk factor for poor outcomes. The effect of COPD on mortality after TAVI was not observed, which might indicate the predominance of this method in this group of patients. The postprocedure respiratory failure eliminates this advantage.

Another independent predictor of long-term mortality after TAVI was RBC distribution width (RDW), which is an anisocytosis parameter and illustrates differences in the volume of RBCs. The influence of RDW on mortality was observed in cardiovascular diseases, such as heart failure, myocardial infarction, coronary artery disease, pulmonary embolism, stroke, and sudden cardiac arrest [10–14]. Thus, RDW is recognized as a risk factor for mortality after non-cardiac surgeries and cardiac surgeries [15, 16]. These results are in line with previous findings in our clinic [17–20]. Duchnowski et al. [17–20] observed the impact of RDW on the occurrence of perioperative multiple organ dysfunction, stroke, acute kidney injury (AKI) requiring dialysis, and 30-day dismal prognosis. The explanation of this correlation is ambiguous. The most likely mechanisms are oxidative stress, inflammation, and malnutrition [21]. RDW is a possible indicator of low stroke volume and organ hypoperfusion. Renal ischemia and decreased erythropoietin production [22] might result in erythropoiesis disturbances. Patients who suffered from anemia frequently required RBC transfusion during the perioperative period, which is a predictor of long-term mortality and AKI [23]. Anemia leads to platelet dysfunction, thus increasing the risk of bleeding. Moreover, DAPT, by increasing the risk of bleeding, probably elevates RDW and the risk of mortality.

Interestingly NOAF was an independent predictor of long-term mortality after TAVI, but not after SAVR. The results of the PARTNER study indicate, that NOAF after TAVI is associated with a higher risk of permanent pacemaker (PPM) implantation and renal failure requiring dialysis [24]. NOAF is a risk factor for prolonged hospitalization, and staying in the intensive care unit [25] also increases the risk of rehospitalization [24]. After TAVI, patients with NOAF had a higher functional class in the New York Heart Association (NYHA), which unfortunately might reduce the effectiveness of the procedure in this subset of patients [25]. So far, there have been no clear recommendations regarding anticoagulation after TAVI with NOAF. Therefore, it appears that patients after TAVI complicated by NOAF received an oral anticoagulant (OAC) drug in addition to dual antiplatelet therapy (DAPT). In the vast majority of cases, elderly patients with many comorbidities, such as chronic kidney disease and hypertension, were qualified for TAVI. Hence, they were at higher risk of bleeding complications during DAPT and OAC combined therapy. A question about perioperative anticoagulant therapy needs to be considered. Due to the risk of AF complicated by stroke or peripheral embolism (often asymptomatic), hemorrhagic complications, and subclinical valve thrombosis [26, 27] DAPT, one should consider replacing them with OAC.

The independent risk factor of long-term mortality after SAVR were stroke and TIA. Stroke was a risk factor

for 6-, 12- and 24-month mortality in other centers [29]. Knowledge about causes, timing, and risk factors of stroke is crucial because stroke leads to disability, reduces quality of life, and cost-effectiveness of treatment. The DeNOVO and Neuro-TAVI studies revealed the underestimation of the cerebrovascular events [28, 29]. The use of protective systems during TAVI appears to reduce the incidence of major disabling strokes, but non-disabling cerebrovascular events occur more commonly [30, 31]. Most likely, therefore, periprocedural stroke after TAVI has not increased the risk of death in long-term follow-up.

RBC transfusion was a risk factor for long-term mortality after SAVR and TAVI. Transfusions had deleterious effects, especially on the kidneys. During storage, erythrocytes undergo structural changes and are more susceptible to deformation. Hence, RBCs adhere to the vessel walls more easily, accumulate pro-inflammatory molecules, and release pro-thrombotic lipids [23]. Therefore, transfused erythrocytes promote a pro-inflammatory state and oxidative stress, which leads to the activation of leukocytes and clotting pathways [32]. The necessity of RBC transfusion indicates hemodynamic instability and thus the risk of renal ischemia and mortality.

Our study confirmed the usefulness of TAVI, especially in elderly patients, with coexisting COPD and neurological disorders. Long-term outcomes of TAVI and SAVR in elderly patients with an increased risk of bleeding were comparable. In a group of patients assigned to SAVR, preoperative anemia was a predictive factor for dismal prognosis while the occurrence of NOAF after TAVI with indications of OAC was related to bleak outcomes.

Limitations

The study is a retrospective analysis of single-center outcomes of aortic stenosis treatment, which may have a bias in the selection of the patient group.

Patients were not randomly assigned to particular groups but enrolled in the TAVI or SAVR groups, based on the heart team's decision, which may illustrate a specific preference for aortic stenosis treatment in our hospital.

Although the study group reflects the real population, the number of TAVI-treated patients was relatively small, which may limit the value of the analysis.

CONCLUSIONS

- No significant differences regarding the prognosis for up to 24 months between SAVR and TAVI were found in groups of consecutive patients in the propensity score model.
- Independent predictive factors of late mortality after both procedures elevated risk score (EuroSCORE II >4%) and postprocedure respiratory failure.
- Independent risk factors of late mortality specific for TAVI were the new onset of atrial fibrillation and the increased red blood cell distribution.
- Independent predictive factors of late mortality specific for SAVR were stroke/TIA, COPD, and RBC transfusion.

