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Hemodialysis arteriovenous fistula ligation after renal transplantation: Impact on graft resistive index

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Abstract

Background: Kidney allograft resistive index (RI) is prognostic for graft and recipient survivals. Recipient hemodynamics could influence RI. In particular, dialysis arteriovenous fistula (AVF) has been involved in heart function changes, reversible after AVF ligation. Knowledge about AVF and RI is lacking. In this study, we prospectively evaluated RI changes after AVF ligation in kidney transplanted patients.

Methods: We enrolled 22 stable transplanted patients. Mean RI was measured before AVF ligation (T0), 18 to 24 h (T1) and 6 months (T6) after surgery; mean blood pressure (mBP), heart rate (HR), serum creatinine (sCr), estimated glomerular filtration rate (eGFR), 24 h proteinuria (24 h-P), immunosuppressive drug blood levels (IS) and antihypertensive drugs were also recorded.

Results: AVF ligation was performed 3.1 years (IQR: 2.1–3.8) after transplantation. Median AVF flow (Qa) was 1868 mL/min (IQR: 1538–2712) and 8 AVF were classified as high flow (Qa $\geq$ 2 L/min). At baseline, median sCr was 1.32 mg/dL (IQR: 1.04–1.76) and median eGFR was 57.1 mL/min. Median RI was 0.71 at T0, 0.69 at T1, 0.66 at T6. RI reduction at T1 and T6 was statistically significant ($p < 0.05$ and $p < 0.001$ respectively); in particular, 90.4% of patients had persistently improved values at T6. Furthermore, mBP increased while HR decreased. These changes were independent from sCr, 24 h-P, IS, antihypertensive drugs number, Qa and AVF type.

Conclusions: AVF ligation improves kidney allograft RI; it may reflect better kidney perfusion.

Keywords

Arteriovenous fistula, renal transplant, renal resistive index, graft perfusion

Introduction

Arteriovenous fistula (AVF) is the preferred vascular access for hemodialysis since it associates with improved treatment efficacy and reduced infection risk than central venous catheter (CVC).[1] However, AVF creation affects systemic hemodynamics by reducing peripheral vascular resistance and by increasing heart venous return. Left ventricular hypertrophy, left ventricular diameters, and cardiac index increase, bi-atrial dilatation, tricuspid annular plane systolic excursion (TAPSE) decrease, cardiac oxygen supply and demand changes, atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) increased secretion are reported.[2–5] Different authors reported greater hemodynamic impact due to high-flow AVF (Qa $\geq$ 2 L/min) and they highlight the risk of heart failure, myocardial infarction, and lung hypertension.[6,3]

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In kidney transplant (KT) patients, changes of these heart morpho-functional alterations are reported after spontaneous or surgical AVF closure.[7–9] Recently, Rao et al.[10] demonstrated that AVF ligation in adults with stable KT function resulted in significant reduction of left ventricular myocardial mass and improved clinical heart performance. However, routine AVF ligation after KT is not recommended as it is not clear whether the benefits outweigh the risk of restarting dialysis without an appropriate vascular access.[11,12] To date, the most common reasons for AVF ligation are heart failure, high-flow fistula, local complications, and aesthetic.[13] Conversely, little is known about AVF impact on peripheral tissue perfusion and in particular on kidney graft.[14]

Kidney allograft RI is an indirect and dynamic marker of intrarenal vascular perfusion.[15,16] RI is calculated by Doppler ultrasound measure of blood speed within the arciform renal arteries; higher RI corresponds to increased resistance.[17] Initially, RI was considered as marker of intrarenal vascular injury; more recently it has been demonstrated that RI is also influenced by aortic vascular compliance and cardiac function.[18–20] Other factors influencing RI in KT are sex, body weight, height, and KT recipient age.[21]

Several studies reported RI evaluations as prognostic factor for long-term KT function, allograft and recipient survival, and both cardiovascular morbidity and mortality.[22–28] We herein hypothesized that AVF ligation induces RI changes related to hemodynamics variation; thus, we prospectively measured RI in a cohort of KT patients before and after AVF ligation.

Methods

We recruited all consecutives patients with stable KT function who underwent AVF ligation between April 2016 and February 2017 in AOU Città della Salute e della Scienza—Presidio Molinette, Turin. An internal commission approved the study protocol and the investigation was performed in accordance with the Helsinki declaration.

