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REVIEW

Endovascular management of spinal vascular malformations

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Abstract Spinal vascular malformations are rare diseases with a wide variety of neurological presentations. In this article, arteriovenous malformations (both from the fistulous and glomerular type) and spinal dural arteriovenous fistulae are described and an overview about their imaging features on magnetic resonance imaging (MRI) and digital subtraction angiography is given. Clinical differential diagnoses, the neurological symptomatology and the potential therapeutic approaches of these diseases which vary depending on the underlying pathology are given. Although MRI constitutes the diagnostic modality of first choice in suspected spinal vascular malformation, a definite diagnosis of the disease and therefore the choice of suited therapeutic approach rests on selective spinal angiography. Treatment in symptomatic patients offers an improvement in the prognosis. In most spinal vascular malformations, the endovascular approach is the method of first choice; in selected cases, a combined or surgical therapy may be considered.

Keywords Spinal vascular malformations · Spinal AVM · Spinal dural AV fistula

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Introduction

Spinal vascular malformations are rare and still underdiagnosed entities that, if not treated properly, can lead to considerable morbidity with progressive spinal cord symptoms. Their clinical diagnosis rests mainly on magnetic resonance imaging (MRI), whilst, for a thorough understanding of the diseases and for planning the therapeutic strategy, selective spinal subtraction angiography (DSA) still is necessary. Depending on the type of spinal vascular lesion, initial symptoms may vary between acute or chronic onset. Pathophysiologic mechanisms include intramedullary or subarachnoidal haemorrhages or subacute venous congestion leading to progressive myelopathy. The space-occupying nature of some of these lesions and circulatory "steal" phenomena are additional possible pathophysiologic mechanisms. Whilst acute manifestations of spinal vascular malformations typically lead to a diagnosis early in the course of the disease, the subacute venous congestion might lead to unspecific neurological

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symptoms which in turn delays proper diagnosis. The aim of the following article is to review the imaging features, clinical symptomatology and potential endovascular therapeutic approaches of spinal vascular malformations.

Since an understanding of spinal vascular malformations both from an aetiological and from a pathophysiological standpoint is based on the spinal vascular anatomy, we will briefly describe the salient features of spine and spinal cord arterial supply and venous drainage in the following section.

Spinal vascular anatomy

Segmental arteries (i.e., segmental feeders from the vertebral arteries, the deep and ascending cervical arteries for the cervical levels; intercostal or lumbar arteries at the thoracic and lumbar levels; and, for the sacral levels, the iliolumbar arteries) supply the spine (including the vertebral bodies, paraspinal muscles, dura and nerve roots) and the spinal cord with blood. The bony spine is supplied by anterior and posterior central arteries that arise directly from the segmental and radicular arteries. A spinal radicular branch supplying the dura and the nerve root as a radiculomeningeal artery is present at each segment. From these radicular arteries, radiculomedullary and/or radiculopial arteries might branch, following the anterior or posterior nerve root to reach the anterior or posterior surface of the cord where they form the anterior or posterior spinal artery [33]. In the adult patient, not all lumbar or intercostal arteries have a radiculomedullary or radiculopial feeder and their location for a given patient is not predictable. The anterior and posterior spinal arteries constitute a superficial longitudinal anastomosing system [24]. The anterior spinal artery travels along the anterior sulcus and typically originates from the two vertebral arteries, whilst the typically paired posterolateral spinal arteries originate from the preatlantal part of the vertebral artery or from the postero-inferior cerebellar artery. These three arteries run from the cervical spine to the conus medullaris but are not capable of feeding the entire spinal cord. Instead, they are reinforced from the above-mentioned anterior radiculomedullary and posterior radiculopial arteries that derive from various (and unpredictable!) segmental levels [13]. The most well known of the anterior radiculomedullary arteries is the artery radiculomedullaris magna (i.e. the Adamkiewicz artery). The anterior radiculomedullary arteries branch in a very typical way to reach the spinal cord. The ascending branch continues along the direction of the radicular artery in the midline of the anterior surface. The descending branch, being the larger one at thoracolumbar levels, forms a hairpin curve as soon as it reaches the midline at the entrance of the anterior fissure [33].

The intrinsic network of the spinal cord arteries can be divided in central or sulcal arteries from the anterior spinal artery on the one hand and, on the other hand, in the rami perforantes of the vasocorona that supplies the periphery of the spinal cord and is derived both from the anterior and from the paired posterolateral arteries [24].

