Research Article

Interventional Cardiology

High-output cardiac failure secondary to arteriovenous fistula: a wide-based literature review

Abstract:

Performing arteriovenous fistula (AVF) has become common among patients requiring hemodialysis. Apart from complications like bleeding, stenosis, infection, and thrombosis, even high-output cardiac failure has been a matter of concern for these patients. Sever risk factors like upper arm AVF with brachial artery anastomosis, male sex, and volume expansion has been identified. The pathophysiology contributing to this disease process is the shunting of blood from the high-resistance arterial system into the lower resistance venous system, increasing the venous return and eventually cardiac failure. It is also believed cardiac failure in AVF is also a reflection of the underlying cardiovascular disease. This is diagnosed using an echocardiogram and cardiac biomarkers (brain natriuretic peptide & atrial natriuretic peptide). AVF ligation has been shown to improve symptoms of cardiac failure at the expense of losing vascular access. Current guidance recommends schematic predialysis planning process with the aim of optimizing the route of dialysis for an individual patient's unique psychological, medical and wider social implications.

Keywords: Arteriovenous fistula, haemodialysis, high-output cardiac failure

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Received Date: June 27, 2020, Accepted Date: July 21, 2020, Published Date: July 30, 2020

Abbreviation list:

AVF: Arteriovenous Fistula; RRT: Renal Replacement Therapy; Qa: vascular access flow; HD: haemodialysis; HOCF: High-Output Cardiac Failure; CHF: Congestive Heart Failure; ESRD: End-Stage Renal Disease; CO: Cardiac Output; LVEDV: Left Ventricular End-Diastolic Volume; LVH: Left Ventricular Hypertrophy; Qa-CO: Vascular Access Flow- Cardiac Output; BNP: b-natriuretic peptide; ANP: Atrial Natriuretic Peptide; KT: Kidney Transplant; LVEDD: Left Ventricular End-Diastolic Dimension.

Introduction

The adoption and development of techniques to establish arteriovenous fistula (AVF) as a viable form of renal replacement therapy (RRT), notwithstanding the shortage of kidney transplant, revolutionised the treatment of patients with end stage renal disease [1]. Satisfactory vascular access flow (Qa) is essential for adequate haemodialysis (HD). A low-resistance venous pathway where the AVF is a typically utilised conduit, follows the consideration of balancing pressure and flow to prevent thrombosis and maximise haemodialysis efficiency [1]. Although the development of this form of vascular access rendered haemodialysis a long-term viable treatment method, there are significant considerations that ought to be reconciled for both the patient and nephrologist. Several complications have AVF have been described and these include but are not limited to, bleeding, stenosis, infection and thrombosis [2]. Furthermore, the decision-making process around timing, location and duration of AVF creation is varied in different centres [3].

The haemodynamic effects of AVF construction were first reported in the mid- 20th century [4]. An association between AVF and high-output cardiac failure (HOCF) was described in the 1970s [5] and 36% of dialysis patients have pre-existing heart failure [6]. Furthermore, a significant burden owing to mortality and morbidity in the dialysis population is secondary to cardiovascular disease [7]. Traditional cardiovascular risk factors which often compound patients with chronic kidney disease do not entirely

constitute the burden of cardiovascular disease in this subset of patients. The haemodynamic processes involved in high-output shunting of blood in the pathogenesis of HOCF is unclear. Low Qa would suggest HD dysfunction whereas a high Qa may represent an attributing or predisposing factor for the development of HOCF. Several case series have reported on this complication but there is a paucity of evidence base surrounding mechanisms and natural history [5,8-11]. The normal range of cardiac index is between 2.5 and 4.2 L/min/m^2 and indices above this range increase the risk of HOCF consequential of supraphysiological cardiac output [12].

An important question surrounds whether ligation of AVF leads to improvement in the pathological changes in the cardiovascular system, and resolution of the supraphysiological changes. Hence with respect to potential adverse cardiovascular consequences, the nephrologist is often faced with a clinical dilemma of maintaining a viable method for RRT whilst avoiding potential adverse cardiovascular outcomes. Consequently, there may be an argument for developing standardized screening methods to identify HD patients at highest risk for adverse cardiovascular outcomes due to placement of an AVF. In this review we aim to highlight the relationship between AVF and HOCF with respect to risk factors, pathogenesis, clinical correlates, and report on the dilemmas facing clinicians and patients. We aim to delineate the gap in evidence for this entity to explore potential methods of reducing the overall burden of this disease.

