OPTIC NEURITIS & NEUROMYELITIS OPTICA: AN UPDATE

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INFLAMMATORY OPTIC NEUROPATHIES
OPTIC NEUROPATHY

CONGENITAL

GLAUCOMA

ISCHEMIC

OPTIC NERVE HYPOPLASIA

POAG

NTG

NON-ARTERITIC

ARTERITIC

OTHERS

INFLAMMATORY

DEMYELINATING

WITH ANTIBODY, WITHOUT A SYSTEMIC CONDITION

WITH ANTIBODY, WITH A SYSTEMIC CONDITION

WITHOUT ANTIBODY, WITHOUT A SYSTEMIC CONDITION

WITHOUT ANTIBODY, WITH A SYSTEMIC CONDITION

NON-IMMUNE MEDIATED

IMMUNE MEDIATED

INFECTIONOUS

SYMPHILIS

TUBERCULOSIS

LYME DISEASE

EHRLICHIOSIS, BARTONELLOSIS, TOXOPLASMOsis Q FEVER

HV, OTHER VIRUS

AUTO-IMMUNE OPTIC NEUROPATHY (AON)

MENINGIOMA

GLIOMA

PITUITARY TUMORS

ANEURYSM

SINUS MUCOCELE

ORBITOPATHIES

METASTASIS

LEUKEMIA

LYMPHOMA

OTHERS

COMPRESSIVE/INFLTRATIVE

TRAUMATIC

GENETIC

NUTRITIONAL/TOXIC

RADIATION-INDUCED

DOA

LHON

DEGENERATIVE (SCA, FA)

VITAMIN B-12 DEFICIENCY

METHANOL ETHYLENE GLYCOL

DRUGS
CASE:  JB

PRESENTATION

• 26 YO WOMAN
• PRESENTS WITH 2 WEEK HISTORY OF VISION LOSS OS
• SENSATION THAT THE EYE WAS SWOLLEN OS
• PMHX: CONTACT DERMATITIS
• POCHX: UNREMARKABLE
• NO REGULAR MEDICATIONS
• ALLERGIES: ENVIRONMENTAL
• SHX: NONE.

EXAMINATION

• 20/20 OD, 20/60 OS
• CP: 16/16 OD AND 8/16 OS
• LEFT AFFERENT PUPILLARY DEFECT
• SLEX: NORMAL
CLINICAL DIAGNOSIS OF DE MYELINATING OPTIC NEURITIS

1) ACUTE MANAGEMENT STRATEGIES FOR CURRENT OPTIC NEURITIS

2) RELATIONSHIP BETWEEN CURRENT OPTIC NEURITIS TO MULTIPLE SCLEROSIS
CLINICAL DIAGNOSIS OF DEMYELINATING OPTIC NEURITIS

- OPTIC NEURITIS TREATMENT TRIAL (ONTT):
- COMPARED ORAL PREDNISONE (1 MG/KG/D X 14 DAYS) VS. IV METHYL PREDNISOLONE VS. ORAL PLACEBO
- NO LASTING DIFFERENCES IN AFFERENT VISUAL FUNCTION
- VISION FUNCTION RECOVERED FASTER IN THE GROUP RECEIVING IV METHYL PREDNISOLONE
- PARTICIPANTS GIVEN ORAL PREDNISONE GROUP SUFFERED A HIGHER RATE OF NEW ATTACKS OF OPTIC NEURITIS THAN PATIENTS IN EITHER OF THE OTHER TWO GROUPS
<table>
<thead>
<tr>
<th>Test</th>
<th>Placebo (N=133), Median Score (25th, 75th Quartiles)</th>
<th>Intravenous (N=137), Median Score (25th, 75th Quartiles)</th>
<th>Prednisone (N=139), Median Score (25th, 75th Quartiles)</th>
<th>P*</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual acuity</td>
<td>20/16 (20/20, 20/13)</td>
<td>20/16 (20/20, 20/13)</td>
<td>20/16 (20/25, 20/13)</td>
<td>.68</td>
<td>.25</td>
</tr>
<tr>
<td>Contrast sensitivity</td>
<td>14 (14, 15)</td>
<td>14 (14, 15)</td>
<td>14 (13, 15)</td>
<td>.59</td>
<td>.71</td>
</tr>
<tr>
<td>Visual field</td>
<td>(-1.45 (-3.21, -0.48))</td>
<td>(-1.63 (-3.35, -0.05))</td>
<td>(-1.90 (-3.97, -0.44))</td>
<td>.75</td>
<td>.41</td>
</tr>
<tr>
<td>Color vision</td>
<td>79.9 (47.6, 140.9)</td>
<td>76.0 (44.0, 119.9)</td>
<td>71.1 (39.4, 153.0)</td>
<td>.15</td>
<td>.37</td>
</tr>
</tbody>
</table>

