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CLINICAL RESEARCH

Evolution of chronic kidney disease after surgical aortic valve replacement or transcatheter aortic valve implantation

Évolution des patients insuffisants rénaux chroniques après un remplacement valvulaire aortique chirurgical ou percutané

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Summary
Background. — Immediate improvement in kidney function has been reported after surgical aortic valve replacement or transcatheter aortic valve implantation. Long-term data, however, are not available.

Glomerular filtration rate;
Kidney function;
Transcatheter aortic valve implantation;
Surgical aortic valve replacement

Aim. — To assess the evolution of kidney function in chronic kidney disease stage 3b—5, 1 year after surgical aortic valve replacement or transcatheter aortic valve implantation.

Methods. — All patients with chronic kidney disease stage 3b—5 undergoing surgical aortic valve replacement or transcatheter aortic valve implantation for aortic stenosis in a single centre were included. Kidney function was assessed 1 year postprocedure. Improvement or deterioration in estimated glomerular filtration rate was defined by an increase or decrease of 5 mL/min/1.73 m^2, respectively.

Results. — Overall, 127 procedures were analysed (54 surgical aortic valve replacements and 73 transcatheter aortic valve implantations). Kidney function improved in 51% of patients at 1 year (45% of the surgical aortic valve replacement group versus 57% of the transcatheter aortic valve implantation group; $P = 0.21$), and deteriorated in only 14% of patients at 1 year (18% of the surgical aortic valve replacement group versus 10% of the transcatheter aortic valve implantation group; $P = 0.22$). Almost a quarter of patients (23%) had an improvement in estimated glomerular filtration rate of > 15 mL/min/1.73 m^2, and this was consistent at later follow-up. Few patients went onto chronic dialysis at 1 year (three after surgical aortic valve replacement and one after transcatheter aortic valve implantation). Acute kidney injury was an independent prognostic factor for long-term deterioration in kidney function (odds ratio 2.1, 95% confidence interval 1.4—3.6; $P = 0.006$).

Conclusion. — Aortic valve replacement, whether by surgical aortic valve replacement or transcatheter aortic valve implantation, improved estimated glomerular filtration rate at 1 year in more than half of patients with chronic kidney disease stage 3b—5.

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MOTS CLÉS
Insuffisance rénale chronique ;
Débit de filtration glomérulaire ;
Fonction rénale ;
Remplacement valvulaire aortique chirurgical ;
Remplacement valvulaire aortique percutané

Résumé

Contexte. — Une amélioration de la fonction rénale immédiatement après un remplacement valvulaire aortique chirurgical (RVAC) ou un remplacement aortique valvulaire percutané (TAVI) a été observée. En revanche, des données à plus long terme ne sont pas décrites.

Objectif. — Évaluer l'impact d'un RVAC ou d'un TAVI à un an sur la fonction rénale d'insuffisants rénaux chroniques (IRC) de stade 3b à 5.

Méthodes. — Tous les patients IRC de stade 3b à 5 bénéficiant d'un RVAC ou d'un TAVI dans un centre étaient inclus. La fonction rénale était évaluée durant l'année suivant la procédure. Le critère de jugement principal était une amélioration ou une dégradation à un an du débit de filtration glomérulaire estimé (DFGe) de 5 mL/min/1,73 m^2.

Résultats. — 127 patients ont été inclus (54 RVAC et 73 TAVI). Le DFGe s'est amélioré à un an parmi 51 % des patients (RVAC 45 % vs TAVI 57 % ; $p = 0,21$) et détérioré chez seulement 14 % (RVAC 18 % vs TAVI 10 % ; $p = 0,22$). Presque un quart des patients ont présenté une amélioration de plus de 15 mL/min/1,73 m^2. Seulement 4 patients ont nécessité une mise en dialyse chronique à un an (3 vs 1 patient après RVAC et TAVI, respectivement). L'insuffisance rénale aiguë était un facteur pronostique indépendant d'une détérioration de la fonction rénale à un an (OR 2,1, IC95 % 1,4—3,6 ; $p = 0,006$).

Conclusion. — Un RVAC ou un TAVI a permis une amélioration significative de la fonction rénale à un an chez plus de la moitié des patients IRC stade 3b à 5.

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Background

Chronic kidney disease (CKD) with an estimated glomerular filtration rate (eGFR) of < 60 mL/min/1.73 m^2 [1] is found in 30—50% of patients undergoing surgical aortic valve replacement (SAVR) [2] or transcatheter aortic valve implantation (TAVI) [3,4] for symptomatic aortic stenosis. Risk factors for calcific aortic stenosis and CKD are the same; consequently, both diseases may occur independently [5]. Aortic stenosis may also contribute to the onset of chronic kidney failure by reducing blood flow, leading to chronic renal hypoperfusion [6], or by inducing increased central venous pressure secondary to postcapillary pulmonary arterial hypertension, leading to chronic cardiorenal syndrome [7,8].

