Pediatr Nephrol (2007) 22:1377–1382
DOI 10.1007/s00467-007-0491-5

BRIEF REPORT

Neurologic and ophthalmologic complications of vascular access in a hemodialysis patient

Roxana Cleper · Nitza Goldenberg-Cohen · Liora Kornreich · Irit Krause · Miriam Davidovits

Received: 9 January 2007 / Revised: 12 March 2007 / Accepted: 16 March 2007 / Published online: 9 May 2007

Abstract Patients on long-term hemodialysis undergo multiple interventions, including insertion of central catheters and arteriovenous anastomoses for creation of vascular access. The need for high-flow vessels to maintain hemodialysis efficiency leads to wear on the central veins and consequent stenosis and occlusion. In addition to local signs of impaired venous drainage, abnormal venous flow patterns involving the upper chest, face, and central nervous system might develop. We describe the first pediatric case of devastating intracranial hypertension presenting with visual loss in the eye contralateral to a high-flow vascular access in a patient on long-term hemodialysis. The literature on this rare complication of hemodialysis is reviewed.

Keywords Cerebral venous infarction · Hemodialysis · Intracranial hypertension · Papilledema · Vascular access (high-flow) · Visual loss

R. Cleper (✉) · I. Krause · M. Davidovits
Department of Pediatric Nephrology,
Schneider Children's Medical Center of Israel,
Petah Tiqwa 49202, Israel
e-mail: acleper@zahav.net.il

N. Goldenberg-Cohen
Department of Pediatric Ophthalmology,
Schneider Children's Medical Center of Israel,
Petah Tiqwa, Israel

L. Kornreich
Department of Imaging,
Schneider Children's Medical Center of Israel,
Petah Tiqwa, Israel

R. Cleper · N. Goldenberg-Cohen · L. Kornreich · I. Krause · M. Davidovits
Sackler Faculty of Medicine, Tel Aviv University,
Tel Aviv, Israel

Introduction

The optimal treatment at present for children with end-stage renal failure is kidney transplantation. Young patients awaiting a transplant and patients with transplant failure, especially if uremic, are best treated by peritoneal dialysis or, when unfeasible (e.g., loss of peritoneal function due to recurrent episodes of peritonitis or rejection of intraperitoneally transplanted kidneys), by hemodialysis. In hemodialysis in children, the creation of a permanent vascular access can be technically challenging because of the small native vessel size [1].

The guidelines of the Kidney Disease Outcomes Quality Initiative (K/DOQI) stipulate that fewer than 10% of patients be dialyzed through a central catheter and at least 40% via a native arteriovenous fistula. Nevertheless, studies have shown that, in 79% of children starting hemodialysis, a cuffed catheter is used for vascular access [1, 2]. The major cause of catheter failure is venous thrombosis, which can ultimately lead to stenosis or complete occlusion of the vein, even after excision of the central catheter [3, 4]. Rates of central venous stenosis range from 27% to 38% for cuffed and uncuffed central catheters, respectively [5].

The risk of widespread vascular stenosis or complete obliteration arises in patients receiving prolonged chronic hemodialysis [6, 7], which mandates the intensive use of blood vessels for the creation of a new vascular access or for transient insertion of central venous catheters to bridge the maturation of a new access. In this context, patients may acquire superior vena cava (SVC) syndrome, characterized by facial and arm swelling, cyanosis, dyspnea, chest pain, dysphagia, and, sometimes, headache [7]. Indeed, the long-term use of central venous catheters has displaced malignancy (bronchogenic carcinoma in adults and non-Hodgkin lymphoma in children) as the major cause of SVC syndrome [7].

The aim of this report is to describe the first case of intracranial hypertension and blindness in a child on prolonged hemodialysis with multiple vascular interventions. The literature on the neurological and ophthalmologic complications of hemodialysis vascular access is reviewed.

Case report

A 13-year-old girl who had been treated with hemodialysis for 10.5 years complained of sudden loss of vision in her right eye. Past medical history showed that end-stage renal failure had developed at age 8 months secondary to familial hemolytic uremic syndrome (HUS). The parents were unrelated Ashkenazi Jews of Hungarian origin. Another son had died in infancy of the same disease. The patient was started on peritoneal dialysis, but had to be switched to hemodialysis because of peritoneal failure induced by recurrent peritonitis. She underwent cadaveric kidney transplantations at ages 3.5 years, 5 years, and 10 years, which failed within 2 weeks, 10 months, and 1 week, respectively, owing to the recurrence of HUS. Genetic analysis revealed heterozygosity for membrane co-factor 1 mutation.

