Central Venous Obstruction-Induced Intracranial Hypertension in Hemodialysis Patients: An Under-Recognized Cause of Elevated Intracranial Pressure

Devin D. Mackay, MD[1-3]; Sara M Takacs, MD[1]

From the Departments of Neurology,[1] Ophthalmology,[2] and Neurosurgery,[3] Indiana University School of Medicine, Indianapolis, IN.

Manuscript Category subtype: Disease of the Year, State-of-the-Art Review

Title character count: 146

Manuscript word count: 2997

Abstract word count: 278

Number of references: 46

Number of tables: 1

Number of figures: 1

Address correspondence to:

Devin D. Mackay, MD, 355 W. 16th Street, Suite 4400, Indianapolis, IN, USA 46202 Phone: (317) 948-5450, Fax: (317) 962-2141; Email: ddmackay@iupui.edu.

The authors have no conflict of interest to disclose.

Funding Disclosures: None

Acknowledgements: None

This is the author's manuscript of the article published in final edited form as:

Mackay, D. D., & Takacs, S. M. (2020). Central Venous Obstruction-Induced Intracranial Hypertension in Hemodialysis Patients: An Underrecognized Cause of Elevated Intracranial Pressure. Journal of Neuro-Ophthalmology: The Official Journal of the North American Neuro-Ophthalmology Society, 40(2), 218–225. https://doi.org/10.1097/WNO.0000000000000964

Abstract

Background:

Central venous obstruction (stenosis or occlusion) is common in patients with renal failure on hemodialysis and may be associated with intracranial hypertension (IH). Causes include vein injury from an endoluminal device, lumen obstruction from a device or thrombus, external vein compression, and high venous flow leading to vein intimal hyperplasia. A combination of high venous flow and central venous obstruction can lead to intracranial venous hypertension, impaired CSF resorption, and subsequent IH.

Evidence Acquisition:

We conducted a search of the English literature using the Ovid MEDLINE® Database and PubMed, with a focus on reports involving IH and central venous obstruction in the setting of hemodialysis. We review CSF flow dynamics, the risk factors and causes of central venous obstruction, and the evaluation, management, and outcomes of central venous obstruction-induced IH.

Results:

Twenty-four cases of IH related to central venous obstruction in hemodialysis patients were identified. Twenty patients had headaches (83.3%) and nine had visual symptoms (37.5%). The brachiocephalic vein was the most common site of stenosis or occlusion (20/24, 83.3%). Twenty-one patients (87.5%) had resolution of IH with treatment. Two patients died from complications of IH (8.3%).

Conclusions:

Central venous obstruction-induced IH is likely underrecognized by clinicians and mimics idiopathic intracranial hypertension. Hemodialysis patients with IH should be screened with CT venography of the chest. Optimal treatment is with vascular intervention or a CSF diversion procedure and can help prevent vision loss from papilledema or nervous system damage. Medical management may be appropriate in mild cases or as a bridge to definitive interventional treatment. Increased awareness among clinicians has potential to facilitate the timely diagnosis of this treatable condition with potential for good neurologic and visual outcomes.

Keywords: Intracranial hypertension, papilledema, hemodialysis, central venous

obstruction, venous hypertension

Introduction

Intracranial hypertension (IH) may occur in patients with renal failure on hemodialysis and is likely underrecognized. A variety of mechanisms related to the establishment of reliable hemodialysis access can cause obstruction of central venous outflow in the neck and chest.[1] As cerebrospinal fluid (CSF) dynamics are sensitive to alterations in intracranial venous pressure, an increase in resistance to venous outflow from the head and neck can lead to venous hypertension, decreased CSF resorption, and IH. The approach to management of IH in hemodialysis patients with central venous obstruction (stenosis or occlusion) differs from that of other causes of IH and the condition is treatable with good potential outcomes, which emphasizes the importance of early diagnosis and treatment.

Cerebrospinal Fluid Flow Dynamics

CSF is primarily made by the choroid plexus in the third, fourth, and lateral ventricles and circulates through the ventricular system and subarachnoid space surrounding the brain, spinal cord, and optic nerves. The majority of CSF resorption occurs in the cranial arachnoid granulations, where the fluid joins the venous circulation in the intracranial venous sinuses.[2] A system of granulations consisting of invaginations of the arachnoid mater into the dura permits close approximation of the CSF and blood in the venous sinuses. Arachnoid granulations have a valvular structure, rather than simply a membrane, which only permits unidirectional CSF flow from the subarachnoid space to the venous circulation.[3] The drainage of CSF through the arachnoid villi is dependent on a pressure gradient between the subarachnoid space and the adjacent venous sinus of at least 3-5 mm Hg.[4] A gradient less than this results in collapse of the arachnoid villus, cessation of CSF flow, and buildup of intracranial pressure.[3] Therefore, any process that increases intracranial venous pressure can decrease the pressure gradient for CSF resorption and result in IH. Recent evidence has also shown a role for spinal arachnoid granulations, transependymal flow, and the lymphatic system as additional CSF outflow pathways.[2, 5] Although CSF drains through a variety of mechanisms, the most significant drainage pathway is through the craniocervical veins. A net total overwhelming of the various CSF drainage pathways leads to IH.

