Central Venous Obstruction Management

Sanjoy Kundu, B.Sc., M.D., F.R.C.P.C., D.A.B.R., F.A.S.A., F.C.I.R.S.E., F.S.I.R.[1]

ABSTRACT

A major challenge in the management of hemodialysis patients is central venous stenosis and obstruction. Placement of central venous catheters has been shown to result in a high incidence of central venous stenosis or obstruction. There has been extensive literature on the treatment of this important and prevalent problem. Treatment options include percutaneous balloon angioplasty and bare metal stents. Unfortunately, all the available treatment options have variable rates of patency, requiring repeated intervention. More recently, covered stents have been mentioned in the literature for the treatment of central venous stenosis and obstruction. There is very little data to date, and further randomized controlled trials will be needed to compare the efficacy of percutaneous balloon angioplasty, bare metal stents, and covered stents. It appears prevention of this difficult problem is paramount, by limiting use of central venous catheters.

KEYWORDS: Central venous obstruction, hemodialysis, percutaneous balloon angioplasty, bare metal stents, covered stents

Objectives: Upon completion of this article, the reader should be able to identify the etiology and treatment options for central venous disease in hemodialysis patients, including percutaneous transluminal angioplasty, bare metal stents, and covered stents.
Accreditation: Tufts University School of Medicine (TUSM) is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.
Credit: TUSM designates this educational activity for a maximum of 1 *AMA PRA Category 1 Credit*™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Central venous stenosis and obstruction (CVD) is an important and prevalent problem in the management of hemodialysis (HD) patients. CVD compromises the integrity of the hemodialysis access circuit, by causing venous hypertension with/without debilitating symptoms. This can result in loss of the access site due to access dysfunction or ligation for symptom relief. The incidence of CVD has been reported in the range of 30% in the literature.[1]

ANATOMY

A thorough knowledge of the route of the central veins and their relationship to surrounding structures is critical to why CVD occurs in typical locations. The brachial and basilic veins join at the lower border of the teres major muscle to form the axillary vein, which passes anterior to the subscapularis muscle and posterior to the pectoralis minor muscle near its insertion at the coracoid process. The axillary vein continues to the lateral border of the first rib, where it becomes the subclavian vein, which enters the thoracic inlet posterior to the clavicle and anterior to the first rib and scalenus anticus muscle (costoclavicular space) and joins the internal jugular vein after several centimeters to become the brachiocephalic vein. The right and left brachiocephalic veins, join in the mediastinum to form the superior vena cava.[2]

[1]Department of Medical Imaging, Scarborough Hospital, Toronto, Ontario, Canada.

Address for correspondence and reprint requests: Sanjoy Kundu, B.Sc., M.D., 217 Davenport Road, Toronto, Ontario, Canada M5R 1J3 (e-mail: sanjoy_kundu40@hotmail.com).

Update on Dialysis Intervention; Guest Editors, Sidney Regalado, M.D., and Steven M. Zangan, M.D.

Semin Intervent Radiol 2009;26:115–121. Copyright © 2009 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662.
DOI 10.1055/s-0029-1222454. ISSN 0739-9529.

PATHOGENESIS

There has been a strong association of CVD, with previous placement of central venous catheters and pacemaker wires. In one study, 27% of patients with CVD had a history of previous central venous catheter placement.[3] Furthermore, there is a very high incidence of CVD in patients with a history of subclavian catheters of 42 to 50% compared with internal jugular vein catheters.[4–7] A suggested mechanism for the development of CVD includes central venous catheter-induced trauma to the venous endothelium and secondary inflammatory damage within the vessel wall at the time of insertion. Other proposed mechanisms include the presence of a foreign body in the vein, along with increased flow and turbulence from the creation of an AV access. Turbulent blood flow has been shown to incite an inflammatory response and stimulate intimal hyperplasia.[7–11]

RISK FACTORS FOR CVD

It is rare for CVD to occur in HD patients, without a history of previous central venous catheterization. Multiple central venous catheter placements, with longer catheter dwell times, have been associated with a greater risk of CVD.[3,7,12] The location of the central venous catheter is also an important causative factor for CVD. Central venous catheters placed by a subclavian access, have a particularly high risk, with a 42% incidence of CVD compared with a 10% rate with catheters placed via an internal jugular vein access.[4–7] There is also an increased predilection for CVD to occur with left-sided access for catheter placement. This may be related to the more tortuous course catheters have to traverse from a left-sided access.[6,13–15] Given the high incidence of CVD with hemodialysis catheters, the large caliber of these catheters may be a causative factor in CVD. There is no literature available to assess the impact of catheter caliber on CVD to date.

