Angiographic and MRI/MRA Characterization of Intracranial Vascular Malformations: A Pictorial Review

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Learning objectives

• Discuss and illustrate radiologically the different types of intracranial vascular malformations.
• Describe the radiological descriptors and terminology in radiological reporting including classification.
• Review the complementary role of magnetic resonance imaging (MRI), magnetic resonance angiography (MRA) and angiography in the diagnosis, characterization and management of intracranial vascular malformations.
Background

Introduction

Intracranial vascular malformations are a diverse set of uncommon anomalies that may be asymptomatic or present with devastating hemorrhagic events or serious neurological symptoms. Estimates of the overall incidence of vascular malformations involving the brain range from 0.1% to 4%. Some of the lesions are arteriovenous malformations (AVMs), dural arteriovenous fistulas (DAVFs), cavernous malformations (CMs) and developmental venous anomalies (DVAs). The clinical presentation, evolution and therapeutic approach depend on type, location, size, and overall hemodynamic characteristics of the lesion. Different systems have been proposed for the classification of intracranial malformations, which helps to understand complications, guide management and treatment, and predict potential complications. With more advanced radiological modalities, intracranial vascular malformations are now more commonly identified and better characterized, even when asymptomatic.

Arteriovenous Malformations (AVMs)

I. Definition

- Focal vascular abnormalities consisting of multiple dilated arteries and veins within the brain parenchyma, without an intervening capillary network (Figure 1).

II. General Features

- The prevalence in the general population is approximately 0.1%.
- The mean age at diagnosis is between 30 and 40 years.
- The lesions are thought to be congenital in origin.
- Although occasional cases are associated with other abnormalities (e.g., Osler-Weber-Rendu disease and the Sturge-Weber syndrome), arteriovenous malformations are not regarded as familial, and the overwhelming majority of cases are sporadic.
- The majority arise supratentorially.

III. Clinical Presentation and Natural History

- Approximately 12% of patients with arteriovenous malformations will become symptomatic during the lifetime of the patient (1).
- Signs and symptoms include:
• Intracerebral hemorrhage (most common)
• Seizures
• Mass effect
• Ischemia in adjacent parenchyma secondary to vascular "steal" phenomenon
• Predisposition factors that increased the risk of hemorrhage include:
  • Aneurysms
  • Drainage into the deep venous sinuses
  • Deep location
  • Single draining vein
  • Venous outflow stenosis

IV. Classification

• To help the neurosurgeon estimate the surgical risk, several classifications have been developed, but the most commonly used today is the one proposed by Spetzler and Martin (Figure 2) (2).
  • This classification simplifies the estimation of the surgical risk by considering the lesion size, location, and drainage.

V. Treatment

• Therapeutic options include observation, embolization, surgery and/or radiosurgery.
• The goal of therapy is complete obliteration of the arteriovenous malformation to prevent the risk of hemorrhage.
• Decisions as to which lesions are most amenable to surgery are commonly based on the Spetzler-Martin scale (2).
  • Strong consideration of surgery is recommended for lesions of Spetzler-Martin grade I and grade II, and consideration of endovascular embolization followed by microsurgery is recommended for grade III lesions.
  • Consideration of radiosurgery is recommended for lesions that may be associated with an increased rate of surgical complications, owing to their anatomical location or drainage anatomy, in particular for lesions in eloquent tissue.
  • Grade V and many Grade IV AVMs should be left alone due to the risk of treatment, unless the patient has a serious progressive neurological deficit or has suffered multiple hemorrhages.
• Please note that many AVMs are treated in a multimodal stage approach.

**Dural Arteriovenous Fistula (DAVFs)**

I. Definition
• Intracranial dural arteriovenous fistulas (DAVFs) are pathologic connections between branches of dural arteries and dural venous sinuses, meningeal veins, or cortical veins (Figure 3).
• Unlike arteriovenous malformations, DAVFs have a dural arterial supply and lack a parenchymal nidus.
• DAVFs can involve any dural sinus however, the most common locations include the transverse and sigmoid sinus.
• Most are acquired and present in adulthood.
  • Usually occur in an idiopathic manner.
  • In certain cases a trigger may be identified including:
    • Trauma, cranial surgery, dural sinus thrombosis or cortical venous thrombosis, hypercoagulable states, tumors, or infection.

II. General Features
• Constitute approximately 10% to 15% of all cerebrovascular malformations (3).
• A higher incidence is identified in middle-aged patients (50-60 years).

