Cardiac mechanics in chronic kidney disease

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Part 2

VALVULAR HEART DISEASE IN CHRONIC KIDNEY DISEASE

Chapter 5

VALVULAR HEART DISEASE IN PRE-DIALYSIS AND DIALYSIS PATIENTS: PROGNOSTIC IMPLICATIONS

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Submitted

ABSTRACT

Aims

Patients with chronic kidney disease (CKD) have an increased risk for valvular heart disease (VHD) due to hemodynamic factors and metabolic pathways that promote valvular calcification and dysfunction. The prevalence of VHD in patients with CKD however, is unclear. The aim of the present study was to investigate the prevalence of VHD and its prognostic implications in pre-dialysis and dialysis patients.

Methods and results

From a retrospective cohort of pre-dialysis and dialysis patients (CKD stage 3b-5) who underwent clinically indicated echocardiography, the presence of significant VHD was assessed. Patients were divided into two groups according to the presence of significant VHD, defined as moderate or severe mitral regurgitation, mitral stenosis, tricuspid regurgitation, aortic regurgitation and/or aortic stenosis. Patients were followed-up for the occurrence of all-cause mortality. Of 281 pre-dialysis and dialysis patients (66% men, mean age 62±14 years), 74 (26%) had significant VHD. During a median follow-up duration of 30 months (IQR; 16, 60 months), 14% of patients with significant VHD underwent surgical or transcatheter valve replacement or repair and 33% of patients died. The cumulative mortality rates at 12, 24 and 36 months were 27%, 40% and 45% for patients with significant VHD versus 8%, 12%, and 18% for patients without significant VHD (log-rank P<0.001).

Conclusion

The prevalence of significant VHD in pre-dialysis and dialysis patients is 26%. Surgical or transcatheter valve replacement or repair was performed infrequently. In this population, patients with significant VHD had significantly increased risk of all-cause mortality compared to patients without significant VHD.

INTRODUCTION

The prevalence of significant (moderate and severe) valvular heart disease (VHD) in the general population of the United States of America is 2.5%.[1] Degenerative etiology is the most frequent cause of VHD and an increase in prevalence of VHD is projected due to the larger life expectancy and ageing process.[2] Patients with chronic kidney disease (CKD) have an increased risk for developing VHD due to hemodynamic factors and metabolic pathways that promote valvular calcification and valvular dysfunction.[3,4] However, the prevalence of significant VHD in CKD patients remains unclear.[5] Surgical or transcatheter valve replacement or repair are the only curative or supportive therapies for VHD. However, patients with advanced CKD are often denied or not referred to surgery due to the increased operative risk.[6,7] The prognostic implications of untreated severe VHD in the general population are well known, but have not been studied extensively in patients with CKD.[8] Accordingly, the objectives of the present study were to assess the prevalence of VHD in a well-characterized cohort of pre-dialysis and dialysis patients and its prognostic implications, including frequency of surgical or transcatheter valve intervention and all-cause mortality.

METHODS

Patient population and protocol

Pre-dialysis and dialysis patients from an ongoing registry at the Leiden University Medical Centre were selected for this retrospective study.[9] Pre-dialysis and dialysis patients who underwent clinically indicated transthoracic echocardiography were included in the present study, introducing a selection bias and potentially underestimating the prevalence of VHD. In patients receiving dialysis, echocardiography was performed after dialysis sessions (meaning dry weight), to avoid volume overload that could increase the frequency of significant valvular heart disease (particularly in terms of valvular regurgitation). Patients were diagnosed with CKD stage 3b-5 according to the 2012 Clinical Practice Guideline for the Evaluation and Management of CKD by Kidney Disease: Improving Global Outcomes.[10] Patients with a history of heart valve replacement or repair, younger than 18 years old and with inadequate echocardiographic image quality for off-line analysis were excluded. Clinical data, including demographics, medication

use, cardiovascular risk factors and laboratory results were collected in the departmental cardiology information system (EPD-vision; Leiden University Medical Centre, Leiden, The Netherlands) and electronical medical records (HiX; ChipSoft, Amsterdam, The Netherlands) and retrospectively analysed. Estimated glomerular filtration rate (eGFR) was calculated by the CKD Epidemiology Collaboration (CKD-EPI) equation.[10] In dialysis patients, residual renal function was calculated by the creatinine clearance using the pre-dialysis plasma creatinine concentration and the concentration of creatinine in a 24-hours urine specimen.[11] The occurrence of surgical or transcatheter valve treatment, endocarditis, aortic dissection and renal transplantation during follow-up was registered through case record review. All-cause mortality during follow-up was registered through case record review and the national death registry. The institutional review board approved this retrospective evaluation of clinically acquired data and waved the need for patient written informed consent.