Intracranial Venous Outflow

The sigmoid and transverse venous sinuses accommodate most of the cerebral venous drainage. Numerous connections between the dural venous sinuses of the posterior fossa and the internal and external vertebral plexus provide some flexibility in the distribution of exiting intracranial blood.[6] The predominant pathways of intracranial venous drainage through the neck are position-dependent, with the internal jugular veins (IJV) being the primary drainage route in the supine position, and the vertebral venous systems playing a larger drainage role in the upright position.[7] Therefore, the precise pathway of intracranial venous drainage depends on posture, pressures, and anatomic variations.

Intracranial venous blood en route to the heart, regardless of the pathway taken through the neck, flows through the major central veins of the chest: the brachiocephalic veins and the superior vena cava (SVC). Obstruction of blood flow through the major central chest veins by thrombosis, stenosis, external compression, or other means, can lead to an increase in venous pressure and subsequent IH.

Pathogenesis of IH in the Setting of Central Venous Obstruction

The specific veins that constitute the central venous system have been variable in the medical literature, potentially limiting the usefulness of comparing data from multiple reports. Recognition of this problem led to the development of reporting standards for thoracic central veins, which term includes the SVC, brachiocephalic veins, subclavian veins, IJV below the first rib, and azygous vein, and excludes the axillary veins (lateral to the first rib) (Figure 1).[8] Notwithstanding the past variability in reporting standards, central venous stenosis is common in hemodialysis patients, affecting approximately 16-42% of patients, depending on the series, and is likely underreported in asymptomatic cases.[1, 9, 10] However, despite the relative prevalence of central venous stenosis, the prevalence of papilledema in hemodialysis patients is low. In one cross-sectional observation case series of 44 hemodialysis patients with peripheral arteriovenous dialysis shunts, not one patient had papilledema or symptoms of IH.[11] Therefore, central venous obstruction alone is unlikely to account for IH in hemodialysis patients. This observation lead to the proposal of a “two-hit” hypothesis to explain why only some hemodialysis patients developed IH.[12] The “two-hit” hypothesis proposes that both 1) central venous obstruction and 2) increased venous flow from an arteriovenous shunt are required to cause IH. Increased venous flow through an arteriovenous fistula or graft can overwhelm the flow capacity of an obstructed central vein and lead to retrograde flow through the IJVs, which causes intracranial venous hypertension and subsequent IH. Without both “hits”, retrograde flow through the IJVs is much less likely to occur.

The site of central venous stenosis appears to play an important role in the development of IH. Retrograde flow through the IJV or vertebral vein is a common feature in reported cases of IH from central venous obstruction.[13-16] Most cases involve the brachiocephalic veins or IJVs (table 1), rather than subclavian vein stenosis, as stenosis of the more central veins is more likely to lead to retrograde venous flow in the neck.[17, 18] A valve in the IJV typically prevents pressure variations in the extracranial venous system from affecting cerebral venous drainage, but venous hypertension can lead to valvular incompetence, retrograde IJV flow, and IH.[18-20] Increased venous flow (part of the "two-hit" hypothesis) from an arteriovenous shunt in the setting of central venous obstruction involving the brachiocephalic or IJVs, in particular, likely leads to IH by inducing retrograde venous flow in the neck. Brachiocephalic vein stenosis tends to be less common than stenosis involving other central veins (e.g., subclavian), which may help explain why IH is still an uncommon finding despite the prevalence of hemodialysis arteriovenous fistulas/grafts and central venous stenosis.[9, 13]

How Does Central Venous Stenosis Occur?