Peripherally inserted central catheters (PICC) and central venous port catheters are also becoming an increasingly important risk factor for CVD. Most patients with CVD secondary to peripherally inserted catheters and central venous port catheters, are usually asymptomatic, and present clinically after a hemodynamic challenge, such as placement of a ipsilateral AV access.[16,17] Pacemaker and defibrillator wires can also lead to CVD, with development of clinical symptoms after the placement of an AV access in the ipsilateral extremity.[18–21]

CLINICAL PRESENTATION

CVD can be asymptomatic, detected on a pre-access placement venogram or diagnostic fistulogram.[22,23] Most occult CVD become clinically apparent after development of a functioning AV access in the ipsilateral extremity. The symptoms of CVD depend on the site of stenosis or obstruction. Narrowing or occlusion of the subclavian vein most commonly presents with edema and/or venous hypertension of the corresponding extremity and breast. Innominate vein stenosis or occlusion affects blood flow from the same side of the face as well as the upper extremity and breast.

Approximately, only 50% of patients with significant CVD will develop ipsilateral upper extremity edema.[22] Edema is much more common once a functional ipsilateral upper extremity AV access is created.[24] Use of this access for HD, can lead to further exacerbation of the edema, with swelling, tenderness, pain and associated erythema, which can mimic cellulitis. Associated edema of the breast on the ipsilateral side along with pleural effusions may develop.[25,26]

CVD may lead to aneurysmal dilation and tortuosity of an arteriovenous (AV) access. Progression may be prevented with prompt treatment of the inciting central lesion. Marked aneurysmal dilation may have to be treated with surgical revision of the AV access. CVD leads to the development of collaterals, which divert blood centrally via enlarged collateral veins. The collateral veins are often evident on physical examination on the neck, chest, and ipsilateral extremity. Rarely, the collaterals can bypass sufficient blood flow centrally, leading to improvement or stabilization of the symptoms of CVD.

Superior vena cava syndrome is the most feared complication of superior vena cava stenosis or obstruction or bilateral innominate vein narrowing or occlusion.[27,28] This clinical syndrome is comprised of edema of both upper extremities, face and neck, along with multiple dilated collateral veins over the chest and neck. Acute emergent treatment of superior vena cava syndrome is required.

CVD may also decrease access blood flow, leading to access recirculation and inadequate dialysis. This may also present as elevated venous pressure during HD, and prolonged bleeding from needle sites after dialysis. If there is a significant decline in access blood flow, the AV access may become occluded secondary to thrombosis. Thrombolysis techniques will be ineffective, or lead to recurrent thrombosis, unless the CVD is also treated.

DIAGNOSIS

The diagnosis of CVD is made based on a constellation of clinical and imaging findings. Most patients will have a history of previous central venous catheter placement, and will present with ipsilateral arm, breast, face, or neck swelling. Many patients will have evidence of AV access dysfunction, with decreased access flows. On physical examination, there may be numerous dilated collaterals in the neck or chest and arm edema, on the side of the CVD. In the cases of bilateral innominate vein or

superior vena cava stenosis or occlusion, patients may present with superior vena cava syndrome. CVD can often be diagnosed by duplex ultrasound, with an absence of normal respiratory variation in the diameter of central veins and polyphasic atrial waves.[29] It is difficult to visualize the central veins with duplex ultrasound in patients with an elevated body mass index, or significant chest musculature.