Article information

Conflict of interest: None declared.

Funding: None.

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REFERENCES

1. Vahanian A, Beyersdorf F, Praz F, et al. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J. 2021; 43(7): 561–632, doi: 10.1093/eurheartj/ehab395, indexed in Pubmed: 34453165.
2. Pibarot P, Salaun E, Dahou A, et al. Transcatheter aortic-valve replacement with a balloon-expandable valve in low-risk patients. N Engl J Med. 2019; 380(18): 1695–1705, doi: 10.1056/NEJMoa1814052, indexed in Pubmed: 30883058.
3. Takagi H, Hari Y, Nakashima K, et al. A meta-analysis of ≥5-year mortality after transcatheter versus surgical aortic valve replacement. J Cardiovasc Surg (Torino). 2020; 61(1): 107–116, doi: 10.23736/S0021-9509.19.11030-0, indexed in Pubmed: 31666501.
4. Ludman PF, Moat N, de Belder MA, et al. Transcatheter aortic valve implantation in the United Kingdom: temporal trends, predictors of outcome, and 6-year follow-up: a report from the UK Transcatheter Aortic Valve Implantation (TAVI) Registry, 2007 to 2012. Circulation. 2015; 131(13): 1181–1190, doi: 10.1161/CIRCULATIONAHA.114.013947, indexed in Pubmed: 25637628.
5. Weissman C. Pulmonary complications after cardiac surgery. Semin Cardiothorac Vasc Anesth. 2016; 8(3): 185–211, doi: 10.1177/1089253 20400800303.
6. Ando T, Adegbala O, Akintoye E, et al. Is transcatheter aortic valve replacement better than surgical aortic valve replacement in patients with chronic obstructive pulmonary disease? A nationwide inpatient sample analysis. J Am Heart Assoc. 2018; 7(7), doi: 10.1161/JAHA.117.008408, indexed in Pubmed: 29606641.
7. Dębiński M, Domaradzki W, Fil W, et al. Longterm outcomes of transcatheter self-expanding aortic valve implantations in inoperable and high surgical-risk patients with severe aortic stenosis: a single-center single-valve registry. Kardiol Pol. 2021; 79(3): 319–326, doi: 10.33963/KP.15821, indexed in Pubmed: 33599461.
8. Weismann C. Pulmonary complications after cardiac surgery. Semin Cardiothorac Vasc Anesth. 2004; 8(3): 185–211, doi: 10.1177/108925320 400800303 , indexed in Pubmed: 15375480.
9. Gilard M, Eltchaninoff H, Donzeau-Gouge P, et al. Late outcomes of transcatheter aortic valve replacement in high-risk patients: the FRANCE-2 registry. J Am Coll Cardiol. 2016; 68(15): 1637–1647, doi: 10.1016/j.jacc.2016.07.747, indexed in Pubmed: 27712776.
10. Tonelli M, Wiebe N, James MT, et al. Red cell distribution width associations with clinical outcomes: A population-based cohort study. PLoS One. 2019; 14(3): e0212374, doi: 10.1371/journal.pone.0212374, indexed in Pubmed: 30865651.
11. Söderholm M, Borné Y, Hedblad Bo, et al. Red cell distribution width in relation to incidence of stroke and carotid atherosclerosis: a population-based cohort study. PLoS One. 2015; 10(5): e0124957, doi: 10.1371/journal.pone.0124957, indexed in Pubmed: 25950717.
12. Allen LA, Felker GM, Mehra MR, et al. Validation and potential mechanisms of red cell distribution width as a prognostic marker in heart failure. J Card Fail. 2010; 16(3): 230–238, doi: 10.1016/j.cardfail.2009.11.003, indexed in Pubmed: 20206898.
13. Dabbah S, Hammerman H, Markiewicz W, et al. Relation between red cell distribution width and clinical outcomes after acute myocardial infarction.

