

STATE-OF-THE-ART PAPER

Catheter Interventions for Hemodialysis Fistulas and Grafts

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More than 1 in 1,000 patients in the U.S. has end-stage renal disease, and most patients who require renal-replacement therapy undergo hemodialysis. By the year 2020, more than 750,000 patients are expected to have end-stage renal disease, and over 500,000 will require hemodialysis. The greatest limitation of hemodialysis is the finite durability of hemodialysis accesses, which on average remain patent for <3 years but are the lifeline for hemodialysis patients. Catheter-based interventions are successful in restoring flow in more than 80% of hemodialysis accesses that undergo thrombosis and have replaced surgical revision as the treatment of choice for failing or thrombosed accesses. Catheter-based interventions have improved the quality of life for hemodialysis patients by reducing the need for temporary hemodialysis catheters and have prolonged total survival time by preserving existing access sites and by saving venous segments for future access creation. This review discusses the pathophysiology of dialysis access failure, presents the success rates of catheter-based treatments, and illustrates the interventional approaches for treating failing and thrombosed fistulas and grafts. (J Am Coll Cardiol Intv 2010;3:1–11) © 2010 by the American College of Cardiology Foundation

More than 350,000 persons require hemodialysis for end-stage renal disease in the U.S., and by the year 2020, more than 750,000 patients are expected to need renal-replacement therapy (1). With the rising incidence of end-stage renal disease, the creation of hemodialysis accesses has become the most commonly performed type of vascular surgery (2), but <50% of all hemodialysis accesses remain patent for 3 years (3). Catheter-based interventions are more than 80% successful in restoring flow each time thrombosis occurs and, after publication of the Dialysis Outcomes Quality Initiative guidelines in 1997 (4), catheter-based therapy has gradually replaced surgical revision as the treatment of choice for failing or thrombosed fistulas and grafts. With the rising number of patients requiring hemodialysis, the total outlay for maintaining hemodialysis vascular access already exceeds \$1 billion in the U.S. and is rising at more than 6% per year (5).

This report reviews the pathogenesis, indications, and outcomes for catheter-based interventions for hemodialysis-access failure. Many techniques illustrated in the review exist in the endovascular skill set and are readily adaptable for treating failing or thrombosed hemodialysis accesses. By using catheter-based therapies, endovascular specialists fulfill a critical need by maintaining vascular access patency for the vulnerable population of hemodialysis patients, who consider their fistulas and grafts to be a lifeline.

Anatomy, Pathogenesis, and Incidence of Access Failure

The identification of a suitable site for permanent access placement depends on the adequacy of the venous and arterial anatomy. The selection of a particular access is based on evidence favoring the formation of an autogenous arteriovenous fistula whenever possible before resorting to the use of a polytetrafluoroethylene (PTFE) graft (6–8). The selection of a particular location is based on the recommended sequence of using the nondominant arm before the dominant arm, the forearm before

the upper arm, and the upper extremity before the lower extremity (2); but most hemodialysis patients ultimately will have functioning and failed access sites in multiple locations. **Anatomy.** An autogenous arteriovenous fistula is surgically formed by directly anastomosing an endogenous artery to an outflow vein. The pertinent venous anatomy in the upper extremity is straightforward and involves the basilic and cephalic veins (Fig. 1). The basilic vein courses along the medial aspect of the forearm, continues in the upper arm, and then courses into the axillary vein. The cephalic vein arises along the lateral aspect of the forearm, continues in the upper arm, and then drains into the axillary vein after traversing the pectoral groove. The axillary vein continues as the subclavian vein and then becomes the brachiocephalic vein at the confluence of the internal jugular vein. The confluence of the right and left brachiocephalic veins defines the origin of the superior vena cava.

The arterial anatomy of the upper extremity is similarly straightforward (Fig. 2). The subclavian artery courses under the clavicle, becomes the axillary artery, and then becomes the brachial artery. The brachial artery divides into the radial and ulnar arteries distal to the level of the antecubital crease.

Abbreviations and Acronyms

PTA = percutaneous
transluminal angioplasty

PTFE = polytetrafluoroethylene

Autogenous arteriovenous fistulas have several different configurations. In the forearm, the common type is the Brescia-Cimino radiocephalic fistula constructed at the wrist between the radial artery and the cephalic vein (Fig. 3), usually in an end-

to-side configuration. Another configuration uses the proximal radial artery anastomosed in a side-to-side manner with the median antebrachial vein (9). In the upper arm, the basilic vein may be mobilized and tunneled laterally and superficially to form a transposed brachio basilic fistula.

Prosthetic arteriovenous grafts also have several configurations. An arm graft, which is created by surgically interposing a PTFE connector between the brachial artery and an arm vein, may have either a looped or straight configuration. The most common forearm bridge-graft pattern is the medial-to-lateral configuration of the brachiocephalic graft (Fig. 3). The most common upper-arm bridge-graft pattern is the lateral-to-medial configuration of the brachio-basilic graft. Other configurations involve a position in the thigh in which a loop of PTFE is connected end-to-side with superficial femoral artery and end-to-end with the greater saphenous vein.

Pathophysiology of access failure. Primary failure of an autogenous arteriovenous fistula, defined as an access that never provided reliable hemodialysis after surgical creation, occurs in up to 50% of cases (10–14). Fistula failure is more common in diabetic or elderly patients. In diabetic patients, the patency of upper-arm brachiocephalic and transposed

