The life cycle of the vascular access: from creation to ligation

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Chapter 6

The pros and cons of preserving a functioning arteriovenous fistula after kidney transplantation

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Abstract

The autologous arteriovenous fistula (AVF) for hemodialysis burdens the cardiovascular system with increased cardiac output and pulmonary artery pressure, increasing cardiovascular risk. This article reviews literature on the benefits and drawbacks of a functioning AVF after kidney transplantation and discusses the cardiovascular effects of AVF closure. Several cohort studies demonstrate a significant cardiac burden of an AVF and improvement of cardiac dimensions after AVF ligation. However, no randomized trials have been conducted on routine AVF closure after successful kidney transplantation. Therefore, clinical trials are warranted to evaluate whether the cardiovascular benefits of routine AVF closure outweigh the potential harm for patients after successful kidney transplantation.

Introduction

In maintenance hemodialysis patients, the autologous arteriovenous fistula (AVF) is the preferred type of vascular access. Benefits of the AVF are superior long-term patency and low risk of infections, compared to central venous catheters and prosthetic arteriovenous grafts [1, 2]. However, important drawbacks of AVFs are the occurrence of hand ischemia and high-output heart failure by volume overload [3-6]. The presence of a functioning AVF is associated with a higher left ventricular mass and pulmonary hypertension. The true incidence of high-output heart failure is probably underestimated, as fluid retention can be managed by adapted ultrafiltration in hemodialysis patients.

Enormous efforts are made to create and maintain an adequately functioning vascular access when patients are on hemodialysis. With the increasing kidney allograft survival, we face the dilemma of deciding what to do with a functioning AVF after successful kidney transplantation.

The optimal approach towards asymptomatic patients after kidney transplantation is a topic of debate. In current clinical practice, the AVF is often neglected if the patient with a functional kidney allograft is not reporting any symptoms related to their AVF. However, in some clinics, routine closure is performed [7]. Routine surgical closure of the AVF might be beneficial for these patients in reverting left ventricular dysfunction, or preventing its progression. It is striking to notice that despite the sheer magnitude of care for vascular access related complications for patients on hemodialysis, not a single remark is made about vascular access care after transplantation in any of the current vascular access guidelines from EBPG and NKF KDOQI nor the KDIGO guideline on post-transplantation care [1,8,9]. In the present review, we discuss the benefits and drawbacks of a functioning AVF after kidney transplantation, as well as the previous literature on the cardiac effects of AVF closure.

Why should we aim to maintain a functional vascular access after kidney transplantation?

The advantage of maintaining the AVF after kidney transplantation is to have a functional vascular access if the allograft fails and hemodialysis is again required. Whether or not this strategy of vascular access preservation is defensible, depends on the chance that a specific patient will lose allograft function in the near future. The report from the US Renal Data System from 2014 revealed that the 10-year probability of allograft failure after transplantation in 2002, is 35.5% for recipients of a deceased donor transplant and 25.8% for living donor transplants [10].

The percentage of patients with a failed allograft declined in subsequent years, illustrating an ongoing improvement in long-term kidney allograft survival. In the Netherlands, the current 5-year survival with a functioning allograft is 84% for living donor transplants and 70% for deceased donor transplants [11].

Another issue that should be taken into account is the chance of spontaneous occlusion of the AVF after transplantation. In a retrospective study of 542 kidney transplantation recipients in 2005, long-term AVF patency was evaluated with an up to 10 year follow-up [12]. Spontaneously occluded AVFs were observed in 45% of patients, both in patients with a functioning transplant as well as in cases of transplant failure. In 55% of patients requiring hemodialysis, their previous AVF was used. Another European study found similar results in a cohort of 160 patients [13].

These studies suggest that long-term AVF patency after kidney transplantation is approximately 50%. Approximately 30% of all kidney transplantation recipients return to hemodialysis within 10 years. With routine closure in all kidney transplantation recipients, 15% would have the disadvantage of needing a new vascular access, when the previous AVF could have been used otherwise.