Transthoracic echocardiography

Echocardiographic data were obtained with the patient in the left lateral decubitus position using commercially available systems (Vivid 7 or E9, General Electric Vingmed, Milwaukee, WI, USA) equipped with 3.5 MHz or M5S transducers and digitally stored in cineloop format for off-line analysis (EchoPac 112.0.1, GE Medical Systems, Horten, Norway). On M-mode recordings from the parasternal long-axis view, linear dimensions of the left ventricle were measured and left ventricular (LV) mass index was calculated and indexed to body surface area.[12] LV end-diastolic and end-systolic volumes were measured from the apical 4- and 2-chamber views and LV ejection fraction was calculated using the biplane Simpson's method.[12] Using the disk summation technique, the left atrial (LA) volume was measured in the apical 4-chamber view and indexed to body surface area. In the focused apical 4-chamber view of the right ventricle, the tricuspid annular plane systolic excursion, a measure of right ventricular function, was assessed on anatomical M-mode.[12] LV diastolic parameters consisted of peak early diastolic (E) and late diastolic (A) wave velocities, measured using pulsed wave Doppler recordings of the mitral inflow. Lateral E' mitral annulus velocity was measured with tissue Doppler imaging in the apical 4-chamber view at the lateral side of the mitral annulus and the E/E' ratio was calculated.[13]

The severity of mitral regurgitation (MR) was graded semi-quantitatively by measuring the width of the vena contracta in the parasternal long-axis or apical

4-chamber views.[14] MR was characterized as mild (vena contracta of 0.1-2.9 mm), moderate (vena contracta between 3 and 6.9 mm) or severe (vena contracta ≥7 mm).[14] In patients with calcified leaflets, MR was graded quantitatively, the proximal isovelocity surface area (PISA) was used to calculate the effective regurgitant orifice area (EROA) and the regurgitant volume (Rvol). The etiology of MR was defined as primary, when the leaflets of the mitral valve were structurally abnormal, or secondary, when the lack of leaflet coaptation was due to LV dilation and tethering of the leaflets. Mitral stenosis (MS) grade was estimated by measuring the mean gradient and mitral valve area using the pressure half-time method in the apical window.[15] MS was defined as mild (mean gradient <5 mmHg and valve area >1.5 cm²), moderate (mean gradient 5-10 mmHg and valve area 1.0-1.5 cm²) or severe (mean gradient >10 mmHg and valve area <1.0 cm²).[15] Tricuspid regurgitation (TR) severity was classified qualitatively by the continuous wave signal of the TR jet and semi-quantitatively by measuring the width of the vena contracta in the apical 4-chamber view.[14] TR was defined as mild (faint/parabolic continuous wave signal of TR jet), moderate (dense/parabolic continuous wave signal of TR jet and vena contracta < 7 mm) or severe (dense/triangular with early peaking continuous wave signal of TR jet and vena contracta ≥7 mm).[14] Aortic regurgitation (AR) was graded semi-quantitatively by measuring the pressure half-time of the continuous wave spectral signal of AR on the apical 5-chamber view.[14] AR was characterized as mild (pressure half time >500 ms), moderate (pressure half time of 500-200 ms) or severe (pressure half time <200 ms).[14] Aortic stenosis (AS) grade was estimated by measuring the aortic valve area, aortic jet velocity and the mean gradient in the parasternal and apical long-axis view or 5-chamber view.[15] AS was defined as mild (aortic valve area >1.5 cm²), moderate (aortic valve area 1.0-1.5 cm²) or severe (aortic valve area <1.0 cm²).[15] Patients were divided into two groups according to the existence of one or more significant VHD, defined as moderate or severe MR, MS, TR, AR and/or AS.

