

Arteriovenous malformations

Arteriovenous malformation (AVM) is a congenital convolute (bundle) of dilated vessels in which arterial blood flows directly into the drains, which lack a normally developed capillary system. They can be congenital (98%) or acquired (2%). These malformations can occur anywhere in the body, but they are the most common in the brain and cause even the most significant problems ^[1]. This article, therefore, focuses on AVM in the **CNS**.

AVM classification

Based on the morphological arrangement, we divide:

1. AVM with **compact** (glomerular) **nid** – typically these are abnormal vessels without the presence of brain tissue, they occur more often than the diffuse type.
2. AVM with **diffuse** (proliferative) **nid** – there is no well-formed nidus with functional neuronal tissue that would be scattered between anomalous vessels.

S-M grading

In the clinic, we encounter a classification known as **S-M grading** according to the authors R. Spetzler and N. Martin. By scoring, the size of the malformation, the type of venous drainage, and whether the AVM is located in a functionally important area of the brain are assessed. According to this classification, we divide AVM into five levels: ^[2]

- **Low-grade** – grade I, II a III (1–3 points),
- **High-grade** – grade IV a V (4–5 points).

Scoring table according to SM grading:: [\[Expand\]](#)

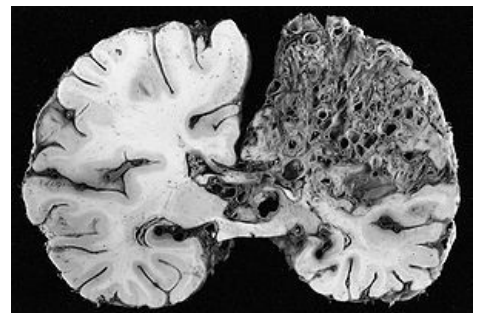
S-M grading is used primarily for surgically resectable AVMs. The SM classification lacks many parameters that are fundamental to deciding the progression of radiosurgical or endovascular treatment. Therefore, other classifications were made within these modalities, for radiosurgery they are, for example, Virginia grading ^[3] or Pollock-Flickinger grading ^[4], Feliciano grading ^[5] and Buffalo score are best known for endovascular treatment . ^[6].

Angioarchitecture

The AVM usually has a confined center (**nidus** – Latin "nest") and dilated supply arteries along with wide drainage veins that are filled with arterialized blood. This type of vascular malformation lacks capillary flow resistance, so the flow is quite high.

Epidemiology

The incidence in the population is relatively low, newly diagnosed cases reach the value of 1-1.5 cases per 100,000 inhabitants. AVM most often manifests itself at a younger age, typically between the ages of 15 and 20. year of life. ^[7]



Arteriovenous malformations filling a substantial part of the parietal lobe

Localization

CNS

In the brain, AVMs are localized, **AVMs are localized** (86 %), of which those in the neocortex, the deeply deposited AVM's (diencephalon, basal ganglia, capsula interna etc.) and combined AVM's archi- and paleocortex lead in incidence. Infratentorial AVMs are uncommon, affecting mainly the neocerebellum and brainstem, and cases of intraventricular AVMs. 80% of spinal AVMs are intramedullary or extramedullary. ^[8]

Hemorrhage from AVM is approximately 30% subarachnoid, 23 % intraparenchymal, 16% intraventricular, and 31% is a combination of these. ^[9]

Other locations

Other areas of AVM include:

1. **lungs**,
2. **liver**,
3. **kidneys**,
4. **uterus and others**.

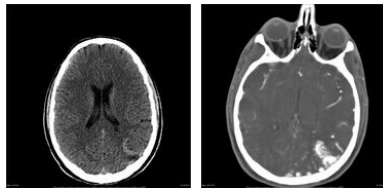
For more details related to AVM in these localizations, see the external links at the end of this article.

Diagnostics

CT

It is often performed as the first imaging method in patients with acute neurological deficits, but without the use of contrast, the accurate diagnosis of AVM is relatively complicated. Nidus AVM without previous bleeding is hyperdense to the surrounding brain tissue. After administration of a contrast agent (especially in CTA), the diagnosis is clearer, but angiography is recommended for more detailed imaging of the arteries. In the case of an AVM rupture, the CT is sensitive enough to image an acute hematoma.

CT - difference in AVM diagnosis when contrast was administered / not



CT without a contrast agent shows a hyperdense lesion (AVM) in the left parietal lobe, in the next image we see the difference in contrast.

