The Evolution and Prognostic Value of N-Terminal Brain Natriuretic Peptide in Predicting 1-Year Mortality in Patients Following Transcatheter Aortic Valve Implantation

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ABSTRACT: Aims. N-terminal pro-brain natriuretic peptide (NT-proBNP) has been found to correlate with the severity of aortic valve stenosis and to provide prognostic information in aortic stenosis patients undergoing surgical aortic valve replacement. There is a paucity of data describing the association between clinical outcomes after TAVI and NT-proBNP levels. We investigated the evolution and prognostic value of NT-proBNP levels after TAVI. **Method and Results.** We prospectively collected data on the baseline characteristics, NT-proBNP levels (baseline, post-treatment and discharge) and adverse clinical outcomes of patients undergoing TAVI from 2007 to 2010. Using a univariable and multivariable Cox regression model, pre- and postimplantation NT-proBNP tertile levels were correlated to 30-day and 1-year mortality. A total of 373 patients underwent TAVI with either the Medtronic CoreValve or Edwards SAPIEN prosthesis. The cumulative 30-day and 1-year mortality was 7.3% and 18%, respectively. Rehospitalization for heart failure was observed in 0.8% at 30 days and 7.8% at 1 year. The tertile baseline NT-proBNP levels were identified as ≤1570 ng/L, 1571 to 4690 ng/L and ≥4691 ng/L. In the univariable analysis, baseline (HR, 1.01; 95% CI, 1.001-1.02; *P*=.02) and post-treatment NT-proBNP (HR 1.02; 95% CI, 1.002-1.04; *P*=.04) were predictors for 1-year mortality. In the multivariable analysis, however, only baseline NT-proBNP and atrial fibrillation were identified as predictors for the 1-year mortality (HR, 1.02; 95% CI, 1.01-1.05; *P*=.006 and HR, 3.4; 95% CI, 1.25-9.5; *P*=.017, respectively). **Conclusions.** NT-proBNP and atrial fibrillation were predictors for 1-year mortality, offer independent prognostic information, and identify patients being at increased risk for mortality. Thus, NT-proBNP reveals more incremental value for patient selection and should be included in the risk stratification of patients undergoing TAVI.

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Key words: NT-proBNP, TAVI, prognostic value of BNP, biomarkers in TAVI

Abbreviations

NT-proBNP	N-terminal brain natriuretic peptide
SAVR	surgical aortic valve replacement
AS	aortic stenosis
TAVI	transcatheter aortic valve implantation
NYHA	New York Heart Association
LVEF	left ventricular ejection fraction

Brain type natriuretic peptide (BNP) is an amino acid protein synthesized by the myocardium that is cleaved into two fragments yielding BNP and NT-proBNP. The 2008 European Heart Failure Guidelines highlight BNP as a diagnostic and prognostic marker in heart failure patients.[1] In patients with aortic stenosis, BNP levels correlate with functional status and can also serve as a prognostic marker for patients with asymptomatic or symptomatic aortic valve stenosis undergoing surgical aortic valve replacement (SAVR)[2-6] or those undergoing balloon aortic valvuloplasty.[7]

There is very little information, however, about the prognostic implications of BNP levels from patients undergoing transcatheter aortic valve implantation (TAVI). The aims of this study are to: (1) characterize pre- and postprocedural BNP levels in patients with severe AS undergoing TAVI; and (2) to examine the correlation between NT-proBNP levels and clinical outcomes.

Methods

Study population. From November 2007 to April 2010 we prospectively collected the pre- and post-treatment data of 373 consecutive patients with severe aortic valve stenosis who underwent TAVI with either the Medtronic CoreValve (Medtronic CoreValve) or Edwards SAPIEN (Edwards Lifesciences) prosthesis. Patient screening and TAVI (including feasibility and safety, vascular access site and prosthesis size selection) were discussed among a multidisciplinary team consisting of cardiac surgeons, invasive and non-invasive cardiologists, and anesthesiologists. All patients provided informed consent prior to treatment. Patients without complete data were excluded from the study.