The venous drainage of the cord is via radially symmetric intrinsic spinal cord veins and small superficial pial veins that open into the superficial longitudinal median anastomosing spinal cord veins. These veins are following more or less the arteries, the anterior and posterior median spinal vein, but have many anastomoses (including transmedullary anastomoses) creating a network with commonly more than one anterior and posterior vein [13]. They also are using the roots as a vehicle to reach the epidural plexus and the extraspinal veins and plexus with a reflux-impeding mechanism within the dura mater [36]. It is important to note that the transition of a median vein into a radicular vein shows the same hairpin shape as the artery. At the superior cervical part, they can run through the occipital foramen to connect the vertebral plexus the inferior dural sinus. Drainage of blood from the spine occurs through the valveless internal and external venous vertebral plexus that is connected to the azygos and hemiazygos venous systems.

Classification of spinal vascular malformations

Multiple different classification schemes have been proposed for spinal vascular malformations. Recently, the Bicetre group classified spinal cord arteriovenous malformations (AVM) into three main groups [28]: (1) genetic hereditary lesions that are caused by a genetic disorder affecting the vascular germinal cells. Spinal cord malformations associated to hereditary hemorrhagic telangiectasia fall into this category. (2) Genetic non-hereditary lesions that share metameric links such as the Cobb syndrome (or spinal arteriovenous metameric syndrome) that affects the whole myelomere. These patients typically present with multiple shunts of the spinal cord, the nerve root, bone, paraspinal, subcutaneous and skin tissues. Klippel–Trenaunay and Parkes–Weber syndromes also belong to this group. (3) Single lesions that may reflect the incomplete expression of one of the previous mentioned situations and that include spinal cord, nerve root and filum terminale lesions.

Since the majority of spinal vascular malformations fall into the last group, in the following, we use a classification that is based on the vascular anatomy of the spinal cord as described above [22]. According to this classification, spinal vascular shunting malformations can be differentiated, similar to vascular malformations of the brain, into pial

and dural arteriovenous shunting lesions depending on the vessels feeding the shunt. Spinal cord AVMs are like their cerebral counterparts shunts that are fed by arteries normally supplying the neural tissue, i.e. the intrinsic arteries of the spinal cord, whereas spinal cord dural arteriovenous (AV) fistulae (like their cranial counterparts, the dural AV fistulae) are fed by radiculomeningeal arteries (which are in fact similar to meningeal arteries) [35].

Dural AV fistulae

Spinal dural arteriovenous fistulae (SDAVF) are the most frequent vascular malformation of the spine and account for 70% of all AV shunts of the spine [35]. They are presumably acquired lesions; however, the exact aetiology is not known. Usually, the disease becomes symptomatic in elderly men (between 40 and 60 years) [34]. Most fistulae are found in the thoracolumbar region.

The arteriovenous shunt is located inside the dura mater close to the spinal nerve root where the arterial blood from a radiculomeningeal artery (i.e. an artery supplying the root and meninges but not necessarily the spinal cord!) enters a radicular vein where the latter passes the dura. The increase in spinal venous pressure diminishes the arteriovenous pressure gradient and leads to a decreased drainage of normal spinal veins and a venous congestion with intramedullary oedema [7, 17]. This in turn leads to chronic hypoxia and progressive myelopathy [6]. Clinical symptomatology of this congestive myelopathy is rather unspecific and might consist of hypo-esthesia and paraesthesias, paraparesis, back pain that might irradiate to the lower legs, impotence and sphincter disturbances. Usually, the deficits are slowly progressive; however, an acute onset of the disease and a progressive development interrupted by intermediate remissions is also possible [19, 35]. Without therapy, this increase results in irreversible para- or even tetra-plegia.

