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RARE CASE OF CEREBRAL ARTERIOVENOUS MALFORMATION PRESENTING AS PARASTHESIA OF ALL FOUR LIMBS

Radiodiagnosis

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ABSTRACT

Brain arteriovenous malformations (AVM) are abnormal vascular connection between arteries (pial vessels) and veins via a nidus of vessels through which shunting of blood occurs in the absence of true capillary bed. When intracranial AVMs are encountered at cross-sectional imaging, other diagnoses like malignant dural arteriovenous fistulas, and moyamoya disease must also be considered. Secondary effects such as gliosis, neurologic deficits, venous congestion, hydrocephalus should be included in the radiology report. Imaging findings influence the clinical management by neurosurgeons.

KEYWORDS

arteriovenous, nidus, shunting, flow voids, vascular malformation.

INTRODUCTION:

Brain AVM's are almost always developmental or congenitally acquired.The most common type of brain AVM is glomerular and fistulous being less common (1).

Glucose transporter protein (GLUT 1) are present in cranial AVMs, unlike extracranial AVMs (2).

The **Spetzler-Martin arteriovenous malformation (AVM) grading system** allocates points for surgical treatment risk.

The grading is based on the size of nidus, eloquence of adjacent brain & venous drainage. The following areas are considered eloquent: sensory, motor, language, or visual cortex, hypothalamus or thalamus, internal capsule, brainstem, cerebellar peduncles (superior, middle, or inferior), deep cerebellar nuclei.

Higher (or lower) the grade, the higher (or lower) the complication rate. Therefore, patients with smaller and cortical-based brain AVMs are likely to benefit most from surgical resection.

CASE REPORT

A 23 year old female presented with sudden onset of slurred speech, weakness & numbness in all the four limbs. No history of chronic headache/seizures.

On imaging, non contrast MRI brain showed large area of serpiginous abnormal flow voids with perilesional edema involving right parasagittal frontal region measuring 9 x3 x 4cm. Inferiorly, the lesion is reaching up to the right caudate nucleus, causing mild mass effect on body of corpus callosum. Supero-medially, it is abutting anterior half of the superior sagittal sinus as well as falx without causing compression/obliteration. No obvious evidence of invasion either. No evidence of aneurysms/ focus of calcification.

On post contrast images, a nidus is seen with dilated tortuous feeding artery most prominently from A1 segment of right ACA and multiple veins draining the lesion into superior sagittal sinus (SSS). No evidence of intracerebral/subdural bleed/infarct/ischemia.

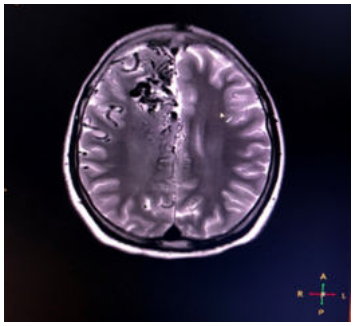

Fig: T2.

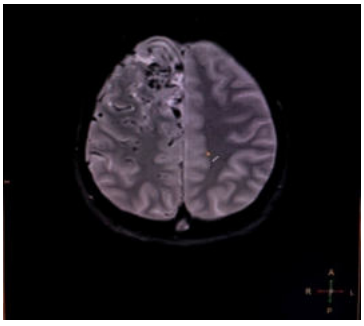

Fig: T2 FFE.

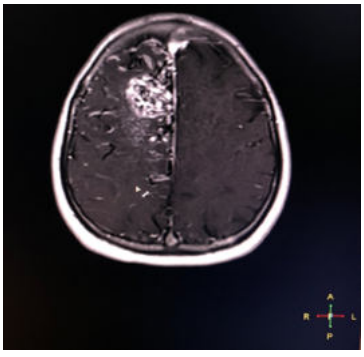

Fig: Post contrast T1.

DISCUSSION:

Cerebral angiography remains the gold standard, able to delineate the location and number of feeding vessels and the pattern of drainage. Ideally, angiography is performed in a bi-plane system with a high rate of acquisition, as shunting can be very rapid. Risk of treatment must be assessed in view of the future hemorrhage.

MR angiography is often useful for subtracting the hematoma components when an AVM complicated by an acute hemorrhage needs to be imaged. On CTA feeding arteries, draining veins & intervening nidus is visible in 'bag of worms' appearance.

- **Treatment:** Endovascular embolisation (adjunct), radiosurgery (lesion < 3cm),microsurgical removal.
- Endovascular embolization can be used to quickly eliminate angiographic risk factors. There are no real contraindications for endovascular therapy; however, the cure rate with embolization alone is relatively low (~10%–20%), except in highflow pial AVFs or small lesions.(1)
- Radiosurgery has a high cure rate with relatively low complication rates. However, its major limitation is that radiation is slow to take effect; it may be up to 2 years before any shrinkage of the brain

AVM is seen. Therefore, radiosurgery may not be well suited for the treatment of brain AVMs with angiographic risk factors for future hemorrhage.(1)

- Risk factors of future hemorrhage: Previous hemorrhage, intranidal aneurysms, venous stenosis or ectasia (pouches) ,deep venous drainage, single venous drainage ,deep or posterior fossa locations & risk of nonhemorrhagic neurologic deficits: High-flow shunt, venous congestion or outflow obstruction, long pial course of draining vein, perifocal or perinidal gliosis, mass effect or hydrocephalus arterial steal. (1) (3).

CONCLUSION:

Cross sectional imaging plays a vital role by providing the information necessary for AVM management. Distal subtraction angiography (DSA) remains the gold standard in diagnosis to delineate location, number of feeding vessels and pattern of drainage. On MRI fast flow generates flow voids, easily seen on T2 weighted images. MRA is useful for subtracting hematoma components. Diagnosis can be difficult on non-contrast CT.

Risk of non-hemorrhagic complications like focal neurological deficit increases with a long pial course of a draining vein, arterial steal, mass effect, and perinidal gliosis (3).

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