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ORIGINAL PAPER

The impact of biventricular heart failure on outcomes after transcatheter aortic valve implantation

Tobias Schmidt[1] · Mintje Bohné[1] · Michael Schlüter[2] · Mitsunobu Kitamura[1] · Peter Wohlmuth[2] · Dimitry Schewel[1] · Jury Schewel[1] · Michael Schmoeckel[3] · Karl-Heinz Kuck[1] · Christian Frerker[1]

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Abstract

Aims We sought to assess the impact of different manifestations of heart failure (HF) at baseline on the short- and long-term outcomes of transcatheter aortic valve implantation (TAVI) for aortic stenosis (AS).

Methods and results Of 361 patients undergoing TAVI between May 2013 and April 2015, 185 (51%) showed clinical signs of HF at the time of admission. HF was diagnosed as isolated left ventricular (LV) and biventricular in 63 (34%) and 122 patients (66%), respectively. Acute device success (VARC-2) was achieved in 97% of patients without HF, in all patients with LV HF, and in 97% of patients with biventricular HF. Follow-up for a median of 427 days revealed significantly poorer survival in patients with biventricular HF (1-year estimate, 72.1% [95% confidence interval, 64.0–80.2%]) than in patients with LV HF (84.5% [75.2–93.8%]; $p=0.0203$) or no HF (94.3% [90.7–97.9%]; $p<0.0001$). Survival in the latter two patient subgroups was statistically not different. A diagnosis of biventricular HF was associated with a hazard ratio of 2.62 ($p=0.0089$) vs. no HF in the likelihood of death; NT-proBNP and the logistic EuroSCORE were not significantly associated with survival. Half of all deaths in patients with biventricular HF occurred within 42 days of TAVI.

Conclusion Biventricular HF is a strong predictor of mortality following TAVI for severe AS. AS in patients with LV HF should be treated without delay to avoid progression to biventricular HF. Patients with AS and biventricular HF should be monitored closely after TAVI to possibly prevent early death.

Keywords TAVI · Heart failure · Acute and long-term outcome

Introduction

Heart failure (HF), as defined by the latest guidelines of the European Society of Cardiology (ESC), is a clinical syndrome characterized by typical symptoms (e.g. breathlessness, ankle swelling and fatigue) that may be accompanied by clinical signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema) caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/or elevated intracardiac pressures [1]. This classification is independent of echocardiographically graded left ventricular function. In patients diagnosed with severe aortic stenosis (AS) clinical symptoms reflecting the disease may or may not be present. AS may remain asymptomatic for a long period of time before symptoms become apparent.[2–4] There are many clinical (older age and atherosclerotic risk factors) as well as echocardiographic (e.g. valve calcification, left ventricular ejection fraction, excessive LV hypertrophy and abnormal tissue Doppler parameters of systolic and diastolic LV function) and laboratory variables (e.g. elevated plasma levels of natriuretic peptides) known to increase the risk of symptom development [2–7]. Asymptomatic AS should be re-evaluated every 6 months, [8] whereas severe symptomatic AS has a class IB recommendation for intervention [transcatheter aortic valve implantation (TAVI) or surgical

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✉ Tobias Schmidt
tobiasschmidtmd@gmail.com

1 Department of Cardiology, Asklepios Klinik St. Georg, Lohmühlenstr. 5, 20099 Hamburg, Germany

2 Asklepios Proresearch, Hamburg, Germany

3 Department of Cardiovascular Surgery, Asklepios Klinik St. Georg, Hamburg, Germany

aortic valve replacement (SAVR)], [8] since symptoms are known to increase the risk of mortality [9]. HF secondary to AS usually manifests itself in symptoms or signs of left ventricular (LV) HF, progressing to biventricular HF in the course of months or years after the initial diagnosis [9]. An association of clinical HF, especially heart failure symptoms, related to AS with mortality has not yet been shown.

Therefore, the primary objective of this study of patients undergoing TAVI was to assess the association of baseline covariates—acute signs of HF in particular—and procedural outcomes with mortality.

