Peripheral Hemodialysis Shunt With Intracranial Venous Congestion

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Background—Intracranial venous congestion is often caused by local venous thrombosis or brain arteriovenous fistulas. Hemodialysis shunts are known to cause venous enlargement in the arm or chest but have not been related to intracranial vascular pathology.

Case Description—A 59-year-old woman who presented with increasing headache, gait instability, and memory loss was a renal transplant recipient who still carried a left upper arm shunt. Cranial CT scan showed enlarged veins in the posterior fossa with incipient hydrocephalus. Extracranial duplex sonography revealed reversed flow in the left internal jugular vein, which normalized on cuff inflation around the shunt-carrying arm. The reversed flow, intracranial venous congestion, and neurological status improved after surgical shunt ligation.

Conclusions—To our knowledge, this is the first case description of an intracranial venous outflow obstruction caused by a peripheral arteriovenous shunt. (Stroke. 2001;32:2945-2946.)

Key Words: arteriovenous shunt, surgical □ intracranial thrombosis

Enlargement of the veins in the posterior fossa can occur in venous thrombosis and intracranial arteriovenous fistulas. It has also been observed in tumors with mass effect and in children with vein of Galen aneurysms. Peripheral venous enlargement and thrombosis have been described as complicating large extracranial arteriovenous shunts placed for hemodialysis. We describe a rare case of intracranial venous complications directly related to a peripheral extracranial shunt.

Case Description

A 59-year-old woman presented to the emergency department of our hospital with increasing occipital headaches for the past 3 days. Her medical history was remarkable for renal failure since 1980 due to chronic abuse of analgesics. She had received hemodialysis during 1983–1986 with the use of a polytetrafluoroethylene (PTFE) left upper arm arteriovenous fistula and subsequently underwent renal transplantation in 1986. Hemodialysis was resumed after transplant rejection in 1988 and was discontinued again after successful second transplantation in 1990. A phlebogram before the surgical shunt placement had been normal on the left side but showed occlusion of the right subclavian vein. This was attributed to the patient’s cardiac pacemaker, which was implanted for sick sinus syndrome in 1983 and replaced in 1993.

On admission the patient’s neurological examination was normal. Systemic blood pressure was 140/80 mm Hg and remained normal on the left side of chest and neck were noted (Figure 1). Electroencephalogram was normal, ECG showed continuous pacemaker activity, chest x-ray was unremarkable with pacemaker in place, and blood chemistry revealed slightly elevated serum creatinine of 120 μmol/L. Contrast-enhanced cranial CT showed enlarged veins in the posterior fossa and mild internal hydrocephalus, suggestive of an intracranial arteriovenous malformation. MRI could not be performed because of the pacemaker, and the patient refused conventional angiography. She left the hospital against medical advice but was readmitted 10 days later with increasing headache. On examination she showed pronounced gait and stance instability and moderate memory loss. There was no orthostatic influence on the symptoms. Follow-up cranial CT (Figure 2) revealed increased hydrocephalus and unchanged prominent vessels in relation to the initial scan. Cerebral angiography demonstrated venous congestion in the posterior fossa and stasis or thrombosis in the left lateral sinus with drainage only toward the right lateral sinus and jugular vein. There was no evidence of arteriovenous shunting. Duplex sonography of the neck vessels revealed retrograde flow, ie, toward the brain, in the left internal jugular vein (Figure 3). In addition, high-grade stenosis of the left brachiocephalic vein was suggested. Venous collaterals were observed along the neck and lower mandibular region. On cuff inflation around the left, shunt-carrying arm above systolic blood pressure, blood flow in the jugular vein reversed to normal flow direction. After surgical ligation of the PTFE shunt, blood flow in the left jugular vein was...
still reversed but with very low flow velocity. On early follow-up cranial CT, signs of hydrocephalus had resolved after 1 week, and the clinical status returned to normal. On long-term follow-up more than 1 year after admission, the patient’s neurological status was normal. She was independent in her activities of daily living, and headache did not recur. A duplex scan performed with the patient in the supine position still revealed slow retrograde flow in the proximal left jugular vein, but orthograde flow was depicted distally in the submandibular region. The retrograde flow was distributed to the facial and neck veins, creating an extracranial watershed that left the intracranial venous drainage unaffected.

**Discussion**

In this patient the combination of peripheral high-flow arteriovenous shunt and central venous stenosis led to collateral venous flow along superficial veins of chest and neck and also to a collateral pathway involving the brain, mimicking an intracranial arteriovenous malformation. The age of the patient argued against the midline venous ectasia sometimes found in children. Conventional angiography failed to reveal other vascular malformations. Among those, dural arteriovenous malformations are the most common in the posterior fossa. The suggestion of a lateral sinus thrombosis initiated the screening for extracranial venous thrombosis. The direct relation of the reversed jugular flow to the hemodialysis shunt was proven by its reversibility on cuff inflation around the shunt-carrying arm, which temporarily interrupted the arterial flow into the venous system. The craniopetal detour of blood flow was further aided by the central venous stenosis found by duplex sonography.

Central venous stenosis in hemodialysis with PTFE grafts has been described in up to 29% of cases, often leading to arm edema rendering the affected limb unavailable for further vascular access. Often associated with prior subclavian cannulation, development of venous stenosis is inferred to result from high-pressure turbulent flow caused by the arteriovenous fistula. Given the large number of patients carrying hemodialysis shunts and the considerable frequency of associated central venous stenoses, it is surprising that syndromes such as that in our patient are not reported more often. The detection rate of such constellations may increase with growing awareness of neurovascular problems in the presence of peripheral arteriovenous shunts. Screening for extraneurological venous disease expedited the diagnosis in our case and is recommended when intracranial thrombosis is suggested.

**References**

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