Intracranial Vascular Malformations: A Pictorial Review

Poster No.: C-1925
Congress: ECR 2017
Type: Educational Exhibit
Authors: M. B. Bolina¹, U. Tazinaffo², L. Ramos², L. Cordoval²; ¹Belo Horizonte, MI/BR, ²Belo Horizonte/BR
Keywords: Neuroradiology brain, Vascular, MR, Education, Arteriovenous malformations, Education and training
DOI: 10.1594/ecr2017/C-1925

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.
As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.
You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.
Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.
www.myESR.org
Learning objectives

• To describe the radiological descriptors of the different types of intracranial vascular malformations.
**Background**

With the widespread use of brain imaging studies, intracranial vascular malformations are now more commonly identified and better characterized, even when asymptomatic.

Vascular malformations of the brain are often found in the workup of intracranial hemorrhage, seizures, focal neurological deficits, or headaches. Most vascular malformations are not easily evaluated on CT and are better seen on magnetic resonance imaging. In the case of AVMs, AVFs, or cavernous malformations (CMs), the lesion may serve as the etiologic source of the symptoms and thus warrant treatment [1].

The best management strategy sometimes is controversial, but it is important to consider the clinical presentation, location, size, and overall hemodynamic characteristics of the lesion.
Findings and procedure details

Intracranial vascular malformations traditionally are divided into the following four basic types [2,3]:

1. Arteriovenous Malformations (AVMs)
2. Capillary Telangiectasias
3. Cavernous Malformations (CMs)
4. Venous Malformations - Mainly represented by developmental venous anomalies (DVAs)

Vascular Malformations with varying mixtures of cavernous and capillary elements, AVMs, and venous anomalies have also been reported [2].

1. Arteriovenous Malformations (AVMs):

It is a connection between artery and vein, with a nidus (a tangle of abnormal vessels) in the transition (Classic Brain AVMs); or direct (fistulous), without a nidus (Arteriovenous Fistula - AVF) [3].

1.1 - Classic Brain AVMs (Parenchymal AVMs or pial AVMs) (Figs. 1 to 4): Consist of clusters of dilated feeding arteries or arterioles that collect to a nidus, where there is a connection to a draining vein or system of veins (Figs 1 to 4) [1,2].

They are the common symptomatic vascular malformations. Although congenital, patients tend to present later in life, most commonly with intracranial hemorrhage, seizures, or other symptoms such as focal neurological deficits or headache [1,3].

Brain AVMs are usually a solitary lesion shaped like a cone with its base on the cerebral cortex and apex pointing toward the ventricle (Fig 1a) [2].

The diagnostic criteria include the presence of a nidus embedded within the brain parenchyma and early venous drainage (the veins are seen in the "arterial" phase) (Figs 2,3,4), which may be identified at standard MR angiography or CT angiography if the shunt volume and draining veins are large enough [3].
The typical type of nidus is the glomerular or compact, which consists of abnormal vessels without any interspersed normal brain tissue (Figs 1 and 2); if normal brain parenchyma is interspersed throughout the tangle of vessels, it is termed diffuse or proliferative type nidus and differential diagnosis must be included [3].

Computed Tomography (CT) findings: Hyperdense serpentine structures (enlarged, tortuous vessels) which show enhancement (contrast-enhanced scanning is needed) and may present calcification (Fig. 2); CT is useful for demonstrating acute hemorrhage from AVMs, and in these cases the most important finding that must be urgently communicated to the attending clinician (apart from life-threatening findings such as midline shift or herniation) is the presence of intranidal aneurysms or venous pouches (Figs. 2 and 3), due to the higher risk of early recurrent hemorrhage [1,3].

Magnetic resonance imaging (MRI) findings: Flow voids on both T1 and T2-W images (Fig 1a); Hemorrhage in different stages of evolution is commonly present; perilesion gliosis evidenced by hyperintensity in the surrounding brain on T2-weighted MR or FLAIR images (Fig 1a) [4]. Gradient-echo T2-weighted sequences may help identify patients with a subclinical hemorrhage, which is relevant because previous hemorrhage is the most important and consistent predictor of future hemorrhage [2,3].