Two certificated experience in vascular ultrasound nephrologists independently performed AVF flow (Qa) Doppler measurement. AVF flow was calculated at the humeral artery. In particular, Qa was measured with a LA523 4.5 to 11.5 MHz linear probe (MyLabFive, Esaote). AVF ligation was performed by vascular surgical team with local or plessic anesthesia.

RI was assessed the day before AVF surgical closure (T0), 18 to 24 h (T1) and 6 months (T6) after surgery. Operators were blinded on AVF blood flow and previous RI results. Kidney interlobular arteries were evaluated by a CA621 3.5 to 8 MHz convex probe (MyLabFive, Esaote) and the RI was calculated with the formula: ((peak systolic velocity – end diastolic velocity)/peak systolic velocity). Three measurements were obtained (lower pole, between poles, and upper pole) and mean values were included in the statistical analysis. At the same time points, we also collected laboratory values, vital parameters, and drugs anamnesis. Collected variables were: serum creatinine (sCr), 24 h proteinuria (24 h-P), immunosuppressive blood levels (IS), estimated glomerular filtration rate (eGFR—calculated according to the chronic kidney disease epidemiology collaboration equation), HR, and mean blood pressure (mBP). Medication anamnesis included all drugs taken by patients, with particular attention to antihypertensive agents. All data were collected prospectively.

Statistical analysis

Statistical analysis was performed with SPSS (IBM SPSS Statistics, vers. 25.0.0). Continuous variables are described by median and interquartile range (IQR), according to their non-Gaussian distribution, and were analyzed with Kolmogorov-Smirnov test. Within-patient comparison was performed and the differences were analyzed with Mann-Whitney or Wilcoxon test. Categorical variables are presented as fraction and Pearson's χ^2 or Fisher's exact test were used to compare data. Significance level for all tests was set at $\alpha < 0.05$. Simple linear regression approach was employed to analyze the relationship between independent variables.

Results

We enrolled twenty-three patients with stable graft function and functioning AVF. One of them experienced severe acute interstitial rejection and restarted dialysis. Twenty-two patients completed the study with valid data. Table 1 reports baseline characteristics of our population. Median age was 60 years and 73% of patients were male. Nobody had a dual KT; one patient had a combined pancreas-KT and another a liver-KT. Two patients had a second KT. AVF ligation was performed after a median of 3.1 years from transplant (IQR: 2.1–3.8).

Median AVF survival was 7.1 years (IQR: 4.2–9.6). In all, 41% was brachiocephalic AVF, 54.5% was radiocephalic AVF at the forearm, 4.5% was brachial–basilic arteriovenous graft. Median Qa was 1868 mL/min (IQR: 1538–2712); 8/22 AVF were classified as high flow (Qa $\geq$ 2 L/min). The most frequent kidney disease resulted to be Autosomal Dominant Polycystic Disease (ADPKD) (36.4%). Twenty-one patients used calcineurin inhibitors (CNi) as immune suppressant: tacrolimus in 19/21 cases, cyclosporine in 2/21. About half of patients had cardiac hypertrophy, only one had a left ventricular hypokinesia and 18.2% had a previous ischemic event (coronary acute syndrome or inducible ischemia to myocardial scintigraphy). Lung hypertension has been detected in 35.7% of patients (defined as estimated pulmonary artery pressure

Table 1. Baseline characteristics of studied population.