Methods

Literature search strategy

Electronic searches were performed on PubMed, Scopus, Embase and Cochrane databases with no limits placed on dates. Search terms included: pathogenesis, high-output cardiac failure, dialysis, arteriovenous fistula, location, risk factors, prevalence, survival rates, surgical intervention, medical therapy, and mortality. Search terms were charted to MeSH terms and combined using Boolean operations. Papers were selected based on title and abstract. The reference lists of selected papers were reviewed to identify any relevant papers that might be suitable for inclusion in the study. We included case reports and case series. Comments, opinions and editorials were excluded.

Risk factors

Several factors have been linked to the development of HOCF in HD patients. These include location (upper arm AVF with brachial artery anastomosis), male sex and volume expansion [8]. The determination of a precise relationship between AVF and cardiac dysfunction has proven to be problematic owing to the observation that patients with end stage renal disease invariably have a degree of volume overload secondary to salt and water retention [13]. Underlying compounding factors add insult to injury and include pressure load due to arterial sclerosis, increased cardiac output (CO) secondary to anaemia and hypertension [14]. Furthermore, a significant proportion of HD patients may have concomitant structural cardiac disease [15]. Despite these pitfalls, several series describe worsening cardiac function following creation of AVF which could suggest a causative relationship [8-11].

Pathophysiological basis of HOCF

Congestive heart failure (CHF) is known to be associated with ESRD. Pathophysiological changes involving chronic activation of the renin-angiotensin-aldosterone system with consequential salt and water retention contribute to progressive ventricular remodeling and cardiac dysfunction [6]. Indeed, a significant proportion of ESRD patients have established CHF prior to initiation of dialysis and ESRD patients with concomitant CHD renders a worse prognosis. The haemodynamic effects of AVF were studied in patients who developed traumatic AVF and here this was associated with increases in CO [4].

The pioneering studies in the mid-1940s found that patients with large AVFs had elevated CO and that this was diminished with compression of the AVF [4,16]. Later it was recognised that the fistula establishes a low-pressure low-resistance circuit in the context of a high-pressure and high-resistance peripheral arterial system. Hence, shunting of flow from the high-resistance arterial system into the lower resistance venous system corresponds with increased venous return and consequential increased CO [17]. Furthermore, AVF may decrease arterial impendence which may contribute to a reduction in effective circulating volume with a consequential activation of baroreceptors and sympathetic tone. The net effects of such changes result in a supraphysiological CO.

In one study the presence of AVF was associated with cardiac dilation [18]. Renal transplant patients with a functioning AVF had increased left ventricular end-diastolic volumes (LVEDV) (53 vs. 49 mm; $P<0.01$) compared to those without. Several studies have reported on the impact of AVF on echocardiographic indices corresponding to cardiac function [18-25]. Consistent findings involving the increase in contractility and CO occur within short periods following construction of the AVF. In some reports, diastolic filling parameters were impaired and an AVF could correspond to a 15-20% increase in CO [26].

Some authors suggest that cardiac decompensation in ESRD and AVF occurs as a result of underlying cardiovascular disease; although there is emerging evidence that AVF creation may present an independent risk factor for de novo CHF [27]. In an observational study of 562 patients the creation of an AVF was more predictive of the development of decompensated heart failure than a prior history of CHF (OR 9.54 vs. 2.52) [28].

Risk factors that interplay the relationship of AVF and HOCF include the location of an AVF and male sex. As the velocity of blood flow decreases with a smaller calibre vessel, one can postulate that proximal fistulas have higher flow