Understanding why central venous stenosis occurs can help identify high-risk patients. There are four main mechanisms by which central venous stenosis occurs in hemodialysis patients: 1) Vein injury related to an endoluminal device, such as a central catheter, 2) endoluminal obstruction related to a thrombus or endoluminal device, 3) extrinsic vein compression, and 4) hemodynamic abnormalities, such as turbulent blood flow, leading to neointimal hyperplasia and stenosis.[1]

Vein injury from an endoluminal device

Central or peripherally inserted central catheters (CICC or PICC), as they are inserted, are typically advanced to the superior vena cava. As the catheter is advanced, several turns may be necessary, depending on the access site, which increases the risk of vein injury as the catheter collides with the vessel wall. The risk may be higher with insertion in either subclavian vein where a turn is needed to follow the course of the brachiocephalic vein into the SVC, or the left internal jugular vein, where two turns are needed (one from the IJV to the brachiocephalic vein, and another to the SVC).[21] Insertion through the right IJV yields a straight access trajectory to the SVC (Figure 1). The leads from a cardiac implantable device can also lead to vein injury. Vein injury leads to neointimal hyperplasia, and consequently vein stenosis.[9]

Endoluminal obstruction related to a thrombus or intraluminal device

The presence of an obstruction in the vessel lumen may also cause a functional stenosis. A thrombus or intraluminal device, such as a catheter, may partially or fully occlude the vessel, also limiting venous blood flow.[22]

Extrinsic vein compression

External compression of the central veins most typically occurs from an adjacent artery, such as an ectatic aorta compressing the left brachiocephalic vein, or from a musculoskeletal cause, such as costoclavicular compression of the subclavian vein. The resulting functional stenosis may cause symptoms when it is sufficient to impede venous flow.[1]

Hemodynamic abnormalities causing neointimal hyperplasia

Some patients may have central venous stenosis from hemodynamic abnormalities, such as turbulent blood flow, without any history of endoluminal device placement or extrinsic compression.[9]

Review of Published Cases of Central Venous Stenosis-Induced IH in Hemodialysis Patients

A systematic review was conducted of currently available English literature regarding cases of presumed IH from central venous stenosis in patients with chronic renal failure on hemodialysis. PubMed and Ovid MEDLINE® databases were searched using search terms “intracranial hypertension”, “venous stenosis”, “venous occlusion”, and “hemodialysis”. The resulting articles were analyzed for relevance, and their references were also scanned in order to identify any additional relevant cases. Twenty-four cases were identified (Table 1).

There were 12 cases receiving hemodialysis through an arteriovenous fistula, ten through an arteriovenous graft, and two through a central venous line. Most patients had undergone venous access multiple times in the past for central venous catheters, surgical correction of shunts,[13] and prior angioplasty for venous stenosis.[17, 23, 24] In some cases, there was previous venous access for another purpose such as total parenteral nutrition[25] or cardiac pacemaker lead placement.[12, 18, 26]

Presenting symptoms included headache in most patients (20/24, 83.3%). Visual complaints such as transient visual obscurations, progressive vision loss, or diplopia were reported in nine cases (37.5%). Papilledema was noted in ten cases (41.7%), although it is very unlikely that all patients underwent a funduscopic exam, and the true prevalence of papilledema in this series may have been substantially higher. No cases in the series reported a lack of papilledema when specifically examined. Cerebrospinal

fluid opening pressure was reported in eight cases, ranging from 27-56 cm H_2O. Many also had evidence of extracranial venous congestion such as unilateral upper extremity or facial edema and development of tortuous collateral veins. Most exhibited an indolent onset of symptoms over weeks to months, although five cases (20.8%) had a fulminant course.[14, 17, 18, 26, 27]

The most common location of central venous stenosis or occlusion was the brachiocephalic vein (20/24, 83.3%) followed by the subclavian vein (7/24, 29.2%), with ten cases involving stenosis or occlusion of multiple central veins. The left brachiocephalic vein was more commonly affected than the right with 13 cases involving the left brachiocephalic vein, four involving the right brachiocephalic vein, and three with bilateral involvement. One patient had hydrocephalus on MRI[26] and another had MRI findings typical of increased intracranial pressure.[17] Several patients developed intracranial hemorrhage or white matter edema, theorized to be due to cerebral venous hypertension.[15, 18, 22, 28, 29] Enlarged venous collaterals and retrograde venous flow, most commonly in the internal jugular vein (18 cases), were noted on vascular imaging in the majority of cases.

Treatment consisted of angioplasty and stent in the region of central venous stenosis in five cases, angioplasty without stent in five cases, shunt ligation in 11 cases, and ventriculoperitoneal shunt placement in one case. One report described treatment with a combination of partial arteriovenous shunt closure, lumboperitoneal shunt, and venous angioplasty.[23] Acetazolamide and optic nerve sheath fenestration were used as temporizing measures in two cases.[18, 30] One patient required repeat venoplasty after IH recurred due to central venous stenosis.[18] Of all reported cases, 21 (87.5%) had complete resolution of IH with treatment. Of those with resolution of IH, only two had residual vision loss while the remainder had recovery of vision.