Digital subtraction central venography is the gold standard for the diagnosis of CVD, and is more sensitive than duplex ultrasound.[1] All patients undergoing diagnostic fistulography for AV access dysfunction, should undergo complete access circuit venography, to rule out CVD. Magnetic resonance venography is an alternative to conventional venography, with no significant literature to date on the assessment of CVD. However, it should be noted that patients with decreased glomerular filtration rate (GFR), are at risk of developing nephrogenic systemic fibrosis.[30]

TREATMENT OPTIONS

Endovascular intervention is the mainstay of treatment in HD patients with CVD. The treatment options include percutaneous transluminal angioplasty (PTA), placement of bare metal stents (BMSs), and more recently placement of covered stents (CSs). The K/DOQI guidelines recommend PTA, with or without stent placement as the preferred treatment approach to CVD.[31]

PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY

Percutaneous transluminal angioplasty (PTA) for CVD was first reported by Glanz et al in 1984, with 100% technical success rate.[32] A subsequent study by Trerotola et al in 1986 demonstrated similar technical and clinical success rates.[33] PTA is the first-generation technology for the treatment of CVD. Unfortunately, at the time of the initial PTA studies, there were no clear defined reporting standards in place, leading to variable study methodology and endpoints. There are no large randomized control level one studies to assess PTA for CVD, making it difficult to draw conclusions on the outcomes of PTA, and make comparisons to alternative technologies.

PTA has demonstrated a variable technical success rate ranging from 70 to 90%.[2,18,34–38] A PTA study by Kovalik et al in 1994 made some interesting observations, including a technical failure rate of 7%, with greater than 50% improvement (nonelastic lesions) in 70% of patients with CVD, and less than 50% improvement (elastic lesions) in 23% of patients with CVD. The study concluded that there were two types of central venous lesion: nonelastic lesions that responded well to PTA, and elastic lesions that were unresponsive or poorly responsive to PTA. It was felt the histology of the two types of lesions was different based on observations on intravascular ultrasound.[34]

Examining the PTA patency results for CVD demonstrates a wide range of variability. There is a 6-month primary patency range of 23 to 63% and a cumulative patency range of 29 to 100%. There is a 12-month primary patency range of 12 to 50% and a cumulative patency range of 13 to 100%.[2,18,34–38] One of the largest studies to date on PTA for CVD by Bakken et al in 2007 comprised of 47 patients, demonstrated a technical success rate of 77%. There was a primary patency rate at 3 months of 58%, 6 months of 45%, and 12 months of 29%. There was a cumulative patency rate at 3 months of 76%, 6 months of 62%, and 12 months of 53%.[38] In summary, technical failures are to be expected when treating CVD with PTA in the range of 10 to 30%. There is clearly a subgroup of CVD patients with elastic lesions, unresponsive to PTA. It is also apparent repeated interventions are required with PTA for CVD, to maintain patency over the long term.

BARE METAL STENTS

Bare metal stents (BMSs) were first placed in the dialysis access circuit, for refractory stenoses by Gunther et al in 1989.[39] BMSs are the second-generation technology for the treatment of CVD. BMSs provide mechanical support to a site of stenosis that is resistant or unresponsive to PTA. BMSs are potentially useful in CVD in the setting of kinked stenoses, collapsing or elastic stenoses post PTA, sealing dissections or circumscribed perforations post PTA, establishing and maintaining patency of chronic central vein occlusions, and after PTA of highly resistant stenoses.

However, there are significant limitations to BMSs. Postdeployment, BMSs may migrate, shorten, or fracture on a subacute or delayed basis. BMS placement may preclude future endovascular procedures or surgical revision. It is also clearly evident that all BMSs incite intimal hyperplasia, leading to recurrent stenoses and multiple repeat interventions to maintain patency. The use of a BMS in HD access PTA interventions has significantly increased from 0% in 1991 to over 9% in 2001 according to the United States Renal Data System.[40] The exponential increase in BMS usage in HD access procedures has led to the development of guidelines for its applications. The Society of Interventional Radiology Quality Improvement Guidelines, recommend BMS be reserved for central vein lesions in which PTA has failed or that recur within 3 months after initially successful PTA; or rupture after PTA.[41] Similarly, the consensus guidelines of the National Kidney Foundation Dialysis Outcomes Quality Initiative recommend that the use of stents be reserved for surgically inaccessible stenoses in which PTA fails.[31,42,43]

The results for BMS demonstrate a wide range of variability. The vast majority of the literature demonstrates a very high technical success rate, in the range of 100%.

