Nmo disease pdf

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add Contributing Editors: add Neuromyelitis Optica/Myelin Oligodendrocytic Glycoprotein. © 2019 Neuro-ophthalmology Virtual Education Library: NOVEL [1] Disclaimer: This article is directed for an Ophthalmology audience, and may not include all non-ocular neurological manifestations of NMO. Neuromyelitis optica, (previously referred to as Devic disease) and now termed neuromyelitis optica spectrum disorders (NMOSD), is an inflammatory, antibody mediated, immunologic disease of the central nervous system that causes demyelination of the optic nerve and spinal cord. Disease Entity Neuromyelitis optica (NMO) is an inflammatory disease that causes demyelination of the central nervous system, primarily affecting the optic nerve (optic neuritis) and the spinal cord. [2] [3] Etiology The precise etiology of NMOSD remains to be completely defined. In the past there was debate as to whether NMO represented a variant of multiple sclerosis (MS), recent evidence however suggests that NMOSD has a completely different pathogenesis, pathology, mechanism of disease, presentation, course, treatment, and prognosis than MS. [3][4] Risk Factors Although NMOSD can occur in any ethnicity, either gender, and any age, the disease has a predilection for females and may be more common in patients of Asian or African descent. [5] General Pathology Segmented demyelination and inflammation of the spinal cord and the optic nerves inducing axonal loss and perivascular lymphocytic infiltration. Pathophysiology NMOSD is primarily an astrocytopathy. It involves demyelination and inflammation of multiple spinal cord segments and the optic nerves.[6] NMOSD produces significant axonal loss associated with perivascular lymphocytic infiltration and vascular proliferation.[6] Necrosis in NMOSD usually involves both gray and white matter, which is distinct from multiple sclerosis.[6] The pathophysiology of NMOSD mainly involves the humoral immune system.[6] NMO is characterized by a disease specific IgG antibody against the astrocytic aquaporin 4 (AQP4) water channel (also known as the aquaporin-4 autoantibody (anti-AQP4 or AQP4-IgG).[7][8] The AQP4 water channel membrane protein is concentrated in the optic nerve, area postrema, and spinal cord.[9] AQP4 rich areas of the CNS account for the clinical findings of NMOSD. The proposed pathophysiology involves anti-AQP4 autoantibodies that are peripherally produced entering the CNS and binding astrocyte foot processes, which then induces complement mediated cell damage, granulocyte infiltration, and astrocyte death.[8] Death of astrocytes induces secondary death of oligodendrocytes, resulting in demyelination and ultimately neuronal cell death.[8] Local CNS water imbalance results in oligodendrocyte damage and demyelinization.[2] The loss of AQP4 immunoreactivity and the astrocyte pathology in the brain and spinal cord lesions distinguish NMOSD lesions from multiple sclerosis (MS) lesions.[7] There are still unknown elements in the pathophysiology of NMOSD including the mechanism for loss of tolerance and anti-AQP4 formation, pathogenesis of seronegative NMOSD, and the mechanisms that anti-AQP4 breach the blood brain barrier.[8] Diagnosis The first association between myelitis and optic nerve disorder was reported in 1870 by Sir Thomas Clifford Allbutt. His case reports were vague and no pathology was documented [10] Almost 80 years later, Stansbury published a thorough review on NMO and afterwards accepted as a separate entity from MS. [4] Ocular Signs and Symptoms Interestingly, patients can initially present with an acute flu-like illness (fever, myalgia, and headache). Later, more suggestive and specific signs and symptoms of NMO may start to develop including optic neuritis or myelitis. Ophthalmologic examination may be within normal limits in asymptomatic or pre-symptomatic patients with NMOSD. Patients with optic neuritis related to NMO often present acutely with optic disc swelling[8] ,however can develop optic atrophy and. Central optic nerve cavitation can be seen as a sequela of demyelination and necrosis in more severe cases.[11] Patients with optic neuritis in NMOSD typically present with decreased visual acuity, visual field, or color vision (red desaturation).[8] A relative afferent pupillary defect may be seen in unilateral or bilateral but asymmetric optic nerve involvement.[8] Spinal cord involvement manifestations are paraparesis or tetraparesis as well as sphincter dysfunction. [11] As opposed to MS related optic neuritis, patients with NMOSD may have a worse visual prognosis after optic neuritis and many patients are left with residual visual disability.[10] NMOSD typically follows a relapsing and chronic course[9] with recurrent acute attacks of transverse myelitis and/or unilateral or bilateral optic neuritis with only partial or no recovery.