On MRI, the combination of cord oedema and perimedullary dilated vessels is the characteristic finding and should lead to the diagnosis [19]. On T2-weighted sequences, the cord oedema is depicted as a centromedullary and not well-delineated hyper-intensity over multiple segments that is often accompanied by a hypo-intense rim, most likely representing deoxygenated blood within the dilated capillary vessels surrounding the congestive oedema [16]. The cord is swollen and might demonstrate contrast enhancement as a sign for chronic venous congestion [8] (Fig. 1). In the further course of the disease, the cord will get atrophic. The peri-medullary vessels are dilated and coiled and can be observed on the T2-weighted images as flow voids. However, if the shunt volume is small, they might only be seen after contrast enhancement. Neither the location of pathological vessels nor the intra-medullary imaging findings seem to be related to the height of the fistula. Localisation of the fistula can sometimes be very difficult leading to lengthy and even multiple catheterisation procedures during spinal DSA. Therefore, non-invasive diagnostic techniques, such as contrast-enhanced magnetic resonance angiography (MRA) with relative fast acquisition protocols, have been developed [24–26].

There are two options in the treatment of SDAVF: surgical occlusion of the intra-dural vein that received the blood from the shunt zone—a relatively simple and safe intervention with exception of sacral fistulae [15, 25]—or endovascular therapy employing glue after superselective catheterisation of the feeding radiculomeningeal artery [27, 30]. The embolic agent must pass the nidus and reach and occlude the proximal segment of the draining vein in order to prevent subsequent intra-dural collateral filling of the fistula (Fig. 2). Therefore, success rates of endovascular therapy have been reported to vary between 25% and 75% [27, 38] whereas a recent meta-analysis suggests complete occlusion of the fistula following surgery in 98% [32]. If the glue stays intra-arterial and does not reach the venous site, we strongly advocate early surgical intervention since a recent study has shown that patients in whom the endovascular occlusion was incomplete and that required surgical intervention had a bad clinical outcome which was likely due to the delay for the secondary intervention [3]. The treatment strategy that is adopted by most centers nowadays includes a tentative of embolisation if this was felt to be a safe approach (i.e. no spinal cord supplying artery that may arise from the same pedicle as the feeder to shunt). In the authors' experience, a slow continuous injection of liquid glue (two parts of lipiodol and one part of glue) has a high chance to reach the draining vein and obliterating the fistula [20]. Following complete occlusion of the fistula, the progression of the disease can be stopped; however, only two thirds of all patients have a regression of their motor symptoms and only one third show an improvement of their sensory disturbances. Impotence and sphincter disturbances are seldom reversible [4].

Spinal cord AVMs

Spinal cord AVMs are fed by radiculomedullary and/or radiculopial, i.e. spinal cord, feeding arteries and drained by spinal cord veins. These shunts might be intra- and/or perimedullarily located and can be differentiated according to their transition from artery into vein into fistulous and glomerular AVMs [31].

Glomerular AVMs (which are sometimes called plexiforme or nidus-type AVMs) are the most often encountered spinal cord AVM with a nidus resembling closely those of a