*P values are for Wilcoxon Rank-Sum tests, adjusted by baseline visual acuity, comparing each steroid group with the placebo group.

†Visual acuity data are presented as Snellen equivalents, contrast sensitivity data as Pelli-Robson chart line numbers, visual field data as Humphrey Field Analyzer (Allegan Humphrey, San Leandro, Calif) mean deviations in decibels, and color vision data as Farnsworth-Munsell 100 hue-test error scores.

(Beck et al., 1993)
ONTT – 15 YEAR FOLLOW-UP

- The long term prognosis of vision is good
- The treatment received did not influence long-term visual outcome
<table>
<thead>
<tr>
<th>Measure</th>
<th>Placebo (n = 102)</th>
<th>Intravenous (n = 91)</th>
<th>Prednisone (n = 101)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visual acuity (logMAR)</td>
<td>−0.01 (−0.10 to 0.06)</td>
<td>−0.04 (−0.10 to 0.06)</td>
<td>−0.02 (−0.10 to 0.08)</td>
</tr>
<tr>
<td>Contrast sensitivity</td>
<td>1.65 (1.50–1.65)</td>
<td>1.65 (1.50–1.65)</td>
<td>1.65 (1.35–1.65)</td>
</tr>
<tr>
<td>(log units)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Visual field (mean</td>
<td>−1.20 (−3.06 to −0.26)</td>
<td>−1.28 (−2.99 to 0.30)</td>
<td>−1.23 (−3.52 to −0.48)</td>
</tr>
<tr>
<td>deviation)</td>
<td></td>
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</tbody>
</table>

(ONTT, 2008)
A RANDOMIZED, CONTROLLED TRIAL OF ORAL HIGH-DOSE METHYLPREDNISOLONE IN ACUTE OPTIC NEURITIS

500 MG MP DAILY X 5 DAYS WITH 10 DAY TAPER

SIMILAR FINAL VISUAL FUNCTION BUT FASTER RECOVERY WITH METHYLPREDNISOLONE

(Sellebjerg et al., 1999)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Methylprednisolone group, n (%)</th>
<th>Placebo group, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal visual acuity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 wk</td>
<td>2/28 (7)</td>
<td>3/30 (10)</td>
</tr>
<tr>
<td>3 wk</td>
<td>10/30 (33)</td>
<td>8/29 (28)</td>
</tr>
<tr>
<td>8 wk</td>
<td>14/28 (50)</td>
<td>13/30 (43)</td>
</tr>
<tr>
<td>Normal contrast sensitivity</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 wk</td>
<td>1/28 (4)</td>
<td>1/30 (3)</td>
</tr>
<tr>
<td>3 wk</td>
<td>6/30 (20)</td>
<td>5/29 (17)</td>
</tr>
<tr>
<td>8 wk</td>
<td>11/28 (39)</td>
<td>8/30 (27)</td>
</tr>
<tr>
<td>Normal color vision</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 wk</td>
<td>6/28 (21)</td>
<td>5/30 (17)</td>
</tr>
<tr>
<td>3 wk</td>
<td>14/30 (47)</td>
<td>8/29 (28)</td>
</tr>
<tr>
<td>8 wk</td>
<td>17/28 (61)</td>
<td>12/30 (40)</td>
</tr>
<tr>
<td>Improvement of visual functional system of the EDSS</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 wk</td>
<td>15/28 (54)</td>
<td>12/30 (40)</td>
</tr>
<tr>
<td>3 wk</td>
<td>24/30 (80)</td>
<td>23/29 (79)</td>
</tr>
<tr>
<td>8 wk</td>
<td>23/28 (82)</td>
<td>28/30 (93)</td>
</tr>
</tbody>
</table>

