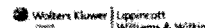


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Immunopathogenesis of acute transverse myelitis.

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Acute transverse myelitis is a group of disorders characterized by focal inflammation of the spinal cord and resultant neural injury. Acute transverse myelitis may be an isolated entity or may occur in the context of multifocal or even multisystemic disease. It is clear that the pathological substrate--injury and dysfunction of neural cells within the spinal cord--may be caused by a variety of immunological mechanisms. For example, in acute transverse myelitis associated with systemic disease (i.e. systemic lupus erythematosus or sarcoidosis), a vasculitic or granulomatous process can often be identified. In idiopathic acute transverse myelitis, there is an intraparenchymal or perivascular cellular influx into the spinal cord, resulting in the breakdown of the blood-brain barrier and variable demyelination and neuronal injury. There are several critical questions that must be answered before we truly understand acute transverse myelitis: (1) What are the various triggers for the inflammatory process that induces neural injury in the spinal cord? (2) What are the cellular and humoral factors that induce this neural injury? and (3) Is there a way to modulate the inflammatory response in order to improve patient outcome? Although much remains to be elucidated about the causes of acute transverse myelitis, tantalizing clues as to the potential immunopathogenic mechanisms in acute transverse myelitis and related inflammatory disorders of the spinal cord have recently emerged. It is the purpose of this review to illustrate recent discoveries that shed light on this topic, relying when necessary on data from related diseases such as acute disseminated encephalomyelitis, Guillain-Barré syndrome and neuromyelitis optica. Developing a further understanding of how the immune system induces neural injury will depend upon confirmation and extension of these findings and will require multicenter collaborative efforts.

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