Approach to an Arteriovenous Access with No Thrill, Bruit, or Pulse

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Introduction

Biochemical and fluid disequilibrium. A patient who has been receiving suboptimal dialysis for a week may predictably have more or more severe biochemical derangements than a patient whose dialysis treatments have been uneventful up until the day of access thrombosis.

The clotted vascular access is not difficult to clinically diagnose. A color flow Doppler examination will verify the absence of flow through the access, but this is rarely needed. A sometimes confusing clinical finding is a thrill close to the artery-vein anastomosis in an autologous fistula. If the thrill comes, in fact, from the fistula and not transmitted from the artery, then one's approach might be simplified to a percutaneous angioplasty of a suspected downstream stenosis. Another useful physical finding is whether or not the fistula or the effluent venous drainage of a prosthetic arteriovenous graft is hard or tumescent suggesting extension of clot to this region. A greater length of soft or collapsible fistula portends a smaller clot burden than a sizable length of hard or turgid vein. Anticipating the amount and extent of thrombus that one might encounter would be beneficial in planning whether or not to utilize thrombolytics, the approach to removing thrombus, and in anticipating the likelihood of complications such as forward embolization of access thrombi to the pulmonary circulation. The clot burden in a typical AVG is between 1.5 and 4.7 mL [1] but in the fistula can vary from minimal to significantly larger volumes especially in the aneurysmal, serpentine brachiocephalic variety.

Figures
Figures 16.1 to 16.7: nothing to disclose, my personal figures on cases I have personally performed.

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It is generally accepted that one's chances of a technical and clinically successful thrombectomy are highest when intervention is performed as soon as possible following the diagnosis. In a chronically thrombosed fistula, gaining entry to the vessel becomes progressively more challenging with time when the absence of blood flow causes it to collapse. This is readily apparent in the case when an angiographically collapsed drainage vein of a thrombosed AVF is found to be widely patent and of large caliber following restoration of flow in the upstream segment without any intervention performed on that specific segment of the collapsed vein.

The patient with a hemodialysis vascular access that has no palpable thrill or pulse and no audible bruit presents the physician with at least two simultaneously critically important and time-sensitive issues: the resuscitation of the vascular access and the patient's need for ongoing life-sustaining renal replacement. Although it may intuitively appear that the former necessarily leads to the latter, the decision regarding how best to assure the immediate and more crucial need for ongoing dialysis often flavors how one approaches vascular access. In this regard, the physician must employ his keen clinical sensibilities and judgment, understand the renal patient's history and physiology, and judiciously utilize the most appropriate approach to the problems at hand. The management of the clotted dialysis vascular access can be a most challenging but ultimately uniquely rewarding situation that a clinical interventionalist will face.

Clinical Considerations

In most circumstances, the patient is referred from the dialysis facility where the health-care professionals assessed the vascular access pre-cannulation and deemed it thrombosed. On occasion, however, they may have attempted cannulation and been unsuccessful in obtaining viable blood return from one or both needles. Invariably, there may have been prodromal symptoms or signs that presaged the clotting of the access. It is useful for the clinical interventionalist to be aware of these because it provides a clue as to the culprit

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lesion(s) that one may anticipate during the procedure. It also aids one to know the duration during which renal replacement has been suboptimal or dysfunctional as this helps stratify procedural and sedation risks based on the patient's biochemical and fluid disequilibrium. A patient who has been receiving suboptimal dialysis for a week may predictably have more or more severe biochemical derangements than a patient whose dialysis treatments have been uneventful up until the day of access thrombosis.

The clotted vascular access is not difficult to clinically diagnose. A color flow Doppler examination will verify the absence of flow through the access, but this is rarely needed. A sometimes confusing clinical finding is a thrill close to the artery-vein anastomosis in an autologous fistula. If the thrill comes, in fact, from the fistula and not transmitted from the artery, then one's approach might be simplified to a percutaneous angioplasty of a suspected downstream stenosis. Another useful physical finding is whether or not the fistula or the effluent venous drainage of a prosthetic arteriovenous graft is hard or tumescent suggesting extension of clot to this region. A greater length of soft or collapsible fistula portends a smaller clot burden than a sizable length of hard or turgid vein. Anticipating the amount and extent of thrombus that one might encounter would be beneficial in planning whether or not to utilize thrombolytics, the approach to removing thrombus, and in anticipating the likelihood of complications such as forward embolization of access thrombi to the pulmonary circulation. The clot burden in a typical AVG is between 1.5 and 4.7 mL [1] but in the fistula can vary from minimal to significantly larger volumes especially in the aneurysmal, serpentine brachiocephalic variety.

