Chapter

Early Detection and Endovascular Intervention to Correct Dialysis Vascular Access Malfunction

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Abstract

Endovascular intervention in hemodialysis vascular access is among the most frequent interventions performed in an angiography suite. Vascular stenosis is the most prevalent lesion causing vascular access malfunction. Vascular access pathology and the outcomes in response to endovascular treatment are quite different from the arterial territory. Treatment strategy must be integrated, multidisciplinary, and with a long-term perspective, as recurrence rates of malfunction are quite common. We will detail our experience managing an extremely busy vascular access center serving a population of 4000 dialysis patients, performing all endovascular techniques in close coordination with the surgical team.

Keywords: hemodialysis, vascular access, angioplasty

1. Introduction

Endovascular interventions have substituted surgical repair as the primary treatment of failing or thrombosed vascular access (VA). Endovascular and surgical techniques, however, are complementary. Optimizing endovascular interventions of VA malfunction is a crucial component for a successful vascular access program. The identification and early treatment of stenosis are essential to prevent access thrombosis and ultimate failure.

Despite recent advances in endovascular techniques and devices, angioplasty continues to be the primary method for the treatment of access-related stenosis. Not all stenosis needs to be treated. When timely applied, angioplasty is a fast, easy, and safe procedure that can extend the patency of a hemodialysis graft or fistula.

The *early detection and endovascular intervention to correct dialysis vascular access malfunction* are reviewed in this chapter, describing the authors' experience in a highly active Vascular Access Center in Lisbon, integrated in a large outpatient dialysis network. We will cover the following topics: (1) vascular access options and its selection, (2) vascular access morbidity and complications, (3) vascular access malfunction detection, (4) endovascular interventions to correct dialysis vascular access malfunction, and (5) endovascular intervention outcomes.

2. Vascular access options and its selection

The VA constitutes the interface between chronic kidney disease (CKD) patient and machine (the dialysis monitor); its function is a key factor that affects most

dialysis treatment quality indicators, such as dialysis dose and adequacy (Kt/V), substitution volume during hemodiafiltration, operating costs, and vital prognosis of the dialysis patient.

In this chapter, we deal only with long-term arteriovenous accesses:

A. The native AV fistula (nAVF), usually result from an end-to-side anastomosis of a vein to an artery, either at the wrist (distal fistula), most commonly a radio-cephalic fistula, or at the elbow/upper arm level (proximal fistula), in this position most commonly a brachiocephalic fistula, or a brachio-basilic fistula, this last one requiring a second procedure the transposition to the surface of the arterialized vein.

B. The arteriovenous graft (AVG), usually a second choice in patients not suitable for a nAVF fistula, has better mechanical strength, can be used earlier, and has lower primary failure rates when compared with nAVF, but has a much higher infection risk, a poorer primary long-term patency, and needs many more interventions to remain functional.

The VA dysfunction and its complications, such as low access flow (Qa), infection, loss of dialysis adequacy, or thrombosis, are the major single cause of hospitalization and morbidity requiring endovascular intervention, as well as one of the most important drivers of the total cost of an end-stage renal failure program.

Whenever a native arteriovenous fistula (AVF) can be built and is able to mature in no more than 8 weeks, it is considered the first and best choice as a vascular access. It results in higher long-term longevity and less thrombotic or infectious morbidity, needs fewer procedures for maintenance, and is overall a big life and money saver.

The nAVF, however, comes with its own set of disadvantages. There is a higher risk of primary failure (nonmaturation) up to 60% prior to cannulation, requiring frequent angiographic procedures to assist maturation [1–3]. Studies have shown that the primary failure rate is two times greater for fistulas (40%) than grafts (19%), with similar cumulative patency; in addition, the number of catheter days before AV access use was more than double in those using a fistula (81 days) than those with AV grafts (38 days); however, grafts require more angioplasties (1.4 vs. 3.2 events) and thrombolysis (0.05 vs. 0.98 events) interventions per 1000 patient-days [2, 4]. The risk of primary fistula failure is much higher for lower arm fistula (28%) than with upper arm fistula (20%), although these last ones produce more than 90% of all cephalic arch stenosis [1].

The secondary patency rates of AV grafts (total life span even if requiring several interventions to maintain its function) are on average around 3 years, all in all identical to AV fistulas, but those improved rates are achieved at the expense of three- to six-fold greater reintervention rates.

There has never been a randomized control trial (RCT) comparing different VA choices regarding mortality or other hard outcomes. All large observational trials compared accesses achieved as opposed to the accesses that were intended (as in intention to treat). As 25–60% of all AVFs created either fail or need several procedures to mature and the central venous catheter (CVC) group in most studies were people in whom AVF failed or CVC was chosen because of a predictable bad prognosis (age, congestive heart failure, short life expectancy, etc.), we really cannot answer the question on which VA is the best. If we exclude patients that begin hemodialysis urgently, mortality between nAVF and CVC patients becomes identical. Using a decision analysis model (fed with data extracted from DOPPS 2, the

REDUCE FTM study, the DAC study, and CMS data) for choosing the best option for patients initiating hemodialysis (HD) with a CVC, a nAVF attempt strategy is associated with better survival and lower annual cost, but that advantage is progressively lost in patients above 60 years or diabetics [5, 6].

Access malfunction is a source of tremendous emotional and physical suffering, dialysis treatments loss, low treatment adequacy, urgent need for a central catheter as a substitution access, and referral for new angiography or surgical procedures at huge costs.

In this chapter, we basically describe our experience on VA management in our dialysis network treating approximately 5000 patients in our Vascular Access Center (VAC) that performs more than 1000 VA surgeries and more than 1600 endovascular interventions per year.