Several studies have reported high rates of acute kidney injury (AKI) within 48 hours after SAVR or TAVI in patients

with CKD stage 3—5 [9,10]. Kidney function, however, was also reported to improve in the week after aortic valve replacement, linked to a survival increase [11,12]. However, data on long-term kidney function progression in this population are scarce, although the haemodynamic changes induced by aortic valve replacement could have a long-term impact on kidney function, by suppressing renin-angiotensin-aldosterone system hyperactivation and the resulting chronic inflammation.

The objective of the present study was to assess the evolution of kidney function 1 year after aortic valve replacement (SAVR or TAVI) for aortic stenosis in patients with CKD stage 3b—5 (i.e. eGFR < 45 mL/min/1.73 m^2).

Methods

Population

Between January 2012 and June 2015, 620 patients with tight symptomatic aortic stenosis underwent isolated SAVR, with coronary bypass if needed, in the University Hospital of Clermont-Ferrand (France). In parallel, between January 2013 and June 2015, 370 TAVI procedures were performed in the cardiology department of the same hospital. According to current guidelines [13], indications for TAVI were determined after a multidisciplinary ''heart team'' discussion, for patients with high surgical risk or contraindications for conventional surgery. Percutaneous coronary intervention was, if necessary, performed ahead of TAVI during systematic coronarography.

The inclusion criterion for both cohorts was preoperative eGFR < 45 mL/min/1.73 m^2, calculated using the Modification of Diet in Renal Disease Study Group equation [14]. Patients were categorized into three CKD stages according to maximal eGFR within 48 hours before the procedure [15]: stage 3b, moderate (GFR 30—44 mL/min/1.73 m^2); stage 4, severe (GFR 15—29 mL/min/1.73 m^2); and stage 5, preterminal (GFR < 15 mL/min/1.73 m^2). To reduce patient characteristics in both groups, we excluded patients aged < 70 years from our analysis. Other non-inclusion criteria were chronic dialysis and an eGFR $\geq$ 45 mL/min/1.73 m^2 if a blood test was available within 3 months before the procedure.

Data collection

Epidemiological data, cardiovascular and non-cardiovascular history, echocardiographic data, procedural technique and type of implant were collected from the national EPICARD registry for the SAVR group, and from the French national TAVI registry for the TAVI group. Missing data were collected retrospectively from medical files.

Serum creatinine concentration and eGFR calculated using the Modification of Diet in Renal Disease Study Group equation [14] were recorded within 48 hours before the procedure, during the hospital stay, at discharge, during the first month postdischarge, at 1—12 months postprocedure, at 1 year postprocedure and at the last available assay during follow-up. AKI was defined, according to the modified RIFLE classification [16], as an absolute increase in serum creatinine concentration of $\geq$ 0.3 mg/dL ($\geq$ 26.5 μmol/L) within

48 hours postprocedure or a relative increase of > 50% within 7 days from baseline. Hospital length of stay, in-hospital mortality and 1-year mortality from any cause were also recorded. All data were registered with the French national data protection commission (CNIL: *Commission nationale de l'informatique et des libertés*) after approval from the institutional review board (*Comité de protection des personnes du sud-est VI*).

Primary endpoint

Improvement in kidney function was defined as an increase in eGFR of > 5 mL/min/1.73 m^2 from baseline, 1 year after SAVR or TAVI; deterioration was defined as a decrease of > 5 mL/min/1.73 m^2. Initiation of chronic dialysis during follow-up was counted as deterioration in kidney function, with eGFR scored 0 mL/min/1.73 m^2.

Statistical analyses

All analyses were performed using Stata software, version 13 (StataCorp, College Station, TX, USA) for a two-sided type I error of α = 5%. Patient characteristics are expressed as mean $\pm$ standard deviation or median [interquartile range] for continuous data (assumption of normality assessed by using the Shapiro-Wilk test), and as numbers and associated percentages for categorical variables. Quantitative variables were compared between TAVI and SAVR groups (and between eGFR improvement and eGFR no change/worsening groups) using Student's t test, or the non-parametric Mann-Whitney test if t test assumptions were not met (normality and homoscedasticity analysed using the Fisher-Snedecor test). For categorical variables, comparisons between groups were made with the χ^2 test or Fisher's exact test. To determine factors associated with eGFR improvement, a multivariable generalized linear model (logistic for dichotomous endpoint) was performed using the stepwise approach on covariates fixed according to univariate results and clinical relevance. Particular attention was paid to the study of multicollinearity and interactions between covariates. Results were expressed as odds ratios (ORs) and 95% confidence intervals (CIs). The same statistical methods were used to compare kidney progression and procedural complications between TAVI and SAVR groups. Concerning hospital length of stay, a multiple linear regression was proposed, after applying a logarithmic transformation on the endpoint. Finally, hospital mortality was studied as censored data. Comparisons were analysed using the log-rank test and Cox proportional-hazards regression. The proportional-hazard hypothesis was studied using Schoenfeld's test and plotting residuals, and the interactions between possible prognostic factors were also tested. Results are expressed as hazard ratios (HRs) and 95% CIs.