Methods

Patients

Between May 2013 and April 2015, 473 patients underwent TAVI for severe AS at our institution. Patient selection for TAVI was performed by an institutional Heart Team. Exclusion criteria were non-femoral access ($n = 66$), severe aortic regurgitation as the indication for TAVI ($n = 9$), missing data ($n = 26$), and emergency TAVI in cardiogenic shock according to the definition of the IABP-Shock II trial ($n = 11$) [10]. The 361 remaining patients were divided into three groups according to the absence (No HF, $n = 176$ [49%]) or presence (LV HF, $n = 63$ [17%]; biventricular HF, $n = 122$ [34%]) of clinical signs of HF at the time of admission.

HF definition

The 2016 ESC guidelines for HF define HF as a clinical syndrome with typical symptoms (e.g. breathlessness, ankle swelling and fatigue) and signs (e.g. elevated jugular venous pressure, pulmonary crackles and peripheral oedema), which result in a reduced cardiac function [1].

Data were collected from clinical examinations, interviews patients underwent at the time of admission, as well as chest X-ray, when indicated, with special emphasis on clinical signs of HF.

Patients were categorized as having (leading) LV HF if clinical signs comprised left-sided congestion like orthopnoea, paroxysmal nocturnal dyspnoea and bilateral pulmonary rales. Biventricular HF was defined if signs of right-sided congestion like jugular venous dilatation, bilateral peripheral oedema, congested hepatomegaly, hepatojugular reflux, ascites and symptoms of gut congestion were present in addition to symptoms of LV HF.

Ethics

This retrospective analysis was approved by the Hamburg Ethics Committee (trial number: WF-55/17).

TAVI procedure

All procedures were performed in a hybrid operating room, under analgosedation and local anaesthesia. Details of TAVI procedures have previously been described [11, 12].

Baseline echocardiography

Transthoracic echocardiography was performed regularly during pre-procedure diagnostics. Transoesophageal echocardiography was only performed if additional information such as unclear aortic valve area in transthoracic echocardiography was needed, for sizing of the aortic annulus, left atrial appendage thrombus, for exclusion of echocardiographic signs of endocarditis.

Invasive haemodynamics

Right-heart catheterization with a 7F Swan-Ganz catheter was regularly performed before and after TAVI. Right atrial pressure, systolic, diastolic and mean pulmonary artery pressures, and pulmonary capillary wedge pressure were recorded; cardiac output was determined using the thermodilution method. In addition, left ventricular and aortic pressures were recorded simultaneously before and after TAVI with pigtail catheters in the left ventricle and aorta. Mean and peak-to-peak transaortic pressure gradients were measured, and aortic valve area was calculated using the Gorlin formula.

Outcome definition

Procedural endpoints were defined according to the Valve Academic Research Consortium-2 (VARC-2) for TAVI in native aortic annuli [13]. Follow-up data were obtained at 30 days and 12 months by clinical visit or telephone interview.

Statistics

Continuous variables are described as mean values and standard deviations. Differences between continuous variables were analysed by one-way analysis of variance with post-hoc Bonferroni correction for multiple comparisons. Categorical variables are described with absolute and relative frequencies. Differences between categorical variables were evaluated with the Chi-square test. Patient survival was assessed using the Kaplan–Meier method.

To differentiate effects on survival, a predefined multivariable survival regression model was used including the logistic EuroSCORE, NT-proBNP, and the type of HF. For this model, multiple imputations were performed to take missing baseline data into account.

A two-tailed p value < 0.05 was considered statistically significant, except for multiple ($n = 3$) two-group comparisons, for which $p < 0.0167$ was considered statistically significant. Calculations were performed with the statistical analysis software R (R Foundation for Statistical Computing, 2018, Vienna, Austria) [14].

Results

Patients

The baseline characteristics of the three patient subgroups are shown in Table 1. There were no differences in age (overall mean age was 82 years) and gender distribution (overall 47% men), and all patients but one had a history of hypertension. However, patients with signs of (LV or biventricular) HF had a significantly higher logistic EuroSCORE (26% and 28% vs. 17%), and markedly higher prevalences of pulmonary hypertension (71% and 80% vs. 47%), atrial fibrillation (57% and 59% vs. 33%), and chronic renal failure (41% and 57% vs. 21%) than patients without such signs. Moreover, in patients with biventricular HF in particular, diabetes (51% vs. 27%), chronic obstructive pulmonary disease (COPD; 32% vs. 14%), and peripheral arterial disease (36% vs. 22%) were markedly more prevalent than in patients without HF. Among the latter were the only patients in our study cohort with a prevalence (30%) of New York Heart Association (NYHA) class II dyspnoea; in contrast, NYHA class III dyspnoea was most prevalent (73%) in patients with LV HF, and NYHA class IV dyspnoea was most prevalent (53%) in patients with biventricular HF.