CT with contrast - AVM parietal lobe on the left



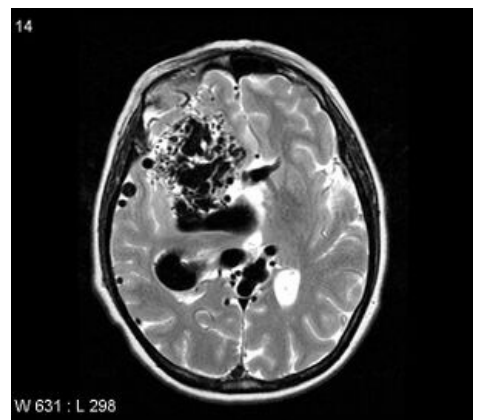
CT without contrast medium - the image shows a large hematoma in the temporo-parietal area on the right, which was caused by the rupture of the AVM.

MRI

MRI is especially important for a clear view of the surrounding structures and their possible damage, and any adjacent edema is well recognizable. Compared to CT, MRI better determines the exact anatomical location of the nidus as well as any previous hemorrhage. ^[10]

MRA

MRA sequences play an important role in displaying three-dimensional angiographic images of AVM. The TOF MRA method is typically one of the first examinations of a given malformation, showing very well most supply arteries and nidus lesions. However, it is often complicated to distinguish supply arteries from venous drainage, especially in AVMs with a more complex arrangement. In addition, it is not very sensitive to smaller AVMs and associated aneurysms. ^{[11][12]}



MRI - cerebral AVM

Angiography - DSA

Digital subtraction angiography (DSA) is the **gold standard for detailed AVM** diagnostics. This malformation appears on angiography as tightly packed masses of enlarged surrounding arteries that supply the nidus of the malformation. The DSA will provide a detailed description of the supply arteries and associated angiopathic changes, an accurate evaluation of the nidus (its size, hemodynamic properties, anatomical characteristics), and the definition of venous drainage (deep or superficial), as well as any signs of stenotic changes or ectasia. If hemorrhage is noted in the malformation, the hematoma typically compresses the nidus, increasing intranidal vascular resistance. The result can be a "delay" in venous outflow and AVM can become **angiographically occult** - the malformation is anatomically present but does not appear on the angiography. ^[13]

Associated abnormalities

The most commonly observed abnormality associated with AVM is **aneurysms**, which typically result from increased blood flow through malformations. They can occur **outside the nid** (intranid - present in up to 41-100% of patients with AVM) or on the **supply vessels** (intrapedicular). Alternatively, non-AVM aneurysms may appear as part of the diagnostic test. appear as part of the diagnostic test.

Furthermore, flow **angiopathy** is relatively common secondary to endothelial hyperplasia.

Symptomatology

The presentation of these vascular malformations is very diverse. Some AVMs may be **asymptomatic** for a long time , and some patients may have posthemorrhagic **neurological deficits**, **headaches** or **seizures**. **Heart murmurs** have been reported in some patients.

We often encounter the so-called **steal syndrome** - - the arteries supplying the AVM "steal" around the vessel, which can result in ischemia of areas whose supply has been redirected to the AVM.

Therapy

AVM therapy is a prime example of multimodal cooperation in several branches of medicine. AVM can be surgically resected, irradiated radiosurgically, or embolized endovascularly. Often, multiple treatment alternatives can be applied to a single patient to achieve the most appropriate outcome with the lowest possible morbidity. Of course, observation is a legitimate procedure if the risk of rupture is not very high and the malformation itself does not cause serious problems for the patient.

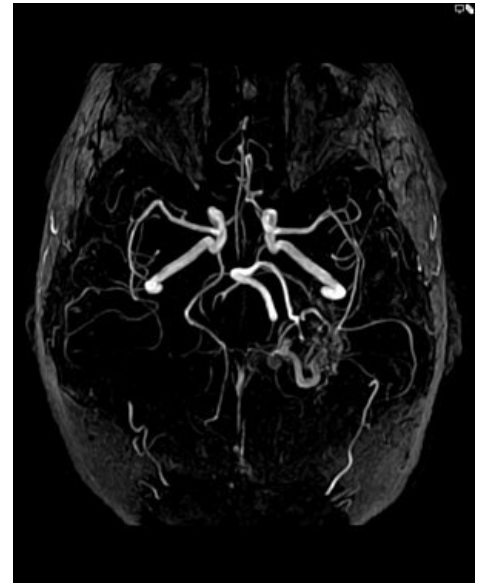
1. **Surgical resection** - especially for smaller and superficially stored malformations, the advantage is the immediate elimination of the circulation and thus definitive treatment, complete resection is necessary, and thanks to resection, possible elimination of epileptic focus.
2. **gamma knife** radiosurgery - usable for AVM nidus up to 2.5-3 cm in diameter, for deep-seated lesions, the advantage is a non-invasive technique, the disadvantage is the gradual closure of the malformation for 1-3 years (during this time the patient is at risk of bleeding from AVM).
3. AVM endovascular **embolization** - rarely used alone, the goal is usually to close the superficial part of the nid and the main supply arteries.