It is generally accepted that one's chances of a technical and clinically successful thrombectomy are highest when intervention is performed as soon as possible following the diagnosis. In a chronically thrombosed fistula, gaining entry to the vessel becomes progressively more challenging with time when the absence of blood flow causes it to collapse. This is readily apparent in the case when an angiographically collapsed drainage vein of a thrombosed AVF is found to be widely patent and of large caliber following restoration of flow in the upstream segment without any intervention performed on that specific segment of the collapsed vein. Successful endovascular intervention on fistulas thrombosed for as long as 9 days has been reported [2].

Thrombectomy procedures being performed successfully in cases of early graft failures suggest that intervention can be safely done as early as 15 days after creation using straightforward endovascular techniques including thrombolysis with 250,000 units of urokinase [3]. Of the two early occlusion grafts treated in this fashion, they experienced only one episode of extravasation at the tapered arterial end of the graft following thrombolysis and angioplasty with a 4 mm balloon, causing them to abort the procedure. Some investigators report acceptable cumulative patency rates of 74% at 3 months and 68% at 12 months [3, 4]; other investigators report dismal findings of median patency rates of 11 days in grafts age ≤30 days and 23 days in grafts 31–60 days [5] and 6-month cumulative patency rates of 26 and 44% for grafts age ≤30 days and 31–60 days, respectively [6]. These values fall far below the recommended benchmarks [7] and have caused the authors to question the value of performing endovascular thrombectomy procedures in these early failure grafts. Since the analysis of their results, one group has now opted to channel all early thrombosed prosthetic grafts to surgery for creation of a new access [6].

Extrapolating these observations and conclusions to the native fistula is unwise and fraught with problems. The fistula, of course, requires a maturation process during which flow progressively increases culminating in the thickening of the walls and dilatation of the vessel lumen to accommodate the increased flow and pressure within the circuit. Apart from the surgical anastomoses healing and incorporating into surrounding tissue and the expected perioperative swelling around the tunneled graft, no such maturation process is required. Angiograms done on 1-week-old grafts have demonstrated incorporation of prosthetic into surrounding tissue [3]. The lesions involved in the thrombosis of the graft are different from those in a fistula. While the graft-vein anastomosis is the most common lesion encountered in clotted AVGs [8–11], the clotted fistula can have a variety of lesions or a combination of them [12–16]. In a series of over 100 immature AVF that thrombosed before they were ever used for dialysis, Miller reports a 79% success rate at endovascular intervention [17]. Although the average age of the fistula at the time of thrombosis was 5.6 months, the average mid-fistula diameter was only 1.5 mm. Regardless of age, these were fistulas that had failed to mature. Following thrombectomy, there was an average maturation time of 46.4 days with 2.64 interventions required to attain maturity, including angioplasties, stent implantation, and coil embolization of side branches. Following maturation, these fistulas required an average of 2.78 interventions/access year to maintain patency and underwent 0.52 thrombectomies/access year. It has previously been reported [18] that fistulas that require two or more interventional procedures to attain suitability for use behave differently from those that attain this state spontaneously or require only one procedure.

Compared to fistulas requiring one or less procedures to attain maturity, those that require two or more have consistently reduced 1-, 2-, and 3-year cumulative survival and require more procedures to maintain patency. For these various different reasons, the experience and practice recommendations with early thrombosed prosthetic grafts should not be translated to fistulas without due caution.

Precautions

There are two contraindications to percutaneous thrombectomy of the dialysis vascular access: access infection and known right-to-left shunt (e.g., patent foramen ovale).

A known active infection of the thrombosed access could prove to be disastrous by disseminating infection in an otherwise contained area. It should be noted that a nonfunctioning, even chronically thrombosed, prosthetic AVG can be the source of bacteremia and sepsis [19, 20] in up to almost a third of cases seen by surgery for excision of the prosthetic [21].

A patent foramen ovale is seen in about 25% of the general population over the age of 45 [22] and in 27% of autopsies of otherwise healthy adults [23]. While right-to-left shunting is not necessarily problematic with a small PFO, the presence of pulmonary hypertension makes the likelihood of significant shunting much more of a concern. With pulmonary hypertension seen in as much as 40% of hemodialysis patients, 14% of whom have moderate to severe levels [24], one must be cognizant that a PFO that would otherwise not be problematic could, indeed, prove to be catastrophic. Unfortunately, there is no efficient and cost-effective way to monitor the confluence of these two processes, especially as the natural history of the patient's dialysis unfolds. Pulmonary hypertension is at least 2.7 times more likely to be seen in the hemodialysis patient than in the general population and 1.6 times more likely than the CKD pre-dialysis population [25].

General Approach

Regardless of specific technical methodology and tools employed, there are some fundamental tenets that one follows in order for the thrombectomy to be successful:

1. Identify and treat all lesions felt to be physiologically significant and have contributed to the dysfunction of the access.
2. Control and minimize risk of peripheral pulmonary or arterial embolization.
3. Keep circuit in the least prothrombotic state as reasonably possible.

