

TEACHING COURSE – ARTERIOVENOUS MALFORMATIONS

NEUROSURGICAL TREATMENT OF CEREBRAL ARTERIOVENOUS MALFORMATIONS

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Introduction

Despite tremendous developments in imaging, surgical techniques and intensive care management the microsurgical resection of cerebral arteriovenous malformations (AVMs) can still be a great challenge even to experienced neurosurgeons. Radiosurgery, endovascular embolisation and surgery are the treatment options with surgery being the treatment of choice.

Epidemiology and Clinical Presentation

The prevalence is calculated 0.2-0.5%/100.000 although lower numbers are discussed (0.01%) Men are slightly more affected than women (1.4:1) [1]. Most patients present with the neurological sequelae of intracranial haemorrhage caused by the AVM (50%), which is a major cause of intracerebral bleeding in younger adults. In 25% patients present with epileptic seizures and in 25% with other symptoms including neurological deficits not related to haemorrhage, headaches and neuropsychological disturbances including e.g. memory deficits and learning disabilities. The annual risk of spontaneous intracerebral bleeding of an AVM has been reported to be 2-4% per year leading to a mortality of 10-15% and a permanent morbidity of 30-50%. The risk of rebleeding is increased up to 18% within the first year after the first bleeding then declining again [4]. AVMs with a high intranidal pressure (highflow feeder or obstructed veins), only one draining vein or AVM associated aneurysms have a higher risk of bleeding. Whether small deep seated

AVMs carry also a higher risk is still a matter of debate. AVMs are generally categorized using the Spetzler-Martin grading scale which is built on size of the nidus, the location of the aneurysm and the type of venous drainage [7].

Imaging

Preoperative evaluation usually includes MRI and angiography of the cerebral vessels. Depending on the location of the AVM close to or in eloquent areas additional imaging including functional MRI or fibre tracking are helpful in planning surgery. Blood flow can be assessed using PET and diffusion/perfusion MRI. The data can be transferred to neuronavigation systems which are extremely helpful in localising these areas during surgery. In the acute setting with haemorrhage computed tomography is still the primary technique of examination. In suspicion of an AVM it can be immediately followed by a CT-angiography which may show the nidus or the draining vein. If no emergency surgery is required cerebral digital subtraction angiography (DSA) can be planned. In some cases DSA can be negative when the nidus is compressed by the haematoma. Then angiography should be repeated after some time delay. MRI can be performed in the subacute stage or even later when blood has resorbed. MRI provides a good anatomical analysis of the lesion and the surrounding brain tissue. DSA is still considered the gold standard to establish the diagnosis and is used in the work-up to assess angioarchitectural and blood flow before treatment.

Treatment Modalities

The risk of bleeding persists until the nidus of the AVM has been excluded from circulation which means complete angiographic obliteration of the AVM. However, despite modern techniques some AVMs are considered incurable with the risk of treatment being higher than the risk of bleeding during the natural course [3]. The treatment modalities include radiosurgery, surgery and endovascular occlusion. All three treatment modalities have an established position in the treatment of patients with arteriovenous malformations. The final decision which treatment should be chosen depends on age of the patient, neurological status, associated clinical risk factors, and size and angioarchitectural features of the lesion. In some cases combined treatments are useful and effective. The big advantage of surgical resection when performed completely is the fact that there is no more risk of rebleeding and that there is no recurrence. Radiosurgery offers an alternative to surgery in smaller AVMs up to 3cm but has the disadvantage that complete occlusion occurs in only up to 80% of the treated cases and takes up to several months, which exposes the patient with an already ruptured AVM to the risk of rebleeding during that time, which would not be the case in surgery.

Surgery

Microsurgical resection is the treatment of choice in AVMs; however, not all AVMs can and should be treated by surgery. In some cases endovascular or radiosurgical pretreatment may be helpful. In smaller AVMs (Spetzler-Martin Grade I to III) complete resection in 98 to 100% with a low morbidity and mortality (0-0.5%) has been reported [5]. Permanent neurological deficit is somewhat higher in AVM surgery in eloquent areas (5-6%) compared to non-eloquent areas (0-1%) but is still acceptable. However, results are worse in patients with high-grade arteriovenous malformations (Grade IV and V). Combined mortality and morbidity rate in this group occurs in up to 22% of the patients [2]. Therefore partial treatment or surgery should only be performed in selected cases with progressive neurological deficit as annual bleeding rate is lower (1.5%) than previously suspected and higher in partially obliterated AVMs (10.5%) [3]. Relevant risk factors associated with postoperative morbidity

are Spetzler-Martin grade of the AVM, eloquent area, nidus size, and presence of deep venous drainage [6]. There is also a delayed postoperative deficit described in patients who had an initially uneventful postoperative period. Most of these patients had hypertension causing intracerebral haemorrhage after resection of a large arteriovenous malformations, or vasospasm.

Conclusion

Surgery should be considered as the first choice treatment in patients after bleeding of the less critical AVMs Spetzler-Martin Grade I-III. In patients with higher surgical risks or Grade IV or V AVMs alternative or multimodal treatment should be considered. Although the risk of bleeding in incidental AVMs may be lower than previously thought treatment should be considered in younger people in order to prevent the risk of neurological deficit caused by haemorrhage or perfusion deficits.

References

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