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Abstract

Purpose: International guidelines recommend that haemodialysis access is provided by an arteriovenous fistula (AVF), which enables frequent, reliable access to the circulation, but there are no guidelines to suggest whether these AVFs need to be ligated after kidney transplantation. Cardiovascular morbidity and mortality remain high in recipients of a kidney transplant, the persistence of a patent arteriovenous fistula (AVF) after transplantation may contribute to ongoing maladaptive cardiovascular remodelling. The ability to reverse this maladaptive remodelling by ligation of this AVF is unknown. We conducted this trial to evaluate the effect of AVF ligation on cardiac structure and function in stable kidney transplant recipients. Also we studied the ability of preoperative echocardiographic and non-invasive hemodynamic measurements, including the effects of acute temporary occlusion of the fistula, to predict postoperative left ventricular diameter and mass reduction, by the closure of the fistula.

Materials and Methods: Nonrandomized controlled trial. kidney transplant recipients (>12 months after transplantation with stable graft function) were divided into 2 groups. The first referred for surgical arteriovenous fistula closure. The second group didn't receive Fistula closure (control). Standard echocardiographic parameters, heart rate, and blood pressure were assessed preoperatively (fistula closure) at baseline. These measurements were repeated 6 months after surgical closure.

Findings: Seventeen kidney transplant patients were prospectively studied with 11 case and 6 controls with no fistula closure. Surgical fistula closure decreased left ventricular end-diastolic diameter and mass indexes (29.9_2.4to 27.4_2.1 mm/m2, P<0.001, and 141_37 to 132_39 g/m2, P<0.05, respectively), whereas no changes were seen in controls after a similar delay. Postoperative left ventricular end-diastolic diameter and mass reductions correlated best with the increases in total peripheral resistance (r_0.85, P<0.0001) and mean arterial blood pressure (r_0.64, P_0.006), respectively.

Conclusions. Surgical closure of arteriovenous fistula reduces left ventricular diameter and mass in kidney transplant recipients. The best predictors of those morphological changes are the rise in blood pressure and total peripheral resistance induced by temporary occlusion of the fistula**.**

Implications to Theory, Practice and Policy: Surgical closure of persistent AV fistula after renal transplantation to correct LV geometry and improve symptoms in terms of exertional dyspnea and palpitations.

Keywords: *Arteriovenous Fistula, Renal Transplant, Left Ventricular Hypertrophy*

1.0 INTRODUCTION

Left ventricular hypertrophy (LVH) is very common in patients with end-stage renal failure, and is an independent prognostic factor (1–3). The cause of LVH in renal patients is multifactorial and includes factors such as hypertension, anaemia, the uraemic state itself, and the presence of an arteriovenous fistula (AV), and is associated with subsequent development of heart failure and high mortality rates (*4–6*). Kidney transplantation is the preferred treatment for end-stage kidney disease, with significant improvements in quality of life and survival among kidney allograft recipients, cardiovascular death is the major cause of mortality after transplantation, it has been shown that left ventricular dimensions may improve after renal transplantation, although complete regression of LVH is usually not obtained. One of the factors that may contribute to the persistence of LVH after renal transplantation is the presence of an AV fistula.

The presence of an AV fistula lowers systemic vascular resistance, resulting in an increase in stroke volume and cardiac output in order to maintain blood pressure. (*7*). The detrimental role of fistula patency has been supported by various case reports of high output cardiac failure subsiding after surgical closure (*8*, *9*). In one study including 162 kidney transplant recipients the maintenance of a long-lasting AVF has been independently shown to be associated with LV hypertrophy and increased LV mass. (10) However, whether surgical closure of AV fistulas reverses cardiac morphological abnormalities remains a matter of debate. The prospective but uncontrolled study of van Duijnhoven et al. (*11*) showed some regression of LV diameter and mass after surgical closure of these fistulas. In addition, the contribution of AV fistula to cardiac performance, hypertrophy, and dilatation in an individual patient is often difficult to ascertain. It could be evaluated through the assessment of the hemodynamic effects of an acute, temporary occlusion of the fistula. Subsequently, and despite the lack of any additional evidence of a predictive value of the changes induced by an acute fistula occlusion, short manual compression has been repeatedly used to determine the hemodynamic burden induced by AV fistulas (*9*, *13–15*). Moreover, there are no data correlating the acute hemodynamic changes induced by a temporary occlusion of an AV fistula to the changes in LV morphology after fistula closure.