3. Vascular access morbidity and complications

The most common VA complications are failure to mature, persistently low Qa, suboptimal dialysis adequacy, pain, aneurysms, rupture/hemorrhage, infection, and thrombosis. Endovascular stenosis is the underlying lesion and the direct culprit behind most of these complications.

Neointimal hyperplasia is the common pathogenic mechanism inducing stenosis, and stenosis is the underlying promoter of thrombosis. Stenotic plaques are composed of myofibroblasts (smooth muscle cells) surrounded by extracellular matrix and macrophages. This cell proliferation begins in the adventitia and migrates toward the lumen of anastomotic areas or endothelial segments exposed to several stresses, such as surgical trauma, shear stress, wall stress, diameter and compliance mismatch, uremic endothelial dysfunction, and wall lesion secondary to repeated needle punctures.

Stenosis is necessary for thrombosis, but it is not enough. Only 30% of stenosis above 50% of lumen compromise will cause thrombosis in the next 6 months; we just do not know which ones. On the other hand, angioplasty induces accelerated NH with recurrent stenosis [7]. In 20% of the cases, recurrent stenosis occurs in 1 week and in 40% in 1 month [8], and although stenosis stenting may delay stenosis recurrence, it did not reduce the incidence of thrombosis [9].

As in other vascular territories, we do not know and have no biomarkers to decide which stenosis will progress to cause thrombosis, which stenosis if dilated will prevent thrombosis, which stenosis once dilated will suffer early recurrence, which is the best option to prevent recurrence, and how to define the successful angioplasty.

4. Vascular access malfunction detection

In hemodialysis vascular access management, just as in general medicine, an early diagnosis of malfunction and prevention of definite failure is considered the best approach to diminish morbidity and costs. This axiom was strongly suggested in several seminal studies [10, 11] and is expressed in most guidelines of scientific societies in this field.

It is recommended that regular monitoring of access function should be performed, preferably by measuring vascular access flow (Qa), and when access stenosis is present, preemptive intervention should be performed percutaneously without further delay. In support of these level 2 recommendations, we can quote: "All types of pressure measurement should be abandoned in favor of access flow

measurement," and "Monitoring plus intervention reduces thrombotic rates, morbidity and costs" [11, 12].

Consensual recommendations for preemptive intervention in malfunctioning grafts are (a) Qa measurement <600 ml/min for grafts or <400 ml/min for native fistulas and (b) a Qa drop higher than 25% over two consecutive measurements [13].

However, recent and quite relevant information has questioned those recommendations, and scanning through recent prospective randomized controlled trials in this field reveals some discordant opinions.

No matter if we are looking at native fistulas or PTFE grafts, using only Qa measurements, or its association with Doppler studies or dynamic venous pressure as surveillance techniques, it is believed that VA stenosis is now very effectively detected and responsible for a large increase in percutaneous vascular access procedures. Surprisingly, however, it has been found that all these diagnostic and therapeutic procedures fail to reduce the thrombosis rate or prolong access longevity, fueling an ongoing controversy regarding its beneficial effects, both in terms of overall access survival and associated costs [6, 8, 14–20].

All presently approved clinical guidelines recommend performing surveillance of vascular access quality and performance, aiming at early detection of access stenosis, which induced a global trend toward implementation of Qa-based monitoring programs in many dialysis units.

The recommended Qa thresholds for angiography referral are based on its putative predictive power of access malfunction and/or failure.

However, even before the final decision on the clinical relevance of periodic Qa determination, the quality and accuracy of the Qa measurements methods must be questioned, as most techniques have a good correlation among them, but high variation in absolute terms (±200 ml/min) [21–30].

We are now in a position where we feel that we must do some form of VA surveillance, but do not know exactly which. Qa, although not perfect, with results that are hard to interpret and need specific calibration to fine tune appropriate alarm thresholds for each measurement technique, is probably the best hemodynamic parameter to follow.

In our unit, we evaluate monthly Qa, together with a trend analysis of other equally not perfect parameters, like physical examination [31], Kt/V in all dialysis treatments, recirculation, and maximum obtainable Qb with circuit arterial pressure above −250 mmHg, and then decide empirically, as physicians always do, when to refer to angiography.

A successful program of surveillance should reduce thrombosis rate by an amount identical to the angioplasty rate it induces. The key to measure surveillance effectiveness is avoidance of thrombosis; no other surrogate is acceptable.

As a matter of fact, absolute flow (Qa) and drop in flow, measured using several different flow indicators (ultrasound, thermal dilution, ionic dilution), are inaccurate predictors of thrombosis. Most thromboses are unpredictable, and interventions based on surveillance likely yield many unnecessary procedures at high cost.

We do not know if a vascular access defined by us as well functioning actually looks normal in angiography. Without that, it is difficult to really appreciate the specificity of our monitoring indicators and, most of all, the meaning of stenosis in the natural history of the VA.

Our data suggest that the presence of what we call a significant stenosis is not correlated with measured Qa and it might not be associated with early thrombosis deserving immediate intervention [20]. Further studies are needed to clarify

the best surveillance protocol and the role of preemptive intervention in significant stenosis.

A proposal for surveillance could well include the following:

A. Each unit should perform sequential measurement and trend analysis of the parameters of their choice.

B. Physical examination done and recorded before each dialysis by the R.N., in an access without dressings and needles. Signs to be looked for include a pounding pulse, an intermittent thrill, arm swelling, increment in collateral veins, difficult hemostasis, a new or an enlarging aneurysm, and pain during treatment, reaching an agreement rate with angiogram to detect stenosis of 80% [31].

C. Access flow measurement (Qa) in:

i. High-risk grafts—Every 2 weeks.

ii. Other grafts—Quarterly.

iii. Native fistulas with a Qa < 1000 ml/min—Quarterly.

iv. Native fistulas with a previous Qa ≥ 1000 ml/min—Once a year or whenever clinically indicated.

