Spinal Vascular Lesions

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Abstract: Spinal vascular lesions are relatively rare. For the the detection and treatment of such lesions, understanding their anatomical and pathophysiological basis is essential. Spinal ischaemic infarctions are probably extremely rare. Spinal dural AV fistulas are the commonest. These lesions may present acutely or chronically. MRI, MRA, CT scan, CTA of the spine, and spinal DSA are necessary investigation modalities for diagnosis and evaluation. Microsurgery is the mainstay of treatments; however, endovascular treatment options are sometimes very useful. In this chapter, spinal vascular malformations (including spinal AVM and AVF), spinal cord infarctions, spinal cord cavernomas and haemangiomas will be discussed briefly encompassing their management options.

Abbreviations

ASA	anterior spinal artery	AVM	arteriovenous malformation
AVF	arteriovenous fistula	CM	cavernous malformation
CT	computed tomography	CTA	computed tomographic angiography
dAVF	dural AVF	dAVM	dural AVM
DSA	digital subtraction angiogram	MRI	magnetic resonance imaging
MRA	magnetic resonance angiography	PSA	posterior spinal artery
SVM	spinal vascular malformation	ST	soft tissue
VB	vertebral body	VH	vertebral haemangioma

1. Spinal Vascular Anatomy

The posterior and anterior spinal arteries are the main blood vessels that perfuse the spinal cord's intricate blood supply system. To appreciate the pathophysiology of spinal vascular pathologies, one must have a comprehensive understanding of the usual spinal blood supply. The posterior and the anterior artery systems supply the spinal cord. The anterior arterial system is created by the anterior spinal artery (ASA), which remains in the anterior median fissure. The anterior horn's grey matter and the corticospinal tracts, including the spinothalamic tracts, are all supplied by sulcal arteries that emerge from the anterior spinal artery. Between the two posterior spinal arteries (PSAs), the posterior artery system creates a plexiform network of collaterals. It nourishes the posterior third of the spinal cord (Singh et al. 2016; Greenberg 2010).

The posterior and anterior spinal arteries typically receive a feeding from 6 to 10 medullary arteries. The vertebral arteries, including branches of the thyrocervical trunk, give rise to the medullary arteries in the cervical region. These medullary arteries arise from the intercostal and lumbar arteries, which in turn develop from the aorta and the iliac arteries in the dorsal and lumbar regions. The biggest of these medullary arteries, the artery of Adamkiewicz, normally arises on the left side to supply the spinal cord from D8 to L2 (Singh et al. 2016; Greenberg 2010; Lindsay et al. 2011).

2. Spinal Cord Ischaemic Conditions

2.1. Anterior Spinal Artery (ASA) Syndrome

2.1.1. Introduction

Ischaemia of the ASA, which results in functional abnormalities of the anterior two-thirds of the spinal cord, is the cause of ASA syndrome. The corticospinal tract, reticulospinal tract (autonomic fibres), spinothalamic tract, and grey matter are among the spinal structures that are impacted. Motor deficit, loss of pain, loss of warmth, and hypotension are signs that are found in this syndrome. The most typical type of spinal cord infarction is ASA syndrome. The anterior spinal cord is more susceptible to ischaemia due to the single anterior spinal artery's limited collaterals (as opposed to the posterior spinal cord, which is perfused by two PSAs) (Schneider 2010).

2.1.2. Aetiology

- Aortic insufficiencies: aortic dissections, aneurysms, direct aortic trauma, surgeries, and atherosclerosis.

- Pathology of the spinal column: acute intervertebral disc herniation, kyphoscoliosis, cervical spondylosis, compromised spinal column, and neoplasms.
- Other aetiologies: vasculitis, sickle cell disease, polycythaemia, decompression sickness, and collagen and elastin diseases (Schneider 2010).
- Any thrombus or embolus in the artery of Adamkiewicz can result in ASA syndrome (Yoon et al. 2002).

2.1.3. Clinical Features

- Total motor paralysis caudal to the level of the infarction.
- Loss of temperature and pain sensation at and caudal to the level of the infarction (Greenberg 2010).
- Intact sense of proprioception (Foo and Rossier 1983.)
- Autonomic dysfunctions: hypotension (either frank or orthostatic), sexual dysfunction, and/or bladder and bowel dysfunction (Cheshire et al. 1996; Cheung et al. 2002).
- Areflexia, flaccid external and internal sphincter tone leading to anal and urinary incontinence, and intestinal obstruction can also be found (Schneider 2010).