[8] Transverse myelitis in NMOSD is frequently severe causing a complete spinal cord syndrome involving all three major neurological pathways (motor, sensory, and autonomic). This may result in permanent symptoms and signs (e.g., paraparesis or quadriparesis, paroxysmal tonic spasms, bladder dysfunction, and sensory loss) as well as radiographic progression to spinal cord atrophy.[7][8][9] Although the optic neuritis in NMOSD superficially may resemble MS, NMOSD related optic neuritis tends to be more severe, more extensive, more likely to be bilateral, recurrent and be less likely to recover than the optic neuritis seen in MS.[8] The optic neuritis seen in NMOSD is more likely to have rapidly sequential or simultaneous bilateral involvement and as well as involve the optic chiasm. Many patients with or without treatment are left with severe residual visual loss with acuity 20/200 or worse.[7][8] An area postrema syndrome may also develop in NMOSD due to involvement of the medulla and manifests as intractable hiccups or nausea and vomiting and symptomatic narcolepsy.[7][8] Diagnosis There are different diagnostic criteria for NMOSD with serum positive AQP4-IgG and NMOSD without serum positive AQP4-IgG detailed by the International consensus diagnostic criteria for NMOSD.[7] Diagnostic criteria for NMOSD with serum positive AQP4-IgG:[7] At least 1 core clinical characteristic[7] Positive test for AQP4-IgG using best available detection method[7] Exclusion of alternative diagnoses[7] Diagnostic criteria for NMOSD without AQP4-IgG or NMOSD with unknown AQP4-IgG status:[7] At least 2 core clinical characteristics occurring as a result of one or more clinical attacks and meeting all of the following requirements:[7] At least 1 core clinical characteristic must be optic neuritis, acute myelitis with longitudinally extensive transverse myelitis lesions (LETM), or area postrema syndrome[7] Dissemination in space (2 or more different core clinical characteristics)[7] Fulfillment of additional MRI requirements as applicable[7] Negative tests for AQP4-IgG using best available detection method, or testing unavailable[7] Exclusion of alternative diagnoses[7] Core clinical characteristics:[7] Optic neuritis[7] Acute myelitis[7] Area postrema syndrome: episode of otherwise unexplained hiccups or nausea and vomiting[7] Acute brainstem syndrome[7] Symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions[7] Symptomatic cerebral syndrome with NMOSD-typical brain lesions[7] Additional MRI requirements for NMOSD without AQP4-IgG or NMOSD with unknown AQP4-IgG status:[7] Acute optic neuritis: requires brain MRI showing (a) normal findings or only nonspecific white matter lesions, OR (b) optic nerve MRI with T2-hyperintense lesion or T1-weighted gadolinium enhancing lesion extending over >1/2 optic nerve length or involving optic chiasm[7] Acute myelitis: requires associated intramedullary MRI lesion extending over > 3 contiguous segments (LETM) OR >3 contiguous segments of focal spinal cord atrophy in patients with history compatible with acute myelitis[7] Area postrema syndrome: requires associated dorsal medulla/area postrema lesions[7] Acute brainstem syndrome: requires associated peri ependymal brainstem lesions[7] NMOSD may coexist with Systemic Lupus Erythematosus, Sjögren Syndrome, or myasthenia gravis and presence of these diagnoses tends to strengthen the confidence in NMOSD diagnosis.[7] Laboratory test AQP4 antibody serum levels are not only specific to NMO, but also correlate with the degree of disease activity. Testing of AQP4 is recommended during an acute attack and before starting immunosuppressive therapy.[11] Testing for serum AQP4-IgG is recommended to be done with cell-based serum assays (microscopy or flow cytometry-based detection) since they optimize autoantibody detection and have the best sensitivity and specificity.[7] Indirect immunofluorescence assays and ELISAs are sometimes used due to cell based assays not yet being widely available.[7] However, they have a lower sensitivity compared to cell based assays and occasionally yield false positive results, so interpretive caution is necessary.[7] Confirmatory testing using 1 or more AQP4-IgG assay techniques is recommended in equivocal or seronegative but clinically/radiographically suggestive cases of NMOSD.[7] A small number of patients with clinical characteristics of NMOSD, mostly all AQP4-IgG seronegative, have detectable serum myelin oligodendrocyte glycoprotein (MOG) antibodies.[7] Lack of CSF oligoclonal bands support a diagnosis of NMOSD over a diagnosis of MS, but they can be transiently detectable during an attack in NMOSD.[7] CSF pleocytosis with >50 leukocytes/microliter or presence of neutrophils or eosinophils are useful in distinguishing NMOSD from MS.[7] Neuroimaging An MRI demonstrating specific lesion patterns is an important factor in NMOSD diagnosis.