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## CHAPTER 47

# VASCULAR DISEASE OF THE SPINAL CORD

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Blood supply to the spinal cord and nerve roots originates in the vertebral, thyrocervical, costocervical, intercostal, and lumbar arteries, which give rise to radicular and medullary arteries. The segmental radicular arteries supply the nerve roots, originating near the vertebral foramina. Six to nine large medullary arteries originate from the vertebral, subclavian, or iliac arteries and the aorta (Fig. 47.1). Branches of the medullary arteries form a single anterior median spinal artery and two posterior spinal arteries, which perfuse the spinal cord. The anterior median spinal artery arises from the vertebral artery, and it runs along the entire length of the cord. The pial arteriolar plexus and posterior spinal arteries supply the dorsal aspect of the cord.



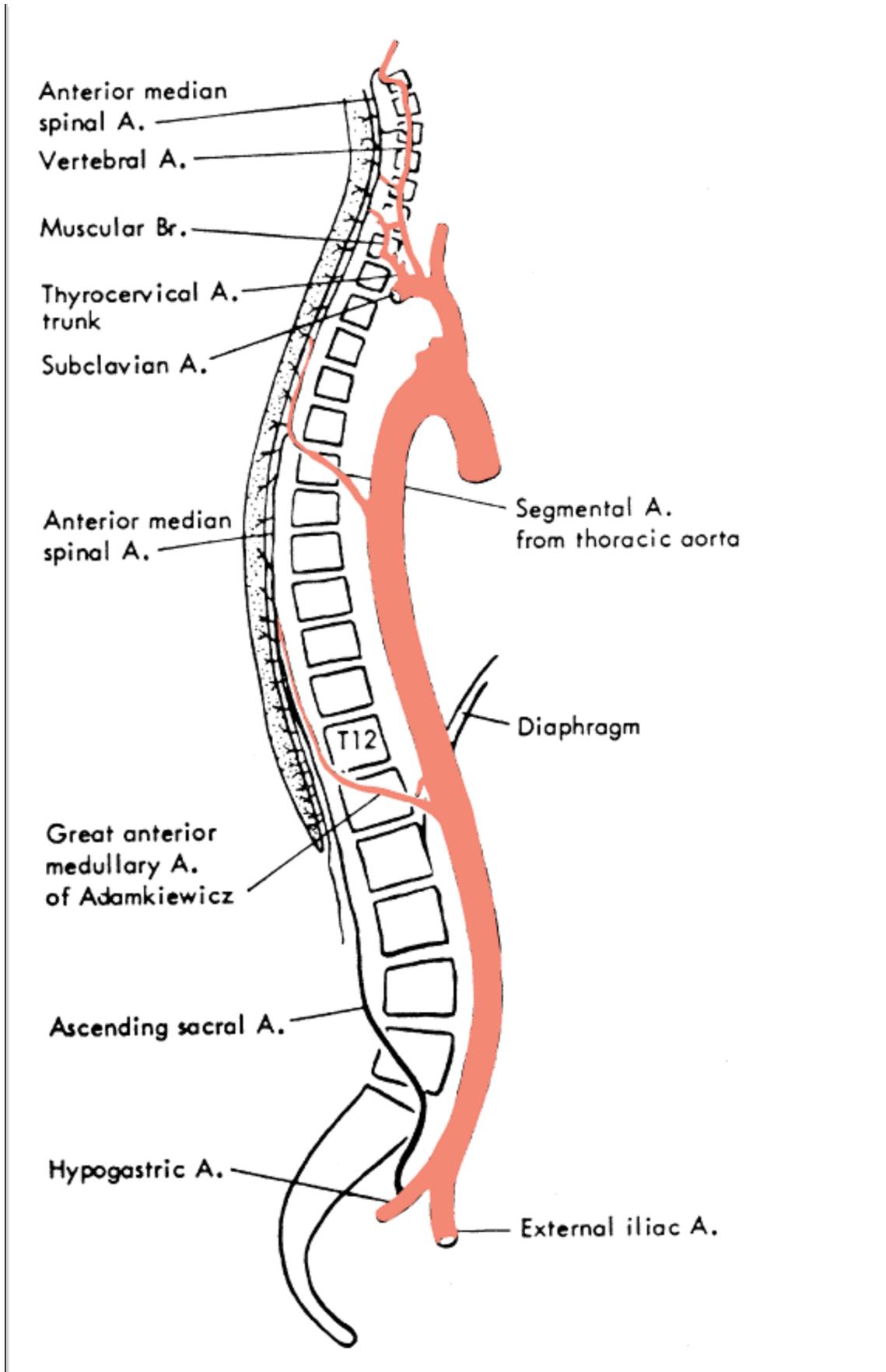


FIG. 47.1. The anterior median spinal artery is joined at various levels by arteries arising from the vertebral and subclavian arteries, the aorta, and the iliac arteries.