Statistical analysis

Continuous data were reported as mean ± standard deviation and median with interquartile range (IQR), as appropriate. Categorical data were reported as frequencies and percentages. Patients were divided into two groups according to the existence of significant VHD. Differences between the two groups were analysed using the Student's t-test or Mann-Whitney U-test for continuous data

and the chi-square test or Fisher's exact test for categorical data, as appropriate. The Kaplan-Meier method was used to calculate the cumulative event-free survival rates for all-cause mortality from the time of echocardiography. To compare the cumulative event-free survival rates between the two groups, the log-rank test was used. Univariable and multivariable Cox's proportional hazard analysis were performed to determine the independent association between significant VHD and all-cause mortality. All statistical tests were two sided. A P-value <0.05 was considered to indicate significance. Statistical analyses were performed using SPSS software (Version 20.0. Armonk, NY: IBM Corp)

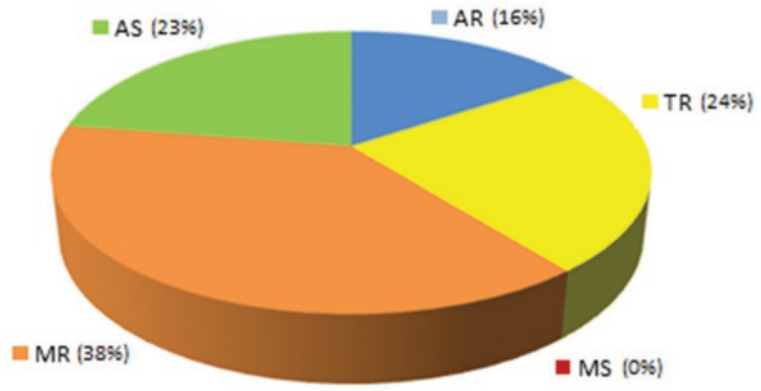

Figure 1. Prevalence of mitral regurgitation (MR), mitral stenosis (MS), tricuspid regurgitation (TR), aortic regurgitation (AR) and aortic stenosis (AS), in pre-dialysis and dialysis patients with significant valvular heart disease.

RESULTS

Patient population

Of 281 pre-dialysis and dialysis patients (66% men, mean age 62 ± 14 years), 74 (26%) patients had one or more significant VHD. More specifically, 39 (38%) patients had significant MR (23 patients with moderate MR, 16 patients with severe MR), 24 (24%) patients had significant TR (18 patients with moderate TR, 6 patients with severe TR), 16 (16%) patients had significant AR (16 patients with moderate AR) and 23 (23%) patients had significant AS (19 patients with moderate AS, 4 patients with severe AS) (Figure 1). There were no patients with

significant MS. Of the 39 patients with significant MR, 24 patients had secondary MR and 15 patients had primary MR (Table 1).

Table 1. Prevalence of significant valvar heart disease in pre-dialysis an dialysis patients

Variable	Frequency	Percent
Aortic stenosis	16	22%
Aortic regurgitation	9	12%
Mitral regurgitation	18	24%
Tricuspid regurgitation	8	11%
Aortic stenosis and aortic regurgitation	2	3%
Aortic stenosis and mitral regurgitation	2	3%
Aortic regurgitation and mitral regurgitation	3	4%
Mitral regurgitation and tricuspid regurgitation	11	15%
Aortic regurgitation, mitral regurgitation and tricuspid regurgitation	2	3%
Aortic stenosis, mitral regurgitation and tricuspid regurgitation	3	4%

Patients with significant VHD were older, received less often kidney transplantation, had a higher heart rate and more frequently presented with NYHA class III-IV heart failure symptoms, peripheral artery disease and atrial fibrillation compared to patients without significant VHD. Furthermore, patients with significant VHD more often used oral anticoagulation and had lower albumin levels compared to patients without significant VHD (Table 2).

Table 3 shows the echocardiographic characteristics of the groups. Patients with significant VHD had larger LV end-diastolic and end-systolic diameters and volumes, lower LV ejection fraction, larger left atrial volume index and higher LV mass index compared to patients without significant VHD. LV filling pressures were higher in patients with versus without significant VHD.