Characteristics	Value
Gender male—%; (n)	73 (16/22)
Age in years—median value; (IQR: 25°–75°)	60 (50–70)
Transplant vintage in years—median value; (IQR: 25°–75°)	3.1 (2.1–3.8)
Single transplant—%; (n)	100 (22/22)
Pancreas-KT—%;(n)	4.5 (1/22)
Liver-KT—%;(n)	4.5 (1/22)
Second KT—%;(n)	9 (2/22)
AVF vintage age in years—median value; (25°–75°)	7.1 (4.2–9.6)
Brachiocephalic AVF—%; (n)	41.0 (9/22)
Radiocephalic AVF—%; (n)	54.5 (12/22)
Brachial—basilic graft—%; (n)	4.5 (1/22)
Qa AVF mL/min—median value; (IQR: 25°–75°)	1868 (1538–2712)
Qa $\geq$ 2 L/min—%; (n)	36.4 (8/22)
Kidney disease	
ADPKD—%; (n)	36.4 (8/22)
ESRD Unknown– %; (n)	22.7 (5/22)
IgA GN– %; (n)	13.7 (3/22)
IgM GN– %; (n)	4.5 (1/22)
Diabetic GS– %; (n)	4.5 (1/22)
MGN—%; (n)	4.5 (1/22)
SLE—%; (n)	4.5 (1/22)
Nephroangiosclerosis—%; (n)	4.5 (1/22)
CNi toxicity– %; (n)	4.5 (1/22)
Immunosuppression regimen with CNi	95.5 (21/22)
TAC– %; (n)	90.5 (19/21)
CyA– %; (n)	9.5 (2/21)
Immunosuppression regimen without CNi—%; (n)	4.5 (1/22)
Heart features	
Cardiac hypertrophy– %; (n)	52.4 (11/21)
Left ventricular hypokinesia—%; (n)	4.8 (1/21)
Ischemic event—%; (n)	18.2 (4/22)
Lung hypertension (PAPs > 25 mmHg) —%; (n)	35.7 (5/14)

AVF: Arteriovenous fistula. Qa: AVF blood flow. ADPKD: Autosomal Dominant Polycystic Disease. ESRD: end stage renal disease. IgA GN: IgA glomerulonephritis. IgM GN: IgM glomerulonephritis. Diabetic GS: diabetic glomerulosclerosis. MGN: membranous glomerulonephritis. LES: systemic lupus erythematosus. CNi: calcineurin inhibitors; TAC: tacrolimus; CyA: cyclosporin A. PAPs: pulmonary artery pressure systolic

Table 2. Donors characteristics.

Characteristics	Value
Gender male —%; (n)	36.4 (8/22)
Age in years—median value; (IQR: 25°–75°)	56 (45–67)
Donor sCr in mg/dL– median value; (IQR: 25°–75°)	0.71 (0.4–0.87)
Donor eGFR with Cockcroft–Gault formula mL/min—median value; (IQR: 25°–75°)	102 (65–148)

sCr: serum creatinine. eGFR estimated glomerular filtration rate.

systolic over 25 mmHg). Table 2 reports baseline donor characteristics.

In high-flow AVF the reason for surgical ligation was heart failure risk; in the others cases patients required ligation due to aesthetic reasons or local complications (aneurysm). No major surgical complications (i.e. bleeding or hemodynamic instability) were recorded during the procedure. Two patients needed re-surgery to drain hematoma, both at 6 days after ligation. No infections were recorded. In Figure 1, RI values, vital parameters, and laboratory exams recorded at three time points (T0–T1–T6) are reported.

Median RI was 0.71 (IQR: 0.66–0.74) at T0, 0.69 (IQR: 0.63–0.72) at T1, 0.66 (IQR: 0.61–0.69) at T6. RI changes from T0 to T1 and from T0 to T6 were statistically significant ($p = 0.034$ and $p < 0.001$ respectively—Figure 1). RI was reduced from T0 to T6 in 90.4% of cases, unchanged in only one case (4.8%) and increased in another one (4.8%). T0 to T6 median RI decrease was 0.04 (0.01 – 0.09).

Furthermore, median HR significantly decreased from T0 to T1 (from 68 to 64 bpm; $p = 0.004$) and median mBP significantly increased from T0 to T6 (from 98 to 105 mmHg; $p < 0.05$).

No significant changes in renal function (sCr, eGFR or 24 h-P) were observed at T6; median sCR was 1.33 mg/dL (IQR: 1.04–1.76) at T0, 1.32 mg/dL (IQR: 1.03–1.84) at T1, and 1.35 mg/dL (IQR: 1.15–1.72) at T6; median eGFR was 57.1 mL/min (IQR: 35.7–73.0) at T0, 57.1 mL/min (IQR: 32.7–72.5) at T1, and 55 mL/min (IQR: 37.7–73.2) at T6; 24 h-P was 0.166 gr/24 h (IQR: 0.10–0.23) at T0, 0.19 gr/24 h (IQR: 0.11–0.23) at T1, and 0.18 gr/24 h (IQR: 0.09–0.38) at T6.