Results

Population characteristics

Of the 620 patients who underwent SAVR, 407 were aged > 70 years, among whom 60 had impaired baseline renal function, with eGFR < 45 mL/min/1.73 m^2 within 48 hours before the

Table 1 Population characteristics.

	TAVI ($n = 73$)	SAVR ($n = 54$)	P
Age (years)	83.5 ± 5.6	77.3 ± 4.1	< 0.001
Male sex	55	56	0.93
EuroSCORE II (%)	8.6 ± 5.9	4.4 ± 3.2	< 0.001
Cardiovascular risk factors			
BMI (kg/m^2)	26.9 ± 4.8	26.9 ± 3.8	0.99
Diabetes mellitus	34	33	0.91
Hypertension	81	85	0.52
Dyslipidaemia	53	54	0.97
Medical history			
Peripheral arterial disease	30	15	0.04
Chronic respiratory insufficiency	25	11	0.05
Atrial fibrillation	62	30	< 0.001
Anticoagulant therapy	58	30	0.002
Haemoglobin (g/dL)	11.8 ± 1.6	12.8 ± 1.6	0.002
Neoplasia	16	2	0.007
Stroke	8	6	0.6
Ischaemic heart disease	38	20	0.03
Coronary artery bypass	10	2	0.1
NYHA class 3/4	54	52	0.63
Transthoracic echocardiography			
LVEF (%)	58.4 ± 15	57.4 ± 11	0.34
Mean transvalvular gradient (mmHg)	42.9 ± 17	42.0 ± 15	0.38
Aortic valve surface (cm^2)	0.70 ± 0.19	0.74 ± 0.23	0.85
Moderate PAP (systolic PAP 41—60 mmHg)	47	24	< 0.001
Severe PAP (systolic PAP > 60 mmHg)	27	4	< 0.001
CKD characteristics			
Baseline eGFR (mL/min/1.73 m^2)	34.3 ± 8	37.8 ± 7	0.01
CKD stage			0.19
3b	74	87	
4—5	26	13	
Identified nephropathy (biopsy)	20	15	0.46
Type of nephropathy			
Nephroangiosclerosis	8	2	
Diabetic nephropathy	6	6	
Kidney transplantation	1	0	
Membranoproliferative glomerulonephritis	0	1	

Data are expressed as mean ± standard deviation or %. BMI: body mass index; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; EuroSCORE II: European System for Cardiac Operative Risk Evaluation II; NYHA: New York Heart Association; LVEF: left ventricular ejection fraction; PAP: pulmonary arterial hypertension; SAVR: surgical aortic valve replacement; TAVI: transaortic valve implantation.

procedure. Six patients were excluded: three for chronic dialysis and three for eGFR ≥ 45 mL/min/1.73 m^2 on a blood test during the 3 months preceding surgery.

Of 370 patients undergoing TAVI, 85 had a baseline eGFR < 45 mL/min/1.73 m^2; all were aged ≥ 70 years. Twelve patients were excluded: eight for chronic dialysis and four for eGFR ≥ 45 mL/min/1.73 m^2 during the 3 months preceding the procedure. As expected, Table 1 shows significantly different epidemiological profiles between the two groups, with more co-morbidity in the TAVI cohort. Overall, 80% of the study population were CKD stage 3b, and 20% were stage 4—5. Nephropathy was demonstrated by biopsy in only 18% of patients. Type of nephropathy and procedural data for the 127 patients are shown in Tables 1 and 2.

Evolution of eGFR at 1 year (Figs. 1 and 2)

Five patients in the SAVR group and six in the TAVI group died before 12 months after aortic valve replacement, without a serum creatinine measurement at 12 months, and were excluded from the 1-year eGFR analysis.

In the SAVR group, 22 patients (45%) showed improved kidney function, including 11 (50%) with an eGFR increase of > 15 mL/min/1.73 m^2; 18 patients (37%) had no change and nine patients (18%) showed deterioration. Three patients went onto chronic haemodialysis therapy during the year after SAVR.

In the TAVI group, 60 patients (90%) experienced no change ($n = 22$; 33%) or an improvement ($n = 38$; 57%) in

Table 2 Procedural data.