III. Clinical Presentation and Natural History
• Patients may be completely asymptomatic or suffer catastrophic intracranial hemorrhages.
• Signs and symptoms are related to the location and hemodynamic pattern of the lesion.
  • Transverse and sigmoid sinus: pulsatile tinnitus.
  • Cavernous sinus: ophthalmoplegia, proptosis, chemosis, retroorbital pain and decreased visual acuity.
  • Headache, dementia, seizures or progressive encephalopathy.
  • Intracranial hemorrhage (approximately 2% per-year depending on the site and hemodynamics of the lesion) (4).
  • DAVF venous drainage pattern and location determines the severity of symptoms.
    • Retrograde venous cortical drainage is associated with a more severe clinical presentation and an aggressive natural history.

IV. Classification
• Borden classification is based on the venous drainage pattern and the presence or absence of cortical venous drainage (Figure 4)(5).
• Cognard classification is based on the direction of dural sinus drainage (antegrade or retrograde), the presence or absence of cortical venous recruitment, and venous outflow characteristics (nonectatic/ectatic cortical
veins or spinal perimedullary veins) (Figure 5) (6). This classification enables accurate comparison of clinical and radiological parameters.

V. Treatment

- Available therapeutic options include no treatment, conservative therapy, palliative or definitive endovascular treatment, surgery, a combination of endovascular therapy and surgery, and radiosurgery.
- Low-grade lesions without angiographic evidence of retrograde sinus or cortical venous drainage and presenting with non-debilitating symptoms can be managed conservatively with observation.
- Low-grade lesions with debilitating symptoms can be managed with palliative arterial endovascular embolization to diminish focal symptomatology.
- Aggressive intracranial DAVFs presenting with intracranial hemorrhage or progressive neurological symptoms need definitive therapy with surgery or embolization. Disconnection of the cortical venous reflux is necessary to protect the patient against the sequela of intracranial hemorrhage or severe neurological deficits.
- Radiosurgery carries a time delay of 2-3 years for DAVFs obliteration, therefore is not recommended as a primary therapeutic measure for these lesions. This method should be considered if occlusion by surgical or endovascular means is not possible or carries high morbidity.

Cavernous Malformations (CMs)

I. Definition

- Also known as cavernous hemangiomas, cavernous angiomas, or cavernomas.
- Usually presents as a discrete, honeycomb-like mass of endothelial-lined sinusoidal vascular spaces with no intervening brain parenchyma (Figure 6).

II. General Features

- The prevalence in the general population is approximately 0.4% to 0.8% and represents about 10% to 15% of cerebrovascular lesions (7).
- Most often diagnosed in patients between 40-60 years.
- Occur in two forms: a sporadic (nonhereditary) and a familial form.
  - The sporadic is usually solitary and the familial presents earlier with multiple lesions.
  - The majority arise supratentorially within the brain parenchyma.
III. Clinical Presentation and Natural History

- Most CMs are found incidentally and remain asymptomatic.
  - If the patient becomes symptomatic, the clinical presentation may include: seizures, focal neurologic deficits and hemorrhage.
  - Hemorrhagic risk: estimated at 0.6% to 4.5% per lesion-year; higher in brainstem lesions, in familial cases and in cases with previous hemorrhage (8).
- May be associated with other abnormalities such as developmental venous anomaly, superficial siderosis and cutaneous malformations.
- Present with a broad range of dynamic behavior including lesion enlargement, regression and even de novo lesions in other locations may develop.
- CMs located in the cortex or white matter of the cerebral hemispheres are classified as superficial lesions.
  - These lesions typically follow a more indolent course with surgery usually reserved for patients with intractable epilepsy.
- Deep lesions located in the brainstem, cerebellar nuclei, basal ganglia, and thalamus have a higher hemorrhagic rate and may be clinically devastating due to their close proximity to eloquent structures.

IV. Classification

- Zabramski and coworkers suggested a MRI classification ranging from types I to IV (Figure 7) (9).

V. Treatment

- Treatment options include conservative therapy such as MRI follow up, medical management of symptoms associated with the lesion such as seizures, surgical resection or stereotactic radiation.
- Although microsurgical resection of symptomatic CMs is well established, surgery itself carries a risk of neurological deficit especially in eloquent locations.
- Radiosurgery provides an alternative for such cases, but its efficacy remains in doubt.

