Transverse myelitis is an acute inflammatory condition of the spinal cord characterised by rapid onset of bilateral motor, sensory and autonomic dysfunction. While a relatively rare condition, having a reported incidence of 1 – 8 cases per million, the diversity of its underlying etiologies makes it an important diagnostic challenge.

An approach to the classification and work up of transverse myelitis outlined by the Transverse Myelitis Consortium Working Group in 2002 (1) aims to standardise the diagnostic criteria and terminology in order to facilitate the design and analysis of clinical research, and also forms a useful tool in the clinical work up of patients at presentation. Important diagnostic features include an appropriate clinical picture with symptoms referable to the spinal cord, maximal clinical severity within 4 hours and 21 days, and imaging or CSF evidence of an acute inflammatory process.

The pathogenesis of transverse myelitis can be grouped into four broad categories:
1. multiple sclerosis (MS) and neuromyelitis optica (NMO)
2. systemic autoimmune syndromes such as systemic lupus erythematosis (SLE) and Sjogren’s disease
3. infectious causes including Herpes viruses, Picornaviruses, Flaviviruses and HIV
4. idiopathic

Radiation myelopathy and vascular conditions such as cord ischemia and spinal vascular malformation are other conditions which often fall within the differential diagnosis, but are classified separately to transverse myelitis.

Imaging is done in the first instance to exclude cord compression. MRI appearances in transverse myelitis can be relatively nonspecific with typical findings consisting of central T2 hyperintensity extending for more than 2 vertebral segments and involving greater than 2/3 of the cord cross sectional area. In the acute stages there maybe cord expansion and enhancement (2, 3). Careful analysis of the morphology of cord involvement, enhancement pattern and presence of coexistent abnormalities on MRI brain can provide clues as to the underlying etiology. For example, MS lesions are usually smaller and involve the dorsolateral cord, polio and other picornaviruses typically involve the anterior horn cells and certain infective etiologies can cause nerve root or leptomeningeal enhancement. Together with the clinical background and specialised tests, neuroimaging is extremely important in identifying subgroups that may benefit from specific treatment.

Interval MRI follow up is of use in assessing response to treatment, but is also an important diagnostic tool in difficult or equivocal cases. Resolution of expansion and enhancement over time is helpful in excluding cord neoplasm. Follow up brain and spinal cord MRI may also demonstrate the appearance of new lesions, which will confirm progression of an isolated cord syndrome to clinically definite multiple sclerosis.

References: