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Chapter 9

Role of Collateral Vein Occlusion in Autologous Dysfunctional Hemodialysis Fistulas

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1. Introduction

An autologous arterial-venous fistula is the most desirable vascular access for end-stage renal disease (ESRD) patients who are dependent on hemodialysis. [1] Autologous arterial-venous fistulas have a lower risk of thrombosis and infection, and have shown to have a longer patency as well. [1] Kidney Dialysis Outline and Quality Initiative (KDOQI) guidelines mandate that 50% of all new hemodialysis accesses and 40% of all hemodialysis accesses be autologous arterial-venous fistulas.

Unfortunately, not all fistulas mature enough to be used for hemodialysis. The failure rate of a fistula to mature in the earlier studies ranged from 10%-55%. [2,3] As the mean survival age of hemodialysis patients has increased, surgeons are creating fistulas in elderly patients with less than ideal vessels, thereby increasing the rate of non-maturing fistulas and reducing their long-term patency. The later studies have shown a significant increase in the number of native fistulas failing to reach maturity without intervention. [4-9]

The Fistula First initiative has made a concerted effort to increase the number of patients on hemodialysis to have autologous fistulas rather than synthetic grafts or tunneled hemodialysis catheters. An increase in the number of native fistulas has also created an unintended consequence of a higher number of these fistulas not reaching maturation. [10] Aggressive early intervention in a non-maturing autologous arterial-venous fistula results in a high percentage of these fistulas reaching maturation. [9]

The two most common causes of non-maturing and mal-functioning autologous fistulas are anastomotic and juxta-anastomotic stenoses reducing the blood flow through the outflow vein and collateral veins which drain part of the blood away from the main outflow vein, thereby making the volume of blood flow in the main outflow vein suboptimal for hemodialysis. [11]

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Anastomotic or juxta-anastomotic stenosis is the most common cause of a fistula failing to mature. The stenosis most likely occurs because of operative trauma. In case of transposed vein fistulas, the loss of vasa vasorum in the mobilized portion of the vein may also contribute to causation of stenosis. Secondly, there is element of neo-intimal hyperplasia. Because of the large pressure gradient between arterial and venous circulation, the blood accelerates while passing from the artery to the vein causing turbulent flow that is considered responsible for endothelial damage as a result of micro trauma.

The role of preventing blood flow through the competing accessory or collateral veins by embolization or ligation is questionable. [1] There are no prospective studies which evaluate the effect of embolization or ligation of the collaterals on fistula maturation or patency.

In this chapter, the approach to treating non-maturing fistulas will be discussed as it applies to the embolization of competing collaterals.

2. Rationale

Traditional surgical practice has been to abandon autologous arteriovenous fistulas that fail to mature in three to four months in favor of a new fistula, or a graft. Recently however, there has been a drive to intervene aggressively on the non-maturing fistulas to assist them to mature.

With advances in medicine and better healthcare delivery, life spans of patients on hemodialysis are increasing and these patients are outliving their hemodialysis access sites. There are only a finite number of sites in a patient suitable for access creation. As these fistulas fail and are abandoned in favor of new ones, the patients eventually run out of options for access sites. It is therefore imperative for physicians responsible for maintaining these accesses to treat these fistulas aggressively to maintain their patency for as long as possible in order to maximize the patients' life expectancy.

The role of angioplasty of anastomotic or juxta-anastomotic stenosis in assisting non-maturing fistulas to reach maturity is well documented. [1,2] Tessitore et al. demonstrated in their landmark prospective study that even in the absence of hemodynamic blood flow abnormality, preemptive angioplasty of the anastomotic and juxta-anastomotic stenosis leads to a four-fold increase in the median survival of the fistula and almost a three-fold decrease in the risk of their failure.

The role of collateral vein embolization, however, is less well established. Intuitively, and hemodynamically, it makes sense to maximize blood flow through a single outflow vein, thereby providing adequate flow for hemodialysis. If there are competing parallel outflow veins for which there is a single inflow artery of a finite size, the flow through individual venous channel will decrease despite the overall decrease in the resistance offered by multiple parallel channels. The parallel outflow veins, which have different calibers, lengths, tortuosity, and flow patterns, further complicate the flow dynamics (Figure 1).

