International Journal of Innovative Research in Medical Science (IJIRMS)
Volume 09, Issue 02, February 2024,
https://doi.org/10.23958/ijirms/vol09-i02/1828

Original article

Central Venous Occlusion Management in Nigeria: Six Year Review of Our Experience

Emmanuel Auchi Edafe [1,2,3], Maclean Rumokere Akpa [3]

[1]Bayelsa Specialist Hospital, Yenagoa, Nigeria.
[2]Cardiocare Multispecialty Hospital, Abuja, Nigeria.
[3]University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria.

*Corresponding Author: Edafe EA, *dremmanueledafe@gmail.com*

Received 20 January 2024; Accepted 10 February 2024; Published 15 February 2024

Abstract

Introduction: The majority of cases of stenosis or occlusion of central veins are the result of prolonged use of central catheters, neoplasms or traumas. Central vein occlusion is also common among patients with cardiac devices or chronic repeated thrombi. Central venous occlusion could be severe due to engorged neck veins, thrombosis, venous hypertension and edema of the extremities. The objective of this study is to discuss endovascular management of central venous occlusion in three Nigerian patients. ***Methods:*** This is a retrospective study of patients seen in cardiac catheterization laboratory and the Vascular C-Arm of the Bayelsa Specialist Hospital and Cardiocare Multispecialty Hospital over a period of 6 years from January 2018 to December 2023. The data were extrcated from the patients' record and the data analyzed with SPSS version 25.0 for windos. ***Results:*** There were 31 patients in the 6-year of review. There were 19 males and 12 females. The average age of patients was 64.0 ± 9.8 years. The commonest symptoms were facial swelling, upper extremity swelling and pain. The commonest etiology was use of long indwelling venous catheters. ***Conclusion:*** Central vein stenosis and its risk factors are common in Nigeria. The endovascular treatment included use of balloon and stents.

Keywords: ***central venous occlusion, management of venous stenosis, venous stenosis.***

Introduction

Central venous occlusions (CVOs) refers to the occlusion of any of the body's central veins [1,2]. The central veins include jugular, subclavian, brachiocephalic, superior and inferior vena cava veins [3-5]. The occlusion results from injury to the vein or thrombus formation. Central veinous injuries result majorly from indwelling veinous catheters or cardiac device leads inserted for various medical conditions [4].

The majority of cases of stenosis or occlusion of central veins are the result of prolonged use of central veinous catheters, neoplasms or traumatic injuries [6,7]. Central vein occlusion is also common among patients with cardiac devices or chronic repeated thrombi [8,9]. Central venous occlusion could be severe and results in engorged neck veins, thrombosis, venous hypertension, and edema of the extremities [6-9]. The objective of this study is to discuss endovascular management and outcomes of central venous occlusion in two Nigerian hospitals.

Methods

This is a retrospective study involving the review of the medical records of patient that were treated at the Bayelsa Specialist Hospital, Yenagoa and Cardiocare Multispecialty Hospital in Abuja. The review covered the period of 6 years from 1st January 2018 to 31st December 2023.

The records of the cardiac catheterization laboratory and the Vascular C-Arm of two tertiary hospitals in Nigeria, The Bayelsa Specialist Hospital, Yenagoa and the Cardiocare Multispecialty Hospital in Abuja provided the data for this review; are tertiary hospitals located in Yenagoa and Abuja respectively in Nigeria. These are referral centres for cardiac catheterization laboratory and vascular C-arm cases across Nigeria. The demographic and clinical records of all the patients managed for central veinous occlusion were extracted from the laboratories of the two hospitals. The data were analyzed using SPSS version 25.0 for windows

Results

There were 31 patients in the period under review, 19 males 61.3% and 12 females 38.7% aged 44 to 79 years. The average age of the patients at presentation was 64.0 ± 9.8 years. The common symptoms were facial swelling, upper extremity swelling and pain. The common etiologies were use of long indwelling catheters, thrombus formation, pacemakers/defibrillator leads and a small number resulted from neoplasms, radiation injury and trauma. The cause of veinous occlusion in 17 patients was indwelling catheters

[54.8%], cardiac devices in 6 subjects [19.4%]. This is shown in table 1. A procedure images are shown in figure 1 and 2.

