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CENTRAL VENOUS STENOSIS – ENDOVASCULAR AND SURGICAL TREATMENTS

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Introduction

The global incidence of subclavian, jugular, and femoral vein stenoses is 15.6%, 2.7% and 0-3.8%, respectively. Asymptomatic subclavian vein stenoses, detected by venograms, represent only 23-33% of all subclavian vein stenoses. Most reports show a higher incidence of asymptomatic versus symptomatic lesions: the rates of occurrence for subclavian vein stenoses are 41% versus 3.3%; the rates for nonsymptomatic jugular vein stenoses are 9% versus 1.6%.[1] Superior vena cava (SVC) syndromes are uncommon and usually caused by malignant diseases. In about 20% of the cases, however, the cause is benign. In addition to chronic mediastinitis, there are a growing number of reported cases of thrombosis resulting from endovenous devices (central catheters, pacemaker leads, etc.). Onset is often slow and insidious, with good tolerance in the early stages explained by the development of an effective collateral circulation.

While bilateral upper extremity venography is still the gold standard, currently computerized tomography and magnetic resonance imaging are more frequently used to make the diagnosis. Symptoms usually regress after medical treatment, sometimes requiring thrombolysis; however, in 10% of the patients, major functional impairment may require bypass surgery or transluminal angioplasty.[2] The surgical treatment of central venous stenosis has been reserved for either significantly symptomatic patients who have an obstruction or stenosis that is not amenable to an endovascular approach or for selected patients who have venous resection as part of a planned en block tumor resection.[3] The etiology of central venous obstruction is usually extension of bronchopulmonary neoplasm or mediastinal disease. Stenosis or obstruction from venous thrombosis or fibrosis related to indwelling dialysis catheters, pace makers, defibrillators, or central venous lines is increasingly common.[4]

Management of Superior Vena Cava Occlusion

In the presence of known malignant disease of the thorax, percutaneous management of complete SVC occlusion with thrombolysis and/or clot aspiration followed by stent insertion is safe and effective, giving sustained symptomatic relief.[5] Primary patency in malignant and benign cases at one year is 64% and 76%, respectively. Overall symptom-free survival ranges from one to 34 months.[6] Resection for malignant tumor involvement was for a long period considered an absolute contraindication to resection. Increasingly, resection and graft replacement have been used in selected cases where en bloc resection could be achieved.[7,8]

Infiltration of the superior vena cava due to advanced non-small cell lung cancer (NSCLC) or thymoma can be treated by prosthetic replacement or tangential resection. It should not be considered as palliative treatment because of the perioperative risks. SVC tangential resection involves fewer surgical problems. However, since this procedure is used mostly for N2 NSCLC subjects, patients have a low mean survival in spite of adjuvant therapy.[9] Surgical reconstruction of the superior vena cava with an expanded polytetrafluoroethylene (ePTFE) prosthesis provides immediate and long-term relief of symptoms of SVC obstruction with a low surgical morbidity, even in patients with unresectable malignancy.[10] When replacing the superior vena cava combined with

resection of mediastinal malignancies, reconstruction of a left brachiocephalic vein alone results in a significant rate of occlusion and development of superior vena cava syndrome. Single right brachiocephalic vein reconstruction or bilateral brachiocephalic vein reconstruction in this setting, and separate reconstruction of the veins, is preferable to the use of a Y graft.[11]

Highly symptomatic patients will benefit from central venous resection and reconstruction. Replacement grafts can include PTFE,[1] spiral vein graft,[12] or pericardium, although no data exists to support the superiority of one over the other. Our preference for superior vena cava reconstruction has been a self-constructed pericardial tube graft (Figures 1-3). Vessel involvement by soft tissue sarcoma can be classified as type I, artery and vein; type II, only artery; type III, only vein; and type IV, neither artery nor vein.

A study by Schwarzbach et al. found that in patients with retroperitoneal soft tissue sarcoma (STS), the most common vascular involvement pattern was vein only (64%). The inferior vena cava, iliac vein, and superior mesenteric vein were preserved or replaced in 80% of these patients. Morbidity was 36% (hemorrhage, others), and mortality was 4%. At a median follow-up of 19.3 months, venous patency rate was 93.8% (primary and secondary).[13] Venous reconstructions for iliofemoral or IVC obstruction offer 3-year patency rates of 62%. The Palma procedure with autologous saphenous vein