Stent structure and composition may be a factor in the initial technical success rate and long-term patency, although this has not been clearly demonstrated in the literature to date. As a general rule, self-expanding stents have been utilized for CVD. The first generation self-expanding stent is the Wallstent® (Boston Scientific, Natick, MA). The Wallstent® is constructed of 18 filaments of Elgiloy woven into a mesh. The advantages of this stent include low profile, flexibility, and radiopacity. The disadvantages of this stent include foreshortening at the time of placement: eccentric loading (stenosis) can lead to concentric narrowing and decreased radial strength as well as rare delayed shortening and migration.[44–48] The second-generation self-expanding stent are the nitinol stents. Nitinol is an alloy of nickel and titanium. It has a crystalline structure, which exists in two types of temperature-dependent forms. Nitinol undergoes a reversible shape transformation (martensitic transformation), which is preset by the ratio of nickel and titanium and high temperature heating. When nitinol transforms to its higher temperature crystalline form (28 to 33°C), it will expand to its preset size and become relatively more rigid. Nitinol, also has the characteristic of superelasticity, which will cause an applied external force to deform it, but attempt return to its original shape over time, or if the external force is removed.[47–50]

The results for BMS in the setting of CVD have been quite variable. There is a 3-month primary patency range of 63 to 100% and a cumulative patency range of 72 to 100%. There is a 6-month primary patency range of 42 to 89% and a cumulative patency range of 55 to 100%. There is a 12-month primary patency range of 14 to 73% and a cumulative patency range of 31 to 91%.[34–38,51–58] One of the largest retrospective studies to date on BMSs with Wallstent™ for CVD by Haage et al published in 1999 with 50 patients demonstrated a 3-month primary patency rate of 92% and a 6- and 12-month primary patency rate of 84% and 56%, respectively. There was a cumulative patency rate at 6 and 12 months of 97%.[55] Unfortunately, these results have not been replicated elsewhere in the literature. A more recent retrospective study on nitinol BMS for CVD by Vogel et al in 2004 with 16 patients demonstrated 3-, 6-, and 12-month primary patency rates of 81%, 74%, and 67%, respectively. Cumulative patencies were not reported in this study.[51] There are no randomized control trials to date comparing PTA and BMS in the setting of CVD. A recent retrospective study by Bakken et al published in 2007 comparing PTA and BMS for CVD demonstrated 3-, 6-, and 12-month primary patencies with PTA of 58%, 25%, and 29% in comparison with 3-, 6-, and 12-month primary patencies with BMS of 65%, 54%, and 45%. There were 3-, 6-, and 12-month cumulative patencies with PTA of 76%, 62%, and 53% in comparison with 3-, 6-, and 12-month cumulative patencies with BMS of 72%, 55%, and 46%. There was no significant difference in patency results between the PTA or BMS group.

In summary, it appears BMS for CVD demonstrate a high technical success rate. There is clearly a group of CVD patients, who are unresponsive to PTA and will require a BMS to achieve technical success. However, there is no literature to date demonstrating the superiority of BMSs over PTA in the setting of CVD. Future randomized control trials will be needed to determine the appropriate role of BMSs for CVD.

