



Case Report

Central Vein Stenosis in Patient with Routine Haemodialysis: From Diagnosis and Prompt Treatment

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ABSTRACT

Background: Central vein stenosis (CVS) is common in hemodialysis patients and is caused by the implantation of cardiac intravascular devices, venous access, and limited vascular access. The effect may develop until an arteriovenous fistula (AVF) or graft is established in the ipsilateral arm or forearm for hemodialysis. Arm edema, significant venous dilatation, and recurring infections are common complications of having an ipsilateral arteriovenous fistula or graft.

Case Illustration: We present the case of a 50-year-old female who developed symptomatic CVS while being on regular hemodialysis. Venography using a catheter revealed significant stenosis of the subclavian vein. Due to central venous stenosis, this patient had substantial edema of her left arm after undergoing an ipsilateral arteriovenous graft. Multiple endovascular treatments have failed to alleviate the symptoms.

Discussion: The incidence and danger of central vein stenosis in hemodialysis patients are discussed, as well as the success of endovascular therapies. The initial line of therapy should be percutaneous balloon angioplasty.

Conclusion: Unfortunately, after angioplasty or stenting, primary patency is poor. If there is recurring stenosis, the procedure can be repeated.

1. Introduction

The incidence of Central Vein Obstruction (CVO) ranges between 25% and 40%. The previous history of venous catheterization is the most common cause and serious complication of CVO in hemodialysis access. For individuals with end-stage renal disease (ESRD), hemodialysis is the most commonly prescribed treatment. This treatment should be given through an arteriovenous fistula (AVF) whenever possible, as this affords the best vascular access. This procedure has fewer infection concerns, as well as a lower rate of morbidity and mortality when compared to other techniques of kidney replacement therapy. In the treatment of hemodialysis (HD) patients, central venous stenosis (CVS) and obstruction (CVO) is a common and serious conditions. By producing venous hypertension with or without severe symptoms, CVD jeopardizes the integrity of the hemodialysis access circuit. This can lead to the access site being lost due to access malfunction or being ligated for symptom relief. In the literature, the incidence of CVD has been estimated to be in the 30 percent range. Stenoses develop in about 40% of patients who had previously undergone a subclavian vein hemodialysis catheter.¹

Cannulation of the central venous system can lead to central venous stenosis. In dialysis patients, ipsilateral CVC placement is the most common cause. However, it can develop without prior catheter implantation history. While endovascular therapy has a high initial technical success rate, it is associated with a low primary patency rate and a high failure rate, both of which have adverse consequences (e.g., the development of upper limb edema). The therapy of CVS is changing, and the best management method is yet unknown. The findings of trials examining the safety and efficacy of endovascular methods such as balloon angioplasty and venous stenting in CVS were positive; nonetheless, aided primary patency rates tend to decline during the first 12 months, necessitating additional treatments to preserve AVF patency. This case study aimed to examine central venous thrombosis caused by a hemodialysis catheter from a clinical standpoint, from diagnosis through treatment.²

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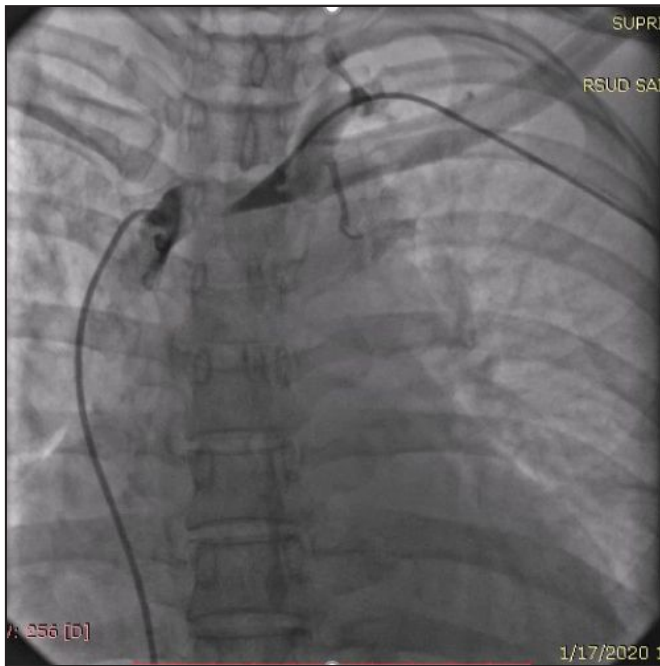