Transcatheter heart valve prostheses

Overall, most of the patients (81%) were treated with the Sapien XT or Sapien 3 heart valve prostheses (Edwards Lifesciences Corp., Irvine, CA), with no difference in valve type distribution between the three groups of patients (Supplemental Fig. 1).

Echocardiographic variables

Pertinent echocardiographic variables pre- and post-TAVI are shown in Table 2. Overall, the severity of AS was reflected in a mean aortic valve area of 0.76 ± 0.17 cm^2 and a mean transaortic pressure gradient of 41 ± 16 mmHg. At baseline, aortic valve area was significantly smaller in patients with biventricular HF (0.71 ± 0.19 cm^2) than in

Table 1 Baseline patient characteristics

	No HF ($n = 176$)	LV HF ($n = 63$)	BiV HF ($n = 122$)	p value
Age, (years)	81 ± 7	83 ± 6	82 ± 7	0.08
Male	81 (46)	31 (49)	57 (47)	0.91
Log. EuroSCORE, (%)	17 ± 11	26 ± 17*	28 ± 17*	< 0.0001
LV ejection fraction, (%)	53 ± 6	48 ± 10*	44 ± 12*	< 0.0001
NT-proBNP, (pg/ml)	931[522, 2080]	4162*[1835, 8988]	5667*[2441, 16,310]	< 0.0001
Hypertension	176 (100)	62 (98)	122 (100)	0.09
Coronary artery disease	107 (61)	43 (68)	70 (57)	0.0006
Pulmonary hypertension†	83 (47)	43 (71)	93 (80)	< 0.0001
Atrial fibrillation	58 (33)	36 (57)	72 (59)	< 0.0001
Chronic renal failure‡	37 (21)	26 (41)	69 (57)	< 0.0001
Diabetes	47 (27)	20 (32)	62 (51)	< 0.0001
COPD	24 (14)	11 (17)	39 (32)	0.0005
Peripheral arterial disease	39 (22)	11 (18)	44 (36)	0.0062
NYHA functional class				< 0.0001
II	52 (30)	0 (0)	0 (0)	
III	124 (70)	46 (73)	58 (48)	
IV	0 (0)	17 (27)	64 (53)	

Values are mean ± SD, median [first quartile; third quartile] or n (%)

BiV biventricular, *COPD* Chronic obstructive pulmonary disease, *LV* left ventricular, *NYHA* New York Heart Association

*$p < 0.0167$ vs. No HF

†Mean pulmonary artery pressure ≥ 25 mmHg

‡Glomerular filtration rate < 60 ml/min/1.73m^2

Table 2 Echocardiographic variables

	Pre-TAVI				Post-TAVI			
	No HF	LV HF	BiV HF	*p* value	No HF	LV HF	BiV HF	*p* value
Aortic valve area, (cm^2)	0.79 ± 0.15	0.74 ± 0.19	0.71 ± 0.19*	0.0003	1.81 ± 0.35	1.83 ± 0.42	1.77 ± 0.39	0.50
Mean transaortic pressure gradient, (mmHg)	41 ± 14	39 ± 19	41 ± 17	0.66	11 ± 4	11 ± 5	10 ± 5	0.27
Maximum transaortic pressure gradient, (mmHg)	65 ± 22	64 ± 29	69 ± 27	0.43	20 ± 7	19 ± 6	19 ± 9	0.38
Moderate/severe MR	48/171 (28)	31/62 (50)*	63/119 (53)*	< 0.0001	45/164 (27)	29/87 (51)*	46/105 (44)*	0.0013
Moderate AR	15 (9)	9 (14)	8 (7)	0.21	1/172 (0.6)	1/57 (1.8)	3/108 (2.8)	0.33
LV ejection fraction, (%)	53 ± 6	48 ± 10*	44 ± 12*	< 0.0001	53 ± 5	49 ± 10*	47 ± 11*	< 0.0001

Values are mean values ± SD or *n* (%)

AR aortic regurgitation, *BiV* biventricular, *LV* left ventricular, *MR* mitral regurgitation

