**Transverse myelitis**

What is Transverse Myelitis (TM)?

Transverse myelitis (TM) is an inflammation of the spinal cord. “itis” means inflammation and “myel” refers to the spinal cord. So, “myelitis” means inflammation of the spinal cord. “Transverse” indicates that the inflammation tends to affect the diameter of the cord rather than the length of the spinal cord. The inflammation in TM is usually limited to a short segment of the spinal cord rather than its entire length.

What are the symptoms of TM?

TM is usually noticed over a few hours and reaches a maximum severity over a few hours or days. The symptoms depend on what parts of the spinal cord are affected.

The spinal cord carries signals from the brain down the back. A second nerve then takes these signals from the back out to the rest of the body. Sensory signals from the body are brought to the spinal cord by nerves, and then are taken up the spinal cord to the brain. The spinal cord is the main connection between the brain and the rest of the body.

Motor nerves carry signals to muscles. If these nerves are affected, the result is referred to as spasticity. Spasticity will affect the muscles from the level of injury to the spinal cord downwards. For example, if the spinal cord is injured at the waist, then weakness will occur from the waist down. Spasticity has several components. This includes weakness, tightness/stiffness of the muscles, spastic leg jumps and brisk reflexes.

Sensory nerves carry signal from the skin, bones, joints and muscles to the brain. If these nerves are damaged, sensory symptoms will occur below the level of injury. If the injury is severe enough that no signal gets to the brain, then numbness results. If the signal gets to the brain, but is jumbled, then a number of odd sensations can result. These include pins and needles, burning, pain, tingling or other odd sensations. A feeling of tightness or constriction at the level of the injury is so common that patients have named it the “MS Hug”.

Damage to the nerves serving the bowels leads to constipation. Damage to the nerves serving the bladder can lead to a number of bladder symptoms including urgency, retention and hesitancy. Sexual dysfunction can also be seen.

These symptoms are discussed in greater detail in the section on Management of Symptoms.

What diseases cause TM?
The most common cause of TM is MS. However, many other diseases must be considered. These include neuromyelitis optica, compression by masses in spine (discs), infections (Herpes simplex 1 and 2, Lyme disease, syphilis, zoster, cytomegalovirus, enteroviruses, influenza, Epstein Barr virus, HIV, HTLV 1, tuberculosis, mycoplasma, parasitic diseases), blockage of the blood supply to the spinal cord (vascular myelopathy, fibrocartilagenous embolism), increased pressure in the veins (venous infarct, dural arteriovenous malformation), vitamin deficiencies (B₁₂, Vitamin E, copper), radiation myelopathy, and other inflammatory diseases (lupus, Sjogren’s syndrome, sarcoid).

What diagnostic tests are needed for TM?

If TM occurs in a patient for the first time, evaluation by a neurologist is recommended. This is often done in the emergency room in patients without a prior diagnosis.

Evaluations include a careful neurology examination. An MRI is usually needed to look for masses which might compress the spinal cord. It is important that the MRI look high enough on the spinal cord. For example, weakness in the legs may be due to an abnormality of the spinal cord in the neck.

A lumbar puncture (spinal tap) may be needed to evaluate possible infections or other causes of TM.

Blood tests are indicated to evaluate infections and inflammatory causes of MS.

What are the diagnostic criteria for TM?

The diagnosis of TM must meet the following criteria (Transverse Myelitis Consortium Working Group. Neurology 2002;59:499):

- Development of sensory, motor or autonomic dysfunction attributable to the spinal cord
- Bilateral signs and/or symptoms (though not necessarily symmetric)
- Clearly-defined sensory level
- Exclusion of extra-axial compressive etiology by neuroimaging (MRI or myelography; CT of spine not adequate)
- Inflammation within the spinal cord demonstrated by CSF pleocytosis or Elevated IgG index or gadolinium enhancement. If none of the inflammatory criteria is met at symptom onset, repeat MRI and LP evaluation between 2-7 days following symptom onset meets criteria
- Progression to nadir between 4 hours to 21 days following the onset of symptoms (if patient awakens with symptoms, symptoms must become more pronounced from point of awakening)
The diagnosis of TM is divided into two types: Idiopathic and disease-associated. The cause of idiopathic TM is unknown. Disease-associated TM occurs in the presence of another disease which is believed to be the cause of the TM. The following diseases must be considered before diagnosing idiopathic TM:

- History of previous radiation to the spine within the past 10 years
- Clear arterial distribution clinical deficit consistent with thrombosis of the anterior spinal artery
- Abnormal flow voids on the surface of the spinal cord c/w AVM
- *Serologic or clinical evidence of connective tissue disease (sarcoidosis, Behcet’s disease, Sjogren’s syndrome, SLE, mixed connective tissue disorder etc)
- *CNS manifestations of syphilis, Lyme disease, HIV, HTLV-1, mycoplasma, other viral infection (e.g. HSV-1, HSV-2, VZV, EBV, CMV, HHV-6, enteroviruses)
- *Brain MRI abnormalities suggestive of MS
- *History of clinically apparent optic neuritis
*Do not exclude disease-associated ATM

**How often does TM lead to MS?**

TM may be the first attack of MS. Overall, 60-70% of people with TM go on to have MS, if there is no other explanation for the TM. The risk of having MS is best determined by the MRI. If the MRI is normal, then the risk of MS is 21% after 20 years. If the MRI is abnormal, the risk of MS is 82% after 20 years. This is discussed in more detail in the section on clinically isolated syndrome (CIS)

**How is TM treated?**

Acute attacks of TM are treated like MS attacks. This generally involves corticosteroids if the symptoms are severe enough to warrant it. If symptoms are severe and if they fail to respond to corticosteroids, then plasma exchange may be used. These treatments are discussed further in the section on treating acute attacks of MS.

If patients have an abnormal MRI, and thus have a high risk of having MS, treatment with medications to decrease the frequency of MS attacks may be warranted. These are discussed further in the section on treating MS.