Central Venous Stenosis: What Should a Nephrologist Know?

Central venous stenosis (CVS) is known to occur following the insertion of central lines inserted through neck vessels in ICUs, peripherally inserted central catheters, chemotherapy catheters, pacemaker wires (30%–60% cases), and hemodialysis catheters, both noncuffed and cuffed catheters. CVS is often asymptomatic in nondialysis patients, but can result in edema of ipsilateral extremity and breast when challenged by increased flow from an arteriovenous fistula (AVF) or AV graft. When brachiocephalic vein is involved, it can cause hemifacial swelling(published as a case report in this issue of journal, titled "innominate vein stenosis in association with ipsilateral hyperdynamic brachio-basilic fistula causing ipsilateral limb and hemifacial swelling).[1] A similar case has been reported earlier, titled "Brachiocephalic Vein Stenosis in Association with Ipsilateral Hyperdynamic Brachio-Basilic Fistula Causing Ipsilateral Facial Swelling and Contralateral Papillodema and Visual Loss-A Case Report.[2] Furthermore, the presence of CVS prevents the creation of new vascular access and results in increased morbidity and mortality, related to inadequate dialysis. Although this problem is widely prevalent among long-term dialysis patients in whom stiff noncuffed catheters are kept for a longer time, there is a lack of awareness among treating personnel. This commentary is being written to spread the awareness about the problem and how to tackle it.

How it Occurs?

The high venous pressure and blood flow due to the fistula may overwhelm the collateral venous and lymphatic drainage, resulting in the development of dilated and tortuous collateral veins over the ipsilateral upper arm, neck, and upper chest.[3] In severe cases, venous hypertension may eventually lead to disabling arm edema with pain and discomfort[4] [Figure 1]. Endothelial injury with subsequent changes in the vessel wall results in the development of microthrombi, smooth muscle proliferation, and CVS.

Closure of vascular access results in relief from symptoms and signs of CVS, but many a times, it is not possible to close the functioning vascular access. Subclavian catheters and catheters placed in the left-sided neck veins are more prone to CVS. Longer duration of stiff noncuffed catheter is more responsible to cause CVS. Hence, the KDOQI guidelines do not recommend using noncuffed catheters for more than 7 days.[5] Although CVS is primarily related to the placement of an ipsilateral central venous catheter, it can also occur without a previous history of catheter placement in up to 40% of the patients.[6,7] Most of the catheter-related cases occur following the insertion of stiff noncuffed catheters, but the author has experienced brachiocephalic vein occlusion due to soft-cuffed tunneled catheters also [Figures 2-4].

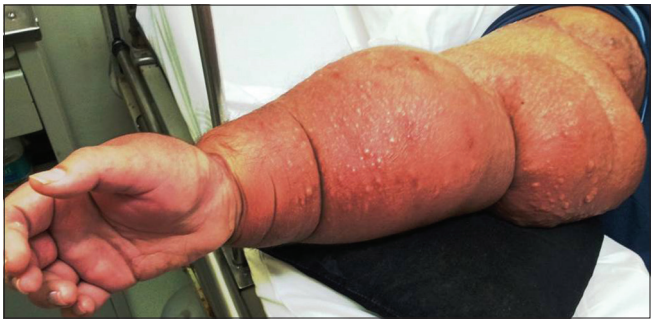

Figure 1: Hand edema due to central venous stenosis

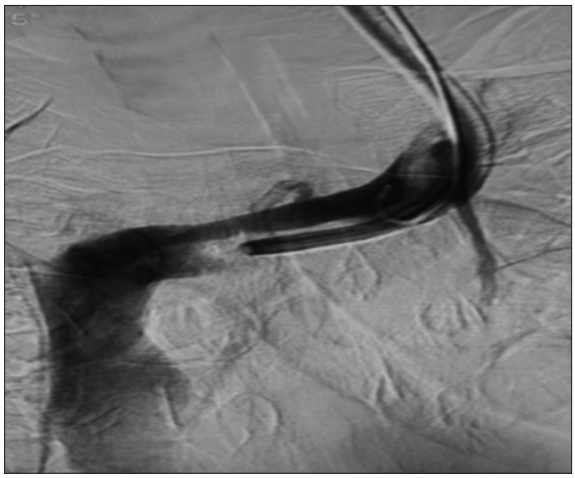

Figure 2: Venous dissection of the left brachiocephalic vein due to short length left internal jugular vein tunneled cuffed catheter

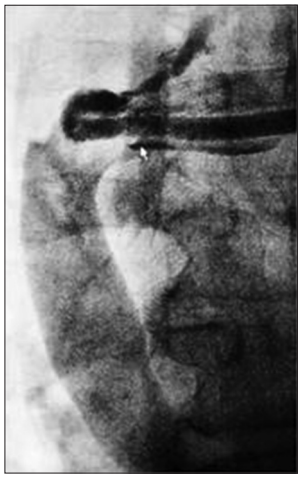

Figure 3: False passage in the left brachiocephalic vein due to tunneled cuffed catheter

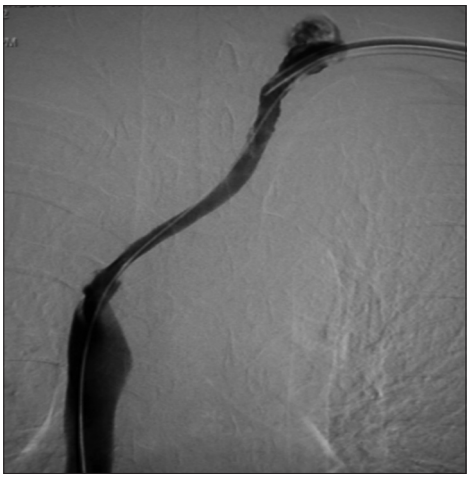

Figure 4: Left brachiocephalic vein stenosis due to previous tunneled cuffed catheter