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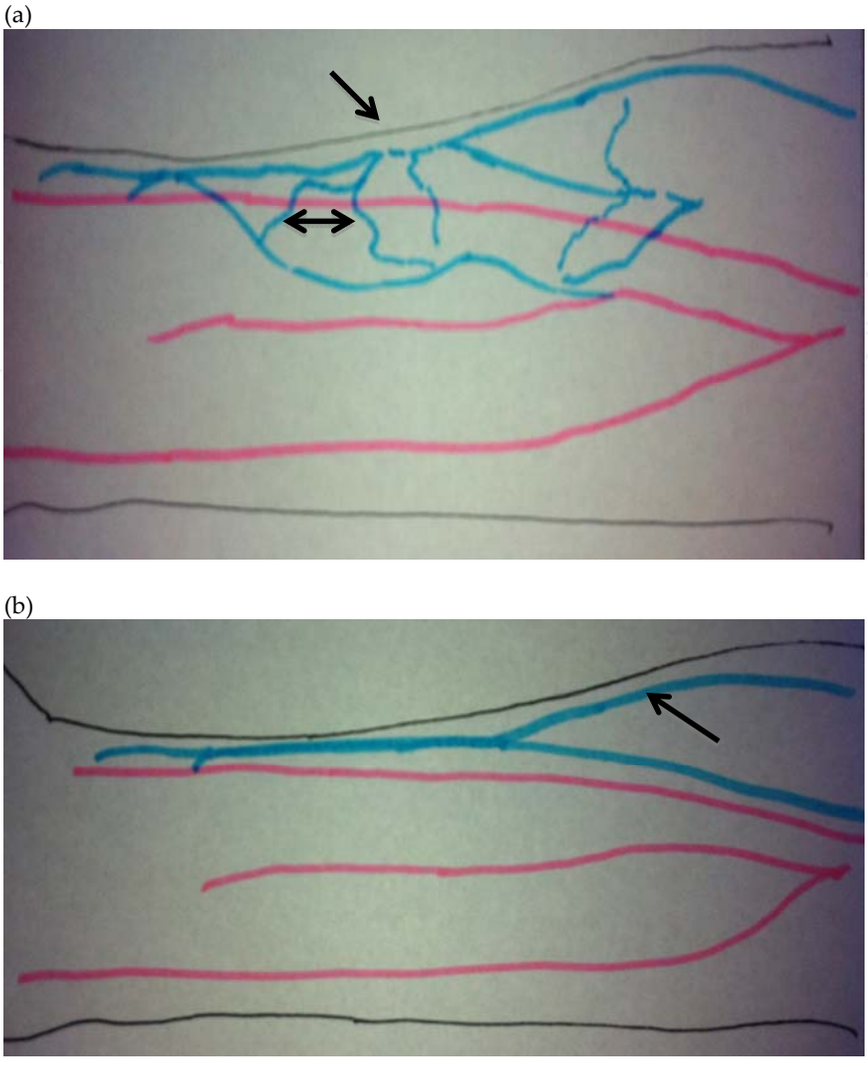


Figure 1. Demonstrates schematic diagram of a radial artery to cephalic venous fistula demonstrating multiple collaterals (double headed arrow in figure 1 a) due to a tight stenosis in the cephalic outflow vein (single headed arrow in figure 1 a). After treatment of the culprit stenosis, there is single inline flow in the main cephalic vein (single headed arrow in figure 1 b) and no further filling of the collaterals.

Arterial inflow increases in response to decreased resistance in the venous outflow. In most cases, arterial inflow is insufficient to sustain adequate outflow through each collateral channel to for adequate hemodialysis.

