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SM Journal of Nephrology and Kidney Diseases

Review Article

Arterio-Venous Fistula (Avf) in Dialysis Patients: understanding the Pathophysiology behind Arteriovenous Fistulae Non-Maturation

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Article Information

Received date: Mar 16, 2018
Accepted date: Mar 26, 2018
Published date: Apr 02, 2018

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Article DOI 10.36876/smjnkd.1013

Abstract

A well-functioning Arteriovenous Fistula (AVF) has been shown to be the best modality for vascular access in patients with End-Stage Renal Disease (ESRD) that require haemodialysis (HD). A mature AVF has lower incidence of thrombosis and stenosis compared to the other two modalities; Arteriovenous Grafts (AVGs) and Central Venous Catheters (CVCs). That translates into prolonged patency rates and lower risk for infection. However, it has been reported that around 20% - 50% of fistulae fail to mature into a useable access for haemodialysis. Non-maturation remains a major concern with as many as one third of first time created fistulae expected to fail. Intimal hyperplasia induced by altered biomechanical forces plays an integral role in stenosis within an AVF. The aim of this review is to give an analysis of the factors implicated in the process of AVF maturation.

Introduction

A well-functioning Arteriovenousfistula (AVF) has been shown to be the best modality for vascular access in patients with End-Stage Renal Disease (ESRD) undergoing haemodialysis (HD) [1-5]. A mature AVF has lower incidence of thrombosis and stenosis compared to the other two modalities- Arteriovenous Graft (AVG) and Central Venous Catheter (CVC), resulting in prolonged patency rates and a lower risk for infection. With exclusion of fistulae that fail to mature primarily, the cumulative patency from formation to permanent failure is superior to grafts and fewer interventionsare required (angioplasty, stenting, thrombectomy or surgical revision) to maintain patency [6-10]. In addition, fistulae are associated with fewer deaths and hospitalisations [9,10].

However, around 20%-50% of fistulae fail to mature into a useable access for haemodialysis [11-14]. The widely definition used for AVF maturation is the one published in the updated Kidney Disease Outcomes Quality Initiative (K-DOQI) guidelines which define mature fistulae as those placed within 6 millimetres from the skin surface, at least measure 6 millimetres in diameter and finally allow for flow rate of ≥ 600 ml/minute.

The aim of this review is to summarise the current understanding of the various processes implicated in the pathogenesis of AVF non-maturation.

Intimal Hyperplasia

Intimal Hyperplasia (IH) was first described by Noble Prize winning surgeon Alexis Carrel in 1906 when he noticed that few days following a vascular bypass procedure, the stitches placed to make the anastomosis were covered with a "glistening substance similar in appearance to thenormal endothelium" [15]. However, it was not until 1971 when Grondin et al published a paper on "Progressive and late obstruction of an aorto-coronary venous bypass graft" that the link was established between intimal hyperplasia and graft restenosis [16].

Intimal Hyperplasia is the process of cellular proliferation within the inner most layer of the blood vessel. It is defined as the abnormal migration and proliferation of vascular smooth muscle cells provoked by injury, inflammation or stretch with associated deposition of extracellular matrix in the intimal layer of the vein [17-19].

Maturation of AVF depends on variable biomechanical forces induced into the vascular system following the creation of the new fistula. The remodelling process of the arterial limb of the AVF results in dilatation and outward hypertrophic changes of the intimal layer. Remodelling at the venous end can be accompanied by aggressive thickening of the intimal layer resulting in inward

OPEN ACCESS
ISSN: 2576-5450

hypertrophic remodelling, which ultimately can lead to stenosis and failure of the AVF to mature. Marked increase in the flow rate and the accompanying abnormal distribution in Wall Shear Stress (WSS) are believed to contribute significantly to intimal hyperplasia and ultimately AVF non-maturation [20-22]. In the absence of significant injury, wall stretch can lead to a less remarkable smooth muscle cells proliferation [18,23].

Inflammatory Markers

ESRD patients are believed to be predisposed to more inflammatory changes within the vascular endothelial layer, even before the formation of newAVFs [24-26]. Wali et reported wall thickening and IH with loss of the internal lamina layer in 45% of AVFs created for HD access in ESRD patients,loss of the endothelial cell layer in 30%, inflammatory cell infiltration in 25%, mural calcifications in 3 patients (15%) and telangiectasia in 10% of their patients [24]. Liang et al induced Chronic Kidney Disease (CKD) into a group of mice, then they anastomosed the common carotid artery to the internal jugular vein. Their results showed that mice with CKD had 45% more neointima formation than controls. Also CKD mice had more inflammatory cells and showed increased endothelial barrier dysfunction [26].

Wasse et al suggested that IH pre-exist in patients with CKD before creation of the fistula. They also observed that inflammation and oxidation markers were present within the veins at least one year before commencement of haemodialysis [27]. Conversely, Allon et al in a series of 50 patients with CKD undergoing AVF formation found that intimal hyperplasia was not present at baseline, but rather developed later in non-maturing fistulae. They also found that medial fibrosis and micro calcifications are common in arteries used for AVF [28].