Figure 1. Venography before percutaneous transluminal venoplasty

2. Case Illustration

A 50-year-old woman with the chief complaint of a swollen left hand. She had Swollen on the left hand for 1.5 years before admission, accompanied by pain, the skin on the left hand was reddish, and sometimes felt pins and needles. She went to the nearest hospital. They performed an ultrasound examination of the left hand and continued with an imaging test of the left arm blood vessels. The result was a narrowing in the left arm vein. The patient was then referred to our hospital for further treatment. For the past medical history, she was suffered from Chronic Kidney Failure 2 years ago and routinely got dialysis two times a week. She had implantation of the double lumen in the left hand during the initial 5 months of dialysis. Because the left hand began to swell, then the patient performed AV-Shunt implantation 1.5 years ago. For the risk factors, she got no Diabetes mellitus. However, she has had hypertension since 4 years ago. Routine medications were Lisinopril 1x10 mg, Amlodipine 1x10 mg, Bisoprolol 1x2.5 mg, Atorvastatin 1x20 mg. From family history, her mother had hypertension.

Clinical Manifestation BP 160/95 mmHg HR 96 bpm RR 18 tpm SpO2 99% room air, Weight: 70 kg, Height: 155 cm, BMI 29.1 kg/m², BSA 1.69 m². JVP R+2 cmH₂O, Ictus cordis was invisible, palpable at ICS VI MCL. S1-S2 normal, no murmur and gallop. Vesicular breath sound, no crackles or rales, no wheezing. Extremities edema at the left arm. Left arm locational status was arm circumference 36 cm with edema and redness. Chest X-ray showed Cardiomegaly. For the Duplex Ultrasound examination in August 2019, the result was suspicious of stenosis in anastomose AV-Shunt of the left ulnar artery – left cephalic vein, and there were no aneurysms or DVT along the upper left limb. Then we continued to perform Peripheral Arteriography in Sept 2019, the result was normal arteriography, AV – Shunt patent, and there was an enlargement in the proximal one-third radial vein up to the superior vein of upper left extremities. From all the examinations, we conclude that she got suspicious of central venous stenosis. Then we performed venography and continued with venoplasty. The venography upper left extremity revealed stenosis 100% at the Subclavian vein, irregularity at the axillary vein, and stenosis 70% at the radiocephalic shunt anastomosis (Figure 1).

We performed a percutaneous transluminal venoplasty procedure. The patient was laid on the operating table and disinfected with 10% povidone-iodine in the left radial region and right and left inguinal. The patient was closed with a sterile cover and given local anesthesia with 2% pehacaine in the left radial and right inguinal regions. The 6F radial sheath with the Selinger technique was inserted in the left radial artery. Then the MPA-1 5F diagnostic catheter was advanced to the Cephalic Vein via the AV Shunt with the guidance of Terumo hydrophilic wire J-stiff 260 cm. Wire Entering the left Subclavian vein, cine-angiography was performed. There was stenosis in the left subclavian vein. Another puncture was performed on the right femoral vein, and 7F femoral sheath was inserted. Then, JR 3.5 6F diagnostic catheter with a Guidewire Terumo hydrophilic wire J-stiff 260 cm directed towards the left subclavian vein for cine-angiography. There was stenosis in the left subclavian vein. Heparin 5000 IU IV was given, and a Mustang 5x40mm balloon was directed through the left radial artery to the left Subclavian vein. The balloon successfully penetrated the stenosis and was inflated several times with a pressure of 10 atm for 2 minutes. After that, cine-angiography revealed minimal flow in the left subclavian vein. The balloon was directed to radiocephalic shunt anastomosis and then inflated for 8 atm for 2 minutes, followed by 10 atm for 2 minutes. We changed the Mustang 5x40mm balloon with the Mustang 10x40mm balloon to the left Subclavian vein (Figure 2). The balloon was inflated gradually along the left Subclavian vein with 8 atm for 2-minute. After that, the cine-angiography showed adequate flow in the left Subclavian vein (Figure 2). All devices were removed, and the procedure was completed. We successfully performed percutaneous transluminal venoplasty at the stenosis left Subclavian vein with adequate flow and suggested optimal medical treatment.