However, Digital Subtraction Angiography (DSA) remains the gold standard for excluding BAVM. [4]

Several imaging findings in brain AVMs have an impact on decision making with respect to clinical management. The most important are: evidence of previous hemorrhage, intranidal aneurysms, venous ectasias, venous stenosis, deep venous drainage, single venous drainage, posterior fossa locations and deep location of the nidus [1,3]. Particular care should be given to note all potential arterial feeders, including meningeal vessels and perforator supply [1]. The nidus morphology (compact vs. diffuse), size, and its relationship to normal brain structures are also important factors in deciding how to treat these lesions [1,4].

Other imaging findings that should be included in the radiology report are secondary effects caused by brain AVMs that may lead to nonhemorrhagic neurologic deficits, such as venous congestion, a long pial course of the draining vein, gliosis, mass effect, hydrocephalus, or arterial steal [3].

A long pial course of the draining vein may interfere with normal brain drainage, increasing the risk of venous congestion and subsequent epilepsy [3].

The arterial steal phenomenon occurs when arterial blood is shunted through the relatively low-resistance arteriovenous fistulae of the AVM and away from the higher-resistance capillary bed in adjacent normal brain, which may cause atrophy and gliosis of brain tissue that is not directly part of the AVM [2]. It has been associated with clinical findings (eg, migraine and focal neurologic symptoms) that are most often transitory in nature [3].
Treatment options: microsurgery, endovascular embolization, radiosurgery or conservative management [1,3].

Factors to consider when evaluating the risk of AVM treatment include AVM's size (< 3 cm, 3-6 cm, or > 6 cm), patient age, AVM rupture status, and nidus diffuseness [1,3].

When feasible, microsurgical resection is the optimal treatment option; endovascular embolization may serve as a crucial adjunct to microsurgery; and radiosurgery may be a viable treatment alternative for inoperable AVMs [1]. Conservative management is typically used when the risk posed by treatment is too high or in asymptomatic patients who are believed to have a low risk of future hemorrhage [3].

1.2 - Arteriovenous Fistula (AVF): In contrast to AVMs, AVFs are direct fistulous connections between cerebral arteries and veins in the absence of an intervening nidus[1,2]. They can be stratified based on arterial supply into dural AVF (meningeal artery-fed) and pial AVF [1].

- **Dural AVF (Figs. 5 and 6):** lesions that comprise arteriovenous fistula(s) situated in the meninges and supplied partly or wholly by dural arteries [1,3,4]. Venous drainage is to dural sinuses and/or leptomeningeal venous channels. They can occur at any dural site, but most frequently involve the cavernous or transverse/sigmoid sinuses. Dural AVFs are more common than pial AVFs, and account for 10%-15% of all intracranial arteriovenous shunts [1,3]. The presenting symptoms and signs of patients with DAVFs, such as bruit, headaches, progressive neurological deficit and hemorrhage, are related to the venous drainage pattern [4]. The simplest way to classify these lesions is to group them into those with cortical venous reflux (malignant fistulas - Borden types 2 and 3) and those without cortical venous reflux (benign fistulas - Borden type 1). Benign fistulas almost never lead to neurologic deficits, whereas malignant dural AVFs often have an aggressive clinical course, including intracranial hemorrhage, seizure, dementia, altered consciousness, and focal nonhemorrhagic neurologic symptoms due to venous congestion or rupture of the venous pouches [3]. Imaging findings: Dural AVFs with cortical venous reflux will manifest with dilated cortical veins, seen as abnormal enhancing tubular structures or flow voids within the cortical sulci with no true nidus within the brain parenchyma [1,4]. Dynamic studies will demonstrate early venous filling, the contribution from external carotid artery branches (rather than pial vessels), and shunt location. Curvilinear subcortical calcifications can be seen at CT in patients with long-standing cortical venous reflux [1]. The dilated vessels of the cortex (veins), meninges or extracranial tissues are more easily identified on MRI, and Gadolinium enhancement will help [4]. However, CT or MRI are likely relatively less sensitive for these fistulae in comparison to AVMs, as many are supplied by small tortuous vessels adjacent to bone. Given the absence of a nidus, their presence may be
obscured, and thus their discovery may not be made until shunting is seen on formal digital subtraction angiography [1]. CT is rarely diagnostic. Catheter angiography is the 'gold-standard' [4]. Following haemorrhage the findings are those of lobar haematoma and may simulate venous infarction with secondary haemorrhage - a search for thrombosis of a major venous sinus is important [4]. Treatment is mandatory for malignant dural AVFs due to their poor natural history if left untreated [3]. Treatment options: microsurgery, endovascular embolization, radiosurgery or conservative management [1].