Clinical follow-up exams, during a period of 12 ± 2 months, were obtained by clinical visits and/or through telephone contacts. Death and cardiac rehospitalization were recorded from all patients.

Procedural technique. Procedural details have been published elsewhere.[8] Briefly, under conscious sedation or general anesthesia, patients underwent implantation of the Medtronic

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Table 1. Baseline characteristics.

	Entire Cohort n = 373
NT-proBNP (ng/L)	2710 (1177-6332)
Age (years)	81 (77-85)
Male	139 (37.2%)
Height (cm)	164.7 ± 8.7
Weight (kg)	70.8 ± 14.4
Body mass index	26.1 ± 4.35
Body surface area (m^2)	1.77 ± 0.19
NYHA class III or IV	360 (96.3%)
Previous cerebrovascular event	49 (14.2%)
Coronary artery disease	206 (55.1%)
Previous percutaneous coronary intervention	98 (26.2%)
Peripheral vascular disease	95 (25.5%)
Creatinine (mg/dL)	1.2 ± 0.6
Chronic obstructive pulmonary disease	72 (19.3%)
Atrial fibrillation	83 (22.2%)
Logistic EuroSCORE (%)	20.4 ± 13.2
STS score (%)	6.17 ± 4.1
Echocardiographic data	
Left ventricular ejection fraction <35%	56 (16.1%)
Mean gradient	47 ± 16.5
Aortic valve area (cm^2)	0.67 ± 0.2
Interventricular septal wall thickness	14.6 ± 2
Pulmonary hypertension	82 (22%)
Left ventricular end diastolic dimension	47.7 ± 7.2
Left ventricular outflow tract	20.3 ± 2.3
Aortic valve annulus by TEE (mm)	23.3 ± 2
Procedural data	
Medtronic CoreValve	261 (69.8%)
Post-implant dilatation	66 (17.6%)
Contrast media used (mL)	126 ± 56.7

Data given as median (interquartile range), n (percentage), or mean ± standard deviation. NYHA = New York Heart Association; STS score = Society of Thoracic Surgeons Predicted Risk of Mortality; TEE = transesophageal echocardiogram.

CoreValve (Medtronic CoreValve) or Edwards SAPIEN (Edwards Lifesciences, Inc) device in a hybrid procedural suite via a transfemoral, transaxillary, transapical, or transaortic route. The transfemoral procedures were performed using a surgical cut-down or pre-closure techniques with one ProStar and one ProGlide device (Abbott).

Measurement of BNP. Blood samples for BNP levels were drawn into chilled EDTA acid test tubes the day before TAVI (baseline), immediately after (post-treatment) and on the day of hospital discharge (7 ± 4 days post implantation). After 20 minutes of centrifugation, separated plasma samples were processed by a fluorescence immunoassay for measurement of B-type natriuretic peptide levels (Elecsys proBNP; Roche Diagnostics GmbH).

Echocardiographic data. At study entry, all patients underwent echocardiographic evaluation at baseline, at discharge, and at 1 year after TAVI. The echocardiographic protocol contain measurement of the aortic annulus, the interventricular septum, the aortic valve peak and mean gradient, aortic valve area and the left ventricular ejection fraction using commercially available ultrasound systems (Siemens, ACUSON. Sequoia. 512). Left ventricular ejection fraction (LVEF) was estimated according to Simpson's rule. The simplified Bernoulli equation was used to calculate the transaortic gradients and the aortic valve area was calculated by the continuity equation. All echocardiographic examinations were performed by experienced echocardiographers.

Study endpoints. Primary endpoints were all-cause mortality and rehospitalization for heart failure at 30 days and 1-year after TAVI. Outcome variables were collected from patient interviews (ambulatory clinic or telephone calls) using a standardized questionnaire or review of electronic hospital records.

Statistics. Continuous variables were presented as means ± standard deviation or medians ± interquartile ranges (IQRs). Comparisons of continuous variables between groups were performed with the Student's t-test or the Mann-Whitney U-test in case of non-parametric distribution. Categorical variables were compared with the chi-square or Fisher's exact test if the expected frequency was less than 5. A two-sided *P*-value <.05 was considered to be significant.