Efficient hemodialysis was performed (KT/V 1.6, hemoglobin up to 11.6 g%, ferritin 660 ng/ml). Intensive dietary supplementation, together with 8 years' treatment with growth hormone (GH), had resulted in improved but still suboptimal growth. However, despite the prolonged dialysis sessions, only partial phosphate control was achieved, and severe hyperparathyroidism necessitated several courses of calcitriol pulse therapy and temporary discontinuation of GH therapy. At age 11 years, she underwent right tibial osteotomy for the correction of severe angulation of the right lower limb.

Hypertension was treated with three or, intermittently, four drugs with relatively good control (blood pressure 115–125/70 mmHg).

Three vascular accesses had been created during the patient's 10.5 years on hemodialysis. The first, in her left arm, failed within a short time, and the second, in her right arm, failed after multiple interventions for venous outflow stenosis, including stent insertion. Venography performed before creation of the third access revealed occluded left innominate, left and right brachiocephalic trunks and calcified right subclavian stent protruding into a stenosed SVC. The left upper limb veins drained into an anomalous left hemiazygos vein and into the SVC above the stenotic segment. A two-step procedure was performed to create a left brachio-basilic arteriovenous fistula.

Within 2 months, progressive left facial edema was observed, together with mild transient hoarseness and orthopnea, which subsided spontaneously. Findings on otolaryngology examination with filter X-ray of the trachea were consistent with mild laryngitis ("pencil-tip" stenosis). Funduscopic examination demonstrated mild-to-moderate optic disc edema, with mild attenuation of the retinal arteries, considered to be an early stage of papilledema or grade I hypertensive retinopathy, despite relatively satisfactory blood pressure control at the time. There were no physical complaints consistent with intracranial hypertension or neurological abnormalities. Brain computed tomography (CT) scan was normal.

Balloon percutaneous transluminal angioplasty (PTA) successfully dilated the SVC diameter to 7 mm, which was followed by a transient decrease in left facial edema. PTA was repeated periodically with each recurrence of the edema, but the results were less impressive, and, gradually, severe left facial, peri-orbital, and upper chest edema developed, with grossly dilated tortuous venous collaterals. The patient remained asymptomatic.

Repeated funduscopic examinations revealed progressive bilateral papilledema, more pronounced on the right side, and venous congestion without hemorrhage. No changes in visual acuity or in findings on repeated neurological examinations and brain CT scans were observed. However, enlarged blind spots and bilateral nasal defects appeared on the first visual field examination, performed approximately 9 months after the appearance of blurred optic discs.

Three months later, and almost 1 year from the appearance of the blurred discs, the patient presented with sudden blindness in her right eye. Examination revealed no light perception in the right eye, in addition to right optic atrophy and the long-standing papilledema on the left. Brain CT revealed no new findings. Lumbar puncture was performed, and cerebrospinal fluid (CSF) pressure measured more than 500 mm H_2O, with normal CSF content. A lumboperitoneal shunt was inserted, with rapid improvement of the funduscopic findings in the left eye but no change in the vision of the right eye. Elevated intraocular pressure (up to 34 mmHg) was diagnosed in the left eye, and therapy was started. The right eye pressure was within normal limits.

Within 24 h of shunt insertion, the patient began to complain of severe headaches, which required frequent narcotics administration. Brain CT showed "slit-like" ventricles and diffuse brain edema. As acetazolamide is not recommended for intracranial hypertension in patients with end-stage renal failure, treatment with dexamethasone was started. The headaches gradually and partially resolved, but upper back pain appeared.

One month later, after 2 days of weakness and resurging headaches, the patient presented with generalized tonic–clonic seizures and decerebrate posturing. Magnetic resonance (MR) imaging (MRI) and MR venography (MRV) revealed bilateral parieto-occipital venous brain infarction (Fig. 1a) due to superior saggital venous thrombosis