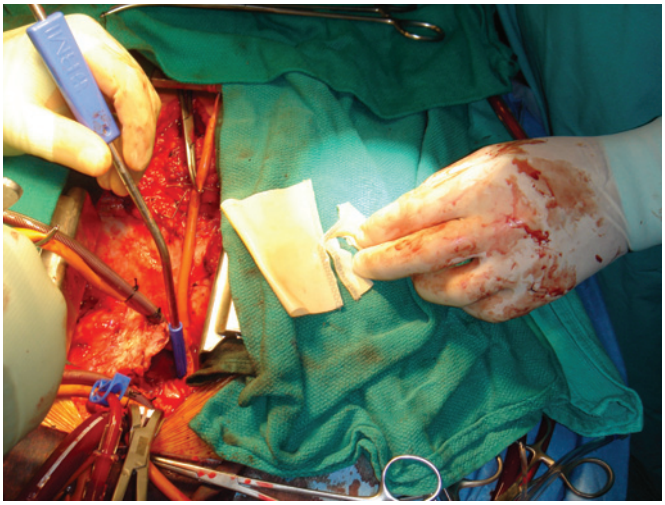


Figure 1. Construction of pericardial graft to replace the SVC and innominate vein.

Figure 2. Operative picture of replaced SVC and innominate vein with pericardial graft.

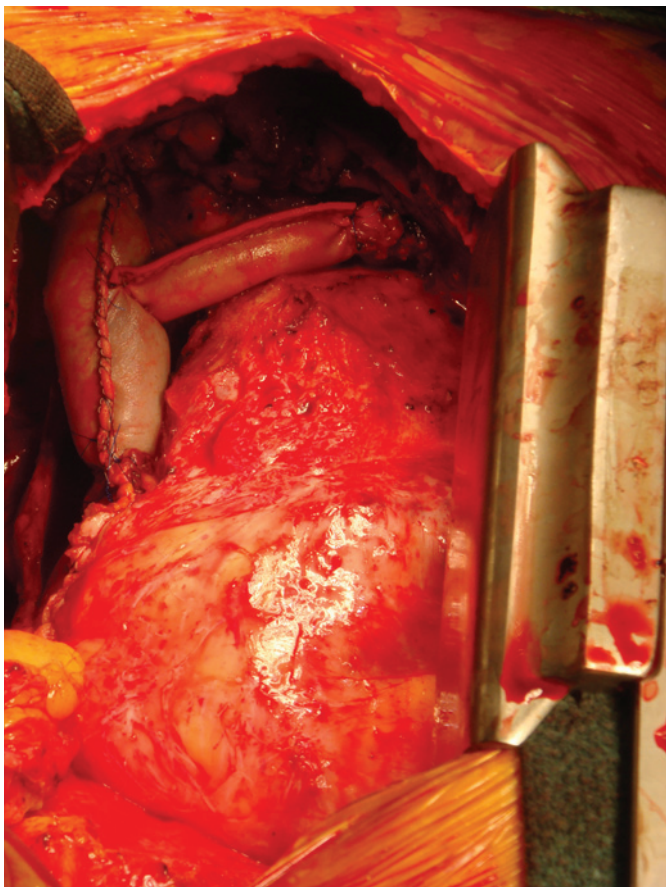
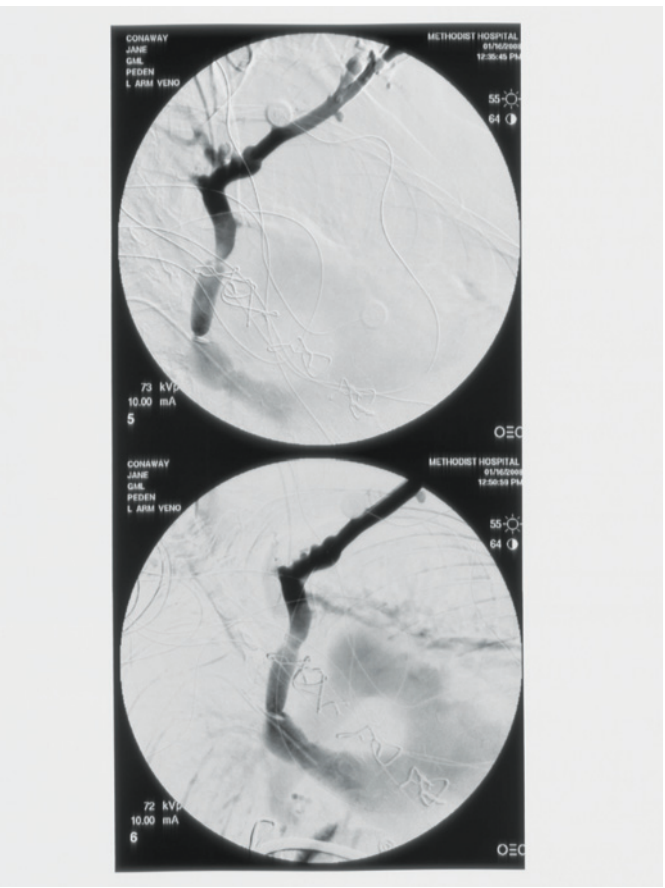