Two patients (8.3%) died from complications of IH, one with deferral of treatment due to sepsis with subsequent fatal intracranial hemorrhage,[22] and one who developed venous sinus thrombosis after treatment, with a prolonged convulsion that lead to brain death.[23]

Risk Factors

Identification of risk factors associated with IH in hemodialysis patients can help clinicians adjust their index of suspicion for a specific patient, justify appropriate additional testing, and facilitate an earlier diagnosis to minimize permanent vision damage from papilledema or avoid other complications.

Hemodialysis patients with symptoms and signs of IH, including headache, pulse-synchronous tinnitus, papilledema, and transient visual obscurations should certainly

prompt further imaging for causes of secondary IH. Additional helpful information includes a history of venous access procedures including the location of the access site, any history of central venous angioplasty or stent placement (as recurrent stenosis is common), arteriovenous shunt failure, or catheter-related infection.[9, 31]

Uremia has been proposed as a cause of optic neuropathy with optic disc edema, but without convincing evidence regarding a direct toxic effect. At least some of the cases attributed to uremia toxicity are more likely accounted for by nonarteritic anterior ischemic optic neuropathy (NAION) or possibly papilledema associated with IH due to unrecognized central venous obstruction.[32-34]

Establishing the Diagnosis

Evaluation of a hemodialysis patient with suspected IH should begin with a thorough clinical history screening for symptoms of IH, such as headaches, pulsatile tinnitus, transient obscurations of vision, and binocular diplopia, and the clinical exam must include a funduscopic examination screening for papilledema. Additional symptoms seen in hemodialysis patients that may indicate venous hypertension, as seen in superior vena cava syndrome, include facial or upper extremity edema, pain, bluish discoloration and pigmentation of the skin, increasing difficulty with access and efficacy of dialysis, and respiratory distress (from laryngeal edema, pleural effusion, or chest swelling).[1, 8, 13, 35] Bilateral ophthalmoplegia and exophthalmos have been reported as a result of retrograde IJV flow in hemodialysis patients with central venous stenosis.[16, 36] Intracranial imaging should be performed, preferably with MRI (+/- MRV) of the head, to exclude structural intracranial causes of IH, such as a mass lesion or cerebral venous sinus thrombosis. Intracranial imaging can also identify other manifestations of IH due to venous hypertension, such as cerebral venous infarction or hemorrhage.[15, 35] Many patients with IH have radiographic signs on MRI, such as a partially empty sella, optic nerve sheath distension, posterior globe flattening, tortuosity of the optic nerves, and transverse venous sinus stenosis.[37] These radiographic signs may be associated with chronic IH, and are not specific to idiopathic intracranial hypertension (IIH). Therefore, in hemodialysis patients with IH (and selected patients with atypical IH), chest vascular imaging is necessary before a diagnosis of IIH can be made.

Central venous imaging options

A CT venogram of the chest is the study of choice for screening for central venous stenosis as it includes the central veins most likely to cause IH when narrowed (superior vena cava, brachiocephalic veins, and the inferior portion of the internal jugular veins).[17] The requisition should include a note to the radiologist protocolling the study requesting special attention to screening for central venous stenosis, as the scan protocols may

vary according to local practice and the timing of contrast administration will need to be optimized for the venous phase. Preference should be given to scheduling the study immediately prior to a hemodialysis session to allow the clearance of contrast dye. Although MRV of the chest with contrast can also be considered, opinions among radiologists vary regarding the use of gadolinium-based contrast dye (GBCD) in patients with advanced renal disease, with or without hemodialysis, due to the risk of nephrogenic systemic fibrosis (NSF).[38] Although there is thought to be a lower risk of NSF with newer GBCDs, MRI is likely not necessary to screen for central venous obstruction in most cases.[39] If a CT of the chest with contrast proves to be inadequate and suspicion remains high for central venous stenosis, catheter-based venography may be pursued. Ultrasonography is ineffective in the detection of most central vein stenosis.[40]

Lumbar puncture with measurement of the opening pressure is also essential to confirm a diagnosis of central venous obstruction-induced IH. Demonstration of elevated opening pressure may exclude other causes of bilateral optic disc edema and is necessary prior to consideration of any intracranial pressure-lowering interventions.

Management and Treatment Options

Management strategies for IH related to central venous obstruction in hemodialysis patients include the possibility of an interventional procedure to relieve the venous obstruction. Options include percutaneous transluminal angioplasty and stent placement. Many central vein lesions are elastic, which may limit improvement in stenosis following percutaneous transluminal angioplasty. Furthermore, many stenoses that do improve following the procedure restenose within months.[41]

Although intracranial pressure lowering medications, such as acetazolamide, may help lower intracranial pressure in patients with central venous obstruction-induced IH, their use should be limited to patients with only mild papilledema without visual dysfunction and without other symptoms who have a high surgical risk. Moreover, the effect of medication may be limited, and acetazolamide may be difficult to effectively dose in hemodialysis patients. Medical management may be most appropriate as a bridge to surgery for more definitive treatment of IH.

