Vascular Access: Core Curriculum 2008

Ivan D. Maya, MD, and Michael Allon, MD

More than 90% of dialysis patients in the United States are on hemodialysis therapy. These patients require a reliable vascular access to deliver dialysis treatment thrice weekly. The ideal access would be: (1) easy to place, (2) ready for immediate use, (3) able to deliver sufficient blood flow to achieve adequate dialysis, (4) have a long patency, and (5) have a low rate of complications. None of the 3 types of vascular access (arteriovenous [AV] fistulas, AV grafts, and tunneled dialysis catheters) fulfill all these requirements (Table 1). Fistulas have the longest patency and require the fewest interventions after they are cannulated successfully for dialysis. However, they have the greatest primary failure rate and require the longest time for maturation (6 to 12 weeks). Grafts have a lower primary failure rate than fistulas and can be cannulated fairly quickly (within 2 to 3 weeks). However, they are prone to recurrent stenosis and thrombosis and have a greater frequency of salvage procedures (angioplasty, thrombectomy, and surgical revision) to maintain their long-term patency for dialysis. Finally, catheters are easily placed and can be used for dialysis immediately. However, they deliver lower blood flows, predispose to central vein stenosis, and have the greatest rates of thrombosis and infection. The Kidney Disease Outcomes Quality Initiative (KDOQI) Vascular Access guidelines promote an increase in fistula use for dialysis. An unintentional byproduct of increased fistula placement has been the concurrent increase in use of dialysis catheters. Optimizing vascular access outcomes requires advanced planning; close collaboration among nephrologists, access surgeons, radiologists, and dialysis staff; close monitoring and intervention for complications; and maintenance of prospective computerized access databases. Achieving this goal can be expedited by having dedicated access coordinators.

HISTORY

I. First hemodialysis: 1924
II. First vascular access: 1943
III. Quinton-Scribner shunt: 1960 (Fig 1)
IV. Brescia-Cimino fistula: 1966
V. Synthetic polytetrafluoroethylene (PTFE) AV grafts: 1970s
VI. Permanent tunneled cuffed indwelling hemodialysis catheters: 1980s
VII. Synthetic polyurethane (Vectra; Thoratec Laboratories Corporation, Pleasanton, CA) AV grafts: 1990s
VIII. KDOQI Vascular Access guidelines first publicized in 1997, revised in 2001 and 2006. Highlights include:
   A. Concerted measures to increase fistula placement and maturation
   B. Concerted efforts to minimize use of dialysis catheters
   C. Earlier guidelines strongly promoted surveillance for graft stenosis; latest version is more equivocal about its value

EPIDEMIOLOGY

I. More than 20% of dialysis patient hospitalizations are access related
II. Vascular access complications are associated with morbidity and mortality

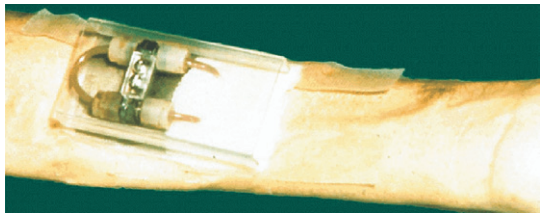

Figure 1. Scribner shunt. The invention that made long-term hemodialysis possible in the early days was the removable U-shaped Teflon shunt connecting an artery to a vein in the arm of a patient. Reproduced with permission from Blagg et al.[28]

From the Division of Nephrology, University of Alabama at Birmingham, Birmingham, AL.

Originally published online as doi:10.1053/j.ajkd.2007.10.046 on March 3, 2008.

Address correspondence to Michael Allon, MD, Division of Nephrology, PB, Rm 226, 728 Richard Arrington Blvd, Birmingham, AL 35294. E-mail: mdallon@uab.edu

0272-6386/08/5104-0022$34.00/0
doi:10.1053/j.ajkd.2007.10.046

Table 1. Comparison of Vascular Access Types

Feature	Fistula	Graft	Catheter
Primary failure rate (%)	20-50	10-20	<5
Time to first use (wk)	6-12	2-3	Immediate
Frequency of intervention	Very low	Moderate	High
Dialysis blood flow	Excellent	Excellent	Moderate
Frequency of thrombosis (after use for hemodialysis)	Very low	Moderate	High
Frequency of infection	Very low	Moderate (~8/100 patient-years)	Very high (~2 times/y/patient)
Longevity (after in use)	Longest (~5 y)	Intermediate (~2 y)	Shortest (<1 y)