	TAVI(n = 73)	SAVR(n = 54)
Coronarography		
Coronary angiography	100	100
Coronary angioplasty	22	—
Anaesthesia		
Local	49	—
General	51	100
TAVI approach		
Transfemoral	78	—
Transapical	7	—
Subclavian	14	—
Transaortic	1	—
Contrast agent volume (mL)	156 ± 55	—
Surgery		
Sternotomy	—	100
Associated coronary bypass	—	35
Extracorporeal circulation time (minutes)	—	103 ± 47
Cross-clamp time (minutes)	—	82 ± 39

Data are expressed as % or mean $\pm$ standard deviation. SAVR: surgical aortic valve replacement; TAVI: transaortic valve implantation.
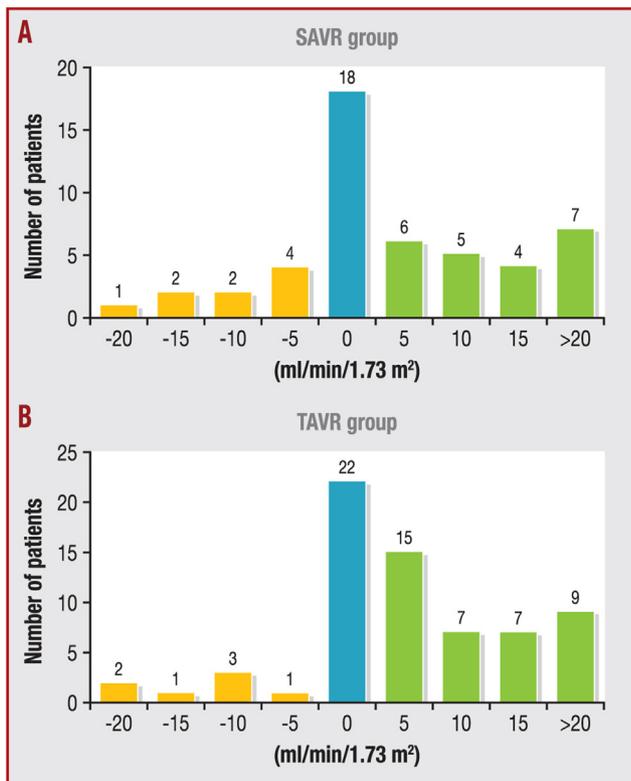

Figure 1. Evolution of estimated glomerular filtration rate (eGFR) 1 year after the procedure, compared with baseline eGFR. SAVR: surgical aortic valve replacement; TAVI: transaortic valve implantation.

Figure 2. Evolution of estimated glomerular filtration rate (eGFR) from the preprocedural period to the last follow-up for each patient according to chronic kidney disease stages 3b and 4—5 in the surgical aortic valve replacement (SAVR) group (n = 49) and the transaortic valve implantation (TAVI) group (n = 57).

kidney function, only one (CKD stage 5) required initiation of chronic dialysis, at 5 months.

In our overall population, no statistically significant difference in kidney function improvement ($P = 0.21$) or deterioration ($P = 0.22$) at 1 year was found between the TAVI group and the SAVR group. Multivariable analysis confirmed this result (kidney function improvement for TAVI versus SAVR: OR 2.07, 95% CI 0.38—11.19; $P = 0.40$).

Postprocedural data (Table 3)

During their hospital stay, 17 patients (31%) undergoing SAVR and 17 patients (23%) undergoing TAVI had acute stage 1 kidney failure; three patients (6%) in the SAVR group and three (4%) in the TAVI group had acute stage 3 kidney failure requiring dialysis as a result of haemorrhagic shock, except

their kidney function; almost a quarter of patients (n = 16; 24%) had an eGFR improvement of > 15 mL/min/1.73 m². Fig. 2 displays a linear increase in each CKD stage. Of the seven patients (10% of the TAVI group) with deterioration in
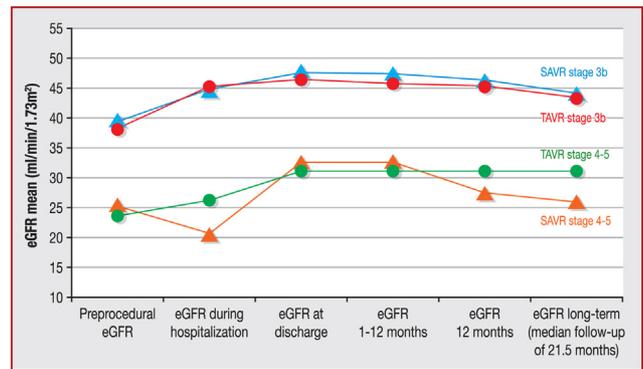
Table 3 Postprocedural data.