than distal fistulas. In some analyses, Qa could be increased by a factor of 2 in proximal fistulas (1336 ± 689 vs. 645 ± 332 mL/min) [29]. Furthermore, a proximal AVF closely related to the incidence of new CHF compared to distal AVF (40% vs. 8%) [28]. AVF contribute to LV remodelling by virtue of increased wall mass and diameter, which may be secondary to increases in CO. This relationship may be independent of underlying chronic activation of RAAS, as patients who have undergone renal transplants may have persistent LV hypertrophy in the context of a residual functional AVF. Furthermore, ligation of an AVF post-transplant has associated with a significant regression of LV hypertrophy [30]. Whether this leads to a reduction in adverse cardiovascular events remains to be seen. The sirtuin family proteins, which are histone deacetylases, have been implicated in a wide range of physiological and pathological processes, including aging, stress resistance and inflammation [31]. The endogenous Sirtuin-1 (Sirt 1) gene may have a vital role in the pathogenesis of heart failure, and in animal models, an overexpression of Sirt1 had a protective effect against mitigating oxidative stress [32]. Furthermore, epigenetic alterations of this family of genes may be implicated in cardiovascular disease and diabetes hence could be a potential area for targeted therapeutics [33-35].

Non-invasive techniques for identification and monitoring AVF-induced HOCF

With respect to the deleterious cardiac outcomes observed in the proportion of ESRD patients with AVF, efforts to identify these patients at risk of cardiac decompensation have been undertaken. These broadly involve the use of ultrasound dilution techniques and echocardiography [23,36,37]. Many studies have investigated the impact of AVFs on echocardiographic indices of cardiac function. These demonstrate an increase in in left ventricular end diastolic volumes, stroke volume and CO shortly following AVF creation [7,14,18,21,25,30]. Regarding non-invasive investigations, there is compelling evidence that the development of HOCF in AVF patients without prior cardiac disease may be proportional to Qa [8,38].

Notably Qa may be increased two-fold in proximal vs. distal fistulas and most cases of HOCF were reported in patients with Qa>2 L/min and one analysis reports an average Qa of 1.13-1.72 L/min [8]. Qa can be measured routinely by the non-invasive ultrasound dilution technique. Prior attempts to correlate cardiac failure with Qa have reported conflicting data due in part to the wide variation between individuals. Therefore, the ratio of access flow to cardiac output (Qa:CO) is a proposed method where values >0.3 may associate with increased risk for the development of HOCF [8,39].

However, this finding is yet to be validated in a prospective study. Unfortunately, there is a paucity of evidence around the significance of these parameters to adverse outcomes and mortality. Furthermore, Qa may need to be interpreted in the context of body size. Nevertheless Qa ≥ 603 mL/min when indexed for height did predict the occurrence of heart failure symptoms and correlated with cardiac indices such as left ventricular mass and diastolic dysfunction [40]. Clearly, high-flow AVF with greater increases in LVEDV increase the risk of developing heart failure.

Cardiac biomarkers such as brain natriuretic peptide (BNP) and atrial natriuretic peptide (ANP) are released in response to ventricular and atrial stretch and have a prognostic role in cardiac failure management and assessment but are nonspecific indicators [41]. Elevations of these biomarkers have a role in stratifying the severity of cardiac failure and in monitoring the degree of ventricular dysfunction. In one study, levels of BNP and ANP were increased following the creation of AVF [17] but Qa may not correlate with increases in BNP. A change in LV diastolic filling pattern (specific E-A ratios) occurred in response to AVF, and these ratios were altered in response to a consistent pattern of diastolic dysfunction [25,42,43]. ANP correlated well with elevated CO and volume loading whereas BNP levels were linked with the degree of diastolic dysfunction. Hence AVF may induce diastolic dysfunction via volume expansion secondary to the fistula flow that increases the LV diastolic filling pressure.

Owing to the complex interplay of multiple factors, the decision of predicting the optimal location and type of vascular access anastomosis, presurgical evaluation with surgical experience alone is fraught with problems. Recently authors have suggested computational models which may alleviate these difficulties; a patient-specific haemodynamic model may predict the increase in Qa after AVF construction.

Between a rock and a hard place: considerations for management

HOCF due to AVF creation represents a unique clinical challenge. Although there is an increased prevalence of cardiovascular disease in ESRD population, a significant proportion develops cardiac failure because of AVF creation. Due to the relative scarcity of kidney donors and in view of the disadvantages of peritoneal dialysis, the clinician is left with an interim option of HD. On the one hand, one would endeavour to preserve vascular access whilst minimising and preventing the progression of cardiac failure, though these goals may not be mutually compatible. Closure of an AVF including in patients who have had kidney transplants is a matter of ongoing debate [21,30,44].

