MRI assessment of acute optic neuritis (ON) at the first episode. Can we predict the visual outcome (VO) and the development of multiple sclerosis (MS)?

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Aims and objectives

Optic neuritis (ON) is an acute inflammatory demyelinating disorder, primarily an isolated phenomenon or secondarily associated with other neurological diseases as neuromyelitis optica or multiple sclerosis (MS) [1].

Its most common symptoms include unilateral, subacute visual loss with variable degree of severity, periocular pain on eye movements, dyschromatopsia, without systemic or other neurological symptoms.

The presentation is mostly monophasic but can also rarely be polyphasic with recurrent relapses.

ON is most frequently seen in females, F:M=3:1, diagnosed in young adults aging 20-45 years; mean age at onset is 36 years.

ON may be the initial presentation in ~20% of MS patients, and ON may occur during the course of the disease in 50% of patients with MS.

ON is normally a self-limiting event and recovery of visual acuity typically occurs within the first few weeks of symptom onset, however some patients have persistent visual problems [2,3].

Magnetic Resonance Imaging (MRI) may help ON diagnosis by detecting optic nerve inflammation.

Application of MRI to the assessment of optic nerve damage in a single episode of acute ON has been performed rarely [4] and its role in predicting the prognosis of visual impairment is not clear [3, 4].

Therefore, our aim was to assess MRI findings in the acute phase of ON and their correlation with visual acuity at presentation, visual outcome, MS development.
**Methods and materials**

We retrospectively revised the ophthalmological, neurological and imaging data of patients who presented to our Emergency Department (ED) with the first episode of acute ON from January 2015 to January 2017 (N=85).

We excluded some patients according to the following criteria:

- patients aged<18 years
- previous episodes of ON
- history of MS or NMO
- patients affected by known ophthalmological diseases
- patients affected by known immunological / infective disorders
- patients with previous neurological events
- patients with family history of Leber hereditary optic neuropathy
- lack of complete ophthalmological, neurological, imaging data
- time between symptoms onset and presentation > 14 days
- time between ED arrival and MRI execution > 14 days
- MRI executed in other Institutions
- lack of follow up data
- clinical follow up < 1 year

We therefore included in our study 37 patients (age range: 21-57 years; mean age: 33 years), 26 female and 11 male, 20 had right ON, 14 had ON in the left eye and 4 had bilateral ON, for a total of 41 affected eyes.

After the arrival, every patient underwent a complete ophthalmological examination, including fundoscopic examination, visual acuity assessment, visual field and Optic Coherence Tomography (OCT), brain and orbits unenhanced Computed Tomography, blood tests to exclude infective/autoimmune causes of ON and to test AQP4 antibodies.
Following the neuro-ophthalmological confirmation of the diagnosis of optic neuritis, steroid therapy was administered intravenously within 24 h from the patient access (methylprednisolone 1 g/d for five days, followed by a low-dose steroid oral regimen for 15 days).

MRI of brain, orbits, cervical spine was executed within 7 +/- 6 days from ON onset with the following acquisition protocol:

Imaging exams were transferred to the MRI workstation (Leonardo, Siemens, Forchheim, Germany) and revised by 2 experienced neuroradiologist in consensus.

Brain MRIs were classified as:
- normal
- nonspecific
- suspected demyelination (lesion with MS-like shape, localized in typical MS sites - periventricular, juxtacortical, infratentorial, or spinal cord with dissemination in space) [5]
- MS-like (lesions with dissemination in space and time: coexistence of asymptomatic gadolinium-enhancing and non-enhancing lesions) [6]

Optic nerves MRIs were reported as:
- normal
- STIR alteration without contrast enhancement (CE)
- STIR signal abnormalities + CE

Optic nerves pathologic findings were localized in 3 sites:
- intraorbital (IO)
- canalicular (CA)
- chiasmal (CH)

The extension of altered signal or CE was measured in cm.

OCT
OCT test was acquired using the same machine (Spectralis version 6.3.2, Eye Explorer Software 1.6.1.0, Heidelberg Engineering™, Heidelberg, Germany) to be reproducible, after mydriasis induced with 1% tropicamide.

OCT produces a cross sectional image of the peripapillary retina which is color-coded and shows the temporal, superior, nasal, inferior and temporal sections on the same image.

The software automatically recognizes the retinal nerve fiber layer (RNFL) layer, measures the RNFL thickness along the scan and reports the results on a graphic display showing the RNFL thickness (in µm). We use the average global value of RNFL

VISUAL ACUITY

Visual acuity was measured at patients' arrival and at 6 months follow up, according to Snellen chart (decimal). Light perception and motus manus (mm) were assigned a decimal visual acuity of 0.

Follow up

Each patient underwent a clinical follow up of at least 12 months.