Except in few and rare instances of hypercoagulability, insufficiently low perfusion pressures from marginal cardiovascular reserve or function, or an inordinately long access circuit relative to feeding arterial flows, an access will have thrombosed because of an anatomic inflow or outflow abnormality or a combination of these. Unless this pathology is found and fixed, or at the very least mitigated, the thrombectomy will not be technically or clinically successful. It is not enough to remove clot and restore flow without addressing the fundamental reasons why the dysfunction occurred to begin with.

Decisions regarding specific methodology that one will employ for a thrombectomy are flavored, among other things, by how most efficiently to get the job done successfully and in a cost-effective fashion but also how to minimize the risk of complications. One of the complications that one might anticipate is the risk for downstream pulmonary embolization. The risk is generally felt to be minimal because the clots are small in size. Paired pre- and post-procedure scintigraphy scans on 13 patients failed to show any difference [26] although larger studies using similar methodology of thrombectomy and scintigraphy revealed new perfusion defects in about 35–40% of patients [27, 28]. In these series, all but 1 of 50 patients studied were symptomatic. Another interesting note is that baseline V/Q scan abnormalities were noted in over 70% of patients [28].

Similar to pulmonary embolism, the incidence of this symptomatic arterial embolism is significantly lower than asymptomatic embolism. The incidence is quoted as between 0.4% and 0.6% [29, 30]. Treatment is generally limited to the symptomatic patient and/or one whose quality and intensity of peripheral pulses have changed during the course of the procedure.

All implements employed during a thrombectomy procedure are potentially thrombogenic. The trauma of the procedure, especially against the vessel wall, and its attendant biochemical and hormonal effects also contribute to the prothrombotic state of the circuit. Systemic anticoagulation is typically given at the start of a procedure, although we have successfully performed thrombectomies without the benefit of heparin in patients with heparin-induced thrombocytopenia. The brisk and robust return of blood flow to the circuit has an antithrombotic effect but can be attenuated by small-caliber vessels that are occluded by sheaths and other similar implements and the wall trauma of instrumentation. Once flow is restored, the physician must move quickly but deliberately and decidedly and address all pathologic lesions that are felt to have caused or contributed to the thrombosis.

Specific Approach

Although there are variations dictated by practice or by the particular case at hand, the approach to the thrombosed prosthetic graft follows the steps in Table 16.1.

Initial Cannulation

The initial step should be to enter the graft and obtain access to the venous outflow tract and central venous system. While it is fairly easy to determine the direction of the cannulation

Table 16.1 Steps in thrombectomy of a prosthetic dialysis graft

1.	Cannulate graft with intent to gain access to the venous effluent tract
2.	Cross vein-graft anastomosis
3.	Perform central venous angiography ± recanalization/ angioplasty of central venous occlusion
4.	Administer anticoagulation and sedation analgesics if not already done
5.	Treat clot within graft and within effluent vessels
6.	Perform venous angioplasty of vein-graft anastomosis
7.	Cannulate graft with intent to gain access to the arterial inflow tract
8.	Remove arterial fibrin-platelet plug
9.	Evaluate and address inflow pathology
10.	Evaluate and address outflow pathology
11.	Completion angiography of entire circuit

needle in an open graft, in a straight configuration graft or a graft that has been studied before and for which images and notes are available, one must rely on usual or typical graft architecture. The loop AVG typically has its venous anastomosis/loop at the lateral aspect of the arm, while its arterial anastomosis/loop is at the medial aspect.

Cross the Vein-Graft Anastomosis

If one is unable to cross this anastomosis, then there is no purpose in restoring arterial inflow for which no outflow is available. Several standard endovascular techniques may be called upon if the guidewire will not traverse the anastomosis readily. Remember also that lesions may be eccentric or may have an orifice that allows wire passage more readily from one direction rather than another. It might be necessary to cannulate the predicted draining vein and pass a guidewire through the vein-graft anastomosis in a retrograde fashion. If this approach is necessary, an angioplasty of this area will allow much easier guidewire passage through an antegrade direction should draining or central venous angioplasty be required.

Perform Central Venography

As much as 30% of dysfunctional dialysis accesses have a physiologically significant central venous stenosis [31–33]. These lesions may be suspected on the basis of clinical history or physical examination. When central occlusions are diagnosed and are felt to be clinically significant based on clinical data, these will need to be treated before restoring flow to the dialysis access; otherwise, the improved/increased