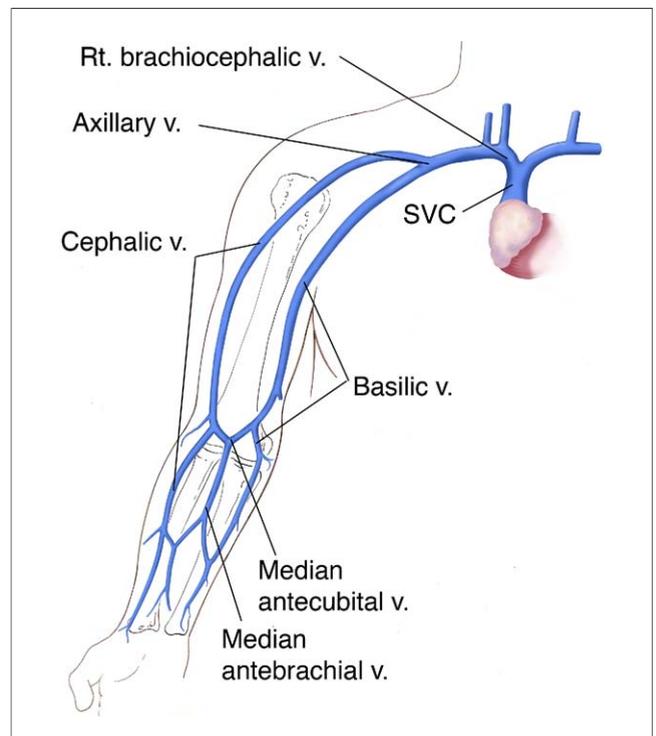


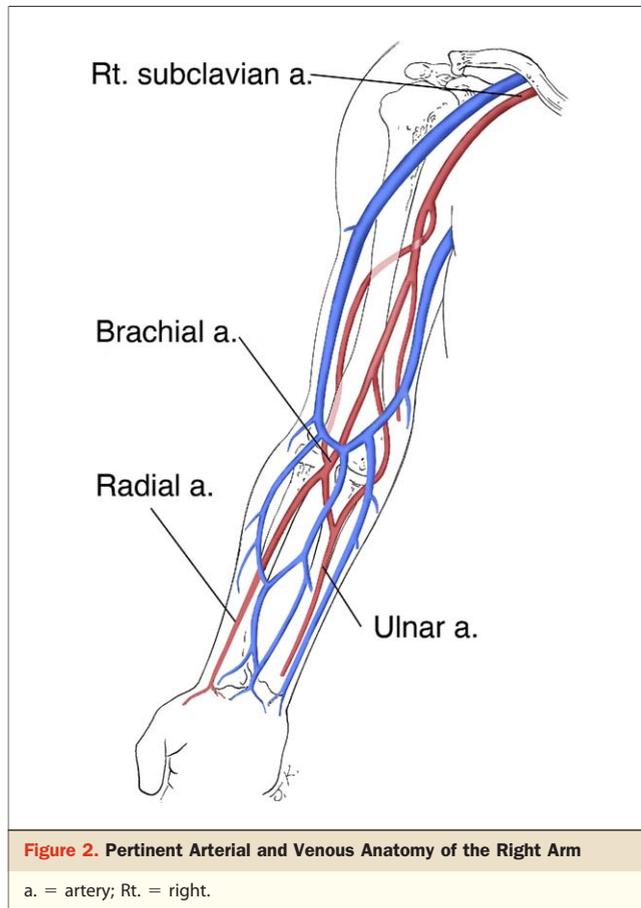
Figure 1. Venous Anatomy of the Right Arm

Rt. = right; SVC = superior vena cava; v. = vein.

basilic vein fistulas at 18 months may be better than the patency of forearm fistulas (78% vs. 33%) (15).

The failure of autogenous arteriovenous fistulas to mature within several months of surgical creation has been attributed to several etiologies, but the presence of an inflow stenosis caused by neointimal hyperplasia at the juxta-anastomotic site (16) is a mechanism amenable to interventional therapy (17–19). Percutaneous transluminal angioplasty of hypoplastic fistulas may increase blood flow, lead to adaptive remodeling, and allow hemodialysis to be carried out successfully (20).

The late failure of either autogenous arteriovenous fistulas or prosthetic grafts is caused by the appearance of a stenosis within an outflow vein that reduces flow and leads to thrombosis. The underlying mechanism is the marked increase in shear stress in the thin-walled outflow vein, which triggers focal fibromuscular hyperplasia and causes a fibrotic venous lesion to appear (21,22). The pathogenic stenoses causing access failure occur in various locations, but the most common site in prosthetic arteriovenous grafts is at the anastomosis between the graft and outflow vein, as identified in 47% (23,24) to 60% of cases (25), or in the outflow vein itself in autogenous arteriovenous fistulas. When critical flow reduction and clotting ensue, the bulk of the thrombus is probably red thrombus, which is rich in fibrin and red cells and easily extracted with rheolytic



methods or pulse-spray thrombolysis, but a platelet-rich white clot at the arterial anastomosis may require mechanical removal with Fogarty thrombectomy in 35% to 60% of cases to restore flow (23,26,27).

Although secondary thrombosis is a commonly associated finding in failed hemodialysis accesses, primary thrombosis of chronically used accesses is unusual. Primary thrombosis may be seen during the hypercoagulable state associated with hyperfibrinogenemia after major surgery or after profound hypotension associated with sepsis. Other nonanatomic causes of access thrombosis are excessive post-dialysis fistula compression, polycythemia, hypovolemia, or thrombophilic conditions associated with Factor V Leiden or the antiphospholipid syndrome.

In summary, 2 modes of hemodialysis-access failure are amenable to interventional treatment (Fig. 4). In newly placed fistulas, the development of an arteriovenous anastomotic stenosis prevents flow-mediated vasodilation, enlargement, and maturation of the fistula. In chronically used accesses, the development of an outflow-vein stenosis causes flow reduction, stasis, and thrombosis.

Incidence of access failure. Autogenous fistulas have a higher rate of primary failure than prosthetic grafts (~50% vs. 15%),

but the long-term patency of fistulas is superior to that of grafts. Autogenous fistulas fail after a median of 3 to 7 years, whereas prosthetic arteriovenous grafts fail after a median lifetime of only 12 to 18 months (28-31). For this reason, professional societies from several countries recommend an autogenous fistula as the access of choice for hemodialysis (6-8).

Signs of Access Failure

The National Kidney Foundation-Dialysis Outcomes Quality Initiative (32) recommends an organized program to identify failing fistulas and grafts. Monitoring is carried out with regular physical examinations and assessment of dialysis adequacy. Surveillance refers to the performance of noninvasive testing to gather further information about access structure and function. Diagnostic testing refers to the performance of procedures, usually invasive, to define access anatomy and hemodynamics.

Monitoring. Physical examination performed on a weekly basis by nephrologists can identify the presence of inflow or outflow stenoses in hemodialysis accesses. The mature

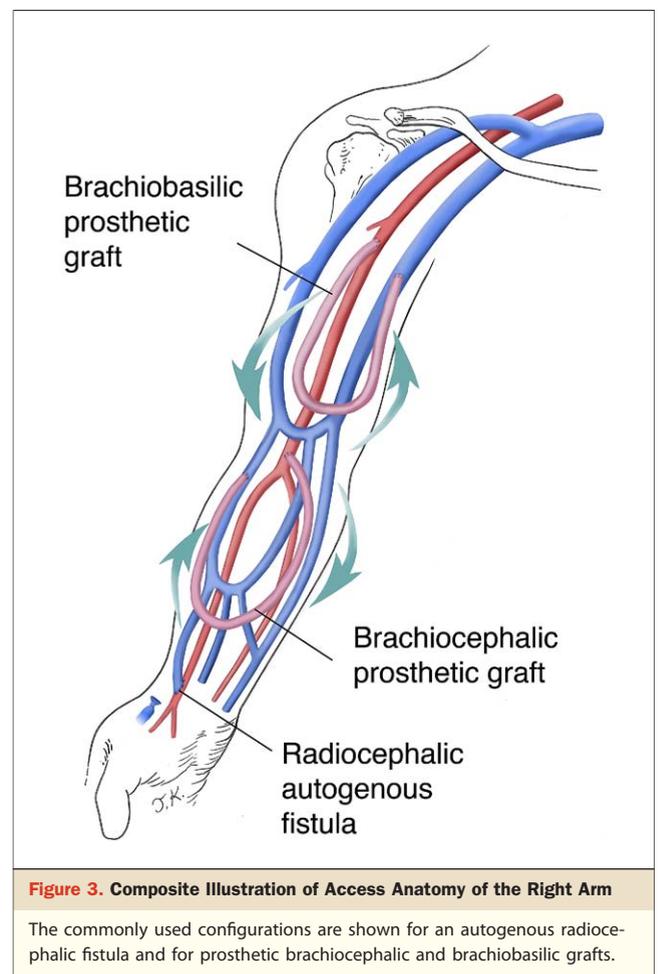
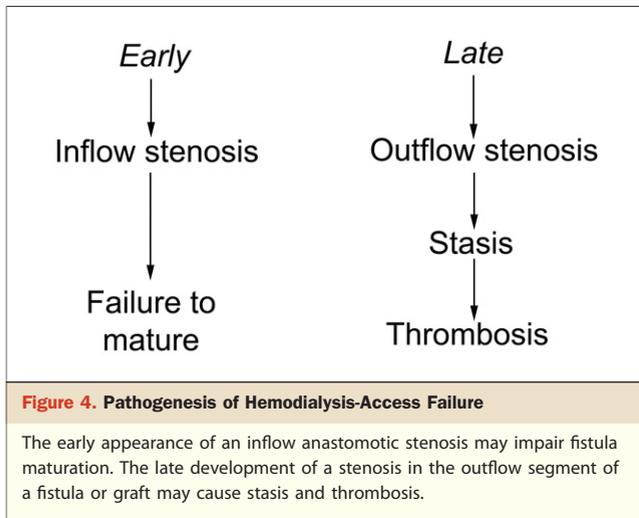


Figure 3. Composite Illustration of Access Anatomy of the Right Arm

The commonly used configurations are shown for an autogenous radiocephalic fistula and for prosthetic brachiocephalic and brachiocephalic grafts.



fistula or graft should course conspicuously in the subcutaneous tissue and have a prominent thrill and continuous medium-pitched bruit similar to the continuous murmur of a patent ductus arteriosus. Disappearance of the thrill and bruit is diagnostic of hemodialysis-access thrombosis.