We consider high-risk grafts:

a. "Last" available vascular access site of that patient.

b. Frequent clotter.

c. Frequent recurrence of significant stenosis (less than 3 months apart).

Patients are referred from the dialysis unit to our VAC by their nephrologists, the indication for intervention is confirmed upon arrival, and an ultrasound/Doppler study will be performed if needed, to decide if it should be referred to the surgical or endovascular arm of the VAC and to help planning the endovascular approach localizing eventual stenotic lesions, their location, and preferred puncture site.

Our referral criteria to surgery: (a) Native AVF thrombosis; (b) VA rupture; (c) infection with visible abscesses or purulent discharge at puncture sites; (d) need for a new VA; (e) steal syndrome, VA limb distal ischemia; (f) primary malfunction of a VA created or submitted to open surgery less than 1 month ago; (g) growing aneurysm; and (h) hemorrhage.

Referral criteria to endovascular intervention: (a) Growing edema of the VA limb, (b) VA pain during dialysis treatment, (c) recent increment of VA venous pressure associated with a drop in dialysis adequacy, (d) unexplained drop in dialysis adequacy, (e) a drop of VA flow (Qa) in 2 measurements <600 ml/min in a AVG or <400 ml/min a nAVF, (f) need for assisted maturation of a nAVF, (g) superior vena cava syndrome, and (h) AVG thrombosis.

The techniques we perform in the angiography suite are (a) diagnostic angiography in no more than 7% of all cases, (b) percutaneous angioplasty (PTA) of stenotic lesions, (c) thrombolysis for thrombosed AVGs, and (d) stenting of elastic or frequently relapsed stenosis.

In our unit, prospective results of 1-year follow-up in 71 new AV grafts with monthly surveillance revealed the following:

a. A Qa < 600 ml/min had the same predictive value with that ΔQa of 25%, and dynamic venous pressure was useless.

b. After 1 year only 35% of PTFEs did not need any kind of intervention. We demonstrated then a sensitivity of 82% and a specificity of 90% to detect stenosis.

c. "Successful" PTA in 91% and Qa↑ on average 142%.

d. A sensitivity of 39% and a specificity of 21% to detect thrombosis.

e. A thrombosis rate—0.46 thru/pt. year.

f. In 60% of cases, previous monitoring was normal.

5. Endovascular intervention to correct dialysis vascular access malfunction

The initial treatment recommended for stenotic lesions in both nAVF and AVG is endovascular intervention, primarily angioplasty. Endovascular intervention is employed to maintain or even rescue AV access [32].

The recommendation within the K/DOQI guidelines is to treat hemodialysis access stenosis of more than 50% of the vessel lumen, if those are related with reduced flow rate and high venous pressure. PTA is considered a standard of care in failing hemodialysis access due to its high rates of success and satisfactory patency rate [33].

5.1 Stenosis location

The stenotic lesions in an AV fistula can occur in any location of the access system, with a higher incidence in specific spots for each type of VA. This is the case of stenosis at the proximal "swing segment" (the vein segment immediately after the arteriovenous anastomosis of a nAVF, which was dissected and brought close to the artery to create the anastomosis) either in the upper arm in transposed brachio-basilic fistulas, or in the lower forearm, in radio-cephalic fistulas, which are relatively more frequent than lesions at any other site [34]. Another example is the cephalic arch region, in patients with brachiocephalic fistulas [32].

Below we describe some types of stenosis of the vascular circuit, selected for their particularities, namely, their frequency, risk of restenosis, and predictable danger of VA failure.

5.2 Cephalic arch stenosis

The proximal cephalic vein is characterized by a curved shape, which occurs as the cephalic arch passes though the coracoclavicular ligament, just before joining the subclavian vein.

Several reasons have been put forward to justify the development of cephalic arch stenosis, such as increased blood flow rates, hemodynamic factors associated with the vessel shape, external compression by the outer structures surrounding the vein, and hypertrophy of valves that are often present in the cephalic arch.

Although angioplasty is the accepted initial treatment of cephalic arch stenosis, it can be problematic because lesions in that location are more resistant to dilatation. When dealing with resistant stenotic lesions, it is shown that employing cutting balloons (see below) may improve outcomes. On the other hand, complications are more likely (vein rupture), and patency is reduced compared with other vein location. Stent placement in the arch is a delicate task because the stent should invade the subclavian vein lumen, which can result in its partial or total occlusion, impeding the future creation of an AVF or AVG using the basilic or axillary vein, thereby consuming vascular patrimony. In view of the recurrent problems with angioplasty of the cephalic arch, the stent placement can be an alternative to rescue the vascular access (**Figure 1**).

For several reasons, it is not possible to make any evidence-based recommendations on best practices for management of CAS (endovascular or surgical). There is profound heterogeneity in the studies retrieved, from their initial design to their presentation of data. Few studies were prospective, few studies involved more than one or two centers, and the lack of uniformity of outcomes is another weakness of current published studies. CAS is often managed alternatively by interventionists and surgeons, in our experience with identical success.

5.3 Central vein stenosis

The prior placement of a central venous catheter is by far the most common cause of central vein stenosis (CVS) in dialysis patients. Transvenous wires of cardiac rhythm devices are more and more related with central veins stenosis in this population of high cardiovascular morbidity. Hemodialysis patients are, therefore, primary candidates for new wireless pacemakers or epicardial pacemaker leads.

The surgical approach to central vein stenosis is difficult because they can hide behind the bone structure. Therefore, endovascular intervention with angioplasty and/or stent placement becomes a logistically more receptive proposal for treatment of CVS. Still, anatomically and functionally, central veins have several specific characteristics including the diameter, angle, and elasticity that make treatment and maintenance of their patency after intervention difficult.