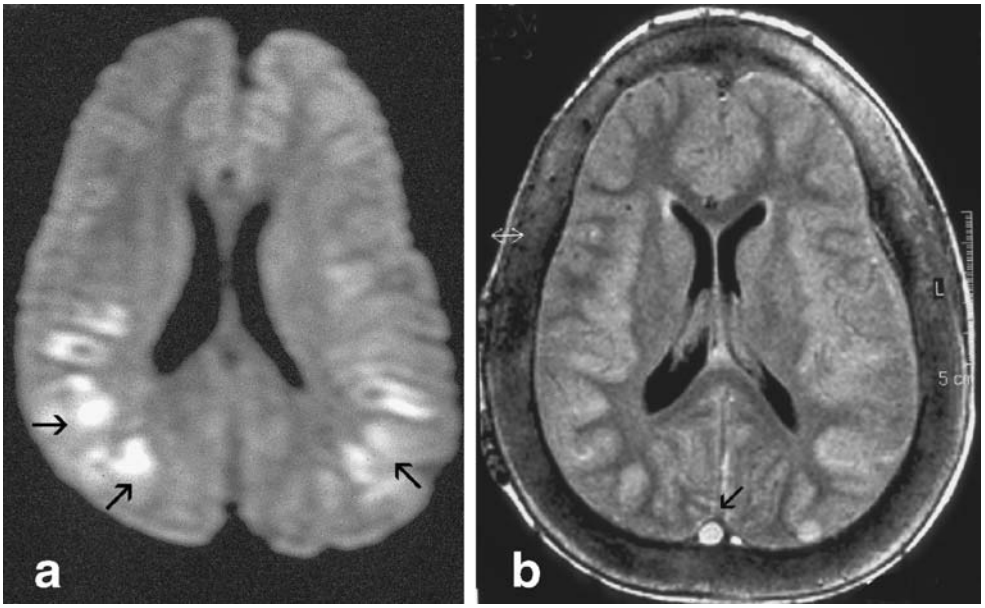

Fig. 1 Brain MRI. **a** Diffusion-weighted MR axial image. Note the bilateral extensive high signals in the parieto-occipital subcortical regions. The location is typical for venous infarcts. **b** Axial T2-weighted MR image. Note the hyperintense signal in the sagittal sinus, compatible with thrombosis (*arrow*)

(Fig. 1b). Anomalous venous drainage of the left arm fistula into the left internal jugular vein was noted, in addition to drainage of the posterior intracranial venous collateral into the right external jugular vein, which, itself, drained into the SVC via multiple small and tortuous collaterals (Fig. 2). There was no significant SVC stenosis.

The anomalous retrograde venous blood flow seemed to be driven by the high flow in the vascular access in the left arm. Therefore, closure of the fistula was mandatory to relieve the intracranial pressure. Meanwhile, until a new vascular access was built in the lower limbs, partial surgical stenosis was induced. One day after partial fistula closure, the patient's neurological status began to improve, and we were able to wean her from the ventilator. Full gross motor function returned, but the patient remained in delirium, with almost no signs of understanding her close environment. However, creation of a temporary access in the lower limbs failed, so continued use of the still-functioning left arm fistula was necessary for hemodialysis. During our planning of a graft to create a new vascular access in the lower limb, the patient experienced a repeated, prolonged convulsion, followed by severe cerebral damage and brain death.

Discussion

Sudden visual loss in the context of optic disc edema in patients with severe chronic renal failure might be caused by several conditions: uncontrolled hypertension with hypertensive retinopathy with or without encephalopathy; advanced uremia with optic neuropathy (similar to uremic peripheral neuropathy or encephalopathy), a condition rarely encountered in patients with accurate nephrological follow-up; anterior ischemic optic neuropathy (AION), mostly described in reduced perfusion states (such as relative hypovolemia and hypotension at the end of hemodialysis sessions or the morning after in patients on night intermittent peritoneal dialysis); and finally, long-standing papilledema secondary to intracranial hypertension