Symptoms generally occur very rapidly and are frequently experienced within 1 h of the initial damage.

2.1.4. Investigations

Ten to fifteen hours after the onset of symptoms, MRI can determine the extent and site of the damage. It is possible to employ diffusion-weighted imaging since it can detect impairment within a few minutes after the onset of symptoms (Schneider 2010). MRA, CTA, and DSA can be performed but have little value. Investigations for the identification of causes are needed.

2.1.5. Treatment

The root cause and symptoms will determine the course of treatment. The prognosis is bad when the diagnosis is hidden. The mortality rate is about 20%, and 50% of people die. Typically, there are only minor or no changes in symptoms (Schneider 2010).

2.2. Posterior Spinal Artery Syndrome (PSAS)

PSAS is very rare, as the white matter in the spinal cord is more resistant to ischaemia.
Clinical features:

- Absence of tendon reflexes/motor weakness.
- Absence of joint position sense.

Investigation: MRI T2W and DW images will easily diagnose the condition.
Treatment: Treatment is symptomatic and directed toward the aetiology (Greenberg 2010).

2.3. Venous Infarction (Total Cord Syndrome)

This is a swift "total" cord syndrome with poor outcomes, frequently linked to pelvic sepsis (Greenberg 2010).

3. Spinal Vascular Malformations (SVMs)

SVMs, also commonly termed spinal AVMs, are spinal vascular malformations. Approximately 4% of primary intraspinal masses are SVMs and they mostly occur in middle age (Youmans 1982).

3.1. Classification

The American/English/French Connection Working Formulation Classification (Greenberg 2010; Lindsay et al. 2011; Youmans 1982; Gueguen et al. 1987; Mourier et al. 1993).

3.1.1. Dural AVM (Low Flow)

Type 1: dAVM or dAV–fistula (AVF); 80% of SVMs in adults are type 1 (Strugar and Chyatte 1992). They take their vascular supply from the radicular artery, which creates a fistula (AV shunt) at the dural root sleeve (in the intervertebral foramen); then, it drains into a distended vein on the posterior spinal cord. Generally, it affects the lower thoracic or lumbar spine. These SVMs have a sluggish flow. Congestion of the cord's veins may

result from high pressure in the draining vein. Cord involvement could be far from the fistula. Back pain, cauda equina syndrome, progressive myeloradiculopathy, and urine retention are common symptoms in middle-aged individuals. Ninety percent of affected patients are men. Up to 35% of patients report experiencing pain. About 15–20% are connected to other AVMs (cutaneous or other). Rarely does a type 1 SVM bleed. A type 1B SVM contains two or more arterial feeders, whereas type 1A SVM has a single artery feeder.

3.1.2. Intradural AVMs (High Flow)

About 20% of spinal SVMs are intradural. Nearly 75% of intradural AVMs present clinically with sudden onset of symptoms, generally from haemorrhage (intramedullary or SAH).

Type 2 (spinal glomus AVM): These are intramedullary AVMs and the real AVM of the cord. Fifteen to twenty percent of all SVMs are glomus AVMs. A compact nidus supplied by medullary arteries with the AV shunt occupies at least partly the pia or the cord. They may be accompanied by supplying artery aneurysms. They have worse outcomes than dural AVMs. Usually one or at most two to three feeders occur in 80% of cases.

Type 3 (juvenile spinal AVM): It is an enormously engorged glomus AVM that occupies the whole cross-section of the spinal cord and extends down the vertebral body. It may lead to spinal deformity.

Type 4 (intradural perimedullary AVM): Arteriovenous fistulae (AVF) is another name for these. They are direct fistulae between the veins which drain the cord and the ASA, which typically supplies the spinal cord. Varices frequently occur in the vein immediately distal to the fistula. They are usually lethal, with bleeding into the subarachnoid space, and occur in younger individuals than type 1 (Bederson and Spetzler 1996). Based on the size and intricacy of the nidus, Merland and colleagues divided these lesions into three groups (Merland et al. 1980; Singh et al. 2016).

Subtype 1 pathologies are low-flow arteriovenous shunts in the conus or filum terminale composed of a small fistula fed by a solitary small ASA.

Subtype 2 perimedullary AVF are high- or moderate-flow fistulas. Their nidus is medium- or small-size and composed of multiple discrete shunts which are fed by several enlarged ASA sor PSAs.

Subtype 3 are multiple high-flow shunts composed of multiple dilated arteries feeding into a solitary large fistula.