Some central vein stenoses are not symptomatic. Asymptomatic central veins stenosis, involving less than 50% of the vessel lumen, does not require treatment and is best managed by simple supervision [35].

Angioplasty with or without stent placement has been the recommended preferred approach to CVS. The guideline 20 NKF-K/DOQI suggests that the percutaneous intervention with transluminal angioplasty is the preferred treatment for CVS [36]. PTA has very high initial technical success rates, ranging from 70 to 90% [37, 38]. Primary and cumulative patency rates are widely variable and can

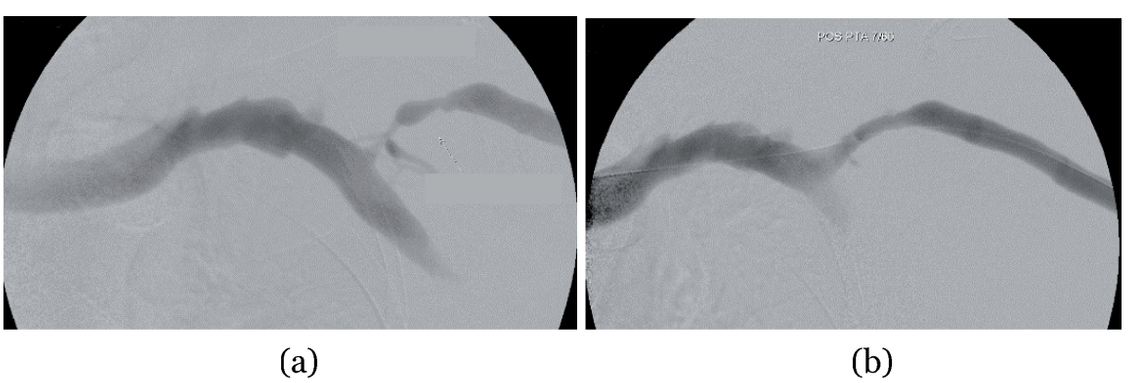
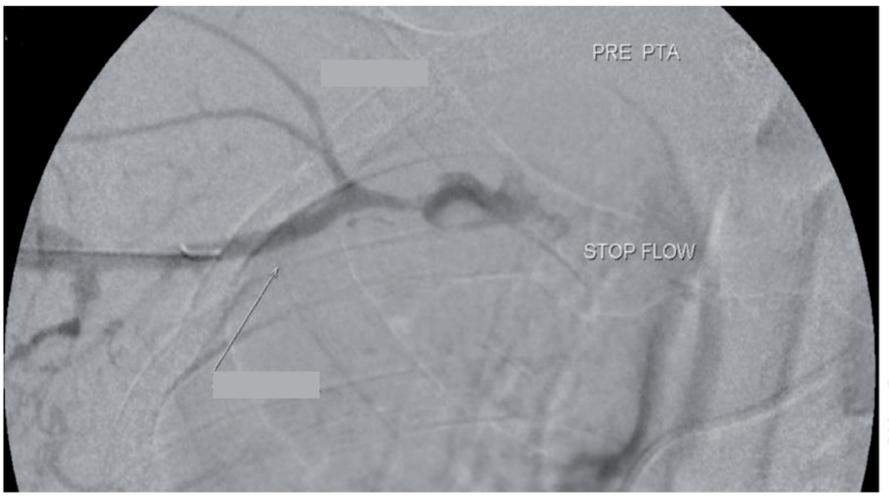
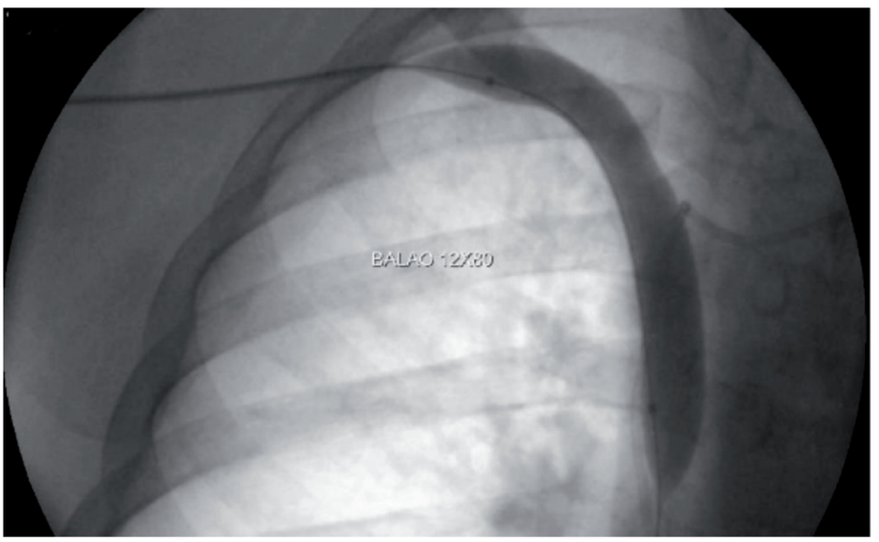


Figure 1.
(a) and (b). Stenosis affecting cephalic arch; this lesion responded well to balloon angioplasty.



