Brain AVM

Relationship of Angioarchitecture and Clinical Symptoms and Implications for Treatment

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The clinical variability in the presentation of patients with brain arteriovenous malformations (B-AVM) may in part be due to the difference in host response to the presence of an AVM as demonstrated in the angioarchitecture.

Prenidal

Prenidal arterial abnormalities such as arterial ectasias and stenosis almost certainly represent an individual host response to the increased flow associated with the downstream fistula. Stenosis may be so profound as to become occlusive and secondarily induce extensive collateral circulation phenomena (Moya Moya type).

Clinical symptoms under these circumstances may entirely be unrelated to the AVM itself. Aneurysms involving the arterial circulation on the way to the AVM occur at a much higher frequency (15 to 25%) and in different locations (beyond the circle of Willis), as compared to patients without AVMs. These aneurysms proximal to an AVM may rupture and be responsible for clinical presentation. Current experience suggest that in the natural history the risk of haemorrhage is twice as high (7 versus 3.5%) in patients with brain AVM and arterial aneurysms versus those without arterial aneurysms. The Toronto experience has

also shown that regression of aneurysms on the feeding vessels to the AVM may occur following complete obliteration of the AVM, but his effect is to be anticipated only on feeding vessel aneurysms, which are at a close distance from the AVM.

The presence of direct versus indirect feeders towards the AVM is of major importance with regard to the feasibility of endovascular treatment. Indirect feeders often occur in AVM locations near the watershed zone of major arterial territories. They can make angiographic assessment of the size and extent of the AVM difficult and for that purpose MRI examinations are not only helpful but also mandatory. It appears unlikely that indirect feeder supply to the AVM is associated with different or increased incidence of neurological symptoms caused by so called "steal phenomena", and this angiographic feature is mostly important from an endovascular versus surgical management point of view.

Nidus

Nidus angioarchitecture has previously been analyzed mostly with regard to neurosurgical parameters emphasizing size, location, venous drainage, etc. Superselective angiography has demonstrated intranidal aneurysms, pseudoaneurysms, venous ectasias and fistulous com-

munications. The significance of these intranidal abnormalities has become more apparent over the last five years. The presence of intranidal aneurysms is most likely responsible for the two-fold risk of haemorrhage (7%) of patients with AVM and associated aneurysms. These aneurysms should be considered for endovascular treatment (targeted, partial) in particular in those patients where for various reasons a complete cure of the entire AVM cannot be achieved, or a cure is anticipated to take several years (following stereotactic radiosurgery).

Venous

The venous abnormalities associated with B-AVM have increasingly been recognized as critical to the understanding of B-AVM symptomatology. Venous hypertension and congestion similar to that occurring in dural AVF with cortical venous reflux is perhaps responsible for symptoms previously believed to be caused by the so called "arterial steal phenomenon". The hydrodynamic influences of venous hyperpressure are exemplified in patients with vein of Galen malformations.

The angiographic appearance of venous ectasias, stenosis and occlusion have clinical expressions which can be clearly identified and have therapeutic implications. Deep venous drainage and single draining veins are recognized markers for haemorrhagic presentation. Elimination of this aspect of the angioarchitecture by endovascular or other means should be anticipated to result in a change in the natural history outcome. Careful analysis should also be carried out with regard to the venous

drainage pattern of the normal brain, as it may share certain venous exits with the AVM, and if so, this may create potential increased risk at the time of treatment (venous thrombosis and infarction).

Further analysis of the angioarchitecture of brain AVMs will lead to improved understanding of the clinical symptomatology and almost certainly result in modified and improved strategies in the future.

References

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