- **Pial AVF:** constitute true pial arteriovenous shunts and consist of a direct fistulous communication between a pial artery and a vein without any intervening nidus [1,3]. Pial AVFs represent a specific subgroup of brain arteriovenous shunts and account for approximately 5% of all brain AVMs [3]. They are less common than dural AVFs, are generally discovered in a younger patient population (children) and are frequently associated with hereditary hemorrhagic telangiectasia [1,3]. Clues to the diagnosis: the presence of dilated vessels, mainly at the brain surface; and asymmetric dilatation of the pial feeding artery - either the middle, anterior or posterior cerebral artery - which is best seen at the level of the circle of Willis [3]. Pial AVFs are often considered for treatment: microsurgery or embolization [1].

2. **Capillary Telangiectasias** *(Figs 7 to 10 and 20)*: nests of dilated capillaries with interposed normal brain [2,5], without mass effect [1]. Most clinically silent and found incidentally [1,2,5]. They have a predilection for the pons *(Figs 7 and 10)*, spinal cord, and cerebellum but can be anywhere [1,2]. They have been reported in association with cavernous malformations and described as precursors [1,2,4,5], but their radiographic characteristics, however, are distinct [1]. Capillary telangiectasias are typically only visible on MRI, show hypointense or isointense signal on pre-contrast T1-weighted and T2-weighted images with homogeneous enhancement after the administration of contrast and hypointensity on gradient echo and Susceptibility-Weighted sequences [1,4,6,7]. Xanthochromic pigmentation or gliosis is not usually found in the surrounding brain parenchyma [4]. Hemorrhage in association with these lesions should raise the question of an associated cavernous malformation [1,5], which may become symptomatic [5]. Capillary telangiectasias are usually occult to cerebral angiography and CT scan, but faint areas of increased density on CT scans can be seen following contrast administration. No treatment is indicated [1,5].

3. **Cavernous Malformations (CM)** *(Figs. 11 to 15, 19 and 20)*: also referred to as cavernous hemangiomas and cavernomas [1]. They consist of closely packed thin-walled vessels without normal interposed brain parenchyma [1,2,4]. CM may cause seizures, intracranial hemorrhage, focal neurologic deficits, or headaches [1,8]. These lesions were thought to be rare until the advent of MRI and they have since been identified in
0.4% of the population [2]. Occurrence is usually sporadic, but a significant proportion of cavernous angiomas are familial. They can be found in any part of the brain but 80% are supratentorial, with the frontal and temporal lobes most frequent sites, usually in subcortical white matter [2,4]. 50-80% are multiples, with a typical age at presentation between 20 to 40 [2]. Their occurrence has also been found in association with capillary telangiectasis and at sites of previous stereotactic biopsy or radiation therapy [4].

Noncontrast CT may show a small focus of hyperdensity, reflecting intraparenchymal hemorrhage/calcification suggestive of a possible underlying CM (Figs. 11a and 12a), but MRI remains the gold standard in their diagnosis [1, 2].

MRI studies have sensitivity and specificity nearing 100% showing a typical "popcorn-like" lesion (Fig. 11b) with well-delineated complex reticulates core of mixed signal intensities representing hemorrhage in different stages of evolution, due to repeated minor haemorrhages [2,4,9]. A rim of signal loss due to hemosiderin demonstrates prominent blooming on susceptibility weighted sequences (Fig 11c). Zabramski described four types of CM appearance on MRI [1]:

- Type I: lesions are T2-bright due to an acute or subacute hemorrhage.
- Type II: lesions are the typical "popcorn" CMs with mixed hyperintensity and hypointensity on T2-weighted MRI, with a classic surrounding hypointense hemosiderin rim.
- Type III: lesions are T2-hypointense
- Type IV: lesions are only visualized on gradient echo (or susceptibility weighted) sequences (Figs. 12d, 13b, 13c). These are rarely of concern [1].

