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ORIGINAL ARTICLE

Cardiac impact of the arteriovenous fistula after kidney transplantation: a case-controlled, match-paired study

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Summary

In kidney transplant (KT) recipients, cardiac impact of the persistence of an asymptomatic arteriovenous fistula (AVF) for hemodialysis has not been fully elucidated. Seventy-six patients (mean age: 49 years) without history of diabetes or cardiovascular disease underwent an echocardiography. Thirty-eight had a functioning AVF and were match-paired for age, gender and KT duration. Left ventricular mass index (LVMI) was significantly higher in patients with functioning AVF: 135.1 ± 30.3 vs. 112.4 ± 28 g/m^2 ($P = 0.001$). Exposure to AVF increased the risk of developing high LVH fourfold. Search for a dose-effect of AVF flow revealed a trend towards increasing LVMI with higher flow: 142.6 ± 30 vs. 126.9 ± 23.9 g/m^2 ($P = 0.084$) (median flow of the population as cut-off). Other significant changes were observed in left ventricular dimensions: greater end diastole- and systole diameters, both larger left and right atria, and left atrium diameter. Our study suggests that, in stable asymptomatic KT patients, functioning AVF has significant impact on cardiac mass, cardiac index and left ventricular dimensions. The effects on morbidity and mortality were to be investigated.

Introduction

In chronic kidney disease (CKD), cardiovascular disease (CVD) is the leading cause of morbidity and mortality [1]. From the initial stage of CKD to end stage requiring renal replacement therapy, several cardiac abnormalities and dysfunctions may occur, such as left ventricular hypertrophy (LVH), left ventricular dilatation with or without hypertrophy and systolic dysfunction [2,3]. In the end, LVH is found in almost 75% of the patients at the start of dialysis [4]. Concentric or eccentric LVH is an adaptative remodeling of the heart to maintain its functions, respectively in response to pressure overload (blood pressure, arteriosclerosis) and volume (extracellular volume fluid expansion, arteriovenous fistula (AVF), anemia) [5]. These effects are combined with nonhemodynamic factors linked to uremic toxins. Furthermore, hemodialysis and peritoneal dialysis are, in a different way, associated with the progression of LVH [6,7]. But, survival seems to be identical whatever be the dialysis modality [8,9]. The prevalence of LVH associated with CVD in kidney transplant (KT) recipients remains high, despite the expected beneficial cardiovascular effects of KT, by the correction of some major risk factors [10–13]. Persisting patency of the AVF may contribute to the persistent LVH.

The specific long-term contribution of AVF on LVH is not clearly determined. And closure after KT is still a matter of debate. First of all, transplant patients are reluctant to undergo this procedure, because, even after successful KT, they always view their AVF as a crucial access to hemodialysis. Nonetheless, in line with studies after AVF creation or persistence [14–17], studies after mandatory AVF closure because of infectious or cosmetic

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complications suggest that AVF may lead to progression of LVH and high cardiac output [18,19]. Last but not least, cardiac repercussions of AVF may be present even in asymptomatic patients with high AVF flow. To date, no randomized, controlled study of persistence versus systematic closure has been performed. The present case-controlled, match-paired study between KT recipients with and without AVF was undertaken to precisely evaluate the cardiac impact of a functioning AVF using standard echocardiography and Doppler tissue imaging.

Materials and methods

Patients: inclusion criteria and patient pairs

To control confusion biases on the potential cardiac impact of AVF, inclusion criteria formulated were: age between 30 and 70, absence of diabetes, absence of history of CVD, estimated creatinine clearance (Cockcroft and Gault formula) above 30 ml/min, and proteinuria under 1 g/day. In a cohort of 488 patients, transplanted between January 1995 and December 2002 and followed at our department, 155 patients met all the inclusion criteria. Fifty-six had an AVF still functioning. According to gender, age and KT duration, 38 pairs of patients (one with an AVF, one without) were created. Among those without AVF, nine were on peritoneal dialysis before KT. Two had a tunneled venous catheter for hemodialysis. Twenty-three had lost their access by spontaneous AVF thrombosis, four by planned closure. The delay of AVF close up or thrombosis was not registered in our study. Thrombosis usually appeared a few days after transplantation. Nevertheless, the AVF lost, by closure or thrombosis, had occurred at least 5 years before our study.