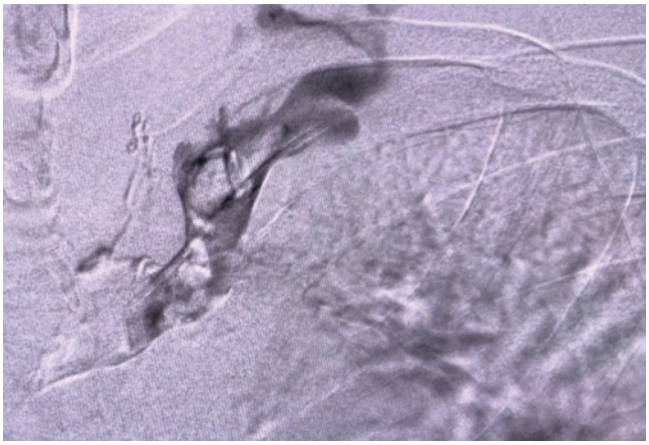

Fig. 16.1 *Central venogram* showing a large central clot; avoid removing the arterial plug since this is high risk for developing a pulmonary embolism

flow will cause significant hemodynamic effects centrally that will result in arm, facial, neck, or breast swelling; dilatation of superficial veins; or other similar changes reflective of flow obstruction. Please see Figs. 16.1 and 16.2.

Administer Medications

Depending on the point of service and specific practice setup, the anticoagulation and sedation/analgesia medications may be administered peripherally by the appropriate licensed health-care professional. We have chosen to have the interventionalist administer these medications in an open peripheral or central vein after having been able to cross the vein-graft anastomosis with a guidewire and ensuring that the central veins are not occluded or, if they are, have been successfully treated. This also reduces the oftentimes arduous task of finding and maintaining an intravenous line in the patient's contralateral arm. Standard heparin doses of between 2000 and 5000 IU are given at the start of the case, and this can be augmented with additional doses as clinically needed or titrated to a target ACT, generally ≥250 s.

Moderate sedation in the form of midazolam and fentanyl are given. In a series of over 12,000 patients treated, Beathard found the median dose of midazolam to be 3.0 mg and fentanyl 75 μg for most interventional cases, including thrombectomies [34]. These doses were only slightly less when used in combination rather than singly. With these doses, even high-risk patients tolerated the procedures without incident, and pain levels were adequately managed. A trained RN is given the responsibility of monitoring the patient and the response to sedation/analgesia.

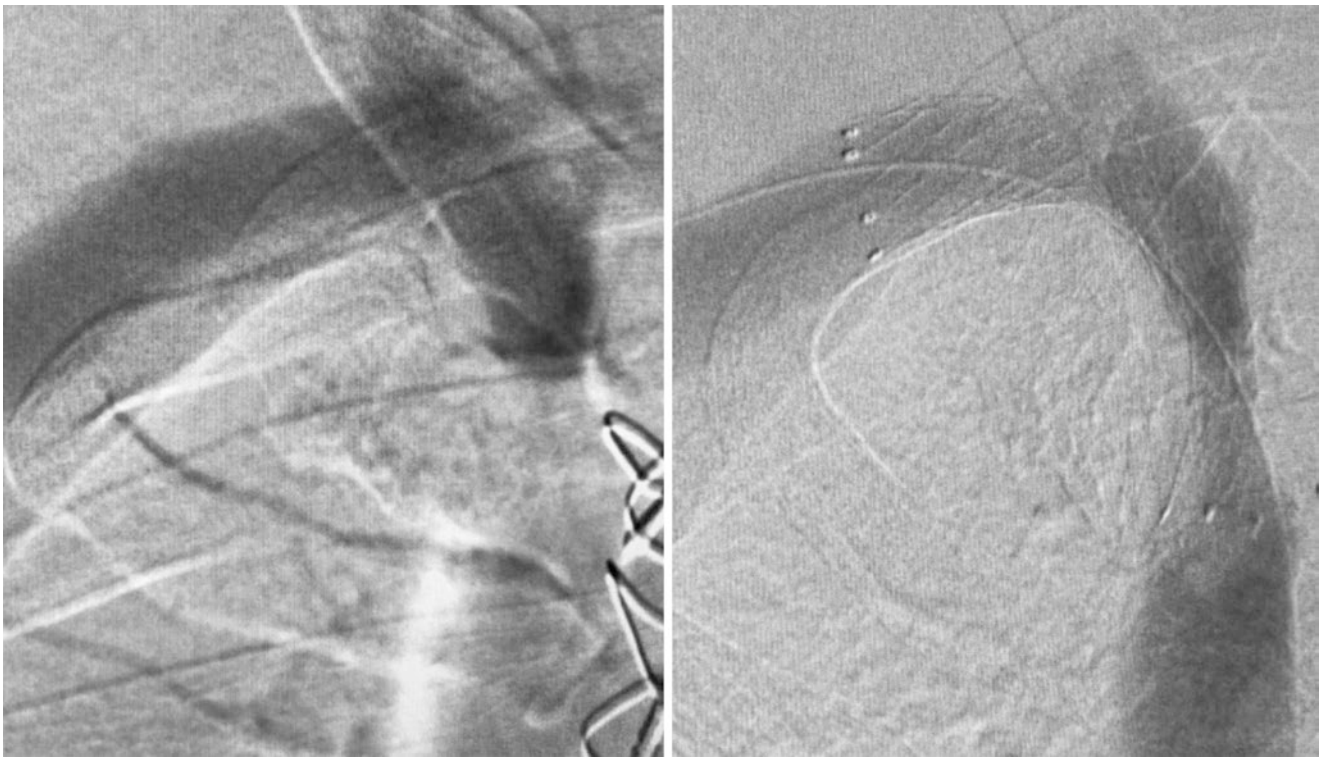

Fig. 16.2 *Central venogram*: on the left, a clinically significant central occlusion with right breast swelling and prominent superficial skin veins. On the right, recanalized and stented, the right breast swelling resolved