Figure 3. Angiogram of endovascular dilatation of stenosis at graft anastomosis.



showed the best long-term patency, whereas long-term success with ePTFE was moderate.[14] Tumor lesions of the inferior vena cava can originate from the vein or can develop by malignant tumor infiltration from the surrounding tissue. The resection rate was 83%, with surgical reconstruction of IVC achieved in each case (100%). The perioperative morbidity was 33%, whereas the hospital mortality was 8.3%. There was a mean post-operative observation period of 20 months (range, one to 58 months). Complete follow-up documentation was obtained for all of the patients (100%). While the overall mortality through the follow-up observation period was 27.5%, the tumor-specific mortality was 16%. The variable prognosis of the various tumor lesions depends on tumor entity, stage, resection status and individual risk factors.[15]

Treatment Strategies for Benign Etiologies

The need for intervention for benign etiologies is increasing as the use of indwelling catheters for dialysis and cardiac therapy such as pacemakers and implantable defibrillators expands. Endovascular treatment of benign iliocaval occlusive disease is a safe and efficient minimally invasive technique with good mid-term patency rates. Moreover, it improves cases with obstruction only and cases with associated reflux and obstruction. Primary stenting should always be performed by using self-expanding stents deployed under general anesthesia to avoid lumbar pain. In case of failure, the endovascular procedure does not preclude further surgical reconstruction.[16]

Endovascular repair is emerging as a first-line treatment for patients with SVC syndrome of benign etiology. Open surgical repair of benign SVC syndrome is effective, with durable long-term relief from symptoms. Endovascular repair is less invasive but equally effective in the mid-term, albeit at the cost of multiple secondary interventions, and is an appropriate primary treatment for benign SVC syndrome. Open surgical repair remains an excellent choice for patients who are not suitable for endovascular repair or in whom the endovascular repair fails.[17] Pacemaker wires can result in stenosis of the SVC and other central veins. Superior vena cava stenting is safe and effective in patients who develop the SVC obstruction after cardiac pacemaker insertion. No pacemaker function dysfunction was encountered in several case series.[18]

Reconstruction is usually limited to patients who are significantly symptomatic and have failed endovascular attempts at correction. Reconstruction can be done via mediasternotomy or upper hemisternotomy. Proximal access is generally not difficult, and we have replaced the SVC and used the right atrium as the outflow site with both working well. For isolated brachiocephalic obstruction, reconstruction has generally been done with ringed PTFE. The major technical issue is distal vein exposure and control.

Central venous obstruction is a common problem in patients with chronic renal failure who undergo maintenance hemodialysis. Significant stenosis or occlusion of the subclavian vein is known to occur in 20% to 50% of patients who have had central venous catheters inserted into the subclavian vein or the internal jugular vein.[19] Stents provide a temporary benefit in most patients with central or peripheral upper-extremity symptomatic venous obstruction. Incidences of central vein stenosis reported in hemodialysis patients have ranged from 11% to 40%.[20-21] Regular follow-up and reinterventions are required to maintain patency and achieve long-term clinical success. Stents used for central venous lesions have higher clinical success rates than stents used for peripheral venous lesions. However, endovascular therapy for central venous stenosis, whether by angioplasty or stenting, is safe in the dialysis access patient, with low rates of technical failure. Multiple additional interventions are the rule with both treatment modalities. Although neither offers truly durable outcomes, stent placement does not improve the patency rates associated with balloon angioplasty and does not add to the longevity of ipsilateral hemodialysis access sites.[22] Thus, patients with a reasonable life expectancy or who are unable to return for subsequent procedures should be considered for alternative therapy.[23] Right atrial bypass grafting has been used to restore central venous patency in the carefully selected patient in whom all other access sites are exhausted and in whom percutaneous dilation and/or stenting has failed.[24]

Summary

Central venous stenosis remains a difficult disease to diagnose and treat. The etiology of the lesion and the condition of the patient dictates the treatment strategy. Both endovascular and surgical approaches are successful and are best accomplished by the endovascular cardiovascular surgeon.