EDSS = Expanded Disability Status Scale.
ORAL VS. IV STEROIDS

• COCHRANE REVIEW (2015):
  • 6 DIFFERENT STUDIES, 750 PARTICIPANTS
  • IV STEROIDS DO NOT IMPROVE VISUAL OUTCOMES
  • ORAL STEROIDS (ALL DOSES) DO NOT IMPROVE VISUAL OUTCOMES
  • LIMITED ANALYSIS: UNABLE TO COMPARE HIGH-DOSE ORAL AND IV STEROID TREATMENT
PHENYTOIN IN DEMYELINATING OPTIC NEURITIS

- OPTIC NEURITIS, CLINICAL DIAGNOSIS
- DURATION OF 14 DAYS OR LESS
- TREATED WITH LOADING AND THEN MAINTENANCE DOSE OF PHENYTOIN
- DID NOT EXCLUDE: USE OF STEROIDS, DMD OR MS
- OCT WAS THE OUTCOME MEASURE – RNFL AND MACULAR VOLUME

(Raftopoulous et al., 2016)
• NATIONAL, RANDOMIZED TRIAL

• COMPARE ERYTHROPOIETIN VS. PLACEBO, GIVEN IN ADDITION TO METHYLПREDNISOLONE

• INCLUSION: FIRST EPISODE OF OPTIC NEURITIS, 10 DAYS

• EXCLUSION: PREVIOUS OPTIC NEURITIS, DIAGNOSIS OF MS

• TREATMENT: 3 DAY TREATMENT

• TRIAL STARTED SEPTEMBER 2014, EXPECTED DURATION 30 MONTHS

BMJ Open Treatment of optic neuritis with erythropoietin (TONE): a randomised, double-blind, placebo-controlled trial—study protocol
DEMYELINATING OPTIC NEURITIS

• OPTIC NEURITIS TREATMENT TRIAL UPDATE:

• THE ASSOCIATION BETWEEN OPTIC NEURITIS & MS IS SIGNIFICANT

• RISK STRATIFICATION:
  1. MRI
  2. CLINICAL EXAMINATION
DEMYELINATING OPTIC NEURITIS

• OPTIC NEURITIS TREATMENT TRIAL:

• THE ASSOCIATION BETWEEN OPTIC NEURITIS & MS IS SIGNIFICANT

• RISK STRATIFICATION:
  1. MRI

  NO MRI LESIONS: 22% RISK OF MS IN 10 YEARS

  ≥ 1 MRI LESION: 56% RISK OF MS IN 10 YEARS
**TABLE 4: The 2010 McDonald Criteria for Diagnosis of MS**

| 1 attack\(^a\); objective clinical evidence of \(\geq 2\) lesions | Dissemination in time, demonstrated by:  
Simultaneous presence of asymptomatic gadolinium-enhancing and nonenhancing lesions at any time; or  
A new T2 and/or gadolinium-enhancing lesion(s) on follow-up MRI, irrespective of its timing with reference to a baseline scan; or  
Await a second clinical attack\(^a\) |
|---|---|
| 1 attack\(^a\); objective clinical evidence of 1 lesion (clinically isolated syndrome) | Dissemination in space and time, demonstrated by:  
For DIS:  
\(\geq 1\) T2 lesion in at least 2 of 4 MS-typical regions of the CNS (periventricular, juxtacortical, infratentorial, or spinal cord)\(^d\); or  
Await a second clinical attack\(^a\) implicating a different CNS site; and  
For DIT:  
Simultaneous presence of asymptomatic gadolinium-enhancing and nonenhancing lesions at any time; or  
A new T2 and/or gadolinium-enhancing lesion(s) on follow-up MRI, irrespective of its timing with reference to a baseline scan; or  
Await a second clinical attack\(^a\) |

*(Polman et al., Diagnostic criteria for Multiple Sclerosis, 2010 revisions to McDonald Criteria)*
FURTHER STRATIFICATION

• IF NO LESIONS SEEN ON MRI, REDUCED RISK OF DEVELOPING MS BASED ON CLINICAL EXAMINATION:
  • MALE (HR: 0.35)
  • OPTIC NERVE SWELLING (HR: 0.41)
  • MS DID NOT DEVELOP IF: PAINLESS, NLP VISION, DISC HEMORRHAGES OR EXUDATES

