#### HOW I DO IT



# How I do it: surgery for spinal arteriovenous malformations

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## Abstract

**Background** Spinal arteriovenous malformations (AVM) are rare lesions. They may present with intramedullary hemorrhage or edema, often inducing severe neurological deficits. Active treatment of spinal AVMs is challenging even for experienced neurosurgeons.

**Method** Anticipation of anatomy and AVM angiocharacteristics from preoperative imaging is key for successful treatment. Information gathered from MRI and DSA has to be then matched to intraoperative findings. This is a prerequisite for reasonably safe and structured lesion removal.

**Conclusion** We provide a structured approach for surgical treatment of spinal AVMs, supplemented by high-resolution video and imaging material.

Keywords Arteriovenous malformation · AVM · Spinal · Multimodality · Treatment · Exoscope

#### Abbreviations

ASA	Anterior spinal artery
AVM	Arteriovenous malformation
CSF	Cerebrospinal fluid
EVT	Endovascular treatment
ICG	Indocyanine green
LMWH	Low molecular weight heparin
PSA	Posterior spinal arteries

# **Relevant surgical anatomy**

The spinal cord receives segmental blood supply on multiple levels via radiculomedullary arteries, feeding the anterior (ASA) and two posterior spinal arteries (PSA) [1]. Number and course of radiculomedullary arteries in the thoracic spine, including the Adamkiewicz artery, are highly variable. Small

This study has not been presented previously.

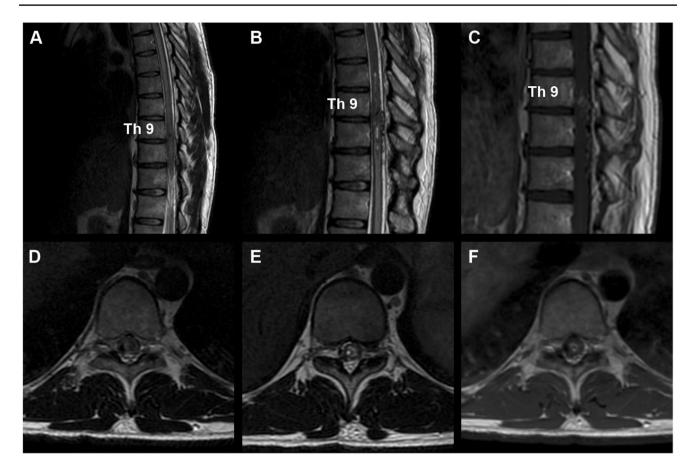
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arteries may not be visualized on DSA [8]. ASA and PSA are connected via anastomoses surrounding and traversing the spinal cord [1]; however, there is little tolerance to ischemia in the thoracic spinal cord [8]. As such, great care must be taken when occluding arteries during AVM treatment. While larger spinal AVM feeders are thought to almost solely supply the nidus, smaller vessels may also supply the medulla [1]. According to recent literature, superficial AVM niduses are mainly fed by primary branches of the ASA and PSA, while transmedullary feeders were only found in few cases [7]. Spinal AVMs are rare compared to other spinal arteriovenous lesions. They are located in the thoracic spine in the majority of cases [3].

The AVM demonstrated in Figs. 1, 2, and 3, and the video accompanying this manuscript (Online Resource 1) had the majority of its nidus volume located superficially at the pial level of the spinal cord. It received arterial supply from Th8-11 segmental arteries; venous drainage was found mainly along the right Th11 root. Based on MRI, intramedullary vasculature of the nidus was also present. The AVM presented with intramedullary hemorrhage, causing edema and severe neurological deficits.

Several classification systems for spinal arteriovenous lesions exist [4, 7]. They differ fundamentally and have been a matter of considerable debate [5], their discussion is beyond the scope of this text. Being based on intraoperative findings, imaging, and clinical presentation [9], as well as angioarchitecture and histogenetic location [7], their review may be helpful for understanding these lesions.