3.1.3. Miscellaneous Vascular Lesions of the Spine

(i) Cavernomas of the spinal cord.
(ii) Venous angiomas of the spinal cord: very rare. Difficult to see in angiography.
(iii) Vertebral body haemangiomas.

Spetzler et al's classification of spinal vascular lesions are shown in Table 1.

3.2. Presentation

Depending on the form of AVM, the clinical characteristics of spinal AVMs can vary. The majority of spinal AV anomalies are seen in the lower dorsal and dorsolumbar spine, and the dural type is the most prevalent variety.

Table 1. Spetzler, et al.'s classification (this system of classification reincorporates vascular spinal tumours).

1. Neoplastic Vascular Lesions	**2. Spinal Aneurysms (Rare)**	**3. Arteriovenous Lesions**	
		AVF	**AVMs**
- Haemangioblastoma - Cavernoma	----- (very rare)	- Intradural: dorsal ventral - Extradural	- extradural–intradural - intradural - intramedullary - intramedullary–extramedullary - conus medullaris

Source: Authors' compilation based on data from Greenberg (2010); Spetzler et al. (2002).

Patients with the dural type typically complain of a myelopathy that is slowly progressing, and many of them have a history of recurrent myelopathy. The engorging vein's direct mechanical compression or a blood flow disturbance (steal/venous hypertension) that results in cord ischaemia are the two causes of the myelopathy.

The most frequent symptom on clinical presentation is motor weakness, which is preceded by paraesthesia plus sphincter abnormalities.

This type of spinal AV malformation is quite uncommonly associated with pain and acute myelopathy onset. Back pain with recent onset and myelopathy are common acute symptoms of the perimedullary and intramedullary types. Here, a burst nidus can result in intramedullary haemorrhage or subarachnoid haemorrhage. The clinical presentation may include indications of meningeal inflammation. However, a low-flow malformation or fistula might manifest symptoms gradually and can mirror the dural type's aetiology and clinical presentation (July and Wahjoepramono 2019; Dumont and Oldfield 2011; Jellema et al. 2003).

Eighty-five percent or so of patients have a growing neurodeficiency (back pain plus quadriparesis or paraparesis over months to years). Less than 5% of SVMs are present, comparable to spinal cord "tumours". About 10–20% of SVMs cause abrupt onset of myelopathy, which typically occurs in individuals under 30 years old (Greenberg 2010; Tobin and Layton 1976) and is brought on by bleeding (SAH, SDH, epidural haematoma, haematomyelia, or watershed infarction). In 2–3% of cases, a bruit is heard during auscultation above the spine. Some 3–25% of people have a lesion across their back; the Valsalva technique may make the angioma redder (Greenberg 2010; Barnwell et al. 1990; Tobin and Layton 1976).

3.3. Evaluation

3.3.1. Magnetic Resonance Imaging (MRI)

While MRI can identify some SVMs more reliably and safely than spinal DSA, it is insufficient for treatment planning. Extramedullary flow voids are present in 82% of lesions. Additionally, several levels of cord contrast enhancement are visible (from venous infarction or venous congestion). The diagnosis is not ruled out by a negative MRI. On T2WI MRI, the core region of the spinal cord may exhibit a diffuse hyperintense signal indicative of spinal cord oedema (Figure 1).

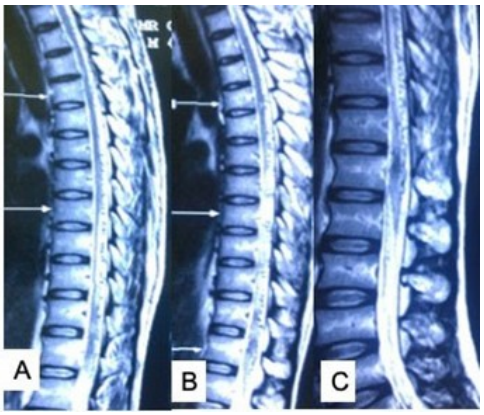

Figure 1. MRI of dorsal (**A**,**B**) and dorsolumbar spine (**C**) (T2W images, sagittal views) showing intradural vascular flow voids suggestive of spinal AVM. Source: Figure by authors.

A hypointense signal, which is more likely to be present at the cord's dorsal surface, may be present in conjunction with this lesion. An obstructed perimedullary venous plexus is represented by these hypointense signals (vascular voids). The spinal cord is frequently seen to be hypointense and enlarged on T1W imaging. When the contrast agent is infused, diffuse enhancement that corresponds to chronically obstructed veins may be visible along with a breach in the blood–spinal cord barrier.