Most published cases of IH related to central venous obstruction were treated with AV graft ligation/occlusion or angioplasty +/- stent (22/24 cases, 91.6%), and 20 of those 22 (90.9%) cases experienced resolution of IH (including one with a successfully-treated recurrence)[18] and all 22 had some symptom improvement initially. Although direct treatment of central venous obstruction is ideal, it may be difficult or impossible in some patients, including those with a history of multiple previous endovascular procedures.

Optic nerve sheath fenestration may be considered in patients with severe papilledema threatening visual loss while awaiting more definitive treatment. CSF shunting procedures, such as a ventriculoperitoneal or lumboperitoneal shunt, may be appropriate when endovascular intervention for central venous obstruction may jeopardize currently reliable hemodialysis access or interventions for central venous obstruction are otherwise high-risk or contraindicated.[17] More long-term follow-up data is necessary to assess the long-term efficacy and prognosis of patients undergoing interventional treatment for central venous obstruction-induced IH.

Conclusions

Central venous obstruction-induced IH is likely more common than previously recognized in hemodialysis patients. The presenting signs and symptoms are very similar to IIH. Therefore, before a diagnosis of IIH is made in any hemodialysis patient, venous imaging of the chest must be performed to rule out central venous obstruction. Medical management is less likely to be effective, and clinicians must be familiar with central venous obstruction-induced IH to facilitate appropriate treatment with endovascular intervention for central venous obstruction, arteriovenous shunt ligation, or a CSF diversion procedure. There is significant risk for visual loss or permanent nervous system damage without treatment. Clinicians familiar with the risk factors, presenting signs and symptoms, indications for central venous imaging, and management options will be well-positioned to diagnose and arrange for intervention early in the course of this treatable condition with potential for good neurologic and visual outcomes.

References

1. Agarwal AK, Patel BM, Haddad NJ. Central Vein Stenosis: A Nephrologist's Perspective. Semin Dial. 2007;20(1):53-62.
2. Chen L, Elias G, Yostos MP, Stimec B, Fasel J, Murphy K. Pathways of cerebrospinal fluid outflow: a deeper understanding of resorption. Neuroradiology. 2015;57(2):139-47.
3. Welch K, Friedman V. The Cerebrospinal Fluid Valves. Brain. 1960;83:454-69.
4. Pollay M. The function and structure of the cerebrospinal fluid outflow system. Cerebrospinal Fluid Res. 2010;7:9.
5. Kapoor KG, Katz SE, Grzybowski DM, Lubow M. Cerebrospinal fluid outflow: an evolving perspective. Brain Res Bull. 2008;77(6):327-34.
6. San Millan Ruiz D, Gailloud P, Rufenacht DA, Delavelle J, Henry F, Fasel JH. The Craniocervical Venous System in Relation to Cerebral Venous Drainage. American Journal of Neuroradiology. 2002;23(9):1500-8.
7. Valdueza JM, von Munster T, Hoffman O, Schreiber S, Einhaupl KM. Postural Dependency of the Cerebral Venous Outflow. Lancet. 2000;355(9199):200-1.