The only prospective evaluation of the effects of surgical closure of AV fistulas in renal transplant recipients did not assess the predictive value of acute compressions of AV fistulas and provided only limited information on which baseline hemodynamic or echocardiographic parameters best predicted the postoperative changes (*11*). The conclusions that can be drawn from the few available studies are that high flow fistulas causing symptomatic heart failure should be subjected either to reconstruction or ligation. One study clearly showed high predictive power for the occurrence of cardiac failure with high arterial flow (Qa) exceeding 2.0 l/min.(3)

Although closure of an AV fistula may benefit the patient, it also jeopardizes a valuable vascular access should kidney function deteriorate, and the patient need further haemodialysis. Thus, more information is required to determine which patients, if any, are most likely to benefit from surgical closure. The present prospective study was undertaken to assess the changes in LV morphology and function induced by elective surgical fistula closure in stable renal transplant patients and to determine the value of several preoperative echocardiography, and non-invasive hemodynamic measurements, at baseline and during the temporary AV fistula occlusion, in predicting postoperative changes in LV diameter and hypertrophy.

Null Hypothesis

Closure of arteriovenous fistula after renal transplantation doesn't reduce left ventricular diameter and mass.

2.0 METHODOLOGY

A cohort of 20 patients referred for surgical closure of an AV fistula were considered for enrolment. Three patients were excluded for the following reasons: regional wall motion abnormalities on echocardiographic examination, valvular heart disease, and CABG. All patients were in sinus rhythm. Thus, the study group consisted of 17 patients (Table 1) with AV fistulas considered large by Doppler measurement.

Fistula flow (QA) was assessed using an ultrasound colour-flow scanner with a linear-array transducer operating at 5 MHz in Doppler mode and 7.5 MHz in imaging mode. Fistula flow was expressed as the flow in the brachial artery 2 cm above the elbow (in the fistula arm) proximal to the fistula, assessed at the same height. This method was used as measurement of flow over the fistula itself can be unreliable because of the possible presence of irregularities. Measurements were performed in triplicate and a mean Qa value calculated. Most experts agree that high flow means a flow rate higher than 1600 ml/min. In our study "high-flow fistula" was defined as a Qa ≥1600 mL/min. (25). Echocardiography, blood pressure measurements, and blood chemistry analysis were performed within 4 weeks before the surgical closure and 6 months after the procedure for the 2 groups.

Echocardiographic studies were performed using GE Ultrasound System with standard imaging views. These measurements included LV end-diastolic and systolic diameters, shortening fraction, left atrial dimensions, interventricular septal, and posterior wall end-diastolic wall thickness (*16*). Doppler echocardiography allowed the measurements of stroke volume and cardiac output at the level of the LV outflow tract (*17*). LV ejection fraction was calculated using the Teichholz method (*18*). LV mass was calculated based on the American Society of Echocardiography convention using the Devereux formula (*19*). The changes in LV end-diastolic diameter and mass indexes induced by surgical closure of the fistula were expressed as: (Xpostsurgery -Xbaseline/Xbaseline)

The AV fistula was occluded at the end of the echocardiographic study by inflating a sphygmomanometer cuff to a 50-mmHg higher than the systolic pressure for 30 sec. This pressure was shown in preliminary experiments (27) To offset any increase in systolic blood pressure induced by the occlusion of the AV shunt. We also verified that the application of this pressure on the contralateral arm (sham occlusion) did not induce any hemodynamic changes. Cardiac output was determined within 1 minute before pneumatic occlusion and during the last 10 sec of the AV fistula closure.

