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Case Report

Cerebral hemorrhage due to intracranial venous reflux associated with left brachiocephalic vein occlusion in a hemodialysis patient

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ABSTRACT

Background: Although central venous occlusion is sometimes seen in hemodialysis (HD) patients, neurological symptoms due to intracranial venous reflux (IVR) are extremely rare.

Case Description: We present a case of a 73-year-old woman with cerebral hemorrhage due to IVR associated with HD. She presented with lightheadedness and alexia, and was diagnosed with subcortical hemorrhage. Venography through the arteriovenous graft showed occlusion of the left brachiocephalic vein (BCV) and IVR through the internal jugular vein (IJV). It is extremely rare that IVR occurs and causes neurological symptoms. This is because that there is the presence of a valve in the IJV and the communication between the right and left veins through the anterior jugular vein and thyroid vein. Percutaneous transluminal angioplasty for the left obstructive BCV was performed, but the obstructive lesion was only slightly improved. Hence, shunt ligation was performed.

Conclusion: When IVR is found in HD patients, central veins should be confirmed. Early diagnosis and therapeutic intervention are desirable when neurological symptoms are present.

Keywords: Brachiocephalic vein, Central vein stenosis, Hemodialysis, Intracranial venous reflux

INTRODUCTION

Although central venous stenosis (CVS) or occlusion is sometimes seen in hemodialysis (HD) patients, neurological symptoms due to intracranial venous reflux (IVR) are very rare. Chronic intracranial venous stasis can cause venous infarction and hemorrhage, so immediate intervention is required when IVR is suspected. We present a case of cerebral hemorrhage due to IVR associated with the left brachiocephalic vein (BCV) occlusion in a HD patient.

CASE PRESENTATION

A 73-year-old woman suffering from end-stage renal disease due to glomerulonephritis and receiving HD through an arteriovenous fistula (AVF) in the left upper arm for 4 years presented with lightheadedness and alexia. Computed tomography showed a high-density area in the left occipital lobe, which was hypointense on T2 star weighted image of magnetic resonance imaging

(MRI), and was diagnosed with subcortical hemorrhage [Figures 1a and b]. T2-weighted image showed flow voids in the cerebellar hemispheres, left occipital lobe, left parietal lobe, and around the brainstem [Figure 1c]. Magnetic resonance angiography (MRA) showed abnormal signals in internal jugular vein (IJV), sigmoid sinus, transverse sinus, inferior petrosal sinus, anterior condylar vein, and cavernous sinus such as dural AVF(dAVF) [Figure 1d]. Head digital subtraction angiography (DSA) and venography through the arteriovenous graft were performed. DSA showed that the blood flow in the left cerebral hemisphere was mainly perfused through the right transverse sinus contralaterally [Figure 2a], but partially refluxed into cortical veins such as the vein of Trolard, leading to venous stasis and venous perfusion failure [Figures 2b and c]. Shunt points on the dura were not observed. Venography showed occlusion of the left BCV and IVR through the IJV [Figure 3a]. The development of the subcutaneous veins, which serve as collateral blood circulation to the contralateral side of the anterior neck and chest, was poor, and ectasia of the subcutaneous veins in the left upper arm and neck was observed. Based on these findings, we diagnosed subcortical hemorrhage due to IVR associated with the left BCV occlusion.