*$p < 0.0167$ vs. No HF

patients without HF (0.79 ± 0.15 cm^2, $p < 0.0001$), whereas no statistical difference between the three patient groups was observed for the transaortic pressure gradient. Concomitant moderate-to-severe mitral regurgitation (MR) was significantly more prevalent in patients with LV HF (50%) and biventricular HF (53%) than in patients without HF (28%). Moderate aortic regurgitation accompanying AS was diagnosed in 9% of the overall population, with no statistical difference in prevalence between the patient groups. Left ventricular ejection fraction was significantly lower in HF patients than in patients without HF and decreased with increasing severity of HF. Post-TAVI, aortic valve area was significantly increased and no longer different between the patient groups, and transaortic pressure gradients were significantly reduced to roughly the same level in all patient groups. Of note, the prevalence of moderate-to-severe MR after TAVI was significantly higher, and left ventricular ejection fraction significantly lower, in HF patients than in patients without HF.

Haemodynamic variables

At baseline, haemodynamically assessed aortic valve area was less in HF patients (overall 0.78 ± 0.24 cm^2) than in patients without HF (0.85 ± 0.23 cm^2) (Supplemental Table 1). Pulmonary artery pressures and pulmonary capillary wedge pressure were higher in HF patients than in patients without HF. Moreover, patients with biventricular HF had significantly higher right atrial (12.4 ± 5.9 vs. 8.8 ± 4.5 mmHg, $p < 0.0167$) and left ventricular end-diastolic pressures (18.1 ± 9.3 vs. 4.5 ± 6.8 mmHg, $p < 0.0167$) than patients without HF. Cardiac output of 4.6 ± 1.3 l/min in the pooled cohort of HF patients and 5.0 ± 1.4 l/min in patients without HF, was significantly lower ($p = 0.0056$).

Post-TAVI, aortic valve area was no longer different between the patient groups (overall mean, 2.65 ± 0.55 cm^2). However, pulmonary artery pressures, pulmonary capillary wedge pressure and right atrial pressure remained increased in HF patients, particularly in patients with biventricular HF, but left ventricular end-diastolic pressure was no longer different between groups (Supplemental Table 1). Cardiac output, although significantly increased compared to baseline in both groups (in HF patients [4.9 ± 1.3 l/min pooled], in patients without HF [5.4 ± 1.5 l/min]).

Acute outcomes

Acute device success according to the VARC-2 definition was achieved in 174 patients (99%) without signs of HF, in all patients with LV HF, and in 118 patients (97%) with biventricular HF (Table 3). Device failures in two patients without HF were due to a mean transaortic pressure gradient ≥ 20 mmHg ($n = 1$, in a valve-in-valve procedure) and moderate paravalvular regurgitation ($n = 1$). Device failures in the four patients with biventricular HF were due to post-procedural death within 72 h ($n = 2$), a mean transaortic pressure gradient ≥ 20 mmHg ($n = 1$), and moderate paravalvular regurgitation ($n = 1$).

The incidence of acute kidney injury in patients with biventricular HF (14.8%) was not significantly higher than in patients with LV HF (7.9%, $p = 0.24$) but significantly higher than in patients without HF (5.7%, $p = 0.0141$).

Complications

Overall, a total of 14 patients (3.9%) sustained a periprocedural stroke and 45 patients (12.5%) required new permanent pacemaker implantation, with no statistically significant differences in incidence noted between the three patient groups (Table 3). Acute kidney injury was observed more often in patients with biventricular HF (14.8%) than in patients with LV HF (7.9%, $p = 0.24$) and patients without HF (5.7%,

Table 3 Outcomes according to VARC-2 criteria

	No HF ($n=176$)	LV HF ($n=63$)	BiV HF ($n=122$)	p value
Device success	174 (99)	63 (100)	118 (97)	0.34
Stroke	9 (5.1)	3 (4.8)	2 (1.6)	0.29
New pacemaker implantation	24 (13.6)	7 (11.1)	14 (11.5)	0.80
Acute kidney injury*	10 (5.7)	5 (7.9)	18 (14.8)	0.0263
Vascular complication				0.67
Major	0 (0)	1 (1.6)	1 (0.8)	
Minor	16 (9.1)	6 (9.5)	11 (9.0)	
Bleeding				0.0119
Life-threatening or disabling	2 (1.1)	2 (3.2)	7 (5.7)	
Major	18 (10.2)	7 (11.1)	25 (20.5)	
Coronary obstruction	0 (0)	0 (0)	0 (0)	–
72-h mortality	0 (0)	0 (0)	2 (1.6)	0.14
30-day mortality	1 (0.6)	0 (0)	18 (14.8)	< 0.0001