For the analysis, NT-proBNP levels were divided into tertiles (≤1570 ng/L, 1571 to 4690 ng/L, and ≥4691 ng/L). Consequently, three groups were identified: first tertile with 124 patients, second tertile with 126 and third tertile with 123 patients. Unpaired Student t-tests or repeated-measures ANOVA with Bonferroni correction for multiple comparisons were used to compare continuous variables between the tertile groups.

Kaplan-Meier estimator curves were used to examine the distribution of adverse outcomes on the basis of NT-proBNP tertiles. Log-rank test was used to compare differences between stratified groups.

Using the Cox's proportional hazards model, we studied the univariable and multivariable association between mortality and NT-proBNP (at baseline, post-treatment and discharge) and baseline characteristics (see variables in Table 1). Models were generated with a forward stepwise analysis. Factors with a *P*-value <.20 in the univariable analysis were entered into the multivariable analysis.

All data were analyzed with SPSS software (version 15 for Windows; SPSS Inc).

Results

Baseline characteristics. A total of 373 consecutive patients with severe aortic valve stenosis were enrolled in the study.

Table 2. Clinical and biochemical characteristics stratified according to baseline NT-proBNP tertiles.

	First Tertile ≤1570 ng/L n = 124	Second Tertile 1571 to 4690 ng/L n = 126	Third Tertile ≥4691 ng/L n = 123	*P*-Value
Baseline data				
Age (years)	79.3 ± 6.4	80.7 ± 6.7	81.8 ± 7.3	.014§
Male	40 (32%)	43 (34)	56 (45.5)	.061
Height (cm)	163.7 ± 8.4	164.2 ± 8.8	165.8 ± 8.7	.15
Weight (kg)	72.7 ± 15.6	70.8 ± 14	68.7 ± 13.2	.08
CoreValve implantation	89 (71%)	85 (67)	87 (70)	.75
Body surface area (m^2)	1.78 ± 02	1.76 ± 0.19	1.75 ± 0.19	.6
NYHA class III or IV	121 (96%)	117 (92%)	122 (99%)	.02*
Previous cerebrovascular event	20 (16.3%)	23 (18%)	14 (11%)	.29
Coronary artery disease	65 (52%)	69 (54%)	72 (59%)	.62
Previous percutaneous coronary intervention	36 (29%)	29 (23%)	33 (26.8%)	.5
Peripheral vascular disease	25 (20%)	30 (23%)	40 (32%)	.078
Creatinine (mg/dL)	1.02 ± 0.38	1.10 ± 0.37	1.48 ± 0.83	<.001*
Chronic obstructive pulmonary disease	21 (16.9%)	21 (16.7%)	30 (24%)	.21
Atrial fibrillation	12 (9.6%)	41 (32%)	31 (25%)	<.001*
Logistic EuroSCORE (%)	13.9 ± 8.4	19.4 ± 12.4	27.9 ± 14	<.001*
STS score (%)	4.8 ± 2.8	5.38 ± 3	8.3 ± 5.2	<.001#
Echocardiographic data				
Left ventricular ejection fraction >50%	109 (87.2%)	79 (62.7%)	45 (36.6%)	<.001*
Left ventricular ejection fraction 35%-50%	12 (9.6%)	37 (29.4%)	35 (28.5%)	<.001*
Left ventricular ejection fraction ≤35%	4 (3.2%)	10 (7.9%)	43 (35%)	<.001*
Mean gradient (mm Hg)	45.4 ± 13.6	48.7 ± 15.5	46.7 ± 19.8	.28
Aortic valve area (cm^2)	0.71 ± 0.18	0.68 ± 0.19	0.63 ± 0.23	.89
Interventricular septal wall thickness (mm)	14.7 ± 2	14.5 ± 2	14.5 ± 2.1	.89
Left ventricular end diastolic dimension (mm)	44.7 ± 6.5	47.1 ± 7.5	50.3 ± 6.7	<.001†
Left ventricular outflow tract (mm)	20.2 ± 2	20.4 ± 2.1	20.3 ± 2.3	.73
Aortic valve annulus (mm)	24 ± 1.9	23.2 ± 2	23.5 ± 2.1	.005§
Outcomes				
Cardiac rehospitalization	5 (4.3%)	11 (9.8%)	12 (10.7%)	.16
30-day mortality	8 (6.4%)	8 (6.3%)	6 (4.9%)	.84
1-year mortality	18 (14.9%)	24 (20.2%)	35 (29.2%)	.024*