In the cervical region, the anterior median artery is collateralized at several levels by unpaired medullary arteries derived from the vertebral and subclavian arteries; this blood supply is rich in collateral branches. In the thoracic region, the anterior median spinal artery is joined by only a few branches of the thoracic aorta, and blood supply is relatively sparse, especially in the lower segments. The midthoracic spinal cord is supplied by terminal vessels descending from the subclavian and vertebral arteries or ascending from the abdominal aorta; this watershed is particularly vulnerable to vascular insufficiency, and spinal cord infarction is most likely to occur at T-4 to T-9. Because of its relative hypovascularity, the midthoracic region is particularly vulnerable to effects of hypotensive infarction. Midthoracic spinal syndrome (e.g., T-4 myelopathy) may be “false localizing,” and apparent clinical dysfunction at this level may actually be due to impaired perfusion at higher or lower cord levels or to global ischemia. Lumbar and sacral spinal areas are supplied by the largest and most constant of medullary arteries, the “great anterior radicular artery” of Adamkiewicz. This is usually found at L-1 or L-2 (occasionally as high as T-12 or as low as L-4). This artery, paired or single, travels through the vertebral foramen and anastomoses with the anterior medial spinal artery; the largest branch supplies the lumbosacral spinal cord and conus

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medullaris. Conus and cauda equina are also supplied by sacral branches ascending from the iliac arteries.

The central (sulcal) arteries originate in the anterior spinal artery to supply the anterior two-thirds and central area of the spinal cord. Penetrating branches from the pial arterial plexus supply the periphery and posterior one-third of the cord. Within the spinal cord, these arterial feeders anastomose in their most distal parts, creating border zones similar to those in the brain. These vascular border zones may explain the development of incomplete or partial syndromes seen after some spinal cord infarctions (see Fig. 47.1).

The plexiform venous system interconnects freely with the radicular arteries within subarachnoid space. The radicular veins empty into the epidural venous plexus, which in turn communicates with the inferior vena cava and azygos system through the perivertebral plexus.

## INFARCTION OF THE SPINAL CORD

Softening or infarction of the spinal cord (myelomalacia) results from occlusion of major vessels. Anterior spinal artery infarction is much more common than the posterior spinal artery syndrome because of the difference in collateral supply between these two regions.

### *Etiology*

Spinal cord infarction is most often caused by atheromas involving the aorta and results as

a potential complication of thoracoabdominal aneurysm repair. In one series, spinal infarction represented 1.2% of stroke admissions. Less common causes of spinal cord infarction include collagen vascular disease (including systemic lupus and polyarteritis), syphilitic angiitis, dissecting aortic aneurysm, embolic infarction (bacterial endocarditis, nucleus pulposus), pregnancy, sickle-cell disease, neurotoxic effects of iodinated contrast material used in angiography, compression of spinal arteries by tumor, systemic arterial hypotension as a consequence of cardiac arrest, and decompression sickness. Paraplegia may follow surgical repair of an aortic aneurysm when cross-clamp time is longer than 25 minutes; the risk of neurologic deficit may be lessened by avoiding systemic arterial hypotension, placing the shunt around the cross-clamp, and using thiopental anesthesia. Spinal cord ischemia may occur as an early complication of spinal arteriovenous malformation (AVM) repair (surgery, embolization).

### ***Symptoms and Signs***

The symptoms of spinal stroke usually appear within a few minutes or hours of the onset of ischemia. The first symptom may be local or radicular back pain. This may be lancinating or burning and is usually transient. There may also be diffuse, deep, aching pain in both legs, or a burning, dysesthetic pain may start in the feet and rapidly ascend to calves, thighs, and abdomen. These sensory symptoms are followed by rapid onset of leg weakness; the patient is soon unable to walk, reaching a peak of disability within minutes. Occlusion of the cervical part of the anterior spinal artery causes tetraplegia, incontinence of urine and feces, and sensory impairment below the level of the lesion. Proprioception and vibration sensations are spared because the posterior columns are supplied by the posterior arterial plexus. If proprioception and vibration sensation are impaired, the lesion is most likely not an anterior spinal artery infarction, and alternative diagnoses should be considered. Spastic weakness in the legs results from lesions of the lateral corticospinal tract. Sometimes, signs are restricted to those of either upper or lower motor neurons (or both) in a pattern similar to that of amyotrophic lateral sclerosis (ALS), but with major differences in mode of onset and lack of progressive worsening in spinal stroke. If the spinal ischemia involves only the gray matter supplied by sulcal arterial branches, there may be only lower motor neuron deficit (amyotrophy). The sudden onset of the motor deficit is consistent with a vascular etiology; however, if the deficit develops more slowly and progressively, clinical differentiation from ALS or spinal cord tumor may be difficult.