Cardiovascular parameters (mean arterial blood pressure (MABP), cardiac index (CI), stroke volume index (SVI), and vascular resistance index (SVR)were non- invasively measured using thoracic bio-impedance technique (ICON) (13, 14).*)*. Hemodynamic parameters are measured by Electrical Cardiometry Technology (EC). Electrical Cardiometry is a method for the non-invasive determination of stroke volume (SV), cardiac output (CO), and other hemodynamic parameters in adults. Electrical Cardiometry has been validated against "gold standard" methods such as thermodilution and is a proprietary method trademarked by Cardiotronic, Inc

The placement of four skin sensors on the neck and left side of the thorax allow for the continuous measurement of the changes of electrical conductivity within the thorax. By sending a low amplitude, high frequency electrical current through the thorax, the resistance that the current faces (due to several factors) is measured. Through advanced filtering techniques, Electrical Cardiometry (EC) is able to isolate the changes in conductivity created by the circulatory system. One significant phenomenon, which is picked up is associated with the blood in the aorta and its change in conductivity when subjected to pulsatile blood flow. This occurrence is mainly due to the change in orientation of the erythrocytes (RBCs).

During diastole, the RBCs in the aorta assume a random orientation, which causes the electrical current to meet more resistance, resulting in a lower measure of conductivity. During systole, pulsatile flow causes the RBCs to align parallel to both the blood flow and electrical current, resulting in a higher conductivity state. By analyzing the rate of change in conductivity before and after aortic valve opening, or in other words, how fast the RBCs are aligning, EC technology derives the peak aortic acceleration of blood and the left ventricular ejection time (flow time). The velocity of the blood flow is derived from the peak aortic acceleration and used within our patented algorithm to derive stroke volume.

Occlusion of the AVF: Participants assigned to the intervention group then underwent surgical AVF ligation, which was performed as a same-day procedure under local anaesthesia.

Data collection: Basic patient demographic details including age, site of AVF, were recorded along with any evidence of symptomatic heart failure or prior major adverse cardiac events (MACE). The presence or absence of symptomatic heart failure was classified according to New York Heart Association (NYHA) criteria (15).

Statistical Analysis

For statistical analysis (Statview, SAS), Student's two-tailed t test for paired and unpaired data was used to compare the mean of the data obtained before and after surgical closure and to compare the controls with the group that underwent fistula occlusion, respectively. Data are reported as mean ± SD. Comparisons of means of data obtained at baseline, during temporary occlusion, and after surgical closure were analysed by a paired t test with a Bonferroni correction for multiple comparisons. Correlations between variables were assessed by the Pearson coefficient. Forward stepwise regression analysis was performed to determine which parameter best predicted LV mass and diameter regression after fistula occlusion. P values ≥ 0.05 were considered non-significant.

3.0 FINDINGS

All patients had stable kidney graft function. There were nine radio-cephalic, six brachiocephalic, one brachio-basilic, and one radio-basilic fistulas. For safety reasons, medications were not interrupted for this study. During the preoperative examination, 12 subjects used one or more antihypertensive drugs: 6 patients were treated with a calcium channel blocker, 10 with α-blocking agent, 6 with an angiotensin converting enzyme inhibitor, and 3 with diuretics. No patient was receiving nitrate therapy. Immunosuppression consisted of cyclosporine (n=10), mycophenolate mofetil (n=8), tacrolimus (n=5), azathioprine (n=5), and prednisolone (n=11). Except for one patient who had the dosage of enalapril reduced from 10 to 5 mg/day, there was no change in medication during the study period.

Controls

Six consecutive kidney transplant recipients with patent AV fistulas referred for routine echocardiographic follow-up served as controls (Table 1). These patients were matched for the time elapsed since renal transplantation and fistula creation to the patients who underwent surgical closure of their AV fistulas (Table 1).

Table 1: Patients' Clinical Characteristics

Patients	(n =17)	Controls (n=6)
Age (years)	46±13	6
Gender	8 male, 9 female	4 male, 2 female
Body surface area (m2)	1.76±0.14	1.89±0.26
Time after transplantation (months)	37±28 (range 10–116)	38±13 (range 17–51)
Time after fistula creation (months)	108±58 (range 39–248)	98±51 (range 45–180)

As compared with baseline, plasma haematocrit did not change after the surgical closure of the AV fistula (39.4±4.4versus 39.0±4.1%; *P*=NS). Creatinine clearance remained unchanged (54.3±13.1 versus 56.0±13.5 ml/min; *P*=NS). There was a slight postoperative decrease in plasma creatinine (1.55±0.41 versus 1.48±0.38 mg/dl; *P*_0.04), whereas blood urea nitrogen remained unchanged (29.6±12.6 versus 27.6±10.8 mg/dl; *P*=NS). There was no significant difference in baseline echocardiographic parameters between patients' group and control group, and parameters remained unchanged between the two studies in the control group.