3.3.2. Digital Subtraction Angiography (DSA)

Because DSA may be used to analyse the angiographic architecture of the vessels within or around the lesion, it becomes the preferred course of action. Additionally, DSA provides insightful data regarding the flow dynamics of the lesions, making it a significant tool for choosing treatment approaches (Figures 2 and 3).

For type 1dural AVMs, DSA must include all dural supplying arteries of the neuro-axis, including the following:

1. ICAs: the arteries of Bernasconi and Cassinari;
2. Every radicular artery, including the artery of Adamkiewicz;

3. Internal iliac arteries in search of sacral feeders.

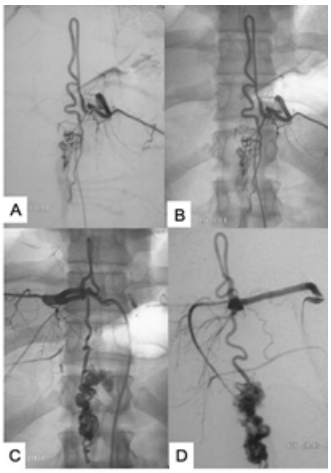

Figure 2. (**A**–**D**) Spinal DSA showing spinal AVM at dorsolumbar junction. Source: Figure by authors.

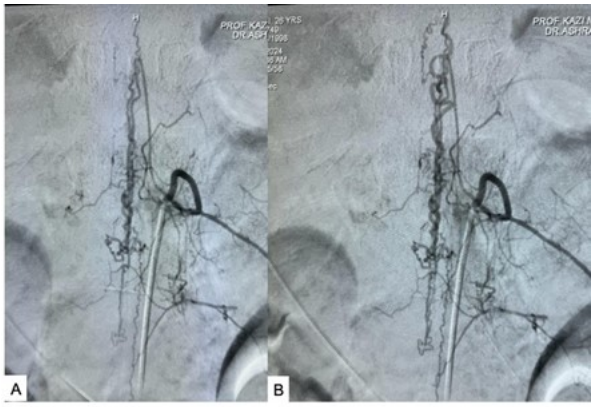

Figure 3. (**A**,**B**) Spinal DSA showing lower dorsal spinal AVM with feeding artery. Source: Figure by authors.

3.3.3. MRA

Spinal MRA might be useful if it can identify the fistula at a level that is suspiciously close to a vertebral level (Figure 4), as this information would speed up investigation via DSA. MRA is very important where DSA or CTA contraindicated.

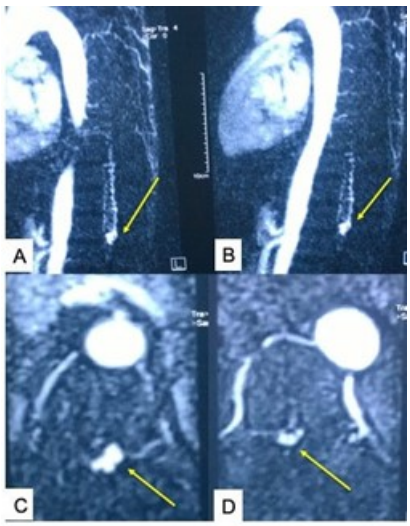

Figure 4. (**A**–**D**) Spinal MRA of the patient in Figures 1 and 3, showing the feeder and nidus of the AVM (arrow marked). Source: Figure by authors.

3.3.4. CTA

Spinal CTA can also define spinal AVMs with feeding arteries in the intervertebral foramen (Figures 5 and 6). Three-dimensional CTA is an attractive option for planning microsurgical excision (Singh et al. 2016; Greenberg 2010; Lindsay et al. 2011; July and Wahjoepramono 2019; Dumont and Oldfield 2011; Barnwell et al. 1990).

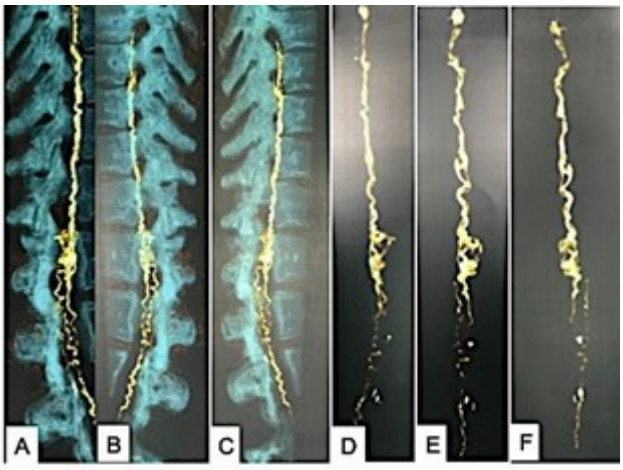

Figure 5. (**A**–**F**) CTA of dorsolumbar spine showing lower dorsal spinal SVM. Source: Figure by authors.