	TAVI (n = 73)	SAVR (n = 54)	P[a]
Postprocedural complication			
Blood transfusion	8	44	< 0.01
Aortic regurgitation ≥ grade 2	16	4	0.02
Postprocedural stroke	1	0	1.0
Pacemaker	30	2	< 0.01
AKI	27	37	0.25
AKI requiring haemodialysis	4	6	0.67
In-hospital mortality	3	6	0.65
Length of stay (days)	10 [7—13]	12 [10—14]	0.01
1-year mortality			
Overall population: CKD stage 3b—5	12	11	0.8
CKD stage 3b	9	6	0.72
CKD stage 4—5	21	43	0.34
1-year kidney function evolution	(n = 67)	(n = 49)	
eGFR worsening > 5 mL/min/1.73 m^2			
Overall population: CKD stage 3b—5	10	18	0.22
CKD stage 3b	10	18	0.26
CKD stage 4—5	12.5	25	0.51
eGFR improved > 5 mL/min/1.73 m^2			
Overall population: CKD stage 3b—5	57	45	0.21
CKD stage 3b	57	44	0.23
CKD stage 4—5	56	50	0.83
Beginning of chronic dialysis	1	6	0.31

Data are expressed as % or median [interquartile range]. AKI: acute kidney injury; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; EuroSCORE II: European System for Cardiac Operative Risk Evaluation II; SAVR: surgical aortic valve replacement; TAVI: transaortic valve implantation.

[a] P-value resulting from univariate analysis. Results with P < 0.05 remained statistically significant in the multivariable analysis, adjusting for age, EuroSCORE II, CKD stage, diabetes, pulmonary artery hypertension, left ventricular ejection fraction, AKI (except for length of stay, with P = 0.68 in the multivariable analysis).

one case in the TAVI group, which was attributed to iodized contrast agent.

The 1-year death rate from any cause was similar after TAVI or SAVR in the univariate analysis (TAVI 12% vs. SAVR 11%; HR 1.34, 95% CI 0.45—4.01; P = 0.60) as well as in the multivariable analysis, adjusted for age, European System for Cardiac Operative Risk Evaluation II (EuroSCORE II), implantation year, neoplasia yes/no and anticoagulant drug yes/no (TAVI versus SAVR: HR 0.78, 95% CI 0.15—3.96; P = 0.77). Regarding advanced CKD (stage 4—5), the death rate was twice as high 1 year after SAVR (SAVR 43% vs. TAVI 21%: HR 1.8, 95% CI 0.04—75.9; P = 0.75) without statistical significance, while the EuroSCORE II was much lower (SAVR 5.9 ± 1.9 vs. TAVI 8.2 ± 4.8; P = 0.45).

Predictors of kidney failure reversibility or worsening (Table 4)

In the univariate analysis, AKI and diabetes emerged as the only risk factors for worsening kidney function at 1 year (P < 0.001 and P = 0.01, respectively). Factors identified as being associated with eGFR improvement were absence of AKI (P < 0.001), absence of diabetes (P = 0.026), absence of peripheral arterial disease (P = 0.047) and absence of chronic pulmonary disease (P = 0.038). We also found significant trends for absence of ischaemic cardiopathy

(P = 0.061) and presence of pulmonary arterial hypertension (P = 0.055).

In the multivariable analysis, adjusting for age, EuroSCORE II, CKD stage, blood pressure, diabetes, pulmonary artery hypertension, left ventricular ejection fraction, AKI and type of procedure (SAVR or TAVI), only AKI emerged as an independent predictor of poor prognosis for kidney function (OR 2.1, 95% CI 1.4—3.6; P = 0.006). Conversely, absence of AKI and improvement in kidney function during hospital stay and at discharge were predictive of good improvement in kidney function at 1 year (P < 0.001 for each).

Discussion

This first study of long-term kidney function after SAVR or TAVI among a severe CKD population (stage 3b—5) enlightened us about two major points:

- there is an improvement in eGFR in half of this population, 1 year after both SAVR and TAVI;
- AKI is a strong predictor of worsening kidney function longer term.

This present study found an improvement in eGFR of > 5 mL/min/1.73 m^2 1 year after aortic valve replacement in 51% of patients with CKD stage 3b—5 (SAVR 45%;

Table 4 Predictors of improved kidney function 1 year after surgical aortic valve replacement or transaortic valve implantation.