All patients were informed about the protocol study.

Data collection

Clinical, biological and echocardiographic measurements took place on the same day.

Blood pressure was measured and checked after several minutes of sitting in a quiet room, using a standard bladder. Hypertension was defined as systolic blood pressure higher than 140 mmHg and/or diastolic blood pressure higher than 90 mmHg, or as present in patients taking antihypertensive drugs.

Laboratory data included: serum creatinine, blood urea, hemoglobin, calcium, phosphorus, parathormone, C-reactive protein. Proteinuria was measured on 24-h urine collection.

Echocardiography was performed by three trained cardiologists (CSS, AC, AP) who standardized their measures. To limit inter-observer variability, all examinations were reviewed by the same senior echocardiographer (CSS). Echocardiographic studies were performed using a Vivid 7 Dimension (General ElectricHealthcare Company, Milwaukee, WI, USA) ultrasound system with standard imaging transducer. Analysis was performed according to the guidelines of the American Society of Echocardiography recommendations [20] and indexed to the body surface area: left ventricular end diastolic diameter (LVEDD), left ventricular end diastolic septal wall (LVEDPW) and left ventricular end diastolic septal wall (LVEDSW) were measured at end diastole. Left ventricular mass index (LVMI) was calculated as $0.8 \times [1.04(\text{LVEDD} + \text{LVEDSW} + \text{LVEDPW})^3 - \text{LVEDD}^3 + 0.6]$, divided by body surface area and expressed in g/m^2. Left ventricular systolic function, depending on pre- and postload, was evaluated by left ventricular end systolic (LVESD) and LVEDD, LVEDSW and LVEDPW, in mm, Simpson 2-dimension (2D) ejection fraction (EF, in %), and fractional shortening (FS in %). Left atrial diameter was measured by parasternal M mode and LA and right atrial area were obtained from apical 4 cavities (4C) view. Diastolic left ventricular function, which depends both on relaxation and compliance, was then studied using the mitral valve diastolic flow velocity curve defined by first the protodiastolic E wave, corresponding to the initial mitral inflow velocity peak, and the telediastolic A wave, corresponding to the inflow velocity caused by atrial contraction. The ratio E/A was calculated, together with the time deceleration curve of the E wave (TDCE). Right ventricular systolic function was assessed by the tricuspid annular plane systolic excursion (TAPSE, in mm). In order to detect any pulmonary effect of the possible cardiomyopathy, the pulmonary pressure was also measured.

Pulsed Doppler was used to measure AVF flow and diameter.

Statistical analysis

Clinical, biological data and echocardiographic parameters were compared using Student's *t*-test and Pearson's chi-squared tests for distributions. The α-error was 5%. The association between the presence of the AVF and echocardiographic abnormalities was tested with adjustment for gender, age, and KT duration. Multiple linear regression was used for quantitative criteria and logistic regression for binary criteria.

Results

We studied 76 patients divided in two groups: 38 with functioning AVF and 38 without AVF (Table 1). In each group, 65.7% were male. Mean age was 49 years and KT duration was 5.4 years, in each group. More than 85% of these transplant recipients were hypertensive (35

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Table 1. Comparison of baseline characteristics of kidney transplant recipients according to arteriovenous fistula (AVF) exposure.

