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Aqueductal stenosis caused by an atypical course of a deep collector vein draining bilateral cerebellar developmental venous anomalies

Sir,

We report a 31-year-old woman who presented with intermittent mild occipital headache for six months without focal neurological deficits or papilledema. Mild hydrocephalus involving lateral and third ventricles with prominent aqueductal flow void was noted. Contrast-enhanced MRI revealed large developmental venous anomalies (DVAs) in both cerebellar hemispheres draining into a prominent collector vein in the fourth ventricle [Figure 1]. Axial and sagittal T1 contrast-enhanced images demonstrated the subependymal collector vein coursing upwards through the aqueduct to join the vein of Galen [Figure 2A,B]. Cardiac-gated cine phase contrast MRI revealed absence of CSF flow in the aqueduct. A coexistent pontine cavernous angioma was also noted.

DVAs are common intracranial vascular malformations that are thought to represent primary dysplasias of the capillaries and small transcerebral veins or a compensatory mechanism of an intrauterine accident that caused thrombosis of the normal venous pathways. It follows that these veins, although abnormal, are often the only venous drainage available and thrombosis or surgical excision of the draining vein have resulted in infarction.

The typical DVA consists of multiple radially arranged abnormal veins that converge and drain into superficial

Figure 1: Contrast-enhanced axial T1-weighted MR image (460/15 [repetition time/echo time]) shows bilateral cerebellar developmental venous anomalies (arrowheads)
or deep collector veins forming the ‘caput medusae’. The superficial collectors drain into a venous sinus or cortical vein. The deep collectors join subependymal veins.

Supratentorially, the deep collectors drain to the vein of Galen via the subependymal veins of the lateral ventricles. Infratentorially, the deep collectors drain into the subependymal veins of the fourth ventricle. Further drainage may be to through the anterior or lateral transpontine veins or the veins of the lateral recess of the fourth ventricle. Superior drainage is to the precentral cerebellar vein and thence to the Galenic system.

In this patient, the cerebellar DVAs drained into a subependymal vein in the fourth ventricle. Further drainage was atypical, through a subependymal vein that coursed through the aqueduct of Sylvius to enter the vein of Galen causing obstruction to cerebrospinal fluid (CSF) flow.

Venous angiomas have been very rarely associated with CSF obstruction. Aqueductal stenosis caused by the midbrain DVAs and unilateral foramen of Monro have been described. This case illustrates the fact that draining deep collector veins of cerebellar DVAs can follow atypical drainage patterns and cause obstruction at the aqueduct of Sylvius.

As this condition is an obstructed hydrocephalus, an endoscopic third ventriculostomy is preferable over shunting as and when the symptoms deteriorate or the ventricle size increases.

Srinivasaraman Govindarajan, Lakshmi Narasimhan Ranganathan
Barnard Institute of Radiology, Madras Medical College, 1Department of Neurology, Stanley Medical College, Chennai, Tamil Nadu, India.
E-mail: srinivasaraman@rediffmail.com

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Srinivasaraman Govindarajan, Lakshmi Narasimhan Ranganathan
Barnard Institute of Radiology, Madras Medical College, 1Department of Neurology, Stanley Medical College, Chennai, Tamil Nadu, India.
E-mail: srinivasaraman@rediffmail.com

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