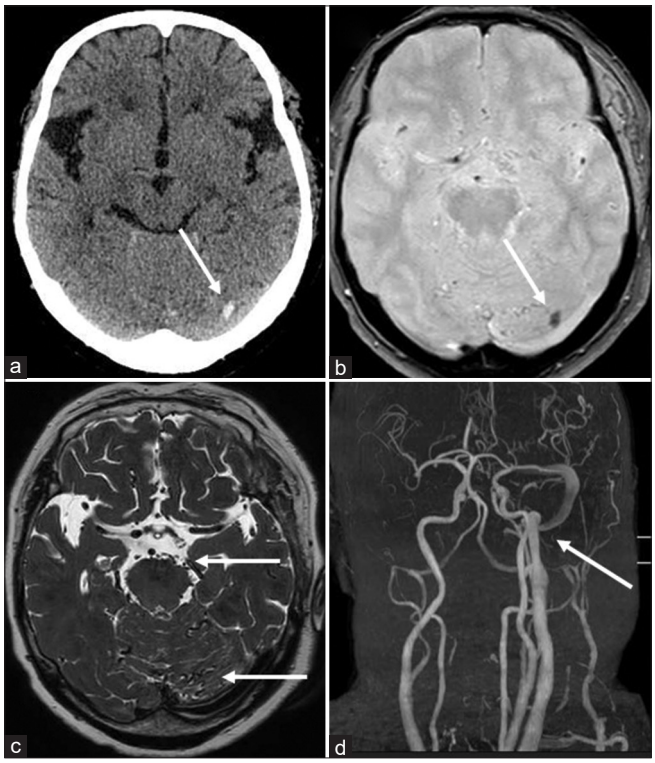

Figure 1: Imaging findings on admission (a and b) CT shows a slight high-density area in the left occipital lobe (arrow), which is hypointense on T2 star weighted image of MRI (arrow). (c) T2-weighted image of MRI shows flow voids around the cerebellum and brainstem (arrows). (d) MRA clearly visualizes the IJV and sigmoid sinus (arrow), and venous reflux into the anterior condylar vein and cavernous sinus can also be confirmed. CT: Computed tomography, MRI: Magnetic resonance imaging, MRA: Magnetic resonance angiography, IJV: Internal jugular vein.

Percutaneous transluminal angioplasty (PTA) for the left obstructive BCV was performed, but the obstructive lesion was only slightly improved [Figures 3b and c]. Three weeks later, chemosis developed, so shunt ligation was performed. The chemosis disappeared the day after the operation and subcutaneous edema of the neck and the left upper arm also improved. MRI on the day after surgery showed complete disappearance of venous reflux [Figure 4]. Since then, neurological symptoms have not recurred.

DISCUSSION

It is said that HD patients sometimes develop CVS. The causes of CVS are thought to be intimal hyperplasia due to chronic elevation of venous pressure, vasoconstriction, platelet aggregation, changes of shear stress and blood flow velocity, and intimal thickening due to oxidative stress.[1,5] Mechanical irritation from the previous central venous catheterization is also a risk factor of CVS.[1] In recent reports, CVS is defined as stenosis of at least 50% of the central thoracic veins, including inferior and superior vena cava, BCV, subclavian vein, and IJV.[2] Although CVS is said to occur in 16–50% of HD patients, it is extremely rare that IVR occurs and causes neurological symptoms.[3] This is because that there is the presence of a valve in the IJV and the communication between the right and left veins via the anterior jugular vein and thyroid vein. However, chronic venous hypertension has been reported to cause jugular valve dysfunction.[3] In addition, there are individual differences in the development of the right and left collateral blood circulation. These factors appear to cause IVR in CVS patients.

To the best of our knowledge, 23 cases of neurological symptoms due to IVR have been reported, but only three of them presented with cerebral hemorrhage.[7] In many cases, there is no organic change in the brain parenchyma. The most frequent symptom is headache, followed by motor deficits, and no case of alexia has been reported.[7] In our case, alexia was caused by inadequate venous perfusion in the left parietal lobe, and theoretically, various neurological symptoms can occur depending on the site of venous stasis. Furthermore, it has been reported that compression of the left BCV by the sternum and aortic arch causes cerebral venous reflux in 1.6% of healthy individuals.[4] The characteristics of IVR in HD patients are similar to those in healthy individuals, such as venous reflux in the left side. However, among patients with IVR, HD patients appear to be about 10 years younger than healthy individuals.[4,7]

Although it is relatively easy to detect abnormal findings from the visualization of the venous sinuses by MRA, it is difficult to distinguish them from dAVF by MRI alone.

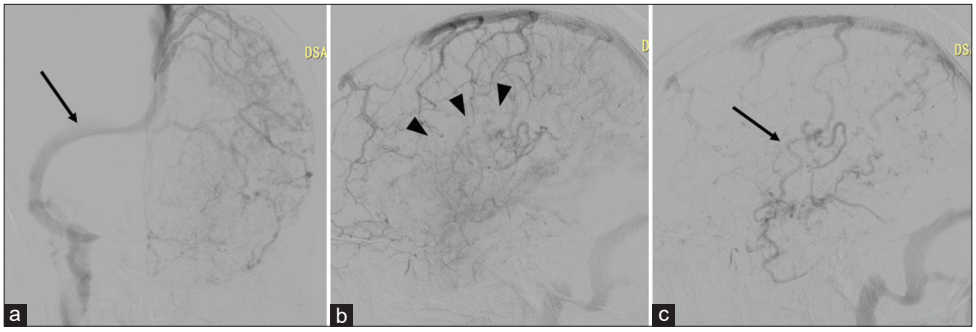

Figure 2: Left common carotid angiography (a) Venous perfusion of the left cerebral hemisphere uses the right transverse sinus (arrow), and the left transverse sinus is not visualized. (b and c) The venous blood flow of the left cerebral hemisphere reflux into cortical veins (arrow). Venous stasis and venous perfusion failure are detected (arrowheads).