3. Technique

It is best to approach a non-maturing autologous arterial-venous fistula, by obtaining a detailed history of hemodialysis access, in particular, as it pertains to the access in question. This should be followed by a thorough physical examination of the fistula. The physical exam can reveal much of what one might expect on fistulogram. Robbin et al. have shown that experienced

operators on physical examination alone can predict with 80% accuracy if the fistula will mature. [12]

Physical examination can reveal the cause of the underlying anatomical abnormality quite accurately. [13] In a normally developed fistula, there is a continuous thrill from the anastomosis all the way centrally along the outflow vein. The thrill gradually decreases in intensity from the anastomosis centrally. When patients have a critical stenosis in the outflow vein, the physical examination often reveals a strong pulse peripheral to the stenosis and a weak thrill centrally in the outflow vein. The pulse often has a water hammer character. [14] In the absence of a stenosis, the pulse is soft and easily compressible. The presence of collaterals can also be determined by physical examination. Compression of the outflow vein abolishes the thrill at the anastomotic site and converts it into a strong pulse. But if competing collaterals are present, the thrill persists even after compression of the main outflow vein, as blood continues to flow through the collateral. In addition, the character of the thrill changes central to the point of takeoff of the collateral veins. The thrill becomes significantly weaker and difficult to palpate.

The next step in approaching the non-maturing autologous hemodialysis fistula is to perform an ultrasound. This helps to confirm the findings of the physical examination. It also provides an idea as to the best site to puncture the fistula for intervention. Not infrequently, the arteriovenous anastomosis is difficult to identify because of severe anastomotic stenosis or because of a number of collateral venous channels close to the anastomosis stealing the blood from the main outflow. In these cases a reflux fistulogram is non-diagnostic as all the collateral veins fill up without correctly identifying the arteriovenous anastomosis. In these cases, it becomes imperative to puncture the brachial artery in the mid upper arm and perform an angiogram to identify the arterial-venous anastomosis. (Figure 2) The author restricts brachial artery access to a 3F inner dilator of the micro-puncture set. The arteriogram only serves the purpose of identifying the arteriovenous anastomosis. A retrograde puncture of the outflow vein is also needed in these cases, as all interventions are performed from the retrograde venous access in order to avoid a large caliber hole in the brachial artery.

The author initially identifies all the areas of stenosis and competing collateral veins by initial fistulogram. The stenosis responsible for non-maturation of the fistula is then treated with an appropriate size balloon angioplasty. Once the anastomosis is treated, the a fistulogram is performed with the catheter placed in the inflow artery to identify the result of angioplasty and whether the collaterals still fill. It is imperative that the fistulogram both before and after angioplasty of the stenotic lesion be performed with the catheter tip in the inflow artery, and not by means of a reflux fistulogram by occluding the outflow vein. If the fistulogram is performed using the reflux technique, the collaterals are likely to appear more prominent than they actually are.

The fistulogram is then reviewed in conjunction with the clinical exam. If the post angioplasty fistulogram and the clinical exam indicate that there has been no significant decrease in the filling of the collaterals, occlusion of the blood flow through the collaterals is indicated. (Figure 3) The occlusion of the blood flow in the collaterals is performed either by embolization or surgical ligation of the vein.

