Neuroimaging Highlight

*Editors: Richard Farb, David Pelz*

Recurrent Thalamic Haemorrhage Attributed to a Cerebellar Arteriovenous Malformation

Submitted by: R. Sparing, T. Krings, M. Dafotakis, J. Noth, A. Thron

A 55-year-old man presented with acute-onset right-sided hemiparesis. Cranial CT on admission revealed an intracranial haemorrhage in the left thalamus and parts of the internal capsule (Figure 1A). The patient had no history of arterial hypertension, bleeding diathesis or tobacco or alcohol abuse, and did not receive any anti-platelet agent or anticoagulant. Four weeks later cranial magnetic resonance imaging (MRI) and MR angiography (0.5T, gadolinium-enhanced T1-SE, T2-TSE, FLAIR, 3D-dynamic contrast-enhanced angiography) showed normal blood resorption, with no signs of a vascular disorder or neoplasm. The patient was discharged to a rehabilitation centre. Six months later he was re-admitted after the acute onset of headache followed by left-sided hemiparesis and dysarthria. The CT scan showed an intracranial haemorrhage in the right thalamus, with ventricular rupture (Figure 1B). Following six months of rehabilitation, a second MRI scan (1.5T, gadolinium-enhanced T1-SE, T2-TSE, T2-FFE, FLAIR) failed again to demonstrate a cause for the bilateral thalamic lesions. Digital subtraction angiography (DSA), however, revealed a small arteriovenous malformation (AVM) located superficially in the lower third of the superior vermis (Figures 2A and B). Arterial blood supply was derived from vermian branches of the left posterior inferior cerebellar artery. The venous phase showed AVM drainage to the transverse sinus via the superior vermian veins to the basal vein of Rosenthal, draining to the vein of Galen and the straight sinus (Figure 2C and D). Two years following radiosurgery, DSA confirmed complete occlusion of the AVM and normal venous drainage pattern. On long-term follow-up more than six years after treatment, the patient's neurological examination revealed a mild spastic tetraparesis and moderate neuropsychological deficits. There were no recurrent episodes of bleeding reported.

Since no other cause for the recurrent bleedings was found, we presume that the change of local haemodynamics, especially the increased venous pressure, within the common drainage pathway of the thalamic veins and the remotely located
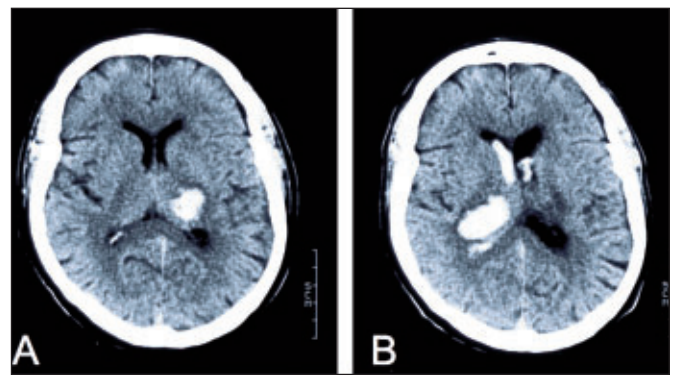

Figure 1: A: First CT scan revealing haemorrhage in the left thalamus and parts of the internal capsule. **B:** The CT scan six months later showed haemorrhage in the right thalamus and parts of the internal capsule with rupture into the ventricle.