Data given as mean ± standard deviation or n (percentage).
NYHA = New York Heart Association; STS score = Society of Thoracic Surgeons predicted risk of mortality.
**significant difference between all tertiles; §significant difference between first und third tertile; †significant difference between first and third, second and third tertile.*

Clinical characteristics and echocardiographic data of all patients are summarized in Table 1. Figures 1A and 1B illustrated the relationship between NT-proBNP levels and NYHA class, NT-proBNP and LVEF. Plasma levels of NT-proBNP correlates positively with the NYHA classification but negatively with the LV ejection fraction.

NT-proBNP tertile analysis. NT-proBNP tertiles were ≤1570 ng/L, 1571 to 4690 ng/L, and ≥ 4691 ng/L. Table 2 summarizes the differences between the baseline NT-proBNP according to tertiles. Patients in the 3rd BNP tertile exhibited significantly higher preoperative creatinine levels than patients in the 1st tertile (1.48 ± 0.83 mg/dL (3rd tertile) vs 1.02 ± 0.38 mg/dL (1st tertile); *P*<.001), a 2-fold higher logistic EuroSCORE and STS score (Logistic EuroSCORE, 27.3 ± 14% vs 13.9 ± 8.4%, respectively; *P*<.001) (STS score, 8.3 ± 5.2% vs 4.8 ± 2.8%, respectively; *P*<.001). Furthermore, patients in the

Table 3. Changes on NT-proBNP levels.

	Transfemoral	Transapical	*P*-Value	Female	Male	*P*-Value
Baseline	2430 (1050-6330)	2520 (965-6275)	.78	1980 (853-5275)	2970 (1320-7680)	.2
Post treatment	1550 (633-4030)	1530 (872-3975)	.27	1390 (682-2985)	2580 (583-5850)	.02
Discharge	2980 (1170-6520)	3650 (1915-8595)	.94	2630 (6630-1120)	3750 (1600-6980)	.41

Data given as median (interquartile range).

3rd tertile had a 3 to 4-fold higher frequency of atrial fibrillation than patients in the 1st tertile (32% vs 9.6%, respectively, *P*<.001). There were no significant differences in 30-day mortality (*P*=.84) and cardiac rehospitalization rate (*P*=.16) among the NT-proBNP tertiles but a higher 1-year mortality in the 3rd tertile (*P*=.024).

Evolution of NT-proBNP levels during hospitalization. The median baseline NT-proBNP level was 2710 ng/L (IQR, 1177-6332). NT-proBNP changes are shown in Table 3. Median NT-proBNP levels decreased by approximately 40% immediately after TAVI (baseline 2460 ng/L (IQR, 1080-6410) vs post-treatment 1550 ng/L (IQR, 681-4030; *P*<.001). From post-treatment to hospital discharge, median NT-proBNP levels increased from 1550 ng/L (IQR, 681-4030) to 3070 (IQR, 1270-6670 ng/L), (*P*<.001). There were no statistically significant differences in NT-proBNP levels between the transfemoral and transapical subgroups either at baseline or at hospital discharge. Comparing the NT-proBNP levels in men and women, men presented a higher baseline NT-proBNP than women. The post-treatment decrease in NT-proBNP is higher in females than males (*P*=.02).