Table 1: Demographic characteristics and central vein involvement.

Variable	Frequency	Percentage
Sex		
Male	19	61.3%
Female	12	38.7%
Total	31	100
Age		
≤ 50years	13	41.9%
>50years	18	58.1%
Total	31	100
Etiology		
1. Indwelling Tunnel catheters	17	54.8%
2. Thrombi	4	12.9%
3. Cardiac devices leads	6	19.4%
4. Others	4	12.9%
Central veins involved		
1 right subclavian	12	38.7%
2.left subclavian	6	19.4%
3. right internal Jugular	9	29%
4. left internal Jugular	7	22.5%
5.bronchocephalic vein	8	25.8%
5, Superior Vena Cava	6	19.4%
Treatment		
1.Venoplasty with balloons	19	61.3%
2. Venoplasty with balloons and peripheral stents	12	38.7%
3. Anticoagulation	31	100%

Figure 1a: Our patient with central venous occlusion. The distended next veins marked the clinical presentation.

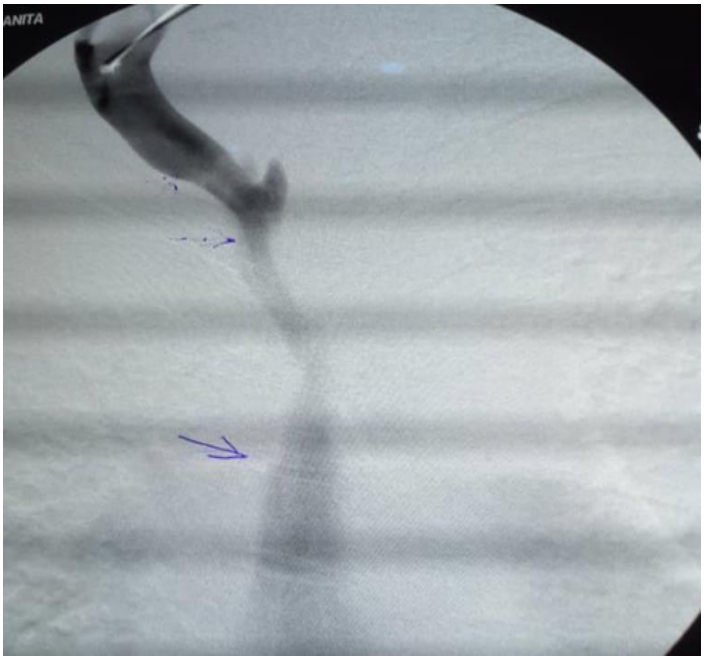


Figure 1b: Venogram of the SVC, Internal Jugular vein and brachiocephalic vein

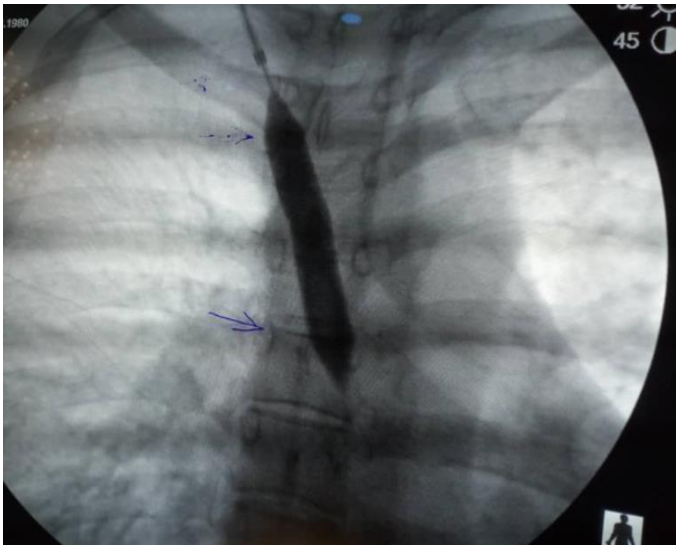
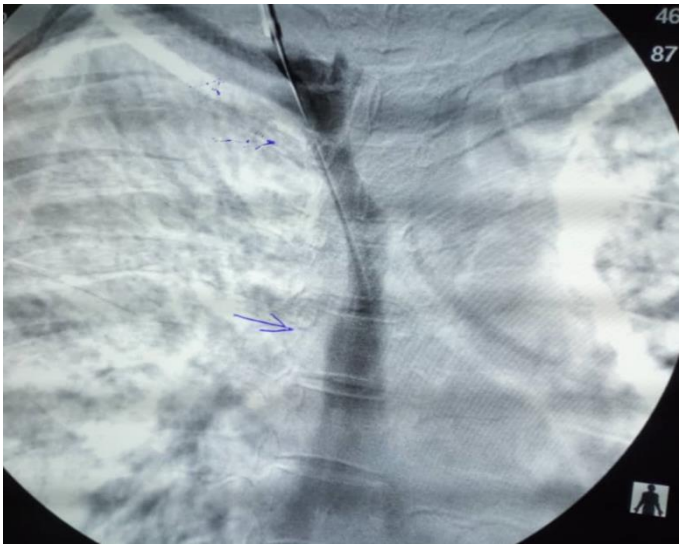


Figure 1c. during the balloon venoplasty of the IJV, and SVC



1d. Post balloon venoplasty

Figure 1: showed venoplasty of the central vein. The access was through the right internal Jugular vein.

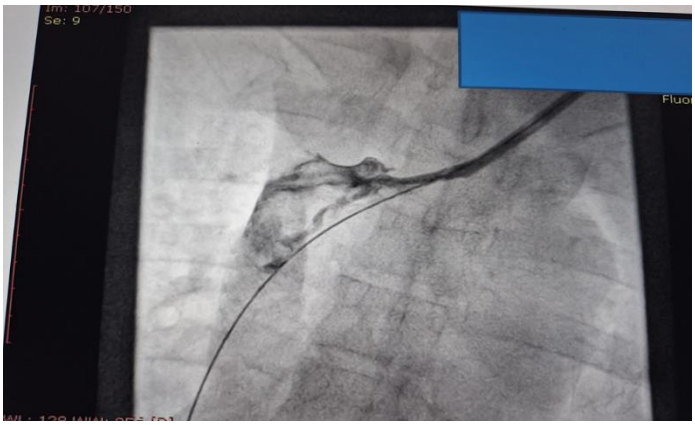


Figure 2a: Venography of the left subclavian and the brachiocephalic veins.

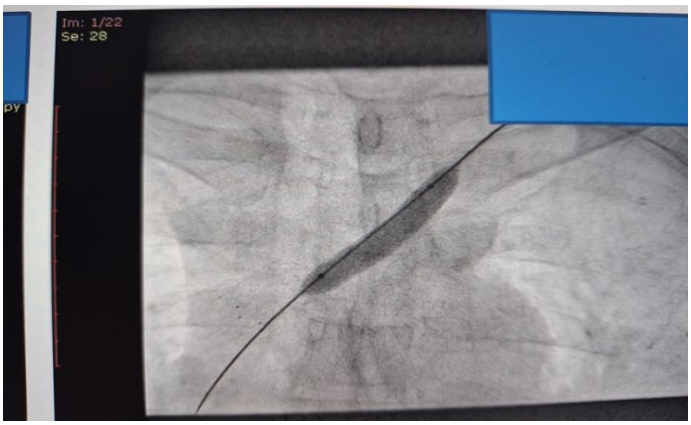


Figure 2b: during balloon venoplasty of the left subclavian and brachiocephalic vein

