classic clinical findings: optic neuritis, paresthesias, INO and bladder symptoms

Visual disturbances

Disturbances of visual acuity may be caused by optic or retrobulbar neuritis which is the presenting symptom of MS in 15% of cases, and which occurs at some time in 50% of MS patients. The percentage of patients with an attack of optic neuritis and no prior attack that will go on to develop MS ranges from 17–87%, depending on the series [1)]. Symptoms: acute visual loss in one or both eyes with mild pain (often on eye movement).

Diplopia may be due to internuclear ophthalmoplegia (INO) from a plaque in the MLF. INO is an important sign because it rarely occurs in other conditions besides MS or brainstem stroke.

Voiding symptoms

50–90% of patients develop voiding symptoms at some point. The demyelination primarily involves the posterior and lateral columns of the cervical spinal cord. Detrusor hyperreflexia is the most common urodynamic abnormality (in 50–99% of cases), with bladder areflexia being less common (5–20%). Patients have DO with DSD without upper tract injury or loss of compliance.

Motor findings

Extremity weakness (mono, para, or quadriparesis) and gait ataxia are among the most common symptoms of MS. Spasticity of the LEs is often due to pyramidal tract involvement. Scanning speech results from cerebellar lesions.

Sensory findings

Posterior column involvement often causes loss of proprioception. Paresthesias of extremities, trunk, or face occur. Lhermitte Sign (electric shock-like pain radiating down the spine on neck flexion) is common, but is not pathognomonic. Trigeminal neuralgia occurs in ≈ 2%, and is more often bilateral and occurs at a younger age than the population in general.

Mental disturbances

Euphoria (la belle indifference) and depression occur in ≈ 50% of patients. Reflex changes Hyperreflexia and Babinski signs are common. Abdominal cutaneous reflexes disappear in 70–80%. GU symptoms Urinary frequency, urgency, and incontinence are common. Impotence in males and reduced libido in either sex is often seen.

The limited efficacy of immunomodulatory treatments (e.g. interferons) for MS, means that more rational, if not, better approaches in the treatment of MS have been sought, such as enhancement of neuroregeneration (e.g. remyelination) via stem cell transplantation. Oligodendrocytes are a good

stem cell source because lineage differentiation of these cells is primarily altered in MS (20,32). Moreover, we hypothesize that transplanting BMDOs loaded with Ephrin (BMDO+Ephrin) may increase migratory capacity of these grafts and eventually promote remyelination, behavioral recovery and improvement of motor functions in MS animals. Of course, several issues remain to be addressed prior to establishing the optimal efficacy of BMDO+Ephrin transplantation in MS animal models. For instance, timing of transplantation after EAE induction is critical for successful functional outcomes. Therefore, early transplantation post- immunization should be performed, especially for chronic EAE. Furthermore, aside from looking into remyelination in white matter tracts of the cerebral hemispheres after BMDO+Ephrin transplantation in animal models of MS, we also need to characterize the effects of these transplants in the spinal cord. It would also be worthwhile to explore other proteins aside from ephrin (e.g. syndecans) that could enhance migration of transplanted oligodendrocytes [2].

1)
Rowland LP. Merritt's Textbook of Neurology. Philadelphia 1989
2)
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