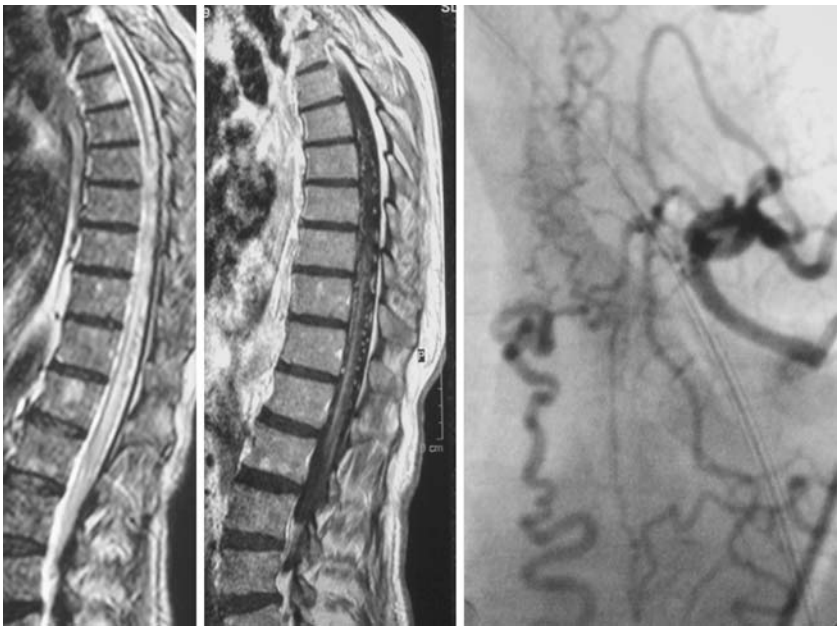

Fig. 1 Spinal dural arteriovenous fistula: MRI and DSA. On T2-weighted images, cord oedema and peri-medullary flow voids can be appreciated; after contrast enhancement, the peri-medullary vessels can be even better appreciated. The combination of pathologically dilated vessels and oedema of the cord is pathognomonic for spinal dural AV fistulae; angiography, however, is necessary to define the exact height of the fistula and to rule out a small peri-medullary fistula. After injection into the left Th6 intercostal artery, the fistulous zone can be appreciated that also receives blood from a small ascending dural branch and that drains into two distinct veins. This presents a challenging situation for the endovascular neuroradiologist since the glue has to pass into both veins to securely occlude both feeding arteries

brain AVM (Fig. 3). This type of malformation usually is located intra-medullarily; superficial nidus compartments can, however, also reach the subarachnoid space. Because of the many anastomoses between the anterior and posterior intrinsic arterial system of the spine, these AVMs have typically multiple feeding arteries derived from both the posterior and anterior system. The drainage is directed into dilated spinal cord vessels.

Fistulous AVMs (which are also called AVM of the peri-medullary fistula type or intra-dural AV fistulae) are direct arteriovenous shunts located superficially on the spinal cord and only rarely possess intra-medullary compartments [14]. Feeding vessels are again radiculomedullary arteries (which differentiates them from the dural AV fistulae where radiculomeningeal arteries are the feeders). Draining veins are superficial peri-medullary veins. The arterialised blood

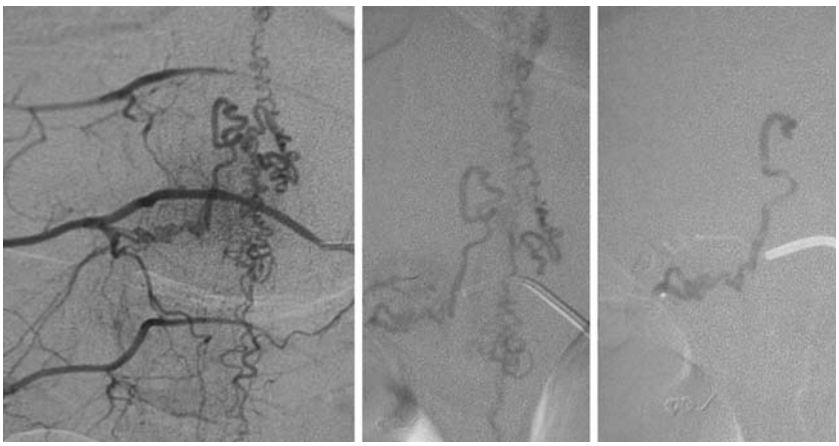

Fig. 2 Angiography and therapy in a spinal dural AV fistula: the goal of endovascular treatment is the permanent occlusion of the shunt that can only be performed with a liquid embolic material that has to pass from the arterial to the proximal venous segment. The superselective injection demonstrates the fistulous point and the glue cast verifies that the liquid embolic material has passed from the arterial to the venous site and therefore securely closed the shunting zone