Treat Clot

As mentioned earlier, the amount of clot involved in a typical graft thrombectomy is typically around 5 mL. In a graft that is studded with pseudoaneurysms, the clot volume may be larger, but more importantly, the organized clot that is often laminated and adherent to the graft wall is removed only with the use of thrombectomy devices. In the vast majority of cases, maceration of the clot with an angioplasty balloon, thromboaspiration through a sheath or catheter, and use of locally instilled tPA are sufficient for clot removal. But in those cases where tenacious adherent clot remains and the clinical interventionalist feels it is necessary to remove, direct wall contact, rheolytic, or hydrodynamic mechanical devices are available (see Table 16.2). In our experience, these are not often necessary.

Table 16.2 Examples of some mechanical devices for access thrombectomy

Direct wall contact	Argon Medical Cleaner®
	Arrow-Trerotola® percutaneous thrombectomy device (PTD)
	Datascope ProLumen®
	Catañeda® OTW Brush
Hydrodynamic	eV3 Helix Clot Buster® (formerly Amplatz thrombectomy device)
	eV3 X-Sizer®
	Edwards Thromex®
Rheolytic	Boston-Scientific Oasis Thrombectomy System®
	Cordis Hydrolyser®
	Medrad Medical Angiojet® (AVX)
	Spectranetics ThromCat® Thrombectomy Catheter System

Many studies have evaluated the various devices [29, 35–45], and it is apparent that the success and patency rates are unrelated to the device but more to treating the underlying pathology leading up to the stenosis. Some studies have also shown a tendency to higher complication rates when devices are utilized, but this could well be related to a learning curve in the use of the device.

Alteplase (rt-PA, Genentech, South San Francisco, CA) is employed by some, but the indications and doses vary widely. The "lyse-and-wait" first described in 1997 [46] remains popular because it is simple, inexpensive, and easy to follow. We have inconsistently used this method, but when we have the doses have been between 2 and 4 mg of rt-PA. Others have reported similar doses of rt-PA [47, 48]. Using a multipurpose angiographic catheter or via the side arm of the sheath depending on location of cannulation, 2 mg of rt-PA is delivered close to the venous anastomosis and another 2 mg close to the arterial anastomosis. We dilute the drug in only 2 mL of sterile water in order to minimize

the volume delivered to a closed circuit and subsequent risk of arterial embolization. A small final volume of flush sterile water is used to empty the catheter or sheath of its "dead space." During injection of the lytic, we ensure that arterial embolization is minimized by digital manual pressure on the arterial inflow. The amount of time that the lytic is allowed to dwell varies, but a "no wait" technique compared to longer dwell times suggests that there is no difference in success or complication rates and similar 3-month primary, primary-assisted, and reanastomosis rates but statistically significant lower procedure and radiation times [49].

Thromboaspiration with or without maceration of the clot with an angioplasty balloon catheter is performed to remove as much clot as possible. Although the exact sequence of when this is performed varies between operators, the aim is to extract as much clot as possible and minimizing downstream embolization. This should therefore be done before there is both free and unimpeded flow of blood from arterial inflow to venous outflow. Some operators would do this after restoring arterial inflow but before addressing the venous anastomotic stenosis, while others would do this after angioplasty of the venous anastomosis but before dislodging the arterial platelet-fibrin plug.

Angioplasty of Vein-Graft Anastomosis

Since most dysfunctional dialysis prosthetic grafts will have the critical culprit lesion at the vein-graft anastomosis [50–53], some operators will preemptively perform an angioplasty in this area before restoring arterial inflow. Most prosthetic grafts used for hemodialysis access are 6 mm diameter, and the appropriately sized angioplasty balloon catheter used will be a 7 or 8 mm diameter.

Remove Arterial Platelet-Fibrin Plug

Following the second cannulation directed towards the arterial inflow, the fibrin plug at the arterial anastomosis is removed using a compliant Fogarty embolectomy catheter. They are both over-the-wire and plain versions of the catheter. The choice of one over the other depends on operator preference but should be flavored by anatomy, amount of manipulation needed to cross the anastomosis, and the security obtained by having a wire across a treated area. The arterial plug is a whitish dense tissue made up of fibrin and platelets and may be aspirated out of the sidearm of the arterial sheath. If performed under fluoroscopy, the compliant balloon will be noted to deform as it crosses the anastomosis and dislodges the plug. This motion is performed until the plug is retrieved, until the inflated balloon pulls back with minimal resistance, or until no further clots are aspirated. Please see Fig. 16.3.