(a)



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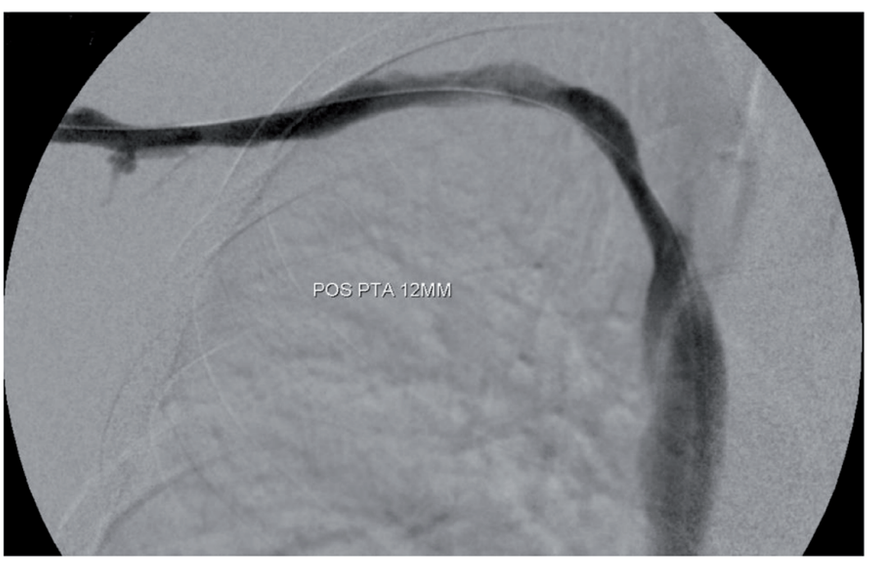

(c)

Figure 2.
(a) Right brachiocephalic trunk stop flow. (b) PTA of stenosis with 12 mm balloon. (c) Final angiogram result.

range between 23 and 63% at 6 months and 12 and 50% at 12 months in the case of primary patency rate, as well as 29 and 100% at 6 months and 13 and 100% at 12 months in the case of cumulative patency rate (**Figure 2**) [36, 39–42].

Our cumulative experience shows that angioplasty and stent placement is undermined by frequent and rapid recurrence. It can also happen that an asymptomatic lesion can become symptomatic upon intervention. Indeed, one study showed that stenosis can progress faster after intervention [37]. The venous response may be worsening, and the stenosis process can be accelerated due to angioplasty.

Correction of CVS with endovascular approaches remains therefore limited and suboptimal and may even be harmful in certain cases. After angioplasty, more aggressive neointimal hyperplasia and proliferative lesions were found in restenosis areas than in the original stenotic lesions [38].

A major problem with lesions in the central veins is that many are quite elastic. For this reason, endovascular stents are used more frequently for central veins stenosis than for other types of dialysis access lesions. Cost considerations are highly relevant and also the fact that we are left without any option to treat effectively a restenosis inside a stent. Even if we extend 100% the half-life of a recurrent stenotic access (from a procedure every 3 months to every 6 months), it may look as an impressive achievement, but with little clinical relevance.

5.4 Juxta-anastomotic location

The vein immediately adjacent to the arteriovenous anastomosis (commonly referred to as juxta-anastomosis) is a common location of stenosis. This is in part due to injury, which occurred while "swinging" the vein to form the AV anastomosis. Some studies demonstrate that the frequency of juxta-anastomotic stenosis may be up to 55% [43].

Angioplasty and surgery are two treatment options. Percutaneous angioplasty has 1-year patency rates of 44–79% [44–46]. For surgery, 1-year patency rates are between 64 and 88% [45–47]. In this location, we usually need very high-pressure balloons to deal with very hard lesions. If we elect a surgical solution, we get better results at the expense of a few more centimeters of vascular territory. Regrettably, randomized studies comparing endovascular treatment and surgery for this lesion are not available (**Figure 3**).

5.5 Type of angioplasty balloons

There are several types of balloons that we can use in angioplasty: (i) "high-pressure," (ii) "ultrahigh-pressure (UHP)," (iii) "cutting," and (iv) "drug-eluting."

5.5.1 High-pressure balloons

High-pressure, noncompliant balloons (e.g., Conquest from Bard Peripheral Vascular Inc., Tempe, Arizona) have rated burst pressures of 20 to 24 atm and are used to treat dialysis vascular access stenosis.

5.5.2 Ultrahigh-pressure balloons

Venous stenosis is characterized by extensive fibrosis and the need for ultrahigh-pressure balloon inflations [48] or cutting balloon atherotomy for optimal treatment [49, 50].