Developmental Venous Anomalies (DVAs)

I. Definition

- Also known as cerebral venous angioma.
Characterized by a network of dilated medullary veins (caput medusa sign) converging in a radial fashion to a single larger collecting vein, which in turn drains into either a dural sinus or into a deep ependymal vein (Figure 8).

The etiology of DVAs is uncertain. However, arrest of developing venous structures has been proposed as a possible etiology.

II. General Features

- They are the most common cerebral vascular malformation, conforming approximately 63% and 50% of all malformations in autopsy and MRI series, respectively (10).
- Usually diagnosed during the fourth and fifth decades of life.
- The majority arise supratentorially within the frontal-parietal lobes, usually draining towards the frontal horn of the lateral ventricle.
- The second most common location is infratentorially in the cerebellar hemisphere draining towards the fourth ventricle.

III. Clinical Presentation and Natural History

- DVAs are typically asymptomatic and found incidentally during a neuroimaging study.
- Signs and symptoms may include (when present)
  - Headache (most common)
  - Seizure
  - Hemorrhage (usually presents with an associated cavernous malformation)
    - Hemorrhagic risk of DVAs has been estimated in approximately 0.34% per lesion per year.
    - Certain factors may increase the risk of hemorrhage including stenosis or thrombosis or a coexisting cavernous malformation.
- DVAs are associated with other abnormalities including cavernous and cervicofacial venous or lymphatic malformations.

IV. Treatment

- Solitary DVA are not treated, since an attempt at removal may cause venous infarction. However, they may be followed with MR if hemorrhage or thrombosis with associated edema or ischemia is detected.
- In case of a mixed venous anomaly, the coexisting lesion dictates the therapeutic approach.
Fig. 1: Arteriovenous Malformation (AVM) Graphic Representation

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Fig. 2: Spetzler-Martin Grading Scale for Arteriovenous Malformation * Graded on a 1-5 scale. Total points = size + venous drainage + eloquence. † Eloquent areas include sensorimotor, visual, or language cortex; thalamus; internal capsule, hypothalamus; brainstem; cerebellar peduncles; and deep cerebellar nuclei. Non-eloquent area = anterior frontal and temporal lobes, cerebellar hemisphere.

**Fig. 3:** Dural Arteriovenous Fistula (DAVF) Graphic Representation

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<table>
<thead>
<tr>
<th>Type</th>
<th>Venous Drainage Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>Dural venous sinus</td>
</tr>
<tr>
<td>Aggressive</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Dural venous sinus with cortical venous drainage</td>
</tr>
<tr>
<td>III</td>
<td>Directly into the cortical veins</td>
</tr>
</tbody>
</table>

**Fig. 4:** Borden Classification of DAVFs

**Fig. 5:** Cognard Classification of DAVFs

Fig. 6: Cavernous Malformation (CM) Graphic Representation

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<table>
<thead>
<tr>
<th>Classification</th>
<th>T1</th>
<th>T2</th>
<th>Gradient Echo</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>Hyperintense core</td>
<td>Hypointense halo and hyperintense core</td>
<td>Subacute hemorrhage</td>
<td></td>
</tr>
<tr>
<td>Type II</td>
<td>Reticulated mixed core signal</td>
<td>Mixed core signal surrounded by a hypointense rim</td>
<td>Lesions with hemorrhages and thromboses of different age</td>
<td></td>
</tr>
<tr>
<td>Type III</td>
<td>Hypo- to isointense</td>
<td>Hypointense</td>
<td>Hypointense</td>
<td>Chronic resolved hemorrhage with hemosiderin staining within and around the lesion</td>
</tr>
<tr>
<td>Type IV</td>
<td>Not visible</td>
<td>Not visible</td>
<td>Small punctate hypointense lesion</td>
<td>Small lesion similar in appearance to telangetasia</td>
</tr>
</tbody>
</table>

**Fig. 7:** Cavernous malformations (CMs) Classification (Zabramski and coworkers)

Fig. 8: Developmental Venous Anomaly (DVA) Graphic Representation

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Imaging findings OR Procedure details

Arteriovenous Malformations (AVMs)

I. Diagnostic Imaging

• MRI and angiography provide complementary information that is necessary to understand the AVM anatomy and determine the best course of treatment.
• Angiography is still the gold standard by which to evaluate the architecture of arteriovenous malformation, including the presence or absence of associated aneurysms, obstruction of venous outflow, and pattern of venous drainage.
  • MRI is a sensitive test that can delineate details of the architecture of the AVM.
  • The spatial relationships of the nidus, feeding arteries, and draining veins, as well as architectural relationships between AVM and adjacent brain.