	Patients with AVF (n = 38)	Patients without AVF (n = 38)	P-value
Gender ratio (male), *n* (%)	25 (65.8)	25 (65.8)	1
Weight (kg), mean ± SD	74.2 ± 16.1	70.7 ± 12.8	0.31
Age (years), mean ± SD	49.5 ± 8.1	49.07 ± 10.4	0.81
Hypertension (present), *n* (%)	35 (92.1)	30 (78.4)	0.22
Systolic blood pressure (mmHg), mean ± SD	133.9 ± 12	131.8 ± 11.4	0.47
Diastolic blood pressure (mmHg), mean ± SD	80.5 ± 8.7	80.7 ± 8.1	0.89
Anti-hypertensive drug, *n* (%)			
ACE inhibitor	27 (71)	22 (57.8)	0.23
Angiotensin receptor antagonist	4 (10.5)	4 (10.5)	1
β-Blocker	9 (23.6)	13 (34.2)	0.31
Calcium antagonist	12 (31.5)	12 (31.5)	1
Diuretic	2 (5.2)	1 (2.6)	0.52
Other	7 (18.4)	6 (15.7)	0.76
Biological values, mean ± SD			
Estimated creatinine clearance (ml/min)	62.3 ± 20.8	56.3 ± 17.5	0.18
Proteinuria (g/day)	0.20 ± 0.4	0.11 ± 0.2	0.12
Calcium (mg/dl)	9.86 ± 0.4	9.77 ± 0.52	0.46
Phosphorus (mg/dl)	3.01 ± 0.5	3.01 ± 0.7	0.97
Parathormone (pg/ml)	84.2 ± 53.4	73.8 ± 46.5	0.39
Hemoglobin (g/dl)	12.9 ± 1.4	13.3 ± 1.3	0.31
C reactive protein (mg/dl)	0.21 ± 0.5	0.23 ± 0.8	0.86
Attributed cause of renal failure, *n* (%)			
Glomerular	13 (34.2)	12 (31.5)	0.99
Vascular	1 (2.6)	1 (2.6)	
Interstitial	1 (2.6)	1 (2.6)	
Hereditary	6 (15.7)	7 (18.4)	
Malformative	6 (15.7)	6 (15.7)	
Unknown	11 (28.9)	11 (28.9)	
Dialysis duration (years), mean ± SD	1.8 ± 1.7	1.9 ± 2.9	0.85
Time after transplantation (years), mean ± SD	5.4 ± 2.2	5.4 ± 2.1	0.95
Patient with acute rejection episode, *n* (%)	11 (29)	16 (42.1)	0.23
Immunosuppressive drugs, *n* (%)			
Cyclosporine	27 (71)	18 (47.3)	0.03
Tacrolimus	8 (21)	18 (47.3)	0.01
Mycophenolate mofetil, azathioprine	28 (73.6)	27 (71.0)	0.79
mTor inhibitor	4 (10.5)	2 (5.2)	0.39
Corticosteroid	33 (86.8)	36 (94.7)	0.23

exposed and 30 nonexposed patients). Among them, 39.5% are treated with one drug, 34.2% with two, 11.8% with three or more and the last 14.5% with none. Renin-angiotensin system blockers were the main first-line anti-hypertensive drugs used. Few patients were taking diuretics. The most frequent cause of CKD was glomerulonephritis. Immunosuppressive drugs consisted of the combination of calcineurin inhibitors, mycophenolate mofetil or azathioprine, mTor inhibitor, and corticosteroid. Univariate analysis did not reveal any significant differences in clinical and biological characteristics, demonstrating the accuracy of the matching process. The only significant difference concerned the calcineurin inhibitor use, as patients with AVF were using less cyclosporin and more tacrolimus.

Echocardiographic characteristics

The results of univariate analysis are illustrated in Table 2. In patients with AVF, LVMI was statistically higher. The left ventricle was significantly enlarged both in systole and diastole. Ejection fraction was not different between the two groups. In case of exposure to AVF, right and left atria diameters and areas were significantly enlarged in patients and cardiac index higher. There were no statistically significant differences concerning left ventricular diastolic function, and left and right ventricular systolic and diastolic velocities.