Am J Cardiol. 2010; 105(3): 312–317, doi: 10.1016/j.amjcard.2009.09.027, indexed in Pubmed: 20102941.
14. Kim J, Kim K, Lee JH, et al. Red blood cell distribution width as an independent predictor of all-cause mortality in out of hospital cardiac arrest. Resuscitation. 2012; 83(10): 1248–1252, doi: 10.1016/j.resuscitation.2012.01.038, indexed in Pubmed: 22322281.
15. Abdullah HR, Sim YE, Sim YT, et al. Preoperative red cell distribution width and 30-day mortality in older patients undergoing non-cardiac surgery: a retrospective cohort observational study. Sci Rep. 2018; 8(1): 6226, doi: 10.1038/s41598-018-24556-z, indexed in Pubmed: 29670189.
16. Sim YE, Wee HE, Ang AL, et al. Prevalence of preoperative anemia, abnormal mean corpuscular volume and red cell distribution width among surgical patients in Singapore, and their influence on one year mortality. PLoS One. 2017; 12(8): 1–19, doi: https://doi.org/10.1371/journal.pone.0182543.
17. Duchnowski P, Hryniewiecki T, Kuśmierczyk M, et al. Red cell distribution width as a predictor of multiple organ dysfunction syndrome in patients undergoing heart valve surgery. Biol Open. 2018; 7(10), doi: 10.1242/bio.036251, indexed in Pubmed: 30127093.
18. Duchnowski P, Szymanski P, Orłowska-Baranowska E, et al. Raised red cell distribution width as a prognostic marker in aortic valve replacement surgery. Kardiol Pol. 2016; 74(6): 547–552, doi: 10.5603/KP.a2015.0213, indexed in Pubmed: 26502945.
19. Duchnowski P, Hryniewiecki T, Kusmierczyk M, et al. Red cell distribution width is a prognostic marker of perioperative stroke in patients undergoing cardiac valve surgery. Interact Cardiovasc Thorac Surg. 2017; 25(6): 925–929, doi: 10.1093/icvts/ivx216, indexed in Pubmed: 29049563.
20. Duchnowski P, Hryniewiecki T, Kuśmierczyk M, et al. Anisocytosis predicts postoperative renal replacement therapy in patients undergoing heart valve surgery. Cardiol J. 2020; 27(4): 362–367, doi: 10.5603/CJ.a2019.0020, indexed in Pubmed: 30799549.
21. Semba RD, Patel KV, Ferrucci L, et al. Serum antioxidants and inflammation predict red cell distribution width in older women: The Women's Health and Aging Study I. Clin Nutr. 2010; 29(5): 600–604.
22. Ronco C, Chionh CY, Haapio M, et al. Cardiorenal syndrome: refining the definition of a complex symbiosis gone wrong. Intensive Care Med. 2008; 34(5): 957–962, doi: 10.1007/s00134-008-1017-8, indexed in Pubmed: 18251008.
23. Najjar M, Salna M, George I. Acute kidney injury after aortic valve replacement: incidence, risk factors and outcomes. Expert Rev Cardiovasc Ther. 2015; 13(3): 301–316, doi: 10.1586/14779072.2015.1002467, indexed in Pubmed: 25592763.
24. Biviano AB, Nazif T, Dizon J, et al. Atrial Fibrillation Is Associated With Increased Mortality in Patients Undergoing Transcatheter Aortic Valve Replacement: Insights From the Placement of Aortic Transcatheter Valve (PARTNER) Trial. Circ Cardiovasc Interv. 2016; 9(1): e002766, doi: 10.1161/CIRCINTERVENTIONS.115.002766, indexed in Pubmed: 26733582.
25. Chopard R, Teiger E, Meneveau N, et al. Baseline Characteristics and Prognostic Implications of Pre-Existing and New-Onset Atrial Fibrillation After Transcatheter Aortic Valve Implantation: Results From the FRANCE-2 Registry. JACC Cardiovasc Interv. 2015; 8(10): 1346–1355, doi: 10.1016/j.jcin.2015.06.010, indexed in Pubmed: 26315738.
26. Makkar RR, Fontana G, Søndergaard L, et al. Possible subclinical leaflet thrombosis in bioprosthetic aortic valves. N Engl J Med. 2015; 373(21): 2015–2024, doi: 10.1056/NEJMoa1509233, indexed in Pubmed: 26436963.
27. Hansson NC, Grove EL, Andersen HR, et al. Transcatheter aortic valve thrombosis: incidence, predisposing factors, and clinical implications. J Am Coll Cardiol. 2016; 68(19): 2059–2069, doi: 10.1016/j.jacc.2016.08.010, indexed in Pubmed: 27580689.
28. Messé SR, Acker MA, Kasner SE, et al. Stroke after aortic valve surgery: results from a prospective cohort. Circulation. 2014; 129(22): 2253–2261, doi: 10.1161/CIRCULATIONAHA.113.005084, indexed in Pubmed: 24690611.
29. Lansky AJ, Brown D, Pena C, et al. Neurologic complications of unprotected transcatheter aortic valve implantation (from the Neuro-TAVI trial). Am J Cardiol. 2016; 118(10): 1519–1526, doi: 10.1016/j.amjcard.2016.08.013, indexed in Pubmed: 27645761.
30. Kapadia SR, Kodali S, Makkar R, et al. Protection against cerebral embolism during transcatheter aortic valve replacement. J Am Coll Cardiol. 2017; 69(4): 367–377, doi: 10.1016/j.jacc.2016.10.023, indexed in Pubmed: 27815101.
31. Seeger J, Kapadia SR, Kodali S, et al. Rate of peri-procedural stroke observed with cerebral embolic protection during transcatheter aortic valve replacement: a patient-level propensity-matched analysis. Eur Heart J. 2019; 40(17): 1334–1340, doi: 10.1093/eurheartj/ehy847, indexed in Pubmed: 30590554.
32. Karkouti K, Wijeysundera DN, Yau TM, et al. Acute kidney injury after cardiac surgery: focus on modifiable risk factors. Circulation. 2009; 119(4): 495–502, doi: 10.1161/CIRCULATIONAHA.108.786913, indexed in Pubmed: 19153273.