	eGFR		
	Improved (n = 60)	No change or worsening (n = 56)	P^a
Preoperative characteristics			
Mean age (years)	81 ± 6.3	81 ± 5.7	0.27
Male sex	50	58	0.34
BMI (kg/m^2)	26.3 ± 4.6	27.5 ± 4.1	0.15
EuroSCORE II (%)	6.6 ± 5.9	6.5 ± 4.5	0.75
NYHA class 3/4	61.5	38.5	0.19
Diabetes mellitus	36	64	0.03
Hypertension	51	49	0.75
Dyslipidaemia	49	51	0.56
Peripheral arterial disease	35	65	0.05
Ischaemic heart disease	38	62	0.06
Stroke	50	50	0.92
Chronic pulmonary disease	68	32	0.04
Anticoagulant therapy	60	40	0.13
Haemoglobin (g/dL)	12.3 ± 1.7	12.2 ± 1.7	0.74
History of neoplasia	36	64	0.35
LVEF (%)	59.6 ± 15.5	56.6 ± 11.8	0.26
Pulmonary arterial hypertension	61	39	0.06
CKD stage			0.28
3b	51	49	
4—5	61	39	
Type of procedure			
SAVR	45	55	0.21
TAVI	57	43	
TAVI with transfemoral approach		42	0.18
SAVR + non-transfemoral TAVI		56	
Postprocedural data			
Aortic regurgitation > grade 2	73	27	0.14
Blood transfusion	39	61	0.18
AKI	24	76	< 0.001
Mean eGFR (mL/min/1.73 m^2)			
Preprocedural	36.4 ± 7.0	36.3 ± 7.8	0.86
In-hospital	47.1 ± 16.9	35.9 ± 14.5	< 0.001
Discharge	50.8 ± 15.4	37.5 ± 10.1	< 0.001
1—12 months	52 ± 14.7	34.9 ± 8.9	< 0.001

Data are expressed as mean ± standard deviation or %. AKI: acute kidney injury; BMI: body mass index; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; EuroSCORE II: European System for Cardiac Operative Risk Evaluation II; NYHA: New York Heart Association; LVEF: left ventricular ejection fraction; SAVR: surgical aortic valve replacement; TAVI: transaortic valve implantation.
^{a} P-value resulting from univariate analysis. In the multivariable analysis, adjusting for age, EuroSCORE II, CKD stage, blood pressure, diabetes, pulmonary artery hypertension, LVEF, AKI and type of procedure (SAVR or TAVI), only absence of AKI and improved eGFR during in-hospital stay, at discharge and 1—12 months postprocedure were statistically significant for improved eGFR at 1 year (P < 0.001 for each).

TAVI 57%), and an improvement of > 15 mL/min/1.73 m^2 in almost a quarter (23%) of this population. A previous study has already reported an eGFR improvement after SAVR: two-thirds of patients with CKD stage 3—4 had an increased eGFR 1 week after SAVR. However, this evaluation was shorter term and with a less-relevant threshold ($\geq$ 1 mL/min/1.73 m^2), which could explain the lower rate in our study [11]. To our knowledge, no other study has analysed kidney function long after SAVR in patients with CKD. After TAVI, kidney function improvement was slightly higher in our cohort

than was recently reported in a larger multicentre study in North America, which found an improvement (> 10% baseline eGFR) 30 days after TAVI in 42% of patients with a baseline eGFR $\leq$ 60 mL/min/1.73 m^2 [17]. This may be the consequence of selection bias, as they could not include consecutive patients; half of the population with baseline eGFR $\leq$ 60 mL/min/1.73 m^2 did not have an eGFR measurement 30 days after, and were excluded. Those patients may have had less care because of an improvement in eGFR immediately after TAVI. Moreover, we investigated patients

with more severe CKD, excluding CKD stage 3a and including CKD stages 4—5, which is the population that has recently been shown to derive the most benefit to kidney function [18,19]. At last, relief of chronic cardiorenal syndrome with chronic systemic inflammation may last long beyond 1 month after aortic valve replacement. As with our study, two other studies have shown kidney function improvement beyond 1 month after TAVI, but did not report the number of patients with CKD stage 3—5 or the number lost to follow-up [19,20].

Our study did not find a statistically significant improvement in kidney function 1 year after TAVI compared with SAVR. This is consistent with another small retrospective study that found no statistical difference 48 hours after both procedures [21]. However, this may be the consequence of both studies being underpowered to detect this difference.

Assessment of predictors of long-term eGFR improvement or worsening revealed that AKI was a strong predictor of eGFR worsening the year after SAVR or TAVI. Conversely, improvement in kidney function at discharge was predictive of good improvement in kidney function at 1 year. These results are supported by Thongprayoon et al., who found greater eGFR improvement 6 months after TAVI in patients with severe CKD without AKI than in those with AKI after TAVI [19]. Other predictors of long-term eGFR improvement (significant or with significant trends) in our univariate analysis, but not in the multivariable analysis, were absence of diabetes, peripheral arterial disease, ischaemic cardiomyopathy, chronic pulmonary disease and female sex. This is explained by a statistically significant connection between these characteristics and AKI, which is well established [22]. Those patients with cardiovascular factors are probably involved in more serious chronic organic kidney lesions that are likely to sustain AKI and be irreversible, despite haemodynamic normalization. Finally, it is worth noting a significant trend towards improved kidney function in case of pulmonary artery hypertension (i.e. $P = 0.055$ in univariate analysis and $P = 0.052$ in multivariable analysis); the reduction in postcapillary pulmonary artery hypertension after aortic valve replacement may account for this finding, supporting the hypothesis that excessive venous pressure impairs kidney function [7,8].