The question rises if patients at high risk of kidney allograft failure can be identified to allow an individualized decision regarding vascular access management after transplantation, excluding them from routine AVF closure. In this respect, it is important to notice that the incidence of kidney allograft loss is highest in the first year after transplantation [14]. Therefore, the estimated glomerular filtration rate (eGFR) at one year after transplantation could be used to predict long-term allograft survival, especially when combined with the slope of eGFR in the first year [15].

Another reason to maintain the arteriovenous access is the use of this conduit for venipunctures and intravenous administration of medication in patients with severely damaged superficial veins. Routine closure should therefore only be considered in patients whose venous anatomy is suitable for venipunctures and future vascular access creation. Obviously, also for patients with a functional kidney allograft, vein preservation is of vital importance.

Potential harm of a functional vascular access after kidney transplantation

Local symptoms like aneurysm formation, steal, cosmetic objections, infection or functional limitations of the extremity are common reasons for surgical AVF closure. While these local

complications are very obvious, the hemodynamic and cardiac effects of the AVF are often much more insidious in nature [6].

To appraise this so-called arteriovenous cardiotoxicity, one needs to know how the cardiovascular system responds to AVF surgery. As already described by Guyton in 1961, an immediate increase in cardiac output occurs upon creation of an AVF [16]. This increase is required to compensate for the drop in vascular resistance and the additional blood flow through the AVF, whilst maintaining organ perfusion.

As vascular resistance decreases after AVF surgery, blood pressure lowers and venous return increases [16,17]. As a result of the increased venous return, more blood is available for diastolic filling of both the left and right ventricle [18]. This increased diastolic filling causes an immediate increase in left ventricular diastolic diameter and volume. In accordance with the Frank-Starling mechanism, stroke volume and thus cardiac output increase [18]. Serum concentrations of atrial and brain natriuretic peptide rise, reflecting the immediate hemodynamic burden of the AVF [17].

Figure 1 provides a schematic representation of the effect of AVF creation on cardiac output in an otherwise healthy cardiovascular system. As demonstrated by Basile, a near linear correlation exists between vascular access flow and cardiac output (Figure 2) [19]. In this study, the mean cardiac output was 5.6 L/min in patients with a forearm AVF and 6.9 L/min with an upper arm AVF.

Even more important than the acute functional adaptation, persisting structural cardiac remodeling also occurs. A 13% increase in left ventricular mass at 6 months after AVF surgery has been observed [18]. As the resulting cardiomyopathy progresses and the heart begins to fail, the initially high cardiac output may decrease to within the normal range. The effective cardiac output, defined as the cardiac output minus the AVF flow, steadily decreases, resulting in systemic hypoperfusion. This state of pseudonormalization is often overlooked, as the cardiac output appears normal if the AVF flow is not subtracted from it. Therefore, it is of vital importance to take the AVF flow into account when assessing cardiac output and to recognize pseudonormalization as a sign of severe cardiac impairment.

