Developmental venous anomaly diagnosis

Radiographic features

VAs are seen on both CT and MRI as a leash of vessels draining towards a central vein.

CT

If large, the draining vein may be seen on non-contrast CT, and is confirmed with contrast administration as a linear or curvilinear enhancing structure.

MRI

There may be some T2 hyperintensity on FLAIR.

May be visible on most sequences, but is most easily seen on postcontrast T1 sequences. If there is an associated cavernous haemangioma then susceptibility weighted sequences will be most sensitive for this component.

SWI is the preferred sequence in venous anomalies and proved to have better detectability of venous structures than conventional T2*-weighted imaging. Signals on SWI are not compromised by low-velocity venous flow. Therefore, SWI has successfully demonstrated low-flow vascular formations such as DVA. DSA: angiography

Angiographically the caput medusae appearance (collection of dilated medullary veins converge in an enlarged transcortical or subependymal collector vein) is pathognomonic and seen only in the venous phase. Arterial phase appears normal although late capillary blush may be present. No shunting is present. Treatment and prognosis

If isolated, than no treatment is necessary. If part of an MVM then treatment will be predicated by the other component. Informing the surgeon of the presence of a DVA is however essential as cautery of the the collecting vein can lead to venous infarction.

When isolated, a DVA has a very low complication rate (0.15% per annum) mainly from spontaneous thrombosis of the collecting vein leading to venous infarction and haemorrhage.

However, DVAs are commonly associated (15-20%) with other vascular malformations, most commonly a cavernous malformation in which case they should be referred to as mixed vascular malformations. Their risk of complication is then elevated to that of the associated lesion. There is an association with cortical dysplasia.

A more recent study has demonstrated that in exceedingly rare cases, the DVA can become symptomatic by various vascular complications. The authors suggested identification of the underlying pathomechanism by MR and DSA for proper management. Importance of preserved integrity of the DVA itself still holds true 6. Differential diagnosis

General imaging differential considerations include:

Dural sinus thrombosis with collateral drainage

Sturge Weber syndrome with leptomeningeal angiomatosis

demyelination may also have enlarged medullary veins

Practical points

Think of an associated cavernoma when a DVA is found in an intraparenchymal hemorrhage investigation (DVA rarely bleed)

Best options to find it could be a gradient-echo or blood oxygen level-dependent sequences on MRI [1].

Angiogram

Occasionally may be angiographically occult; however, they classically produce a distinct caput-medusae (other descriptive terms include: a hydra, spokes of a wheel, a spider, an umbrella, a mushroom, or a sunburst or starburst).

Other angiographic characteristics: appears as a long draining vein (longer than a normal vein) draining an excessive amount of brain tissue (it is theorized that venous restrictive disease occurs because of the length), the arterial phase should show no AV shunting (characteristic of AVM).

1)
http://radiopaedia.org/articles/developmental-venous-anomaly