Table 2. Characteristics of pre-dialysis and dialysis patients with and without significant valvular heart disease

Variable	No significant VHD (n=207)	Significant VHD (n=74)	P-value
Clinical characteristics:			
Age (years)	59 ± 14	68 ± 13	<0.001
Men	136 (66%)	49 (66%)	0.936
Dialysis (vs. pre-dialysis)	70 (34%)	29 (39%)	0.406
Dialysis type (HD)[1]	52 (74%)	23 (79%)	0.595
Dialysis vintage (days)[1]	172 (70-385)	121 (40-300)	0.260
Renal transplant	90 (43%)	11 (15%)	<0.001
Heart rate (beats per minute)	71 ± 13	77 ± 20	0.017
Systolic BP (mmHg)	137 ± 20	135 ± 26	0.576
Diastolic BP (mmHg)	78 ± 11	76 ± 15	0.307
Body mass index (kg/m^2)	26 ± 5	25 ± 4	0.231
NYHA class III-IV	6 (3%)	14 (20%)	<0.001
Smoker	119 (60%)	47 (68%)	0.220
Diabetes mellitus	63 (30%)	18 (24%)	0.319
Hypertension	173 (84%)	61 (82%)	0.821
Hypercholesterolemia	83 (40%)	22 (30%)	0.114
Previous MI	45 (22%)	21 (28%)	0.248
Previous CABG/PCI	47 (23%)	18 (24%)	0.777
Peripheral artery disease	26 (13%)	19 (26%)	0.008
Atrial fibrillation	22 (11%)	25 (34%)	<0.001
Medications:			
Diuretics	126 (62%)	50 (70%)	0.223
ACE inhibitor/ARB	129 (64%)	42 (59%)	0.481
B-blocker	114 (56%)	48 (68%)	0.099
Calcium antagonist	90 (45%)	23 (32%)	0.074
Statin	125 (62%)	40 (56%)	0.411
Antiplatelet	71 (35%)	26 (37%)	0.824
Oral anticoagulation	36 (18%)	25 (35%)	0.002
Nitrates	18 (9%)	11 (15%)	0.122

Table 2. Continued

Variable	No significant VHD (n=207)	Significant VHD (n=74)	P-value
Laboratory results:			
RRF (ml/min/1.73m²)[1]	4.9 (2.1-8.6)	5.3 (2.8-7.7)	0.844
eGFR CKD-EPI (mL/min/1.73m²)[2]	18 ± 7	19 ± 7	0.498
Creatinine (umol/L)[2]	327 ± 122	302 ± 110	0.227
Urea (mmol/L)	22 ± 7	23 ± 8	0.340
Corrected calcium (mmol/L)	2.2 ± 0.1	2.2 ± 0.1	0.692
Phosphate (mmol/L)	1.4 ± 0.4	1.4 ± 0.4	0.759
Parathyroid hormone (pmol/L)	15 (7-25)	16 (10-28)	0.189
Albumin (g/L)	42 ± 5	39 ± 6	0.001
Glucose (mmol/L)	6 ± 3	6 ± 2	0.749
LDL-cholesterol (mmol/L)	2.4 ± 1.2	2.4 ± 1.1	0.956
Hemoglobin (mmol/L)	7.3 ± 1.0	7.1 ± 1.0	0.260

[1]Measured only in dialysis patients.[2] Measured only in pre-dialysis patients.
ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure; CABG, coronary artery bypass graft; CKD-EPI, chronic kidney disease epidemiology collaboration; eGFR, estimated glomerular filtration rate; HD, hemodialysis; LDL, low-density lipoprotein; MI, myocardial infarction; NTX, renal transplantation; NYHA, New York Heart Association; PCI, percutaneous coronary intervention; RRF, residual renal function; VHD, valvular heart disease.
Continuous data are presented as mean ± SD or median (interquartile range). Categorical data are presented as numbers and percentages.