Gradient echo or susceptibility weighted sequences are able to delineate these lesions better than T1 or T2 weighted images, being very important in identifying the number of lesions missed by conventional spin echo sequences in patients with familial or multiple cavernous angiomas (Fig. 13 and 14) [2,4]. The lesions generally do not enhance, although enhancement is possible [10].

Symptomatic lesions due to either hemorrhage or seizures should be considered for microsurgical resection when possible, once complete resection is curative [1]. CMs are angiographically occult and thus not amenable to any endovascular approaches [1].

4. Venous malformations:

Venous malformations are mainly represented by developmental venous anomalies (DVAs) (Figs. 15 to 20), historically also referred to as venous angiomas or venous anomalies [1]. It is the most common type of intracranial vascular malformation, but may instead represent extreme anatomic variants, being frequently an incidental radiographic
finding [1]. DVAs are often discovered incidentally and although asymptomatic in most cases, they may become symptomatic, with venous ischemia or infarctions if the outflow of the venous collector is compromised [1,3]. Seizure or focal neurologic deficit are less common [2].

DVAs are thickened veins arranged in a radial pattern (described as a "caput medusa"), draining into a common distal vein, with normal brain interposed [2]. This pattern is pathognomonic and may be visible on most MRI sequences, but is most easily seen on postcontrast sequences (Figs. 16 to 20) [2]. The venous collector can be identified as a linear or curvilinear enhancing structure or flow void. The classic angiographic feature is the caput medusae (or "inverse umbrella") appearance of the transmedullary veins (better seen in the venous phase) draining into the venous collector, which in turn drains into either a superficial or a deep venous system. A dense capillary stain can be seen in larger lesions, but arterial phase appears normal (Fig. 20c), with no shunting (differentiating it from brain AVMs), although late capillary blush may be present [2,3]. Non-enhanced CT scans can show an ill-defined slightly hyperdense area (Fig. 16a) and also may reveal an enhancing tuft of rounded or linear enhancing area. Common location: deep cerebral or cerebellar white matter, most often near the margin of the adjacent ventricle [2].

As they drain normal parenchyma, DVAs should not be viewed as treatment targets [1]; however, they may be associated with CM and capillary telangiectasias (Figs. 15, 19 and 20) [1]. Because DVAs rarely bleed (sometimes as a result of thrombosis), if a DVA encountered during investigation appears to be the cause of an intraparenchymal hemorrhage, an associated cavernoma must be sought and can best be seen with gradient-echo sequences [2,3].
Fig. 1: Classic superficial type brain AVM in a 43-year-old woman - Presence of a compact (glomerular type) nidus, seen as tangle of abnormal vessels, which measures 43 x 24 mm, localized in the left lower parietal lobe, shaped like a cone with its base on the cerebral cortex and apex pointing toward the lateral ventricle (a). Note the flow voids and perilesion gliosis on FLAIR image (a); irrigation by branches of the left middle cerebral artery on Time-of-Flight (TOF) - Maximum intensity projection image (MIP) (b) and middle meningeal artery ipsilateral; dilated and tortuous drainage veins on T1 post-contrast - MIP (c), one draining into the superior sagittal sinus and another vein with deep drainage communicating with the left internal cerebral vein.

© HOSPITAL MATER DEI - Belo Horizonte/BR

Fig. 2: An 88-year-old man, presenting generalized tonic-clonic seizures. Post-contrast CT images with MIP reconstructions showing a brain AVM with a compact and calcified nidus (enlarged, tortuous and calcified vessels), irrigated by branches of the left middle cerebral artery (a), with drainage to the ipsilateral transverse sinus (c - sagittal); it is important to register the presence of an intranidal aneurysm (arrow in b).

