Multiple Sclerosis Symptoms and Diagnosis

Multiple sclerosis (MS) is a demyelinating disorder that demonstrates unclear pathogenesis but has significant evidence of autoimmune involvement. Clinical presentation is variable with subtypes delineated by symptom exacerbation, but it classically presents with Charcot's neurologic triad, which consists of scanning speech, intention tremor, and nystagmus, with symptoms characteristically worsening after a hot shower. However, a wide range of clinical manifestations may be seen, including optic neuritis, internuclear ophthalmoplegia, urinary and fecal incontinence, motor abnormalities such as intention tremor and paresis, sensory changes ranging from pain to numbness, and depression, all of which typically, though not exclusively, display a relapsing pattern. Diagnosis of MS includes the gold standard presence of simultaneous periventricular plaques on MRI, as well as elevated CSF immunoglobulins, most commonly IgG, and finally, identification of oligoclonal IgG bands on immunoelectrophoresis.

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Symptoms

Optic Neuritis

Optics Nerve-on-fire

This inflammatory demyelination of the optic nerve is the presenting symptom in approximately 20% of MS patients, with 50% of MS patients developing optic neuritis at some point in the disease course. It is clinically represented by a classic triad composed of visual loss (commonly in the form of a unilateral, central visual field defect), periocular pain, and dyschromatopsia (deficiency in the ability to perceive colors fully).

Internuclear Ophthalmoplegia

Nuclear-eye-looking Beside Paralyzed Opal-eye

Internuclear ophthalmoplegia is a restrictive gaze defect in which the affected eye displays weakened horizontal eye adduction while the contralateral eye demonstrates abduction nystagmus. More specifically, due to a demyelinating lesion of the medial longitudinal fasciculus (abbreviated MLF, denoted by the alternative name "MLF syndrome"), the motor impulse to the ipsilateral medial rectus muscle from the oculomotor cortex is inadequately propagated, such that adduction of the affected eye is limited and stops near the midline. Meanwhile, the contralateral eye makes it all the way to the lateral periphery of vision, though with nystagmus usually present. Convergence is usually preserved since additional information is relayed that bypasses the MLF and communicates directly with the cranial nerve III nucleus. This usually occurs bilaterally.

Scanning Speech

Scanner-mouth

This type of speech pattern is characterized by inappropriate pauses and breaks between syllables and words that disrupt the typically melodious diction and flow of normal speech. Cerebellar white matter plaques are responsible for causing this disjointed elocution as well as the associated slurring of speech that is often present secondary to weakness and incoordination of speaking musculature.

Urinary and Fecal Incontinence

Urine and Feces in Continents

Exhibiting a similar pathophysiology to the other characteristics presented here, bladder (75% of MS patients) and bowel incontinence (50% of MS patients) are also related to white matter plaque formation in the spinal cord. Most commonly, this manifests as urinary urgency in the setting of unopposed contraction of the detrusor muscle resulting from a suprasegmental lesion.

Motor Issues (Intention Tremor, Paresis)

Motor with Issues (Shaking Wheelchair)

As expected with a demyelinating disorder, motor dysfunction is a significant cause of disability in these patients. The development and progression of motor impairment is highly variable, even within the same clinical subtype. This is the result of white matter plaques in the spinal cord and typically leads to motor deficiencies, such as intention tremors and paresis.

Numbness and Pain

Broken-sensors and Pain-bolts

Sensory changes are the most common presenting symptom in MS, usually described as numbness, tingling, pins-and-needles, or swelling. White matter plaques in the sensory tracts of the spinal cord remain the culprit for this diminished sensation.

Depression

Depressed-emo

Approximately two-thirds of MS patients suffer from some form of affective alteration, with depression as the primary disturbance, while some evidence suggests that rates of depression are even higher in MS than in other chronic medical conditions. This unfortunate development occurs in the setting of an extensive list of comorbidities, including pain, anxiety, fatigue, cognitive impairment, and substance abuse.

Relapsing Symptoms

Relapse-boomerang

There are four primary clinical subtypes of MS: clinically isolated syndrome (CIS), relapsing-remitting (RRMS), 1° progressive (PPMS), and 2° progressive (SPMS). Most MS cases begin with a CIS, which is the initial attack of an inflammatory, demyelinating characteristic such as optic neuritis, transverse myelitis, or a brainstem or spinal cord syndrome. RRMS is defined by relapses (usually 1.5 years in duration) with full recovery or residual deficits upon recovery with minimal disease progression between relapses. Most MS patients will then transition into SPMS, which is not a concrete transition but rather a retrospective diagnosis in the progression of the disease. PPMS is less common and develops in a more continuously deteriorating manner from onset without relapsing-remitting.

Diagnosis

Gold Standard = Plaques on MRI

Gold Plaque with M-R-eyes

A requirement in diagnosing MS is the dissemination of CNS lesions in time and space, which is determined clinically or in conjunction with MRI imaging. The McDonald criteria specify MRI findings that support the diagnosis of MS but are not intended to differentiate MS from other neurological abnormalities. According to the McDonald criteria, dissemination in space is verified by the presence of T2 lesions in at least two of the four classically-associated MS regions (juxtacortical, periventricular, infratentorial, and spinal cord) of the CNS or by the development of another clinical attack indicating the involvement of an additional, alternative CNS site. The McDonald criteria also provides guidance on establishing an MS diagnosis by delineating the dissemination of time. This process can be accomplished by discovering the presence of asymptomatic, simultaneous, enhancing, or non-enhancing lesions on MRI, a new lesion on follow-up MRI, or evidence of a subsequent clinical attack.

Increased CSF Immunoglobulins (IgG)

Up-arrow In-moon-goblins

Patients with MS exhibit an elevation in cerebrospinal fluid (CSF) immunoglobulins, primarily IgG, though IgM and IgA are also increased, indicating intrathecal production. Total protein is typically normal to mildly elevated; however, protein levels can be substantially higher if patients are undergoing a marked relapse.

Oligoclonal Bands

Old-clown Band

When the clinical presentation is unclear, atypical, confounded, or equivocal, CSF assessment undertakes significant importance as it provides helpful diagnostic indication about the current or future presence of MS. Identification of oligoclonal IgG bands is visualized utilizing immunoelectrophoresis and, while their presence is a strong supporter of an MS diagnosis, it is not sufficient for exclusive diagnosis given the rare, yet unacceptably high

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number of false positives found in patients with certain other chronic neurological disorders.

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