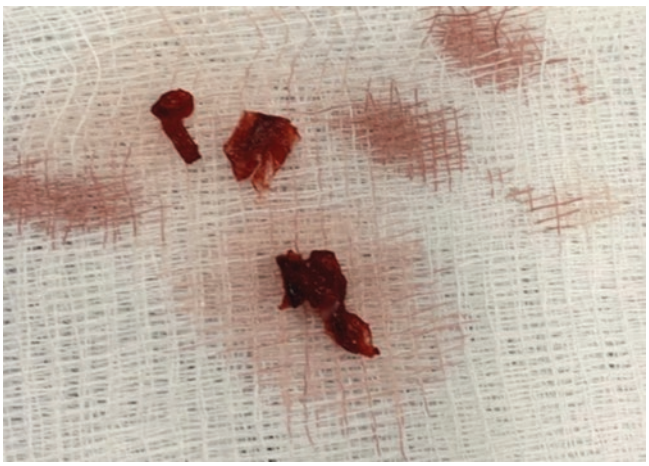

Fig. 16.3 *Arterial plug* removed using a Fogarty balloon and aspirated from the arterial sheath

Evaluate Arterial Inflow

An antegrade arteriogram is performed in order to evaluate the inflow, the artery-graft anastomosis, the juxta-anastomotic stenosis, and the arterial limb of the graft. Additionally, if the patient develops symptoms consistent with a distal arterial embolus and/or peripheral pulses change in quality, the arteriogram should also evaluate the more distal arterial circulation. Appropriate treatment of the symptomatic embolus should be promptly initiated. How far cephalad the arterial inflow must be evaluated is dictated primarily by one's degree of suspicion based on clinical presentation, history, and physical examination. A recurrently thrombosed or dysfunctional graft without compelling evidence for outflow stenosis and hemodynamic or systemic prothrombotic diathesis should raise one's suspicion for an inflow pathology. A dedicated and deliberate evaluation of the inflow circuit should then ensue and identified lesions appropriately treated. Please see Figs. 16.4 and 16.5.

Evaluate Venous Outflow

At this point, circulation has been restored to the graft, and attention is turned to the efferent arm of the circuit. Angiography of the entire graft, the vein-graft anastomosis, and the venous effluent tract is performed and identified lesions appropriately treated. High-pressure balloon angioplasty with a 1–2 mm oversize of the balloon relative to the non-stenotic diameter of the vessel is standard. Some lesions

