

Spinal Ischemic Stroke

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PREVALENCE ≈ 1.2% of all strokes (in general, spinal stroke is rare).

Pathologically – [myelomalacia](#).

- infarction of adjacent vertebral body may be associated.

ETIOLOGY

Diabetes mellitus is affecting ≈ 50% patients.

According to location of vascular pathology:

- A. **Aorta** (major source for spinal strokes!):
- 1) aortic atherothrombosis
 - 2) dissecting aneurysm
 - 3) aortitis
 - 4) aortic surgery (esp. with aortic cross-clamping above renal artery; below that level artery of Adamkiewicz provides protective circulation)
 - 5) aortography, mediastinal angiography
 - 6) aortic coarctation:
 - a) adult type - blood diversion from cord by retrograde flow in anterior spinal artery to bypass narrowed region (→ neurogenic intermittent claudication)
 - b) classic type - cervicothoracic myelopathy from cord compression by enlarged collateral vessels or from steal phenomenon.
- B. **Arterial feeders** (e.g. thoracic, intercostal, or cervical branch from subclavian or vertebral artery)
- 1) thromboembolic disease!
 - 2) complications of abdominal surgery (esp. sympathectomy)
 - 3) dural AV fistulas (between radicular arteries and veins outside dura mater) – cause venous hypertension → characteristic dilated veins that course on spinal cord surface.
- C. **Anterior spinal artery:**
- 1) arteritis (e.g. collagenosis, syphilis, tuberculosis, Lyme borreliosis, schistosomiasis)
 - 2) trauma
 - 3) complication of spinal angiography, spinal anesthesia
 - 4) compression by cervical spondylosis, tumors in spinal canal, vertebral fracture, herniated intervertebral disk
 - 5) spinal adhesive arachnoiditis
 - 6) spontaneous
- D. **Intrinsic cord vessels:**
- 1) arteritis
 - 2) emboli:
 - a) atheroma
 - b) nucleus pulposus fragments (cartilaginous emboli) – unclear how fibrocartilage enters into circulatory system
 - c) decompression sickness (nitrogen emboli)
 - 3) hypoperfusion (cardiac arrest, prolonged hypotension)
- E. **Spinal veins** (rare); e.g. extensive thrombosis of local pial veins in association with AVM

N.B. *intrinsic disease of spinal arteries* is rare cause (vs. vascular compression, disorders of remote vessels).

CLINICAL FEATURES

- **acute (often apoplectic) onset** evolving over minutes:

N.B. all other etiologies (e.g. acute transverse myelopathy, viral myelitis, Guillain-Barré syndrome, mass lesions) evolve more slowly!

1. Sudden severe sharp **SPINAL (BACK) PAIN** (> 80%); at level of lesion, may radiate caudad (radicular pain); usually transient.
 Why spinal infarcts are painful? - unexplained difference from cerebral infarction!
2. **Loss of motor & sensory function:**
 At level of infarct – segmental flaccid areflexic **weakness, sensory loss** (for all modalities).
 Distal to level of infarct – bilateral spastic **weakness** (reversible leg weakness ÷ quadriplegia), **paresthasias, sensory loss**.
 N.B. in acute stage (usually for several days), “**spinal shock**” is observed commonly.
3. Within few hours - loss of sphincter control - **inability to void / defecate**.

LEVELS

- most commonly in "watershed" areas - **T₁₋₄** segments and **L₁** segment.

MIDTHORACIC REGION is most susceptible to ischemia!

VESSELS

Arteries

Anterior spinal artery – anterior 2/3 (or central “watershed”) of spinal cord: paraplegia or quadriplegia, dissociated sensory loss, loss of sphincter control.

- spared *posterior columns* (vibration, position, and light touch sense).

N.B. anterior spinal artery is discontinuous in some individuals, increasing importance of feeders to lower cord!

Posterior spinal artery – uncommon (posterior radicular arteries enter at every level from both sides):

- 1) *below level of lesion* - loss of proprioception and vibration sense
- 2) *segmental* - anesthesia & loss of reflexes.