After adjustment for age, gender and KT duration, multiple linear regression was performed, which demonstrated that patients with functioning AVF exhibited an

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Table 2. Comparison of echocardiographic characteristics according to arteriovenous fistula (AVF) exposure.

	Patients with AVF(N = 38)	Patients without AVF(N = 38)	P-value
LVMI (g/m^2)	135.1 ± 30.3	112.4 ± 28.0	0.001
LVD (mm)			
LVEDD	52.1 ± 7.1	48.5 ± 6.0	0.02
LVESD	34.3 ± 6.3	30.4 ± 5.3	0.004
W (mm)			
LVEDSW	11.1 ± 1.7	10.5 ± 1.6	0.1
LVEDPW	12.2 ± 1.7	11.5 ± 1.8	0.007
Ejection fraction (%)			
Teicholz	62.4 ± 8.6	66.5 ± 10.1	0.06
4 cavities	57.7 ± 8.8	61.4 ± 9.7	0.15
Cardiac index (l/min/m^2)	2.9 ± 0.6	2.4 ± 0.5	0.002
Shortening fraction (%)	32.3 ± 6.7	37.7 ± 8.0	0.06
E/A	1.06 ± 0.2	1.13 ± 0.2	0.22
TDCE (ms)	202.1 ± 43.2	188.9 ± 40.0	0.17
TAPSE (mm)	26.0 ± 5.4	25.7 ± 4.0	0.86
LAD (mm)	41.1 ± 5.4	37.3 ± 4.9	0.002
Area (cm^2)			
LA	19.3 ± 4.8	16.4 ± 3.8	0.008
RA	17.8 ± 5.1	15.3 ± 3.5	0.02
PAPs (mmHg)	28.9 ± 5.2	28.1 ± 3.7	0.50

LVMI, left ventricular mass index; LVD, left ventricular diameter; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; W, wall; LVEDSW, left ventricular end diastolic septal wall; LVEDPW, left ventricular end diastolic posterior wall; TDCE, time deceleration of the E wave; TAPSE, tricuspid annular plane systolic excursion; LAD, left atrial diameter; LA, left atria; RA, right atria; PAP, pulmonary arterial pressure.

Table 3. Flow effect of the arteriovenous fistula on echocardiographic parameters.

	High flow > 680 ml/min (n = 19)	Low flow ≤ 680 ml/min (n = 19)	P-value
LVMI (g/m^2)	142.6 ± 30.02	126.9 ± 23.9	0.08
LV (mm)			
LVDED	52.5 ± 7.6	52.1 ± 6.1	0.85
LVDES	34.1 ± 6.9	35.0 ± 5.4	0.63
Ejection Fraction (%)			
Teicholz	63.8 ± 8.3	60.3 ± 7.5	0.17
4 cavities	59.9 ± 7.9	56.8 ± 9.8	0.29
Cardiac index (l/min/m^2)	3.1 ± 0.7	2.6 ± 0.4	0.02
Shortening fraction (%)	35.3 ± 6.1	32.6 ± 5.5	0.16
E/A	1.1 ± 0.2	1.1 ± 0.2	0.88
TDCE	203.6 ± 39.5	201.8 ± 46.6	0.9
TAPSE (mm)	27.6 ± 5.4	23.7 ± 5.9	0.04
LAD (mm)	42.1 ± 5.8	40.0 ± 5.1	0.23
Area (cm^2)			
LA	17.2 ± 4.5	18.4 ± 5.1	0.52
RA	18.8 ± 5.1	15.9 ± 4.8	0.08
PAPs (mmHg)	29.6 ± 5.2	27.4 ± 4.6	0.21

LVMI, left ventricular mass index; LVD, left ventricular diameter; LVEDD, left ventricular end diastolic diameter; LVESD, left ventricular end systolic diameter; TDCE, time deceleration of the E wave; TAPSE, tricuspid annular plane systolic excursion; LAD, left atrial diameter; LA, left atria; RA, right atria; PAP, pulmonary arterial pressure.