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 3:** A 24-year-old female patient presenting an AVM with a left-sided pontomesencephalic nidus (a and b) measuring 24 x 19 mm, small intranidal aneurysm of 3 mm (small arrow in a), complex nutrition, and early venous drainage to the vein of Galen and straight sinus - both being seen in the "arterial" phase (c) - compatible with arteriovenous shunt. a: sagittal T1 post-contrast MIP. b and c: axial and sagittal TOF angiography.

© HOSPITAL MATER DEI - Belo Horizonte/BR

**Fig. 4:** A 60-year-old male patient presenting a brain AVM with a left paramedian frontobasal nidus (arrow in a) irrigated by anterior marginal branches of the anterior cerebral artery (a - axial TOF), with early venous drainage to the superior sagittal sinus, which is seen in the "arterial" phase (b - sagittal TOF) due to arteriovenous shunt.
Fig. 5: A 69-year-old man with cervical pain and paresthesia of lower limbs. MRIs showed arteriovenous malformations compatible with dural arteriovenous fistulas (AVFs), one at the level of the tentorium, with a small aneurysm at the shunt location, anterior to the confluence of sinuses (a and b); and another dural AVF within the vertebral canal, at the level of C7 (c and d). Note the dilated veins on the surface of the spinal cord, seen as abnormal enhancing tubular structures (d) or flow voids (c), and the diffuse signal alteration of the spinal cord (c), possibly related to venous reflux. After this diagnostic, the patient was submitted to embolization of the dural fistula, obtaining partial improvement of the symptoms.
**Fig. 6:** TOF angiography with MIP reconstructions of a 55-year-old male patient, showing a vascular malformation suggestive of a dural arteriovenous fistula located at the right skull base. Note the abnormal enhancing tubular structures (arrows in b) with no true nidus, and the early ipsilateral dural venous filling (a and b - right transverse and sigmoid sinus seen in the "arterial" phase) related to the shunt location.

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 7:** Incidental finding of capillary telangiectasias in a 66-year-old female patient. It is located in the pons and presents the classical hyposignal on susceptibility-weighted sequence (a) and enhancement on post-contrast T1-weighted MRI (b).

© HOSPITAL MATER DEI - Belo Horizonte/BR

---

**Fig. 8:** Incidental finding of capillary telangiectasia in a 61-year-old woman MRI, located at the posterior end of the right putamen, presenting typically low signal in susceptibility weighted imaging (a) and demonstrating ill-defined focal enhancement in postcontrast T1-weighted sequences (b).

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 9:** A 58-year-old female patient showing punctate image with accentuated hyposignal on magnetic susceptibility study (a) and contrast medium enhancement (b), located at the lateral extremity of caudate nucleus’ left head, suggestive of capillary telangiectasia.

© HOSPITAL MATER DEI - Belo Horizonte/BR

**Fig. 10:** MRI of a 55-year-old male patient showing capillary telangiectasias in the pons with hyposignal on magnetic susceptibility sequence (a) and faint enhancement after the administration of contrast (b and c).

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 11:** A 23-year-old male patient has presented his first episode of generalized tonic-clonic convulsive crisis and performed CT and MRI to investigate it. CT image showed a small focus of hyperdensity (arrow in a) in the left frontal lobe. MRI evidenced in the same location a lesion compatible with a Cavernoma: with the typical "popcorn" appearance (b - mixed hyperintensity and hypointensity on T2-weighted sequence), with a classic surrounding hypointense hemosiderin rim, blooming on susceptibility weighted sequence (c), spontaneous internal hyperintensity on T1-weighted MRI (d), without enhancement on post-contrast sequence (e).

© HOSPITAL MATER DEI - Belo Horizonte/BR
Fig. 12: A 17-year-old female patient - Noncontrast CT showing a small focus of hyperdensity (a), located in the anterior region of the left cingulate gyrus. MRI showed, in the same location, a rounded focus measuring 10 mm in the largest diameter, with an accentuated hyposignal on magnetic susceptibility study (c and e), permeated by a small hyperintense focus, notably on T2-weighting sequence (b), compatible with cavernous hemangioma. Other focal supratentorial images in both cerebral hemispheres, markedly hypointense on magnetic susceptibility study (d) and undetected on the other pulse sequences, also suggesting small cavernomas.