Few data exist comparing outcomes after SAVR versus TAVI in patients with CKD stage 3b—5. Indeed, this population is under-represented in major randomized trials comparing SAVR with TAVI, with an overall rate of < 10% [23—25]. Here, we found that 1-year mortality was not statistically different in the two groups. D'Errigo et al. made the same observation, using a propensity score to match 170 SAVR procedures and 170 TAVI procedures in patients with CKD stage 3b—5 [26], and reported no statistically significant difference in 2-year mortality; However they did not investigate the differences regarding advanced CKD (stage 4—5), which has a higher risk complication. A single investigation assessed survival according to CKD stage between 1336 SAVR and 321 TAVI procedures [27]; the investigators found similar in-hospital mortality rates in CKD stage 3, but a better prognosis for TAVI in CKD stage 4—5, with an in-hospital mortality rate 2-fold lower after TAVI than after SAVR. This finding was confirmed in a recent large observational study using propensity-score matching, but long-term survival was not analysed [28]. In our present study, the 1-year mortality rate was not statistically different between SAVR and TAVI among patients with the most severe CKD (stage 4—5) (43% vs. 21%, respectively; $P = 0.34$). Nevertheless, a larger randomized trial will be necessary to assess this point, as both groups were not similar in our study, and the small number of patients may have limited the statistical power of this analysis.

Study limitations

They were several limitations to our study. Firstly, it was based on a retrospective registry and, for some patients, data may have been defective or kidney function poorly defined. eGFR is a dynamic variable that, both physiologically and pathologically, changes over time. This possible selection bias was reduced by using several serum creatinine measurements taken during the 3 months before the procedure, and by excluding patients with eGFR > 45 mL/min/1.73 m^2. Improved or deteriorated kidney function was then defined by a change of > 5 mL/min/1.73 m^2, to ensure stronger clinical relevance than other authors who used a variation of 1 mL/min/1.73 m^2 or 10% from baseline eGFR. A further limitation was that data on co-morbidities and treatments initiated or pursued were not recorded exhaustively in parallel to kidney function, so as to rule out confounding factors. Our body mass index data at 1 year after aortic valve replacement were also limited, and we cannot exclude an eGFR increase as a result of a loss of skeletal muscle mass [29]. Nevertheless, several reports have shown a substantial improvement in generic health status in the elderly after aortic valve replacement, suggesting that loss of muscle is limited [30]. Consequently, it is unlikely that the improved kidney function is only the result of a loss of muscle.

Conclusions

The present study found significant improvement in kidney function (> 5 mL/min/1.73 m^2) at 1 year in more than half of patients with CKD stage 3b—5 after SAVR or TAVI, without statistical difference between both groups. AKI was the major predictor of worsening kidney function at 1 year.

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Disclosure of interest

The authors declare that they have no competing interest.

References

[1] National Kidney F. K/DOQI clinical practice guidelines for chronic kidney disease: evaluation, classification, and stratification. Am J Kidney Dis 2002;39:S1—266.

[2] Sarnak MJ, Levey AS, Schoolwerth AC, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney

in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. Circulation 2003;108:2154—69.

[3] Ferro CJ, Chue CD, de Belder MA, et al. Impact of renal function on survival after transcatheter aortic valve implantation (TAVI): an analysis of the UK TAVI registry. Heart 2015;101:546—52.

[4] Oguri A, Yamamoto M, Mouillet G, et al. Impact of chronic kidney disease on the outcomes of transcatheter aortic valve implantation: results from the FRANCE 2 registry. EuroIntervention 2015;10:e1—9.

[5] Iung B, Vahanian A. Degenerative calcific aortic stenosis: a natural history. Heart 2012;98(Suppl 4):iv7—13.

[6] Ljungman S, Laragh JH, Cody RJ. Role of the kidney in congestive heart failure. Relationship of cardiac index to kidney function. Drugs 1990;39(Suppl 4):10—21, discussion 2-4.

[7] Damman K, van Deursen VM, Navis G, Voors AA, van Veldhuisen DJ, Hillege HL. Increased central venous pressure is associated with impaired renal function and mortality in a broad spectrum of patients with cardiovascular disease. J Am Coll Cardiol 2009;53:582—8.

[8] Dini FL, Demmer RT, Simioniuc A, et al. Right ventricular dysfunction is associated with chronic kidney disease and predicts survival in patients with chronic systolic heart failure. Eur J Heart Fail 2012;14:287—94.