• THESE FACTORS DO NOT SEEM TO INFLUENCE THE RISK OF MS IN INDIVIDUALS WITH MRI LESIONS
SO FAR

• INFLAMMATORY OPTIC NEUROPATHIES
  • OPTIC NEURITIS
    • FINAL UPDATE FROM ONTT
    • TRIALS OF TREATMENT STRATEGIES FOR ACUTE DEMYELINATING OPTIC NEURITIS [ORAL VS. IV STEROIDS; PHENYTOIN]
CASE: YT

PRESENTATION

• 52 YO WOMAN
• 3 WEEK HISTORY OF EYE PAIN OS
• NOTICED A CHANGE IN HER VISION – “BLURRED ALL OVER WITH CENTRAL SLIT”
• HEADACHE / PAIN AROUND THE EYE
• PMHX: HYPOTHYROID (S/P RAI FOR HYPERTHYROID)

EXAMINATION

• 20/20 OD AND HM OS
• CP: 16/16 OD AND UNABLE OS
• LEFT RAPD
• ANTERIOR SEGMENT: UNREMARKABLE
ATYPICAL FEATURES:

✓ PAIN FOR > 2 WEEKS
✓ SEVERE VISION LOSS
✓ PROGRESSIVE VISION LOSS

Box 1 | Red flags implying diagnosis other than MSON

Atypical clinical presentation
- Pain or loss of vision presenting for more than 2 weeks
- Absence of pain
- Retinal abnormalities
- Unexplained optic atrophy
- Severe loss of vision in patients with a non-white ethnic background
- Severe loss of vision without early recovery

Atypical course
- Progressive loss of vision
- Absence of recovery for more than 3 months
- Worsening of visual function after reducing or stopping steroids or immunosuppression

Bilateral optic neuritis*

Past medical history of cancer
*Simultaneous binocular visual loss must be distinguished from sequential bilateral visual loss. Both visual acuity and visual field need to be documented; sole assessment of visual acuity may miss a peripheral visual field defect. Abbreviation: MSON, multiple sclerosis-associated optic neuritis.
Optic neuritis

Typical
(Pain, retrobulbar or mild disc swelling, visual loss does not progress beyond 2 weeks, age 20–50 years)

Atypical
(No pain, retinal exudates, retinal haemorrhages, severe disc swelling, no visual recovery after 1 month)

Consider

Ischaemic optic neuropathy
Infiltrative optic neuropathy
Inflammatory (sarcoid, lupus)
Infection (syphilis, lyme, viral, cat scratch)
Compressive optic neuropathy

Brain MRI

Normal

Abnormal (consistent with demyelination)

