

## TRANSVERSE MYELITIS

### *SUBTYPES*

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## Definition

- Intrinsic disorder of the spinal cord
- Temporal features
- Spatial features
- Etiology

## Temporal and spatial patterns

- Peak reached at 10 days
- Damage in both longitudinal and horizontal planes
- All functions of SC are disturbed to various degrees

## Semiology

- Prodromes
- Paresthesiae, paraparesis, backache, sphincter dysfunction
- Degree of paresis varies between slight weakness and total paralysis

## Signs (cont.)

- Early, muscle tone is decreased (spinal shock), later spasticity develops
- DTR initially not obtainable or reduced, later they can brisk
- Babinski sign
- Trunkal sensory level

## Etiology

- Parainfectious
- Postvaccinial
- Autoimmune
- MS
- Devic
- Paraneoplastic
- Vascular

## Postinfectious & Postvaccinial Transverse Myelitis

- Prodrome
- Monophasic
- Autoimmune

• Jeffery DR, Mandler RN, Davis LE. *Arch Neurol* 1993;50:532-535

## Etiology

Postinfectious	Postvaccinial
Rubella	Rabies
Rubeola	MMR
Varicella	Varicella
Influenza	PPD
EBV	Influenza
Hepatitis A	
Mycoplasma	
Lyme	
Syphilis	

## CSF

- Pleocytosis (10-100 WBC)
- High protein
- Normal glucose

## Infection

- Viral: Echo, Coxsackie, HSV 1&2, Human herpesvirus6, VZV, CMV, HIV, Hepatitis A, EBV, Mumps, Rubella
- Bacteria: Mycoplasma pneumoniae, Borrelia, Yersinia, Chlamydia, Rochalimaea, Mycobacteria
- Parasites: Schistosoma, Cysticercosis, Toxocara, Toxoplasma gondii

Table III: Bacteria associated with ATM

Infectious agent	Special characteristics	Diagnostics	Therapy
<i>Borrelia</i> (Linszen et al. 1991)	Mostly radiculomyelitis, pure myelitis rare	Search for specific intrathecal AB synthesis. In exceptional cases first positive after 4 wk	High-dose penicillin G or third-generation cephalosporins for 21 days IV (e.g. ceftriaxone)
<i>Mycoplasma pneumoniae</i> (Heller et al. 1990, Mills and Schollfeld 1992)	High fever, ESR accelerated, peripheral leukocytosis	AB in serum	Tetracyclines in combination with corticosteroids
<i>Yersinia enterocolitica</i> (Saebo et al. 1993)	Rare	AB in serum (ELISA)	Antibiotics
<i>Chlamydia psittaci</i> (Williams and Sunderland 1989)	Rare	AB in serum	Antibiotics
<i>Rochalimaea henselae</i> (Bogue et al. 1989)	In immunocompetent persons until now, only reports with simultaneous symptoms of cat-scratch disease	AB in serum (IF or EIA)	Gentamicin or cotrimoxazole, perhaps also other antibiotics
<i>Mycobacteria</i> (Bahemuka and Murungi 1989)	Only very seldom pure intrathecal forms; glucose concentration is reduced in CSF	MRI, intradermal test	Antituberculous chemotherapy, very rarely surgical therapy required

Table IV: Parasites causing ATM

Infectious agent	Special characteristics	Diagnostics	Therapy
<i>Schistosoma mansoni</i> and <i>Haematobium</i> (Haribhai et al. 1991, Nazer et al. 1993)	Sometimes intramedullary granulomas, also diffuse and necrotizing ATM	History of visiting infectious areas. If positive: serum AB (ELISA), MRI	Praziquantel, possibly corticosteroids in addition; laminectomy at CSF block or worsening while drug therapy is given
<i>Cysticercus cellulosae</i> (cysts from <i>Taenia solium</i> ) (Kishore et al. 1991)	Eosinophile lymphocytes in serum and CSF	MRI; AB in serum and CSF (CF, ELISA); proof only by biopsy	Surgical removal of cysts, postoperative praziquantel
<i>Toxocara canis</i> (Linden and Berlit 1994)	Rare, eosinophils in serum, IgE level raised	MRI; AB in serum (ELISA)	Thiabendazole or diethylcarbamazepine plus corticosteroids
<i>Toxoplasma gondii</i> (Wende-Fischer et al. 1993)	Rare, sometimes if patients are HIV positive, multifocal necrotizing course	MRI, CT, AB in serum. If HIV positive AB findings are difficult to interpret	Sulphonamides, pyrimethamin, spiramycin; plus corticosteroids

AB, antibody; ESR, erythrocyte sedimentation rate; ELISA, enzyme-linked immunosorbent assay; IF, immunofluorescence; EIA, enzyme immuno assay.