cerebellar AVM were causative for the bleedings. A review of other cases reporting cerebral haemorrhages or ischaemia in areas remote from the site of the pathological AV-shunting[1-7] led us to hypothesize that the venous drainage of the cerebellar AVM into the superior vermian veins had probably affected the venous flow of the vein of Galen and the straight sinus. This might have resulted in a change of venous haemodynamics, increased retrograde venous pressure and venous rupture causing consecutively the recurrent haemorrhages in both thalami, which are drained by the same venous system. Venous hypertension and congestion have been reported to play a major role for the occurrence of haemorrhages in distant brain areas.[2,5,6] Intraoperative measurements of intravascular pressure demon-
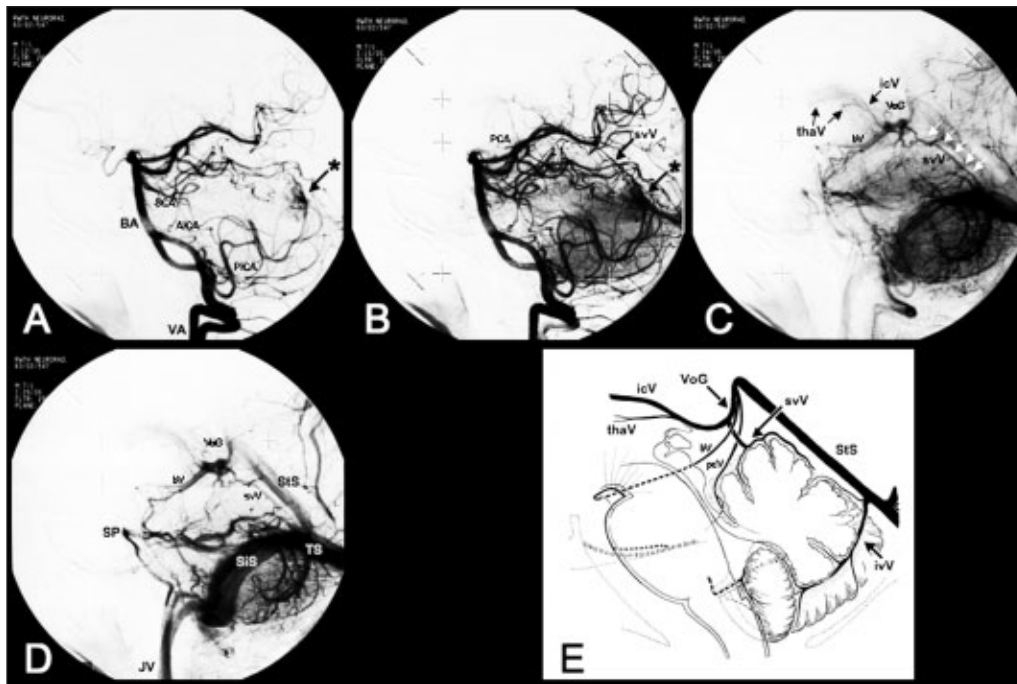
Figure 2: *Series of lateral angiograms. **A-B:** Early arterial phases demonstrating a small-sized (15x15x10 mm) single AVM (*) in the vermis of the cerebellum which was fed mainly by branches from the left PICA and drained early to the superior vermian veins (svV). **C-D:** Later venous phases reveal venous drainage via the svV (white arrows), to the vein of Galen (VoG, great cerebral vein) and in the straight sinus (StS). The thalamic venous system can be divided into: (1) a superior group including the superior thalamic vein, large and constantly present and the anterior thalamic vein, an inconstant accessory; (2) a medial group; (3) an inferior group; and (4) a posterior group, which includes a variable number of perforating veins.[8,9] The thalamic veins (thaV) are depicted relatively early, before the end of the cerebellar or occipital capillary phase and before the choroid plexuses; in the final venous phases they are no longer visualized, a fact which is mostly related to the shortness of the arteriovenous circuit in the basal nuclei.[8] In the present case, the locations of the bleedings suggest that the hemorrhages originated in the posterior segment of the deep nuclei. Since the superior (anterior) group of the thalamic venous perforators drain into the internal cerebral veins, and the medial thalamo-perforators into the epithalamic vein, it may be that venous hypertension occurred in the basal vein of Rosenthal (bV) because of the abnormal blood flow. AICA-Anterior inferior cerebellar artery, BA-Basilar artery, bV-Basal vein of Rosenthal, icV-Internal cerebral vein, JV-Internal jugular vein, PCA-Posterior cerebral artery, PICA-Posterior inferior cerebellar artery, pcV-precentral cerebellar vein, SCA-Superior cerebellar artery, SiS Sigmoid Sinus, SP-Sinus petrosus (sup./inf.), thaV-Thalamic veins (ant./sup.), TS-Transverse Sinus, VA-Vertebral artery. (**E:** adapted from Huang Y.P. & Wolf B.S., 1970 - reprinted with permission from the American Journal of Roentgenology).*

strated indeed a significant increase in cortical venous pressure in parts of the venous draining system with an enlarged local susceptibility to haemorrhages.[4] Our case is in thus far interesting, as we were not able to detect the classical signs of venous congestion on DSA, i.e., stagnation of contrast media within the thalami or retrograde venous flow. Since AVMs are dynamic, i.e. they may undergo anatomic and haemodynamic changes including regression which in turn can alter the blood flow and drainage pattern.

REFERENCES

1. Fardoun R, Adam Y, Mercier P, Guy G. Tentorial arteriovenous malformation presenting as an intracerebral hematoma. J Neurosurg. 1981;55:976-8.
2. Hurst RW, Hackney DB, Goldberg HI, Davis RA. Reversible arteriovenous malformation-induced venous hypertension as a cause of neurological deficits. Neurosurgery. 1992;30:422-5.
3. Reul J, Thron A, Laborde G, Bruckmann H. Dural arteriovenous malformations at the base of the anterior cranial fossa: report of nine cases. Neuroradiology. 1993;35:388-93.
4. Miyasaka Y, Kurata A, Tokiwa K, Tanaka R, Yada K, Ohwada T. Draining vein pressure increases and hemorrhage in patients with arteriovenous malformation. Stroke. 1994;25:504-7.
5. Ito M, Sonokawa T, Mishina H, Sato K. Reversible dural arteriovenous malformation-induced venous ischemia as a cause of dementia: treatment by surgical occlusion of draining dural sinus: case report. Neurosurgery. 1995;37:1187-92.
6. Greenough GP, Mamourian A, Harbaugh RE. Venous hypertension associated with a posterior fossa dural arteriovenous fistula: another cause of bithalamic lesions on MR images. Am J Neuroradiol. 1999;20:145-7.
7. Kurozumi K, Onoda K, Tsuchimoto S. Large cerebral arteriovenous malformation presenting with venous ischemia in the contralateral hemisphere. J Neurosurg. 2002;97:995-7.
8. Christoforidis GA, Bourekas EC, Baujan M, Abduljalil AM, Kangarlu A, Spigos DG, et al. High resolution MRI of the deep brain vascular anatomy at 8 Tesla: susceptibility-based enhancement of the venous structures. J Comput Assist Tomogr. 1999;23:857-66.
9. Giudicelli G, Salamon G. The veins of the thalamus. Neuroradiology. 1970;1:92-8.