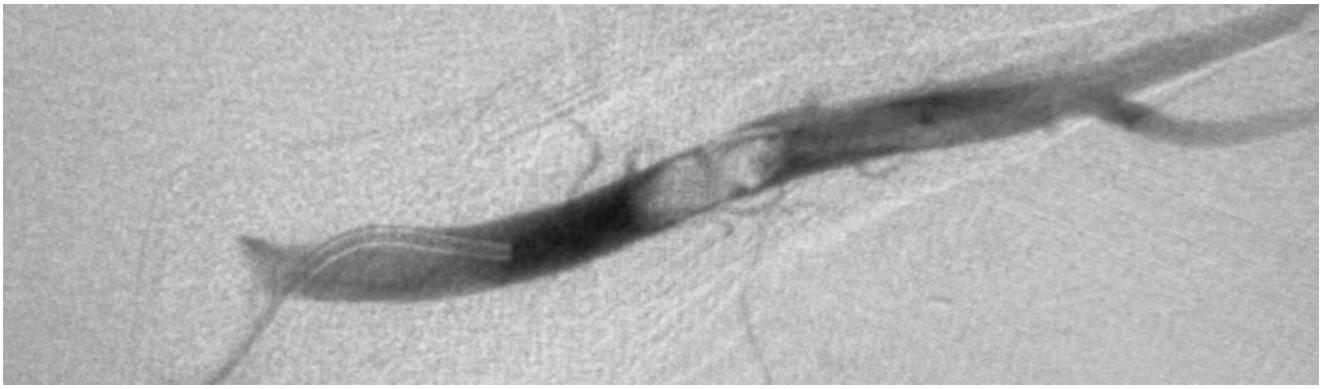

Fig. 16.4 *Distal arterial embolus* post thrombectomy, with acute hand numbness

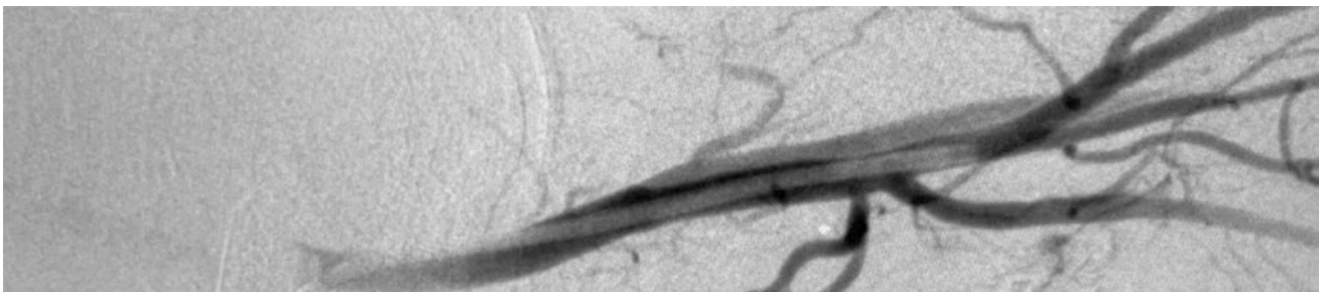

Fig. 16.5 *Appropriate treatment promptly initiated* using a Fogarty balloon with improvement inflow and resolution of hand symptoms

will necessitate ultra-high-pressure balloon angioplasty and endovascular stent placement. The indications, methodology, and precautions for stent placement in the setting of an access thrombectomy are no different than in a non-thrombosed access. These are covered in another section of this book.

Perform Completion Angiogram

Once radiologic and clinical parameters indicate that robust flow has been restored to the graft, a final completion angiogram is performed to assure that all physiologically significant lesions have been adequately treated and that there are no complications for which further treatment is required. Based on the completion angiogram, additional studies or surgical referrals may be considered. Endovascular treatment is an essential and important aspect of dialysis vascular access care but should not supplant surgical evaluation and management. Indeed, the most successful vascular access programs have seamlessly integrated endovascular and surgical approaches at all stages of care.

Autogenous Fistula Thrombectomy

We have described the prototypic thrombectomy approach to a prosthetic dialysis graft. The approach to the thrombosed autogenous fistula, however, is more nuanced and will require a greater degree of operator technical proficiency and clinical acumen.

There are a few important differences between the prosthetic graft and the autogenous fistula that make the approach to thrombectomy different.

Anatomy

While the anatomy of the anastomoses and the inflow/outflow arms are fairly straightforward for a graft, they are variable and may be quite complex for the fistula, for example, the fistula may have a radial arterial anastomosis with a transposed basilic vein or a translocated vein. A proximal radial artery may be anastomosed to the median antebrachial vein which will drain off cephalad and caudad to a number of different veins.

Anastomosis

The prosthetic graft has an artery-graft and a vein-graft anastomoses. Occasionally, one may encounter a graft-graft anastomosis if a patch angioplasty or a bridge graft may have been part of a surgical revision. The autogenous fistula, however, has only the artery-vein anastomosis. In the absence of flow, predicting anatomy of the anastomosis can be daunting. Please see Figs. 16.6 and 16.7.

Lesions

While most dysfunctional grafts will exhibit critical stenosis of the venous anastomosis, the distribution of culprit lesions in the fistula is far more variable [53–55]. A review of the dialysis treatment record with a focused evaluation of laboratory and clinical data may prepare the physician by anticipating the location of the lesions. Nonetheless, even after flow is restored to the fistula, an assessment of the radiologic and clinical record to explain the thrombosis of the fistula may remain challenging.

Thrombectomy

While the graft will almost always require removal of the arterial fibrin-platelet plug in order to restore flow, the fistula may not always require this maneuver. Sometimes, an angioplasty of an occlusive artery-vein or juxta-anastomotic stenosis may be sufficient to restore flow. This is particularly true of the radiocephalic fistula where the arterial inflow pathology tends to be more prominent [53–55].

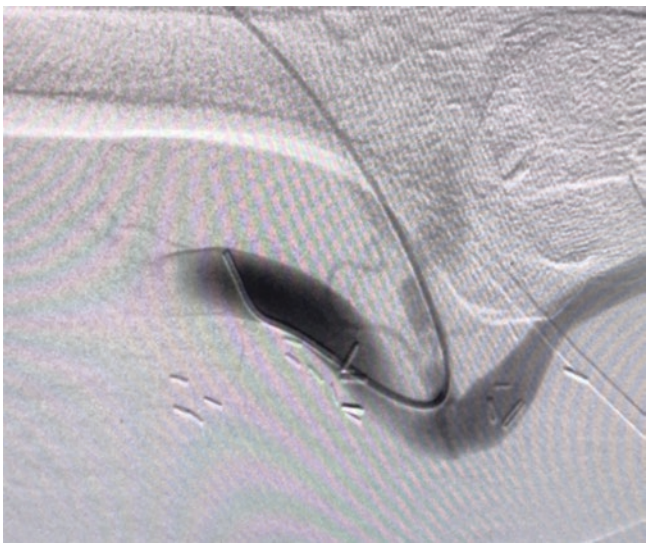

Fig. 16.6 *Arteriogram* of a clotted brachiocephalic fistula. No flow seen into the cephalic vein