Changes in Echocardiographic Parameters after Fistula Closure

The changes in echocardiographic parameters are shown in Table 2. Surgical closure decreased LV end-diastolic diameter and mass indexes and reduced left atrial diameter (*P*-0.05). The postoperative decrease in LV mass was the result of a decrease in LV end-diastolic diameter rather than a decrease in wall thickness. Indeed, interventricular septum thickness was unchanged, and a slight but significant increase in posterior wall thickness was observed.

Correlation between Changes in Hemodynamic Parameters

Induced by Temporary Occlusion and by Surgical Closure, Hemodynamic data at baseline, during acute pneumatic occlusion, and 1 month after surgical closure are listed in Table 3. Heart rate decreased during acute compression of the fistula. (*P*-0.001) but returned to baseline values after surgery. As compared with baseline, MABP increased during compression. (*P*-0.001) and, to a lesser degree, after the surgical procedure (*P*-0.05). Temporary AV occlusion led to a slight decrease in stroke volume and stroke volume index, which was more pronounced after surgery (*P*-0.001).

Cardiac output and index were markedly reduced both during the acute occlusion and after surgery (*P*-0.001). Cardiac output decreased by less than 1 L/min in only two patients during the compression of the fistula. These two patients had exertional dyspnoea as the main complaint. The reduction in cardiac output, cardiac index, and stroke volume observed after surgery correlated only moderately with the acute response. The net effect of the closure of the AV fistula on cardiac output (and to a lesser degree on blood pressure) resulted in a large increase in TPR that was still present after surgery (*P*-0.01), but the correlation between acute and postsurgical increase in TPR was not significant.

Predictive Factors of LV Diameter and Mass Regression

The parameters associated with a regression in LV end diastolic. Diameter and mass indexes in univariate analysis are shown in Table 4. The changes in heart rate, MABP, cardiac index, and TPR induced by acute fistula occlusion were significantly associated with a decrease in LV end-diastolic diameter index (*P*-0.05), whereas the acute increases in MABP and TPR were associated with LV mass index regression (*P*-0.05).

Using forward stepwise regression analysis, the acute increase in TPR during the pneumatic fistula occlusion emerged as the only independent predictor of the changes in LV end-diastolic diameter index induced by surgery (*r*-0.85, *P*-0.0001). Using the same analysis, MABP changes during acute occlusion were the only independent predictor of LV mass index regression (*r*-0.64, *P*-0.006).

Table 2: Echocardiographic Parameters Before and After Surgical Fistula Closure

	Patients			**Controls**		
	Baseline	**After surgery**	**P Baseline Vs. after Surgery**	**Study 1**	**Study 2**	**P (Study 1Vs.Study 2)**
LVEDDI (mm/m2)	29.9±2.4	27.4±2.1	0.0001	29±3.3	29.2±3.6	NS
LVESDI (mm/m2)	17.9±2.6	16.5±2.3	0.003	19.4±2.8	18.6±3.7	NS
IVS (mm)	12.4±2.7	13±2.9	NS	13.4±3.9	12.9±3.1	NS
PW (mm)	11±1.7	11.7±1.9	0.005	11.2±1.7	11.4±1.8	NS
LVMI(g/m2)	141±37	132±39	0.023	153±63	151±59	NS
LAD (mm)	45.3±5.2	43.0±4.9	0.014	46.4±6.2	46.8±5.8	NS
RVEDD(mm)	30.2±4.5	29.6±4.3	NS	28.9±4.5	27.8±4.5	NS
FS (%)	0.40±0.08	0.40±0.08	NS	0.33±0.04	0.36±0.07	NS
EF (%)	0.70±0.10	0.69±0.10	NS	0.61±6	0.65±0.10	NS

Table 3: Hemodynamic Parameters at Baseline, During Acute Pneumatic Occlusion, and After Surgical Closure