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**Table II: Viral causes of ATM\***

Virus	Special characteristics	Diagnostics	Therapy
Varicella-zoster virus (Heiler et al. 1990)	If infection is reactivated, mostly segmental paresis, early serious course. No invasion of spinal cord if associated with chickenpox. Consider VZV-ATM in immunosuppressed patients	PCR, AB search	If reactivation: acyclovir IV if no improvement in course corticosteroids in addition
Herpes simplex virus 1 and 2 (Radhakrishnan et al. 1994)	Necrotizing myelitis	PCR, AB search	Acyclovir IV in combination with corticosteroids
Human herpesvirus 6 (Hill et al. 1994)	Rare	PCR, AB search	Symptomatic
B-virus/herpes simiae (Holmes et al. 1990)	Rare, survival only if therapy is started very early, until now only adult patients	Possible only if there was direct contact to monkeys. If history is positive try virus isolation from discharge from the wound, AB search in serum	Acyclovir IV
Epstein-Barr virus (Landgren et al. 1994, Junker et al. 1994, Tsunomi et al. 1994)	Relatively good prognosis	PCR, AB search in serum and CSF (anti VCA-IgG and IGM, antiD, antiEBNA)	Symptomatic
Cytomegalovirus (Güngör et al. 1993)	Reported only in HIV-positive patients	PCR, search for pp65 in blood cells, conventional and shell vial culture	Ganciclovir IV, if resistance is suspected foscarnet
HIV (Geny et al. 1991)	Detailed diagnostics only if there is a positive history or risk factors	Diagnosis according to CDC criteria	Systemic therapy; recommendations change quickly
Coxsackievirus (Berlit 1988)	Rare	AB search in serum and CSF	Symptomatic
Echovirus (until now only subtypes 2, 5, 19, 25) (Barak and Schwartz 1988)	Rare, symptoms and signs can resemble polio	Culture from CSF, stool, throat swabs, AB search in serum and CSF	Symptomatic
Hepatitis A virus (Tyler et al. 1986)	Rare, reported only in association with typical general symptoms	Hepatitis serology	Symptomatic
Rubella virus (Bitzan 1987)	Very good prognosis, not always contemporary general symptoms	PCR from serum and CSF, AB search in serum	Symptomatic

## Autoimmune-Rheumat.

- ✱ SLE
- ✱ MCD
- ✱ PSS
- ✱ SS

## Laboratory

- ✱ ANA
- ✱ Double stranded-DNA Ab
- ✱ Soluble Nuclear Antigen Ab
- ✱ Anti-Ro
- ✱ AntiSS/A and SS/B
- ✱ RF
- ✱ CBC,APTT, Cardiolipin Ab
- ✱ CSF in 1<sup>st</sup> 24 hours(Low glu,high protein,pleocytosis)

## Lupus myelitis

- ✱ 17% of SLE-TM patients presents with TM as first SLE symptom.
- ✱ Fever as prodrome
- ✱ Pathology:Thrombotic complications due to cardiolipin antibody

## Elsberg Syndrome

- ✱ Sacral radiculomyelitis
- ✱ Urinaryretention
- ✱ Hypo/paresthesia –sacral dermatomes
- ✱ CSF pleocytosis without elevated protein
- ✱ Primary HSV type-2 infection
- ✱ Prognosis-good

## Anterior Spinal Artery Syndrome

- ✱ Posterior cord function is preserved
- ✱ Acute onset of motor symptoms with dissociated sensory symptoms
- ✱ Etiology (60 cases)
  - ✱ Unknown (14)
  - ✱ Spinal angioma (10)
  - ✱ Postinfectious (9)
  - ✱ A. spinal artery occlusion (9)

## MS and TM

- TM is common initial presentation of MS
- MS presenting as an isolated spinal cord or brainstem syndrome is 42% (Miller et al.)
- Lack of relation to antecedent infection
- Polyphasic course
- Chronic recurrent
- Progressive

## Differentiation

- 1. Symmetry of both sensory and motor impairments (>94% accuracy; Scott et al. Neurology 1998,50;2)
- 2. Oligoclonal bands in CSF

## Complementary studies

- 3. MRI
- 4. Physiology

## Prognosis

- Combination of severity of weakness and EMG had had 90.3% predicting power.
- Severity of weakness and denervation on EMG are most useful for predicting the outcome of TM at 6 months

## Devic's Neuromyelitis Optica Diagnostic Criteria

- **C.** Acute involvement of spinal cord and optic nerves, coincidental or separated  
No cerebral, cerebellar or cortical features
- **I.** Normal brain MRI, enlarged, cavitated spinal cord
- **L.** No OCB-No CNS IgG-Incr CSF alb

## Devic's NMO (cont'ed)

- **Pathology:**  
Necrosis, cavitation of spinal cord, thickened vessel walls  
no inflammatory cells.  
No plaques in brain, brainstem or cerebellum

## Devic's NMO Therapeutic Protocol

- W&H, CXR, BP, UA, HbA-1C, Lytes, CBC, Stool guiac, PPD, LFT
- Solumedrol 500 mg IV bid/2 hours x5 days
- Prednisone 1 mg/kg/d qam for 2 months
- Azathioprine 2 mg/kg/d at 3<sup>rd</sup> week
- Very slowly convert prednisone to qod