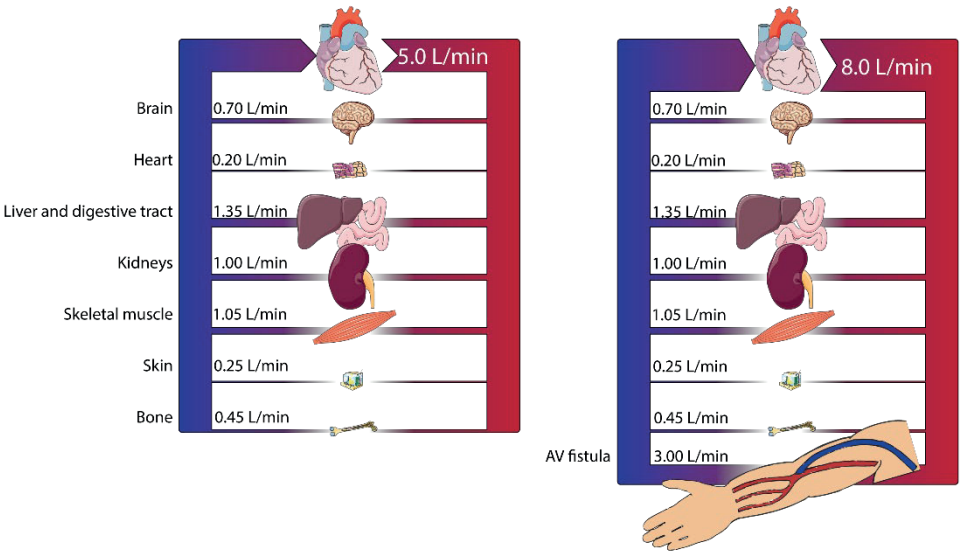

Figure 1 Systemic circulation with organ sizes reflecting relative perfusion, without AVF, with an AVF, resulting in increased cardiac output. AVF; arteriovenous fistula.

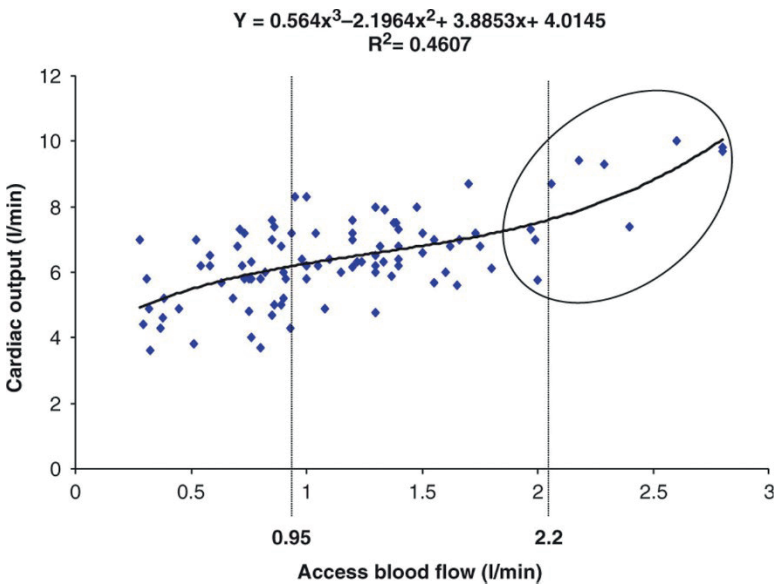

Figure 2 Correlation between vascular access blood flow and cardiac output. Reproduced from Basile et al [19], with permission from European Renal Association-European Dialysis and Transplant Association.

Inevitably, AVF surgery also affects the pulmonary circulation, with increased pulmonary artery pressure (PAP) [20,21]. This increase, along with the increase in left ventricular mass might have serious consequences for the prognosis of patients since both are independent predictors for mortality in hemodialysis patients [22,23] as well as in renal allograft recipients [24–27]. After initiating hemodialysis, the left ventricular mass increases significantly [28]. LVH is common in hemodialysis patients, with a recent study demonstrating a 71% prevalence [26]. Although these observations may also be in part caused by altered hemodynamics and fluid overload due to progressive chronic kidney disease itself, volume overload due to the AVF is an important contributor to left ventricular hypertrophy and pulmonary hypertension.

Since the fluid status of hemodialysis patients is in part regulated by the nephrologist by modifying ultrafiltration rate, the diagnosis of high-output heart failure is easily overlooked. Dyspnea and weight gain are often attributed to non-compliance with the prescribed fluid restriction for hemodialysis patients.

Several observational studies on cardiac changes after kidney transplantation have shown a substantial decline in left ventricular mass, (LVM) although complete normalization of the LVM rarely occurs [29–31]. The cardiac burden of persistent hypertension and the AVF might play a role in this incomplete normalization of LVM.

Improvement of left ventricular dimensions and function after AVF closure

Two different cohort studies of kidney transplant recipients revealed that the mean LVM was significantly higher in patients with a functioning AVF, when compared to patients without a patent AVF [32,33]. These observations suggest that the unfavorable effect of the AVF remains relevant after kidney transplantation [33]. The question arises whether AVF ligation could indeed result in further normalization of LVM after kidney transplantation.