Recent studies have shown that Matrix Metalloproteinases (MMPs) are important in the process of AVF maturation [29-31]. MMPs belong to a group of zinc-dependent proteases capable of degrading Extracellular Matrix (ECM) proteins [32,33]. In particular, MMP-2 is expressed by a variety of cell types and is activated by the membrane-bound type-1 MMP (MT1- MMP) and is inhibited by tissue inhibitor metalloproteinases type 2 (TIMP-2). MMP-9 is also expressed by a variety of cell types and is inhibited by TIMP type 4 (TIMP-4) [34]. Because increased expression of MMP-2 and MMP-9 has been found in the outflow vein tissue after AVF construction [30,35], MMP expression in the patient serum at the time of the initial surgery may serve as an important biomarker of intimal hyperplasia and can possibly predict maturation outcomes for the newly created fistulae [31].

Owens et al in a prospective study of lower extremity bypass surgery using autogenous vein, used Enzyme-Linked Immunosorbent Assay (ELISA) to measure High-Sensitivity C-Reactive Protein (hsCRP) and the adipokines resistin and High-Molecular Weight Adiponectin (HMWA). They concluded that serum biomarkers of insulin resistance and inflammation might be predictive of clinical outcomes following lower extremity bypass [36]. So far, no studies have investigated the use of those markers in predicting maturation outcomes from new fistulae.

Tsapenko et al in 2012 examined the relationship between increased anion production and AVF stenosis in a rat model. They concluded that the increased production of superoxide anion could be due to decreased levels of scavenging promoted by Super-Oxide Dismutase (SOD) combined at the same time with increased generation of superoxide anions by uncoupled Nitric Oxide Synthase. This increase in superoxide anion production promotes juxta-anastomotic stenosis of AVFs and ultimately result in nonmaturation [37]. Recently, short term oxygen supplementation following creation of fistulaehas been shown to reduce both intimal hyperplasia and smooth muscle cell proliferation in a group of rabbits which were subjected to short term 30% oxygen therapy for forty two days [38].

Haemodynamic

AVF maturation is an active process of vascular remodelling that occurs in response to the altered biomechanical forces induced in the vascular system by the creation of the fistula. In order for anAVF to mature into a functioning fistula, sufficient blood flow needs to be obtained through the AVF circuit to insure successful HD repeatedly. This depends on pressure gradient and total resistance of the circuit. Mean arterial blood flow needs to be increased several fold to sustain sufficient blood flow-typically around 500 ml/min. This leads to increase in cardiac output to maintain blood pressure and prevent loss of perfusion to other vascular beds, as well asdilatation of the artery to insure adequate perfusion distal to the fistula and reduces the risk of steal syndrome [39]. However, several factors should be considered when measuring expected arterial dilatation; distal arterial flow occurs in about 75% of forearm AVF and contributes an average of 25% of venous flow of the fistula [40]. Moreover, blood flow is pulsatile rather than steady, and the average pressure gradient increases following creation of AVF, as well as the decrease in blood viscosity with increasing flow rate limiting Wall Shear Stress (WSS). All of that willlimit the expected arterial dilatation to maintain required blood flow through the circuit as per Pouseuille's law [39,41,42].

Arterial remodelling is a response to pressure and flowchanges, and is controlled by the endothelium, sensing Wall Shear Stress (WSS) changes. The changes are caused by creating opposing forces within the vessel wall as a result of deformation that occurs in three direction; longitudinal, circumferential and radial creating both normal tensile stress and shear stress resulting in nine static forces (three static deformation and six static stresses) that can affect vascular dilatation and remodelling [39]. The flow mediated remodelling affects both artery and vein of an AVF. Arterial dilatation has been shown to lower WSS to baseline levels [21,43]. Fashioning AVF in a way that will result in decreased WSS overtime is thought to positively impact on maturation [21]. Venous response to WSS is variable, as some studies have shown venous dilatation, while others reported reduction in luminal area with similar levels of WSS [21,29,44-46]. Dobrin et al examined mechanical and histological changes in femoral vein applied as a graft to bypass ligated femoral artery in dog models. They found that intimal hyperplasia correlated with low WSS, while medial thickening correlated with circumferential deformation, and concluded that intimal hyperplasia and medial thickening are different responses to different stimuliinfluencing vascular dilatation and remodelling [47]. Ben Driss et al examined the effects of chronic increase in blood flow on arterial wall remodelling in Aortocaval Fistulae in rats. They found that shear stress promotes expansive remodelling in relation to flow dependant vasodilatation, whereas medial thickening was related to increase in tensile stress [48].