Immunosuppressive regimen was unchanged in all but one case in which mycophenolate mofetil was added. Tacrolimus through levels were unchanged.

No correlation was found with tacrolimus blood levels and RI. No correlation was investigated for cyclosporine, due to the limited patient number. Antihypertensive therapy remained unchanged from T0 to T6; patients used an average of two antihypertensive drugs at each time. No correlation between AVF flow before ligation and changes in RI was seen, nor ligation of high-flow AVF was associated with larger RI drop. No cardiac events were recorded in the follow-up.

Discussion

AVF surgical ligation after KT is controversial. No definitive evidences are available and the procedure is not recommended in asymptomatic patients.[12] Different studies recorded echocardiographic improvements after AVF closure,[7–10] while some authors have reported a blood pressure raise.[14]

Kidney RI is a prognostic index for cardiovascular morbidity and mortality and for KT survival.[22–28] RI is

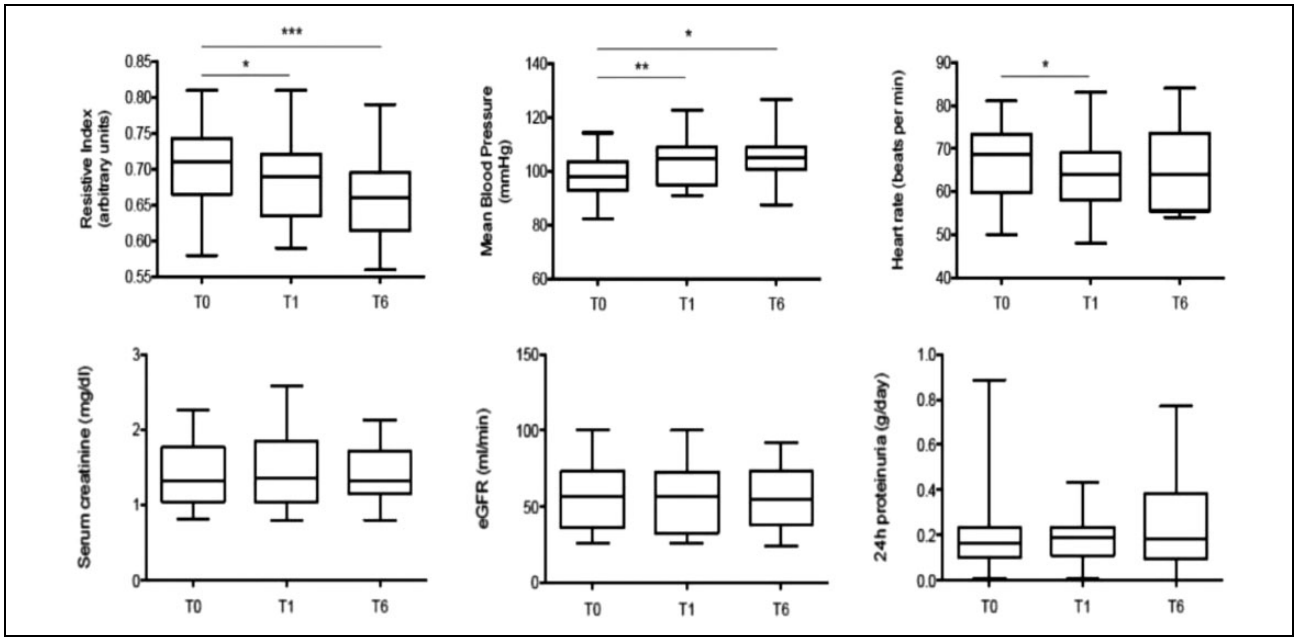

Figure 1. Results observed in the studied population.

influenced by a complex balance between renal and systemic hemodynamics. However, it is a reliable marker of parenchymal perfusion. In this study we hypothesized that the hemodynamic impact of AVF ligation could affect RI and thus renal blood flow.

Only one study assessed the impact of AVF closure on RI but the authors performed a temporary manual compression; thus, no follow-up data were reported.[28] The study demonstrated a significant decline in renal graft RI during AVF compression, and the drop was higher after the occlusion of accesses with higher Qa.