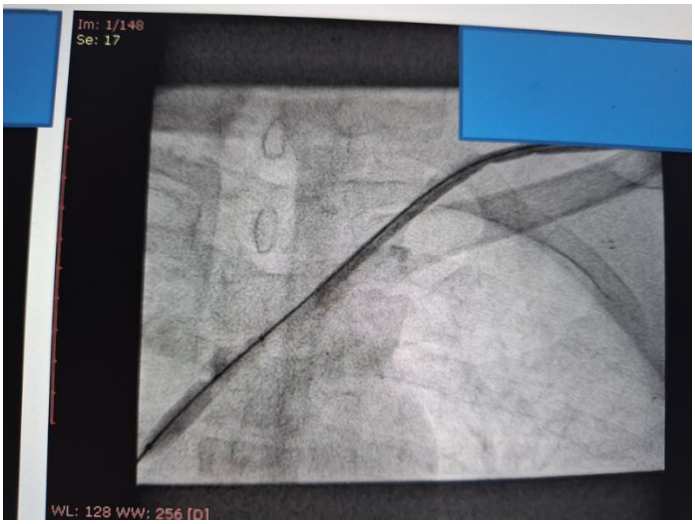


Figure 2c: balloon venoplasty

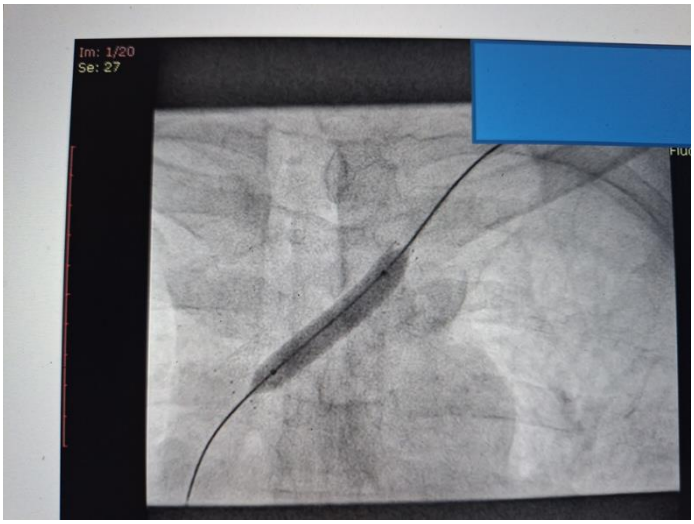


Figure 2d: Venoplasty with peripheral stent and post balloon

Figure 2: Showed a central venous occlusion from Tunnel dialysis catheter. The patient had balloon and stent venoplasty.

Discussion

In Central Venous occlusions, the upper limbs are commonly involved than the lower limbs [3]. The superior vena cave [SVC] is commonest vein involved in upper limb venous occlusion. The benign etiologies include chronic dialysis catheters, cardiac Devices-leads, trauma, in some the etiology is idiopathic [1-3]. In the lower extremities, the etiologies include deep vein thrombosis and post-thrombotic and Venous composure (May-Thurner Syndrome) [4,5]. Most of our subjects in this review had end stage renal disease and were on chronic hemodialysis.

Criteria for significant occlusion

The criteria for significant stenosis include: [6-9]

1. Collateral circulation 2. Pressure gradient (2-3 mmHg) 3. Area of stenosis > 50% There is no available curative treatment for this condition [5-8]. The goal of treatment is to prevent complications and minimize symptoms as much as possible [9]. Endovascular treatment is the first line therapy of choice in central venous occlusions but this may not be readily available to practitioners [10-16]. The endovascular procedures include balloon venoplasty, stent angioplasty or a combination of both. Noninvasive medical treatment involves the use of thrombolytics and or anticoagulation [17-25]. In addition to anticoagulation, these 31 patients received either balloon venoplasty alone or with the use of stent.

Etiologies and Risk factors of Central venous occlusion [CVO].

There are many risk factors for central vein occlusion. These include prolonged indwelling catheters for hemodialysis and chemotherapy, cardiac defibrillators/leads, chronic thrombosis, neoplasm, trauma, radiation etc [26-29].