III. Adjusted mortality is 40% to 70% greater in patients dialyzed through a catheter than those dialyzed through a fistula or graft. Mortality risk decreased in patients switched from catheter to AV access compared with patients who remain catheter dependent
IV. Fistula prevalence is lower in United States than Europe or Japan
V. Fistula prevalence varies by geographic region in United States:
   A. Northeast and Northwest: 49% to 57%
   B. Midwest: 36% to 42%
   C. Southwest: 36% to 48%
   D. Southeast: 30% to 35%
VI. 75% of US patients initiate dialysis therapy with a catheter
VII. Fistula First initiative has increased both fistula and catheter use in United States

PREOPERATIVE VASCULAR MAPPING

I. Routine preoperative vascular mapping with ultrasonography or venography increases fistula placement
II. High primary fistula failure persists despite preoperative mapping
III. Elements of preoperative mapping:
   A. Minimum vein diameter, 2.5 mm for fistula
   B. Minimum vein diameter, 4 mm for graft
   C. Minimum artery diameter, 2 mm for fistula or graft
   D. Exclude stenosis or thrombosis of proximal vein
IV. Order of preference of vascular access to be placed:
   A. Distal (radiocephalic) fistula
   B. Proximal (brachiocephalic) fistula
   C. Proximal transposed brachiobasilic fistula
   D. Upper-extremity graft
   E. Thigh graft
   F. Unusual grafts: necklace, unilateral chest wall

VASCULAR ACCESS MONITORING AND SURVEILLANCE

I. Identify access dysfunction as early as possible
II. Establish a monitoring/surveillance program
III. Clinical monitoring:
   A. Physical examination: absent thrill, abnormal bruit, or distal edema; pulsatile swelling aneurysm (fistula) or pseudoaneurysm (graft)
   B. Dialysis abnormalities: difficult cannulation, aspiration of clots, or prolonged bleeding from needle site
   C. Unexplained decrease in Kt/V on constant dialysis prescription
IV. Access surveillance parameters (abnormal values in parentheses):
   A. Static dialysis venous pressures (DVPs; ratio of DVP to systemic blood pressure >0.4)
   B. Access blood flow (<600 mL/min or decrease by >25% from baseline)
   C. Doppler ultrasound: peak systolic velocity (PSV) ratio greater than 2:1
   D. Dynamic DVP and recirculation are not useful
V. Positive predictive value for greater than 50% stenosis is 70% to 100% for clinical monitoring, static venous pressures, flow monitoring, or duplex ultrasound

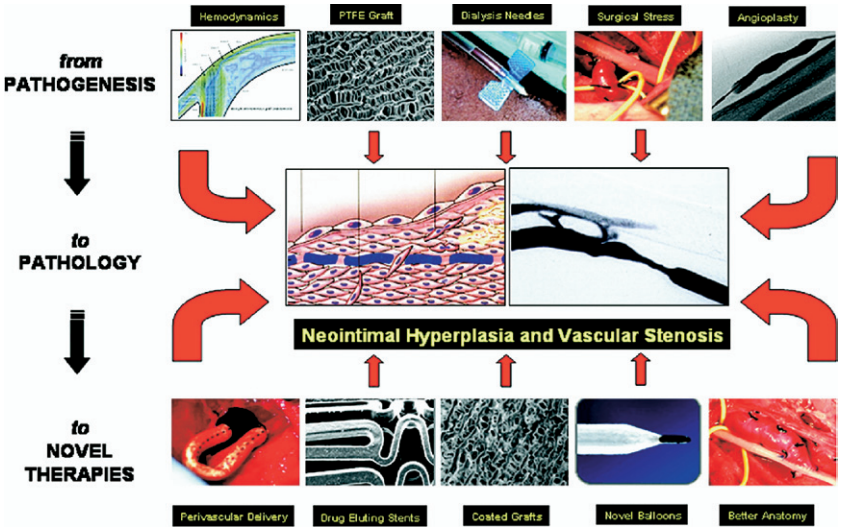

Figure 2. From pathogenesis to pathology to novel therapies. This figure identifies the different pathogenetic mechanisms that result in dialysis access stenosis and directs attention to potential novel therapies. Pathogenetic factors include hemodynamic and surgical stressors, inflammatory stimuli from dialysis needles and polytetrafluoroethylene (PTFE) graft material, and the unavoidable vascular injury that occurs at the time of angioplasty. Novel therapeutic modalities include perivascular drug delivery, drug-eluting stents, coated grafts, and novel balloons. Reproduced with permission from Roy-Chaudhury et al.[24]