MRI Findings (Figures 9, 10, 11 and 12)

T1/T2WI
- Tangle of vessels with flow voids
- May have hemorrhage with variable signal

T1WI C+
- Enhancement of nidus, draining veins
- Rapid flow may not enhance ("flow void")

FLAIR
- Flow voids with surrounding hyperintense gliosis may be better demonstrated

T2* GRE
- Blooming artifact if hemorrhage present

MRA
- Provides additional information regarding the nature of the feeding arteries and draining veins such as their location in space and relation to other vascular structures
• **Angiography**
  - Every patient with a suspected AVM must have a cerebral angiogram. This is an obligatory step in the preoperative evaluation of a patient with an AVM (*Figure 10, 11, 12*).
  - Can localize the nidus, the feeding arteries and draining veins. Is also able to detect associated aneurysms and other AVM-related factors that are associated with a high hemorrhage risk (*Figure 13*).

**Dural Arteriovenous Fistula (DAVs)**

**I. Diagnostic Imaging**

- Conventional MR imaging is unable to directly visualize the shunt in DAVFs.
  - Its main roles includes:
    - Documenting parenchymal complications.
    - Suggesting venoocclusive disease by defining abnormal cortical venous drainage.
    - Suggest venous hypertension and venous outflow obstruction by delineating enlarged deep medullary veins.
    - Exclude other causes of venous dilation (e.g., parenchymal AVM, isolated dural sinus thrombosis).
    - MR is usually unable to delineate the fistula; however, the presence of dilated pial vessels without an AVM nidus is suggestive of a possible DAVF.

**MRI Findings** (*Figure 15*)

<table>
<thead>
<tr>
<th>Sequence</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1/T2WI</td>
<td>• Extra-axial flow voids may be appreciated with an adjacent thrombosed dural sinus</td>
</tr>
<tr>
<td>T1WI C+</td>
<td>• Chronic sinus thrombosis usually demonstrates some level of enhancement</td>
</tr>
<tr>
<td>T2WI/FLAIR</td>
<td>• Surrounding edema secondary to venous congestive changes and/or ischemia</td>
</tr>
<tr>
<td>T2* GRE</td>
<td>• Blooming artifact in thrombosed dural sinus and/or parenchymal hemorrhage</td>
</tr>
<tr>
<td>DWI</td>
<td>• Normal, unless venous infarct or ischemia is present</td>
</tr>
</tbody>
</table>
MRA

- Phase contrast is useful for characterization of DAVFs feeding vessels, spatial relation to other vascular structures and hemodynamics
- TOF is positive in larger DAVFs however, a false negative study may be obtained with many smaller or slow-flow lesions

MRV

- Demonstrates thrombosed sinus with collateral flow

Angiography

- Conventional angiography remains the gold standard for DAVF diagnosis, classification and treatment planning.
  - It allows a systematic evaluation of the feeding vessels and demonstrates the presence and extent of retrograde venous drainage (Figure 15 and 16).

Cavernous Malformations (CMs)

I. Diagnostic Imaging

- MRI is the standard imaging technique to diagnose CMs, showing characteristic features that have been described as resembling a "mulberry" or "popcorn" appearance.
  - The presence of blood products at different stages of evolution is responsible for its typical MRI signal characteristics.
- CMs are angiographically occult due to the slow blood flow.

MRI Findings (Figure 17 and 18)

T1WI

- Hemorrhage appearance is variable depending on stage of evolution.
- "Mulberry" or "popcorn" appearance with hyper- and hypointense blood-containing locules.

T1WI C+

- Minimal or no enhancement
- Contrast may show associated venous malformation.
T2WI

- "Mulberry" or "popcorn" appearance with hyper- and hypointense blood-containing locules and a complete hypointense hemosiderin rim.
- Surrounding parenchymal hyperintense signal representing edema.

T2 GRE

- Blooming artifact depicted as numerous punctate hypointense foci.

MRA

- Usually normal, unless a mixed malformation is present.

**Angiography**

- Digital subtraction angiography is considered an unnecessary diagnostic tool in evaluating CMs. Reserved only for equivocal cases (Figure 18).

**Developmental Venous Anomalies (DVAs)**

I. Diagnostic Imaging

- MRI is the primary imaging method for DVA diagnosis.

**MRI Findings** (Figure 18 and 19)

**T1WI**

- Variable signal intensity depending on vascular flow and size of the lesion.
- Small DVA may yield false negative results.