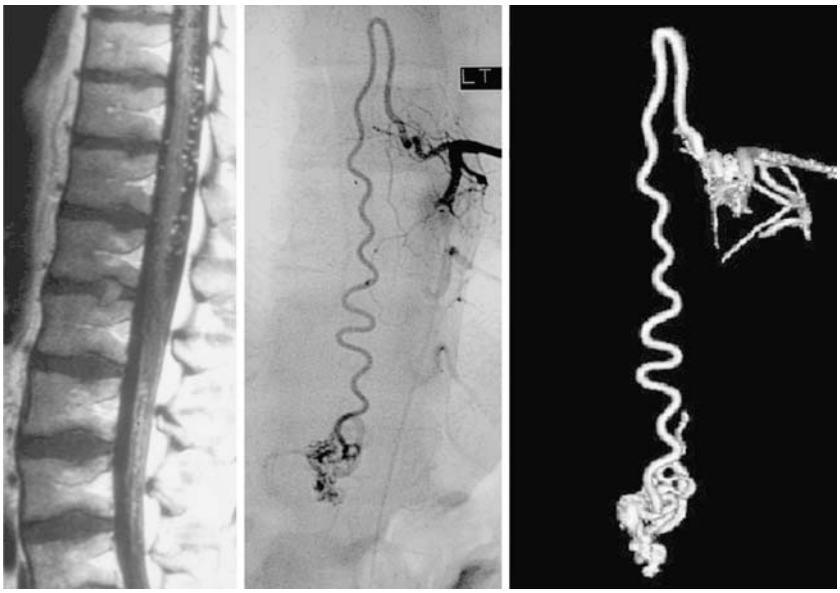

Fig. 3 Spinal glomerular AVM: this patient presented with subacute onset of progressive weakness of both legs over a 3-month period. MRI demonstrated increased signal within the conus and multiple flow voids along the spinal cord, which enhanced with contrast. Ventral spinal arterial supply was demonstrated towards a SCAVM located along the posterior aspect of the conus that is well appreciated on 3D rotational angiography

sometimes even ascends via the foramen magnum into the posterior fossa.

Depending on the size of the feeding vessels, the shunt volume and the size of the draining veins, these fistulae can be further categorised into those with a low shunt volume and only moderately enlarged feeding veins and arteries and those with a high shunt volume that lead to a massive remodelling of the blood vessels with enlarged arteries and dilated venous pouches. The latter types are typically encountered in children with a history of hereditary haemorrhagic telangiectasia [23].

Pathophysiologic mechanisms in spinal cord AVMs include venous congestion and haemorrhage. Space-occupying effects and a "steal phenomena" have also been attributed to the pathogenesis [1, 2]. If the AVM does not present initially with an acute haemorrhage, symptomatology is unspecific. Patients may complain about hypo-esthesia or paraesthesia, weakness and diffuse back and muscle pain. Progressive sensorimotor symptoms can slowly develop or acutely worsen followed by some improvements over time. Whilst fistulous AVMs often get symptomatic with a subarachnoid haemorrhage due to their intra-dural peri-medullary location, glomerular AVMs can become symptomatic by venous congestion alone [18], by intra-parenchymal haemorrhage and/or a subarachnoid haemorrhage.

On MRI, the AVM type cannot always be differentiated. The typical appearance of spinal cord AVMs is a conglomerate of dilated, peri- and intra-medullary located vessels that are demonstrated on T2-weighted sequences as flow voids, whilst they appear on T1-weighted sequences depending on their flow velocity and direction as mixed hyper- or hypo-intense tubular structures. Contrast enhancement may vary. A venous congestive oedema may be present as an intra-medullary hyper-intensity on T2-weighted images with concomitant swelling of the cord. The image might get even more complicated if intra-parenchymal haemorrhages are present that might demonstrate varying signal intensities depending on the time elapsed between bleeding and imaging [12]. A subarachnoid haemorrhage might be present. MRI should be able to identify the location of the AVM in relation to the myelon and the dura. Especially in low-flow peri-medullary fistulous AVMs, contrast media must be given to detect subtle venous dilatations [11, 19, 37]. Recently, it has been shown that fast MRA is capable of detecting the main arterial feeder of glomerular-type AVM and peri-medullary fistulous type AVM with a single large arterial feeder. Spinal cord AVMs with small or multiple feeders have not yet been investigated. Therefore, selective spinal angiography remains necessary to define the exact type of the AVM and to plan subsequent treatment.

The therapeutic approach of asymptomatic AVM is difficult since data concerning the spontaneous prognosis are not available; however, in symptomatic AVMs, therapy ameliorates the prognosis of the patient. The therapy of choice for all spinal cord AVMs is the endovascular embolisation with coils, glue or particles after careful analysis of the selective spinal angiography [5] with the embolising agent being dependant on the specific angioarchitecture.