Increased post-dialysis bleeding suggests the presence of an outflow stenosis. A prominent pulsation over the access is abnormal and may signify elevated pressure within the access caused by an outflow stenosis. This may be accompanied by a change in the bruit on serial examinations from a continuous medium-pitched bruit to a short high-pitched bruit. Multiple aneurysmal segments in the distribution of a large, mature, serpiginous fistula or graft used for many years may actually be pseudoaneurysms caused by impaired hemostasis and chronic venous hypertension. Marked fibrotic venous wall thickening may produce palpable subcutaneous nodules anywhere along the course of an outflow vein.

Marked arm edema usually indicates dual venous obstruction in both the cephalic and basilic veins or an isolated occlusion of a central vein related to prior central venous catheter placement in the axillary, subclavian, or brachiocephalic veins or in the superior vena cava. Depending on the level of central venous obstruction, this may be accompanied by the development of facial plethora or extensive venous collateral veins on the arm or chest wall.

The inability to insert dialysis needles into a patent but hypoplastic fistula or the finding of a soft bruit and fine thrill over a recently created fistula may indicate the presence of an anastomotic inflow stenosis. The coexistence of inflow and outflow stenoses may produce mixed physical findings, balanced hemodynamics, and difficult needle entry for hemodialysis.

Infection. Signs of infection are indicated by the presence of cellulitis, fluctuance, skin breakdown, or purulent discharge. Mild isolated erythema without tenderness or edema is usually not a sign of infection, but uremia may mask fever or

leukocytosis. Anorexia, weight loss, and myalgias may be constitutional symptoms suggestive of infection. Access infection is a contraindication to interventional treatment because of the resistance of infected thrombus to endovascular therapies and the risk of sepsis.

Surveillance. Hemodynamic surveillance should prompt referral for angiographic evaluation if dynamic venous pressures on hemodialysis exceed 120 mm Hg, fistula flow falls to <500 ml/min, graft flow decreases to <650 ml/min, or access blood flow falls by more than 25%. Evidence of incomplete dialysis may be suggested by the recirculation fraction using urea concentrations or clinical parameters such as body weight, volume status, or serum potassium concentration, but these are probably relatively late predictors of graft failure and become abnormal at the time of impending thrombosis (8). The observation of rising pressures >340 cm H₂O at a constant flow of 200 ml/min may also indicate the presence of an outflow stenosis (8). The enthusiasm for regular ultrasonographic surveillance for early detection of access failure (33) has been tempered by the expense, limited availability, and high likelihood of excessive referral for invasive testing (32).

Pre-emptive invasive evaluation. The ability of early angiography and pre-emptive angioplasty to prevent access thrombosis remains uncertain. In 1 study (34) of 21 patients with prosthetic arteriovenous accesses that had not previously clotted or required intervention, pre-emptive angioplasty reduced the risk of thrombosis from 44% to 10% per 100 patient-years ($p = 0.01$). Another study (35) reported that prophylactic angioplasty ($n = 32$) was superior to the standard treatment of fistulas ($n = 30$) in reducing thrombosis rate from 25% to 16% per 100 patient-years. Tessitore et al. (36) evaluated the cost-effectiveness of access blood-flow measurements and pre-emptive angioplasty in 159 patients and observed that a 3-fold increase in the number of angiographic procedures offset a 77% reduction in thrombosis events and a 65% reduction in fistula loss, thus defining an “economically dominant therapy” (i.e., cost-saving).

Several randomized studies have suggested that early angiography fails to prevent access thrombosis. A prospective randomized trial (37) of 64 patients monitored with monthly static venous-to-systolic blood pressure ratios compared prophylactic angioplasty with the strategy of delayed invasive management at the time of thrombosis and observed similar rates of thrombosis rates and access loss (37). Another randomized trial (38) of 112 patients compared monthly access blood-flow measurements with standard surveillance and reported that the greater number of interventions performed in the surveillance group than in the control group did not reduce the rate of access thrombosis (41% vs. 51% per 100 patient-years, $p = \text{NS}$). A recent economic analysis (39) has suggested that early angiography

and pre-emptive percutaneous transluminal angioplasty (PTA) produced a decline in access thrombosis from 27.6 to 22.0 events per 100 patient-years ($p = 0.029$) but the net cost was \$34,586 per 100 patient-years and the incremental cost-effectiveness ratio for invasive surveillance was \$6,177 per thrombosis event avoided. The Vascular Access Work Group has concluded that as a preventive strategy, “there is considerable debate concerning whether PTA interventions improve long-term outcomes” (40).

Interventional Therapy

Angiographic evaluation is generally recommended if monitoring or surveillance suggests that thrombosis either is imminent or has already occurred. The inability to insert dialysis needles in patent but hypoplastic fistulas is an indication for angiographic evaluation. Access thrombosis should trigger urgent angiographic referral before a single hemodialysis treatment is missed (Table 1). Contraindications to angiographic management of thrombosed accesses include the presence of a right-to-left intracardiac shunt, pulmonary hypertension, infected access, or surgical revision <30 days before referral.

Interventional procedure. The approach for thrombosed fistulas and grafts requires a sequential series of endovascular procedures (Table 2). Percutaneous needle entry into the occluded fistula or graft is straightforward, even though no flashback of blood occurs and no contrast or saline can be injected. The clotted access is ballotable. The usual entry sites are characterized by thickened skin and healing needle tracks. If a guidewire cannot advance easily through an 18-gauge thin-wall needle, an alternative approach is to enter the graft with a 4-F micropuncture set (Cook, Inc., Bloomington, Indiana) and exchange for a full-size sheath. In either case, a distinct “pop” is felt when the dura is penetrated with a needle. It is important to avoid puncturing the back wall of the graft because an external hematoma may compress the access.

The accompanying figures illustrate the standard thrombectomy methods for prosthetic arteriovenous grafts, but the techniques are immediately transferable for declotting autogenous fistulas as well. The procedure is begun by placing two 6-F sheaths within the access, 1 in the direction

Table 2. The 4-Step Approach for Thrombosed Hemodialysis Fistulas and Grafts

1. Thrombectomy of outflow and inflow segments
2. Angiography of fistula or graft and central veins
3. Percutaneous transluminal angioplasty of venous outflow stenosis
4. Fogarty thrombectomy of adherent clot at the arterial inflow anastomosis

of the arterial inflow and the other in the direction of the venous outflow (Fig. 5). The term “cross-sheath technique” is commonly used but misleading because the tips of the sheaths face each other but do not overlap. Thrombus within a segment of overlapping sheath tips would be inaccessible to catheter thrombectomy.

Advancement of 0.018-inch hydrophilic guide wires (V18, Boston Scientific, Natick, Massachusetts) in the outflow and inflow directions provides tracks for the catheters needed for thrombectomy and treatment (Fig. 5). If it is difficult to identify or penetrate the outflow venous stenosis with the 0.018-inch guidewire or to enter the inflow artery using fluoroscopy alone, replacement of the wire with a 5-F multipurpose catheter for limited angiography is useful. Large amounts of contrast must never be injected into a clotted access because thrombus will protrude into the inflow artery and embolize distally to the hand. After limited angiography is performed, penetration of the resistant stenosis can usually be achieved with a 0.035-inch hydrophilic curved wire and then replaced with the 0.018-inch guidewire for thrombectomy.