**T1WI C+**

- Enhancement in a stellate pattern of tubular vessels converging into a larger collecting vein

**T2WI/FLAIR**

- Flow void representing the collector vein
- Peripheral hyperintensity may represent the presence of
gliosis, venous ischemia and/or hemorrhage.

T2* GRE
- Blooming artifact if lesion is large or if there are coexisting mixed malformations.

DWI
- Normal, unless venous infarct or ischemia is present

MRA
- Normal in the arterial phase. Slow flow on phase-contrast MRA.

MRV
- Helps to demonstrate the "Medusa head" and better depicts the drainage pattern.

- **Angiography**
  - In angiographic studies DVAs present with normal arterial and capillary flow.
  - The venous phase demonstrates the pathognomonic angiographic appearance consisting of wedge-shaped collections of dilated medullary veins ("Medusa head") converging in an enlarged subependymal or transcortical collector vein (*Figure 18*).
Fig. 9: Axial T2 WI (a) and Sagittal T1 WI (c) MR shows a tangle of innumerable tiny serpiginous flow voids within the splenium of the corpus callosum representing the AVM nidus. Note the markedly enlarged draining vein of Galen (yellow arrow). There is relative lack of mass effect and no discernable normal brain parenchyma within the lesion. Axial (b) and sagittal (d) T1 WI post contrast MR in the same patient demonstrates corresponding avid enhancement of the AVM nidus and dilated draining vein of Galen.

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Fig. 10: Axial T2 WI (a) MR and coronal TOF MRA (c) shows an AVM with a very large tangle of serpiginous flow voids within the left parietotemporal lobe representing the AVM nidus with multiple draining veins. This malformation appears to have feeders from branches of the left middle cerebral and left posterior cerebral arteries which are markedly engorged. There is also marked enlargement of the superior sagittal and transverse sinuses on side of drainage. Note that besides the size of the lesion there is minimal mass effect as is seen with AVMs. Axial T1 WI post contrast (b) MR at the same level shows avid enhancement of the nidus and draining veins. Note that rapid flow may not enhanced showing areas of flow voids. Lateral (d) and anteroposterior (e) left ICA DSA demonstrate previously described very large AVM nidus with multiple draining vessels. No flow-related aneurysms were noted.

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**Fig. 11:** Axial T1 (a) and T2 (b) WI MR shows a tightly packed mass with a tangle of serpiginous "honeycomb" flow voids within the body and atrium of the right lateral ventricle consistent with an AVM. There is associated extension into the adjacent brain parenchyma; specifically, it includes the right thalamus and the right splenium of the corpus callosum, better demonstrated in T2 images. Axial T1 WI post contrast (c) MR at the same level shows strong enhancement of the nidus and draining veins. Note that rapid flow may not enhance showing areas of flow voids. Axial TOF MRA MIP (d) confirms the right intraventricular/perimesencephalic AVM demonstrating dilated branches of the right middle cerebral and right posterior cerebral feeding arteries with drainage into an enlarged galenic system. Lateral right vertebral (e) and ICA (f) DSA demonstrate a large AVM nidus filling from the right posterior cerebral artery and a small contribution from the right middle cerebral artery. Deep venous drainage into the galenic system and notable cortical venous reflux was demostrated (not shown).

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Fig. 12: Axial T2 WI (a) and FLAIR (b) MR shows a left frontoparietal tangle of multiple serpiginous flow voids consistent with an AVM nidus with enlarged feeding and draining vessels. No normal brain identified within the lesion. There is associated increased signal intensity within the surrounding parenchyma consistent with gliosis and/or edema. Axial T2* GRE (c) MR of the same patient shows prominent hypointense "blooming" (susceptibility effect). Axial T1 WI post contrast (d) MR in the same patient demonstrates corresponding avid enhancement of the AVM nidus. There are areas of rapid flow that does not enhance and presents as flow voids. Axial TOF MRA MIP (e) confirms the left frontoparietal arteriovenous malformation with enlarged feeding arteries arising from the opercular segment of the left middle cerebral artery. Anteroposterior left ICA DSA (f) demonstrates the left frontoparietal AVM nidus composed of tightly packed vessels. This lesion is feeding from left middle cerebral artery branches and draining into deep venous system and cortical venous system into the sagittal sinus.