LVMI which on average was 21.8 g/m^2 higher than in patients not exposed to AVF (P = 0.0006). In case of exposure to AVF, end systole and end diastole left ventricular diameters were higher, respectively on average 3.4 mm (P = 0.002) and 3.8 mm higher (P = 0.005), mean left atria diameter higher on average 3.6 mm (P = 0.001), the cardiac index higher 0.4 l/min/m^2 (P = 0.001). Logistic regression demonstrated that exposure to AVF increased the risk of developing LVH 4.3-fold [1.4–12.8].

In order to determine whether AVF flow had an impact, patients were divided into two subgroups according to AVF flow using 680 ml/min as cut-off, i.e. the median flow for the whole population. Mean AVF flow was 1040 ± 916 (range 315–4500) ml/min. Results presented in Table 3 are in favor of a correlation between flow and LVMI, cardiac index and right ventricular function.

Discussion

Our study suggests that, after successful KT, asymptomatic functioning AVF is associated with obvious cardiac abnormalities. As the population is relatively young, without diabetes and free of CVD, one strength of the study is the correct control of well-known cardiovascular risk factors [21]. Eighty percent of the patients presented with hypertension that was controlled. Proportion of ACE inhibitor or angiotensin receptor antagonist was similar in terms of differences between the two groups. Moreover, KT function was stable and good as estimated by a mean creatinine clearance over 55 ml/min, avoiding the cardiac effects of impaired renal function. A delay of 5 years since KT limited possible residual cardiac effects of dialysis [22]. Furthermore, the echocardiographies were performed by trained cardiologists and reviewed by the same operator. Last but not least, by means of the match-paired design, the two groups of transplant recipients are quite similar, except for AVF. For all these reasons, a functional AVF could be considered as an additional CVD risk factor in patients with a well-functioning KT.

We showed that this specific vascular access is associated with a significant cardiac remodeling, consisting in an increased LVH in KT recipients. Results of studies evaluating the cardiac impact of functioning AVF have been discordant. Creation of a AVF seems to lead to early functional cardiac adaptation to the high flow, as shown by the cardiac index, left ventricle diastolic function and

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late structural changes observed with a higher LVMI [15]. Interestingly, after closure of the AVF, reversible adaptative results were found, with at least some regression of the LVMI [23]. This regression is even more pronounced after 21 months of follow up: LVMI decreased from 139 ± 44 to 127 ± 45 and 117 ± 40 g/m^2 respectively at baseline, 1 month and 21 months ($P < 0.001$). A direct comparison of echocardiographic data between studies is hindered by methodological differences. Nevertheless our results were in agreement with the previously published data and add further support to the likely hypothesis of cardiac remodeling after correction of the specific risk factor of AVF. It is also noteworthy that the observed reduction in left ventricular mass, after AVF closure, was larger than that the reduction observed in a large study on the effect of anti-hypertensive therapy on LVH [24]. These patients were not KT recipients, were not CKD patients, and had not been exposed to a high flow access. Although a beneficial effect of AVF closure was observed, the optimal time to evaluate regression of LVMI remains to be determined. The main criticism is that all these studies were conducted in small populations followed for a very short period. The results obtained in one selected population that included a majority of patients with an AVF, suggested that the AVF was closed because of exercise-induced dyspnea, palpitations, heart failure, local fatigue, limb swelling or cosmetic reasons [18]. In contrast, most of the failed AVF of our population were consequences of spontaneous closure. Our results partly disagree with the findings of other comparative studies, based on the same model as ours, comparing cardiac parameters in exposed and nonexposed patients, and which showed no effect of the functional vascular access on LVH [19,25]. Samples in that study were small and were not match-paired. Moreover, patients with diabetes mellitus were included, adding a higher confusion bias. One other study showed that the decrease in LVMI after closure of the AVF in KT recipients was related to a decrease in LVEDD [17]. LVMI decreased significantly from 135 ± 34 vs. 119.8 ± 23 ($P < 0.01$) and LVEDD from 51.5 ± 5.8 vs. 49.3 ± 5.4 mm ($P < 0.01$). So closure of the AVF seemed to result partly in a decrease of eccentric LVH, without any effect on left ventricular wall thickness. We found similar LVEDD shortening. These results suggest that the effects of the AVF on cardiac structures were at least potentially reversible, or more probably explained by the reversal of the volume overload induced by this high-flow access. In previous study on AVF exposed patients, investigators did not perform exercise tests or myocardial stress tests to detect symptomatic patients. In our study, no patient had history of coronary diseases or reported clinical cardiac symptoms. However, no exercice tests or myocardial stress tests were performed. The clinical impact of these cardiac remodeling is to be investigated, so as to select patients in whom AVF closure could be beneficial.