Thirty-day and 1-year mortality and cardiac rehospitalization Patients were followed up for a median of 1.1 (1.0-1.4) years. The 30-day and 1-year mortality rate were 7.3% and 18%, respectively. Cardiac rehospitalization for acute heart failure was observed in 28 patients (7.5%). There were no statistically significant differences in baseline NT-proBNP in those patients with and without rehospitalization (1590 [671-5410] ng/L vs 2455 [1197-6432] ng/L; P=.28).

Kaplan Meier survival analyses stratified according to tertile NT-proBNP levels at baseline and post-treatment are shown in Figure 2. The survival rates after 12 months were 88.5%, 77.1%, and 77.7% among the 1st, 2nd, and 3rd tertile, respectively.

In the 30-day mortality, there was no significant difference between the baseline and post-treatment NT-proBNP tertiles (Figures 2A and 2C). In the 1-year mortality however, there was a significant difference in the 1-year mortality in baseline and post-treatment NT-proBNP tertiles (Figures 2D and 2B).

Predictors of 1-year mortality. Table 4 summarizes predictors for 1-year mortality. In the univariable analysis: age, gender, NYHA class, chronic obstructive pulmonary disease (COPD), left ventricular ejection fraction, left ventricular end-diastolic dimension, peripheral vascular disease, creatinine level, atrial fibrillation, logistic EuroSCORE, STS Score, NT-proBNP at baseline, post-treatment, and at hospital discharge were identified as univariable predictors for mortality in patients undergoing

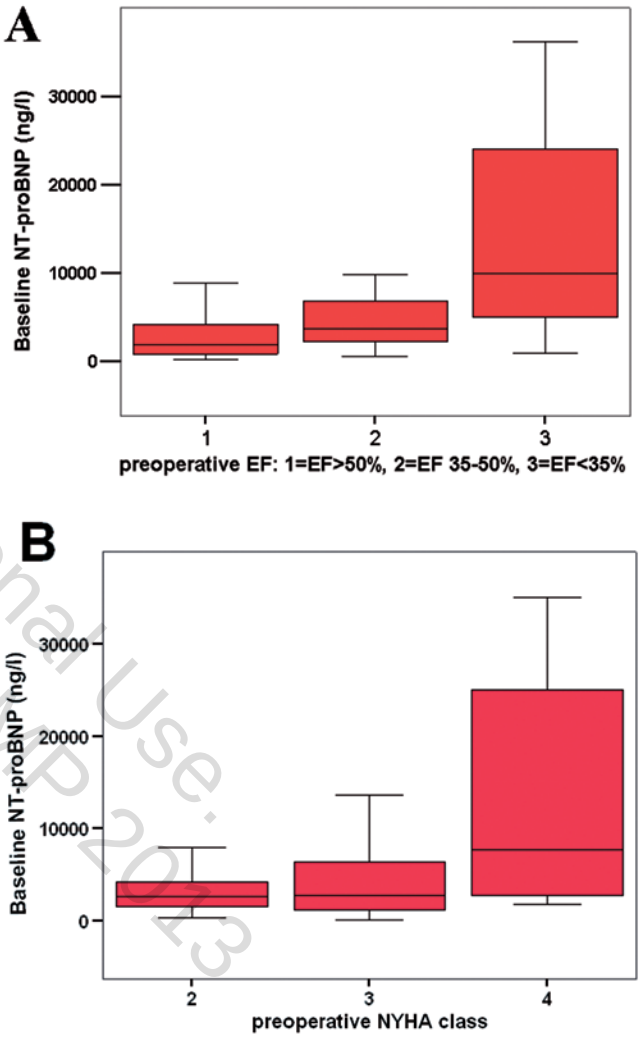

Figure 1. *Plasma level of baseline NT-proBNP and correlation with* **(A)** *preoperative ejection fraction (EF) and* **(B)** *the preoperative NYHA classification at beginning of study.*

TAVI. On multivariable analysis, only baseline NT-proBNP and atrial fibrillation remained as independent predictors for 1-year mortality (HR, 1.02; 95% CI, 1.01-1.05; *P*=.006 and HR, 3.4; 95% CI, 1.25-9.5; *P*=.017, respectively).