Step 1: thrombectomy. Various mechanical and thrombolytic approaches are available to declot thrombosed accesses. The devices include pulse-spray infusion catheters (Cook, Inc.), pulse-spray side-slit catheters (Angiodynamics, Inc., Glens Falls, New York), the Amplatz Thrombectomy Device (Microvena, White Bear Lake, Minnesota), the Arrow-Terrotola Percutaneous Thrombectomy Device (Arrow International, Reading, Pennsylvania), and the Gelbfish Endo-Vac device (Neovascular Technology, New York, New York). Rheolytic thrombectomy with the Possis AngioJet (Possis Medical, Minneapolis, Minnesota) is popular because of its widespread availability in cardiac catheterization laboratories and its rapid ability to declot grafts with the 60-cm, AngioJet AVX catheter (Possis Medical).

Rheolytic thrombectomy should be performed first in the direction of venous outflow (Fig. 6). This is immediately followed by thrombectomy in the direction of the arterial inflow (Fig. 7). If the blunt facing tip of 1 sheath prevents catheter advancement, insertion of sheath dilator presents a smoother transition for advancing the thrombectomy catheter.

Step 2: angiography and angioplasty. After flow is achieved, the access sheaths are flushed clear with saline and the pathogenic stenosis can be identified using angiography (Fig. 8). Venous PTA in the arm requires 6- to 10-mm balloons. Venous stenoses are characterized by extensive

Table 1. Specific Indications for Catheter Intervention of Hemodialysis Accesses

Early failure of hypoplastic autogenous arteriovenous fistulas
Inflow stenosis at the arteriovenous anastomosis
Early development of venous outflow stenosis
Late failure of chronic fistulas and grafts
Access thrombosis (urgent)
Isolated venous or outflow anastomotic stenosis
Arm edema
Central vein or double outlet stenosis

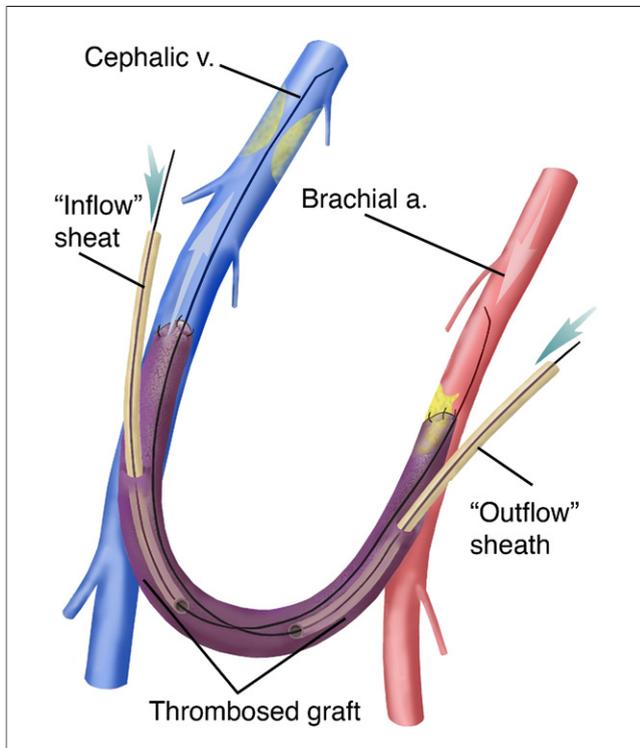


Figure 5. Facing-Sheaths Technique for Thrombosed Prosthetic Grafts

A 6-F sheath is inserted into the access near the arterial anastomosis and directed into the direction of the outflow, and a 6-F sheath is inserted into the access near the venous anastomosis in the direction of the inflow. Guidewires are advanced in the direction of the inflow and outflow under fluoroscopic guidance. No contrast is injected into a thrombosed access. Abbreviations as in Figures 1 and 2.

fibrosis and the need for ultra-high pressure balloon inflations (41) or cutting balloon atherotomy for optimal treatment (42–44). High-pressure, noncompliant balloons (Conquest or Dorado, Bard Peripheral Vascular Inc., Tempe, Arizona) with rated burst pressures of 20 to 24 atm can be used. Cutting Balloons (Boston Scientific) can be used when high-pressure balloons are unsuccessful (42–44), but the use of a peripheral cutting balloon device in 1 study (45) was associated with an increased risk of rupture.

Step 3: Fogarty thrombectomy. An important step to restore adequate flow involves the use of mechanical thrombectomy. The over-the-wire 4-F Fogarty Thru-Lumen Embolectomy Catheter (Edwards Lifesciences, Irvine, California) is almost always required to extract resistant thrombus from the arterial inflow (Fig. 9). After the embolectomy catheter has been used, the disrupted thrombus lodges against the sheaths and can be removed with rheolytic thrombectomy. Pulmonary embolization is rarely encountered. Petronis et al. (46) observed no scintigraphic evidence of pulmonary embolism in a systematic evaluation of patients after pharmacologic and mechanical declotting of thrombosed hemodialysis accesses.

The problem of persistent thrombosis and no reflow after treatment is challenging but may be successfully treated with repeated passes with Fogarty embolectomy catheter through the inflow anastomosis. The catheter must be withdrawn forcefully to dislodge the resistant inflow thrombus. If this fails and pressure within the access remains low, PTA of the arterial inflow anastomosis with a 6-mm balloon can be tried. Another cause of low fistula or graft pressure is a stenosis within the native radial or brachial artery, which may be amenable to PTA.

Step 4: central venography. The final step entails venography of the outflow and central veins. This is necessary to rule out the presence of a central stenosis that may have been caused by the previous use of hemodialysis catheters. However, treatment of incidental central vein stenoses remains controversial. Levit et al. (47) evaluated the success of pre-emptive angioplasty or stenting for central venous stenoses ipsilateral to hemodialysis accesses in 35 patients who underwent 86 angiograms over a 6-year period. Angioplasty or stenting of asymptomatic stenoses was associated with more rapid stenosis progression and escalation of lesions than the strategy of watchful waiting. If central venous stenoses are clinically significant and causing arm edema (Fig. 10), they may be suitable for treatment with large-diameter devices such the XXL balloon (Boston Sci-

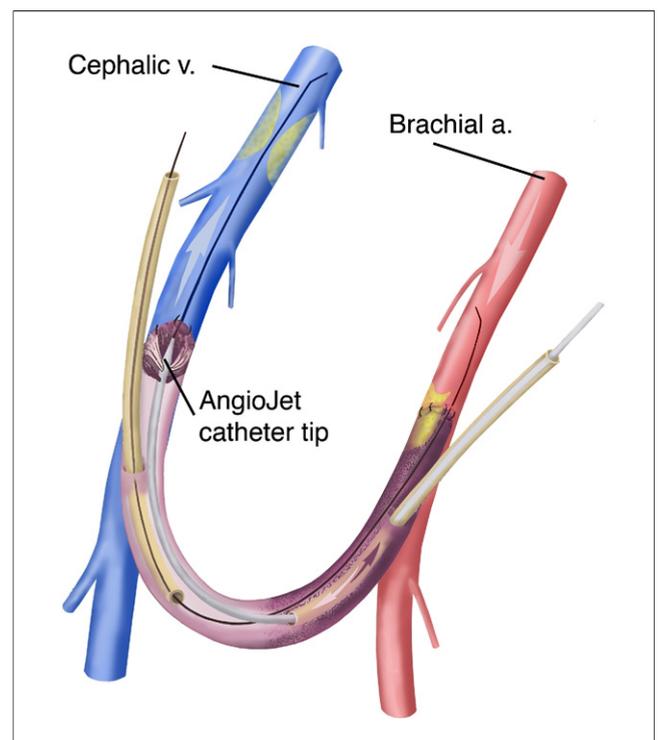


Figure 6. Thrombectomy of Outflow Segment

The AngioJet AVX catheter (Possis Medical) is activated and advanced over the guidewire in the direction of the venous outflow and then withdrawn at a rate of 1 mm/s. Abbreviations as in Figures 1 and 2.