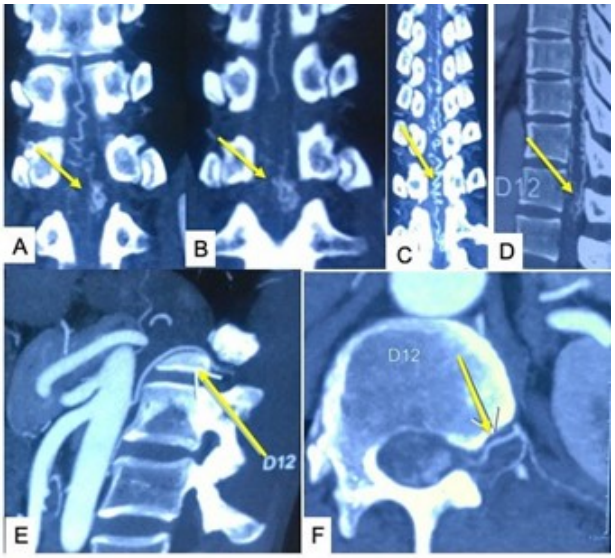

Figure 6. (**A**–**F**) Spinal CTA of the patient in Figures 1 and 4, showing the feeder and nidus of the AVM (marked with an arrow). Source: Figure by authors.

3.4. Treatment

There are two streams of treatments: (1) microsurgery and (2) endovascular therapy.

3.4.1. Microsurgery

Microsurgery (Figure 7) can safely deal with almost all spinal SVMs. The recurrence rate following microsurgery is very low in comparison to endovascular neurosurgery. While spinal AVM microsurgery is comparable to that for cerebral AVMs, the parenchyma cannot be retracted and haemorrhages are seldom life-threatening. Moreover, arteries of passage must be protected to prevent permanent impairments. A preoperative ICG angiography is frequently beneficial. The nidus is small, and the hemosiderin ring surrounding it on MRI frequently serves as a plane that can be used for imaging.

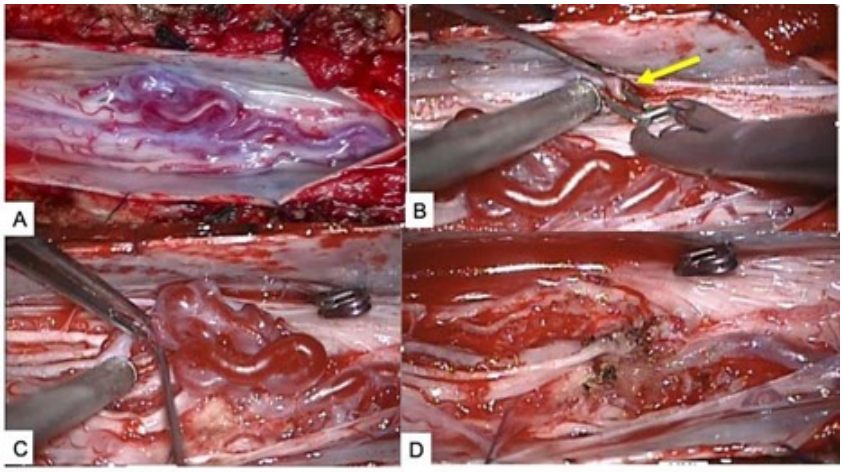

Figure 7. Intraoperative pictures of the patient in Figures 1, 4 and 6. (**A**) After laminotomy and dural exposure; (**B**) feeder occlusion with mini aneurysm clip (marked with an arrow), (**C**) excision of AVM; (**D**) after excision of AVM. Source: Figure by authors.

3.4.2. Endovascular Neurosurgery

It can be used in many cases of spinal AVMs, specially when the feeder vessel is limited with no mechanical compression. Type 1 (dural AVMs) is possibly receptive to glue-based endovascular therapy, in which case the proximal vein also needs to be sealed off. If a dural fistula is not entirely removed by the surgeon, it will return. Some type 2 (spinal glomus AVMs) SVMs, particularly type 2A SVMs (single-feeder), may be treatable with interventional neuroradiologic techniques like embolization. Surgery is frequently used for type 2B (>2 feeders) because recurrence may be more common with endovascular management than with surgical intervention.