[7] Certain brain, optic nerve, and spinal cord patterns are characteristic of NMOSD and detection of a LETM spinal cord lesion associated with acute myelitis is the most specific neuroimaging characteristic of NMOSD.[7] These MRI lesions usually involve the central gray matter and are associated with cord swelling, central hypointensity on T1 weighted sequences, and enhancement following IV gadolinium administration.[7] Cervical lesions in NMOSD characteristically extend into the brainstem.[7] Cord lesions in MS differ from NMOSD in that they are typically 1 vertebral segment long or less, occupy peripheral white matter tracts, and may be asymptomatic.[7] The International consensus diagnostic criteria for NMOSD details neuroimaging characteristics of NMOSD:[7] Spinal cord MRI, acute[7] LETM lesion associated with acute TM[7] Increased signal on sagittal T2-weighted (standard T2-weighted, proton density, or STIR sequences) extending over 3 or more complete vertebral segments[7] Central cord predominance (more than 70% of the lesion residing within the central gray matter)[7] Gadolinium enhancement of the lesion on T1-weighted sequences (no specific distribution or pattern of enhancement is required)[7] Other characteristic features that may be detected[7] Rostral extension of the lesion into the brainstem[7] Cord expansion/swelling[7] Decreased signal on T1-weighted sequences corresponding to region of increased T2-weighted signal[7] Spinal cord MRI, chronic[7] Longitudinally extensive cord atrophy (sharply demarcated atrophy extending over > 3 complete, contiguous vertebral segments and caudal to a particular segment of the spinal cord), with or without focal or diffuse T2 signal change involving the atrophic segment[7] Optic nerve MRI[7] Unilateral or bilateral increased T2 signal or T1 gadolinium enhancement within optic nerve or optic chiasm; relatively long lesions (e.g., those extending more than half the distance from orbit to chiasm) and those involving the posterior aspects of the optic nerves or the chiasm are associated with NMO[7] Cerebral MRI: NMOSD-typical brain lesion patterns (increased signal on T2-weighted MRI sequences unless otherwise noted) [7] Lesions involving the dorsal medulla (especially the area postrema), either small and localized, often bilateral, or contiguous with an upper cervical spinal cord lesion[7] Peri ependymal surfaces of the fourth ventricle in the brainstem/cerebellum[7] Lesions involving the hypothalamus, thalamus, or peri ependymal surfaces of the third ventricle[7] Large, confluent, unilateral, or bilateral subcortical or deep white matter lesions[7] Long (1/2 of the length of the corpus callosum or greater), diffuse, heterogeneous, or edematous corpus callosum lesions[7] Long corticospinal tract lesions, unilateral or bilateral, contiguously involving internal capsule and cerebral peduncle[7] Extensive peri ependymal brain lesions, often with gadolinium enhancement[7] Differential diagnosis Management Initial treatment for patients with suspected NMO or patients with acute attacks is intravenous glucocorticoids. Prevention of recurrent attacks is treated with long-term immunosuppression. AQP4-IgG seropositive patients are assumed to be at risk for relapse indefinitely and preventive treatment should be considered, even in patients with prolonged clinical remission.[7] General treatment Guidelines for NMO management have been difficult to establish, since most studies involve a small number of patients. Treatment for acute episodes consists mainly of steroids (methylprednisolone 500-1000mg daily for 5-10 days) followed by plasmapheresis or intravenous immunoglobulin.[10] Eculizumab and inebilizumab are humanized antibodies that have been studied in randomized controlled trials in patients with NMOSD and have shown efficacy in long term treatment.[12][13] Other immunotherapies have also been used for long term management of NMOSD such as azathioprine, mycophenolate mofetil, rituximab, methotrexate, mitoxantrone, tocilizumab, and oral glucocorticoids.[8][14] Medical follow up Side effects such as hepatotoxicity, immunosuppression, lymphoma and other malignancies should be evaluated in patients receiving these medications [10]. Complications Permanent myelopathy and blindness can occur in NMOSD even after an initially monophasic course. [11]. Prognosis Patients with NMOSD have a variable prognosis with many patients suffering high levels of disability.[8] One study demonstrated that only 22% of patients had full recovery but 6% showed no recovery at all.[8] Severe visual defects or motor impairment is present in about half of patients within 5 years of disease onset.[8] Disease related mortality in NMOSD is most commonly due to neurogenic respiratory failure.[8] References

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