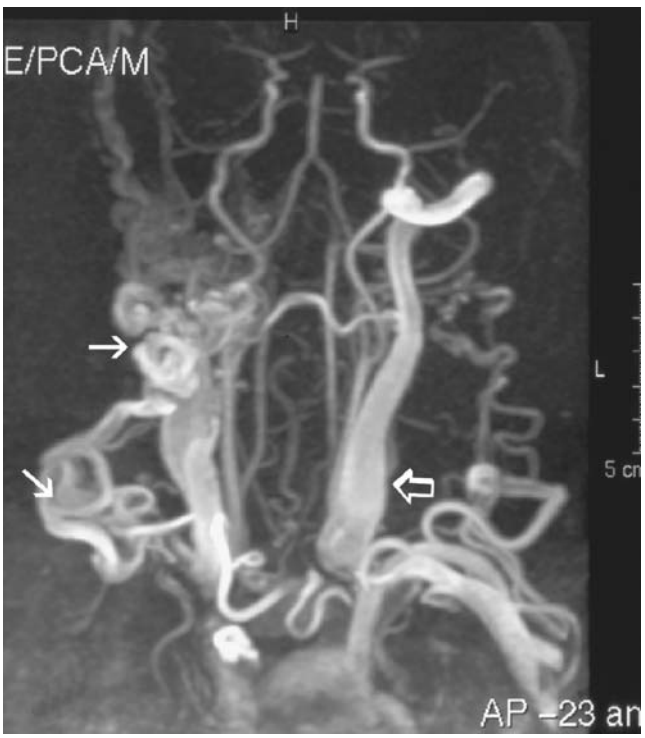

Fig. 2 MR venogram. Note the occlusion in the right subclavian and brachiocephalic veins and the numerous tortuous collateral veins in the neck and scalp, draining into the jugular vessels (*thin arrows*). The internal jugular vein is distended secondary to the increased retrograde flow (*thick arrow*)

[8]. In children, the most common culprit is AION [9–11]. Our patient's blood pressure control (which was satisfactory) and efficient hemodialysis ruled out a diagnosis of hypertensive and uremic retinopathy. Also, the finding of protracted bilateral papilledema was incompatible with the characteristic finding of unilateral swollen disc in AION.

The long-standing optic disc swelling in our patient eventually evolved into full-blown papilledema with severely increased intracranial pressure (>550 mm H_2O). Findings on CSF analysis were within normal range, and brain imaging did not reveal a mass lesion or ventricular enlargement, clearly establishing the diagnosis of intracranial hypertension (pseudotumor cerebri). The initial absence of visual complaints or headache, reported in 80–90% of patients with pseudotumor cerebri, combined with systemic hypertension (albeit not severe enough for high-grade retinopathy), contributed to the delay in measuring CSF pressure and the late diagnosis.

The incidence of intracranial hypertension in the pediatric population is 0.4–2.2 cases per 100,000, or ten-times lower than in adults [12]. The relationship of intracranial hypertension to renal compromise has been attributed to various factors: uremia or dialysis disequilibrium, iron-deficiency anemia, hypo-adrenalism, calcium and water metabolism abnormalities, hypervitaminosis A, and various drugs used in the treatment of chronic renal failure and after renal transplantation (GH, cyclosporine, corticosteroids, nitrofurantoin, minocycline) [13–17]. In the present patient, uremia, high blood pressure, and severe anemia were ruled out. However, GH therapy had been administered for 8 years. Although GH therapy is rarely associated with intracranial hypertension in patients with renal failure [18], and then, mainly within the first 13 weeks of therapy [19], later onset has been reported in some cases. In addition, the severe hyperparathyroidism and the high-dose calcitriol pulse therapy could have produced microscopic arachnoid villi calcification which impaired CSF reabsorption [20].

Nevertheless, the imaging findings of an anomalous retrograde venous flow draining the high-output arteriovenous fistula in the left arm into the ipsilateral internal jugular vein, together with the increased intraocular pressure in the left eye (where venous drainage was impaired), pointed to impaired cerebral venous flow as the probable cause. This assumption was supported by the later development of brain edema with the introduction of a lumbo-peritoneal shunt, in addition to sagittal vein thrombosis with venous cerebral infarction.

Several reports in the literature link increased intracranial hypertension with abnormalities of cerebral venous drainage, mainly SVC obstruction or serious stenosis (SVC syndrome). Most of these cases, however, occurred in the setting of cardiothoracic surgery (acquired hydrocephalus after Mustard operation, switch operation for transposition of great arteries) or extracorporeal membrane oxygenation or intensive care with central venous catheterization [21, 22]. The mechanism, described in early studies in dog models [23], involves the decreased absorption of the cerebral spinal fluid at the level of the arachnoid granulations. Today, cerebral venous outflow obstruction is considered the general pathogenetic mechanism underlying intracranial hypertension [24].

There are only a few reports of intracranial hypertension due to impaired cerebral drainage in patients with renal failure [25–30]. These cases are summarized in Table 1 and involve only adult hemodialysis patients after multiple vascular accesses with "worn out" central veins. The single pediatric case of intracranial hypertension in a patient on chronic hemodialysis was described by Mourani et al. [30], but they provided no vascular details.