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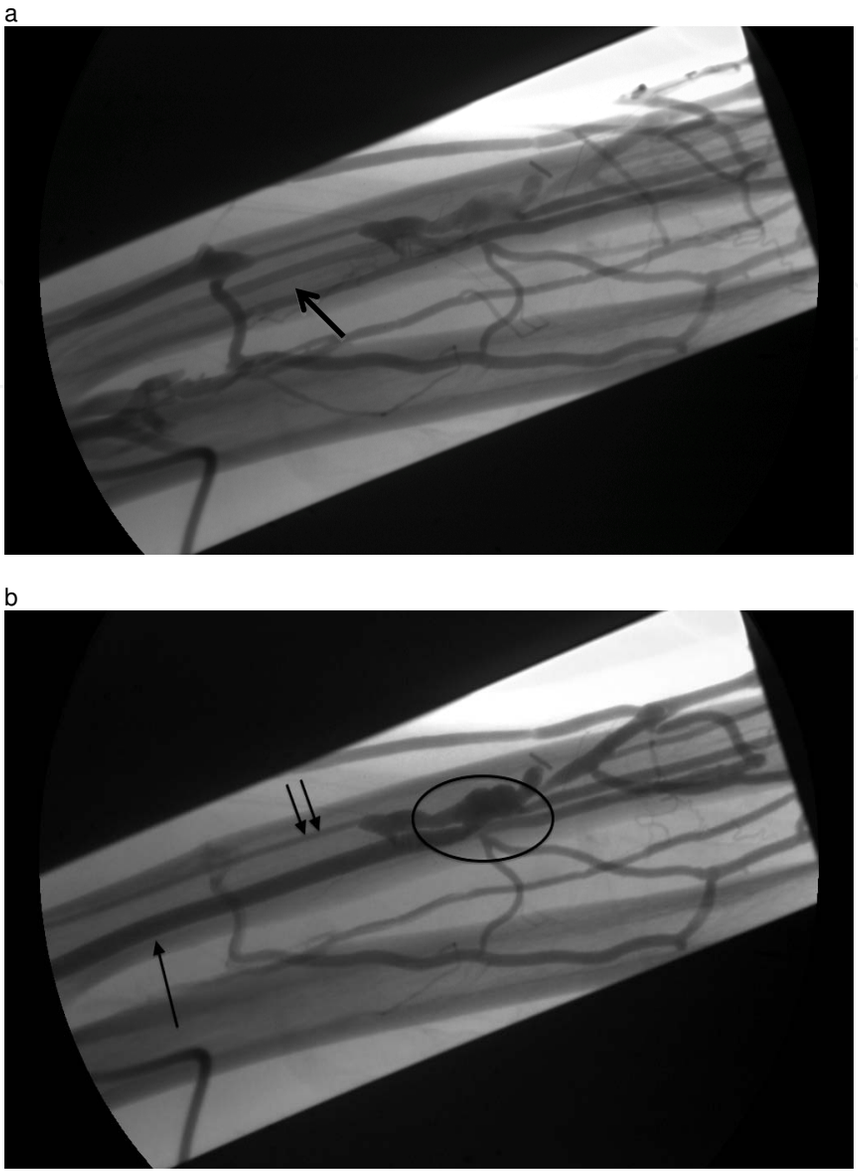


Figure 2. Demonstrates reflux injection of contrast through the cephalic outflow vein in the forearm (single arrow in figure 2 a), which fails to demonstrate the arteriovenous anastomosis. The injection however, fills multiple collaterals. The reason for this occurrence is that reflux injection of contrast encounters un-opacified blood flowing in through the artery and because of the stenosis; the injection pressure is generally unable to get reflux into the artery. There is greater opacification of collaterals for same reason. Figure 2 b shows injection through the brachial artery (single long arrow in figure 2 b), which clearly demonstrates the arteriovenous anastomosis (oval in figure 2 b) with retrograde filling of multiple collaterals due to long segment stenosis of the cephalic outflow vein (double arrow in figure 2 b)

For the collateral veins that are visible on the skin surface, ligation is possible either using a percutaneous or cut down technique. The author performs a percutaneous ligation of the vein using ultrasound guidance. A curved needle is passed deep to the vein under ultrasound guidance while the vein is being visualized in cross-section. A return pass is made superficial to the vein but deep to the skin exiting the needle close to the initial entry site thus placing a purse-string suture around the vein.