Diffuse hypoperfusion → infarction at *boundary zones*:

- 1) midthoracic region in anterior spinal artery territory
- 2) between anterior and posterior spinal artery territories - syndrome of spastic weakness with little sensory change (resembling ALS)

N.B. brain damage usually is more severe than spinal injury!

Veins

- unilateral infarction of one hemicord (hemiplegia or monoplegia, crossed pain and temperature loss) may occur.

DIAGNOSIS

Top priority is to exclude **spinal cord compression by MASS LESION** (neoplasm, abscess, granuloma, hematoma, herniated disk) - urgent need for surgical decompression.

N.B. period during which reversible spinal insult can recover is short (< 24 h).

Another treatable pathology - "transverse myelitis" due to **herpes simplex** or **varicella-zoster** infection.

Avoid pitfall of limiting spinal region studied:

- high cervical regions** have little local symptomatology.
- sensory level may be caudad to lesion** because of topographic lamination with superficial location of ascending sensory pathways (lateral spinothalamic tracts) from lower spinal segments.

1. Spinal MRI – imaging method of choice:

- often normal
- signal changes (focal or diffuse) with mild swelling; rapid evolution over days results in contraction of area of signal change and thinning of involved cord region.

Only MRI can demonstrate pathological changes, other diagnostic modalities serve purely to exclude alternative diagnosis!

2. Cranial MRI:

- for **multifocal CNS diseases** (MS, SLE, infectious disorders, sarcoid)
- "cerebral" paraparesis can occur in **parasagittal disorders** (e.g. parasagittal meningioma) or **bilateral ACA ischemia** (e.g. anomalous common stem of ACAs)

3. CT myelography – if MRI is unavailable / unsatisfactory.

- myelography (in spinal cord infarction) - usually **normal**; edema may cause **intramedullary mass** and **subarachnoid block**.

4. Spinal CT – no real application in spinal ischemia (lacks sensitivity to exclude mass lesions).

5. Spinal angiography – indicated for strongly suspected **spinal AVMs, AV fistulas**.

N.B. spinal angiography per se may cause cord infarction!!!

6. CSF examination – for differential diagnosis (if MRI is negative) - excludes hemorrhagic and infectious disorders.

- CSF (in spinal cord infarction) - may show **slight protein elevation** (but γ -globulin content is normal).

7. SSEP: **normal** (anterior spinal artery) or **prolonged** (posterior spinal artery).

TREATMENT

1. Pathogenetic treatment – **antiaggregants** (limit extension of ischemic lesion + secondary prophylaxis):

- ASPIRIN** – standard drug therapy.
- CLOPIDOGREL**
- ASPIRIN** + controlled-release **DIPYRIDAMOLE**.

- no definitive studies define use of **anticoagulants** (could be indicated for unusual cases – spinal TIA or incomplete infarction with stuttering or progressive course).

2. Treatment of cause (e.g. a/b for infection, obliteration of spinal-dural AV fistula) + control of risk factors (e.g. diabetes mellitus, hyperlipidemia, hypertension).

3. Standard measures **against complications of acute paraplegia** (prevention of pulmonary embolism, pressure sores, spasticity treatment, urinary incontinence and urgency treatment).

4. **Neuroprotective strategies** (e.g. antioxidant, antiglutamatergic, protease inhibition, naloxone, calcium channel blockers) - not reported effective in human cord ischemia.

5. **Diet** not directly relevant.

6. **Activity** – early transfer to chair and ambulation.

PROGNOSIS

Life expectancy is diminished because of vascular, infectious, and other medical **complications**.

- ischemia is in part reversible - **prognosis is better than in traumatic cord injuries**.
- as with all infarcts, deficit is most marked during first few days and may partially resolve with time, but also deficit may progress over next few days to complete transverse myelopathy (with fatal outcome).

BIBLIOGRAPHY for ch. "Neurovascular Disorders" → follow this [LINK >>](#)