Table 3. Echocardiographic characteristics of pre-dialysis and dialysis patients with and without significant valvular heart disease

Variable	No significant VHD (n=207)	Significant VHD (n=74)	P-value
IVSTd (mm)	11 ± 2	11 ± 3	0.053
PWTd (mm)	10 ± 2	10 ± 2	0.694
LVEDD (mm)	51 ± 8	55 ± 11	0.003
LV mass index (gm/m²)	108 ± 35	130 ± 37	<0.001
LVESD (mm)	33 ± 9	40 ± 13	<0.001
LVEDV (ml)	102 ± 39	138 ± 74	<0.001
LVESV (ml)	42 ± 26	78 ± 68	<0.001
LVEF (%)	60 ± 13	49 ± 18	<0.001
LAVI (mL/m²)	25 ± 12	35 ± 16	<0.001
TAPSE (mm)	18 ± 4	17 ± 5	0.174
Peak E-wave velocity (cm/s)	70 ± 23	82 ± 32	0.006
Peak A-wave velocity (cm/s)	80 ± 24	80 ± 29	0.981
Lateral E' (cm/s)	6 ± 3	5 ± 3	0.023
Lateral E/E'	11 (8-16)	16 (10-23)	0.001

IVSTd, interventricular septum thickness in diastole; LAVI, left atrial volume index; LV, left ventricular; LVEDD, left ventricular end diastolic diameter; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; LVESD, left ventricular end systolic diameter; LVESV, left ventricular end systolic volume; PWTd, posterior wall thickness in diastole; TAPSE, tricuspid annular plane systolic excursion; VHD, valvular heart disease. Continuous data are presented as mean ± SD or median (interquartile range). Categorical data are presented as numbers and percentages.

Follow-up

During a median follow-up duration of 30 months (IQR; 16, 60 months), 14% of patients with significant VHD underwent surgical or transcatheter valve replacement or repair. More specifically, 4 patients underwent surgical valve replacement or repair (2 patients under went aortic valve replacement, 1 patient underwent aortic and mitral valve replacement and 1 patient underwent mitral and tricuspid valve repair) , 6 patients underwent transcatheter valve replacement or repair (4 patients underwent mitraclip, 1 patient underwent transfemoral transcatheter aortic valve implantation (TAVI) and 1 patient underwent balloon dilatation of the aortic valve), and 1 patient presented with endocarditis. There

were no patients presenting with aortic dissection during follow-up. Thirty-six percent of patients received renal transplantation and 33% of patients died. Figure 2 shows the Kaplan-Meier curves of the cumulative event-free survival for all-cause mortality in patients with and without significant VHD. The cumulative mortality rates at 12, 24 and 36 months were 27%, 40% and 45% for patients with significant VHD versus 8%, 12%, and 18% for patients without (log-rank P<0.001).