[9] Allende R, Webb JG, Munoz-Garcia AJ, et al. Advanced chronic kidney disease in patients undergoing transcatheter aortic valve implantation: insights on clinical outcomes and prognostic markers from a large cohort of patients. Eur Heart J 2014;35:2685—96.

[10] Thourani VH, Keeling WB, Sarin EL, et al. Impact of preoperative renal dysfunction on long-term survival for patients undergoing aortic valve replacement. Ann Thorac Surg 2011;91:1798—806, discussion 806-7.

[11] Najjar M, Yerebakan H, Sorabella RA, et al. Reversibility of chronic kidney disease and outcomes following aortic valve replacementdagger. Interact Cardiovasc Thorac Surg 2015;21:499—505.

[12] Voigtlander L, Schewel J, Martin J, et al. Impact of kidney function on mortality after transcatheter valve implantation in patients with severe aortic valvular stenosis. Int J Cardiol 2015;178:275—81.

[13] Baumgartner H, Falk V, Bax JJ, et al. 2017 ESC/EACTS Guidelines for the management of valvular heart disease. Eur Heart J 2017;38:2739—91.

[14] Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of diet in Renal Disease Study Group. Ann Intern Med 1999;130:461—70.

[15] Levey AS, de Jong PE, Coresh J, et al. The definition, classification, and prognosis of chronic kidney disease: a KDIGO Controversies Conference report. Kidney Int 2011;80:17—28.

[16] Mehta RL, Kellum JA, Shah SV, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care 2007;11:R31.

[17] Beohar N, Doshi D, Thourani V, et al. Association of transcatheter aortic valve replacement with 30-day renal function and 1-year outcomes among patients presenting with compromised baseline renal function: experience from the PARTNER 1 Trial and Registry. JAMA Cardiol 2017;2: 742—9.

[18] Faillace BLR, Ribeiro HB, Campos CM, et al. Potential of transcatheter aortic valve replacement to improve post-procedure renal function. Cardiovasc Revasc Med 2017;18: 507—11.

[19] Thongprayoon C, Cheungpasitporn W, Kittanamongkolchai W, Srivali N, Greason KL, Kashani K. Changes in kidney function among patients undergoing transcatheter aortic valve replacement. J Ren Inj Prev 2017;6:216—21.

[20] D'Ascenzo F, Moretti C, Salizzoni S, et al. 30 days and midterm outcomes of patients undergoing percutaneous replacement of aortic valve according to their renal function: a multicenter study. Int J Cardiol 2013;167:1514—8.

[21] Bagur R, Webb JG, Nietlispach F, et al. Acute kidney injury following transcatheter aortic valve implantation: predictive factors, prognostic value, and comparison with surgical aortic valve replacement. Eur Heart J 2010;31:865—74.

[22] Najjar M, Salna M, George I. Acute kidney injury after aortic valve replacement: incidence, risk factors and outcomes. Expert Rev Cardiovasc Ther 2015;13:301—16.

[23] Leon MB, Smith CR, Mack M, et al. Transcatheter aortic-valve implantation for aortic stenosis in patients who cannot undergo surgery. N Engl J Med 2010;363:1597—607.

[24] Reardon MJ, Van Mieghem NM, Popma JJ, et al. Surgical or transcatheter aortic-valve replacement in intermediate-risk patients. N Engl J Med 2017;376:1321—31.

[25] Smith CR, Leon MB, Mack MJ, et al. Transcatheter versus surgical aortic-valve replacement in high-risk patients. N Engl J Med 2011;364:2187—98.

[26] D'Errigo P, Moretti C, D'Ascenzo F, et al. Transcatheter aortic valve implantation versus surgical aortic valve replacement for severe aortic stenosis in patients with chronic kidney disease stages 3b to 5. Ann Thorac Surg 2016;102:540—7.

[27] Nguyen TC, Babaliaros VC, Razavi SA, et al. Impact of varying degrees of renal dysfunction on transcatheter and surgical aortic valve replacement. J Thorac Cardiovasc Surg 2013;146:1399—406, discussion 13406-7.

[28] Doshi R, Shah J, Patel V, Jauhar V, Meraj P. Transcatheter or surgical aortic valve replacement in patients with advanced kidney disease: a propensity score-matched analysis. Clin Cardiol 2017;40:1156—62.

[29] Thongprayoon C, Cheungpasitporn W, Kashani K. Serum creatinine level, a surrogate of muscle mass, predicts mortality in critically ill patients. J Thorac Dis 2016;8: E305—11.

[30] Arnold SV, Reynolds MR, Wang K, et al. Health status after transcatheter or surgical aortic valve replacement in patients with severe aortic stenosis at increased surgical risk: results from the CoreValve US Pivotal Trial. JACC Cardiovasc Interv 2015;8:1207—17.