8. Dolmatch BL, Gurley JC, Baskin KM, Nikolic B, Lawson JH, Shenoy S, et al. Society of Interventional Radiology Reporting Standards for Thoracic Central Vein Obstruction: Endorsed by the American Society of Diagnostic and Interventional Nephrology (ASDIN), British Society of Interventional Radiology (BSIR), Canadian Interventional Radiology Association (CIRA), Heart Rhythm Society (HRS), Indian Society of Vascular and Interventional Radiology (ISVIR), Vascular Access Society of the Americas (VASA), and Vascular Access Society of Britain and Ireland (VASBI). J Vasc Interv Radiol. 2018;29(4):454-60 e3.
9. MacRae JM, Ahmed A, Johnson N, Levin A, Kiaii M. Central vein stenosis: a common problem in patients on hemodialysis. ASAIO J. 2005;51(1):77-81.
10. 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(6):1542-5.
11. Taban M, Taban M, Lee MS, Smith SD, Heyka R, Kosmorsky GS. Prevalence of optic nerve edema in patients on peripheral hemodialysis. Ophthalmology. 2007;114(8):1580-3.
12. Simon MA, Duffis EJ, Curi MA, Turbin RE, Prestigiacomo CJ, Frohman LP. Papilledema due to a permanent catheter for renal dialysis and an arteriovenous fistula: a "two hit" hypothesis. J Neuroophthalmol. 2014;34(1):29-33.
13. Eames P, Senthil L, Thomas A, Riley P, Burdon MA. Brachiocephalic Vein Stenosis in Association Ipsilateral Hyperdynamic Brachio-Basilic Fistula Causing Ipsilateral Facial Swelling and Contralateral Papillodema and Visual Loss. Interventional Neuroradiology. 2010;16:322-5.
14. Saha MK, Hamieh T, Larkin B, McMillan W. Cerebral hemorrhage due to internal jugular vein stenosis in a hemodialysis patient. Clin Exp Nephrol. 2012;16(2):345-9.
15. Nishijima H, Tomiyama M, Haga R, Ueno T, Miki Y, Arai A, et al. Venous cerebral infarction in a patient with peripheral hemodialysis shunt and occlusion of the left brachiocephalic vein. J Stroke Cerebrovasc Dis. 2011;20(4):381-3.
16. Varelas PN, Bertorini TE, Halford H. Bilateral ophthalmoplegia and exophthalmos complicating central hemodialysis catheter placement. American Journal of Kidney Diseases. 1999;33(5):966-9.
17. Mackay DD, Biousse V. Hemodialysis graft-induced intracranial hypertension Neurol Clin Pract. 2015;5(6):494-7.
18. Herzig DW, Stemer AB, Bell RS, Liu AH, Armonda RA, Bank WO. Neurological sequelae from brachiocephalic vein stenosis. J Neurosurg. 2013;118(5):1058-62.
19. Silva MA, Deen KI, Fernando DJS, Sheriffdeen AH. The internal jugular vein valve may have a significant role in the prevention of venous reflux: evidence from live and cadaveric human subjects. Clin Physiol & Func Im. 2002;22:202-5.
20. Fisher J, Vaghaiwalla F, Tsitlik J, Levin H, Brinker J, Weisfeldt M, et al. Determinants and Clinical Significance of Jugular Venous Valve Competence. Circulation. 1982;65(1):188-96.
21. Vertemati M, Rizzetto F, Cassin S, Zerbi P, Giordano A, Cariati M, et al. Clinical relevance of the left brachiocephalic vein anatomy for vascular access in dialysis patients. Clin Anat. 2019.
22. Kim CH, Kang J, Choi DS, Park JH. Intracranial Venous Reflux Caused by Occlusion of the Brachiocephalic Vein Mimicking Dural Arteriovenous Fistula. World Neurosurg. 2018;120:438-41.
23. Cleper R, Goldenberg-Cohen N, Kornreich L, Krause I, Davidovits M. Neurologic and ophthalmologic complications of vascular access in a hemodialysis patient. Pediatr Nephrol. 2007;22(9):1377-82.
24. Kwon HJ, Lim J, Park B, Koh HS, Choi SW, Kim SH. Intracranial Venous Hypertension Induced by Superior Vena Cava Syndrome Mimicking Cavernous Dural Arteriovenous Fistula. World Neurosurg. 2018;118:265-8.
25. Lal SM, Twardowski ZJ, Stone JV, Keniston D, Scott WJ, Berg GG, et al. Benign Intracranial Hypertension: A Complication of Subclavian Vein Catheterization and Arteriovenous Fistula. Am J Kidney Dis. 1986;8(4):262-4.