COVERED STENTS

Covered stents (CSs), also known as peripheral endografts, have been proposed as a treatment option for CVD. The potential advantages of a CS would include providing a relatively inert and stable intravascular matrix for endothelialization while providing the mechanical advantages of a BMS. This could potentially reduce the intimal hyperplastic response, causing restenosis post-PTA or BMS placement. CSs are available in balloon-expandable or self-expanding platforms. In practical terms, a self-expanding platform would be preferred, given the rigidity of the balloon-expandable platform. There is minimal literature on CS usage in the hemodialysis access circuit. Most of the literature to date has been on the treatment of graft or outflow vein aneurysms and refractory venous outflow stenoses.[59–62] CSs for CVD has only been mentioned in two publications to date. Sapoval et al in 1996 mentioned the use of a nitinol plus Dacron covered stent (Craig Endopro®, Mintec, LaCiotat, France) for an in-stent restenosis of a Wallstent®, with asymptomatic recurrent restenosis after 6 months.[63] A study by Quinn et al. in 2003 placed six covered stents for CVD, and eleven covered stents for venous outflow stenoses. There was a combined primary patency at 2, 6, and 12 months of: 40%, 32%, and 32%; and secondary patency at 2, 6, and 12 month of: 70%, 55% and 39%. They utilized a Palmaz® stent (P308, Johnson and Johnson, Warren, NJ) with an ePTFE graft material manually sewn on.[64] CSs provide an interesting treatment alternative for CVD. However, further randomized controlled trials, with long-term follow-up will be necessary.

SURGICAL OPTIONS FOR CVD

Percutaneous endovascular therapy continues to be the first-line treatment for CVD. However, in patients refractory to endovascular options, surgical possibilities must be considered. If there is a functioning HD access

in the ipsilateral extremity to the site of CVD, a simple reduction procedure may bring the volume down to something that can be accommodated by collateral circulation and continue to provide adequate flow for dialysis with resolution of symptoms. If not, then the CVD can be addressed by extra-anatomic bypass, including jugular vein turn down procedures, subclavian vein to external or internal jugular vein bypass, or axillary to femoral vein bypass.[65,66] Surgical options for CVD are associated with significant morbidity in patients with CVD and are a second line treatment alternative in patients refractory to percutaneous endovascular treatment options.

FUTURE DIRECTIONS

Future treatments may include drug eluting stents with rapamycin or paclitaxel, or coated stents to improve endothelial healing inside the stent. Other alternatives may include brachytherapy with β radiation, which has shown some benefit in coronary intervention.

Further characterization of hemodynamic, molecular, and pathologic mechanisms of CVD, and development of treatments and preventative strategies are critical to improve long-term patency of the HD access circuit.

SUMMARY

Central venous catheter placement is the most important risk factor for CVD. In patients at risk or with existing renal dysfunction, central venous catheter use should be avoided, particularly in the subclavian vein. The use of other peripheral lines should be minimized to preserve peripheral and central venous capital.

All the current treatment options for CVD are prone to recurrence requiring multiple repeat interventions to maintain patency. Further randomized control trials with long-term follow-up for the currently available treatment options are mandatory to develop appropriate treatment algorithms. Further advancements in treatment technique, technology, and the mechanisms of CVD will be required to continue to improve the outcomes for this difficult problem.