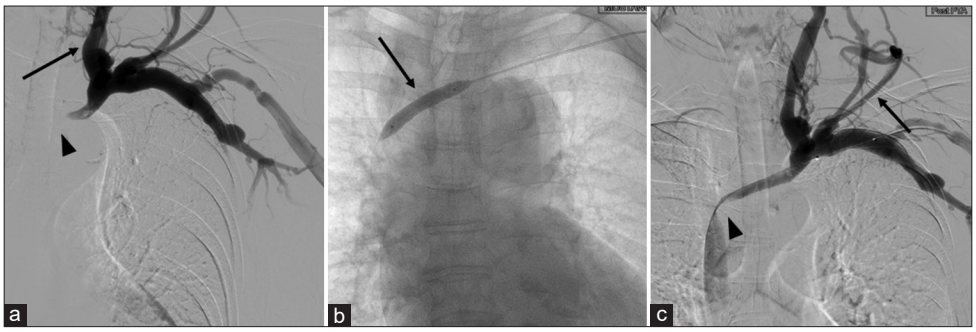

Figure 3: Venography through the arteriovenous graft (a) The left BCV is occluded (arrowhead) and veins flow back into the IJV (arrow), (b) Undergoing PTA for the left obstructive BCV (arrow), (c) The stenosis of the left BCV is only slightly improved (arrowhead). But reflux into the IJV remains and the ectasia of the subcutaneous veins in the left upper arm and neck is observed (arrow). BCV: Brachiocephalic vein, IJV: Internal jugular vein, PTA: Percutaneous transluminal angioplasty.

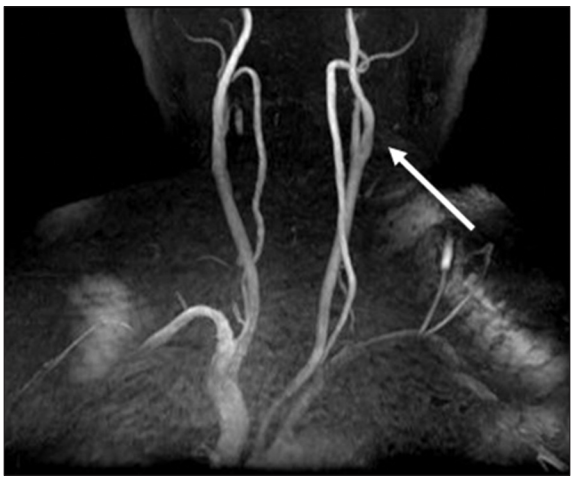

Figure 4: MRI on the day after shunt LIGATION Abnormal signals in the IJV completely disappear (arrow). MRI: Magnetic resonance imaging, IJV: Internal jugular vein.

Angiography is the gold standard for definitive diagnosis. In cerebral angiography, it is important to confirm the denial of dural shunt in the arterial phase, venous stasis in the venous phase, and the use of alternative routes such as the right transverse sinus. Furthermore, it is also necessary to confirm the stenosis or occlusion of central vein, findings of IVR, and the development of collateral circulation, by venography through the arteriovenous graft. When there are abnormal findings of the venous sinuses in HD patients on head MRA, it is important to suspect CVS and perform not only cerebral angiography but also venography through the arteriovenous graft.

According to the National Kidney Foundation Guideline, PTA is the first choice for the treatment of symptomatic CVS in HD patients, and shunt ligation is the second choice.[6] In our case, PTA did not sufficiently improve the stenosis. As a result, recurrence and exacerbation of IVR were observed, so shunt ligation was performed. PTA for incidentally discovered asymptomatic CVS is not generally recommended because the manipulation might accelerate stenotic deterioration. PTA is often selected for symptomatic CVS with swelling of the arm or face because it is minimally invasive, but the high rate of reocclusion is a problem. Shunt ligation is often selected when repeat PTA is required. There

is no therapeutic answer for CVS with IVR. Even if sufficient dilatation is obtained with a balloon, there is a possibility of recurrence in the long-term. Although shunt ligation can be a definitive solution, alternative methods of dialysis must be secured. However, in patients with IVR, if sufficient dilation cannot be obtained with PTA such as our case, early recoil and exacerbation may occur. Therefore, there is a high possibility that shunt ligation must be performed without waiting.

CONCLUSION

When IVR is found in HD patients, central veins should be confirmed. Early diagnosis and therapeutic intervention are desirable when neurological symptoms are present.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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