Anatomy

A thorough knowledge of the route of the central veins and their relationship to the surrounding structures is critical to why CVS 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 anteriorly to the subscapularis muscle and posteriorly 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 posteriorly to the clavicle and anteriorly 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.[8]

Diagnosis

The diagnosis of CVS is made from 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, increased venous pressures during dialysis, and a history of excessive bleeding from the puncture site after removal of needle. On physical examination, there may be numerous dilated collaterals in the neck or chest and arm edema, or dilated tortuous draining veins of fistula [Figure 5], on the side of the CVS. In the cases of bilateral innominate vein or superior vena cava stenosis or occlusion, patients may present with superior vena cava syndrome. CVS can often be diagnosed by duplex ultrasound, with an absence of normal respiratory variation in the diameter of central veins and polyphasic atrial waves. It is difficult to visualize the central veins with duplex ultrasound in obese and muscular patients.

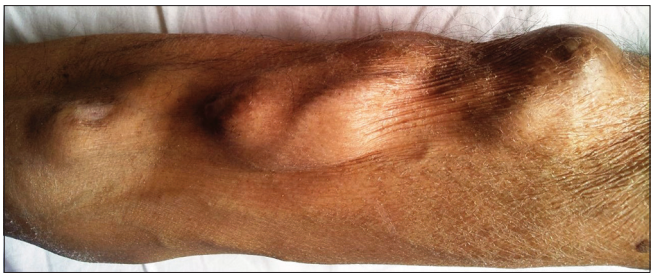

Figure 5: Dilated tortuous veins of arteriovenous fistula due to central venous stenosis

Digital subtraction central venography is the gold standard for the diagnosis of CVS, and it is more sensitive than duplex ultrasound. All patients undergoing diagnostic fistulography for AV access dysfunction should undergo complete access circuit venography to rule out CVS. Magnetic resonance venography is an alternative to conventional venography.[9]

Treatment

Once diagnosed, CVS should be tackled with percutaneous transluminal balloon angioplasty alone or with stent (bare metal nitinol or stent graft). The author has an experience of 30 cases of CVS, 25 of which required venoplasty and 5 required additional bare metal stenting (one required in-stent stenting due to in-stent thrombosis and another due to stent fracture) (unpublished). Hemodialysis vascular accesses with high-flow volumes predispose to a high rate of recurrence after interventional treatment. Recent studies have shown that vascular access flow reduction by banding of the access inflow reduced the rate of re-stenosis and was able to resolve symptoms associated with noncorrectable central venous lesions in patients who had previously undergone angioplasty and stent placement.[10]

In resistant cases with severe symptoms, occlusion of the functioning vascular access will usually provide relief of symptoms. Surgical options (venous bypass with a graft) should be pursued in only refractory cases due to the invasiveness of the intervention.

Clinical pearls

1. Before AV fistula creation, patient's history in terms of central venous catheterization or pacemaker implantation must be carefully reviewed
2. In the case of a previous dialysis catheter or pacemaker implantation, careful examination of the central venous system is mandatory, aimed at ruling out CVS if AVF is to be created on the same arm side
3. Not to keep stiff noncuffed hemodialysis catheters for longer than recommended duration
4. Avoid AVF creation on the same side of neck catheter insertion. Furthermore, avoid central lines or pacemaker implantation on the same side of fistula arm

5. It is important to inform pacemaker implanters of the risk of CVS in CKD patients in order for them to take into consideration the potential need for future AVF when choosing the site of implantation of a pacemaker.

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References

1. Narendra JB, Sreenivas J, Karthikeyan VS, Nagaraja NH. Innominate vein stenosis in association with ipsilateral hyperdynamic brachiobasilic fistula causing ipsilateral limb and hemifacial swelling. Indian J Nephrol 2017;27:452-5.
2. Eames P, Senthil L, Thomas A, Riley P, Burdon MA. Brachiocephalic vein stenosis in association with ipsilateral hyperdynamic brachio-basilic fistula causing ipsilateral facial swelling and contralateral papillodema and visual loss. A case report. Interv Neuroradiol 2010;16:322-5.
3. Davis D, Petersen J, Feldman R, Cho C, Stevick CA. Subclavian venous stenosis. A complication of subclavian dialysis. JAMA 1984;252:3404-6.
4. Tatapudi VS, Spinowitz N, Goldfarb DS. Symptomatic central venous stenosis in a hemodialysis patient leading to loss of arteriovenous access: A case report and literature review. Nephron Extra 2014;4:50-4.
5. Vascular Access Work Group. Clinical practice guidelines for vascular access. Am J Kidney Dis 2006;48 Suppl 1:S176-247.
6. Oguzkurt L, Tercan F, Yildirim S, Torun D. Central venous stenosis in haemodialysis patients without a previous history of catheter placement. Eur J Radiol 2005;55:237-42.
7. Taal MW, Chesterton LJ, McIntyre CW. Venography at insertion of tunnelled internal jugular vein dialysis catheters reveals significant occult stenosis. Nephrol Dial Transplant 2004;19:1542-5.
8. Glanz S, Gordon DH, Lipkowitz GS, Butt KM, Hong J, Sclafani SJ. Axillary and subclavian vein stenosis: Percutaneous angioplasty. Radiology 1988;168:371-3.
9. Kundu S. Central venous obstruction management. Semin Intervent Radiol 2009;26:115-21.
10. Jennings WC, Miller GA, Coburn MZ, Howard CA, Lawless MA. Vascular access flow reduction for arteriovenous fistula salvage in symptomatic patients with central venous occlusion. J Vasc Access 2012;13:157-62.

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