In our study, we prospectively measured RI in grafted kidneys before AVF ligation, 18 to 24 h and 6 months after the surgery. We observed a progressive RI decrease with the lowest values recorded at 6 months. Median magnitude of reduction was 0.04 (IQR: 0.01–0.09) and this occurred in 90.4% of cases. Higher reduction observed at T6 than at T1 could be related to a chronic adaptation mechanism.

This data demonstrates the renal hemodynamic impact induced by AVF closure and may highlight a clinical benefit in selected patients. Of note, no correlation between AVF flow before ligation and changes in RI was seen (data not shown), nor higher RI decrease in high-flow AVF, possibly as a consequence of the relatively high and homogeneous Qa values (median was > 1.8 L/min) in our cohort or the small number of strictly high-flow AVF (defined as Qa > 2.0 L/min). In addition, RI changes were independent from the AVF type.

Naesens et al.[27] observed that allograft recipients with a RI of at least 0.80 had higher mortality than those with a RI of less than 0.80 at 3, 12, and 24 months after transplantation. At protocol-specified biopsy time points, the RI was not associated with renal allograft histologic features. So, they conclude that the RI after transplantation reflects characteristics of the recipient but not those of the graft.

In our study RI was in almost all patients below 0.8, probably due to patient selection for AVF ligation. Only stable graft function patients were admitted for AVF ligation. It could be interesting to study the RI modifications and their clinical impact after AVF ligation in a basal RI more than 0.8 population.

Relation about RI reduction and graft function long-term improvement could be supposed, speculating on an amelioration in allograft perfusion and vascular resistances. In our experience, 24 months after AVF ligation, sCr and 24 h proteinuria were stable (data not shown). We could not detect any functional improvement, possibly because of the relatively small patient sample. In addition, 3.1 years interval between AVF ligation and transplantation could be a suboptimal timing to significantly impact on the allograft outcomes; at last, the high frequency of kidneys from extended criteria donors[29] could influence this observation. In literature, the possibility of renal function improvement after AVF ligation is debated; Vajdic et al.[30] observed improved eGFR after the procedure, while other authors did not.[31,32]

Consistently with the literature, we observed a mBP increase and HR decrease, probably related to peripheral vascular resistances rise.[14] Not all authors confirm this observation. In a recent RCT by Rao et al,[10] no effect of AVF ligation on blood pressure was observed. In our cohort, no significant correlation between RI reduction and mBP or HR changes was seen. This could be explained by the relatively small patient number or by the plethora of

physiological mechanisms regulating both mBP and RI; for instance, an improved kidney perfusion could lead to reduced angiotensin release.

Of note, antihypertensive therapy remained unchanged from T0 to T6; so, hypertension at T6 was not treated aggressively, failing the transplant guidelines recommendations aiming for a lower target blood pressure value. This observation reflects a "real life" situation: our center performs KT in patients coming from all parts of Italy; therefore, our sample included people distributed in many nephrologist centers; the medical treatments, except for immunosuppressive regimen, are managed by local nephrologists after patient discharging. More attention to the pressure management could be advocated in our indications.

At last, RI improvement after AVF ligation could add new elements in AVF closure decision after KT.

To our knowledge, our study is the first one to analyze prospectively the impact of AVF ligation on KT hemodynamics. A significant RI decrease was observed and persisted in 90.4% of cases after six months from the procedure. The small number of patients and the absence of a control group are the main limitations of this study. A power analysis prior to the study was not performed and the study is probably underpowered, in view of the variation in AVF flow and large numbers of variables that are included in the analysis. Further studies with larger samples are needed to prevent these limitations and to assess the long-term clinical outcomes. Furthermore, the relation between RI and spleen resistive index could be studied as a systemic hemodynamic marker.[33–35]

Author contribution

Authors Marica Magnetti and Gianluca Leonardi equally contributed to the work.

Declaration of conflicting interests

The authors declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: The authors declare that the design, the performance, and the data analysis were totally independent. The results presented in this article have not been published previously in whole or part, except in abstract form.

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Informed consent

All patients provided written informed consent before study entry.

Research involving human and animal participants

The study was conducted in compliance with the principles of the Declaration of Helsinki and in accordance with Good Clinical Practice guidelines. The protocols were reviewed and approved by an internal commission.

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