SVC syndrome in patients on hemodialysis may be characterized by progressive edema of the face, neck, scalp and upper chest, as well as the arms, owing to the increased flow through internal and lateral thoracic veins, vertebral veins, or azygos, hemi-azygos or innominate veins. In hemodialysis patients with functioning upper-limb access, more specific signs include ipsilateral vascular access malfunction: elevated venous pressure during hemodialysis sessions, prolonged bleeding from puncture sites, and repeated thromboses [31, 32]. Dilatation of alternative drainage veins might produce more remote signs and symptoms, including esophageal varices and dysarthria [33, 34]. In rare situations, when both the innominate and azygos veins are partially or completely occluded bilaterally, retrograde flow into the internal jugular vein, dural sinuses, and the contralateral collaterals develops, adversely affecting cerebral venous drainage. If there is also a high-flow vascular access in an arm, intracranial venous overload ensues.

The most serious complication of high intracranial pressure is visual loss due to optic atrophy, reported in 30–40% of cases. Patients with chronic renal failure seem to be especially susceptible [14]. Intracranial hypertension is usually treated with repeated CSF drainage and administration of acetazolamide and corticosteroids. If these means prove inefficient, lumbo-peritoneal or ventriculo-peritoneal shunts and even optic nerve sheath fenestration may be used for decompression and vision preservation [14, 16, 17].

In the setting of intracranial hypertension secondary to cerebral venous overload due to vascular stenoses and impaired drainage, the main course of action is immediate decongestion of the cerebral venous system by closure of the active vascular access. An alternative vascular access must be secured for continuing renal replacement therapy. Other options include repeated percutaneous transluminal balloon angioplasty with or without stent insertion, venous recon-

Table 1 Review of the literature: neurological and ophthalmological complications of hemodialysis (*HD*) access

Parameter	Lal et al., [25]	Molina et al., [26]	Varelas et al., [27]	Hartman et al., [28]	Cuadra et al., [29]
Gender, Age	M, 62 years	M, 74 years	F, 58 years	F, 59 years	F, 57 years
HD duration	3 years	5 years	10 months	3+2 years	11 years (cumulative)
Vascular access in use	Brachial AVGF[a]	Forearm AVGF[a]	Arm AVGF[a]	Arm AVGF[b]	AVGF (brachial IJV)[a]
Symptoms, signs	Headache, vision loss, papilledema, CSFP: 550	Headache, visual blurring, papilledema, CSFP: 350	Headache, exophthalmus, 6th nerve palsy, retinal venous congestion	Headaches, gait, memory problems, retinal venous congestion, hydrocephalus	Headache, mental decline, visual loss, optic field defect, optic disc edema, high ocular pressure, CSFP: 370
Diagnostic procedure	Brachial fistulogram	Brachial venogram	SVC venography (via access), Neck MRV	Duplex sonography Cerebral angiography	Cerebral angiography MRV
Vascular abnormality	Innom V occluded IJV retrograde flow	BCV occluded IJV retrograde flow	BCV high-grade obstruction IJV retrograde flow	SCV occluded IJV retrograde flow, lateral sinus stasis/thrombosis	AV, SCV and IJV occluded Intracerebral retrograde flow, superior, transverse, sigmoid sinuses flow stagnation
Intervention	Access ligation	Access ligation	BCV PTA+stent	Access ligation	Optic nerve sheath fenestration, AVGF occlusion (manual pressure)
Outcome	Recovery	Stabilized vision, CSFP: 280	Recovery	Recovery Residual slow retrograde IJV flow	Unilateral eye blindness, Neurologically-normal

M male, *F* female, *AVGF* arterio-venous graft fistula, *CSFP* CSF pressure in mm H_2O, *SVC* superior vena cava, *MRV* MR venography, *Innom V* innominate vein, *BCV* brachio-cephalic vein, *AV* axillary vein, *SCV* subclavian vein, *IJV* internal jugular vein, *PTA* percutaneous transluminal angioplasty, *CVC* central venous catheter

[a] Multiple CVCs

[b] Cardiac pacemaker

struction, including a polytetrafluoroethylene axillary to the saphenous graft bypass [35], an atrial graft bypass [36], a switch to a lower-limb vascular access, or peritoneal dialysis.