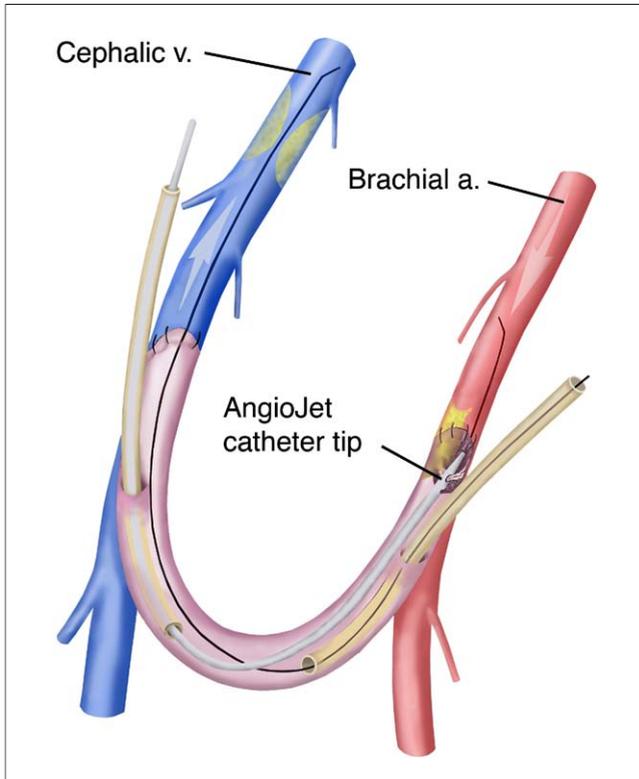


Figure 7. Thrombectomy of Inflow Segment

The AngioJet catheter (Possis Medical) is activated and advanced over the guidewire in the direction of the arterial inflow and then withdrawn. Abbreviations as in Figures 1 and 2.

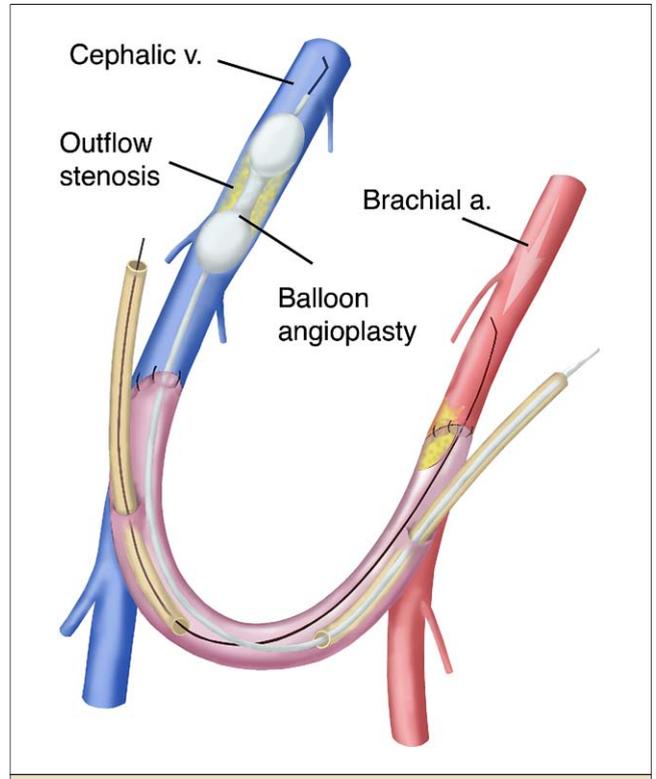


Figure 8. Angioplasty of Stenosis

After flow has been restored, angiography of the entire graft can be completed. The stenosis causing stasis and thrombosis of the graft can be identified and treated with balloon angioplasty. Abbreviations as in Figures 1 and 2.

entific Medi-Tech, Natick, Massachusetts), the high-pressure Atlas balloon (Bard Peripheral Vascular), the Sentinel Self-Expanding Nitinol Biliary Stent System or the Wallstent RX Biliary Endoprosthesis (both from Boston Scientific Corporation).

Final steps. At the end of the procedure, the decision remains whether to proceed with adjunctive stenting of the pathogenic stenosis in the outflow vein. In general, stenting provides no clear benefit over PTA alone and eliminates the option of using the stented vein for future surgical revision. Stents are thus reserved for severe recoil, venous perforations, or stenoses in surgically inaccessible veins. In a series of 65 patients treated with self-expanding stainless steel stents for failing or occluded dialysis accesses (48), repeat thrombosis occurred in 10% of patients within 1 week. In a series of 52 patients (49), the primary patency at 6 months was only 46% after placement of stents. Another study suggested that the provisional use of stents could successfully salvage failed PTA procedures (23). More studies are needed to determine whether nitinol or covered stents have better long-term patency than stainless steel stents (50,51).

The final pressure achieved in the access may determine long-term success. Access pressures that are significantly higher than one-third of systemic pressure are associated

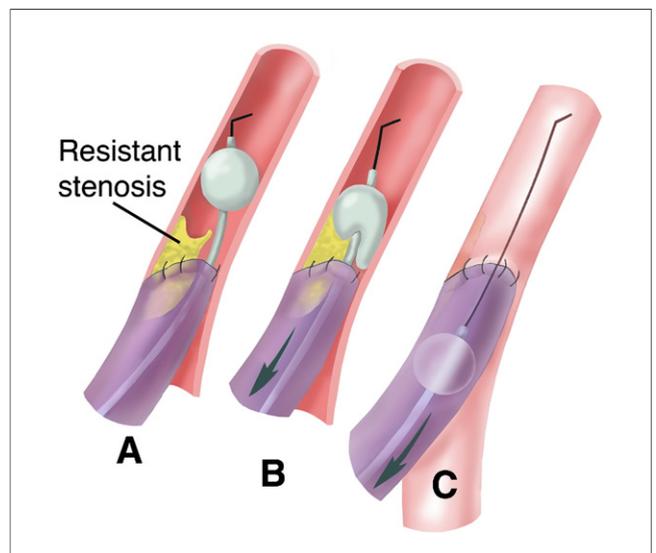


Figure 9. Fogarty Thrombectomy of Resistant Thrombus

A 4-F over-the-wire Fogarty catheter is advanced into the brachial artery beyond the resistant inflow stenosis, and the balloon is inflated (A). The catheter is forcefully withdrawn (B), dislodging the resistant thrombus (C).

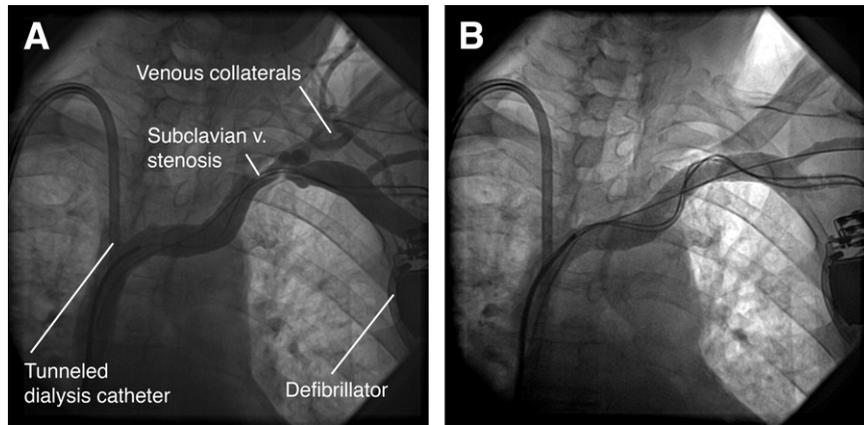


Figure 10. Central Venous Stenting

Recurrent restenosis after repeat balloon angioplasty caused by the presence of defibrillator leads ipsilateral to a fistula was associated with marked arm edema and venous collaterals (A), which were no longer visible after stent implantation (B). v. = vein.

with lower rates of long-term patency than lower access pressures (23,52).