Discussion

In the present study, the median baseline NT-proBNP was 2710 ng/L (IQR, 1177-6332) and BNP tertiles were identified as ≤1570 ng/L, 1571 to 4690 ng/L and ≥4691 ng/L. The major findings of this study were:

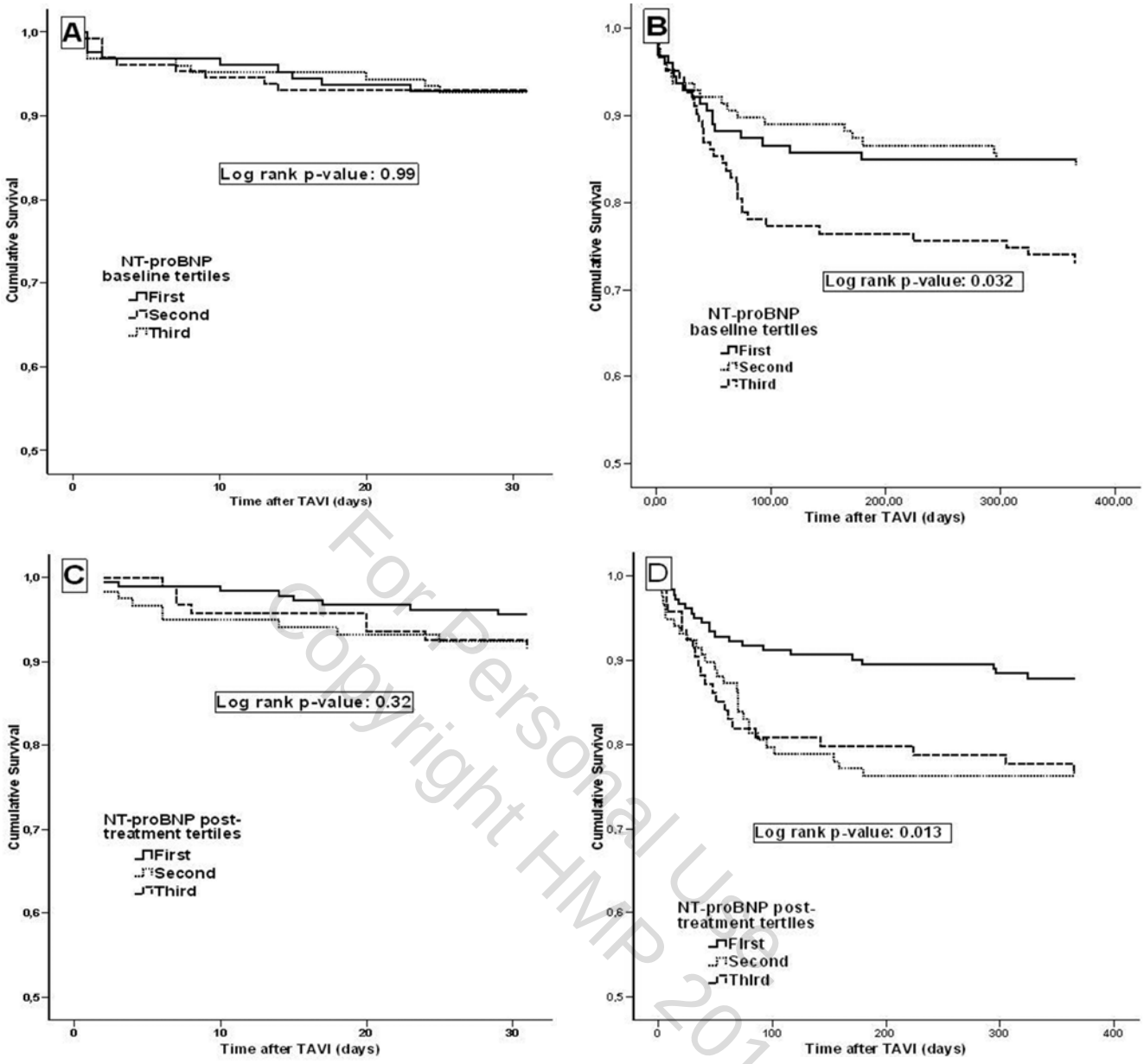

Figure 2. *Results of Kaplan-Meier analysis for 30-day and 1-year mortality between all patients stratified into first, second, and third tertiles based on* **(A, B)** *baseline NT-proBNP and* **(C, D)** *post-treatment NT-proBNP.*