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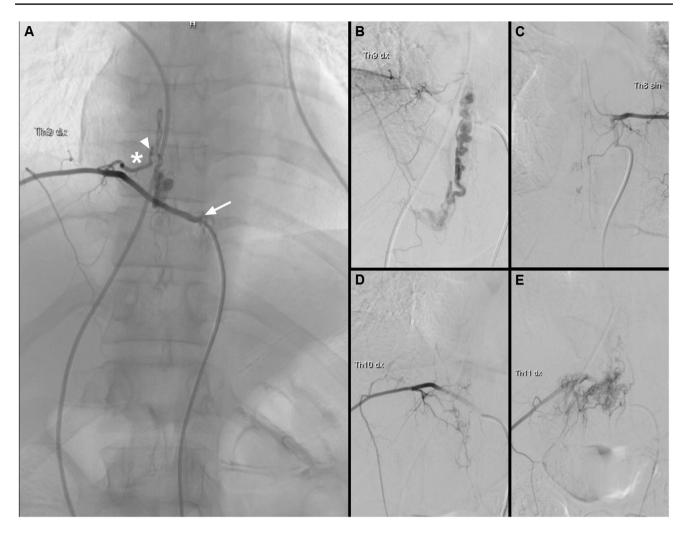
**Fig. 1** Initial MRI examination of the thoracic spine. **A**, **B** Sagittal T2 images showing the large craniocaudal extension of medullary, increased vascularity and the cavity formed by the intramedullary hemorrhage. **C** Enlarged view of nidus and hemorrhage cavity in the

gadolinium-enhanced T1 sequence. **D**, **E** Axial T2 at different levels of the nidus. **F** Axial gadolinium-enhanced T1 images at the height of the intramedullary hemorrhage cavity

# **Description of the technique**

In the upper and mid thoracic spine, we use the angiosuite to identify the correct spinal level preoperatively. With the patient awake in prone position, methylene blue dye is injected on the top surface of the correct lamina under DSA-view. However, any other method one relies on is appropriate. After anesthesia, patients are then put in genupectoral (kneeling) position, as shown in the supplemental video. A standard midline approach to the spine is followed by en bloc laminectomy of the desired levels using the craniotome, for later re-insertion with titanium plates. After dural opening the site is inspected and an initial indocyanine-green (ICG)-angiography is used to clarify the angioarchitecture. Understanding of the AVM architecture, translation of DSA images to the surgical site and identification of the feeding and draining vessels is the key step of the surgery. Enough time should be taken to understand what is part of the lesion and which vessels need to be preserved.

The resection of the spinal AVM follows the same principles as in brain AVM, dissection goes along the nidal borderline without entering the lesion. Initially, a clip is placed on the main feeder, which reduces the risk of intraoperative rupture and softens the lesion during dissection. Tissue planes between the nidus and the medulla are separated by blunt and sharp dissection. Further ICG-angiographies assess residual inflow from the other feeders that are then subsequently coagulated and cut. Disconnection is continued until the complete nidus can be removed. Preservation of neurological function is paramount, and any resection of parenchyma is avoided. Hemostasis is achieved, keeping in mind that tenacious bleedings indicate residual nidus. The final ICG-angiography confirms full resection. Surgery is finalized by watertight dural closure, reinsertion, and fixation of the laminae and wound closure in layers.



**Fig. 2** Preoperative DSA of the spinal AVM fed by segmental arteries from Th8 to Th11, draining mainly along the right Th11 root. **A** shows an overlay of the early arterial phase after injection of the right Th9 segmental artery and the unsubtracted ap view outlining the bony landmarks (55% opacity). **B** Late arterial phase of the same

## Indications

If discovered as an incidental finding, we consider if the lesion should be treated surgically at all, based on feasibility and estimated risk of treatment. In case of rupture or symptoms related to steal or congestion, treatment is always pursued. Others recommend treatment for any discovered spinal vascular malformation, as even partial obliteration of intramedullary AVM may improve the lesions' course [1, 3].

Timing of treatment after hemorrhage is a matter of debate [1]; however, an estimated annual re-hemorrhage rate of 10% should be kept in mind [3]. About one-third of spinal AVM may have associated aneurysms [1], promoting treatment.