At the conclusion of the procedure with demonstration of a widely patent graft with excellent flow, intradermal Z-sutures are placed and tightened to control bleeding after sheath withdrawal. Sutures are removed in 1 to 4 days.

Adjunctive pharmacology. The medical management during invasive treatment is minimal. Aspirin 325 mg is given orally before the procedure. For thrombosed accesses, heparin 5,000 U is given intravenously during the procedure. For nonthrombosed accesses, lower doses of heparin can be considered,

especially if the risk of bleeding or perforation is increased, as in newly placed unused fistulas. Antibiotic prophylaxis with cefuroxime 1 g intravenously is recommended. Vancomycin 1 g intravenously over 0.5 h can be used if an allergy to cephalosporins exists. Warfarin is recommended for the secondary prevention of access thrombosis if no stenosis is found.

Success Rates and Complications

In a recent series (39), catheter-based interventions (Fig. 11) resulted in successful hemodialysis for at least 30 days

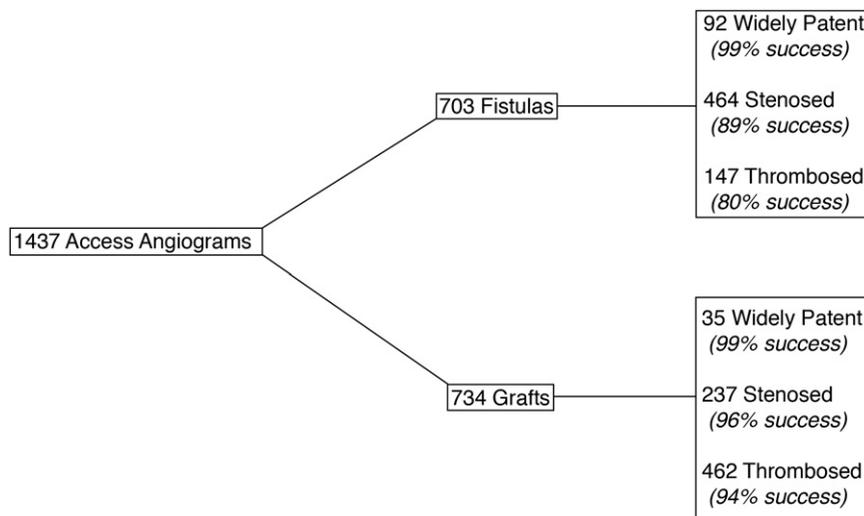


Figure 11. Success Rates

In a consecutive series of 1,437 angiographic procedures, the success of catheter-based interventions is shown resulting in successful hemodialysis for at least 30 days without repeat angiography or surgical revision.

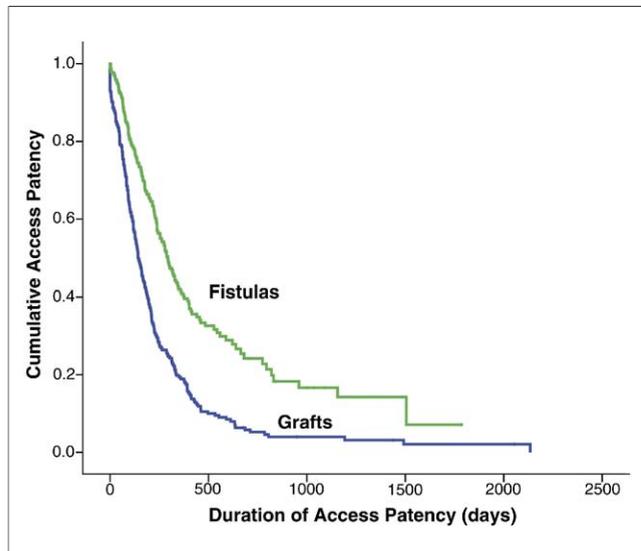


Figure 12. Duration of Access Patency

Kaplan-Meier analysis of patency after percutaneous intervention is presented for 611 autogenous arteriovenous fistulas and 699 prosthetic grafts undergoing pre-emptive angioplasty or thrombectomy for thrombosis.

without repeat angiography or surgical intervention in 1,317 of 1,437 procedures (92%). The angiographic success rate was lower for thrombosed fistulas than for thrombosed grafts (80% vs. 94%, $p = 0.001$). Beathard (53) reported 6-month patency rates of 61% and 1-year patency rate of 38% after a variety of catheter interventions by interventional nephrologists. Several studies (39,54) have shown that autogenous arteriovenous fistulas tend to have longer median patencies than prosthetic grafts after catheter-based intervention (Fig. 12). However, a history of access thrombosis impairs long-term patency and reduces the advantage of autogenous arteriovenous fistulas over prosthetic grafts so that both types of accesses have similar patency durations of about 3 months after declotting (23).

Complications. Catheter treatment of hemodialysis accesses is generally safe. Complications can be categorized as Grade I hematomas (minor, nonflow-limiting), Grade II hematomas (flow-limiting), or Grade III hematomas and perforations (55). Vessel rupture and free perforation have been identified as potential catastrophic complications of percutaneous treatment (45,56–60) and occur in <1% of cases. One study (45) suggested that using a balloon catheter more than 2 mm larger than the diameter of the hemodialysis access or using peripheral cutting balloons increased the risk of rupture or perforation. In some cases of rupture, complete hemostasis requires the insertion of the Fluency Plus Tracheobronchial Stent Graft (Bard Peripheral Vascular), a polyethylene terephthalate-covered stent (WallGraft, Boston Scientific), or a Viabahn Endoprosthesis (W.L. Gore and Associates, Flagstaff, Arizona), after switching to 10- or 11-F sheaths (Fig. 13).

Future Directions

Medical therapy. Several randomized trials of antithrombotic agents to prevent thrombosis have been reported, but none has been unequivocally successful in preventing thrombosis of dialysis accesses. In a randomized trial of 877 patients (10), clopidogrel was no better than placebo for allowing autogenous arteriovenous fistulas to be used successfully for hemodialysis (38% vs. 40%). In another randomized trial of 649 patients (14), dipyridamole was modestly better than placebo in achieved primary unassisted patency of autogenous arteriovenous fistulas at 1 year (28% vs. 23%).