REFERENCES

1. Lumsden AB, MacDonald MJ, Isiklar H, et al. Central venous stenosis in the hemodialysis patient: incidence and efficacy of endovascular treatment. Cardiovasc Surg 1997; 5(5):504–509
2. Glanz S, Gordon DH, Lipkowitz GS, Butt KM, Hong J, Sclafani SJ. Axillary and subclavian vein stenosis: percutaneous angioplasty. Radiology 1988;168(2):371–373
3. Agarwal AK, Patel BM, Farhan NJ. Central venous stenosis in hemodialysis patients is a common complication of ipsilateral central vein catheterization. J Am Soc Nephrol 2004;15:368A–369A
4. Barrett N, Spencer S, McIvor J, Brown EA. Subclavian stenosis: a major complication of subclavian dialysis catheters. Nephrol Dial Transplant 1988;3(4):423–425
5. Cimochowski GE, Worley E, Rutherford WE, Sartain J, Blondin J, Harter H. Superiority of the internal jugular over the subclavian access for temporary dialysis. Nephron 1990; 54(2):154–161
6. Schillinger F, Schillinger D, Montagnac R, Milcent T. Post catheterisation vein stenosis in haemodialysis: comparative angiographic study of 50 subclavian and 50 internal jugular accesses. Nephrol Dial Transplant 1991; 6(10):722–724
7. Vanherweghem JL, Yassine T, Goldman M, et al. Subclavian vein thrombosis: a frequent complication of subclavian vein cannulation for hemodialysis. Clin Nephrol 1986;26(5):235–238
8. Palabrica T, Lobb R, Furie BC, et al. Leukocyte accumulation promoting fibrin deposition is mediated in vivo by P-selectin on adherent platelets. Nature 1992;359(6398):848–851
9. Weiss MF, Scivittaro V, Anderson JM. Oxidative stress and increased expression of growth factors in lesions of failed hemodialysis access. Am J Kidney Dis 2001;37(5): 970–980
10. Fillinger MF, Reinitz ER, Schwartz RA, et al. Graft geometry and venous intimal-medial hyperplasia in arteriovenous loop grafts. J Vasc Surg 1990;11(4):556–566
11. Middleton WD, Erickson S, Melson GL. Perivascular color artifact: pathologic significance and appearance on color Doppler US images. Radiology 1989;171(3):647–652
12. MacRae JM, Ahmed A, Johnson N, et al. Central vein stenosis: a common problem in patients on hemodialysis. ASAIO J 2005;51:77–81
13. Schon D, Whittman D. Managing the complications of long-term tunneled dialysis catheters. Semin Dial 2003;16(4):314–322
14. Moss AH, Vasilakis C, Holley JL, Foulks CJ, Pillai K, McDowell DE. Use of a silicone dual-lumen catheter with a Dacron cuff as a long-term vascular access for hemodialysis patients. Am J Kidney Dis 1990;16(3):211–215
15. Salgado OJ, Urdaneta B, Colmenares B, García R, Flores C. Right versus left internal jugular vein catheterization for hemodialysis: complications and impact on ipsilateral access creation. Artif Organs 2004;28(8):728–733
16. Gonsalves CF, Eschelman DJ, Sullivan KL, DuBois N, Bonn J. Incidence of central vein stenosis and occlusion following upper extremity PICC and port placement. Cardiovasc Intervent Radiol 2003;26(2):123–127
17. Trerotola SO, Kuhn-Fulton J, Johnson MS, Shah H, Ambrosius WT, Kneebone PH. Tunneled infusion catheters: increased incidence of symptomatic venous thrombosis after subclavian versus internal jugular venous access. Radiology 2000;217(1):89–93
18. Beathard GA. Percutaneous transvenous angioplasty in the treatment of vascular access stenosis. Kidney Int 1992;42(6): 1390–1397
19. Korzets A, Chagnac A, Ori Y, Katz M, Zevin D. Subclavian vein stenosis, permanent cardiac pacemakers and the haemodialysed patient. Nephron 1991;58(1):103–105
20. Chuang CL, Tarng DC, Yang WC, Huang TP. An occult cause of arteriovenous access failure: central vein stenosis