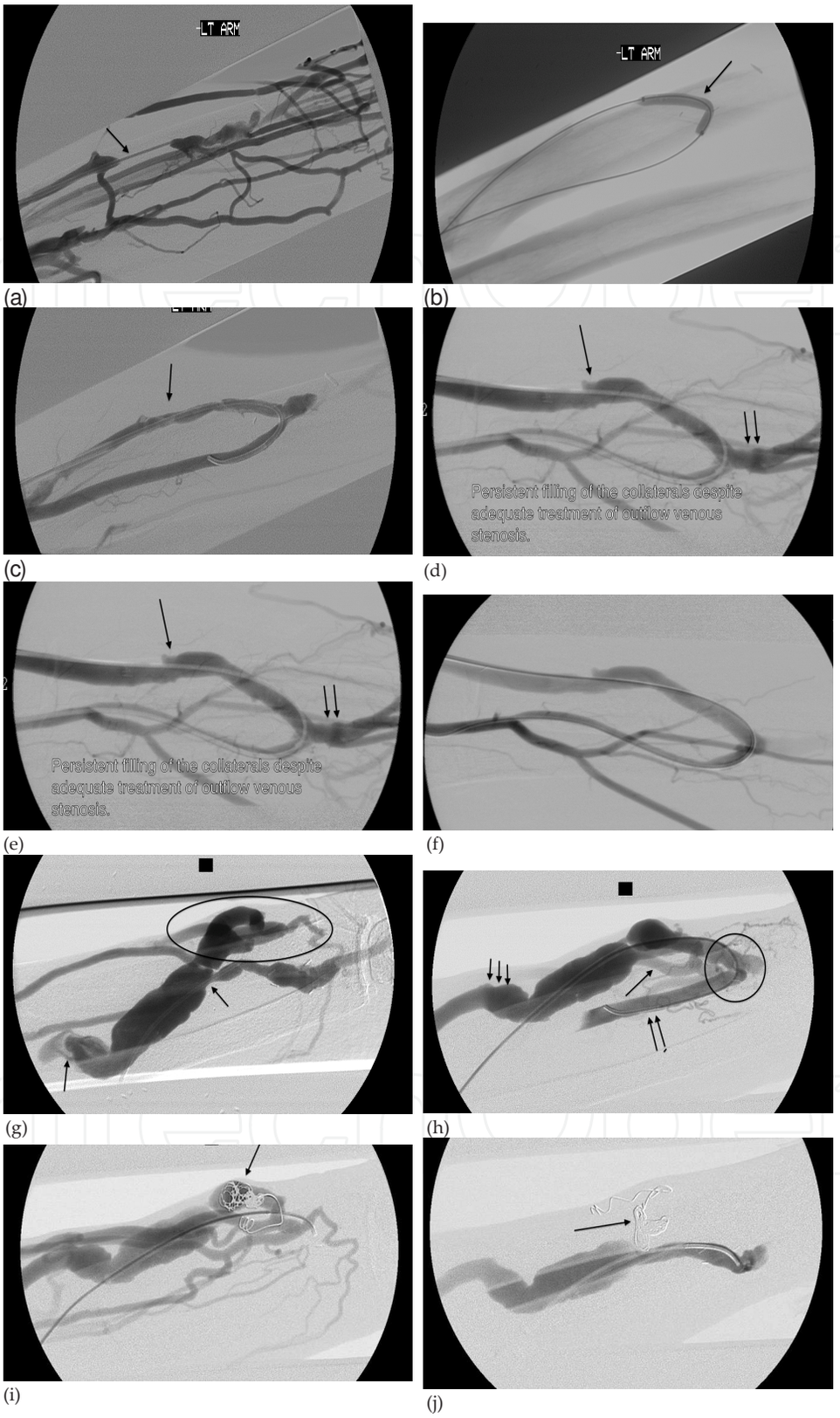


Figure 3. Reflux fistulogram on the same patient as in figure 2 demonstrates filling of multiple collaterals due to a long segment juxta-anastomotic stenosis (single arrow in figure 3 a). This lesion was successfully treated with angio-

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plasty (single arrow in figure 3 b). Post angioplasty fistulogram showed no further filling of collaterals with inline flow through a single outflow vein, (single arrow in figure 3 c) which is significantly larger in caliber. In this case, the collateral veins were not embolized, as they did not fill in post angioplasty fistulogram. The second case demonstrated in figure 3 shows persistent filling of the competing veins in a patient who has a side-to-side radial artery to cephalic vein anastomosis. There is simultaneous filling of cephalic vein central and peripheral to the anastomosis (single arrow and double arrow respectively as seen in figure 3 d). In this case, the cephalic vein peripheral to the anastomosis was clearly visible on the skin, therefore, was ligated percutaneously using ultrasound guidance technique as described in the text. Follow-up fistulogram showed no further flow in the cephalic outflow vein peripheral to the anastomosis, and all the blood flowing centrally in the cephalic vein (figure 3 e) Third case shown in figure 3 demonstrates filling of multiple collaterals (oval in figure 3 f) due to two culprit stenoses (two separate single arrows in figure 3 f). Following successful treatment of these lesions with angioplasty, (triple arrows in figure 3 g) there was persistent filling of the collaterals (single arrow in figure 3 f). Since the collaterals were not clearly visible on the skin, they were treated with embolization using the technique described in the text (single arrow in figure 3 h). Post embolization completion fistulogram demonstrated inline flow in the main cephalic outflow vein without filling of the collaterals. Coils are seen occupying non-filling collaterals (figure 3 j)