3. Discussion

A pathophysiologic venous luminal narrowing that obstructs blood flow is referred to as venous obstruction. The obstruction could be partial or complete. Obstructions were further classified as central, which meant the obstruction was closer to the right atrium, BCVs or SVC, or peripheral, which was farther away from the right atrium, BCVs, and SVC, for example, SCV and IJV obstructions.³ Fibrosis, organized mural thrombus, or De novo smooth muscle hyperplasia could cause venous wall thickening, as could stent, implanted cardiac rhythm device leads, stent graft, or catheter. Wall thickening could be caused by infection, tumor infiltration, inflammation, hematoma, or intramural dissection. The endoluminal obstruction, which is most commonly caused by a thrombus, could also be caused by the implanted endoluminal device (as mentioned above) or the secondary formation of fibrous or neointimal tissues.⁴ Swelling and discomfort in the arm or neck and venous distension were common clinical features. In some cases, patients might experience unusual symptoms, such as jaw or shoulder pain.⁴ In our case, the patient had left arm swelling after having a double lumen for dialysis access implanted.

CVS remained a difficulty for doctors working in vascular access maintenance. It was frequent in HD patients and constituted a significant danger to the AVF's development and patency. Several different factors could cause catheter-related thrombosis (CRT). These factors are linked to Virchow's triad of hypercoagulability, stasis, and endothelial damage described as thrombus-forming components. In the HD population, CVS was thought to be caused by a combination of vein damage generated by central venous catheterization for temporary access and AVF formation causes higher flow and turbulence.⁵

Animal models of vein injury had shown that a "critical area" of injury was required for platelet microthrombi to form within 24 hours, followed by smooth muscle proliferation over the next 7–8 days. Subclavian vein atherectomy specimens from individuals with symptomatic stenosis or occlusion revealed intimal hyperplasia and

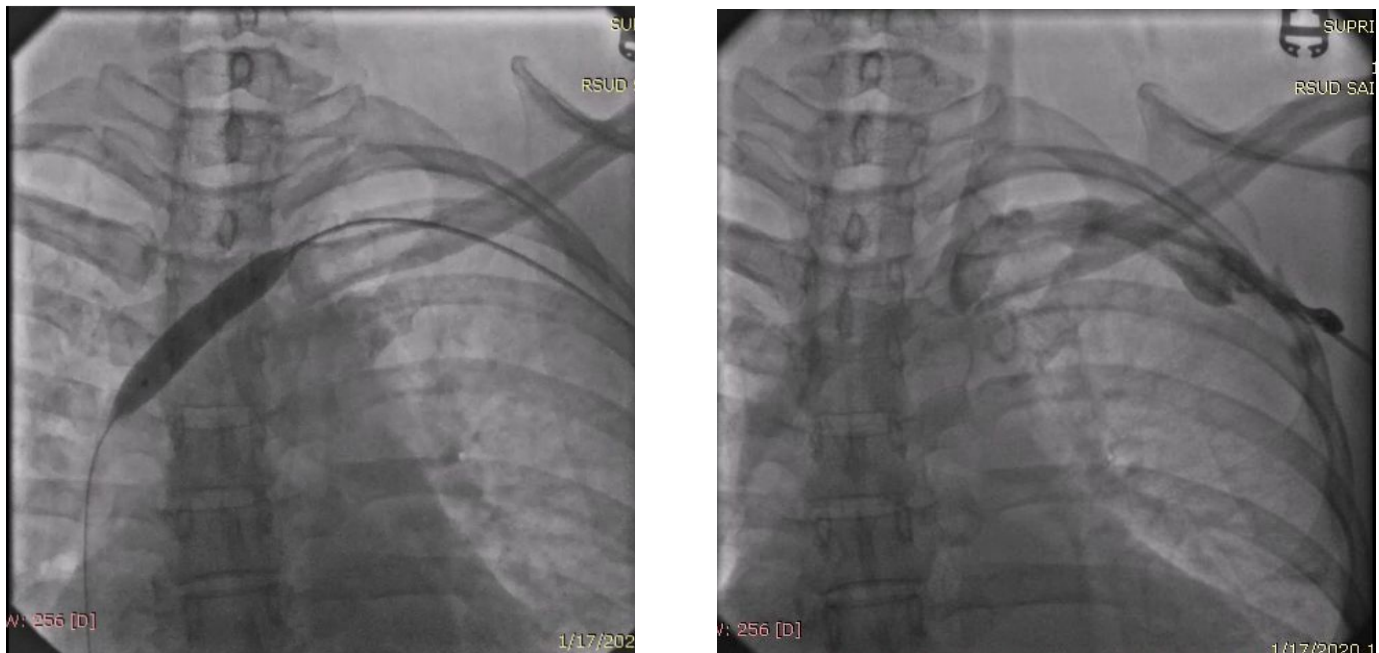


Figure 2. Venography during and after percutaneous transluminal venoplasty procedure