In CVO, the location of the central venous catheter is a key causative factor for occlusion [16-19]. Central venous catheters placed through a subclavian access have a particularly high risk of CVO compared with catheters placed via an internal jugular vein access [30-33]. Also left sided central vein access have more risk of occlusion than right sided access [33-36]. This may be related to the longer route and the more tortuous course catheters have to traverse from a left-sided access [14,15,34-36]. In addition, larger caliber catheters may be more prone to inducing injury compared to smaller caliber ones [19]. Pacemakers and ICD defibrillator leads may also lead to CVO with development of clinical symptoms after the placement of an AV access in the ipsilateral extremity. Other less common sources of CVO include thrombus, neoplasms and radiation injury for cancer treatment [14-18].

Diagnosis of CVO

Digital subtraction central venography is the gold standard for the diagnosis of CVO. It is more sensitive than duplex ultrasound [21-24]. Magnetic resonance venography is an alternative to conventional venography and also has high sensitivity [27,28]. Duplex ultrasound is cheap, readily available and in good operators equally gives good yield for diagnosis. Both venography and duplex ultrasound were used in the diagnosis of our patients.

Endovascular intervention is the mainstay of treatment in HD patients with CVD [26-30]. Our patient received endovascular treatment with either balloon venoplasty with or without a stent. 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 [36-40].

Pathophysiology of CVO

The hard wares like the catheters, pacemakers and defibrillator lead irritate and cause endothelial injury with inflammation that damage the vein wall resulting to stenosis. There is a strong association of CVD, with previous placement of central venous catheters and pacemaker leads. In a study, 27% of patients with CVO had a previous history of central venous catheter placement [4-7]. There is a very high rate 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 injury and trauma to the venous endothelium with secondary inflammatory damage within the vessel wall at the time of insertion. Also, the present of foreign body in the vein causes flow turbulence that may lead to injury of the vein [8-12].

Clinical presentation

The most striking presentation are edema and pain of the extremity. This is due to the narrowing or occlusion of the vein leading to venous hypertension of the corresponding extremity. Innominate vein stenosis or occlusion affects blood flow from the same side of the face as well as the upper extremity and breast [13-17]. Prolonged 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 [25-28].

Superior vena cava syndrome is the most feared complication of superior vena cava stenosis or obstruction or bilateral innominate vein narrowing or occlusion [36-40]. 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.

Treatment

Digital subtraction central venography is the gold standard for the diagnosis of CVD. It is more more sensitive than duplex ultrasound [21-24]. Magnetic resonance venography is an alternative to conventional venography [27,28].

Endovascular intervention is the mainstay of treatment in HD patients with CVD [26-30]. Our patient received endovascular treatment with either balloon venoplasty with or without a stent. 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 [36-40].

Conclusion

Symptomatic CVO requires re-establishment of flow and endovascular approach is the first line of treatment. This therapy is now available in Nigeria with good result.

Limitation of the study

The study involved small number of patients. It is a retrospective and some patients with incomplete data were not involved in the study.

Funding

There is no funding for the study.

References

[1] Kundu S. Central Venous Obstruction Management. Semin Intervent Radiol. 2009 Jun; 26(2): 115-121.

[2] Lumsden A B, MacDonald M J, Isiklar H, et al. Central venous stenosis in the hemodialysis patient: incidence and efficacy of endovascular treatment. Cardiovasc Surg. 1997;5(5):504-509.

[3] Glanz S, Gordon D H, Lipkowitz G S, Butt K M, Hong J, Sclafani S J. Axillary and subclavian vein stenosis: percutaneous angioplasty. Radiology. 1988;168(2):371-373.

[4] Agarwal A K, Patel B M, Farhan N J. Central venous stenosis in hemodialysis patients is a common complication of ipsilateral central vein catheterization. J Am Soc Nephrol. 2004; 15:368A-369A.