(1) Baseline NT-proBNP tertiles correlated with patient risk profile such that patients in the last tertile were older, more symptomatic (NYHA), had higher incidence of peripheral vascular disease, left ventricular dysfunction, atrial fibrillation, higher STS and logistic EuroSCORE;
(2) Median baseline NT-proBNP levels decreased significantly after TAVI;
(3) Patients in the last NT-proBNP tertile had significantly higher 1-year mortality than patients in the first or second tertiles;
(4) Baseline NT-proBNP levels and atrial fibrillation were independent predictors of 1-year mortality.

Patients undergoing TAVI exhibited high levels of baseline NT-proBNP (median 2710 ng/L, IQR 1177-6332). Berry et al also observed high baseline levels of NT-proBNP (mean 10059 ± 12117 ng/L) in patients undergoing TAVI.[9] On the other hand, patients undergoing surgical aortic valve replacement typically have lower baseline NT-proBNP levels (mean values ranging from 157 to 623 ng/L.[10-12] These differences in NT-proBNP levels can be explained by the patient's baseline risk profile.

Very few previous studies on TAVI have reported data on the BNP changes after TAVI. Sherif et el compared early hemodynamic and neurohormonal changes in surgical aortic valve replacement (SAVR) and TAVI-group,[13] they found, 30 days after the implantation, a notable improvement in the LV function in patients with moderately to severely impaired LV function in TAVI patients, suggesting an earlier reverse cardiac remodeling process.

Table 4: Univariable and multivariable analysis of predictors of 1-year mortality.

	Wald	Hazard Ratio (95% CI)	*P*-Value
Univariable Cox regression			
Age (years)	1.5	1.01 (0.94-1.09)	.2
Male (%)	0.16	0.9 (0.55-1.4)	.68
Height (cm)	0.004	0.9 (0.97-1.02)	.95
Weight (kg)	0.5	0.99 (0.97-1.01)	.48
NYHA class III or IV	0.073	1.2 (0.29-4.9)	.78
Peripheral vascular disease	3.12	1.5 (0.95-2.6)	.077
Creatinine (mg/dL)	3.73	1.4 (0.99-1.99)	.05
Chronic obstructive pulmonary disease (%)	3.6	1.5 (0.99-2.3)	.05
Atrial fibrillation (%)	1.68	1.4 (0.83-2.4)	.19
Left ventricular ejection fraction >50%	8.3	0.48 (0.3-0.78)	.003
Left ventricular ejection fraction 35%-50%	1.01	1.32 (0.7-2.2)	.31
Left ventricular ejection fraction ≤35%	8.5	2.2 (1.3-3.8)	.004
Left ventricular enddiastolic dimension (mm)	1.2	1.02 (0.98-1.06)	.26
Aortic valve annulus (mm)	0.2	1.04 (0.85-1.28)	.65
Logistic EuroSCORE (%)	2.9	1.01 (0.99-1.03)	.08
STS score (%)	15	1.08 (1.04-1.12)	<.001
Baseline NT-proBNP (ng/L)	4.9	1.01 (1.002-1.05)	.02
Post-treatment NT-proBNP (ng/L)	3.8	1.02 (1.002-1.04)	.04
Discharge NT-proBNP (ng/L)	3.3	1.01 (1.02-1.05)	.06
Multivariable Cox regression			
Baseline NT-proBNP (ng/L)	7.6	1.02 (1.01-1.05)	.006
Atrial fibrillation (%)	5.7	3.4 (1.25-9.5)	.017
STS score (%)			.91
Logistic EuroSCORE (%)			.87

NYHA = New York Heart Association; STS score = Society of Thoracic Surgeons Predicted Risk of Mortality.