Several experimental methods under investigation have been designed to enhance the long-term patency of arteriovenous grafts by targeting intimal hyperplasia in the venous

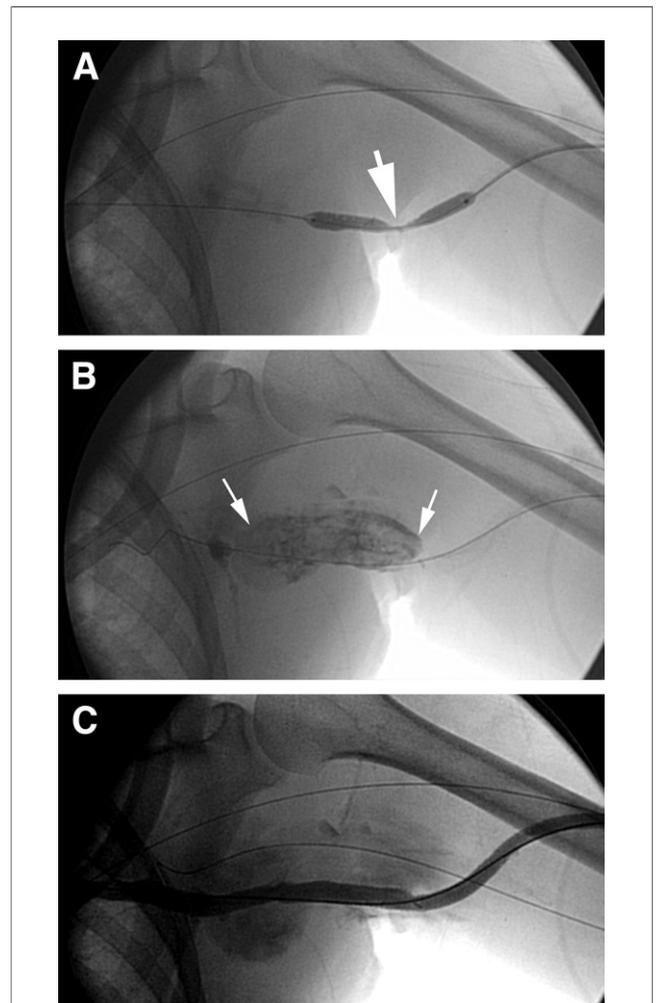


Figure 13. Venous Rupture

Dilation of a stenosis in the basilic vein (A, arrow) was followed by free rupture and extravascular contrast (B, arrows) and successfully treated with a covered stent (C).

outflow. External beam radiation has been tried, but in small series of patients this was unable to reduce the likelihood of repeat restenosis (61).

The use of endothelial cell seeding of PTFE grafts is based on the concept that these cells can form a biologically active lining and reduce the release of mitogens that lead to vascular smooth muscle proliferation. Lining of PTFE grafts with anti-CD34 antibodies, which bind bone marrow-derived circulating CD34(+) endothelial progenitor cells, resulted in almost complete endothelialization of the grafts but paradoxically increased in neointimal hyperplasia at the graft-vein anastomosis (62).

Summary

The catheter-based treatment of thrombosed and failing hemodialysis accesses achieves success in more than 80% of cases and allows patients to undergo immediate hemodialysis without the need for placement of temporary dialysis catheters or surgical consumption of additional venous conduits. Life on hemodialysis can be a Hobbesian existence—"nasty, poor, brutish, and short"—but the availability of catheter-based approaches within many medical centers has preserved a lifeline and improved the quality of life for hemodialysis patients.

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REFERENCES

- Collins AJ, Foley RN, Herzog C, et al. United States Renal System 2008 annual data report abstract. *Am J Kidney Dis* 2009;53 Suppl 1:S8-374.
- Weiswasser JM, Sidawy AN. Strategies of arteriovenous dialysis access. In: Rutherford RB, ed. *Vascular Surgery*. Philadelphia, PA: Elsevier Saunders, 2005:1669-76.
- Schwab SJ. Vascular access for hemodialysis. *Kidney Int* 1999;55:2078-90.
- National Kidney Foundation. DOQI clinical practice guidelines for vascular access. *Am J Kidney Dis* 1997;30 Suppl 3:S150-91.
- Collins AJ, Foley RN, Herzog C, et al. Excerpts from the United States Renal Data System 2007 annual data report. *Am J Kidney Dis* 2008;51 Suppl 1:S1-320.
- Jindal K, Chan CT, Deziel C, et al. Hemodialysis clinical practice guidelines for the Canadian Society of Nephrology. *J Am Soc Nephrol* 2006;17:S1-27.
- Ohira S, Naito H, Amono I, et al. 2005 Japanese Society for Dialysis Therapy guidelines for vascular access construction and repair for chronic hemodialysis. *Ther Apher Dial* 2006;10:449-62.
- Vascular Access 2006 Work Group. NKF-DOQI clinical practice guidelines for vascular access, update 2006. *Am J Kidney Dis* 2006;48 Suppl 1:S176-306.
- Bruns SD, Jennings WC. Proximal radial artery as inflow site for native arteriovenous fistula. *J Am Coll Surg* 2003;197:58-63.
- Dember LM, Beck GJ, Allon M, et al. Effect of clopidogrel on early failure of arteriovenous fistulas for hemodialysis: a randomized controlled trial. *JAMA* 2008;299:2164-71.
- Asif A, Roy-Chaudhury P, Beathard GA. Early arteriovenous fistula failure: a logical proposal for when and how to intervene. *Clin J Am Soc Nephrol* 2006;1:332-9.
- Patel ST, Hughes J, Mills JL Sr. Failure of arteriovenous fistula maturation: an unintended consequence of exceeding Dialysis Outcome Quality Initiative guidelines for hemodialysis access. *J Vasc Surg* 2003;38:439-45.
- Berman SS, Gentile AT. Impact of secondary procedures in autogenous arteriovenous fistula maturation and maintenance. *J Vasc Surg* 2002;34:866-71.
- Dixon BS, Beck GJ, Vazquez MA, et al. Effect of dipyridamole plus aspirin on hemodialysis graft patency. *N Engl J Med* 2009;360:2191-201.
- Hakaim A, Nalbandian M, Scott T. Superior maturation and patency of primary brachiocephalic and transposed basilic vein arteriovenous fistulae in patients with diabetes. *J Vasc Surg* 1998;27:154-7.
- Roy-Chaudhury P, Arend L, Zhang J, et al. Neointimal hyperplasia in early arteriovenous fistula failure. *Am J Kidney Dis* 2007;50:782-90.
- Achkar K, Nassar GM. Salvage of a severely dysfunctional arteriovenous fistula with a strictured and occluded outflow tract. *Semin Dial* 2005;18:336-42.
- Beathard GA, Arnold P, Jackson J, Litchfield T. Aggressive treatment of early fistula failure. *Kidney Int* 2003;64:1487-94.
- Turmel-Rodrigues L, Mouton A, Birmel e B, et al. Salvage of immature forearm fistulas for haemodialysis by interventional radiology. *Nephrol Dial Transplant* 2001;16:2365-71.
- Bittl JA, von Mering GO, Feldman RL. Adaptive remodeling of hypoplastic hemodialysis fistulas salvaged with angioplasty. *Catheter Cardiovasc Interv* 2009;73:974-8.
- Swedberg SH, Brown BG, Sigley R, Wight TN, Gordon D, Nicholls SC. Intimal fibromuscular hyperplasia at the venous anastomosis of PTFE grafts in hemodialysis patients. Clinical, immunocytochemical, light and electron microscopic assessment. *Circulation* 1989;80:1726-36.
- Roy-Chaudhury P, Kelly BS, Miller MA, et al. Venous neointimal hyperplasia in polytetrafluoroethylene dialysis grafts. *Kidney Int* 2001;59:2325-34.
- Bittl JA, Feldman RL. Prospective assessment of hemodialysis access patency after percutaneous intervention: Cox proportional hazards analysis. *Catheter Cardiovasc Interv* 2005;66:309-15.
- Kanterman RY, Vesely TM, Pilgram TK, Guy BW, Windus DW, Picus D. Dialysis access grafts: anatomic location of venous stenosis and results of angioplasty. *Radiology* 1995;195:135-9.
- Beathard GA. Angioplasty for arteriovenous grafts and fistulae. *Semin Nephrol* 2002;22:202-10.
- Valji K. Transcatheter treatment of thrombosed hemodialysis access grafts. *AJR Am J Roentgenol* 1995;164:823-9.
- Asif A, Gadalean FN, Merrill D, et al. Inflow stenosis in arteriovenous fistulas and grafts: a multicenter, prospective study. *Kidney Int* 2005;67:1986-92.
- Huber TS, Carter JW, Carter RL, Seeger JM. Patency of autogenous and polytetrafluoroethylene upper extremity arteriovenous hemodialysis access: a systematic review. *J Vasc Surg* 2003;38:1005-11.
- Perera GB, Mueller MP, Kubaska SM, Wilson SE, Lawrence PF, Fujitani RM. Superiority of autogenous arteriovenous hemodialysis access: maintenance of function with fewer secondary interventions. *Ann Vasc Surg* 2004;18:66-73.
- Schwartz C, McBrayer C, Sloan J, Meneses P, Ennis W. Thrombosed dialysis grafts: comparison of treatment with transluminal angioplasty and surgical revision. *Radiology* 1995;194:337-41.
- Rodriguez JA, Armadans L, Ferrer E, et al. The function of permanent vascular access. *Nephrol Dial Transplant* 2000;15:402-8.
- Anonymous. NKF-K/DOQI clinical practice guidelines for vascular access: update 2000. *Am J Kidney Dis* 2001;37:S137-81.
- Malik J, Slavikova M, Svobodova J, Tuka V. Regular ultrasonographic screening significantly prolongs patency of PTFE grafts. *Kidney Int* 2005;67:1554-8.
- Martin LG, MacDonald MJ, Kikeri D, Cotsonis GA, Harker LA, Lumsden AB. Prophylactic angioplasty reduces thrombosis in virgin ePTFE arteriovenous dialysis grafts with greater than 50% stenosis:

