Fall 2014

The Mystery of Transverse Myelitis: Can it Happen to You?

Jessica Castle
Otterbein University, jessica.castle@otterbein.edu

The Mystery of Transverse Myelitis: Can it Happen to You?

Jessica Castle, RN, BSN

Fall Semester 2014 | Otterbein University, Westerville, Ohio | Advanced Pathophysiology (NURS 5330)

Introduction

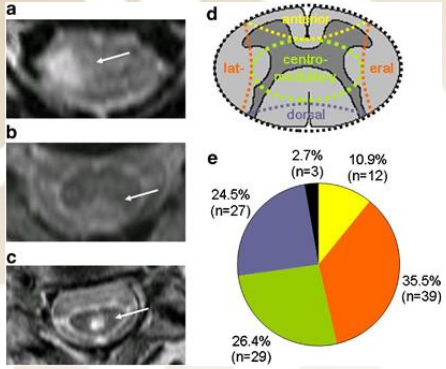
Imagine a day like any other, as a healthy performs daily tasks without any particular difficulty. Then, the first fall happens. After standing and recovering, two more falls occur; however, despite all physical effort, standing proves to be a complete impossibility. Imagine the fear. What is going on?

This unexplainable scenario happened to an 81 woman diagnosed with idiopathic transverse myelitis (TM) in May 2011, leaving her permanently paralyzed below the mysteriously appearing T9 lesion. This woman had only a personal history of asthma and breast cancer, and nothing significant in her family history. After 60 days of lumbar punctures, MRI's, CT's, blood tests, plasmapheresis treatments, high dose steroids, and IVIG transfusions, the neurological damage to her periphery proved to be irreversible, leaving her paralyzed below the T9 level. Still a mysterious disease, researchers continue to search for a cause and a cure.

Raising awareness of TM's prevalence can lead to discovery of new treatments. By researching its etiology and mysterious attacks on neurological cells, permanent damage may be avoided.

Diagnostic Imaging: Figure 1

"Axial topography of 110 spinal cord lesions from 57 patients with acute transverse myelitis. (**a–c**) Examples of the most frequent axial lesion distributions on T2-weighted scans are shown (a) lateral, (b) dorsal and (c) centromedullary. (**d**) Schematic presentation of different lesions types. (**e**) Frequencies of different lesion types."



Sellner, J., Luthi, N., Schupbach, W.M.M., Gebhardt, A., Findling, O., Schroth, G., Mattle, H.P., & Nedeltchev., K. (2009). Diagnostic workup of patients with acute transverse myelitis: Spectrum of clinical presentation, neuroimaging and laboratory findings. *Spinal Cord, 47*(4), 312-317. doi: 10.1038/sc.2008.143.

Pathophysiological Processes

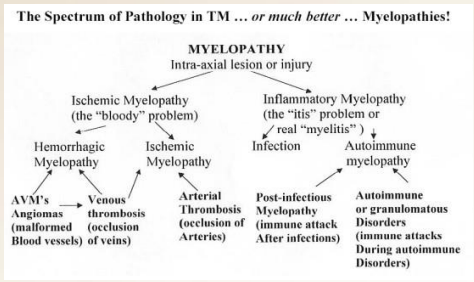
Signs and Symptoms

TM attacks may take three weeks to climax in intensity, however, most patients experience their worst deficits within seven days of the onset of symptoms. Up to 44% of patients have reported viral or bacterial infections prior to TM's classic ascending peripheral sensorimotor losses and autonomic dysfunction. Presentation often includes fever, persistent pain, and parasthesias in the anterior and posterior trunk and periphery, affecting the lower extremities more often than the upper. Maximum effect of symptoms can cause paraplegia and flaccid paralysis. Bowel and bladder dysfunction often accompanies initial presentation (Borchers and Gershwin, 2011). Optic neuritis may also be present. Patients may temporarily recover and relapse at a later time (Brinar, Habek, Brinar, Malojcic, & Boban, 2006).