Values are *n* (%)

BiV biventricular, *LV* left ventricular

*Stages 2 and 3 according to the Acute Kidney Injury Network classification

$p=0.0141$). Vascular complications occurred at similar incidences (9.1% in patients without HF, 11.1% in patients with LV HF, and 9.8% in patients with biventricular HF). Life-threatening or major bleeding ensued most often in patients with biventricular HF ($n=32$ [26%]), a statistically significant increase compared to patients without HF ($n=20$ [11%], $p=0.0011$); the incidence of bleeding complications observed in patients with LV HF ($n=9$ [14%]) was statistically not different from patients without HF ($p=0.09$).

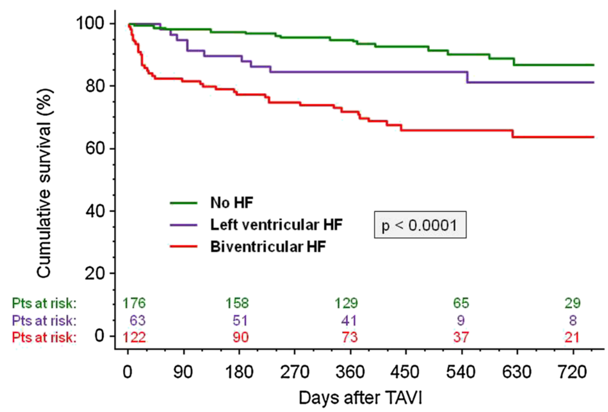
Hospitalization

Post-TAVI, all patients were transferred to the intensive care unit (ICU). They stayed predominantly 1 day on the ICU but HF patients spent significantly more often prolonged periods of time on the ICU. The total duration of hospitalization was significantly longer in HF patients (median 13 and 15 days in patients with LV and biventricular HF, respectively) than in patients without signs of acute HF (median 11 days; Supplemental Fig. 2).

Mortality

By 72 h after TAVI, two patients with biventricular HF (1.6%) but no patient in the other two groups had died; the causes of death were ventricular rupture during implantation of a leadless pacemaker and fatal bleeding. Thirty-day mortality was significantly higher in patients with biventricular HF than in patients without HF and patients with LV HF, 14.8% ($n=18$), 0.6% ($n=1$) and 0%, respectively (Table 3).

The patients were followed for a median of 427 (interquartile range, 315–643) days. Patient survival upto 2 years is shown in Fig. 1, with a steep initial decline of the survival curve apparent in patients with biventricular HF. This decline represents 21 deaths (54%) occurring within 42 days after TAVI, out of a total of 39 deaths occurring in this patient group throughout follow-up. Eleven of the 21 "early" deaths (52%) occurred while the patients were on the ICU, six patients died post-ICU on the regular hospital ward, and another four patients died after hospital discharge. Accordingly, overall survival was significantly worse in patients with biventricular HF than in patients



		1 year		2 years	
Cum. survival w/ 95% CI (%)	No HF	94	91-98	87	80-94
	Left ventricular HF	85	75-94	81	71-92
	Biventricular HF	72	64-80	64	54-74

Fig. 1 Cumulative survival after TAVI in patients without HF (green line), patients with left ventricular HF (purple line), and patients with biventricular HF (red line). The log-rank p value < 0.0001 indicates a statistically significant difference between the survival curves, driven by the markedly impaired survival of patients with biventricular HF. Estimates of 1- and 2-year survival with 95% confidence interval (CI) are given in the box below the graph. *HF* Heart failure

with LV HF (log-rank $p = 0.0203$) and patients without HF (log-rank $p < 0.0001$). Corresponding Kaplan–Meier estimates of 1-year survival were 72.1% [95% confidence interval, 64.0–80.2%], 84.5% [75.2–93.8%], and 94.3% [90.7–97.9%], respectively. At 2 years, estimated survival rates were 63.9% [54.3–73.6%], 81.4% [70.6–92.2%], and 86.9% [80.1–93.8%], respectively.

A total of 65 deaths occurred, 49 in the HF group and 16 in the No-HF group. In a survival regression model, which included the type of HF (no HF, left ventricular or biventricular HF), the logistic EuroSCORE, and N-terminal pro-brain natriuretic peptide (NT-proBNP), the logistic EuroSCORE was only marginally associated with survival (HR 1.47 [30% vs. 10%]; 95% confidence interval [CI] 0.95, 2.29; $p = 0.09$), whereas NT-proBNP showed no significant effect on survival (HR 1.31 [5000 pg/ml vs. 1000 pg/ml], 95% CI 0.86, 2.00; $p = 0.21$). Biventricular HF, as opposed to no HF, was strongly associated with survival (HR 2.62, 95% CI 1.27, 5.38; $p = 0.0089$). In contrast, left ventricular HF was not associated with an increased risk of death compared to no HF (HR 1.26, 95% CI 0.53, 2.99; $p = 0.61$) (Table 4).

Discussion

Main findings

In this study of 361 patients undergoing routine transfemoral TAVI at our institution, we observed that patients presenting with biventricular HF at baseline had significantly poorer survival (64% at 2 years) than patients without acute signs of HF at the time of presentation (87% at 2 years); this difference in long-term survival was primarily driven by an excess of death occurring within 42 days after TAVI in patients with biventricular HF. Interestingly, patients with isolated LV HF at baseline did not experience a statistically worse clinical outcome than that observed in patients without HF. Biventricular HF was a strong independent predictor of survival.

Table 4 Multivariable regression model of mortality

	Hazard ratio	95% CI	*p* value
Logistic EuroSCORE	1.47*	0.95–2.29	0.09
NT-proBNP	1.31†	0.86–2.00	0.21
Left ventricular HF	1.26	0.53–2.99	0.61
Biventricular HF	2.61	1.27–5.38	0.0089

HF heart failure, *CI* confidence interval

*30% vs. 10%

†5000 pg/ml vs. 1000 pg/ml

Biventricular HF as a predictor of mortality

In patients with AS, the severity of HF is assessed during routine physical examination. In patients clinically judged as having biventricular HF, echocardiography and invasive measurements confirm these advanced signs of decompensated AS, including higher cardiac and pulmonary pressures. In this study, baseline echocardiography in HF patients revealed lower aortic valve area and left ventricular ejection fraction and significantly higher percentages of moderate/severe MR than in patients without HF; invasive haemodynamic measurements confirmed signs of decompensated AS in HF patients. It may be hypothesized that these patients, as opposed to patients without acute signs of HF, have possibly suffered for a longer period of time from their AS or have perhaps undergone a more rapid progression of HF. Unfortunately, it appears not to be possible to differentiate the underlying mechanism of HF at the time of admission, which may be due to the AS but may also be a consequence of other causes of HF such as prior myocardial infarction, atrial fibrillation, hypertensive cardiomyopathy or a combination. Nevertheless, clinical assessment of HF seems to be highly predictive of the mortality outcome.

Progression of HF

In this study, a difference in survival between LV and biventricular HF patients, favouring the former, was observed. Therefore, it appears necessary to treat patient diagnosed with severe AS and acute signs of LV HF without delay to possibly avoid progression to biventricular HF. There is presently a paucity of data on the time lag between the diagnosis of severe AS and definite therapy, and the presence or absence and type of HF at the time of diagnosis is usually not factored into the decision-making process on the timing of therapy [15, 16]. Malaisrie et al. [15] observed a prolonged waiting time for SAVR associated with increased mortality. The 2016 heart failure guidelines recommend for patients with severe AS and HF the treatment of the AS in addition to optimal medical treatment for HF [1]. Also, the most recent guidelines for valvular heart disease recommend an early intervention in symptomatic patients without any specific comments regarding the clinical status of HF [8].

In patients with severe AS and acute signs of biventricular HF it can only be hypothesized to perform TAVI without any delay, but progression of HF in this stage may result in a cardiogenic shock. Data on cardiogenic shock and TAVI show high procedural mortality (≤ 72 h) (> 10%) and high 30-day mortality rates (33%) [17], but data in this study show no difference in device success rate and 72-h mortality between patients with biventricular HF compared with patients without clinical signs of HF.