VI. Grafts with abnormal monitoring or surveillance should be referred for angioplasty
VII. Preemptive angioplasty should be performed if greater than 50% stenosis (technical success: <30% residual stenosis)
VIII. Approximately 50% of grafts with stenosis remain patent without preemptive angioplasty
IX. Approximately 25% of graft thrombosis not preceded by abnormal monitoring/surveillance results
X. Five of 6 randomized clinical trials did not observe decreased graft thrombosis or prolonged graft patency with surveillance and preemptive angioplasty

PATHOGENESIS OF ACCESS STENOSIS

I. Stenosis due to venous neointimal hyperplasia (VNH)
II. VNH formed by smooth muscle cells, microfibroblasts, and microvessels
III. Cytokines (platelet-derived growth factor, endothelial growth factor, etc) modulate VNH progression
IV. Other potential factors for VNH:
  A. Hemodynamic turbulence and shear forces
  B. Dialysis needle injury
  C. Surgical vascular damage
  D. Uremia
  E. Vascular damage from angioplasty
  F. Expression of genes for cytokines
V. Novel therapies (local antiproliferative drug delivery systems) have decreased graft stenosis in animal models. Human studies are in progress (Fig 2)

AV FISTULAS

I. Direct anastomosis between artery and vein
II. Three major types: radiocephalic (forearm), brachiocephalic (upper arm), transposed brachiobasilic (upper arm; Fig 3)
III. Time to cannulation: 6 to 12 weeks
IV. High primary failure rate (~40%):
  A. Early thrombosis
  B. Failure to mature
  C. Steal syndrome (1% to 4%)
V. Factors associated with primary failure: age older than 65 years, female sex, nonwhite race, cardiovascular disease, peripheral vascular disease, obesity
VI. Postoperative ultrasound to evaluate maturation (4 to 6 weeks after surgery)
VII. Ultrasound criteria for maturity:
  A. Fistula diameter, 4 mm or greater
  B. Access flow, 500 mL/min or greater
  C. Distance from skin, 5 mm or less
VIII. Common anatomic lesions contributing to immaturity:
  A. Juxta-anastomotic stenosis (repair by angioplasty or surgical revision)