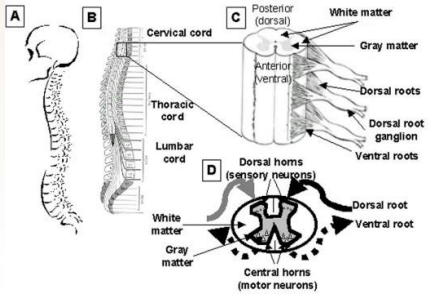
Underlying Pathophysiology

A wide array of pathophysiological processes can cause the "segmental bilateral acute spinal cord" inflammation that results in a TM diagnosis (Bhat, Cheema, Gershwin, & Naguwa, 2009). Prior bacterial or viral infection is present in 50% of cases, with other known causes attributed to various autoimmune diseases that are idiopathic or caused by infections, vaccinations, drug toxicity, spinal cord infarction, demyelinating, inflammatory, autoimmune, paraneoplastic, bacterial, viral, parasitic, or fungal in origin (Bhat et al., 2009; Brinar et al., 2006). Unfortunately, many cases are diagnosed without finding a cause (Jose Sa, 2009).

In most cases of TM, the blood brain barrier is broken down by the infectious agent, resulting in an inflammatory reaction in the CSF and spinal cord with subsequent demyelination (Brinar et al., 2006). Diagnostic criteria show an elevated number of cells present in cerebrospinal fluid (pleocytosis), elevated protein levels without oligoclonal bands, elevated interleukin-6, CF protein level, collapsing-response mediator protein 5-IgG, amphiphysin-IgI, and elevated CSF IgM with later elevated IgG titer or antibodies, and the presence of varying autoantibodies (Bhat et al, 2009; Kitley, Leite, George, & Palace, 2011). SSA/RO antibodies and positive serum aPL are present when TM is associated with lupus (Bhat et al., 2009; Jose Sa, 2009). In cases considered to be viral in origin, infectious agents are not isolated from the nervous system (Bhat et al., 2009). Local demyelinating lesions can be small to large, partial or complete, symmetrical or asymmetrical, and can be found in the spinal cord at varying levels on MRI in 60% of patients; conversely, MRI lesions are not found on 40% of patients (Brinar et al., 2006; Bhat et al., 2009; Jose Sa, 2009).



Pardo, C. A. (2012). The Pathology of Transverse Myelitis. *Transverse Myelitis Association 5(*2), 2. Retrieved from http://myelitis.org/newsletters/v5n2/newsletter5-2-02.htm



Pardo, C. A. (2012). The Pathology of Transverse Myelitis. *Transverse Myelitis Association 5(*2), 2. Retrieved from http://myelitis.org/newsletters/v5n2/newsletter5-2-02.htm

Significance of Pathophysiology

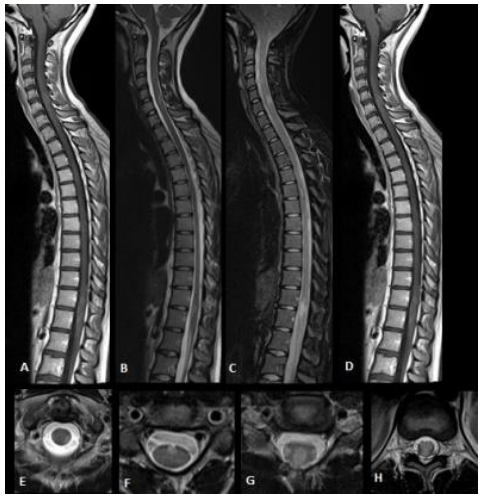
While a rare disease affecting 0.003% of the population (1.34 to 4.6 million people per year), TM deserves special attention because of its potential to physically disable an individual (Bhat et al., 2009; Cree, Hess, & West, 2012). There are multiple etiologies that can cause TM; therefore, identifying the diagnosis itself and discovering the causal etiology can be a complex process, postponing the treatment process (Brinar et al., 2006; Kitley et al., 2011). Delaying the diagnoses may potentially contribute to damaging, lasting effects experienced by two-thirds of cases (Cree et al., 2012; Jose Sa, 2009). Discovering the underlying cause directs treatment options, namely, reversing the inflammatory process via steroid injection, plasma exchange in severe cases, and symptomatic management that accompanies pharmacotherapy and interdisciplinary practices (Jose Sa, 2009).