	Baseline	**Temporary Occlusion**	**Acute Changes (Temporary Occlusion minus Baseline)**	**After Surgery**	**Post Surgical Changes (after Surgery minus Surgery)**	**Correlation between Acute and Post Surgical Changes**
HR	72±9	64-10a	-8±5	71±6	-1± 10	r-0.10
(bpm)						P=NS
MABP	96.5±16.3	107.0±16.9a	10.5±8.2	103.3±13.6b	6.7±11.0	r-0.27
(mmHg)						P=NS
CO (L/min)	7.10±1.32	5.59±1.08a	-1.51±0.66	5.63±1.17a	-1.47±1.10	r-0.57
						Slope-0.95
						P-0.018
CI (L/min.m2)	4.03±0.66	3.17±0.54*a*	-0.85±0.36	3.20 ±0.62*a*	-0.82±0.62	*r*-0.56
						Slope-0.98
						P-0.02
SV (ml)	99±20	88±18*a*	-11±8	79±14*a*	-20±13	*r*-0.55
						Slope-0.94
						P-0.023
SVI (ml/m2)	56±10	50±9*a*	-6±4	45±8*a*	-11±7	*r*-0.51
						Slope-0.89
						P-0.04
TPR(dyn.sec.cm_5)	1127±301	1601±476*a*	474±275	1532±383*a*	405±243	*r*-0.30 P=NS
Abbreviations used in tables: HR, heart rate; CO, cardiac output; CI, cardiac index; SVI, stroke volume (index).						
*a P*_0.001 vs. baseline.						
*b P*_0.05 vs. baseline.						

Table 4: Predictors of postoperative left ventricular end-diastolic diameter and mass indexes regression (Univariate analysis)

Parameter (%)	**Chronic Changes a in LVEDDI (%)**			**Chronic Changes a in LVMI**		
	r	Slope *c*	*P*	*r*	Slope *c*	*P*
Baseline LVEDDI (mm/m2)	0.47	-1.2	0.06	0.12		0.64
Baseline LVMI (g/m2)	0.20		0.44	0.04		0.89
Baseline SVI (ml/m2)	0.19		0.46	0.14		0.60
Baseline HR (bpm)	0.13		0.62	0.006		0.98
Baseline MABP (mmHg)	0.24		0.36	0.05		0.86
Baseline CI (L/min.m2)	0.15		0.57	0.11		0.68
Baseline TPR (dynes.s.cm_5)	0.18		0.48	0.12		0.66
Acute SVI changes *b* (%)	0.39		0.12	0.01		0.97
Acute MABP changes *b* (%)	0.69	-0.46	0.002	0.64	-0.64	0.006
Acute HR changes *b* (%)	0.55	0.45	0.02	0.39		0.12
Acute CI changes *b* (%)	0.73	0.54	0.0008	0.32		0.22
Acute TPR changes *b* (%)	0.85	-0.23	≤ 0.0001	0.58	-0.23	0.01

Discussion

The value of several preoperative echocardiographic and non-invasive hemodynamic parameters in predicting the reduction in LV diameter and hypertrophy after surgical AV shunt closure in renal transplant recipients was determined. The most striking finding of our study is that acute changes in TPR and MABP induced by temporary AV fistula compression are the best predictors of postoperative improvements.

Reduction in LV End-Diastolic Diameter and Mass Indexes

The reduction in LV mass after AV fistula closure was a result of the reduction in LV end-diastolic diameter. The validity of LV mass calculation with M-mode echocardiography in the presence of rapid variations of LV diameter has been well documented (*23*). LV hypertrophy in end-stage renal disease is the result of combined effects of chronic flow and pressure overload and of non-hemodynamic factors associated with uremia. The flow overload and the associated LV dilatation are tightly related to the hyperkinetic circulation caused by anemia, plasma volume overload, and the presence of an AV access (*1*, *4*, *6*). Correction of the uremic state by renal transplantation has been associated with reduced LV dimensions and mass, although the latter is usually only partial (*7, 24*).The present study supports the hypothesis of a deleterious role of persisting large AV fistulas in kidney transplant recipients.