fibrous tissue alterations. Thrombus could form inside, around, or at the catheter's tip. Fibrin sheaths, sock-like structures that formed on the catheter's external surface within 24 hours of insertion, could obstruct flow into and out of the catheter. When blood refluxed into the catheter, an intraluminal thrombus was formed. These occlusions could be partial or complete and could occur because of insufficient flushing, low infusion rates, or a combination of these factors.^{6,7} The link between the placement of a tunneled dialysis catheter in the upper limb and the development of CVS was well known. The risk of CVS was increased for those who have had subclavian vein catheters in the past, and recommendations were made to use the internal jugular vein instead of the subclavian vein for tunneled dialysis catheter placement. To limit the risk of thrombosis, CVCs should be put in the jugular vein on the right side of the body, with the tip near the intersection of the superior vena cava and the right atrium, according to the International Society of Thrombosis and Hemostasis recommendations.⁷

For the diagnosis of CRT, a venous duplex is recommended. If a suspicion of CRT is found from clinical presentation, or if lumen patency is not restored with simple measures, duplex ultrasound is the first imaging modality of choice. It is non-invasive and especially good at detecting thrombi in anatomically accessible veins like the axillary, jugular, arm, and distal subclavian. The left and right arm's superficial and deep venous systems might be evaluated from the wrist to the central veins if technically viable (axillary or distal subclavian vein).⁶ Certain sonographic parameters for assessing veins prior to vascular access implantation were found by Silva et al. When using a tourniquet. An AVF requires a 2.5 mm venous luminal diameter and grafts require a diameter of 4.0 mm. The ipsilateral upper arm's deep venous system had to be connected, and there had to be no blocked segments or segmental stenoses. In ultrasound tests without the application of a tourniquet, a minimum diameter of the cephalic vein of >2.0 mm results in a much larger proportion of well-matured fistulas. Stenosis was also detected using parameters such as the presence of abnormal Doppler finding at the site of stenosis, post-stenotic turbulence, spontaneous contrast, slow flow, poor augmentation, vein dilatation, and contralateral asymmetry in the absence of superior vena cava (SVC) or inferior vena cava (IVC) obstruction, prior to the stenosis.⁸ By comparing the smallest lumen to the usual lumen, planimetry was utilized to

determine the vein diameter decrease. The luminal reduction was measured using B-mode, color, and power Doppler in both the longitudinal and transverse views. The diameter reduction was used to calculate a >50 percent stenosis and an area reduction of >75 percent. The preferred approach to CVS has been angioplasty with or without stent placement. The preferred treatment for CVS is the percutaneous intervention with transluminal angioplasty. The interval between the time of graft deployment and any intervention designed to maintain or reestablish the patency was defined as primary patency (intervention-free stent-graft patency). The interval between graft insertion and access abandonment or measurement of patency was defined as secondary patency, which included any intermediate treatments (surgical or endovascular procedures) to restore thrombosed access functioning (access survival till abandonment).^{9, 10}

In angioplasty, we can use a variety of balloons, including "high-pressure," "ultrahigh-pressure (UHP)," "cutting," and "drug-eluting." We used Mustang balloons in this case, which are high-pressure, non-compliant balloons. They are dedicated to treat dialysis vascular access stenosis and have burst pressures ranging from 20 to 24 atm.¹¹ PTA had a high initial technical success rate, ranging from 70 to 90%. Primary and cumulative patency rates varied widely, with primary patency rates ranging from 23 to 63 percent at 6 months and 12 to 50 percent at 12 months and cumulative patency rates ranging from 29 to 100 percent at 6 months and 13 to 100 percent at 12 months. Following angioplasty, restenosis areas showed more aggressive neointimal hyperplasia and proliferative lesions than stenotic lesions.¹¹

The fact that many central vein lesions were quite elastic was a major issue. With repeated angiography, secondary patency could be significantly improved. The central veins were much more likely than the peripheral veins to recoil, and the success of PTA was determined by the lesion's elastic or nonelastic nature. Cost considerations were important, as was the fact that we had no other option for effectively treating restenosis inside a stent. Even if we double the half-life of recurrent stenotic access (from every 3 months to every 6 months), it might appear to be a significant achievement, but it had little clinical significance.¹²

Endovascular procedures could be completed quickly and without the need for extensive preoperative planning. Second, hemodialysis could be resumed immediately if the AVF was successfully recanalized. Temporary hemodialysis catheter indwelling was not required during wound recovery following surgical thrombectomy. Because of these advantages, endovascular thrombectomy was suggested as the first line of therapy for individuals with thrombosed AVFs.¹²