3.4.3. Summary of Spinal AVM Treatment

All type 1 SVMs can be safely treated microsurgically. Endovascular therapy may also be effective but recurrence is higher. In type 2 A and B, microsurgery is preferable over endovascular therapy. In type 3 (juvenile type of spinal AVMs), the natural course is possibly better than the outcome with any type of management. But when intervention is inevitable, microsurgery is preferable. Type 4:

- Subtype 1: Microsurgery is recommended as endovascular therapy is difficult; treatment is easy on the filum terminale but challenging on the conus medullaris.
- Subtype 2: Microsurgery if preferable (especially with posterolateral AVF) as endovascular occlusion is incomplete.
- Subtype 3: Endovascular therapy is preferred as microsurgery is difficult (Singh et al. 2016; Greenberg 2010; Lindsay et al. 2011; July and Wahjoepramono 2019; Dumont and Oldfield 2011; Endo et al. 2016).

4. Spinal Cavernous Malformation

These lesions are frequently tiny, have little blood flow, and are fed by sinusoidal vessels with thin walls. Cavernous malformations (CMs) frequently have a gliosis and hemosiderin rim around them. MRI can quickly identify them (Figure 8). Despite being more frequent in the brain than in the spinal cord (See-Sebastian and Marks 2013), CMs nonetheless account for up to 12% of spinal vascular pathologies (Killeen et al. 2014). Cases often exhibit myelopathy symptoms between the third and sixth decades of life, while the disease's progression can vary in both intensity and severity (Scherman et al. 2016).

For individuals with asymptomatic CM or patients with modest, static symptoms, nonsurgical treatment with sequential surveillance imaging is a viable choice (Kivelev et al. 2012). Patients who experience a progressive neurological decline require surgical intervention (Liang et al. 2011) (Figure 9). Complete resection or obliteration of the lesion should be the aim of microsurgery because recurring myelopathic symptoms could result from remaining CM bleeding (Singh et al. 2016; Wang et al. 2016) (Figure 10).

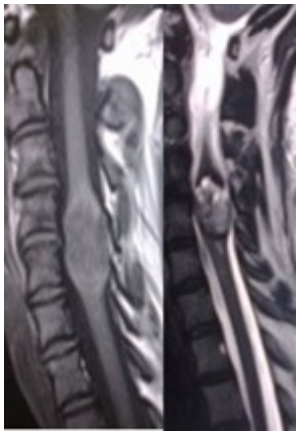

Figure 8. MRI of cervical spine (sagittal views, T1W and T2W images—right and left, respectively) showing intramedullary cavernoma. Source: Figure by authors.

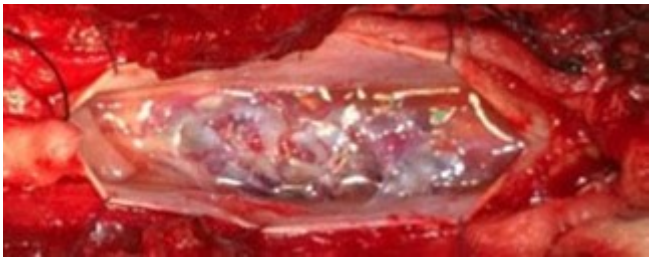

Figure 9. Perioperative picture of cavernoma of patient in Figure 8 after laminectomy, durotomy, and myelotomy. Source: Figure by authors.

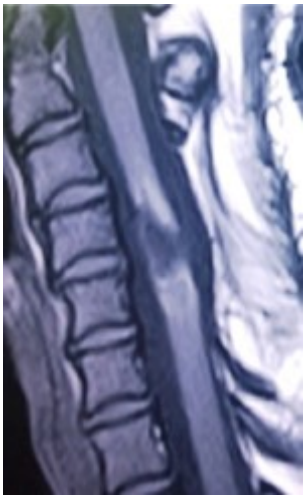

Figure 10. Postoperative MRI of cervical spine of patient in Figures 8 and 9 showing removal of tumour. Source: Figure by authors.