from permanent pacemaker wire. Report of three cases and review of the literature. Am J Nephrol 2001;21(5):406–409
21. Sticherling C, Chough SP, Baker RL, et al. Prevalence of central venous occlusion in patients with chronic defibrillator leads. Am Heart J 2001;141(5):813–816
22. Schwab SJ, Quarles LD, Middleton JP, Cohan RH, Saeed M, Dennis VW. Hemodialysis-associated subclavian vein stenosis. Kidney Int 1988;33(6):1156–1159
23. Clark DD, Albina JE, Chazan JA. Subclavian vein stenosis and thrombosis: a potential serious complication in chronic hemodialysis patients. Am J Kidney Dis 1990;15(3):265–268
24. Nakhoul F, Hashmonai M, Angel A, Bahous H, Green J. Extreme swelling of a limb with A-V shunt for hemodialysis resulting from subclavian vein thrombosis due to previous catheterization. Clin Nephrol 1998;49(2):134–136
25. Gadallah MF, El-Shahawy MA, Campese VM. Unilateral breast enlargement secondary to hemodilaysis arterivenous fistula and subclavian vein occlusion. Nephron 1993;63:351–353
26. Wright RS, Quinones-Baldrich WJ, Anders AJ, Danovitch GM. Pleural effusion associated with ipsilateral breast and arm edema as a complication of subclavian vein catheterization and arteriovenous fistula formation for hemodialysis. Chest 1994;106(3):950–952
27. Baker GL, Barnes HJ. Superior vena cava syndrome: etiology, diagnosis, and treatment. Am J Crit Care 1992;1(1):54–64
28. Khanna S, Sniderman K, Simons M, et al. Superior vena cava stenosis associated with hemodialysis catheters. Am J Kidney Dis 1993;21:278–281
29. Rose SC, Kinney TB, Bundens WP, Valji K, Roberts AC. Importance of Doppler analysis of transmitted atrial waveforms prior to placement of central venous access catheters. J Vasc Interv Radiol 1998;9(6):927–934
30. Marckmann P, Skov L, Rossen K, et al. Nephrogenic systemic fibrosis: suspected causative role of gadodiamide used for contrast-enhanced magnetic resonance imaging. J Am Soc Nephrol 2006;17(9):2359–2362
31. NKF-K/DOQUI clinical practice guidelines for vascular access: Update 2000. Am J Kidney Dis 2001;37:S137–S181
32. Glanz S, Gordon D, Butt KMH, Hong J, Adamson R, Sclafani SJ. Dialysis access fistulas: treatment of stenoses by transluminal angioplasty. Radiology 1984;152(3):637–642
33. Trerotola SO, McLean GK, Burke DR, et al. Treatment of subclavian venous stenoses by percutaneous transluminal angioplasty. J Vasc Interv Radiol 1986;1:15–18
34. Kovalik EC, Newman GE, Suhocki P, Knelson M, Schwab SJ. Correction of central venous stenoses: use of angioplasty and vascular Wallstents. Kidney Int 1994;45(4):1177–1181
35. Quinn SF, Schuman ES, Demlow TA, et al. Percutaneous transluminal angioplasty versus endovascular stent placement in the treatment of venous stenoses in patients undergoing hemodialysis: intermediate results. J Vasc Interv Radiol 1995; 5:851–855
36. Dammers R, de Haan MW, Planken NR, van der Sande FM, Tordoir JH. Central vein obstruction in hemodialysis patients: results of radiological and surgical intervention. Eur J Vasc Endovasc Surg 2003;26(3):317–321
37. Surowiec SM, Fegley AJ, Tanski WJ, et al. Endovascular management of central venous stenoses in the hemodialysis patient: results of percutaneous therapy. Vasc Endovascular Surg 2004;38(4):349–354
38. Bakken AM, Protack CD, Saad WE, Lee DE, Waldman DL, Davies MG. Long-term outcomes of primary angioplasty and primary stenting of central venous stenosis in hemodialysis patients. J Vasc Surg 2007;45(4):776–783
39. Günther RW, Vorwerk D, Bohndorf K, et al. Venous stenoses in dialysis shunts: treatment with self-expanding metallic stents. Radiology 1989;170(2):401–405
40. United States Renal Data System. USRD 2003 Annual Data Report: Atlas of End-Stage Renal Disease in the United States. Bethesda, MD: National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases; 2003
41. Aruny JE, Lewis CA, Cardella JF, et al. Quality improvement guidelines for percutaneous management of the thrombosed or dysfunctional dialysis access. Standards of Practise Committee of the Society of Cardiovascular & Interventional Radiology. J Vasc Interv Radiol 2003;14: S247–S253
42. NKF-DOQI clinical practice guidelines for vascular access. National Kidney Foundation-Dialysis Outcomes Quality Initiative. Am J Kidney Dis 1997;30(Suppl 3):S150–S191
43. Clark TWI. Nitinol stents in hemodialysis access. J Vasc Interv Radiol 2004;15(10):1037–1040
44. Flueckiger F, Sternthal H, Klein GE, Aschauer M, Szolar D, Kleinhappl G. Strength, elasticity, and plasticity of expandable metal stents: in vitro studies with three types of stress. J Vasc Interv Radiol 1994;5(5):745–750
45. Rogers C, Edelman ER. Endovascular stent design dictates experimental restenosis and thrombosis. Circulation 1995; 91(12):2995–3001
46. Trerotola SO, Fair JH, Davidson D, Samphilipo MA Jr, Magee CA. Comparison of Gianturco Z stents and Wallstents in a hemodialysis access graft animal model. J Vasc Interv Radiol 1995;6(3):387–396
47. Dyet JF, Watts WG, Ettles DF, Nicholson AA. Mechanical properties of metallic stents: how do these properties influence the choice of stent for specific lesions? Cardiovasc Intervent Radiol 2000;23(1):47–54
48. Verstandig AG, Bloom AI, Sasson T, Haviv YS, Rubinger D. Shortening and migration of Wallstents after stenting of central venous stenoses in hemodialysis patients. Cardiovasc Intervent Radiol 2003;26(1):58–64
49. Shabalovskaya SA. On the nature of the biocompatibility and on medical applications of NiTi shape memory and superelastic alloys. Biomed Mater Eng 1996;6(4):267–289
50. Duda SH, Wiskirchen J, Tepe G, et al. Physical properties of endovascular stents: an experimental comparison. J Vasc Interv Radiol 2000;11(5):645–654
51. Vogel PM, Parise CP. SMART stent for salvage of hemodialysis access grafts. J Vasc Interv Radiol 2004;15(10): 1051–1060
52. Aytekin C, Boyvat F, Yağmurdur MC, Moray G, Haberal M. Endovascular stent placement in the treatment of upper extremity central venous obstruction in hemodialysis patients. Eur J Radiol 2004;49(1):81–85
53. Chen CY, Liang HL, Pan HB, et al. Metallic stenting for treatment of central venous obstruction in hemodialysis patients. J Chin Med Assoc 2003;66(3):166–172
54. Oderich GS, Treiman GS, Schneider P, Bhirangi K. Stent placement for treatment of central and peripheral venous obstruction: a long-term multi-institutional experience. J Vasc Surg 2000;32(4):760–769