Since the remodelling process is mediated by endothelial response to flow and pressure, loss of endothelial cells impairs remodelling and prevent WSS from dropping to baseline levels following creation of AVF [39]. The physiology of vascular remodelling and reconstruction mediated by endothelial cell response to stress is not fully understood [22,39]. It is believed that an increase in shear stress results in increased production of Nitric Oxide (NO) among other endothelial derived vasodilators in attempt to normalize WSS following fistula formation [49,50]. Miller VM. et al in 1992 showed that chronic increase in blood flow may induce endothelial cellsto either inhibit production of endothelin or promote its depletion,and at the same time enhances the smooth muscle cells response to its contractile effects [49]. However, further arterial dilatation is required to lower WSS than the endothelial-mediated response in the acute phase by generation of NO. This fragmentation of vascular elastic lamina is mediated by MMPs whichis also induced by changes in blood flow and pressure. The activation of MMPs can be inhibited by Doxycycline treatment which inhibits Nitric Oxide Synthaseproduction, hence MMPs are likely controlled by levels of Nitric Oxide [51]. Castier et al examined the effect of homozygous targeted deletion of endothelial Nitric Oxide Synthase (NOS) on MMPs in mice. They found that the increase in MMPs activity and arterial dilatation were both lost. Their findings further underline the role of NOS in regulating MMPs activity and flow induced vascular remodelling [52].

Several studies showed the association between anatomical configuration of AVF and WSS, and consequently the pattern of luminal stenosis as a result of intimal hyperplasia and media thickening and fibrosis. Using computer modelling to alter anatomic configuration has been suggested to reduce WSS deformation and hence improve fistula successful maturation rates [20,21,53].

Remote Ischaemic Preconditioning

To date, no randomised controlled studies have looked into the effect of Remote Ischaemic Preconditioning (RIPC) on the maturation process of fistulae in patients with end stage renal disease. RIPC is a phenomenon that occurs in mammals where by brief periods of ischaemia in a remote distant tissue followed by reperfusion causes subsequent resistance to ischaemia in different organs to a much prolonged period. It has been so far mostly studied in relation to the coronary circulation of the heart. Murry et alspeculated that brief periods of ischaemia slowed the rate of ATP depletion during subsequent ischaemic episodes, and proposed that multiple brief ischemic episodes might actually protect the heart from a subsequent sustained ischemic insult. In their study, repeated ischemia reducedthe infarct size by 25% of that seen in the control group ($p <$ than .001) [54]. RIPC is expected to lower inflammatory markers that are associated with higher risk of intimal hyperplasia and subsequently, graft failure.

Recent studies showed promising results of RIPC when applied to patients undergoing a number of vascular procedures such as elective abdominal aortic aneurysm repair, angioplasty, coronary artery bypass graft surgery, carotid endartrectomy and as a conservative treatment option to improve claudication symptoms in patients with peripheral vascular diseases [55-60].

Discussion

AVF has been proven to be the preferred type of vascular access for haemodialysis patients because of the significantly lower complications rate when compared to AVGs and CVCs. Graftsare inferior to fistulae in terms of cumulativeaccess survival, cost effectiveness and need of salvage procedures. On the other hand, CVCs are high risk for sepsis and result in significant morbidity and mortality. The use of CVCs for HD should be discouraged based on the significantly higher risk of complications. Nonetheless, it is estimated according to the DOPPS II study that 46% of European and 66% of American patients start haemodialysis via a CVC [61]. Reasons for this include late diagnosis with ESRD, late referral to nephrologists, lack of time to wait for AVF to mature, vascular disease, diabetic disease, and preference nephrologists and nursing staff in dialysis centres [5]. Educating primary physicians and general practitioners in making early diagnosis of patients with Chronic Kidney Disease (CKD) coupled with prompt referral to nephrologists was shown to improve the number of patients going to haemodialysis with mature AVF for access [62-64].

However, maturation remains a major concern with as many as one third of first time created fistulae expected to fail. Maturation of AVFs is a complex active process with many variables involved. Haemodynamics of blood flow through the circuit of an AVF seems to be the single most contributing factor in the process of vascular remodelling associated with a newly created fistula. Altered biomechanical forces induces a cascade of reactions induced by the vascular endothelial layer in response to wall shear stress and changes in Haemodynamics. The endothelial response triggers pathways mostly mediated by release of nitric oxide that can result in increased intimal hyperplasia in particular in the venous end of the fistula, as well as increased vascular medial thickening and fibrosis. Arterial dilatation is mediated by increased production of nitric oxide; however, matrix metalloproteinases play an important role in the further arterial dilatation required to normalize WSS in a fistula circuit.

The use of inflammatory markers can prove useful in prediction fistula maturation outcomes, as the process seems to be closely mediated and influenced by inflammatory pathways that are either in existence at the time of fistula formation or are induced by the creation of the new AVF. Also, pharmacological agents that are can alter responses to the inflammatory mediators associated with non-maturation once fully understood can lead to increased numbers of mature AVFs.

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Citation: Bashar K, Bashar H, Ahmed K and Walsh SR. Arterio-Venous Fistula (Avf) in Dialysis Patients: understanding the Pathophysiology behind Arteriovenous Fistulae Non-Maturation. J Nephrol Kidney Dis. 2018; 2(1): 1013.
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