Before creating a new vascular access, the clinician must clearly identify the existing venous circulation. This may be done by spinal CT, venography, MRI, MRV, MR angiography, and color Doppler ultrasonography. The latter is especially useful in demonstrating the abnormal patterns of cerebral circulation and the consequences of closure of a high-flow access [37].

Conclusions

Patients receiving prolonged hemodialysis treatment are prone to extensive vascular abnormalities. The evaluation of increased intracranial pressure is required in those with an active vascular access and central venous stenosis or obstruction, as evidenced clinically by head, face, neck, chest or upper limb edema, intermittent headache or visual complaints (active enquiry has to be pursued), unexplained dysarthria, dysphagia, hoarseness, orthopnea, or back or chest pain, especially in the context of repeated vascular interventions (accesses, surgery, and central catheter insertion). Complete ocular examination, funduscopic examination, and visual field assessment might reveal optic disc swelling and high intraocular pressure—both significant parameters of early intracranial hypertension. CSF pressure determination is obviously imperative in hemodialysis patients with ophthalmological abnormalities. Brain MRI and MRV are indicated for evaluation of the abnormal intracranial circulation and planning of the appropriate intervention to prevent visual loss and a catastrophic neurological outcome.

References

1. Cass DL, Nuchtern JG (2004) Vascular access. In: Warady BA, Schaefer FS, Fine RN, Alexander SR (eds) Pediatric dialysis. Kluwer Academic Publishers, Dordrecht Boston London, pp 65–81
2. Fishbach M, Edefonti A, Schroder C, Watson A, The European Pediatric Dialysis Working Group (2005) Hemodialysis in children: general practical guidelines. Pediatr Nephrol 20:1054–1066
3. Oguzkurt L, Tercan F, Torun D, Yildirim T, Zumrutdal A, Kizilkilic O (2004) Impact of short-term hemodialysis catheters on the central veins: a catheter venographic study. Eur J Radiol 52:293–299
4. Goldstein SL, Macierowski CT, Jabs K (1997) Hemodialysis catheter survival and complications in children and adolescents. Pediatr Nephrol 1:74–77
5. Consalves CF, Eschelman DJ, Sullivan KL, DuBois N, Bonn J (2003) Incidence of central vein thrombosis and occlusion following upper extremity PICC and port placement. Cardiovasc Intervent Radiol 26:123–127
6. Chemla ES, Korrakuti L, Makanjuola D, Chang RW (2005) Vascular access in hemodialysis patients with central venous obstruction or stenosis: one center's experience. Ann Vasc Surg 19:692–698
7. Yim CD, Sane SS, Bjarnason H (2000) Superior vena cava stenting. Radiol Clin North Am 38:409–424
8. Winkelmayer WC, Eigner M, Berger O, Grisold W, Leithner C (2001) Optic neuropathy in uremia: an interdisciplinary emergency. Am J Kidney Dis 37:E23
9. Lapeyraque AL, Haddad E, Andre JL, Bremond Gignac D, Taylor CM, Rianthavom P, Salusky IB, Loirat C (2003) Sudden blindness caused by anterior ischemic optic neuropathy in 5 children on continuous peritoneal dialysis. Am J Kidney Dis 42:E19
10. Chutorian AM, Winterkorn JMS, Geffner M (2002) Anterior ischemic optic neuropathy in children: case reports and review of the literature. Pediatr Neurol 26:358–364
11. Kim JS, Deputy S, Tere Vives M, Aviles DH (2004) Sudden blindness in a child with end-stage renal disease. Pediatr Nephrol 19:691–693
12. Kessler A, Fattal-Valevski A (2002) Idiopathic intracranial hypertension in the pediatric population. J Child Neurol 17:745–748
13. Chang D, Nagamoto G, Smith W (1992) Benign intracranial hypertension and chronic renal failure. Cleve Clin J Med 59:419–422
14. Guy J, Johnston PK, Corbett JJ, Day AL, Glase JS (1990) Treatment of visual loss in pseudotumor cerebri associated with uremia. Neurology 40:28–32
15. Belson A, Alcorn DM, Yorgin PD, Fisher PG, Sarwal M (2001) Visual loss caused by pseudotumor cerebri in an infant on peritoneal dialysis. Pediatr Nephrol 16:216–218