[5] Barrett N, Spencer S, McIvor J, Brown E A. Subclavian stenosis: a major complication of subclavian dialysis catheters. Nephrol Dial Transplant. 1988;3(4):423-425.

[6] Cimochowski G E, Worley E, Rutherford W E, Sartain J, Blondin J, Harter H. Superiority of the internal jugular over the subclavian access for temporary dialysis. Nephron. 1990;54(2):154-161.

[7] 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.

[8] Vanherweghem J L, 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.

[9] 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

[10] 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

[11] 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

[12] 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

[13] 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

[14] 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

[15] 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

[16] Palabrica T, Lobb R, Furie BC, et al. Leukocyte accumulation promoting fibrin deposition is mediated in vivo by Pselectin on adherent platelets. Nature 1992;359(6398):848-851

[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] Forauer A R, Theoharis C.Histologic changes in the human vein wall adjacent to indwelling central venous catheters J Vasc Interv Radiol 200314(9, Pt 1):1163-1168.

[21] 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(02):237-242.

[22] Hernández D, Díaz F, Rufino M. Subclavian vascular access stenosis in dialysis patients: natural history and risk factors. J Am Soc Nephrol. 1998;9(08):1507-1510.

[23] Taal M W, Chesterton L J, McIntyre C W. Venography at insertion of tunnelled internal jugular vein dialysis catheters reveals significant occult stenosis. Nephrol Dial Transplant. 2004;19(06):1542-1545.

[24] MacRae J M, Ahmed A, Johnson N, Levin A, Kiaii M. Central vein stenosis: a common problem in patients on hemodialysis. ASAIO J. 2005;51(01):77-81.

[25] Lumsden A B, MacDonald M J, Isiklar H. Central venous stenosis in the hemodialysis patient: incidence and efficacy of endovascular treatment. Cardiovasc Surg. 1997;5(05):504-509.

[26] Schwab S J, Quarles L D, Middleton J P, Cohan R H, Saeed M, Dennis V W. Hemodialysis-associated subclavian vein stenosis. Kidney Int. 1988;33(06):1156-1159.

[27] National Kidney Foundation Lok C E, Huber T S, Lee T. KDOQI clinical practice guideline for vascular access: 2019 update Am J Kidney Dis 202075(4, Suppl 2): S1-S164.

[28] Cohen E I, Beck C, Garcia J. Success rate and complications of sharp recanalization for treatment of central venous occlusions. Cardiovasc Intervent Radiol. 2018;41(01):73-79.

[29] Keller E J, Gupta S A, Bondarev S, Sato K T, Vogelzang R L, Resnick S A. Single-center retrospective review of radiofrequency wire recanalization of refractory central venous occlusions. J Vasc Interv Radiol. 2018;29(11):1571-1577.

[30] Guimaraes M, Schonholz C, Hannegan C, Anderson M B, Shi J, Selby B., Jr Radiofrequency wire for the recanalization of central vein occlusions that have failed conventional endovascular techniques. J Vasc Interv Radiol. 2012;23(08):1016-1021.

[31] McGuckin J F, Gosnear A M, Williams R L.A new pathway for central venous occlusion Endovasc Today 2009(July):86-88.

[32] 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

[33] 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

[34] Middleton WD, Erickson S, Melson GL. Perivascular color artifact: pathologic significance and appearance on color Doppler US images. Radiology 1989;171(3):647-652

[35] MacRae JM, Ahmed A, Johnson N, et al. Central vein stenosis: a common problem in patients on hemodialysis. ASAIO J 2005; 51:77-81

[36] Schon D, Whittman D. Managing the complications of longterm tunneled dialysis catheters. Semin Dial 2003;16(4):314-322

[37] 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

[38] Salgado OJ, Urdaneta B, Colmenares B, Garcı´a 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

[39] 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

[40] Baetens T R, Rotmans J I, van der Meer R W, van Rijswijk C S. A novel technique to restore access in patients with central venous occlusion using the Surfacer ® Inside-Out ® Access Catheter System. J Vasc Access. 2020;21(05):778-782.