Hence the benefit of AVF closure must be balanced with the infrequent but significant complications associated with closure. The decision of AVF closure has polarised the strategies nephrologists use when facing this difficult proposition. On the one hand, some transplant clinicians suggest that AVF closure should not be routinely performed in kidney transplant (KT) patients who have stable renal function. Whereas there are others who suggest that AVF closure may prevent cardiovascular dysfunction. Nevertheless, reduction of flow through ligation of an AVF is appropriate once HOCF is established.

Successful resolution of symptoms of HOCF have been reported by multiple groups following banding or anastomotic revision which preserved the functioning of the AVF[9,45]. AVF ligation has been shown to improve symptoms of cardiac failure at the expense of losing vascular access; hence this method may be appropriate for those who have had successful KT [9-11]. Several groups have investigated the effect of AVF closure in KT patients on echocardiographic indices including LV wall thickness, LV end-diastolic dimension (LVEDD) and LV wall mass index [18,25,30,43]. Spontaneous closure of AVF (thrombosis) may result in improved cardiac function [30] and a similar benefit was observed in cohorts who underwent surgical AVF closure [23,46].

Cardiac ejection fraction had improved in KT patients compared to pre-transplant. These changes were likely attributable to the improved volume status, correction of uraemia and normalization of hemoglobin that are known to occur after successful transplant. However, there was no difference between functioning and non-functioning AVF post-transplant on echocardiographic indices [20] which were also consistent in other cohorts [21,44]. Conflicting reports dominate the literature, and several reports are retrospective designs with heterogeneous patient characteristics, follow-up and endpoints.

Although several studies have reported on the potential deleterious consequences of AVF on cardiac function, with consistent and inconsistent echocardiographic changes, the hypothesis that AVF closure improves cardiovascular-related survival has not been formally tested. Furthermore, due to the paucity of literature and general limitations of retrospective studies and case reports, recommendations are difficult to ascertain. However, with respect to the data available hitherto on AVF and HOCF it may be reasonable to adopt pertinent general principles.

Management guidelines and the need for risk stratification

The European Best Practice guidelines [47] underline that fistulas ought to be sited as distal as possible, with respect to the suggested increased risk of cardiac dysfunction with proximal AVF. Furthermore, when planning HD in ESRD patients, careful consideration of cardiovascular status may be pertinent. In ESRD patients with pre-existing heart failure the risks of HD may outweigh the risks associated with a dialysis catheter. Patients with mild-moderate pre-existing heart failure might be considered for AVF whilst avoiding a proximal location. Whereas patients with moderate heart failure may receive individualised therapy based on the degree of cardiac dysfunction. Furthermore, these patients may also benefit from continued cardiac surveillance with respect to Qa and echocardiographic parameters on a longer-term basis. Unfortunately, patients may present late during their renal disease hence decisions around HD and vascular access are often made promptly.

Current guidance recommends schematic predialysis planning process with the aim of optimising the route of dialysis for an individual patient's unique psychological, medical and wider social implications [48]. Although several challenges including lack of appropriate access for fistula construction, patient age and premorbid functional status are pertinent in the ESRD patient population, the clinician must balance the risks of alternative dialysis routes i.e. peritoneal dialysis [49] against the propensity of AVF to increase the risk of HOCF in a small subset of patients. Hence a screening approach as part of a structured process of patient selection to identify those most at risk Inof such complications may be beneficial. These considerations ought to be individualised in a population already at significant risk of cardiac failure.

Conclusion and future directions

ESRD represents a significant proportion of morbidity and mortality worldwide. AVFs remain a vital management option in individuals unfit for or awaiting KT. Although AVFs are well tolerated by most patients, a subset of this population is unfortunately at risk of HOCF owing to AVF construction. The development and implementation of standardized screening including echocardiographic parameters and evaluating those with pre-existing heart disease, is crucial to identify HD patients at highest risk of HOCF. Computational models to predict Qa following AVF are in development and could be a fundamental part of screening, as they can aid the clinician in establishing the best suited vascular access. Additionally, further evaluation of flow/cardiac output ratio may help better risk stratify and manage higher risk patients.

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