Transverse Myelopathy

Last updated: September 5, 2017

TRANSVERSE MYELOPATHY - syndrome of spinal cord segment damage across greater part of sectional area.

ETIOLOGY

1. **Trauma** - most frequent cause of ***complete*** lesions.

2. **Demyelinating & inflammatory processes** (**TRANSVERSE MYELITIS**) - most commonly ***incomplete*** lesions (although there is evidence of involvement of entire cross-section of cord):
    1) **MS**, neuromyelitis optica
    2) **infection** – enteroviruses (esp. poliomyelitis*, enterovirus 70-71), herpesviruses (HSV-2**, VZV***, EBV, CMV), mumps, measles, mycoplasma, acute meningovascular syphilis, HIV, HTLV-I.

        *prototypical acute infectious myelitis
        **recurrent sacral myelitis in association with outbreaks of genital herpes
        ***most common cause of acute viral myelitis
    3) **collagenoses** - SLE (!), Sjögren's syndrome, Behçet's disease.
    4) sarcoidosis (subacute transverse myelopathy with severe cord swelling).

3. Spinal cord **ischemia** - ***complete*** or ***incomplete*** lesion (e.g. anterior 2/3 anterior spinal artery syndrome).

4. **Hemorrhage** into spinal cord.

5. Intraparenchymal **abscess**.

6. **IDIOPATHIC acute transverse myelitis**

IDIOPATHIC ACUTE TRANSVERSE MYELITIS

ETIOLOGY

- frequently after ***nonspecific viral infection*** – *direct viral invasion* into cord or *autoimmune* mechanisms.

Pathology - inflammatory demyelination which involves several segments (usually thoracic).

- may progress to necrosis and cavitation.

CLINICAL FEATURES

- **lost all motor & sensory functions below level** of transverse myelopathy.

N.B. sensory & motor findings tend to be *symmetric* (vs. MS – *asymmetric*)!

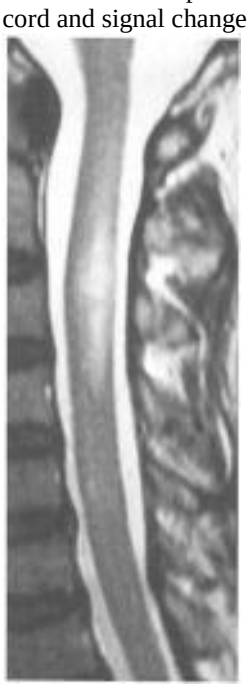
- onset – (sub)acute back pain, ascending leg weakness, paresthesias below level of lesion, sphincter dysfunction.
    - pathological process may be acute - initially produces *SPINAL SHOCK*, but hyperreflexia soon supervenes; persistent areflexic paralysis indicates necrosis over multiple spinal segments (differentiate from Guillain-Barré syndrome).
- common additional features:
    - band of ***disagreeable dysesthesia*** above uppermost border of sensory loss.
    - ***radicular pain*** at lesion level.
    - ***tender*** (on percussion) ***spinous processes*** - in epidural abscess, vertebral metastasis.
- *spontaneous complete recovery* (over weeks ÷ months) occurs in 60% cases.

DIAGNOSIS

CT / MRI - mild ***fusiform swelling*** in affected region.

- diffuse / multifocal ***abnormal bright signal*** on T2-MRI.
- ***contrast enhancement*** (BBB disruption) in acute cases.
- *brain MRI for all cases* - to assess for of MS.

T2-MRI: mild expansion of upper spinal cord and signal change (white) within it:



CSF:

a) normal
b) ***pleocytosis*** (up to several hundred mononuclears; in severe acute cases, PMNs may be present), ***protein normal or mildly elevated***.

Most important differentiation (must be done rapidly with MRI!) - ***compressive myelopathy***:

1) spinal or epidural abscess / hematoma
2) tumor, especially metastatic (may present acutely even though tumor has been present for weeks or longer)
3) herniated intervertebral disk (central herniation may cause acute compression without local pain).

TREATMENT

Corticosteroids (e.g. IV METHYLPREDNISOLONE 500 mg q 12 hours for 3 days → tapering with PREDNISONE) - reduce edema and lead to earlier function restitution.

- indications – idiopathic (postinfectious) transverse myelopathy, MS, cord compression.

SURFER'S MYELOPATHY

Freedman, Brett A "Surfer's Myelopathy: A Rare Form of Spinal Cord Infarction in Novice Surfers: A Systematic Review" Neurosurgery: May 2016 - Volume 78 - Issue 5 - p 602–611

- rare (64 cases reported), acute, atraumatic thoracic/conus medullaris myelopathy that occurs in ***young, healthy, novice surfers who have no pre-existent spinal disease***.
- most definitive support is for a *vascular* cause (angiogram may show the absence of radicular artery and no artery of Adamkiewicz).
  - altered venous return that occurs from lying prone for prolonged periods of time on a surfboard can contribute to vascular insufficiency.
  - occlusion by embolus or vasospasm induced by prolonged hyperextension; there have been no reported cases of a similar acute myelopathy in novice or elite butterfly stroke swimmers, who repetitively and violently hyperextend their flexible trunks for brief periods of time, often while performing a Valsalva maneuver; therefore, it is more likely that prolonged hyperextension plays the putative role.
- clinically start with back pain and rapidly progress to complete / incomplete myelopathy.
- diagnosis: T2 signal in the central cord within 24-72 hours; gadolinium enhancement and DWI are not helpful; at follow up – cord atrophy at involved levels.
- treatment - patients receiving **steroids** improved 55% of the time; optional – **lumbar drain**.

TABLE 2. Prioritized Treatment Recommendations for Acute Surfer's Myelopathy (ie, Presentation <3 Hours From Symptom Onset)[a]

1. Confirm diagnosis by clinical history, physical examination, and emergent routine magnetic resonance imaging of the spine (rule out underlying spinal disease)
2. Consider 1 of these interventions
Emergent aortic spinal angiography and superselective catheter–delivered tPA or nimodipine (rule out underlying aortic disease), or
Administer intravenous tPA, National Institute of Neurological Disorders and Stroke protocol dose, or
Place lumbar drain and maintain cerebrospinal fluid pressure <10-15 mm Hg and send sample
3. Admit to intensive care unit for close monitoring; place Foley catheter; record postvoid residual
4. Elevate mean arterial pressures >85 mm Hg for at least 24 h, first with intravenous fluids, judicious use of narcotic pain medications, and then vasopressors
5. Consider NASCIS III methylprednisolone protocol (24-h dose; 30-mg/kg loading dose; 5.4-mg·kg^{-1}·h^{-1} infusion × 23 h)
6. Bedrest in a position of comfort for 24 h; Immediate passive and active range of motion for all 4 extremities; physical medicine and rehabilitation/physical therapy/occupational therapy consults
7. Evaluate outcome at 24-48 h; those with severe deficits present at 48 h will require transfer to a spinal cord injury rehabilitation center and urological consultation/urodynamic study

[a]NASCIS, National Acute Spinal Cord Injury Studies; tPa, tissue-type plasminogen activator.

- prognosis:
  - incomplete cases improve within 24 hours of onset
  - no improvement has been reported for ASIA A cases (>50% of reported cases)
  - overall neurological recovery rate - 42%;

BIBLIOGRAPHY for ch. "Spinal Disorders" → follow this LINK >>