Implications for Nursing Care

Acute nursing care focuses on thorough patient assessment to detect pathophysiological changes in the disease process. The nurse acts to initiate treatment measures, educate the patient, and monitor daily testing. Accurate assessment and monitoring may detect early changes in patient status, resulting in a needed change in the medical treatment plan (Brinar et al, 2006). Following initial treatment and depending on the intensity of patient symptoms, the nurse's role ranges from supportive care to rehabilitative nursing with a focus on restoring lost function.

Diagnostic Imaging: Figure 2

"Multiple foci of acute partial transverse myelitis: 18 years-old woman presenting progressive paraparesis and sensory loss in right hand. Sagittal spinal cord T1 (A), T2 (B), STIR (C) and T1+Gadolinium (D) show multiple T2 cord hyperintensities affecting less than 3 vertebral bodies with mild and patchy enhancement. Axial T2 images at medullary junction (E), C4 (F), D5 (G) and D11 (H) show partial involvement of the cord section. Brain MRI study was also performed, on axial (I) and parasagittal (J) FLAIR images we can see subcortical white hyperintense lesions, consistent with multiple sclerosis." References: Radiology, CDI, Hospital Clinic de Barcelona - Barcelona/ES."



Ripoll, E., Sarbu, N., Lopez, A., Capurro, S., Oleaga Zufiría, L. (2014). MR imaging of acute non-traumatic spinal cord syndrome. *European Society of Radiology; Electronic Presentation Online System.* DOI: 10.1594/ecr2014/C-0872

Conclusion

TM is a disease of complex etiology but has symptoms that are common to most cases. Attacks can be mild or severe, with effects improving with treatment or permanently disabling the individual. Treatment has been targeted to correct potential underlying causes, reverse spinal cord inflammation, restore physical function, and provide supportive care. Practitioners should think to include TM into the differential diagnoses of cases comprising spontaneous development of sensorimotor debility.

References

Agmon-Levin, N., Kivity, S., Szyper-Kravitz, M., & Shoenfeld, Y. (2009). Transverse myelitis and vaccines: A multi-analysis. *Lupus, 18*(13), 1198-1204. doi: 10.1177/0961203309345730

Bhat, A., Naguwa, S., Cheema, G., Gershwin, E. (2010). The epidemiology of transverse myelitis. *Autoimmunity Reviews 9*(12), 395-399. doi: 10.1016/j.autrev.2009.12.007.

Borchers, A.T., Gershwin, M.E. (2011). Transverse myelitis. *Autoimmunity Reviews 11*(3), 231-248. doi: 10.1016/j.autrev.2011.05.018.

Brinar, V.V., Habek, M., Brinar, M., Branko, M., Boban, M. (2006). The differential diagnosis of acute transverse myelitis. *Clinical Neurology and Neurosurgery, 108*(11), 278-283. doi:10.1016/j.clineuro.2005.11.008.

Kitley, J., Leite, M., George, J., & Palace, J. (2012). The differential diagnosis of longitudinally extensive transverse myelitis. *Multiple Sclerosis (Houndmills, Basingstoke, England), 18*(3), 271-285. doi: 10.1177/1352458511406165.

Pandit, L. (2009). Transverse myelitis spectrum disorders. *Neurology India, 57*(2), 126-133. doi: 10.4103/0028-3886.51278.

Sa, M.J. (2009). Acute transverse myelitis: A practical reappraisal. *Autoimmunity Reviews 9*(2), 128-131. doi: 10.1016/j.autrev.2009.04.005

Sellner, J., Luthi, N., Schupbach, W.M.M., Gebhardt, A., Findling, O., Schroth, G., Mattle, H.P., & Nedeltchev., K. (2009). Diagnostic workup of patients with acute transverse myelitis: Spectrum of clinical presentation, neuroimaging and laboratory findings. *Spinal Cord, 47*(4), 312-317. doi: 10.1038/sc.2008.143.

West., T.W., Hess, C., & Cree, B.A.C. (2012). Acute transverse myelitis: Demyelinating, inflammatory and infectious myelopathies. *Seminars in Neurology, 32*(2), 97-113. doi: 10.1055/s-0032-1322586.

Additional Information

The Johns Hopkins PROJECT RESTORE

THE TRANSVERSE MYELITIS ASSOCIATION

OTTERBEIN UNIVERSITY