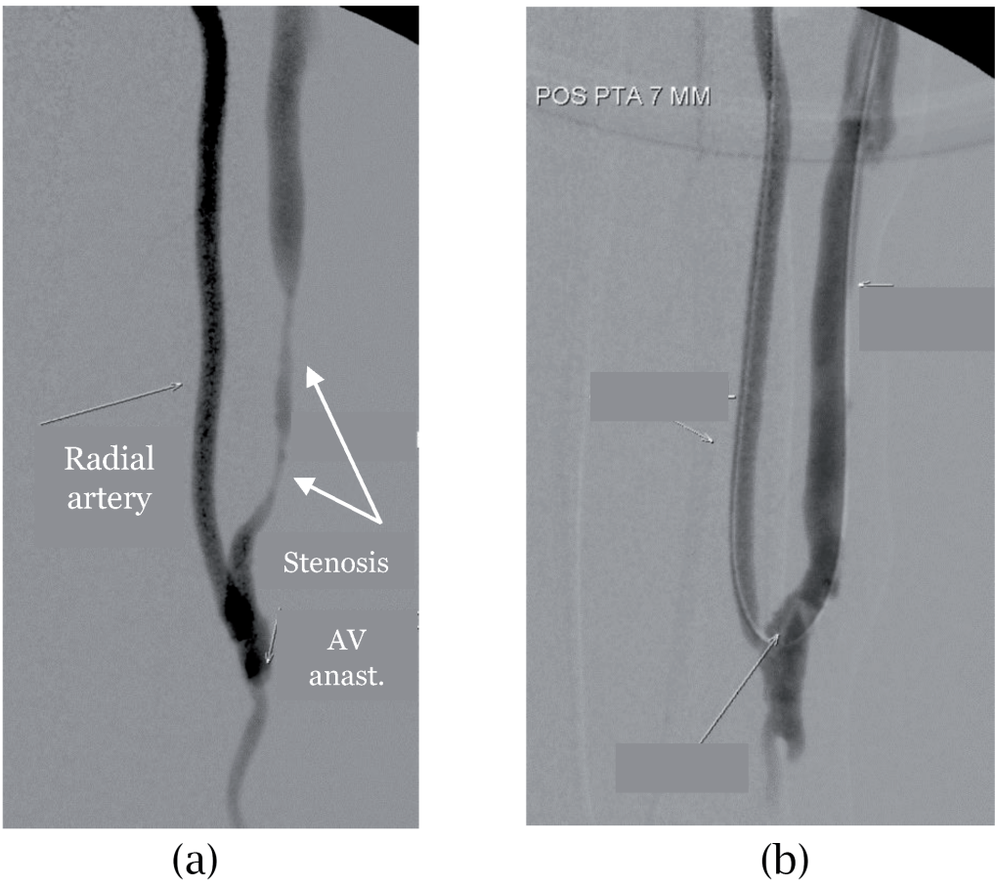

Figure 3.
(a) Severe stenosis in juxta-anastomotic radio-cephalic fistula. (b) Angiogram result after angioplasty with 7 mm PTA balloon.

An UHP balloon is certified for a burst pressure of 27 atm, but higher inflation pressures are possible. Although those balloons do not provide better results in terms of permeability, when compared to conventional ones, it has been suggested that such devices may achieve better patency rates than traditional HP angioplasty balloons [51]. Its use is indicated in the treatment of symptomatic stenosis not responding to conventional high-pressure balloon. The high price of UHP balloon, the need for use thicker inserts, the difficulty of emptying, and its lower compliance and flexibility make it advisable that UHP balloons should not be a first choice in stenosis treatment.

Despite that, ultrahigh-pressure balloons have significantly reduced the incidence of "resistant" lesions [52].

5.5.3 Cutting balloons

The cutting balloons are special angioplasty balloons with three or four cutting edges (atherotomes) fixed longitudinally to its surface. The atherotomes expand radially with balloon inflation and provide longitudinal incisions into the lesion cutting into tenacious neointima. Using cutting balloons has the advantage that disruption of the lesion occurs in a more controlled manner and at lower balloon inflation pressure than with conventional angioplasty.

The use of a cutting angioplasty balloon (CAB) to treat resistant lesions can be found in several reports [49, 51]. Most of these reports are constrained because they are retrospective, lack control, or the size is too small to allow meaningful

conclusions. Taking into account the data reported in the literature and also considering the authors experience, it can be stated that angioplasty with a cutting balloon is safe and can be considered as an alternative treatment for stenosis of hemodialysis AVFs that do not respond to conventional balloons.

There are serious methodologic limitations in the published reports describing the use of cutting balloon angioplasty to treat hemodialysis vascular access stenosis [53–56]. Studies include the concurrent use of cutting and conventional balloon angioplasty, the use of a high-pressure balloon, or a combination with placement of a stent after cutting balloon angioplasty. In other studies, cutting PTA was used only after the failure of high-pressure balloon angioplasty. In these reports, the long-term patency rate does not reflect the results obtainable with cutting balloon angioplasty as a primary, stand-alone treatment. The cutting balloon was designed primarily to reduce vascular trauma, thereby diminishing neointimal hyperplasia, thereby improving hemodialysis access long-term patency. It should be noted that studies comparing cutting balloon and conventional balloon angioplasty in the treatment of vascular access stenosis are fraught with conflicting results.

5.6 Drug-coated balloons

Good results have been obtained with drug-coated balloon (DCB) angioplasty used to prevent restenosis in the treatment of arterial stenosis. This approach (using paclitaxel-coated balloons) was extended to the treatment of stenosis associated with hemodialysis AV access, with mixed results [57].

Drug-coated balloon endovascular technology merges the dilating properties of angioplasty with local drug delivery. Balloon surface excipients enable drug-eluting within the vessel wall, inhibiting cell proliferation, and reducing neointimal hyperplasia, while avoiding the use of permanent metal stents.

DCB angioplasty of vascular access stenosis seem to be safe and effective, providing superior reintervention-free intervals compared to conventional plain balloon angioplasty [58–60]. Recently, Yan and others published a meta-analysis that reveals that DCB is an effective and safe method that can significantly prolong 6-month and 1-year target lesion primary patency for failing hemodialysis access, as compared to conventional plain balloon angioplasty. However, their study was limited by the small number of patients enrolled in each trial, the diversity characteristics of the lesions, the vintage of the dialysis access, and the formulations of paclitaxel (different dose or excipients used). A very heterogeneous group of studies lumped together [61].

The reported number of dialysis patients treated with DCBs is low, and several concerns remain unanswered. First of all, it is uncertain which lesions will benefit from the use of this balloon device. Lesion preparation is another issue that deserves further investigation. Manufacturing companies suggest pre-dilation with a shorter balloon, with the same diameter, to promote drug diffusion within the deeper layers of the vessel wall and to improve the restenosis rate. However, in some RCTs published, pre-dilation was not even performed. Last but not least, although the long-term safety of PCBs in dialysis access treatment has been proven, preclinical and experimental studies in animal models are lacking; consequently, we have no available information on the posttreatment lesion pathology, degree of drug diffusion, and the extent of paclitaxel fixation within the venous wall.

It should be noted that the use of drug-eluting balloons is a novel medical device that aims to decrease the trauma in the endothelium of the vascular wall of a fistula. Although more expensive than the conventional balloon, it is much cheaper than a bare metal stent, and repeated procedures can be performed in case of

recurrences. More trials are needed to find out if this more expensive material can really increase the patency of venous lesions.

5.7 Stent placement

Several challenges must be faced by resistant or recurrent stenosis throughout the access circuit in terms of providing optimal hemodialysis treatment. Those stenoses can be successfully treated by endovascular stent placement, although it usually requires multiple procedures to maintain patency.