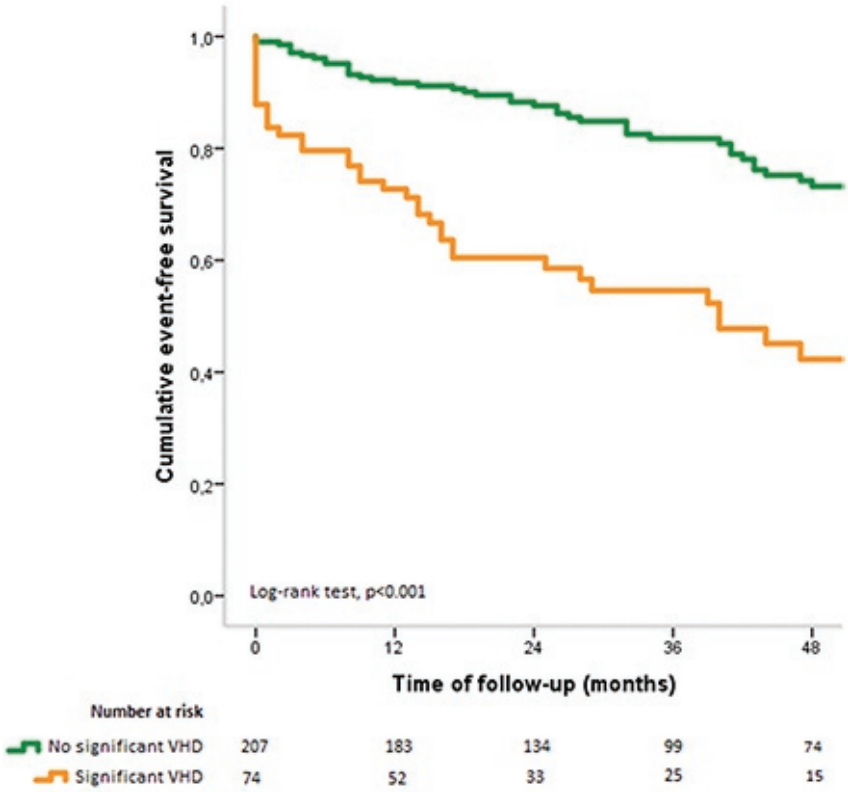

Figure 2. Kaplan-Meier curves demonstrating the relationship between significant valvular heart disease (VHD) and all-cause mortality in pre-dialysis and dialysis patients.

On univariable analyses age, renal transplantation, heart rate, NYHA class III-IV, peripheral artery disease, atrial fibrillation, albumin levels, LV mass index, LV ejection fraction, LA volume index, lateral E/E' and significant VHD were associated with all-cause mortality (Table 4). On multivariable analysis, significant VHD was independently associated with increased risk of all-cause mortality

after correcting for age, renal transplantation, NYHA class III-IV, LV mass index, LV ejection fraction, LA volume index and lateral E/E' (Table 4).

Table 4. Cox proportional hazard model showing the association between clinical and echocardiographic variables and the endpoint of all-cause mortality in pre-dialysis and dialysis patients with significant valvar heart disease

	Univariable HR (95% CI)	P-value	Multivariable HR (95% CI)	P-value
Age	1.07 (1.05-1.09)	<0.001	1.04 (1.02-1.06)	<0.001
Male gender	1.22 (0.78-1.90)	0.384		
RTx future	0.12 (0.06-0.26)	<0.001	0.23 (0.098-0.53)	0.001
Heart rate (bpm)	1.02 (1.01-1.03)	0.006		
NYHA class III-IV	3.38 (1.90-5.99)	<0.001	1.72 (0.76-3.87)	0.192
Peripheral artery disease	3.21 (2.03-5.07)	<0.001		
Atrial fibrillation	2.16 (1.35-3.47)	0.001		
Oral anticoagulation	1.58 (0.99-2.53)	0.056		
Albumin	0.89 (0.86-0.92)	<0.001		
LV mass index (gm/m²)	1.01 (1.00-1.01)	0.046	0.995 (0.98-1.01)	0.213
LVEF (%)	0.97 (0.96-0.99)	<0.001	0.99 (0.98-1.01)	0.436
LAVI (mL/m²)	0.98 (0.95-1.01)	0.027	1.01 (0.99-1.03)	0.370
Lateral E/E'	1.01 (1.00-1.02)	0.005	1.01 (0.99-1.03)	0.236
Significant VHD	3.09 (2.05-4.68)	<0.001	1.90 (1.12-3.22)	0.017

BPM, beats per minute; CI, confidence interval; LAVI, left atrial volume index; LV, left ventricular; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association classification; OR, odds ratio; RTx, renal transplantation; VHD, valvular heart disease.

DISCUSSION

In the present study, the prevalence of significant VHD among pre-dialysis and dialysis patients is as high as 26%. However, only 14% of the patients with significant VHD were referred for surgical or transcatheter valve replacement or repair. The presence of VHD was associated with poor survival in this population.

Prevalence of significant VHD in patients with CKD

Data on the prevalence of significant VHD in patients with CKD are limited.[5] In 62 patients with end-stage renal disease on long-term hemodialysis, mild