The level of the deficit in spinal ischemia may involve high-cervical or low-sacral regions, with the mean level of deficit occurring at T-8. Most clinical spinal cord strokes affect the midthoracic region, where paraplegia, urinary incontinence, and loss of pain and temperature sensation, with sparing of proprioception

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and vibration, localize the level. The weakness is flaccid at first, but Babinski signs are seen, and spasticity and hyperreflexia usually develop in a few weeks.

Arterial insufficiency of the lumbar region causes paraplegia, sphincter symptoms, and loss of cutaneous sensation with sacral sparing. The weakness is more likely to remain flaccid because the anterior horn cells are affected.

Transient ischemic attacks of the spinal cord and cauda equina may occur, but there is no

way to confirm this clinical impression. These attacks may precede spinal artery infarction, sometimes in association with lumbar spondylosis and stenosis. Symptoms may be precipitated by postural change in patients with lumbar stenosis. In cervical spondylosis, the role of arterial compression is uncertain in the subsequent development of myelopathy.

## ***Diagnosis***

Shadow spine radiography, myelography, computed tomography (CT), and magnetic resonance imaging (MRI) are needed to rule out spinal cord neoplasm or cervical spondylosis, which may simulate spinal cord stroke. Lumbar puncture excludes hemorrhagic or infectious disorders. In spinal cord infarction, cerebrospinal fluid (CSF) may show a slight protein content elevation, but gamma globulin content is normal. Two conditions that may simulate spinal infarction are multiple sclerosis (MS) and cord neoplasm. In MS or transverse myelitis, CSF frequently shows elevated gamma globulin content. Neoplasms are more likely to increase CSF protein content to values of several hundred milligrams per deciliter. In spinal cord infarction, myelography is usually normal; however, edema may cause signs of an intramedullary mass and subarachnoid block. Spinal angiography may cause cord infarction and is contraindicated unless spinal vascular malformation is considered likely. MRI may initially be normal in spinal ischemia but may later show focal swelling and abnormal (hyperintense) signal characteristics on T2-weighted sequences. Gadolinium-enhanced MRI would suggest an alternative etiology, such as MS, an infectious-inflammatory condition, neoplasm, vasculitis, or AVM. CT is unlikely to visualize an ischemic spinal lesion; however, it is likely to visualize spinal cord hemorrhage or vertebral abnormalities that may be the cause of the spinal syndrome.

## ***Treatment and Prognosis***

The general principles of care for patients with quadriplegia or paraplegia should be followed. Naloxone hydrochloride (Narcan) and calcium channel blockers have been used experimentally to treat spinal cord ischemia, but no studies have been undertaken in humans. There is no evidence to support utilization of antiplatelets or anticoagulants for spinal ischemia. The prognosis for recovery is varied. In one review, the following was reported: died (22%), unimproved (24%), minimally improved (9%), improved (25%), markedly improved (20%).

The major predisposing factor for spinal ischemia is surgical reconstruction of thoracoabdominal aortic aneurysm. The potential for spinal ischemia and resulting neurologic deficit depends on aneurysm extension and whether dissection has occurred. Despite utilization of hypothermia, intraoperative somatosensory monitoring, reanastomosis of intercostal arteries, short clamp time, distal aortic perfusion, and CSF drainage techniques, spinal ischemia is a potential risk of aortic aneurysm repair.

## **VENOUS DISEASE**

Venous disorders of the spinal cord are even less common than arterial lesions. Venous infarction may occur in patients with sepsis, systemic malignancy, or spinal vascular malformation. Patients experience sudden back pain, and motor, sensory, and autonomic dysfunctions develop. Sensory impairment does not necessarily spare the posterior

columns (as is characteristic of anterior spinal artery ischemia). CSF examination is necessary to exclude infectious–inflammatory or neoplastic conditions in patients with spinal cord ischemia. CT or MRI is necessary to exclude alternative lesions, including vascular malformations.