26. Hartmann A, Mast H, Stapf C, Koch H, Marx P. Peripheral Hemodialysis Shunt With Intracranial Venous Congestion. Stroke. 2001;32:2945-6.
27. Nishimoto H, Ogasawara K, Miura K, Ohmama S, Kashimura H, Ogawa A. Acute intracranial hypertension due to occlusion of the brachiocephalic vein in a patient undergoing hemodialysis. Cerebrovasc Dis. 2005;20(3):207-8.
28. McQuillan R, Bharatha A, Simons M, Bargman J. Sudden Loss of Consciousness During Hemodialysis. Am J Kidney Dis. 2011;57(5):xxiii-xxvi.
29. Chen CF, Hsu SW, Ko SF, Chen KY. High-flow hemodialysis arteriovenous shunt with concurrent central vein stenosis masquerading as sigmoid sinus dural arteriovenous fistula. Clin Neuroradiol. 2013;23(1):59-62.
30. Cuadra SA, Padberg FT, Turbin RE, Farkas J, Frohman LP. Cerebral venous hypertension and blindness: a reversible complication. J Vasc Surg. 2005;42(4):792-5.
31. Aitken E, Honour P, Johnson N, Kingsmore D. Is there an association between central vein stenosis and line infection in patients with tunnelled central venous catheters (TCVCs)? J Vasc Access. 2015;16 Suppl 9:S42-7.
32. Korzets Z, Zeltzer E, Manor R, Bernheim J. Uremic Optic Neuropathy. A uremic manifestation mandating dialysis. Am J Nephrol. 1998;18(3):240-2.
33. Guy J, Johnston PK, Corbett JJ, Day AL, Glaser JS. Treatment of visual loss in pseudotumor cerebri associated with uremia. Neurology. 1990;40:28-32.
34. Knox DL, Hanneken AM, Hollows FC, Miller NR, Schick Jr. HL, Gonzales WL. Uremic Optic Neuropathy. Arch Ophthalmol. 1988;106(1):50-4.
35. Bakradze E, Zampolin RL, Golowa YS, Bhupali D, Pasquale DD, Liberman AL. Teaching NeuroImages: Facial swelling and intracerebral hemorrhage from venous hypertension in a dialysis patient. Neurology. 2019;92(5):e521-e2.
36. Rotellar C, Gelfand MC, Knepshield JH. Exophthalmos: Rare Complication of A-V Fistula Used for Hemodialysis. Nephron. 1987;45:78.
37. Bidot S, Saindane AM, Peragallo JH, Bruce BB, Newman NJ, Biousse V. Brain Imaging in Idiopathic Intracranial Hypertension. J Neuroophthalmol. 2015;35(4):400-11.
38. Zhang B, Liang L, Chen W, Liang C, Zhang S. An Updated Study to Determine Association between Gadolinium-Based Contrast Agents and Nephrogenic Systemic Fibrosis. PLoS One. 2015;10(6):e0129720.
39. Mathur M, Jones JR, Weinreb JC. Gadolinium Deposition and Nephrogenic Systemic Fibrosis: A Radiologist's Primer. Radiographics. 2020;40(1):153-62.
40. Brown PW. Preoperative radiological assessment for vascular access. Eur J Vasc Endovasc Surg. 2006;31(1):64-9.
41. 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):1177-81.
42. Molina JC, Martinez-Vea A, Riu S, Callizo J, Barbod A, Garcia C, et al. Pseudotumor cerebri: An unusual complication of brachiocephalic vein thrombosis associated with hemodialysis catheters. American Journal of Kidney Diseases. 1998;31(5):e3.1-e3.
43. Chang S, Masaryk TJ, Lee MS. Optic nerve edema: complication of peripheral hemodialysis shunt. Semin Ophthalmol. 2004;19(3-4):88-90.
44. Samaniego EA, Abrams KJ, Dabus G, Starr R, Linfante I. Severe venous congestive encephalopathy secondary to a dialysis arteriovenous graft. J Neurointerv Surg. 2013;5(5):e37.
45. Prasad V, Baghai S, Gandhi D, Moeslein F, Jindal G. Cerebral Infarction due to Central Vein Occlusion in a Hemodialysis Patient. J Neuroimaging. 2015;25(3):494-6.

46. Pereira L, Campos Costa E, Nunes T, Saraiva P, Ferreira J, Cruz P, et al. Dynamics of a haemodynamic headache: A case report and literature review of headache secondary to flow inversion of the internal jugular vein. Cephalalgia. 2016;36(14):1370-8.

Figure Legends

Figure 1. Venous anatomy. The thoracic central veins include the superior vena cava, brachiocephalic veins, subclavian veins, internal jugular veins below the first rib, and azygous vein, and excludes the axillary veins (lateral to the first rib). Obstruction of venous flow through these veins along with increased venous flow from an arteriovenous fistula may lead to retrograde venous flow in the neck and intracranial hypertension.

Table 1: Literature review of IH associated with central venous obstruction in the setting of hemodialysis