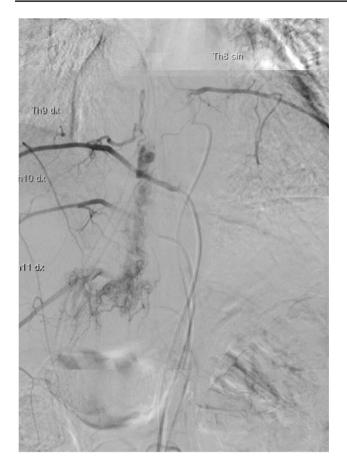
An in-depth analysis of indications for radiotherapy or endovascular treatment (EVT) is beyond the scope of this manuscript. In brief, the rate of complete obliteration by EVT for spinal AVM

injection. **C** Angiogram of left Th8 segmental artery. **D** Right Th10 segmental artery. **E** Right Th11 segmental artery. Long arrow denotes the origin of right Th9 segmental artery from the aorta, asterisk marks the right Th9 vertebral pedicle, arrowhead denotes dural entry of feeding artery. Abbreviations: AVM = arteriovenous malformation

is low, but neurological improvement following EVT has been reported [2]. Radiotherapy is established in brain AVMs, but little is known about its role in spinal AVMs. A recent systematic review [10] only identified 64 patients undergoing radiotherapy for spinal AVM, of which 63% received radiotherapy as an adjunct. Nidus obliteration was found in only 16% of cases, whereas 39% showed no change of the malformation on follow-up.

#### Limitations

Lesions located mainly posterior or lateral to the spinal cord and are partly exophytic (extramedullary) should usually be approachable. Lesions anterior to the spinal cord are difficult to access and manipulation or damage of the ASA may cause bad outcomes.



**Fig. 3** Multiple image overlay of preoperative DSA, to better appreciate the full AVM nidus. Artwork was created using Photoshop CC 2019 (Adobe, Inc.). Five subtracted DSA images (as used in Fig. 2A–E) are stacked above each other. Images are aligned according to unsubtracted views of bony skeleton, order from bottom to top: 2B (100% opacity), 2A (70%), 2D (40%), 2E (40%), 2C (20%)

Apart from the AVM angioarchitecture, it may be assumed that surgical experience represents a major limitation. Expertise on intramedullary spinal tumors, brain AVMs and general cerebrovascular neurosurgery appear as a prerequisite.

#### How to avoid complications

Surgery should start with correct identification of spinal segments and exposure of the whole lesion.

Identification of feeding and draining vessels based on preoperative DSA and ICG-angiography are paramount to safely shut down and remove the AVM. Failure to do so may cause intraoperative rupture.

High levels of magnification should be used when dissecting around the vasculature, preserving small vessels for medullary supply. As in any intramedullary procedure, the use of neuromonitoring (SEP, MEP, D-wave) is advisable, although not based on scientific evidence [1].

Watertight dural closure should be achieved, using additional sealants where needed.

We try to avoid laminectomies in treatment of intradural pathologies using hemilaminectomies or laminoplasty instead. The latter provides a protective landmark in case of revision and may reduce the risk of cerebrospinal fluid (CSF) leaks, while it is not thought to prevent spinal deformity [6].

#### Specific perioperative considerations

Based on our institutional experience, we keep patients normotensive after surgery. Low molecular weight heparin (LMWH) is administered in the perioperative setting only for standard deep vein thrombosis prophylaxis where indicated. Patients are mobilized early after treatment and neurorehabilitation is emphasized.

LMWH is only continued if there is any high-grade paresis; we otherwise do not administer heparin after surgical treatment.

We do not advocate prolonged bed rest after spinal intradural procedures to prevent CSF leaks.

We perform routine DSA before and after surgical treatment; a MRI is performed after 3 to 6 months to assess any spinal cord edema or damage from hemorrhage.

#### Specific information for the patient

Patients need to be aware that unchanged neurological function will be the best possible outcome immediately after surgery. Preexisting deficits may be initially worsened by the surgery, and symptoms may or may not resolve on the long run. It may take 1 to 2 years to evaluate the final clinical outcome.