Indeed, bare metal stents and covered stents have emerged as a potential additional therapeutic intervention in vascular access dysfunction. However, results are not encouraging. For example, bare stents are seldom used due to a high incidence of in-stent stenosis, and covered stents also have problems.

There are three mostly accepted indications for stent deployment: (i) a stenotic lesion that recurs within a 3-month period after initially successful balloon angioplasty in a patient with *exhausted VA sites*, (ii) a stenotic lesion with high elastic recoil (usually in central veins), and (iii) rupture of an outflow vein after balloon angioplasty that cannot be handled using more conventional actions (balloon tamponade). Other special conditions where a stent implantation should be considered include (i) venous outflow stenosis, (ii) pseudo-aneurysms, and (iii) cephalic arch stenosis.

We must take special care not to occlude important collateral veins with implanted stent, namely, the homolateral internal jugular vein, always required for future central vein catheters.

There are several reported complications associated with stent placement, such as stent migration, or stent fracture, which is usually seen on control angiograms. Infection is also a significant complication with potentially tragic outcomes. It should be noted that the combination of the immune-compromised status of patients with ESRD and repetitive cannulations for dialysis treatments is likely factors leading to infection. One unique complication is stent struts protrusion, which results from placing stents in cannulation sites [62]. Damage of the metal part of the stents (struts) can result from repetitive cannulation.

The high cost of stents has to be taken into account, raising the question whether the benefits obtained by placing stents at stenotic lesions outweigh the costs associated with such treatment [9]. One should reflect if the option of creating a secondary AVF should be considered as an alternative treatment for placing a stent (**Figure 4**).

5.8 AVG thrombolysis

Graft thrombosis occurs in one-third of all AVG per year, and of those that thrombose, 60% have more than two episodes per year. Ninety percent of all AVG thrombosis are associated with a stenotic lesion, most commonly in the venous anastomosis, but can occur in any location, in 36% of the cases in more than one site.

In our VAC, AVG thrombosis is primarily referred to interventional nephrology for endovascular thrombectomy, combining pharmaco-mechanical thrombolysis with a multiperforated catheter occluded at its tip, allowing high-pressure lateral injection of heparinized saline to dislodge wall adherent clots, followed by angioplasty with a 8 mm balloon of all stenotic lesions and finally embolectomy of the arterial anastomosis with a 4 French Fogarty catheter, to remove a more adherent, residual, fibrin "white" clot. Alternatively, some of us may use a mechanical device, the Arrow-Trerotola©, that combines clot fragmentation and aspiration, adding quite a substantial extra cost, without improving outcomes.