If the competing collateral vein is not visible on the skin, it is embolized using regular 0.35 inch platform coils. The coils are oversized by about 20%-30%. The author's coil of preference is Tornado coil (large end first), which unfortunately is a special order item from Cook, Inc. (Cook Bloomington IN). Nester coil (Cook Bloomington IN) is also good; however, the Nester coil may prove more challenging to deploy for inexperienced operators. When fully deployed in the vessel, the Nester coil occupy a longer segment of the vein compared to a Tornado coil of the same size vein and coil combination. The collateral veins often have a short stump off the main outflow vein. In these cases precise coil deployment is important. Nester coils, on occasion, can be pushed too far into the collaterals, thereby blocking the blood flow in otherwise unintended veins. Additionally, because Nester coils occupy a longer segment of the vein, they tend to push the catheter tip back, if there is not enough purchase of the catheter tip within the collateral, which results in part of the coil protruding into the main outflow vein. In such a case, the coil will have to be retrieved by snare. The advantage of "large-end-first" Tornado coils over the "short-end-first" coils is that their deployment is more precise. The large end of the Tornado coil encounters the vessel wall immediately after coming out of the catheter tip, anchoring the coil to the vein wall. On the other hand, the short end of the "short-end-first" Tornado coils tends to float away from the catheter tip before the large end engages with the vessel wall to fixate the coil in the vessel.

4. Discussion

A significant number of native arteriovenous fistulas fail to mature [3-10]. Traditionally, the surgeons abandoned these fistulas in favor of new fistulas or synthetic grafts.

There are only a finite number of sites in the human body to create hemodialysis fistulas. All hemodialysis accesses fail at some point; as such, it is imperative for the interventionalist involved in maintaining the fistula to do everything possible within reason to keep each hemodialysis access patent for as long as possible. Keeping this in mind, interventionalists are now addressing non-maturing arteriovenous fistulas much more aggressively. [5-10]

Treatment of a native arteriovenous fistula entails angioplasty of arteriovenous anastomosis or juxta-anastomotic stenoses. [1,5-12] Although the role of angioplasty of swing point and

juxtaanastomotic stenosis is quite clear, the role of collateral vein embolization or ligation remains controversial.

Maturation of the fistula involves adaptation of the vein walls to allow repeated punctures and increase in the blood pressure and flow. When the arteriovenous fistula is created, the high-pressure arterial blood is shunted into low-pressure veins. The thicker walls of the arteries can sustain the high arterial pressure, but the thin wall veins require some time to adapt to this high pressure. In response to the high arterial pressure transmitted to the thin wall veins, the veins respond by progressive thickening of the muscular layer.

There are two reasons for anastomotic or juxta-anastomotic stenosis. One is trauma due to surgical manipulation and possibly some loss of vasa vasorum in the case of mobilized veins, and the second is neo-intimal hyperplasia caused by turbulent blood flow. The laminar blood flow in the artery becomes turbulent when the high arterial pressure is transmitted to the low-pressure veins because of increased pressure gradient.

The blood flow through the fistula increases quite early after creation. [15] Yerde et. al. showed that the flow through the fistula reaches its maximum flow within 48 hours of creation. Thereon, the flow may reduce if anastomotic stenosis occurs. Wong et al. [16] in their publication showed that not only did the flow through the fistula increase, but the diameter of the outflow vein also increased in all patients after the initial fistula creation during the first two weeks. However, in the subset of patients in whom the fistula did not lead to maturity, the flow through the fistula and the diameter of the outflow vein subsequently decreased. The authors also demonstrated that the size of the vein did not correlate closely to the long-term patency of the fistula. Additionally, Wong et al suggested that the flows measured 24 hours after fistula creation are a more reliable predictor of the patency than the intraoperative flows because of inevitable spasm of the veins due to their handling during surgery. The authors also demonstrated that normal side branches were identified in a majority of the patients, and they were numerous in a minority of the patients.