Complications

In the case of **observation**, the biggest complication is bleeding. The risk of haemorrhage in patients without previous bleeding is approximately 2-3% %^[14]. Hemorrhage from AVM (unlike cavernous malformations), can have fatal consequences due to its morphological arrangement - a post-hemorrhagic 30% mortality and a 10-20% probability of persistent neurological deficits are reported in the literature^[15]. V In some cases, cerebral ischemia, can occur, with AVM ruptures representing approximately 1-2% of all cerebral ischemias. The presence of aneurysms is also common in the area of the nidum (or on the supply vessels), which tend to rupture due to the rate of blood flow through the malformations.

Surgical resection is difficult, often several hours, but has very good results (with radical resection) with regard to the long-term prognosis of the patients. The goal of resection is to achieve a complete resection that automatically provides the patient with zero risk of future bleeding from a given AVM. Some morbidity may occur postoperatively, but in most patients it resolves over time - only a fraction (approximately 7% on average) of patients have persistent morbidity^[15].

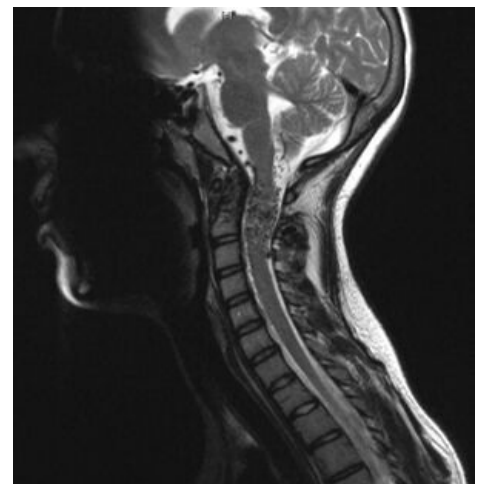
Patients suitable for **radiosurgery** should not be at high risk of hemorrhage - the effect of treatment does not appear immediately after irradiation, so the patient is still at risk of bleeding from AVM for a relatively long time. It is possible to use the AVM first and then operate - experience with this procedure varies, some workplaces operate after radiosurgery only if the irradiation was not effective enough, and others help radiosurgery before surgical resections because malformations usually do not bleed so much after irradiation, etc.



TOF MRA showing cerebral AVM



Angiogram showing AVM



MRI - spinal AVM

The most common complication of **endovascular embolization** is again hemorrhage (arterial perforation may occur during microcatheter insertion or AVM rupture, etc.). Furthermore, there is a risk of thrombus formation and migration to the surrounding cerebral areas, which can result in ischemia.

Video library

ARTERIOVENOUS MALFORMATION (AVM)

* can happen anywhere *

↳ MOST COMMON: Brain, Spinal cord, lungs



Video in English - General about AVM

Links

Related articles

- Hemorrhagic strokes
- Cavernous malformation
- Bleeding into the internal capsule
- Treatment of intracranial aneurysm
- Subarachnoid hemorrhage
- Intracerebral hemorrhage
- Intracranial hypertension
- Brain ischemia
- Bleeding
- Gamma knife

External links

- Cerebral AVM (<https://radiopaedia.org/articles/brain-arteriovenous-malformation>)
- Classification of spinal AVM (<https://radiopaedia.org/articles/spinal-avm-classification?lang=us>)
- Spinal AVM (<https://radiopaedia.org/articles/spinal-arteriovenous-malformations?lang=us>)
- Pulmonary AVM (<https://radiopaedia.org/articles/pulmonary-arteriovenous-malformation?lang=us>)
- Renal AVM (<https://radiopaedia.org/articles/renal-arteriovenous-malformation?lang=us>)
- Michael T. Lawton - Lecture on AVM surgical treatment (https://www.youtube.com/watch?v=Bg_GFWBsZnI)

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