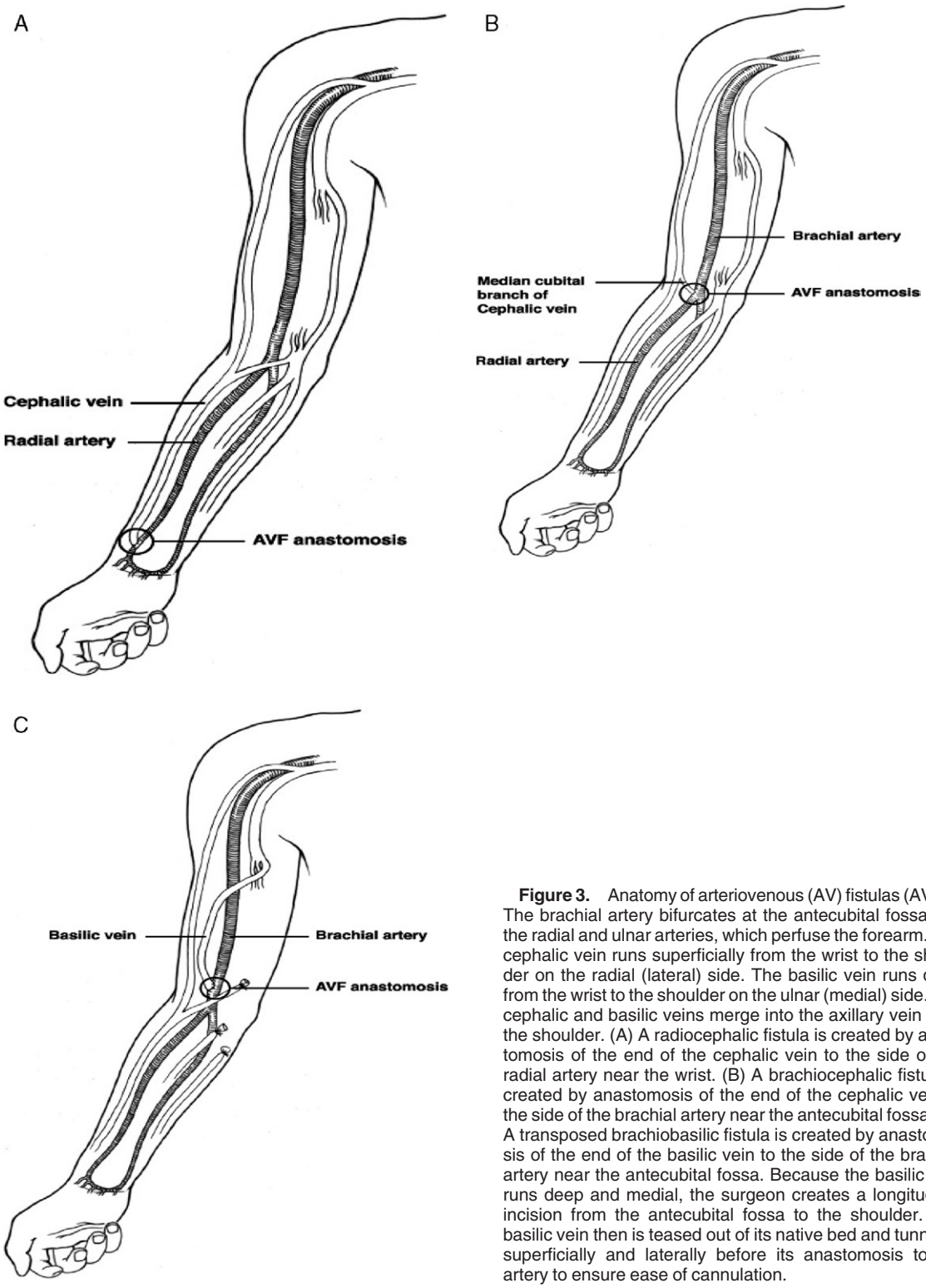

Figure 3. Anatomy of arteriovenous (AV) fistulas (AVFs). The brachial artery bifurcates at the antecubital fossa into the radial and ulnar arteries, which perfuse the forearm. The cephalic vein runs superficially from the wrist to the shoulder on the radial (lateral) side. The basilic vein runs deep from the wrist to the shoulder on the ulnar (medial) side. The cephalic and basilic veins merge into the axillary vein near the shoulder. (A) A radiocephalic fistula is created by anastomosis of the end of the cephalic vein to the side of the radial artery near the wrist. (B) A brachiocephalic fistula is created by anastomosis of the end of the cephalic vein to the side of the brachial artery near the antecubital fossa. (C) A transposed brachiobasilic fistula is created by anastomosis of the end of the basilic vein to the side of the brachial artery near the antecubital fossa. Because the basilic vein runs deep and medial, the surgeon creates a longitudinal incision from the antecubital fossa to the shoulder. The basilic vein then is teased out of its native bed and tunneled superficially and laterally before its anastomosis to the artery to ensure ease of cannulation.

B. Large accessory veins (can be ligated surgically or treated by coil embolization)
C. Excessively deep fistula (can be superficialized)

IX. Correction of anatomic lesions may convert immature to mature fistula
X. Late fistula failure is caused by stenosis:
1. 60% at the venous outlet
2. 25% at the arterial anastomosis
3. 5% at central vessels
4. Rarely, a large aneurysm (unrelated to stenosis) may cause late fistula failure if surgical revision is not feasible

XI. Fistulas require 3- to 4-fold fewer interventions than grafts to maintain long-term patency for dialysis
XII. Thrombosed fistula requires thrombectomy within 48 hours (high technical success)
XIII. Primary patency after thrombectomy: 27% to 81% at 6 months; 18% to 70% at 1 year