Indeed, our prospective assessment of LV diameter and mass demonstrated an early and significant decrease after elective surgical closure, whereas no changes were observed in a control group of kidney transplant recipients with patent AV fistula. Compared with the study group, patients in the control group were studied after a similar post transplantation period and with a similar delay between the two echocardiographic studies. Therefore, the observed improvement is not the consequence of a spontaneous regression of LV hypertrophy after kidney transplantation (*7*). Other factors that might interfere with cardiac structure such as haematocrit and renal function did not significantly change during the study period. There was slightly increase in mean arterial pressure after AV fistula surgical closure. It is therefore most likely that the observed decrease in LV diameter and hypertrophy was induced by the reduction of the chronic volume overload induced by the AV shunt. Although the optimal delay to evaluate LV hypertrophy regression after closure of a fistula remains to be determined, it is interesting to note that these beneficial effects, namely the reduction in LV diameter and hypertrophy as well as the reduction in left atrial dimension, occurred early after the closure of the AV fistula, thereby suggesting that the loading effect of increased peripheral resistance and blood pressure induced by the closure of the fistula is rapidly offset by the reduction in volume overload. These results mirror the effects observed after the creation of an AV fistula, which induces a decrease in TPR coupled with an increase in cardiac output, stroke volume, and LV end diastolic diameter (*25*).

The results of the present study are in accordance with those of Nickerson et al. (*12*), confirming that the acute changes in cardiac output and stroke volume correlate (albeit moderately) with postoperative changes. It has been shown that plasma atrial natriuretic peptide increases while plasma renin activity is suppressed after creation of an AV access, thereby inducing a direct relaxation of blood vessels (*25*). Conversely, opposite hormonal effects may participate in the long-term adaptation to fistula occlusion, namely in the increase in TPR. One might speculate on whether these hormonal changes may be less effective during the sudden cardiovascular changes

induced by the temporary AV shunt closure, thereby explaining the lack of correlation between the increase in TPR during acute and chronic occlusion.

Predictive Factors of LV Diameter and Hypertrophy Regression

The ability of several echocardiographic and non-invasive hemodynamic parameters, all of which are easy to obtain in clinical practice, to predict the regression in LV end-diastolic dimension and hypertrophy after surgical fistula closure was tested. In a recent study, van Duijnhoven et al. (*11*) found a correlation between preoperative LV mass and end-diastolic diameter and the reduction in LV mass as determined 4 to 5 months after surgical closure of the AV fistula. The hemodynamic effects of a temporary closure of the AV shunt were, however, not assessed in this study (*11*).

Our results, therefore, suggest that the presence of abnormalities in LV morphology alone is probably insufficient to guarantee that a patient will benefit from fistula closure. It was detected that postoperative reductions in LV diameter and hypertrophy are best predicted by the dynamic increase in TPR and blood pressure observed during an acute occlusion of the fistula. This acute increase directly relates to the proportion of the vascular bed that is dependent on the fistula. It is, therefore, not surprising that the acute increase in TPR and MABP best reflects the relative load imposed by the AV shunt and thus best correlates with the postoperative regression in LV dimension and hypertrophy. In the present study, an increase in TPR of more than a third of baseline value predicted a -5% reduction in LV end-diastolic diameter index with a sensitivity of 80%, a specificity of 71%, a positive predictive value of 80%, and a negative predictive value of 71%.

Similarly, an increase in MABP during compression of more than 10% of baseline predicted a -5% reduction in LV end-diastolic diameter index with a sensitivity of 70%, a specificity of 86%, a positive predictive value of 88%, and a negative predictive value of 67%. In a recent randomized controlled study, using the gold standard imaging modality (CMR) for evaluation of cardiac structure and function, they demonstrates that AVF ligation causes a reduction in LVM in the first year after kidney transplantation. There was a substantial reduction in LVM observed on the second CMR scan after AVF ligation. In contrast, there was no change in CMR-derived LVM in control patients.

4.0 CONCLUSION AND RECOMMENDATIONS

Conclusion

This study shows that AVF ligation is associated with a significant reduction in LV myocardial mass, and cardiac chamber dimensions, and those changes are associated with important clinical outcomes, these data support formal investigation of the impact of routine AVF ligation in stable kidney transplant recipients on clinical outcomes.

Our data suggest that surgical closure of persistent AV fistula after renal transplantation is safe and has the potential to rapidly correct abnormalities of LV geometry in a significant proportion of patients. In addition, the majority of our patients presented symptomatic improvement, in terms of exertional dyspnea and palpitations.

Recommendations

surgical closure of persistent AV fistula after renal transplantation to correct LV geometry and improve symptoms in terms of exertional dyspnea and palpitations.

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