© HOSPITAL MATER DEI - Belo Horizonte/BR
Fig. 13: A 51-year-old patient with familial multiple cavernous malformation syndrome, presenting multiple cavernomas located bilaterally in the cerebral parenchyma and brainstem. Note that some lesions show the typical "popcorn" appearance, with mixed hyperintensity and hypointensity on FLAIR (a), with a classic surrounding hypointense hemosiderin rim, and other homogeneously hypointense smaller ones are better identified on magnetic susceptibility sequences (b and c). There were no lesions with mass effect and/or significant perilesional edema.

© HOSPITAL MATER DEI - Belo Horizonte/BR

Fig. 14: A Chinese 53-year-old patient with familial multiple cavernous malformation syndrome. MRI demonstrating multiple lesions in cerebral parenchyma (a), cerebellum (b), pons (b) and spinal cord (c), better identified with markedly low signal on susceptibility weighted imaging (a and b).

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 15:** MRI of a 59-year-old man demonstrating a focus of accentuated hyposignal on all pulse sequences, notably on T2-weighted sequences and on magnetic susceptibility study (arrow in a), with subcortical location in the anterior region of the left temporal lobe, compatible with hemangioma cavernous. Note the association with a developmental venous anomaly, visualized mainly on post-contrast T1-weighted sequence (b and c), with drainage to the superficial temporal vein.

© HOSPITAL MATER DEI - Belo Horizonte/BR

**Fig. 16:** A 23-year-old male patient - Non-enhanced CT scan showing an ill-defined slightly hyperdense area (arrow in a) at the base of the left temporal lobe, which on post-contrast T1-weighted sequences (b and c - MIP) represents thickened veins arranged in a radial pattern (caput medusa), with normal brain interposed, draining into an ipsilateral tentorial venous plexus. These findings are pathognomonic of a developmental venous anomaly.

© HOSPITAL MATER DEI - Belo Horizonte/BR
**Fig. 17:** Incidental finding of a developmental venous anomaly in the right frontal lobe of a 37-year-old man - note the caput medusa sign on susceptibility weighted imaging (a), and the venous collector draining into the superior sagittal sinus (b - post-contrast T1-weighted MIP).

© HOSPITAL MATER DEI - Belo Horizonte/BR

**Fig. 18:** A 22-year-old woman presenting a developmental venous anomaly. MRI shows anomaly vein located in the inferior region of left cerebellar hemisphere draining into a collecting vein, which in turn drains into the left transverse sinus. Most easily seen on postcontrast T1 sequences (a, b and c).
**Fig. 19:** A 76-year-old man presenting a left frontoparietal developmental venous anomaly - note the caput medusa sign on susceptibility weighted imaging (a) and on postcontrast T1 MIP (b), draining into a dilated vein which drains into the vein of Galen. Adjacent and associated with the DVA, it is observed a small annular hemorrhagic lesion with markedly low signal on susceptibility weighted imaging (c) compatible with a cavernoma.

© HOSPITAL MATER DEI - Belo Horizonte/BR
Fig. 20: Asymptomatic 12-year-old girl with mixed vascular malformation: DVA - caput medusae sign in left nucleo-capsular region draining into a single vein directed to vein of Galen (a, b - T1 Gd), not seen on TOF (c); Cavernoma associated (d) - "popcorn" appearance and rim of signal loss. There is also a capillary telangiectasias in the pons (e, f).

© HOSPITAL MATER DEI - Belo Horizonte/BR
Conclusion

- It is mandatory for the radiologist the correct and detailed characterization of intracranial vascular malformations, since this is usually the first step in deciding their approach.
- Among the spectrum of intracranial vascular malformations, arteriovenous malformations (AVMs) and cavernous malformations are of particular importance for epilepsy. Seizures are a common mode of presentation for both conditions [11].
- Classic brain AVMs and pial arteriovenous fistulas (AVFs) should be managed according to the risk associated with the disease versus treatment-related risk. Dural AVFs with cortical venous reflux always require treatment [3].
- Capillary telangiectasias and developmental venous anomalies (DVAs) are often incidental findings and may be found in association with cavernous malformations [1].
References


