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MULTIPLE SCLEROSIS: CURRENT REVIEW

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Abstract

This article review our current understanding and modern treatment of multiple sclerosis (MS). MS is a disabling condition resulting in devastating social and economic impacts. As MS can affect any part of the central nervous system, the presentation is often diverse; however, there are key features that can be useful in the clinic. We comment on the diagnostic criteria and review the main subtypes of MS, including clinically isolated syndrome, relapsing remitting MS, secondary progressive MS and primary progressive MS. Although the underlying etiology of MS is still not known, we summarise those with most evidence of association.[1] Finally, we aim to present treatment strategies for managing the acute relapse, disease-modifying therapies and MS symptoms. This review highlights that progressive MS is an area where there is currently a paucity of available disease-modifying treatments and this will be a major focus for future development.

Keywords

Diagnostic criteria, epidemiology, multiple sclerosis, progressive multiple sclerosis, treatments.

MS is an inflammatory disease of the central nervous system (CNS), which causes a heterogeneous array of symptoms and signs because of differential involvement of motor, sensory, visual and autonomic systems. Optic neuritis (inflammation of the optic nerve), Uhthoff's phenomenon (transient fluctuation or worsening of MS symptoms with a rise in body temperature) and Lhermitte's phenomenon (an abnormal electric-shock like sensation down the spine or limbs on neck flexion) are characteristic of MS. MS relapses occur because of focal areas of demyelination evolving over 24 hours and persisting for days or weeks before generally, though not exclusively, improving. There are different phenotypes of MS; here we discuss the most common. Clinically isolated syndrome (CIS) is the initial presentation in 80% of MS cases. CIS encompasses an acute clinical attack affecting one or more CNS sites and can convert to relapsing remitting MS (RRMS). The rate of conversion from CIS to RRMS at 20 years is 21% of patients with a normal MRI scan at baseline versus 82%, if there is one or more clinically silent white matter lesions on MRI. It has been shown that the McDonald 2010 criteria and increased use of MRI are revolutionising the diagnoses of clinically definite MS for the CIS group. [3] With RRMS there is usually good recovery from each clinical episode or relapse. These early stages of MS with underlying demyelination are thought to have an inflammatory pathological substrate with migration of autoreactive lymphocytes across the blood-brain barrier. This triggers a cascade of inflammation with T- and B-cell clonal expansion, and microglia activation and oxidative damage, mitochondrial injury and energy failure, leading to the development of the characteristic plaque. Female: Male sex ratio of MS with relapse-onset is 2:1 and age of onset is around 30 years. Over time, there is accumulation of disability and incomplete recovery from each relapse. 10–15 years after the diagnosis of RRMS, up to 80% of people develop secondary progressive MS (SPMS). There is further axonal injury and atrophy in both white and grey matter, with a likely underlying neurodegenerative pathogenesis, but less inflammation. [4]

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These subtypes of MS should be cross-defined further as active or non-active. If active, there should be occurrence of a clinical relapse or new T2 or gadolinium-enhancing lesions on MRI over at least 1 year.[5]Disease progression is the term used when patients have worsening disability over time compared with those who are worse because of a relapse. 10–15% of patients have progressive disability from the outset, usually due to spinal cord disease. This is defined as primary progressive MS (PPMS). The age of onset is 40 years, roughly a decade older than for relapse onset MS, and the sex ratio is 1:1 in most studies, although there may be a slight male preponderance. Due to spinal cord dysfunction, patients often have a progressive spastic paraparesis. The pathological basis appears to be more diffuse brain axonal loss and further microglial activation with atrophy. There is anterograde and retrograde neurodegeneration with oxidative damage and energy failure. Therefore, SPMS and PPMS are likely to share a common pathological substrate. The etiopathogenesis of the disease is considered as interaction of genetic, epigenetic, exo- and endogenous factors, which leads to demyelination and neurodegeneration. Inflamed- the process can develop both in the direction transfer “from the periphery to the central nervous system” through T-cell mechanisms, and are launched in the central nervous system, through primary damage to brain tissue, and for more later stages - formation of ectopic B-cell follicles and cortical demi- elinization [2–6]. Further improvement understanding of pathogenesis provides insight into increasing the effectiveness of therapy assessed according to NEDA criteria. However, much progress in the treatment of the neurodegenerative component the disease has not yet been achieved, which causes the need to find new approaches to studying changes in brain tissue. At the beginning of this century, a special memagnetic resonance imaging (MRI) technology, allowing to evaluate the activity of neurons in the brain, which is called functional MRI (fMRI) [7]. Functional MRI rest is an fMRI technique in which the limiter The main condition is the absence of motor and cognitive actions (paradigms) on the part of the patient [8]. As an estimated parameter fluctuations in cerebral oxygenation are used blood flow in space and time (BOLD-sig cash). When analyzing this signal, synchronically working areas of the brain, with this fixes the strength of their relationship. These features of the method influenced the choice fMRI to study the features of functional of the central nervous system in patients with MS with autoimmune inflammation and neurodegeneration. Not enough of special interest studied the problem of structural and functional significant changes in the mediobasal regions of the temporal lobe - amygdala complex, hippo- campal region and thalamus, as well as their contribution in the development of the neurodegenerative process and understanding the characteristics of cognitive impairment in patients with MS. The degree of connectivity may become a valuable marker and predictor of damage research of connections between the nuclei of the thalamus and various structures of the limbic system, which will allow highlight the features of their manifestation, including genetic terogenicity of the neurodegenerative process in various types of MS. As for neuroimaging techniques, they use for research purposes justifies given by searching image data for biomarkers in the form of structural and/or functional changes, especially with quantitative assessment, which allows for early identification patients with multiple sclerosis and differential to cause them to have various motor disorders, associated with neurodegeneration. In the diagnosis of various phenotypes, disseminated sclerosis, satisfactory results indicates the use of artificial methods intelligence. Basic visualization techniques include T1-weighted images, T2-weighted enhanced images, diffusion tensor imaging visualization, MR morphometry and functional magnetic resonance imaging. By using various models of machine and deep learning the prospect of overcoming subjective limitations of traditional diagnostic tics, carried out by radiologists.

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