Our data confirm the association between NT-proBNP levels and aortic valve stenosis severity, and preoperative ejection fraction as reported in previous studies.[2,11,14] Similarly, other studies have demonstrated the correlation between NT-proBNP levels and transvalvular aortic pressure gradients.[15,16] This is not surprising given the fact that secretion of NT-proBNP is induced by left ventricular overload and extent left ventricular hypertrophy.

In severe aortic stenosis, the impact of surgical aortic valve replacement on BNP levels is controversial. Several studies have demonstrated up to a 25% decrease in BNP levels after SAVR or balloon valvuloplasty (BAV).[7,17] In the present study, NT-proBNP levels were measured preoperatively, post-treatment and at discharge. Patients undergoing transapical implantation had a more distinct, through not significant decrease than those undergoing transfemoral implantation, and females had a significantly more distinct decrease than males. There was a significant decrease in NT-proBNP levels immediately after the TAVI procedure likely a reflection of lower transventricular pressures. Interestingly, we observed that NT-proBNP levels increased non-significantly prior to hospital discharge. We agree with other investigators who believe, this is a result of anesthetic effects surrounding the periprocedural period[16] or as response of new-onset atrial fibrillation.[18] Other possible explanation of the NT-proBNP increase were post-procedural right ventricular pacing during the implantation.

In SAVR, Qi et al[19] found no significant decrease of NT-proBNP 4 and 12 months after the procedure. Similarly to this finding, Sherif et al[13] observed no significant differences in NT-proBNP levels 30 days after SAVR, in the TAVI group, however, they demonstrated a significant decrease in NT-proBNP in patients with normal or impaired left ventricular function. Another possible explanation is the post-implant aortic regurgitation. Future studies are certainly needed to assess the impact of aortic regurgitation on NT-proBNP levels after TAVI.

Recently, Kefer et al[20] found that both baseline and 24 hours post-treatment BNP were strong predictors of 30-day mortality in patients undergoing TAVI. In contrast, the present study did not find any association between NT-proBNP levels and 30-day mortality. Instead, we observed an association between NT-proBNP levels and 1-year mortality. These observations may indicate that the hemodynamic status of the ventricle influences long-term survival.

Several other studies have described the prognostic value of NT-proBNP in patients undergoing surgical aortic valve replacement and patients with aortic stenosis treated conservatively.[3,12,21] In one study, Bergler-Klein et al demonstrated the prognostic value of BNP in 130 patients with aortic valve stenosis during an average follow-up of 377 days. Their results demonstrated that NT-proBNP provides important prognostic information and independently predicts symptom-free survival, outcome, and left ventricular function.[2]

Other factors influencing NT-proBNP levels are important to mention. It is well established that NT-proBNP correlated with elevated pulmonary artery pressure in patients with right heat failure.[22] Patients with aortic stenosis and diastolic dysfunction presented a higher NT-proBNP levels and this

correlated with poor prognosis.[23] Furthermore, female patients may have a higher NT-proBNP levels.[24] NT-proBNP is influenced by other comorbidities such as advanced age, obesity,[25] and sleep apnea as well.

Recent studies investigate the impact of atrial fibrillation on outcomes of patients following TAVI. New-onset atrial fibrillation was seen in one-third of all patients undergoing TAVI but was not associated with higher 30-day or 1-year mortality.[26] Having said that, B-type natriuretic peptide level is increased in patients with atrial fibrillation.[27]

Study limitations. Although we enrolled a total of 373 TAVI patients, subgroup analyses into NT-proBNP tertiles resulted in relatively small sample sized cohorts. Thus, our results may be viewed as hypothesis generating with larger studies needing to prove the prognostic value of BNP in patients undergoing TAVI. Other confounders of this study are noteworthy. NT-proBNP levels could be higher in patients developing acute kidney injury.

Conclusion

The results of this study suggest an association between baseline NT-proBNP levels and 1-year mortality in patients undergoing TAVI. This information may prove useful in the risk stratification of patients following TAVI and should be considered as a biomarker in future risk stratification models.

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