5. Spinal Epidural and Subdural Haematomas

These could exhibit a sudden onset of paraplegia. When a spinal AVM ruptures, an epidural or less frequently a subdural haematoma may develop. This can also happen after lumbar puncture or minor trauma, or it might develop on its own in patients who have a bleeding condition, liver disease, or are using anticoagulant medication. MRI (or myelography) shows the lesion in detail (Figure 11). Without waiting for spinal DSA, spinal decompression is necessary immediately after addressing any coagulation shortage (rapid CTA or MRA can be done on emergency basis). Angiomatous tissue may be seen during a pathological investigation of the haematoma; in many cases, there is no clear cause (Greenberg 2010; Lindsay et al. 2011; Dumont and Oldfield 2011; Jellema et al. 2003).

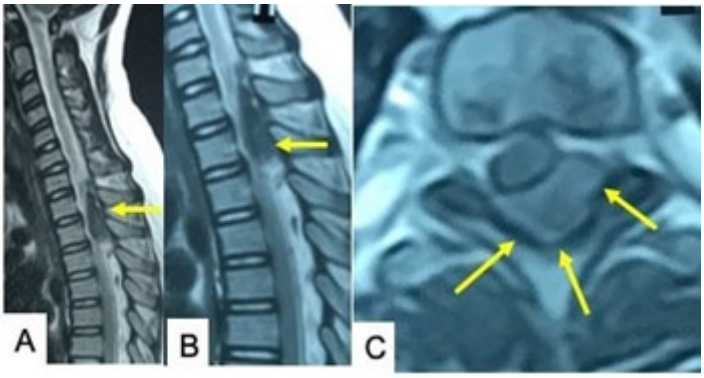

Figure 11. (**A**,**B**) MRI of spine (T2W images, sagittal sections) showing upper dorsal spinal epidural haematoma (marked with an arrow) causing acute paraplegia. (**C**) MRI of spine (T1W axial images) showing epidural haematoma (marked with an arrow). Source: Figure by authors.

6. Vertebral Haemangioma (VH)

6.1. Introduction

VH is also known as spinal haemangioma, cavernous haemangioma, or haemangiomatous angioma. VH classically occurs in post-pubertal female patients. VH is the most common primary spinal bone tumour (10–12% of all primary spinal bone neoplasms). Among these tumours, 70% are solitary and 30% are multiple (Greenberg 2010; Lindsay et al. 2011; Wang et al. 2018; Fox and Onofrio 1993; Healy et al. 1983). The most frequent site for these lesions is the dorsolumbar junction; the cervical and sacral spine are uncommon sites. In 25% of cases, VH only affects the corpus vertebrae; in 25% of cases, it affects the neural arch; and in 50% of cases, it affects both portions. Pure extradural lesions might happen very infrequently (Figure 12). The rarest lesions are intramedullary lesions. These are benign in origin and infrequently (1.2%) present with symptoms; they typically result from compression fractures, disc herniations, and, less frequently, neural compression from bone expansion by tumours. While reported, malignant degeneration is extremely rare (Lindsay et al. 2011; Fox and Onofrio 1993; Healy et al. 1983).

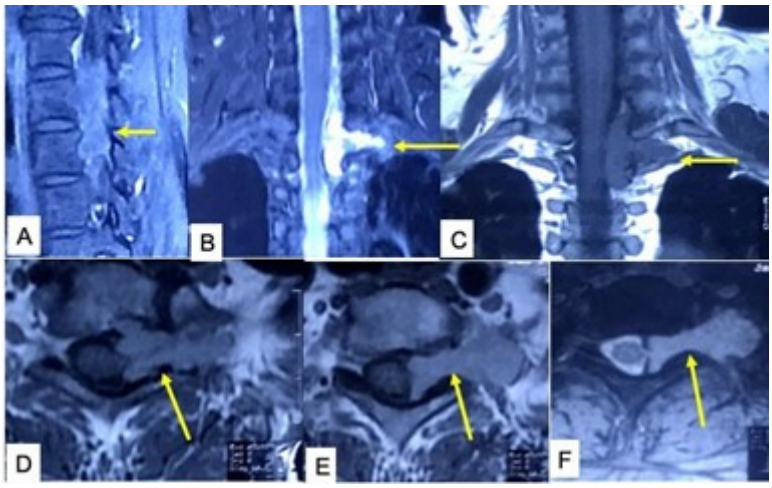

Figure 12. MRI of cervicodorsal spine. (**A**) Contrast sagittal image; (**B**) T2W coronal image; (**C**) T1W coronal image; (**D**,**E**) axial contrast image; and (**F**) T2W axial image showing pure spinal extradural (dumbbell-shaped) haemangioma (marked with an arrow). Source: Figure by authors.