Foix-Alajouanine syndrome (subacute or progressive necrotic myelopathy) is characterized by spinal cord necrosis and evidence of enlarged, tortuous, thrombosed veins. Although this necrotic myelitis is attributed to venous thrombosis, there is usually no angiographic evidence of venous thrombosis or vascular spinal cord malformation. There is pathologic evidence of vascular malformations that are believed to have undergone spontaneous vascular thrombosis. There are multiple small infarcts and hemorrhagic spinal lesions. Pathologically, the necrosis for the most part involves the corticospinal tract, sparing anterior horn cells, and the lesion is most prominent in the thoracolumbar region. Clinically, there is usually subacute or progressive worsening of the condition for several weeks. The prodrome may include back or leg pain. Symptoms include leg weakness, incontinence, and sensory loss. Findings usually include spastic paraparesis, hyperreflexia, bilateral Babinski signs, and sensory level below the lesion. CSF may show a markedly elevated protein content, leukocytic pleocytosis, and red blood cells. Treatment with anticoagulants or corticosteroids has not been effective. Because some venous infarctions of the spinal cord are hemorrhagic, anticoagulation is potentially dangerous in this condition.

## **SPINAL CORD HEMORRHAGE**

Hemorrhage in the spinal cord is rare and may be epidural, subdural, subarachnoid, or intramedullary in location. Hematomyelia (hemorrhage into the substance of the spinal cord) usually immediately follows a spinal injury; however, it may be delayed for hours or days. Nontraumatic causes of spinal cord hemorrhage include blood disorders (e.g., leukemia), anticoagulation therapy, AVM, or venous spinal cord infarction.

### ***Pathology***

In intramedullary spinal cord hemorrhage, the spinal cord is swollen because of an intramedullary central blood clot. The blood dissects longitudinally for several segments below and above the hemorrhage, most severely affecting the gray matter and contiguous white matter. The clot is usually surrounded by

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a rim of normal nervous tissue. With time, the blood is liquefied and removed by phagocytes. Glial replacement is usually incomplete, resulting in a syrinxlike cavity that extends over several cord segments.

### ***Signs and Symptoms***

Localized back or radicular pain is sudden in onset. If hemorrhage is small, there may be only spastic weakness associated with hyperreflexia in the legs and bladder dysfunction. If hemorrhage is large, signs of cord transection include flaccid paralysis, complete sensory loss below the lesion, absent reflexes, Babinski signs, and loss of sphincter control. Autonomic disturbance and vasomotor instability may result in cardiovascular shock. If the patient survives, the hematoma is reabsorbed and symptoms may improve, but outcome is

uncertain. Spinal subdural or epidural hemorrhage is usually due to trauma (including lumbar puncture) or coagulopathy. Initially, patients experience neck or back pain and radiculopathy, or myelopathy may subsequently develop.

## **Diagnosis**

CSF is bloody or xanthochromic (especially if there is an associated subarachnoid hemorrhage), and protein content is increased. Myelography shows evidence of an intradural intramedullary mass with subarachnoid block. CT shows the hyperdense hematoma more clearly than MRI. Spinal angiography may be indicated in nontraumatic cases if spinal vascular malformation is suspected.

Spinal epidural or subdural hemorrhage may cause mass effect that rapidly compresses the cord. This may follow spinal trauma, even without evidence of bone fracture; other causes include anticoagulation and blood dyscrasias, and there are cases without obvious etiology. Epidural hemorrhage may follow lumbar puncture in patients with a coagulation disorder. The symptoms of epidural and subdural spinal hematoma are similar. Symptoms of epidural hemorrhage appear rapidly, with back pain, sensory loss, and sphincter impairment. The diagnosis is established by CT, which directly visualizes the hemorrhage, or myelography, which shows evidence of cord compression by an extradural mass. Spinal subarachnoid hemorrhage may be due to vascular malformation, spinal neoplasm (most commonly ependymoma), blood dyscrasia, or periarteritis nodosa, which is characterized by sudden, severe back pain at the level of the lesion. Symptoms may be due to blood in the subarachnoid space or to blood dissecting into the spinal cord or along the nerve root sheaths. CSF is bloody and xanthochromic. CT shows the hyperdense hemorrhage. Myelography and spinal angiography are necessary to establish the cause. Ruptured intracranial aneurysm occasionally causes severe back pain with clinical findings indicative of spinal hemorrhage; in these cases, cerebral angiography may be necessary to determine the etiology, especially if the patient also reports headache and has nuchal rigidity.

## **Treatment**

Treatment of spinal cord hemorrhage depends on the cause and location of the hemorrhage. For subdural and epidural hemorrhage, surgery is necessary, but the prognosis is poor when paraplegia is present and there is delay in surgical intervention. Patients with spinal cord hematomas caused by anticoagulant therapy should receive fresh whole blood and vitamin K. Spinal cord decompression is carried out if effective hemostasis can be achieved, but should be avoided if bleeding impairment is not correctable.

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