Consider intravenous corticosteroids on case-by-case basis

Intravenous corticosteroids, consider immunomodulatory therapy

Magnetic resonance and serological studies as appropriate

(Frohman et al., 2005)
<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Features</th>
<th>Differential diagnoses</th>
<th>Paraclinical tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Subacute or progressive visual loss following exposure to infectious agent; frequently with broader cellular reaction in the eye</td>
<td>Spirochaetes (syphilis, Lyme)</td>
<td>Serology, PCR, CSF, MRI</td>
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<tr>
<td></td>
<td></td>
<td>HIV</td>
<td>MRI, serology</td>
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<td></td>
<td></td>
<td>Bartonella henselae, neurocysticercosis, tuberculosis</td>
<td>Chest radiography, serology, CSF, MRI</td>
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<td>Sinus pain</td>
<td>Vertebral sinusis*</td>
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<tr>
<td>Reactive</td>
<td>Bilateral and simultaneous; often in childhood and then mostly good prognosis. In contrast, in adults the outcome is more frequently poor</td>
<td>Post-infectious*</td>
<td>Serology, CSF</td>
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<tr>
<td></td>
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<td>Post-vaccination</td>
<td>OCT, ERG</td>
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<td></td>
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<td>Acute disseminated encephalomyelitis (ADEM)</td>
<td>MRI</td>
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<td></td>
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<td>Neurontinitis</td>
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<tr>
<td>Vascular</td>
<td>Sudden-onset visual loss, mostly painless (exception GCA); acutely swollen optic disc (except PION); cardiovascular risk factors</td>
<td>AION</td>
<td>ESR, CRP, glucose</td>
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<tr>
<td></td>
<td></td>
<td>PION</td>
<td>Coagulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>GCA</td>
<td>Biopsy</td>
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<tr>
<td></td>
<td></td>
<td>Diabetic papulopathy</td>
<td>ECG, Doppler ultrasound</td>
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<td></td>
<td></td>
<td>Retinal vasospasms</td>
<td>ECG, Doppler ultrasound</td>
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<tr>
<td></td>
<td></td>
<td>susac syndrome</td>
<td>Audiogram, OCT, CSF, visual fields</td>
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<tr>
<td></td>
<td></td>
<td>CCF</td>
<td>Orbital burst, CTA/MRA</td>
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<tr>
<td>Seizures, neurological</td>
<td>Vascular malformations</td>
<td></td>
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</tr>
<tr>
<td>Nutritional and toxic</td>
<td>Bilateral, painless, progressive; evidence is emerging that cobalt toxicity or joint implants containing cobalt. Pale discs, poor prognosis</td>
<td>Vitamin B12 deficiency</td>
<td>Vitamin B12, MMA</td>
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<tr>
<td></td>
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<td>Tobacco-alcohol, toxic</td>
<td>Full blood, cobalt levels</td>
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<tr>
<td></td>
<td></td>
<td>Endemic</td>
<td>OCT, visual fields</td>
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<td></td>
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<td>Methanol, ethambutol, ethylene glycol</td>
<td>Plasma osmolar gap, ethylene glycol, glycolic acid, formate</td>
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<tr>
<td>Compressive</td>
<td>Painless, progressive, pale disc at presentation, cilioretinal shunt vessels, history of cancer; proptosis, lid lag, diplopia, history of thyroid disease</td>
<td>Primary tumours, metastases, tuberculosis, sinus mucoceles, Graves disease</td>
<td>OCT or MRI, orbits and brain with contrast, MRA, OCT, biopsy, antibiody antibodies (Graves disease only)</td>
</tr>
<tr>
<td>Systemic disease</td>
<td>Painful, progressive and often bilateral, more frequent in non-whites, subacute visual loss, history of migraine</td>
<td>All diagnoses</td>
<td>Brain and orbits with contrast</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sarcoïdiasis</td>
<td>ACE, CSF, biopsy (sarcoïd)</td>
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<td></td>
<td></td>
<td>Behèt disease</td>
<td>OCT, chest radiography, MRI</td>
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<td>SLE</td>
<td>Coagulation, if ANA-positive search for specific antibodies</td>
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<td></td>
<td></td>
<td>Cancer</td>
<td>Paraneoplastic antibodies</td>
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<td></td>
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<td>Persistent migraneous visual aura</td>
<td>Further tests not part of routine work-up</td>
</tr>
<tr>
<td>Ocular</td>
<td>Pain</td>
<td>Posterior scintis</td>
<td>OCT, ultrasound, ANCA</td>
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<td></td>
<td></td>
<td>Painless, metamorphosis</td>
<td>OCT, ERG</td>
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<td></td>
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<td>Preserved colour vision</td>
<td>Fluorescen angiogram</td>
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<td></td>
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<td>Visual field loss, photopsias</td>
<td>Big blind spot syndromes</td>
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<tr>
<td></td>
<td></td>
<td>Preserved colour vision</td>
<td>Visual field, OCT</td>
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<tr>
<td>Hereditary</td>
<td>Family history, Bilateral, painless</td>
<td>LHON, OPA1 and OPA3 mutations</td>
<td>Genetic testing</td>
</tr>
</tbody>
</table>
EVALUATION

- **BLOOD WORK:** RPR - NON-REACTIVE ANCA AND ANA NEGATIVE / ACE = 26 (NORMAL), CALCIUM = 2.40 (NORMAL) CRP = 2.6 CBC = NORMAL
- **MRI BRAIN AND ORBITS (WITH AND WITHOUT GD):** INCREASED SIGNAL ON T2/FLAIR AND POST-GD ENHANCEMENT, OF THE LEFT OPTIC NERVE. LACK OF PARENCHYMAL LESIONS.
- **NMO ANTIBODY:** POSITIVE
NEUROMYELITIS OPTICA

INFLAMMATORY DISORDER OF THE CNS – INITIALLY FELT TO BE RELATED TO MS BUT NOW IDENTIFIED AS A DISTINCT CLINICAL AND PATHOPHYSIOLOGIC ENTITY