Multiple available treatment modalities, or their combination, have to be discussed. The multimodality concept should be reappraised if complete occlusion has not been achieved by the planned treatment.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00701-023-05598-3.

#### **Key points**

 If a vascular malformation of the spinal cord is suspected, at least MRI and DSA should be performed to confirm the diagnosis.
The key to treatment is to understand preoperative MRI and DSA images outlining the anatomy and angioarchitecture of the AVM.
Based on images, neurological exam and patient characteristics, decide whether to treat and if surgery is the correct modality. 4. After the dura has been opened, the first step is to find and recognize pre-identified angiographical structures in situ, by inspection and ICG-angiography.

5. Take sufficient time to understand the anatomy: What is part of the nidus and needs to be taken out and what is not part of the pathology and needs to be preserved?

6. Take down major feeders first to reduce the bleeding risk and ease AVM dissection, then subsequently close other feeders.

7. Dural closure should be watertight, laminoplasty may be preferable to laminectomy.

8. Start mobilization and physiotherapy immediately postoperatively, followed by neurorehabilitation.

9. Preexisting neurological deficits may be (transiently) worsened after surgery and only long-term follow-up will show the final result of the treatment.

10. We advocate performing DSA after surgery and MRI after 3 to 6 months, to verify AVM obliteration and regression of edema.

Author contributions All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by Tobias Rossmann, Michael Veldeman, and Rahul Raj. The first draft of the manuscript was written by Tobias Rossmann, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

#### Declarations

**Ethics approval** The research project was conducted following the principles outlined in the Declaration of Helsinki. Anonymized presentation of a single case does not require an ethics approval by the local university hospital review board.

**Informed consent** The patient provided written informed consent for the use of imaging data and operative video.

Conflict of interest The authors declare no competing interests.

## References

1. Brinjikji W, Lanzino G (2017) Endovascular treatment of spinal arteriovenous malformations. Handb Clin Neurol 143:161–174

- Ehresman J, Catapano JS, Baranoski JF, Jadhav AP, Ducruet AF, Albuquerque FC (2022) Treatment of spinal arteriovenous malformation and fistula. Neurosurg Clin N Am 33(2):193–206
- 3. Gross BA, Du R (2013) Spinal glomus (type II) arteriovenous malformations: a pooled analysis of hemorrhage risk and results of intervention. Neurosurgery 72(1):25–32
- Kim LJ, Spetzler RF (2006) Classification and surgical management of spinal arteriovenous lesions: arteriovenous fistulae and arteriovenous malformations. Neurosurgery 59(5 Suppl 3):S195–S201
- Lasjaunias P (2003) Spinal cord vascular lesions. J Neurosurg 98(1 Suppl):117–119
- McGirt MJ, Garcés-Ambrossi GL, Parker SL, Sciubba DM, Bydon A, Wolinksy J-P, Gokaslan ZL, Jallo G, Witham TF (2010) Shortterm progressive spinal deformity following laminoplasty versus laminectomy for resection of intradural spinal tumors: analysis of 238 patients. Neurosurgery 66(5):1005–1012
- Mizutani K, Consoli A, Maria FD, Condette Auliac S, Boulin A, Coskun O, Gratieux J, Rodesch G (2021) Intradural spinal cord arteriovenous shunts in a personal series of 210 patients: novel classification with emphasis on anatomical disposition and angioarchitectonic distribution, related to spinal cord histogenetic units. J Neurosurg Spine 2:1–11. https://doi.org/10.3171/2020.9. SPINE201258
- Santillan A, Nacarino V, Greenberg E, Riina HA, Gobin YP, Patsalides A (2012) Vascular anatomy of the spinal cord. J Neurointerv Surg 4(1):67–74
- Spetzler RF, Detwiler PW, Riina HA, Porter RW (2002) Modified classification of spinal cord vascular lesions. J Neurosurg 96(2 Suppl):145–156
- Zhan PL, Jahromi BS, Kruser TJ, Potts MB (2019) Stereotactic radiosurgery and fractionated radiotherapy for spinal arteriovenous malformations - a systematic review of the literature. J Clin Neurosci 62:83–87

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