55. Haage P, Vorwerk D, Piroth W, Schuermann K, Guenther RW. Treatment of hemodialysis-related central venous stenosis or occlusion: results of primary Wallstent placement and follow-up in 50 patients. Radiology 1999;212(1):175–180
56. Vesely TM, Hovsepian DM, Pilgram TK, Coyne DW, Shenoy S. Upper extremity central venous obstruction in hemodialysis patients: treatment with Wallstents. Radiology 1997;204(2):343–348
57. 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(2): 479–484
58. 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;195:140–146
59. Vesely TM. Use of stent grafts to repair hemodialysis graft-related pseudoaneurysms. J Vasc Interv Radiol 2005;16(10): 1301–1307
60. Naoum JJ, Irwin C, Hunter GC. The use of covered nitinol stents to salvage dialysis grafts after multiple failures. Vasc Endovascular Surg 2006;40(4):275–279
61. Hausegger KA, Tiessenhausen K, Klimpfinger M, Raith J, Hauser H, Tauss J. Aneurysms of hemodialysis access grafts: treatment with covered stents: a report of three cases. Cardiovasc Intervent Radiol 1998;21(4):334–337
62. Silas AM, Bettmann MA. Utility of covered stents for revision of aging failing synthetic hemodialysis grafts: a report of three cases. Cardiovasc Intervent Radiol 2003;26(6): 550–553
63. Sapoval MR, Turmel-Rodrigues LA, Raynaud AC, Bourquelot P, Rodrigue H, Gaux JC. Cragg covered stents in hemodialysis access: initial and midterm results. J Vasc Interv Radiol 1996;7(3):335–342
64. Quinn SF, Kim J, Sheley RC. Transluminally placed endovascular grafts for venous lesions in patients on hemodialysis. Cardiovasc Intervent Radiol 2003;26(4):365–369
65. Gradman WS, Bressman P, Sernaque JD. Subclavian vein repair in patients with an ipsilateral arteriovenous fistula. Ann Vasc Surg 1994;8:549–556
66. Chandler NM, Mistry BM, Garvin PJ. Surgical bypass for subclavian vein occlusion in hemodialysis patients. J Am Coll Surg 2002;194(4):416–421