AQUAPORIN-4, WATER CHANNEL ON THE ASTROCYTIC FOOD PROCESSES EXPRESSED IN OPTIC NERVE, BRAINSTEM & SPINAL CORD

AQUAPORIN-4 ANTIBODY PATHOGENIC IN NMO
NMO & NMO SPECTRUM DISORDER (NMOSD)

REVISED DIAGNOSTIC CRITERIA, 2015

• CORE CLINICAL CHARACTERISTICS:
  • OPTIC NEURITIS:
    • SIMULTANEOUS, BILATERAL OR SEVERE WITH POOR RECOVERY (20/200 OR WORSE)
    • NON-CENTRAL SCOTOMAS
  • EXTENSIVE OPTIC NERVE INVOLVEMENT ON MRI (POSTERIOR OPTIC NERVE AND CHIASM)
  • TRANSVERSE MYELITIS
  • AREA POSTREMA SYNDROME
  • ACUTE BRAINSTEM SYNDROME
  • SYMPTOMATIC NARCOLEPSY/ACUTE DIENCEPHALIC SYNDROME
NMO SPECTRUM DISORDER: 2015 DIAGNOSTIC CRITERIA

NMOSD with AQP4-IgG
1. At least 1 core clinical characteristic
2. Positive test for AQP4-IgG using best available detection method*
3. Exclusion of alternative diagnoses

NMOSD without AQP4-IgG
1. At least 2 core clinical characteristics occurring as a result of one or more clinical attacks and meeting all of the following requirements
   a. At least 1 core clinical characteristic must be optic neuritis, acute myelitis with LETM, or area postrema syndrome
   b. Dissemination in space (2 or more different core clinical characteristics)
   c. Additional MRI requirements, as applicable
2. Negative tests for AQP4-IgG using best available detection method* or testing unavailable
3. Exclusion of alternative diagnoses
TREATMENT

• ACUTE EXACERBATIONS:
  • IV STEROIDS (IVMP X 5 DAYS) WITH LONG TAPER 2 – 5 MONTHS
  • PLASMA PHARESIS
  • IVIG (LESS OFTEN)

• DISEASE MODIFYING THERAPY:
  • NO PROSPECTIVE, RANDOMIZED CONTROLLED TRIAL
  • PREVENT RELAPSES – OFTEN SEVERE
  • AZATHIOPRINE
  • MYCOPHENOLATE MOFETIL
  • RITUXIMAB
ANTI-MOG OPTIC NEURITIS

MYELIN OLIGODENDROCYTE GLYCOPEPTIDE (MOG)-PROTEIN
CONSTITUENT WITHIN MYELIN
ITS EXACT FUNCTION IS UNKNOWN
MAY PLAY A CRUCIAL ROLE IN THE INITIATION OF AUTOIMMUNITY & DEMYELINATION
### Side by Side Comparison

<table>
<thead>
<tr>
<th></th>
<th>MSON</th>
<th>NMOSD</th>
<th>MOG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Younger</td>
<td>Mean ~50 yrs</td>
<td>Wide range</td>
</tr>
<tr>
<td>Gender</td>
<td>80% F : 20% M</td>
<td>95% F : 2% M</td>
<td>50% F : 50% M but another study showed 3F : 1M</td>
</tr>
<tr>
<td>Nadir of vision</td>
<td>Variable</td>
<td>Poor</td>
<td>Poor</td>
</tr>
<tr>
<td>Visual recovery</td>
<td>Good recovery</td>
<td>Limited recovery</td>
<td>Good recovery but one study showed ~1/3 had visual impairment</td>
</tr>
</tbody>
</table>

(Akaishi et al., 2016)
(Jarius et al., 2016)
SUMMARY

• INFLAMMATORY OPTIC NEUROPATHIES - A GROWING LIST
  • OPTIC NEURITIS
    • FINAL UPDATE FROM ONTT
    • TRIALS OF NEW TREATMENTS [ORAL VS. IV STEROIDS; PHENYTOIN]
  • NEUROMYELITIS OPTICA
    • UPDATED DIAGNOSTIC CRITERIA
  • ANTI-MOG OPTIC NEURITIS
    • RECENT RECOGNITION AND ONGOING CHARACTERIZATION
THANK YOU