Hospitalization

Our data also show prolonged hospitalization and a prolonged stay on the ICU in HF patients. Specific problems with extended hospitalization in elderly patients even without signs of HF are decreases in muscle strength and muscle mass, which are associated with higher mortality rates and impaired functional status [18]. Factors like these might contribute to higher mortality rates in patients with acute HF. Furthermore, mortality rates on the ICU and post-ICU mortality rates are known to be higher in elderly than in younger patients [19]. Since TAVI patients are elderly in the majority of cases and are mostly high-risk patients due to frailty and co-morbidities, each additional day on the ICU increases the risk of mortality and needs to be avoided.

Mortality

Early mortality is increased in patients with clinical biventricular HF, which drove the overall difference in survival between the three patient groups. Of note, overall survival was not statistically different between patients with LV HF and patients without HF, but it was significantly poorer than in either of these patient groups in patients with biventricular HF. In this study, a clinical diagnosis of biventricular HF was predictive of mortality, increasing the likelihood of death in comparison to no HF by a factor of 2.62.

HF is the leading cause of hospitalization in the elderly and associated with increased mortality [20]. Patients treated in the cardiology departments are frail in about 67%, with several co-morbidities [20]. It is not surprising that TAVI for severe AS in these patients is associated with increased mortality. Also, COPD by itself is associated with increased mortality, particularly in hospitalized patients [21]. The combination of COPD and HF, particularly in the presence of reduced left ventricular ejection fraction, is associated with a poor prognosis [1]. Also, HF and chronic impaired renal function frequently coexist and interact to worsen the patients' prognosis [22, 23]. Previous TAVI studies have described a reduced LVEF in patients with AS undergoing TAVI as a risk factor for increased mortality [24–27]. In this study LVEF was significantly lower in HF patients than in patients without HF and decreased with increasing severity of HF. Independent of the fact that LVEF after TAVI was significantly lower in HF patients, the logistic EuroSCORE was only marginally associated with survival, whereas biventricular HF was associated with a statistically significant 2.6-fold increase in the likelihood of death. HF laboratory values like NT-proBNP do not predict mortality in our patient cohort. This underlines our finding that clinical classification of heart failure seems to be very important.

Other clinical risk factors such as reduced mobility, residual impairment of functional capacity or lower body mass index, which are common in TAVI patients and associated with increased mortality, were not collected for this study [28–30].

Clinical relevance

For patients with AS seen in outpatient care our study helps to provide a strategy for further management. Patients without signs of HF can be planned electively after preliminary assessment of all examinations. Patients with signs of LV HF should be sent within the next days, while patients with biventricular HF should be sent directly into a clinic with an existing heart team with experience in the surgical and transcatheter treatment of AS, to avoid progression of HF. Without any intervention, natural progression in patients with severe AS and HF moves on from LV HF to biventricular HF to cardiogenic shock. Both biventricular HF and cardiogenic shock are known to increase mortality, therefore these stages need to be avoided.

Limitations

This is a single-centre study of a real-world TAVI population. The data represent a retrospective analysis without randomization. Also, there were no matched patient groups. The retrospective character of this analysis does not take into account that the results may have been affected by unmeasured confounding variables. Ideally, the impact of cardiac status at the time of admission on TAVI outcomes should be assessed by a standardized protocol in a randomized trial. Unfortunately, it appears not to be possible to differentiate the underlying mechanism of HF at the time of admission, which may be due to the AS but may also be a consequence of other causes of HF such as prior myocardial infarction, atrial fibrillation, hypertensive cardiomyopathy or a combination. Medical treatment, especially HF medication, was not recorded. HF risk scores were not collected for this study.

Conclusions

Biventricular HF is a strong predictor of mortality following TAVI for severe AS. As patients with LV HF fared no worse than patients without HF, AS in patients with LV HF should be treated without delay to avoid progression to biventricular HF. AS patients with biventricular HF should be monitored closely after TAVI to possibly prevent early death.

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Compliance with ethical standards

Conflict of interest Tobias Schmidt has received lecture honoraria from Medtronic, as well as travel expenses from Edwards LifeSciences, Medtronic, and Boston Scientific. Christian Frerker has received lecture honoraria and travel expenses from Medtronic, Edwards Lifesciences, and Abbott Vascular. Karl-Heinz Kuck has received consultation fees from Medtronic, Boston Scientific, Biosense Webster, Edwards LifeSciences, and Abbott Vascular. The other authors report no conflicts of interest.

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