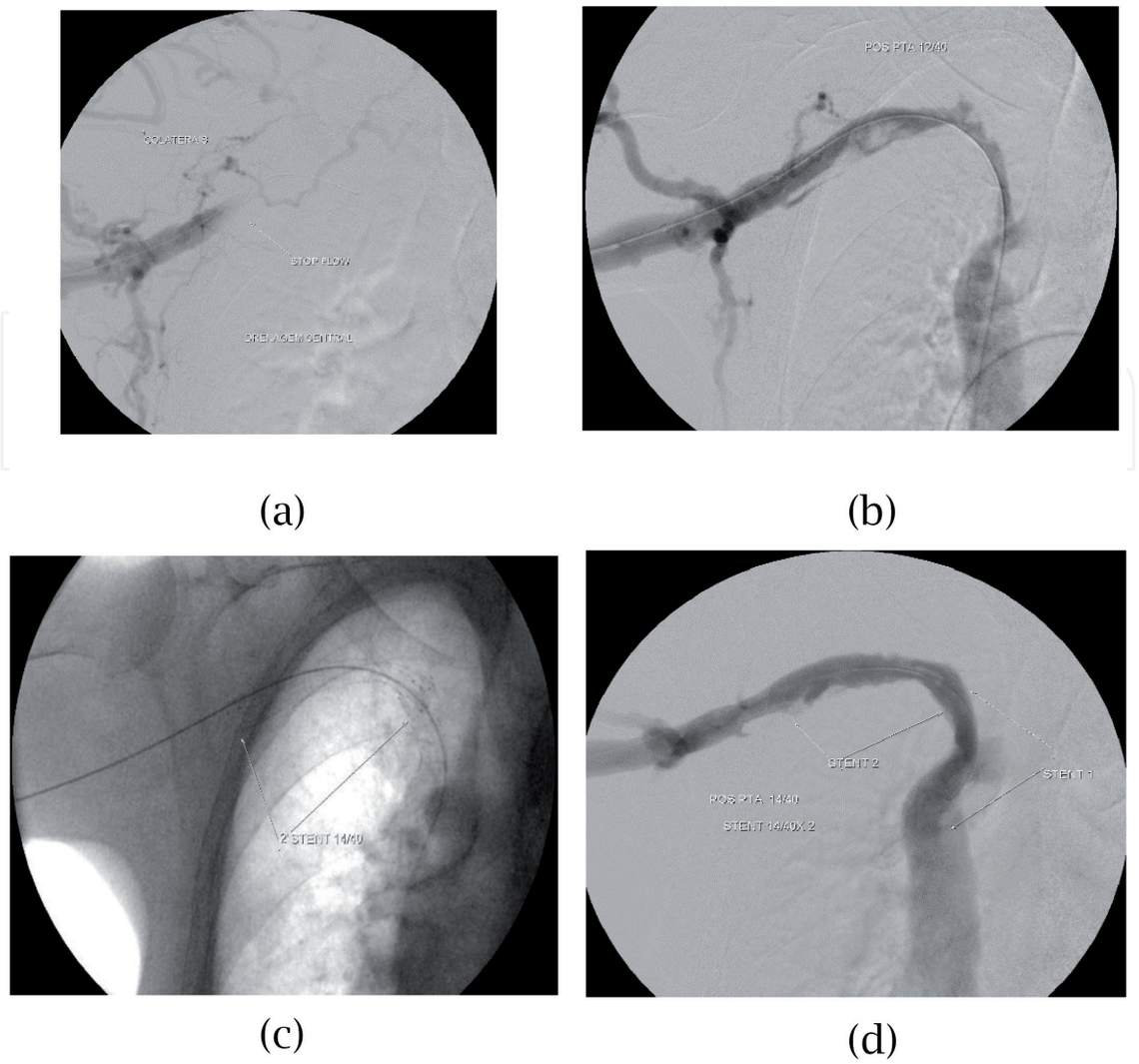

Figure 4.
(a) Stop flow at right BCT. (b) Placement of a stent. (c) Angiogram after 12 mm PTA balloon. (d) Final angiogram result.

The procedure should not be performed if the AVG is suspected to be infected or a graft rupture is detected. At the end of the procedure, we must check AVG flow, the presence of residual clot inside the VA, and the most dreadful complication, symptomatic hand ischemia due to distal arterial embolization, which must be resolved with embolectomy in the angiographic suite or urgently referred to surgery.

6. Endovascular intervention outcomes

The quality indicators achieved in our VAC include: (a) creation of a nAVF as first access in 80% of all patients and in 60% of subsequent accesses, (b) less than 40% primary failure of nAVF at 3 months, (c) less than 55% secondary failure of nAVF at 12 months, (d) less than 30% primary failure of AVGs at 3 months, (e) percent of functioning AVG post-thrombolysis >75% at 7 days and >50% at 3 months, and (f) no VA infections 15 days post-intervention. Regarding VA, the dialysis unit quality indicators are (a) percent of prevalent patients with nAVF >65%, (b) percent of patients with long-term tunneled catheters <20%, and (c) referral's rate to the VAC <0.8/patient years.

Procedures	At 30 days (%)	At 90 days (%)	At 180 days (%)
Diagnostic angiography	83	55	45
Angioplasty	92	60	45
Thrombolysis + angioplasty (AVG)	86	51	40

Table 1.
Primary patency in our VAC at 30, 90, and 180 days, in line with most literature in the field.

In intervening in a nAVF, use when available ultrasound localization of stenosis to plan the best place and direction to puncture the access
In intervening in a AVG, always puncture close to the arterial anastomosis in the direction of the flow toward the venous anastomosis
Use a 7F sheath and a hydrophilic guide wire. It allows balloons up to 14 mm to be inserted more than once
Do not miss any step even when it seems unnecessary. Always check AV anastomosis in nAVFs and arterial and venous anastomosis in AVGs, as well as central venous drainage
Do not accept incomplete balloon dilation. If necessary, use high-pressure balloons or cutting balloons
Avoid stents, only as a last resort
In AVG thrombolysis, after dealing with the venous anastomosis, even if the graft is already working, do approach with a Fogarty the arterial anastomosis
Always test flow at the end of a procedure. An eyeball test as the TIMI for cardiologists. If flow does not look great, it is because there is something else to fix

Table 2.
Clinical pearls to take home.

The technical details of all procedures we perform in our VAC are thoroughly described in a recent textbook [63, 64].

The National Kidney Foundation (KDOQI) guidelines [65] define a successful angioplasty a residual stenosis <30%, with return to acceptable levels of the parameters used to place the indication for angioplasty. Initial success rates using anatomical criteria ranged from 80 to 98%, but in some reports, 20–30% of these patients with anatomical success fail to increase blood flow (residual stenosis, a missed lesion, or elasticity). Primary patency rates are 41–76% at 6 months and 31–45% at 1 year.

Long-term primary patency rates for thrombectomy are not as good as for angioplasty; therefore every effort should be made to prevent thrombosis by the prospective diagnosis and treatment of venous stenosis.

In a thrombosed access, the treatment must be timely to avoid catheters, done as an outpatient, venous stenosis must be detected and corrected, hemodynamic parameters should return to baseline, and patient should be evaluated for a secondary arteriovenous native fistula, created using upper arm veins that have become dilated because of the functioning graft.

In 2019, 139 surgical thrombectomies were performed in 127 patients (69 in nAVF and 70 in AVG). In 49.6%, no new intervention was required, and the average time until a new intervention was 46.7 days. Primary patency at 1 month was 66%, at 3 months 54.4%, and at 6 months 17.5%. In that same period, the angiography suite received 134 patients for 179 procedures (171 in AVGs, 8 with a nAVF), there was immediate success in 159 patients, the average time until a new intervention was 58.1-day, and primary patency at 1 month was 71.6% and at 6 months 42.5%. In our case, Qa average improvement is >50%, and we expect a Kt/V above 1.4.

The immediate success rate of thrombolysis should be 85% or greater according to the NKF/KDOQI guidelines and the primary (unassisted) patency goals at 3 months at least 40% (**Tables 1** and **2**).

7. Conclusion

In conclusion, we are still dealing with quite a number of known unknowns. There has been no RCT to elucidate which percentage of lumen compromise should dictate the indication for angioplasty, and most operators choose 50%. Not all stenotic plaques were created equal, and some will never progress, but we cannot guess which ones. We also have no proof that a successful PTA in a graft improves long-term patency rate [14], angiographic criteria to assess the success of angioplasty are not predictive of changes in blood flow, and there is no correlation between changes in blood flow and changes in the percent of stenosis post-PTA [66]. In an era characterized by less is more, under the imperative of being useful for our patients, creating long-term solutions at sustainable costs, we feel a desperate need for robust scientific evidence to support our decision process and the procedures we perform. Just because it can be done, does not mean it should be done, our intervention is no more a question of know-how, but of know-when.

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