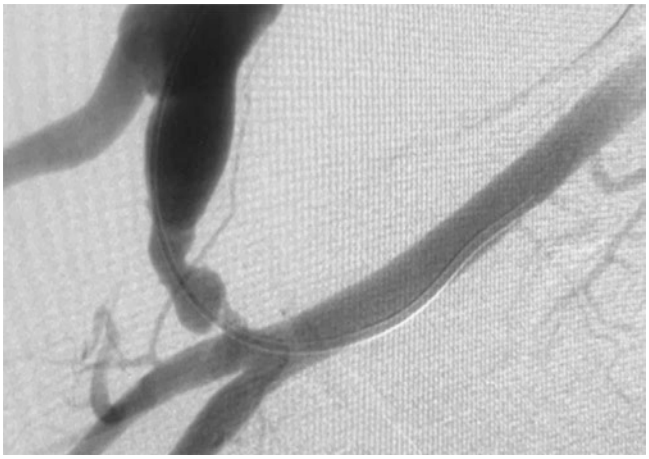

Fig. 16.7 *Post thrombectomy arteriogram of the brachial artery*. Resistance encountered using a Fogarty balloon to remove the arterial plug. Post intervention arteriogram showing a high grade peri-anastomotic stenosis which resulted in clotting of the fistula

Angioplasty

Thrombosed fistulas can vary in size from the immature to the mega-fistula. In the absence of flow, it becomes more difficult to predict the diameter of the vessels and makes angioplasty more complicated. One can size the balloons for the central vessels based on expected normal values [56], but there can be a wide variation in the presence of high-flow fistulas.

Clot Volume

The volume of thrombus within the graft is fairly predictable and is fortunately not typically a large amount. However, the clot burden in a fistula can vary widely, especially with the mega-fistulas. In these instances, the risk of embolization to the pulmonary circulation is significantly higher and must factor in one's approach to the thrombectomy. When the clot burden is predicted to be higher, one must attempt to protect the pulmonary circulation by removing as much clot as possible before restoring flow. Use of thrombolytics, with or without a mechanical device, would be an option in this regard.

HeRO® Graft Thrombectomy

The HeRO® graft (Hemodialysis Reliable Outflow, Hemosphere, Eden Prairie, MN) is a hybrid device approved by the FDA for dialysis-dependent patients whose vascular options have been exhausted. One such indication is central venous stenosis that is poorly responsive to endovascular therapy or is rapidly recurrent, and patient is deemed too

high risk for surgical bypass. There is a single anastomosis of the 6 mm ID graft to the feeding artery, but the venous end is a length of nitinol-reinforced silicone-coated catheter with an ID of 5 mm and opens to the mid-right atrium. The circuit can thrombose because of poor perfusion pressures, but intra-graft or artery-graft stenoses contributing to or causing thrombosis have also been seen.

The findings on examination of a thrombosed HeRO® graft are similar to that of a prosthetic graft. Palpation will show absence of thrill or pulse and auscultation, the absence of bruit. Barring any graft infection, the percutaneous thrombectomy of this device is fairly simple and straightforward and generally follows the same steps as outlined earlier.

The differences in approach between the thrombectomy of the prosthetic graft and the HeRO® graft are the following.

Removal of Thrombus

Using an 80 cm 3 Fr OTW Fogarty catheter of a 5 × 40 angioplasty balloon catheter, the thrombus is drawn from the tip of the catheter in the mid-RA distally to the venous cannulation site in the graft segment. It is suggested that the balloon be followed under fluoroscopy while being pulled to assure that the untethered end of the HeRO® device is not mobilized out of its intended location. If the tip of the catheter is dragged by the Fogarty or angioplasty balloon, deflating the balloon to less than nominal pressure and size will alleviate this problem.

Angioplasty

Because there is no graft-vein anastomosis, the intra-artery, artery-graft, and intra-graft lesions are the lesions one must evaluate as possibly causing or contributing to the thrombosis. Low flow from marginal cardiac function, poor perfusion pressures, or other similar systemic problems should also be considered.

Anticoagulation

Empirically, the amount of heparin needed for the thrombectomy of this device is typically less than that of a prosthetic graft.

Summary

The thrombectomy of a dialysis prosthetic graft or autogenous fistula demands the clinical interventionalist a command of a variety of techniques and tools, coupled with an understanding of the patient's specific clinical history and presentation. The thrombosis of a vascular access is the culmination of progressively critical anatomic and physiologic aberrations that must be identified and corrected if the intervention is expected to be durable. The physician's ability to return the patient to optimum dialysis vascular care depends on a meticulous and assiduous search for and correction of these factors.

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