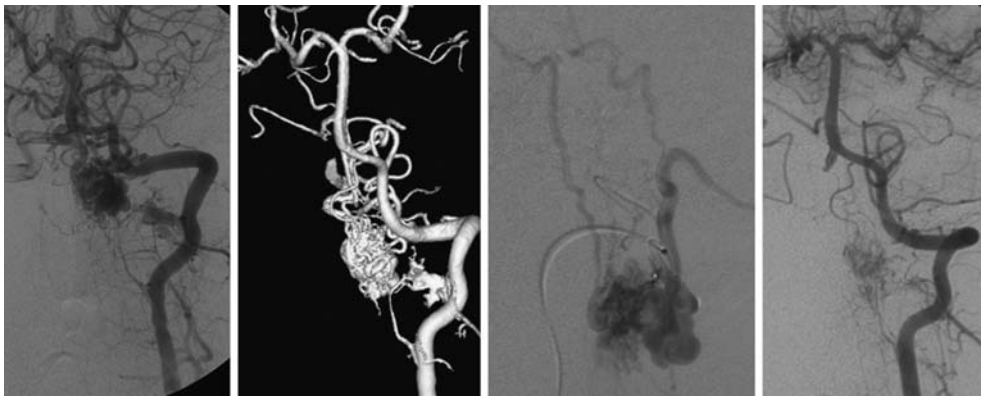

Fig. 4 Glomerular AVM prior and after therapy with liquid embolic agent (glue): after 3D angiography, a pseudoaneurysm as the target in this recently bled AVM can be identified that is subsequently occluded by glue. The control after embolisation demonstrates only a small residuum of the AVM

In glomerular AVMs, glue or particles can be employed to obliterate the nidus; even a partial embolisation seems to ameliorate the prognosis of the patient [9, 10, 29, 35] (Figs. 4 and 5). In low-flow peri-medullary fistulae, glue or sometimes even coils at the fistula zone can be used with the aim of obliterating the most proximal part of the venous receptacle (Fig. 6). A proximal arterial occlusion on the other hand will not be successful because of collateral recanalisation from the radiculopial network. If, therefore, the proximal venous segment cannot be reached by the glue cast or the coils, endovascular therapy should be avoided and a surgical attempt may be favoured. Large high-flow fistulous AVMs can easily be reached by superselective catheterisation close to the fistula and subsequent closure (typically with concentrated glue or coils) is in nearly all instances possible [21, 26] (Fig. 7). Again, the aim is to occlude the most proximal part of the venous part, which in case of glue can be performed using a "mushroom-shaped" glue cast. Some concepts of treatment in these diseases that are related to the fact that there are fundamental differences in spinal AVMs compared to brain AVMs have to be kept in mind: first, a hyper-acute treatment is rarely indicated since in our opinion acute rebleeding rarely occurs [30]. Instead, after a bleeding of a spinal AVM has occurred, we typically wait for approximately 6 weeks for potential vasospasm to resolve or the haemorrhage to absorb. Secondly, whilst brain AVMs have to be completely treated to avoid the risk of rebleeding, this seems not to be the case with spinal AVMs where partial treatment appears to be sufficient to dramatically improve the prognosis, especially in those cases where a complete eradication of the AVM is likely to produce neurological deficits. Especially in unruptured spinal AVMs that have become symptomatic with venous congestion rather than haemorrhage [18], the goal has to be to reduce the shunting volume rather than to make an "angiographically nice" picture that carries a high risk of treatment-related morbidity [29]. Thirdly, because of the low flow in some glomerular AVMs, endovascular therapies with particles seem to have a better and more stable result, if venous stagnation (with subsequent venous thrombosis of the outlet) occurs. Fourth, in the authors opinion, there is no role for radiosurgery in spinal vascular malformations, and the endovascular route should be the modality of choice in most instances. Surgery remains an option especially when the endovascular route is too long (which may be present in AV fistulae located at the filum terminale). Given the rarity of the disease, the constant advances in embolisation material and understanding of the disease and the varying experience of the treating team, exact numbers on technical success and failure rates, as well as data on morbidity and occlusion rate, cannot be extracted from the literature. In the first author's experience as primary treating physician in 11 fistulous spinal AVMs, complete occlusion via an endovascular approach was possible in ten patients; however, in four patients, more than one session was necessary to obtain this goal. In one patient, the fistulous site could not be reached with the microcatheter in a position distal enough to perform a safe occlusion. There were no treatment-related complications. Concerning glomerular spinal AVMs, the first author embolised 23 patients with 24 lesions in a total of 43 sessions. Transient treatment-related morbidity consisted of pain and sensory disturbances in four patients; in one patient, a persistent new neurological deficit occurred