6.2. Clinical Presentation

Most VH cases are asymptomatic and identified incidentally. Only 0.9% to 1.2% of people experience symptoms. The reason why symptoms hardly ever manifest before adolescence may be due to an unproven hormonal effect that causes symptoms to worsen with pregnancy or fluctuate with the menstrual cycle. VH can occasionally result in localized discomfort without radiculopathy at the affected level. However, rather than due to the VH itself, pain is more frequently present in these lesions due to associated pathologies (such as compression fracture, herniated disc, and spinal stenosis). Rarely, progressive neurodeficiency may develop (in the form of myelopathy, generally dorsal). Possible mechanisms of myelopathy include tumour growth into the spinal canal

through the epidural space, bone growth (cortical "blistering") with widening of the pedicles including lamina, and formation of a "bony" spinal stenosis. Pressure by feeding and draining vessels, spontaneous epidural haematoma, compression fracture, and cord ischaemia due to a "steal phenomenon" are the other rare causes of myelopathy in VH (Greenberg 2010; Wang et al. 2018; Fox and Onofrio 1993).

6.3. Evaluation

6.3.1. Plain X-Rays

VH typically shows coarse, vertically depicted striations (corduroy pattern) or a "honeycomb" picture. At least >30% of the vertebral body will have been involve before these findings appear on plain X-ray.

6.3.2. CT Scan

Spinal CT scan is the investigation of choice. The "polka-dot sign", i.e., multiple high-density dots, represents cross-sections through thickened trabeculae.

6.3.3. MRI of the Spine

It demonstrates that tiny haemangiomas are hyperintense, spherical, and focal on T1WI and T2WI. Lesions that are more widespread may be hypointense. On T1WI and T2WI, lesions with a propensity to develop exhibit a mottled enhanced signal. They are also isointense on T1WI and hyperintense on T2WI, and they tend to be symptomatic.

6.3.4. Spinal DSA

DSA may be used to differentiate between symptomatic (normal or slightly enhanced vascularity in contrast to neighbouring bone) and non-evolving (moderate to high hypervascularity) VH. If the supplying artery does not also feed the ASA, therapeutic spinal DSA may be performed. If not, the feeding artery may be embolized beforehand or sacrificed during operation (Greenberg 2010; Wang et al. 2018).

6.4. Treatment

6.4.1. Asymptomatic/Incidental VH

It does not warrant routine follow-up or evaluation.

6.4.2. Symptomatic VH (With Pain or Neurologic Deficit)

Treatment options are as follows:

(i) Radiotherapy: It can be used postoperatively after partial resection, preoperatively as a surgical adjuvant, or alone for symptomatic VH. The sclerotic obliteration of VH makes it radiosensitive. Pain relief may take months or years, and there may be no radiological signs of a response.
(ii) Embolization: Endovascular embolization can provide alleviation of pain more quickly in comparison to radiotherapy. It can also be utilized as a neurosurgical adjunct before surgery. If the major radicular artery (the artery of Adamkiewicz) is embolized, there is a danger of spinal cord infarction.
(iii) Vertebroplasty: It is a better option than kyphoplasty as kyphoplasty destroys the trabecular bone.
(iv) Biopsy: Biopsy is needed in cases where diagnosis is not certain (e.g., when metastases are highly probable).
(v) Surgery: Surgery is utilized for painful lesions that do not respond to the aforementioned treatments or for lesions that result in a growing neurological disability.

Surgical treatment options for symptomatic VH:

- Only neural arch involvement, with or without soft tissue (ST) in canal: radical excision.
- Vertebral body (VB) involvement with anterior canal compression: anterior corpectomy and strut graft.
- VB is affected but no bony expansion, ST in lateral canal: laminectomy with excision of soft tissue.
- Significant involvement of the anterior and posterior vertebral segments along with circumferential bone expansion without ST compression: laminectomy combined with radiation or laminectomy with close monitoring.
- Extensive involvement of anterior and posterior vertebral segments with ST in anterior canal: anterior corpectomy and strut graft plus radiotherapy or anterior corpectomy and strut graft and close follow-up.

- Risk of surgery: major surgical risks include blood loss, destabilization, neurological deficit, or postoperative extradural haematoma. In 20–30% cases, the disease recurs after subtotal resection; recurrence is common within 2 years. Patients with subtotal excision should have radiotherapy (Greenberg 2010; Lindsay et al. 2011; Wang et al. 2018; Fox and Onofrio 1993; Healy et al. 1983).

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