Robbin et al. describe the ultrasonographic characteristics of the maturing and non-maturing fistulas in their article. [12] They suggest that the outflow veins that do not acquire a diameter of at least 4 mm within 16 weeks of fistula creation and that do not have flow greater than 500 ml/min are less likely to reach maturation. The authors also suggest that an experienced hemodialysis nurse is able to predict the likelihood of a fistula reaching maturity successfully in 80% of cases.

Although the practice of ligation or embolization is quite common among interventional radiologists, interventional nephrologists, and vascular surgeons, there is a dearth of evidence in favor of, or against this practice. Although intuitively and hemodynamically it makes sense to promote higher volume of blood flow through a single outflow vein, one can also argue that leaving the collaterals patent may be beneficial as an alternative hemodialysis access if the main outflow vein becomes unusable at some point. [17] This argument may be valid if there is enough blood flowing through each of the outflow channels to sustain adequate hemodialysis or if there is adequate blood flow through the main hemodialysis outflow vein for adequate

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hemodialysis despite the presence of competing collaterals. It has been shown that a blood flow of at least 350–500 cc/min is the minimum required for adequate hemodialysis.

Unfortunately, the arterial inflow in many hemodialysis patients is not enough to sustain such levels. Many of these patients are diabetic with severe diabetic vasculopathy compromising arterial inflow. In a study published by Janicki et. al., authors published results in seven patients in whom, they ligated the collateral veins and in whom, the blood flow increased from 260–370 ml/min to over 700 ml/min. [18] Although the study included only seven patients and was not randomized, it does support the notion that ligation or embolization of the collateral veins enhances blood flow through the main inline outflow venous channel and therefore is more likely to produce blood flows required for adequate hemodialysis.

It has also been argued that competing collaterals develop secondary to complete or partial blockage in the main inline outflow vein. [6,7] The authors of this article argue that if the culprit stenosis is adequately treated, embolization or ligation of the competing collateral is never required or even desirable. Turmel-Rodregues et al. claimed that the competing collaterals develop in response to a central stenosis, creating back-pressure forcing development of collaterals. The authors argue that treatment of these veins is neither indicated nor desirable, as angioplasty of the offending lesion will essentially alleviate the problem. Normal venous anatomy, however, indicates that the cephalic as well as basilic veins, prior to creation of fistulas contain side branches. It is not a routine practice of surgeons creating arteriovenous fistulas to debranch the outflow veins. It is therefore evident that a stenotic lesion central to the side branch does not cause these veins; however, if such stenotic lesion is present, it may exaggerate the blood flow through the side branch. In their article, Turmel-Rodrigues et. al. argue that embolization of the collaterals is never indicated, because collateral channels are normal venous branches which become pronounced due to venous hypertension caused central stenosis. Once the stenosis is adequately treated, the collaterals do not fill. [6] The authors also argue that by not adequately dilating the stenosis and instead ligating the collateral, the operator can make the situation worse, leading to fistula thrombosis.

Tessitore et. al. demonstrated a role of prophylactic angioplasty in otherwise functioning virgin native arteriovenous fistulas in a prospective randomized study, demonstrating improved long term patency and reduced morbidity in the patients assigned to the angioplasty arm. [19] The authors showed that preemptive angioplasty of non-hemodynamically significant stenosis can lead to four-fold increase in the patency of the fistula and almost three fold decrease in the likely hood of their failure.

5. Conclusion

In the author's opinion, embolizing and or ligating collateral channels is effective in assisting non-maturating fistula to get to maturation. It is however, difficult to make that claim convincingly without a prospective randomized study. To the best of the authors' knowledge, there has been no prospective randomized study evaluating the effect of ligation or embolization of the collaterals in non-maturing fistulas with or without angioplasty of the stenotic

lesion. Such a prospective study is needed to further study the role of collateral vein ligation or embolization in non-maturing hemodialysis fistulas.

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