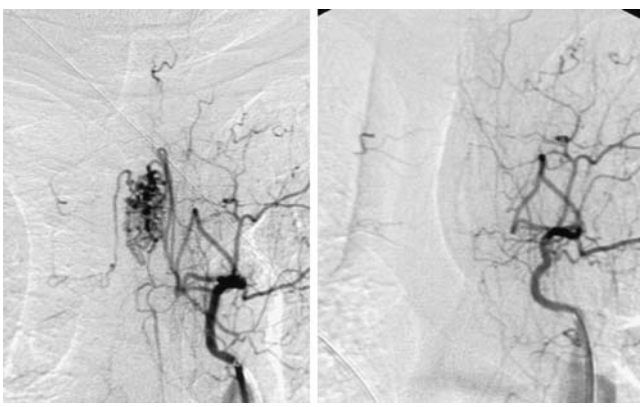

Fig. 5 Complete embolisation of a glomerular AVM using micro-particles with long-term persistence of occlusion in the follow-up DSA

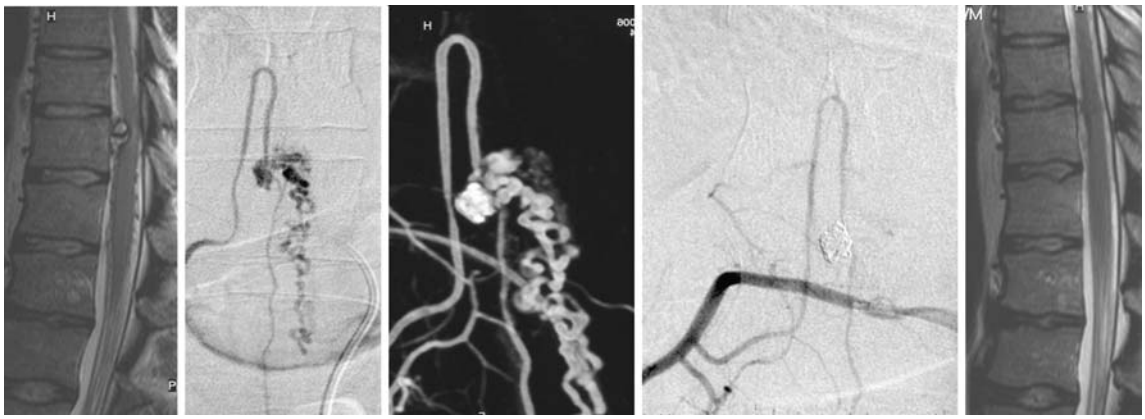

Fig. 6 Endovascular treatment of a fistulous AVM with coils: after subarachnoid hemorrhage in this patient, MRI demonstrated a flow void anterior to the spinal cord indicative of a vascular malformation. Angiography revealed a fistula of the peri-medullary type fed by the anterior spinal artery with a venous aneurysm at the transition between artery and vein that was completely occluded using coils leading to complete obliteration of the fistula with preservation of the flow in the ASA; follow-up MRI demonstrated complete regression of the aneurysm. The patient recovered completely from his deficit