AV GRAFTS

I. PTFE bridge interposed between artery and vein
II. Time to first cannulation: 2 to 3 weeks (24 to 48 hours for Vectra)
III. Primary failure: 15% to 20% (less than for fistulas)
IV. Causes of graft failure:
A. Thrombosis (~80%)
B. Infection (~20%), usually requires surgical excision
C. Occasionally graft failure may be due to a large pseudoaneurysm that is leaking or infected

V. Underlying stenosis in most thrombosed grafts:
A. Venous anastomosis (~60%)
B. Venous outlet (15%)
C. Central veins (10%)
D. Intragraft (10%)
E. Arterial anastomosis (5%)

VI. Intervention-free patency after elective angioplasty: 70% to 85% at 3 months, 20% to 40% at 12 months
VII. Intervention-free patency after thrombectomy: 33% to 63% at 3 months, 10% to 39% at 6 months
VIII. Short-lived benefit of angioplasty: access flow back to preangioplasty level in 20% at 1 week and 40% at 1 month
IX. Stents may prolong patency in selected grafts (elastic lesion):
A. Metal stent (eg, nitinol) may require antiplatelet agent, such as clopidogrel
B. PTFE-covered stents potentially may improve patency compared with metal stents (limited data)

X. No clear advantage of bovine or cadaveric human vein grafts over PTFE grafts. Polyurethane (Vectra) grafts can be cannulated within 24 hours of use versus 2 weeks for PTFE grafts; they may be useful in patients with recurrent catheter dysfunction

HEMODIALYSIS CATHETERS

I. May be nontunneled (temporary) or tunneled (more permanent)
II. Double-lumen and semirigid catheters made of polyurethane, polyethylene, or PTFE
III. Preferred sites of insertion:
A. Right internal jugular vein
B. Left internal jugular (IJ) vein
C. Subclavian vein
D. Femoral vein (if all thoracic veins occluded)
E. Translumbar or transhepatic (last resort)

IV. Catheter thrombosis prevented by using intraluminal anticoagulant (heparin or citrate)
V. Catheter thrombosis treated by instillation of thrombolytic agents (urokinase or tissue-type plasminogen activator); catheter exchange if unsuccessful
VI. Approximately 25% symptomatic ipsilateral deep vein thrombosis with femoral catheter
VII. May cause central vein stenosis (more common with subclavian than IJ)
VIII. Central vein stenosis presents with ipsilateral upper-extremity edema. Some patients have prominent chest wall collateral veins
IX. Treatment of central vein stenosis by using angioplasty (poor patency with or without stent placement)
X. Fibrin sheaths may occasionally encase the catheter tip, thereby impairing flow.

Treatment requires balloon disruption or snare and removal of fibrin sheath by femoral approach

CATHETER-RELATED BACTEREMIA (CRB)

I. Less likely with tunneled than nontunneled catheters
II. Frequency: 2.0 to 5.5 episodes/1,000 catheter-days
III. Suspected when patient has fever or chills; confirmed by positive catheter and peripheral blood culture results
IV. Serious complications in 5% to 10% of patients with CRB: endocarditis, osteomyelitis, septic arthritis, epidural abscess, death
V. 60% to 70% caused by gram-positive organisms, 30% to 40% caused by gram-negative rods
VI. 3 weeks of intravenous (IV) antibiotics for uncomplicated CRB
VII. Systemic antibiotics alone result in 75% recurrent infection
VIII. Catheter removal mandatory with persistent fever or bacteremia on antibiotic therapy or tunnel-track infection
IX. Guidewire exchange of catheter possible if fever resolves on antibiotic therapy
X. Catheter biofilm is the major source of CRB
XI. Antibiotic lock (concentrated antibiotic-heparin solution instilled into catheter lumen after dialysis) can kill bacteria in the biofilm
XII. IV antibiotics plus antibiotic lock cures CRB with catheter salvage in approximately two thirds of cases (Fig 4)
XIII. Success of antibiotic lock depends on organism: 87% to 100% for gram-negative, 75% to 84% for *Staphylococcus epidermidis*, and 40% to 55% for *Staphylococcus aureus*