References

1. Huu TC. Central venous stenosis: review of the literature from 1980 to 2000. Nephrologie. 2001;22(8):479-85.
2. Marlier S, Bonal J, Cellarier G, Bouchiat C, Talard P, Dussarat GV. Superior vena cava syndromes of benign etiology. Presse Med. 1996 Sep 14;25(26):1203-07.
3. Picquet J, Blin V, Dussaussoy C, Jousset Y, Papon X, Enon B. Surgical reconstruction of the superior vena cava system: Indications and results. Surgery. 2009 Jan;145(1): 93-99.
4. Schifferdecker B, Shaw JA, Piemonte TC, Eisenhauer AC. Nonmalignant superior vena cava syndrome: pathophysiology and management. Catheter Cardiovasc Interv. 2005;65:416-23.
5. Crowe MT, Davies CH, Gaines PA. Percutaneous management of superior vena cava occlusions. Cardiovasc Intervent Radiol. 1995 Nov-Dec;18(6):367-72.
6. Barshes NR, Annambhotla S, El Sayed HF, Huynh TT, Kougias P, Dardik A, Lin PH. Percutaneous stenting of superior vena cava syndrome: treatment outcome in patients with benign and malignant etiology. Vascular. 2007 Sep-Oct;15(5):314-21.
7. Suzuki K, Asamura H, Watanbe S, Tsuchiya R. Combined resection of superior vena cava for lung carcinoma: prognostic significance of patterns of superior vena cava invasion. Ann Thoracic Surg 2004;78:1184-89.
8. Thomas P, Magnan PE, Moulin G, Giudicelli R, Fuentes P. Extended operation for the lung cancer invading the superior vena cava. Eur J. Cardiothorac Surg 1994; 8:177-82.
9. Politi L, Crisci C, Montinaro F, Andreani M, Podzemny V, Borzellino G. Prosthetic replacement and tangential resection of the superior vena cava in chest tumors. J Cardiovasc Surg (Torino). 2007 Jun;48(3):363-68.
10. Magnan PE, Thomas P, Giudicelli R, Fuentes P, Branchereau A. Surgical reconstruction of the superior vena cava. Cardiovasc Surg. 1994 Oct;2(5):598-604.
11. Shintani Y, Ohta M, Minami M, Shiono H, Hirabayashi H, Inoue M, Matsumiya G, Matsuda H. Long-term graft patency after replacement of the brachiocephalic veins combined with resection of mediastinal tumors. J Thorac Cardiovasc Surg. 2005 Apr;129(4):809-12.
12. Erbella J, Hess PJ, Huber TS. Superior vena cava bypass with superficial femoral vein for benign superior vena cava syndrome. Ann Vasc Surg 2006;20:834-38.
13. Schwarzbach MH, Hormann Y, Hinz U, Leowardi C, Böckler D, Mechtersheimer G, Friess H, Büchler MW, Allenberg JR. Clinical results of surgery for retroperitoneal sarcoma with major blood vessel involvement. J Vasc Surg. 2006 Jul;44(1):46-55.
14. Jost CJ, Gloviczki P, Cherry KJ Jr, McKusick MA, Harmsen WS, Jenkins GD, Bower TC. Surgical reconstruction of iliofemoral veins and the inferior vena cava for nonmalignant occlusive disease. J Vasc Surg. 2001 Feb;33(2):320-7; discussion 327-28.
15. Eder F, Halloul Z, Meyer F, Huth C, Lippert H. Surgery of inferior vena cava associated malignant tumor lesions. Vasa. 2008 Feb;37(1):68-80.
16. Hartung O, Otero A, Boufi M, Decaridi G, Barthelemy P, Juhan C, Alimi YS. Mid-term results of endovascular treatment for symptomatic chronic nonmalignant iliocaval venous occlusive disease. J Vasc Surg. 2005 Dec;42(6):1138-44; discussion 1144.
17. Rizvi AZ, Kalra M, Bjarnason H, Bower TC, Schleck C, Gloviczki P. Benign superior vena cava syndrome: stenting is now the first line of treatment. J Vasc Surg. 2008 Feb;47(2):372-80.
18. Teo N, Sabharwal T, Rowland E, Curry P, Adam A. Treatment of superior vena cava obstruction secondary to pacemaker wires with balloon venoplasty and insertion of metallic stents. Eur Heart J. 2002 Sep;23(18):1465-70.
19. Chandler NM, Mistry BM, Garvin PJ. Surgical bypass for subclavian vein occlusion in hemodialysis patients. J Am Coll Surg. 2002 Apr;194(4):416-21.
20. Schumacher KA, Wallner B, Weidenmaier W, Friedrich JM. Venous occlusions distant to the shunt as malfunction factors during hemodialysis. Rofo 1989;150:198-201.
21. Jendrisak M. The importance of preoperative evaluation of the subclavian vein in dialysis access planning. AJR Am J Roentgenol 1991;156:623-25.
22. Bakken AM, Protack CD, Saad WEA, Lee D, Waldman DL, Davies MG. Longterm outcomes of primary angioplasty versus primary stenting in the management of central venous stenoses in the hemodialysis patient. J Vasc Surg. 2007; 45:776-83.
23. 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 Oct;32(4):760-69.
24. l-Sabrout RA, Duncan JM. Right atrial bypass grafting for central venous obstruction associated with dialysis access: another treatment option. J Vasc Surg. 1999 Mar;29(3): 472-78.