16. Francis PJ, Haywood S, Rigden S, Calver DM, Clark G (2003) Benign intracranial hypertension in children following renal transplantation. Pediatr Nephrol 18:1265–1269
17. Chamberlain CE, Fitzgibbon E, Wassermann EM, Butman JA, Kettl D, Hale D, Kirk AD, Mannon RB (2005) Idiopathic intracranial hypertension following kidney transplantation: a case report and review of the literature. Pediatr Transplant 9:545–550
18. Fine RN, Ho M, Tejani A, Blethen S (2003) Adverse events with rhGH treatment of patients with chronic renal insufficiency and end-stage renal disease. J Pediatr 142:539–545
19. Koller EA, Stadel BV, Malozowski SN (1997) Papilledema in 15 renally compromised patients treated with growth hormone. Pediatr Nephrol 11:451–454
20. Ozveren MF, Kaplan M, Topsakal C, Bilge T, Erol FS, Celiker H, Akdemir I, Uchida K (2001) Spontaneous cerebrospinal fluid rhinorrhea associated with chronic renal failure. Neurol Med Chir 41:313–317
21. Karmazyn B, Dagan O, Vidne BA, Horev G, Kornreich L (2002) Neuroimaging findings in neonates and infants from superior vena cava obstruction after cardiac operation. Pediatr Radiol 32:806–810
22. McLaughlin JF, Loeser JD, Roberts TS (1997) Acquired hydrocephalus associated with superior vena cava syndrome in infants. Childs Nerv Syst 13:59–63
23. Bering EA, Salibi B (1959) Production of hydrocephalus by increased cephalic-venous pressure. AMA Arch Neurol Psychiatry 81:693–698
24. Karahalios DG, Rekate HL, Khayata MH, Apostolides PJ (1996) Elevated intracranial venous pressure as a universal mechanism in pseudotumor cerebri of varying etiologies. Neurology 46:198–202
25. Lal SM, Twardowski AJ, Van Stone J, Kenistone D, Scott WJ, Berg GG (1986) Benign intracranial hypertension: a complication of subclavian vein catheterization and arteriovenous fistula. Am J Kidney Dis 8:262–264
26. Molina JC, Martinez-Vea A, Riu S, Callizo J, Barbod A, Garcia C, Peralta C, Oliver JA (1998) Pseudotumor cerebri: an unusual complication of brachiocephalic vein thrombosis associated with hemodialysis catheters. Am J Kidney Dis 31:E3
27. Varelas PN, Bertorini TE, Halford HH (1999) Bilateral ophthalmoplegia and exophthalmos complicating central hemodialysis catheter placement. Am J Kidney Dis 33:966–969
28. Hartman A, Mast H, Stapf C, Koch HC, Marx P (2001) Peripheral hemodialysis shunt with intracranial venous congestion. Stroke 32:2945–2946
29. Cuadra SA, Padberg FT, Turbin RE, Farkas J, Frohman JP (2005) Cerebral venous hypertension and blindness: a reversible complication. J Vasc Surg 42:792–795
30. Mourani CC, Mallat SG, Moukarzel MY, Akatcherian CY, Cochat P (1998) Kidney transplantation after a severe form of pseudotumor cerebri. Pediatr Nephrol 12:709–711
31. Bornak A, Wicky S, Ris HB, Probst H, Milesi I, Corpataux JM (2003) Endovascular treatment of stenoses in the superior vena cava syndrome caused by non-tumoral lesions. Eur Radiol 13:950–956
32. Madan AK, Allmon JC, Cheng SS, Slakey DP, Harding M (2002) Dialysis access-induced superior vena cava syndrome. Am Surg 68:904–907
33. Hsu YH, Yang MT, Hsia CC, Tsai DM (2004) Esophageal varices as a rare complication of central venous dialysis tunneled cuffed catheter. Am J Kidney Dis 43:E7
34. Tintillier M, de Suray N, Alexis F, Mathy L, Rombaut E (2004) Systemic-to-pulmonary venous shunt in a hemodialysis patient. Nephrol Dial Transplant 19:2673
35. Kavallieratos N, Kokkinos A, Kalocheretis P (2004) Axillary to saphenous vein bypass for treatment of central venous obstruction in patients receiving dialysis. J Vasc Surg 40:640–643
36. Auarragaray JEF (2003) Surgical treatment of hemodialysis-related central venous stenosis or occlusion: another option to maintain vascular access. J Vasc Surg 37:1043–1046
37. Cockbayir I, Men S, Yanik B, Hekimoglu B (2002) Color Doppler sonographic finding of retrograde jugular venous flow as a sign of innominate vein occlusion. J Clin Ultrasound 30:392–398