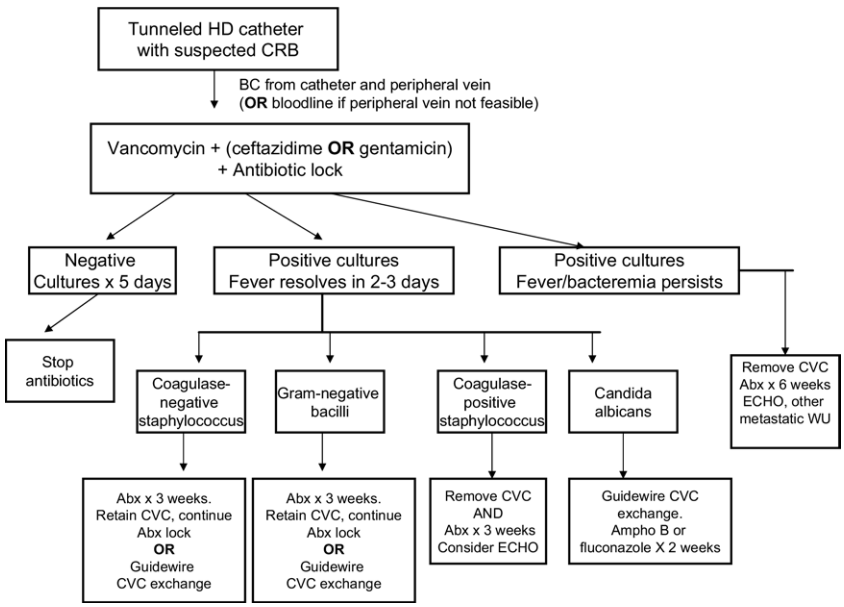

Figure 4. Clinical approach to management of dialysis catheter–related bacteremia (CRB). In catheter-dependent patients with suspected CRB (fever or rigors), after blood cultures (BCs) are obtained, empiric antibiotic therapy (Abx) is started with vancomycin (for coverage of methicillin-resistant *Staphylococcus* species) and ceftazidime or gentamicin (for broad-spectrum gram-negative coverage). In conjunction, an antibiotic lock (Fig 4) is instilled into each catheter lumen at the end of the dialysis session. Abx is discontinued in patients with negative culture results. If culture results are positive for coagulase-negative *Staphylococcus* species or gram-negative bacilli, the 2 treatment options are to: (1) continue appropriate systemic Abx with an antibiotic lock or (2) exchange the infected catheter over a new one over a guidewire. If fever and bacteremia persist despite antibiotic therapy, the catheter is removed and workup (WU) is initiated for metastatic infection. In infections caused by *Staphylococcus aureus* or *Candida* species, catheter replacement is mandatory. Abbreviations: HD, hemodialysis; CVC, central venous catheter; ECHO, echocardiography; Ampho B, amphotericin B. Adapted with permission from Allon.[2]

XIV. Prophylaxis of CRB was reported with a variety of antibiotic locks, 30% citrate, or taurolidine. None is currently approved by the US Food and Drug Administration for this indication

ACKNOWLEDGEMENTS

Support: None.
Financial Disclosure: None.

ADDITIONAL READINGS

1. National Kidney Foundation: KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Vascular Access 2006. Am J Kidney Dis 48:S176-S322, 2006 (suppl 1)

2. Allon M: Dialysis catheter-related bacteremia: Treatment and prophylaxis. Am J Kidney Dis 44:779-791, 2004

3. Allon M: Current management of vascular access. Clin J Am Soc Nephrol 2:786-800, 2007

4. Feldman HI, Kobrin S, Wasserstein A: Hemodialysis vascular access morbidity. J Am Soc Nephrol 7:523-535, 1996

5. Pisoni RL, Young EW, Dykstra DM, et al: Vascular access use in Europe and in the United States: Results from the DOPPS. Kidney Int 61:305-316, 2002

6. Stehman-Breen CO, Sherrard DJ, Gillen D, Caps M: Determinants of type and timing of initial permanent hemodialysis vascular access. Kidney Int 57:639-645, 2000

7. Allon M, Daugirdas JT, Depner TA, Greene T, Ornt D, Schwab SJ: Effect of change in vascular access on patient mortality in hemodialysis patients. Am J Kidney Dis 47:469-477, 2006