In the case of CRT treatment, the consensus is that systemic anticoagulation should be used for at least three months. If the catheter remained in place after three consecutive months of anticoagulation, prophylaxis was advised until the line was removed. Due to a paucity of high-quality information, the duration of anticoagulation after removing a line in people with no persisting thrombotic risk factors is currently debated. Some doctors prescribed anticoagulants for three months, while others prescribed them for less time. Other potential thrombosis risks, the size of the clot, and the extent to which it occluded the vessel should all be factored into the decision-making process. If there were no risk factors and the clot was small and non-occlusive, six weeks of anticoagulation might be sufficient.¹⁰⁻¹²

4. Conclusion

A catheter-based hemodialysis is still a feasible option for many chronic kidney disease (CKD) patients. In addition to catheter-related infections, CRS development was responsible for a considerable share of catheter malfunction. The venous wall's reaction to the catheter and the thrombus it caused was dynamic and continuous. It involved biologically active cell types and bore many resemblances to the thrombus formation process. PTA was a relatively safe and effective treatment for CVS, with a high success rate, few side effects, and a good radiologic and clinical outcome.

5. Declarations

5.1. Ethics Approval and Consent to participate

This study was approved by local Institutional Review Board, and all participants have provided written informed consent prior to involvement in the study.

5.2. Consent for publication

Not applicable.

5.3. Availability of data and materials

Data used in our study were presented in the main text.

5.4. Competing interests

Not applicable.

5.5. Funding source

Not applicable.

5.6. Authors contributions

Idea/concept: YPA, NK. Design: YPA, NK. Control/supervision: NK. Literature search: YPA, NK. Data extraction: YPA, NK. Statistical analysis: YPA, NK. Results interpretation: YPA, NK. Critical review/discussion: NK. Writing the article: YPA. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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References

1. Kreuziger LB, Jaffray J, Carrier M. Epidemiology, diagnosis, prevention and treatment of catheter-related thrombosis in children and adults. *Thrombosis Research*. 2017;157:64-71.

2. Kamphuisen PW, Lee AY. Catheter-related thrombosis: lifeline or a pain in the neck? *Hematology 2010, the American Society of Hematology Education Program Book*. 2012;2012(1):638-44.
3. Geerts W. Central venous catheter-related thrombosis. *Hematology 2014, the American Society of Hematology Education Program Book*. 2014;2014(1):306-11.
4. Grant JD, Stevens SM, Woller SC, Lee EW, Kee ST, Liu DM, et al. Diagnosis and management of upper extremity deep-vein thrombosis in adults. *Thrombosis and haemostasis*. 2012;108(12):1097-108.
5. Monreal M, Raventos A, Lerma R, Ruiz J, Lafoz E, Alastrue A, et al. Pulmonary embolism in patients with upper extremity DVT associated to venous central lines-a prospective study. *Thrombosis and haemostasis*. 1994;72(10):548-50.
6. Forauer AR, Theoharis CG, Dasika NL. Jugular vein catheter placement: histologic features and development of catheter-related (fibrin) sheaths in a swine model. *Radiology*. 2006;240(2):427-34.
7. Suojanen JN, Brophy DP, Nasser I. Thrombus on indwelling central venous catheters: the histopathology of "fibrin sheaths". *Cardiovascular and interventional radiology*. 2000;23(3):194-7.
8. Gunawansa N, Sudusinghe DH, Wijayaratne DR. Hemodialysis catheter-related central venous thrombosis: clinical approach to evaluation and management. *Annals of Vascular Surgery*. 2018;51:298-305.
9. Köksoy C, Kuzu A, Kutlay J, Erden I, Özcan H, Ergin K. The diagnostic value of colour Doppler ultrasound in central venous catheter related thrombosis. *Clinical Radiology*. 1995;50(10):687-9.
10. Wall C, Moore J, Thachil J. Catheter-related thrombosis: a practical approach. *Journal of the Intensive Care Society*. 2016;17(2):160-7.
11. 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. *Journal of vascular surgery*. 2007;45(4):776-83.
12. Gür S, Oğuzkurt L, Gedikoğlu M. Central venous occlusion in hemodialysis access: Comparison between percutaneous transluminal angioplasty alone and nitinol or stainless-steel stent placement. *Diagnostic and Interventional Imaging*. 2019;100(9):485-92.