Through Pubmed, Embase and the Cochrane Collaboration, we identified 8 cohort studies that report on the cardiac effects of AVF closure (table 1). Two studies have shown that temporary occlusion of an AVF instantly improves hemodynamics, reducing both heart rate and cardiac output [34,35]. A couple of cases of AVF closure in patients with high-output heart failure have been described [36–39]. Both cardiac output and PAP decrease after surgical closure of the AVF while the functional performance of patients improves. Timely AVF closure could be very important, since irreversible cardiac changes and possibly fixed pulmonary hypertension can

Study and design	Patient characteristics	Indication for closure	Number of patients	Follow-up (months)	LVMI at baseline (g/m²) ± SD	LVMI at follow-up (g/m²) ± SD	LVMI change (%) ± SD	Difference between groups (g/m²)
Duijnhoven 2001 Prospective observational, single-arm	Renal transplant patients No heart failure or NYHA ≤ II	Routine	20 AVF closed	5	135.0 ± 34.1	119.8 ± 23.2	-11.3% p<0.01	-15.2 ± 9.2
Unger 2004 Prospective observational, case-control	Renal transplant patients 10 (40%) heart failure patients	Cardiac symptoms, venous hypertension, cosmetic	17 AVF closed	21 ± 10	139 ± 44	117 ± 40	-15.8% p=0.0167	-22 ± 14.4
			8 controls	24 ± 14	114 ± 19	115 ± 18	+1% p=0.85	+1 ± 9.3
Movilli 2010 Prospective cohort	Hemodialysis patients No heart failure	Malfunctioning AVF	25 AVF closed	6	135 ± 40	123 ± 35	-8.2% ± 5.6 p < 0.001	-12 ± 8.1 *
			36 controls		138 ± 35	138 ± 33	-1% ± 8.8 p=NS	0 ± 11.3 *
Kurita 2011 Retrospective cohort	Hemodialysis patients All patients refractory heart	Refractory heart failure	33 AVF closed	1	160 ± 79	162 ± 81	+1% p=NS	+2 ± 19.7

	failure NYHA ≥ II							
Glowinski 2012 Retrospective cohort	Renal transplant patients No heart failure	Cosmetic (56%), spontaneous thrombosis (44%)	9 AVF closure	3	118.5 ± 26.3	113.1 ± 21.6	-4% p=0.09	-5.4 ± 11.3
			9 controls		116.0 ± 22.5	115.6 ± 18.5	0% p=0.16	-0.4 ± 9.7
Dundon 2014 Prospective cohort	Renal transplant patients No heart failure	Cosmetic	18 AVF closed	6	166 ± 56	149 ± 51	-10% p=0.0001	-17 ± 17.9
Sheashaa 2004 Prospective cohort	Renal transplant patients	Spontaneous occlusion	17 AVF closed	12	210 ± 80 (Am Soc echocardiogr formula)	169.5 ± 61.3	-19%	-4.5 ± 24.4
			34 controls		248 ± 83.5	176.3 ± 41.4	-29%	-71.7 ± 16.0
Gorgulu 2011 Cross-sectional	Renal transplant patients	Spontaneous occlusion (67%), ligation for: aneurysm (18%), cardiac symptoms (6%), cosmetic (6%), thrombus (2%)	49 AVF closed	Unknown	n/a	125 ± 42	n/a	+4 ± 7.7
			60 AVF patent			129 ± 37		

Table 1 Studies that evaluated change in left ventricular mass index after AVF closure. Data from Movilli, 2010 have been calculated from raw data supplied by the author.

eventually occur [7]. Pulmonary hypertension can be fully reversible if AVF closure is performed timely [39].