8. Xue JL, Dahl D, Ebben JP, Collins AJ: The association of initial hemodialysis access type with mortality outcomes in elderly Medicare ESRD patients. Am J Kidney Dis 42:1013-1019, 2003

9. Agarwal AK, Patel BM, Haddad NJ: Central vein stenosis: A nephrologist's perspective. Semin Dial 20:53-62, 2007

10. Allon M, Lockhart ME, Lilly RZ, et al: Effect of preoperative sonographic mapping on vascular access outcomes in hemodialysis patients. Kidney Int 60:2013-2020, 2001

11. Robbin ML, Gallichio ML, Deierhoi MH, Young CJ, Weber TM, Allon M: US vascular mapping before hemodialysis access placement. Radiology 217:83-88, 2000

12. Silva MB, Hobson RW, Pappas PJ, et al: A strategy for increasing use of autogenous hemodialysis access procedures: Impact of preoperative noninvasive evaluation. J Vasc Surg 27:302-308, 1998

13. Asif A, Cherla G, Merrill D, Cipleu CD, Briones P, Pennell P: Conversion of tunneled hemodialysis catheter-consigned patients to arteriovenous fistula. Kidney Int 67:2399-2406, 2005

14. Beathard GA, Arnold P, Jackson J, Litchfield T, for the Physician Operators Forum of RMS Lifeline: Aggressive treatment of early fistula failure. Kidney Int 64:1487-1494, 2003

15. Moist LM, Hemmelgarn BR, Lok CE: Relationship between blood flow in central venous catheters and hemodialysis adequacy. Clin J Am Soc Nephrol 1:965-971, 2006

16. Ram SJ, Work J, Caldito GC, Eason JM, Pervez A, Paulson WD: A randomized controlled trial of blood flow and stenosis surveillance of hemodialysis grafts. Kidney Int 64:272-280, 2003

17. Robbin ML, Oser RF, Lee JY, Heudebert GR, Mennemeyer ST, Allon M: Randomized comparison of ultrasound surveillance and clinical monitoring on arteriovenous graft outcomes. Kidney Int 69:730-735, 2006

18. Malik J, Slavikova M, Svobodova J, Tuka V: Regular ultrasound screening significantly prolongs patency of PTFE grafts. Kidney Int 67:1554-1558, 2005

19. Poole CV, Carlton D, Bimbo L, Allon M: Treatment of catheter-related bacteremia with an antibiotic lock protocol: Effect of bacterial pathogen. Nephrol Dial Transplant 19:1237-1244, 2004

20. Beathard GA: Catheter management protocol for catheter-related bacteremia prophylaxis. Semin Dial 16:403-405, 2003

21. Beathard GA, Welch BR, Maidment HJ: Mechanical thrombolysis for the treatment of thrombosed hemodialysis access grafts. Radiology 200:711-716, 1996

22. Oliver MJ, McCann RL, Indridason OS, Butterly DW, Schwab SJ: Comparison of transposed brachiobasilic fistulas to upper arm grafts and brachiocephalic fistulas. Kidney Int 60:1532-1539, 2001

23. Dixon BS: Why don't fistulas mature? Kidney Int 70:1413-1422, 2006

24. Roy-Chaudhury P, Sukhatme VP, Cheung AK: Hemodialysis vascular access dysfunction: A cellular and molecular viewpoint. J Am Soc Nephrol 17:1112-1127, 2006

25. Robbin ML, Chamberlain NE, Lockhart ME, et al: Hemodialysis arteriovenous fistula maturity: US evaluation. Radiology 225:59-64, 2002

26. Lok CE, Allon M, Moist LM, Oliver MJ, Shah H, Zimmerman D: REDUCE FTM I (Risk equation determining unsuccessful cannulation events and failure to maturation in arteriovenous fistulas). J Am Soc Nephrol 17:3204-3212, 2006

27. Allon M, Robbin ML: Increasing arteriovenous fistulas in hemodialysis patients: Problems and solutions. Kidney Int 62:1109-1124, 2002

28. Blagg CR: The early history of dialysis for chronic renal failure in the United States: A view from Seattle. Am J Kidney Disease 49:482-496, 2007