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Fig. 13: Anteroposterior (a) and lateral (b) right internal carotid artery (ICA) DSA shows enlarged right middle cerebral artery branches feeders, dilated/tortuous draining veins (right vein of Labbe, orange arrow, cortical veins into sagittal sinus and right sphenoparietal sinus) consistent with an AVM. Anteroposterior left ICA DSA (c) and oblique 3D DSA (d) shows normal cervical, petrous, cavernous and supraclinoid portion of the ICA. Note a very small left A1 perforator aneurysm. Teaching Point: A search for additional vascular lesions such as aneurysm should be performed as they can coexist.

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Fig. 14: Axial non enhanced CT (a) shows a right parietal parenchymal hemorrhage with associated surrounding vasogenic edema. This finding is known to represent an AVM complication. Axial contrast enhanced CT (b) shows a right parietal hematoma with associated surrounding vasogenic edema. In the posterior portion of this hematoma, there is an intense enhancing conglomerate of vessels which arise from a branch of the distal middle cerebral artery and contains a draining vein into the posterior superior sagittal sinus. Lateral ICA DSA (c) shows a right parietal AVM with feeders coming from the left anterior cerebral artery and middle cerebral arteries with an associated aneurysmatic dilatation. Note drainage into the superior sagittal sinus. Lateral ICA DSA post treatment (d) shows no residual right parietal AVM after surgical clipping and dissection.

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**Fig. 15:** Axial T2 WI (a), FLAIR (b), T1 W1 post contrast (c) and coronal TOF MRA (e) show dilatation and tortuosity of the left superior ophthalmic vein (yellow arrow). Note the associated left sided proptosis. Axial T1 W1 post contrast (d) demonstrates enlargement of the left cavernous sinus with evidence of arterialization (orange arrow). These findings are consistent with left direct carotid cavernous fistula. Lateral (f) and anteroposterior (g) ICA DSA demonstrate opacification of the cavernous sinus (orange arrow) and superior ophthalmic vein (yellow arrow).

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Fig. 16: Anteroposterior (a), oblique (b) and lateral (c) ICA DSA images show a dural arteriovenous fistula with feeders from the middle meningeal, right and left tentorial and occipital arteries.

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**Fig. 17:** Axial T2 WI MR (a) shows a reticulated popcorn-like lesion centered within the right posterior midbrain and extending into the superior posterior pons which contain multiple locules with blood-fluid levels surrounded by a complete hypointense hemosiderin rim. These findings are classic of a cavernous malformation. Axial FLAIR MR (b) of the same patient redemonstrate previously described findings. No evidence of surrounding edema. Axial T2* GRE MR (c) shows prominent susceptibility effect (hypointense "blooming") caused by the peripheral rim of hemosiderin and inhomogeneous clot. No evidence of normal brain tissue within the cavernous malformation. Axial T1 WI post contrast (d) shows minimal enhancement of previously described lesion.
**Fig. 18:** Axial T2 WI (a) MR shows a heterogeneous popcorn-like hyperintense lesion which shows a complete hypointense hemosiderin rim on both T2 and in corresponding T2*GRE image (b). These findings are characteristic of a CM. Axial FLAIR (c) MR of the same patient redemonstrate previously described findings. Lateral right ICA DSA (d) does not demonstrate the CM since most of the time these lesions are angiographically occult. Axial T1 WI post contrast (e and f) demonstrate a small linear hyperintense structure associated to the CM, extending posteriorly, consistent with a DVA. This entity is commonly seen in association with CM. Lateral right ICA DSA venous phase (g) nicely demonstrates numerous enlarged medullary veins that converge on an enlarged transcortical "collector" vein consistent with a DVA. Note the classic "medusa head" appearance (yellow arrow) of this lesions.
Fig. 19: Axial T2 WI (a) and FLAIR (b) MR shows umbrella-like enlarged deep white matter veins converging on a single enlarged deep "collector" vein (central dot-like enhancing foci) (yellow arrow) within the right frontal lobe which represents a typical DVA. Axial T1 WI (c) and coronal T1 WI post contrast (d) in the same patient shows avid enhancement of the enlarged stellate, tubular vessels converging on "collector" vein.

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Conclusion

Radiologists play a major role in the diagnosis of intracranial vascular malformations. MRI/MRA and angiography are valuable complementary tools in the diagnosis and management of these lesions. Radiologist should make an effort to adequately characterize the exact nature of the malformations such as location, size, feeding and draining vessels and associated complications. This information will provide an important first step in treatment planning and patient follow up.
References

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