to severe VHD was observed in 18 (29%) patients.[5] In contrast, in the general population the age-adjusted prevalence of moderate to severe VHD was 2.5% (95% confidence interval: 2.2%-2.7%).[1] The higher prevalence of VHD in patients with CKD compared to the general population is caused by increased valvular calcification, cardiac dilatation and infective endocarditis.[3,16-18] Advanced CKD is associated with chronic volume overload leading to hypertension, increased cardiac output and peak flow velocity which generate flow turbulence.[3] Increased flow turbulence causes mechanical stress on the valves, which leads to valvular calcification and fibrosis.[3] In addition, chronic pressure overload, inflammation and dysregulated metabolic pathways, such as disorders in the calcium-phosphate metabolism, may lead to valvular calcification and secondary valvular dyfunction.[3,17,19] Cardiac dilatation, as a result of chronic volume overload, also causes valvular dysfunction by preventing appropriate coaptation of the leaflets.[16] The dynamic nature of valve regurgitation is well known and after intensified ultrafiltration, functional mitral and tricuspid regurgitation can disappear.[16] This highlights the difficulty in estimating the true prevalence of functional mitral and tricuspid regurgitation since their presence will depend on the loading conditions of the patient. In addition, hemodialysis patients have an increased risk for infective endocarditis.[18] In 9,393 patients undergoing hemodialysis, infective endocarditis was diagnosed in 150 patients, compared to 250 patients of 17,6369 age- and gender matched controls, yielding a 38-fold increased risk of endocarditis in hemodialysis patients.[18]

Treatment of patients with CKD and significant VHD

Fourteen percent of pre-dialysis and dialysis patients with significant VHD in our cohort underwent surgical or transcatheter valve replacement or repair during a median follow-up of 30 months (IQR; 16, 60 months). The therapeutic management of patients with CKD and significant VHD has not been extensively studied.[20] In a 5-year multicentre French survey conducted between 1988 and 1992, 98 chronic dialysis patients with severe VHD were identified and 62% of these patients underwent surgical valve replacement or repair.[20] The Euro Heart Survey on VHD conducted in 2001 identified 3,596 patients with mild to severe native valve disease and 31% of these patients underwent surgical valve replacement or repair during the survey period of 30 days.[6] Patients with CKD and severe VHD are often denied or not referred to heart valve replacement or repair, because of the high risk for surgery.[6,7] In high risk and inoperable

patients, transcatheter valve replacement or repair has emerged as a promising therapeutic intervention for severe VHD.[21,22] However, patients with severe renal insufficiency were excluded from these trials.[21,22] Data from the German registry on aortic stenosis, including 28,716 patients, showed a prevalence of CKD (stage 1-5) of 39%.[23] Seventy percent of these patients received a transfemoral TAVI and 30% a transapical TAVI. The in-hospital mortality rates of patients with stage 5 CKD was significantly higher as compared to patients with normal renal function (9.6% for transfemoral TAVI and 17.5% for transapical TAVI vs. 5.2% and 8.2%, respectively).[23] Interestingly, the in-hospital mortality for transfemoral TAVI is lower to that of dialysis patients undergoing cardiac operations (15.3%).[24] Therefore, transcatheter valve replacement or repair could be a promising therapy for patients with CKD and severe VHD.

Prognostic implications of significant VHD in patients with CKD

Pre-dialysis and dialysis patients with significant VHD had significantly increased risk of all-cause mortality compared to patients without significant VHD. Only 14% of patients with significant VHD in the present study were treated with surgical or transcatheter valve replacement or repair. Patients with CKD have an increased risk for all-cause mortality and CKD patients with significant VHD have an even higher risk, partly due to the low percentage of surgical or transcatheter valve replacement or repair in these patients. The high mortality risk in patients with CKD and significant VHD indicates the importance of appropriate treatment of these patients. Prospective studies are needed to investigate the role of transcatheter valve replacement and repair in patients with CKD and severe VHD.

Limitations

Several limitations should be acknowledged. The present study is an observational retrospective study, single-center, with relatively small number of patients and the study population was heterogeneous with different valve pathologies. Furthermore, only pre-dialysis and dialysis patients who underwent clinically indicated transthoracic echocardiography were included in the present study, introducing a selection bias and potentially underestimating the prevalence of VHD. In addition, echocardiography was performed after the dialysis sessions, where the severity of VHD may have been changed by loading conditions. Unfortunately, there were not enough patients to compare the survival of patients treated with surgical or with transcatheter valve replacement or repair.

Conclusion

The prevalence of significant VHD in pre-dialysis and dialysis patients is 26% and 14% of these patients underwent surgical or transcatheter valve replacement or repair. Patients with significant VHD had significantly increased risk of all-cause mortality compared to patients without significant VHD. Significant VHD was independently associated with an increased risk of all-cause mortality.

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