- subset analysis of a prospectively randomized study. *J Vasc Interv Radiol* 1999;10:389-96.
35. Schwab SJ, Oliver MJ, Suhocki P, McCann R. Hemodialysis arteriovenous access: detection of stenosis and response to treatment by vascular access blood flow. *Kidney Int* 2001;59:358-62.
 36. Tessitore N, Bedogna V, Poli A, et al. Adding access blood flow surveillance to clinical monitoring reduces thrombosis rates and costs, and improves fistula patency in the short term: a controlled cohort study. *Nephrol Dial Transplant* 2008;23:3578-84.
 37. Dember LM, Holmberg EF, Kaufman JS. Randomized controlled trial of prophylactic repair of hemodialysis arteriovenous graft stenosis. *Kidney Int* 2004;66:390-8.
 38. Moist LM, Churchill DN, House AA, et al. Regular monitoring of access flow compared with monitoring of venous pressure fails to improve graft survival. *J Am Soc Nephrol* 2003;14:2645-53.
 39. Bittl JA, Cohen DJ, Seek MM, Feldman RL. Economic analysis of angiography and preemptive angioplasty to prevent hemodialysis-access thrombosis. *Catheter Cardiovasc Interv* 2009 Aug 7 [E-pub ahead of print].
 40. Vascular Access 2006 Work Group. NKF-DOQI clinical practice guidelines for vascular access, update 2006: clinical practice recommendations for guideline 4: detection of access dysfunction: monitoring, surveillance, and diagnostic testing. *Am J Kidney Dis* 2006;48 Suppl 1:S269-70.
 41. Trerotola SO, Kwak A, Clark TW, et al. Prospective study of balloon inflation pressures and other technical aspects of hemodialysis angioplasty. *J Vasc Interv Radiol* 2005;16:1613-8.
 42. Bittl JA, Feldman RL. Cutting balloon angioplasty for undilatable venous stenoses causing dialysis graft failure. *Catheter Cardiovasc Interv* 2003;58:524-6.
 43. Vesely TM, Siegel JB. Use of the peripheral cutting balloon to treat hemodialysis-related stenoses. *J Vasc Interv Radiol* 2005;16:1593-603.
 44. Wu CC, Sen SC. Cutting balloon angioplasty for resistant venous stenoses of dialysis access: immediate and patency results. *Catheter Cardiovasc Interv* 2008;71:250-4.
 45. Bittl JA. Venous rupture during percutaneous treatment of hemodialysis fistulas and grafts. *Catheter Cardiovasc Interv* 2009;74:1097-101.
 46. Petronis JD, Regan F, Briefel G, Simpson PM, Hess JM, Contoreggi CS. Ventilation-perfusion scintigraphic evaluation of pulmonary clot burden after percutaneous thrombolysis of clotted hemodialysis access grafts. *Am J Kidney Dis* 1999;34:207-11.
 47. Levit RD, Cohen RM, Kwak A, et al. Asymptomatic central venous stenosis in hemodialysis patients. *Radiology* 2006;238:1051-6.
 48. Vorwerk D, Guenther RW, Mann H, et al. Venous stenosis and occlusion in hemodialysis shunts: follow-up results of stent placement in 65 patients. *Radiology* 1995;195:140-6.
 49. Gray RJ, Horton KM, Dolmatch BL, et al. Use of Wallstents for hemodialysis access-related venous stenoses and occlusions untreatable with balloon angioplasty. *Radiology* 1995;195:479-84.
 50. Vogel PM, Parise C. SMART stent for salvage of hemodialysis access grafts. *J Vasc Interv Radiol* 2004;15:1051-60.
 51. Sreenarasimhaiah VP, Margassery SK, Martin KJ, Bander SJ. Salvage of thrombosed dialysis access grafts with venous anastomosis stents. *Kidney Int* 2005;67:678-84.
 52. Lilly RZ, Carlton D, Barker J, et al. Predictors of arteriovenous graft patency after radiologic intervention in hemodialysis patients. *Am J Kidney Dis* 2001;37:945-53.
 53. Beathard GA. Percutaneous transvenous angioplasty in the treatment of vascular access stenosis. *Kidney Int* 1992;42:1390-7.
 54. Woods JD, Turenne MN, Strawderman RL, et al. Vascular access survival among incident hemodialysis patients in the United States. *Am J Kidney Dis* 1997;30:50-7.
 55. Beathard GA. Management of complications of endovascular dialysis access procedures. *Semin Dial* 2003;16:309-13.
 56. Funaki B, Szymanski GX, Leef JA, Rosenblum JD, Burke R, Hackworth CA. Wallstent deployment to salvage dialysis graft thrombolysis complicated by venous rupture: early and intermediate results. *AJR Am J Roentgenol* 1997;169:1435-7.
 57. Rundback JH, Leonardo RF, Poplasky MR, Rozenblit G. Venous rupture complicating hemodialysis access angioplasty: percutaneous treatment and outcomes in seven patients. *AJR Am J Roentgenol* 1998;171:1081-4.
 58. Raynaud AC, Angel CY, Sapoval MR, Beyssen B, Pagny JY, Auguste M. Treatment of hemodialysis access rupture during PTA with Wallstent implantation. *J Vasc Interv Radiol* 1998;9:437-42.
 59. Sofocleous CT, Schur I, Koh E, et al. Percutaneous treatment of complications occurring during hemodialysis graft recanalization. *Eur J Radiol* 2003;47:237-46.
 60. Beathard GA, Litchfield T. Effectiveness and safety of dialysis vascular access procedures performed by interventional nephrologists. *Kidney Int* 2004;66:1622-32.
 61. Parikh S, Nori D, Rogers D, et al. External beam radiation therapy to prevent postangioplasty dialysis access restenosis: a feasibility study. *Cardiovasc Radiat Med* 1999;1:36-41.
 62. Rotmans JI, Heyliger JMM, Verhagen HJM, et al. In vivo seeding using anti-CD34 antibodies successfully accelerates endothelialization but stimulates intimal hyperplasia in porcine arteriovenous expanded polytetrafluoroethylene grafts. *Circulation* 2005;112:12-8.

Key Words: hemodialysis ■ fistula ■ prosthetic graft ■ end stage renal disease.