Reference	Ophthalmologic and neurologic symptoms	Type of HD access	LP opening pressure (cm H_2O)	Central venous disease	Treatment	Outcome
Lal et al., 1986[25]	Headaches, transient visual obscurations	R arm AV graft	56	R BCV occlusion	Graft ligation	Resolution of IH
Molina et al., 1998[42]	Headaches, transient visual obscurations, progressive vision loss	R arm AV graft	35	Bilateral BCV occlusions	Graft ligation	Improvement in IH without full resolution
Varelas et al., 1999[16]	Diplopia, headaches	R arm AV graft	-	R BCV stenosis	Angioplasty and stent	Resolution of IH
Hartmann et al., 2001[26]	Occipital headaches, gait instability, memory problems	L arm AV graft	-	R SCV occlusion, L BCV stenosis	Graft ligation	Resolution of IH
Chang et al., 2004[43]	Left-sided headaches, pulsatile tinnitus	L brachiobasilic AV fistula	-	L BCV stenosis	Angioplasty	Resolution of IH
Cuadra et al., 2005[30]	Progressive left eye vision loss, encephalopathy, headaches	R brachial artery to IJV graft	37	R axillary vein, SCV, and IJV occlusions	L optic nerve sheath fenestration, graft occlusion	Resolution of IH with unilateral vision recovery
Nishimoto et al., 2005[27]	Headaches, seizures	L arm AV fistula	-	L BCV occlusion	AV fistula ligation	Resolution of IH
Cleper et al., 2007[23]	Right eye vision loss, headaches	L brachiobasilic AV fistula	50	Bilateral BCV occlusions, SVC stenosis	SVC angioplasty, lumboperitoneal shunt, partial AV fistula closure	Resolution of headache; Later developed CVST, seizures, and brain death
Eames et al., 2010[13]	Headaches, right eye vision loss	L brachiobasilic AV fistula	-	L SCV stenosis, L BCV occlusion	Angioplasty and stent	Resolution of IH but persistent vision deficits
McQuillan et al., 2011[28]	Headaches, dizziness, encephalopathy	R IJV catheter	-	R IJV occlusion, L BCV stenosis	Angioplasty	Resolution of IH
Nishijima et al., 2011[15]	Left temporal headaches, seizures, hemiplegia	L forearm AV fistula	-	L BVC occlusion	AV fistula ligation	Resolution of IH
Saha et al., 2012[14]	Headache, encephalopathy	L brachiojugular graft	-	L IJV stenosis, L SCV stenosis	Graft occlusion	Resolution of IH

Reference	Ophthalmologic and neurologic symptoms	Type of HD access	LP opening pressure (cm H_2O)	Central venous disease	Treatment	Outcome
Chen et al., 2013[29]	Dizziness, involuntary movements, seizures, hemiparesis	L radiocephalic AV fistula	-	L BCV stenosis	AV fistula ligation	Resolution of IH
Herzig et al., 2013[18]	Headache, blurred vision, encephalopathy, seizure	L arm AV fistula	35	L BCV occlusion	Acetazolamide, failed recanalization, AV fistula ligation	Resolution of IH
Herzig et al., 2013[18]	Encephalopathy, right arm weakness, myoclonic seizures	AV fistula (location not specified)	-	L BCV stenosis	Angioplasty and stent	Resolution of IH; Recurrent IH from stent occlusion resolved with repeat angioplasty
Samaniengo et al., 2013[44]	Headaches, encephalopathy	R arm AV graft	-	R BCV occlusion	Occlusion of graft	Resolution of IH
Prasad et al., 2014[45]	Encephalopathy, right-sided hypertonia	L arm AV fistula	-	L BCV occlusion	Angioplasty and stent	Resolution of IH
Simon et al., 2014[12]	Headaches, blurred vision, tinnitus	R IJV catheter	30	R SCV and R BCV stenosis	R SCV angioplasty, catheter removal	Resolution of IH; Developed recurrent R SCV stenosis without IH
Mackay and Biousse, 2015[17]	Blurred vision, headaches	R arm AV fistula	38	R SCV stenosis	VP shunt	Resolution of IH
Pereira et al., 2015[46]	Vertigo, left lateropulsion, supine headaches	L brachiocephalic AV fistula	27	L BCV stenosis	AV fistula ligation	Resolution of IH
Kim et al., 2018[22]	Headaches	L arm AV graft	-	L BCV occlusion	Angioplasty and stent	Resolution of IH
Kim et al., 2018[22]	Seizure	L arm AV graft	-	L BCV occlusion	Treatment deferred due to sepsis	Fatal intracranial hemorrhage
Kwon et al., 2018[24]	Headache, right ptosis	R arm AV graft	-	Bilateral BCV stenosis, bilateral SCV occlusion	R BCV angioplasty	Resolution of IH
Bakradze et al., 2019[35]	Headache, aphasia	L arm AV fistula	-	L cephalic vein occlusion	Recanalization of occlusion	Resolution of IH

Abbreviations: AV = arteriovenous; BCV = brachiocephalic vein; CVST = cerebral venous sinus thrombosis; HD = hemodialysis; IH = intracranial hypertension;

Reference	Ophthalmologic and neurologic symptoms	Type of HD access	LP opening pressure (cm H_2O)	Central venous disease	Treatment	Outcome

IJV = internal jugular vein; L = left; LP = lumbar puncture; PTFE = Polytetrafluoroethylene; R = right; SCV = subclavian vein; SVC = superior vena cava; VP = ventriculoperitoneal