A prospective study was performed by Van Duijnhoven, et al. in which the effect of routine closure of vascular accesses was assessed [40]. Nineteen patients with native AVFs and 1 patient with a PTFE-graft were included, with a mean flow of 1790 ml/min, without heart failure higher than NYHA class 2. Cardiac ultrasound examinations were performed 2 and 4-5 months after AVF closure. The mean LVMI decreased from 135 g/m^2 to 120 g/m^2 at 4-5 months after closure. The prevalence of LVH decreased from 60 to 33%. In a subgroup analysis in patients more than 18 months after kidney transplantation, the reduction of LVMI was also significant (136 to 123 g/m^2). The authors assumed LVMI was expected to remain stable in these patients if the AVF would not have been closed, and concluded that the improvement in this subgroup was likely due to the AVF closure.

Smaller studies by Unger and Dundon also demonstrated a significant improvement in LVMI after AVF closure in patients with and without heart failure [41,42].

Movilli, et al. performed a study in which cardiac ultrasound examinations were prospectively performed in 25 hemodialysis patients who underwent AVF closure and conversion to a tunneled central venous catheter (CVC) and 36 controls who continued hemodialysis through an AVF [43]. Cardiac ultrasound examinations were performed at baseline and 6 months after AVF closure. While baseline measurements were not significantly different between groups, LVMI regressed from 135 g/m^2 at baseline to 123 g/m^2 at 6 months after AVF closure, whereas the LVMI in the control group did not change.

In a retrospective study by Sheashaa in 17 patients with a spontaneously occluded AVF after kidney transplantation and 34 controls [44]. The LVM at 1 year was improved in both groups. However, no significant difference was observed between groups. Cardiac output was significantly lower in the group with a closed AVF compared to the group with a patent AVF (4.3 L/min vs 5.8 L/min, p=0.010). This study concludes that even though cardiac output is significantly higher with a patent AVF, this did not result in detectable structural cardiac changes in a 1-year follow-up.

In contrast, the studies by Gorgulu, Glowinski and Kurita did not show a significant improvement of LVM, although it's important to notice that their follow up was limited to 3

months. This limited follow up precludes firm conclusions regarding the effects of AVF closure on cardiac dimensions [45–47]. In addition, a large proportion of patients with spontaneously rather than surgically occluded AVF were included in the studies by Gorgulu, Glowinski. Indeed, spontaneous occlusion of AVFs may be an indication of impaired cardiac function at baseline, resulting in a substantial confounding. In general, one should be cautious with interpreting the results of the above-mentioned studies as none of them had a prospective and randomized trial design.

Another possible source of error is the use of the left ventricular mass index, which corrects for weight or body surface area. In most transplantation patients, body weight changes when fluid retention is resolved, lowering body mass, and when fat mass increases after starting steroids, increasing body mass. Since the body surface area is derived from weight, this parameter also changes. These changes may influence the LVMI even if the actual LVM does not change.

Conclusions and future directions

Although several studies indicate a benefit of AVF closure, the current evidence is not conclusive. The remarkable differences in how AVFs are treated in kidney transplantation recipients in different hospitals, regions and countries, clearly demonstrate the lack of consensus amongst clinicians on this topic. No recommendations for routine AVF closure have yet found their way into vascular access or transplant guidelines. Based on the current literature, it is still unclear how to weigh the pros and cons of AVF ligation in these patients. With the continuously improving outcomes of kidney transplantation, the question arises whether this balance will shift further towards benefit of routine closure.

In order to increase the scientific foundation for recommendations regarding vascular access management after transplantation, we intend to initiate a randomized, controlled trial evaluating the effect of closure of asymptomatic high-output AVFs in post-kidney transplantation patients. Patients will only be included if the renal function is adequate and stable. We believe this strategy will result in a low incidence of patients who need to re-initiate hemodialysis after surgical AVF closure. In addition, patients without other reasonable options to create a new AVF will be excluded. Cardiac MRI will be used at baseline and at two years after AVF closure to evaluate cardiac structure and function.

In conclusion, a patent AVF contributes to the persisting LVH after transplantation and could therefore contribute to the observed high risk of cardiovascular disease in kidney transplant

patients. However, more research is needed to determine whether closure of AVFs in asymptomatic patients is indeed beneficial for their cardiovascular health. Such benefit should then be weighed against the increased risk of future vascular access complications in case of a repeated need for hemodialysis.

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