Results concerning the effect of access flow suggest that the AVF is a new circulation, characterized by low resistance and high flow back to the heart, leading to a higher cardiac flow rate, proportional to the AVF flow rate. Very little data is available about cardiac index and access flow, and none about quantitative access flow and cardiac remodeling. The type of access flow (upper arm versus forearm) may affect cardiac index [26]. The aim of this study, carried out in dialysis patients, was to assess the relationship between access flow and systemic hemodynamics (cardiac output, cardiac index, central blood volume and peripheral vascular resistance) and to compare systemic hemodynamics between patients with upper arm and forearm access types. Mean AVF flow was 878 ± 411 ml/min in forearm and 1350 ± 560 ml/min in upper arm. Access flow was strongly related to systemic hemodynamics. Only a small percentage of patients with an upper-arm AVF seemed to be at risk of developing high cardiac index and failure. There was no difference between native fistula and PTFE grafts. We found similar results, with forearm flow at 848 ± 315 ml/min and upper arm at 1666 ± 900 ml/min. We did not have enough patients to compare native AVF and PTFE grafts. The high-flow group presented a trend towards a higher LVMI, a significant elevation of the cardiac index and of the right ventricular systolic function. These results suggest a dose-effect relationship between AVF flow and cardiac alterations, probably because of the greater venous return. We also pointed out significant left and right atrial enlargement in exposed patients.

Limitations of the study are its cross-sectional nature and the lack of detailed information, in both groups, about the real duration of CKD and hypertension, major cardiovascular risk factors. However, the mean duration of KT is more than 5 years, eliminating effects of factors related to dialysis modalities, and the lack of information regarding the duration of CKD and hypertension. Dates of AVF creation or closure were not documented in either of the groups, but the duration of AVF functional existence was more than 5 years, and the length of time closure was at least 1 year. The possible confounding effects of different immunosuppressive regimen on blood pressure and thus on LVMI have to be studied in a larger-sized population of patients. In fact, calcineurin inhibitors can promote HTA and therefore LVH [27]. In our study, there was no difference between our two groups concerning the use of calcineurin inhibitors and of cardiovascular protective drugs like ACE inhibitors or angiotensin receptor antagonists. Therefore, we cannot exclude that some unmeasured variables might have a

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confounding impact on the association between the functional AVF and left ventricular structure.

In conclusion, our study shows an association between the presence of a functional AVF in KT recipients and echocardiographic abnormalities. While closure of the AVF may be beneficial for the patient, it can also jeopardize a valuable access that the patient would need for further hemodialysis, raising the question of whether or not the AVF access should be systematically closed after a successful KT. Analysis of a larger group of high-flow AVF controlled follow up is also to be investigated, to give more information about the relevance of AVF in symptomatic and asymptomatic cardiac patients. Before recommending systematic closure, prospective and randomized studies are needed to establish evidence-based closure criteria and identify the predictability of LVH reversibility and its incidence on cardiac morbidity and mortality after KT.

Authorship

LF: designed study. CS-S, AC and AP: performed cardiac evaluation. MK and LF: contributed important reagents. JC: collected data. FA: analyzed data. JC: wrote the paper.

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