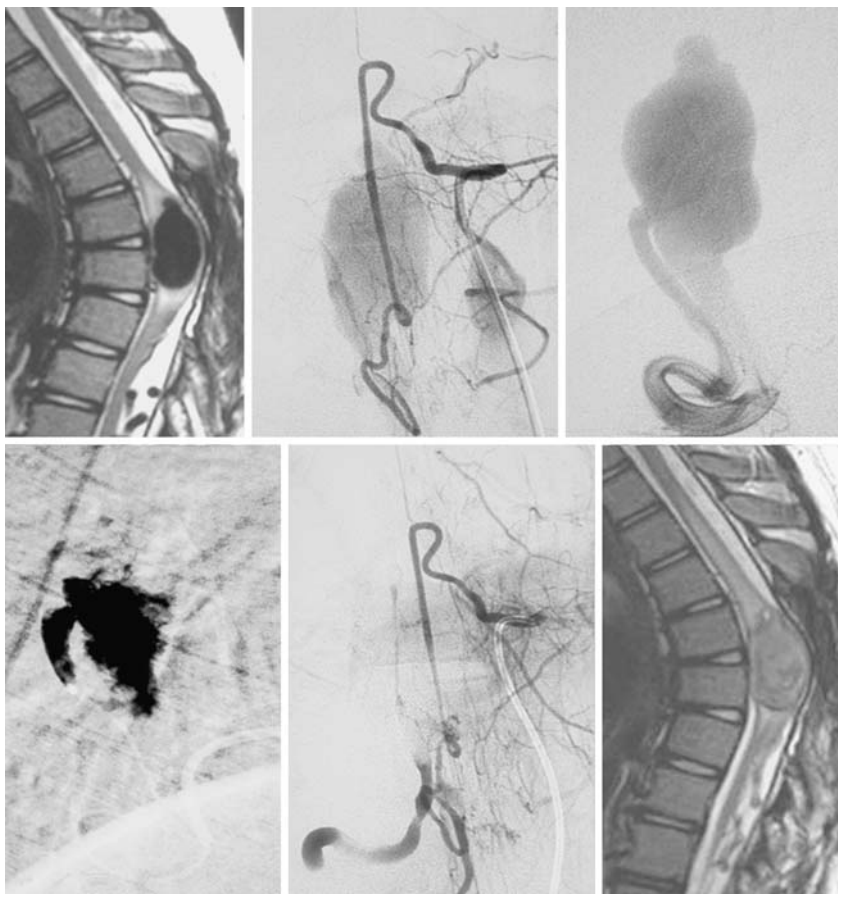

Fig. 7 Macrofistula in Rendu–Osler disease: both the anterior spinal artery and a posterolateral spinal artery converge into the same opening into a massively dilated venous pouch that further drains into dilated peri-medullary vessels. Therapeutic approach is safest via the dorsolateral radiculopial arteries with injection of a liquid embolic agent that occludes the inflow zone into the venous pouch in a "mushroom"-shaped way. Thrombosis of the venous pouch after closure may lead to mass effect with transient worsening of the symptoms

following embolisation (sensory disturbances following glue reflux in a posterolateral spinal artery). No new motor symptoms occurred.

Conclusions

The unspecific neurological symptomatology and the variety of potentially detected vascular malformations make this clinical entity challenging both for the neurologist/neurosurgeon and the neuroradiologist. When spinal vascular diseases are suspected, MRI should constitute the first diagnostic modality to identify the lesion and rule out potential differential diagnoses (spinal cord ischaemia, acute cord compression, tumour, degenerative diseases of the spine, myelitis). Even with the routine sequences, the neuroradiologist should be able to detect intra-medullary pathologies such as intra-medullary haemorrhages, oedema or venous congestion, extramedullary intra-dural alterations (such as dilated vessels or subarachnoidal haemorrhages) or potential extradural manifestations of spinal cord vascular malformations (such as associated haemangiomas). When neurological symptoms and MRI suggest a vascular malformation, spinal angiography is the next diagnostic step to define the type of vascular malformation and, thereby, to decide about the appropriate therapy. Treatment in symptomatic patients offers an improvement in the prognosis.

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Comments

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Krings et al. provide a nice overview of the nosology, imaging findings and treatment options of the main types of spinal dural or intra-dural arteriovenous malformations (AVM), i.e. spinal dural fistulas, on the one hand, and medullary AVMs of the fistulous or glomerular type on the other. Obviously, the relatively low numbers of patients and interventions even in specialised centres renders this field difficult for firm differential therapeutic recommendations, not unlike the situation presently encountered in cerebral AVMs. Nevertheless, the particularly experienced neuroradiological authors from three countries plausibly point out that, based on the available literature and personal experience, open neurosurgery will usually be the treatment of first choice in dural fistulas, whereas a primary endovascular approach may promise more success in most cases of medullary AVMs. Different from the management of cerebral AVMs, radiotherapy rarely appears to be an option. The article gives a profound overview over a complex topic. It demonstrates that similar to many other neurovascular disorders the management of spinal AVMs certainly requires an interdisciplinary approach in specialised neurovascular centres.

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In this comprehensive review, the authors summarise their experience with the endovascular management of spinal arteriovenous malformations along with relevant and useful data from the literature.

It combines the experience from two centres, which made significant contributions to (1) the understanding of the complex vascularisation, (2) the elaboration of a useful classification system and (3) the endovascular treatment of spinal vascular malformations.

The necessity to obliterate the initial segment of the draining vein of dural AVFs and the selection between complete or partially targeted embolisation of pial AVMs or AVFs are key concepts guiding the endovascular management of spinal AVMs.

Concerning the clinical outcome of technically successful embolisation of spinal dural AVFs, in the commentator's experience, this depends mainly on the time interval between the appearance of the first clinical manifestation and the therapeutic intervention.

The authors are to be congratulated for this useful review of a complex and rare subject.