Ophthalmological evaluation including visual acuity assessment was executed at 6 months to establish the visual outcome.

Visual outcome at follow up were classified in 3 groups:

1: complete visual recovery: visual acuity back to the value before NO

2: partial visual recovery: recovery of visual acuity of at least 50% compared to the value before ON

3: stable deficit: recovery of visual acuity < 50% compared to the value before ON, or persistence of the visual deficit, as during ON episode

Assignment of CIS or MS diagnosis was based on revised McDonald criteria [6].
### Images for this section:

<table>
<thead>
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<th>BRAIN</th>
<th>T1 SE sag</th>
<th>PD + T2 TSE tra</th>
<th>DARK FLUID sag</th>
<th>DIFFUSION tra</th>
<th>SPACE DIR sag</th>
<th>T1 VIBE 0.6*</th>
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*after administration of Gadobutrol 1mL/10 kg

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**Fig. 1**

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<table>
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<tr>
<th>ORBITS</th>
<th>T2 TSE cor FS</th>
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<th>SPACE STR tra</th>
<th>T1 SE FS tra</th>
<th>T1 SE FS cor*</th>
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</table>

*after administration of Gadobutrol 1mL/10 kg

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**Fig. 2**

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Fig. 3

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Results

Brain MRIs were:

- normal: 9 patients/37
- nonspecific: 9/37
- suspected demyelination: 13/37
- MS-like: 6/37

Optic nerves MRIs were reported as:

- normal: 14 eyes/41
- STIR alteration without contrast enhancement (CE): 13/41
- STIR signal abnormalities + CE: 14/41

Optic nerves pathologic findings were localized in 3 sites:

- intraorbital (IO): 19 eyes/41
- canalicular (CA): 16/41
- intraorbital + canalicular: 6/41
- chiasmal (CH): 0/41

The extension of altered signal or CE was measured in cm: extension range: 0.5-2 cm; extension mean: 1.5 +/- 0.2 cm

Visual outcomes were:

- complete recovery: 12 patients/37
- partial recovery: 16/37
- deficit persistence: 19/37

Diagnosis:

25 patients received a diagnosis of MS; 12 received a diagnosis of CIS; in our group no patients received a diagnosis of NMO.
RESULTS

BRAIN MRI - DIAGNOSIS

BRAIN MRI pattern was significantly correlated with final diagnosis

![Graph showing correlation between BRAIN MRI pattern and final diagnosis]

<table>
<thead>
<tr>
<th>BRAIN MRI</th>
<th>0: normal</th>
<th>1: nonspecific</th>
<th>2: suspected demyelination</th>
<th>3: MS-like</th>
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<tr>
<td>FINAL DIAGNOSIS</td>
<td>1: MS</td>
<td>2: CIS</td>
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</tbody>
</table>

Fig. 4

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LESIONS NUMBER - DIAGNOSIS

LESIONS NUMBER was statistically correlated with final diagnosis (p=.001; OR:1.342; 95%CI:1.102-1.631) at Mann Whitney test

![Graph showing correlation between lesions number and final diagnosis]

SM | FINAL DIAGNOSIS | CIS
Fig. 5

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LESIONS NUMBER- DIAGNOSIS

[Graph showing ROC curve]

Area Under the Curve (AUC): 0.8.
The presence of 4 lesions as a threshold has a sensitivity of 87.5% and a specificity of 66.6% for the diagnosis of MS

Fig. 6

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ORBITS MRI PATTERN- DIAGNOSIS

[Graph showing histogram]

No statistically significant correlation was observed between orbits MRI pattern and final diagnosis.

FINAL DIAGNOSIS
Pearson chi square: 0.6

ORBITS MRI
1: normal
2: STIR- alteration without contrast enhancement
3: STIR signal abnormalities +

FINAL DIAGNOSIS
1: MS
2: CIS

Fig. 7
Fig. 8

No statistically significant correlation was observed between brain MRI pattern and visual acuity.

Pearson chi square: 0.6

Fig. 9

No statistically significant correlation was observed between ORBITS MRI pattern and visual acuity.

Pearson chi square: 0.4
BRAIN MRI-VISUAL OUTCOME

No statistically significant correlation was observed between brain MRI pattern and visual outcome.

Pearson chi square: 0.2

Fig. 10

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ORBITS MRI-VISUAL OUTCOME

No statistically significant correlation was observed between orbits MRI pattern and visual outcome.

Pearson chi square: 0.9

Fig. 11

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OPTIC NERVE ANOMALIES EXTENSION

At Kruskal-Wallis test no statistically significant correlation was observed between OPTIC NERVE ANOMALIES EXTENSION and VISUAL ACUITY (p=.8), nor between OPTIC NERVE ANOMALIES EXTENSION and VISUAL OUTCOME (p=.2)

Fig. 12

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ORBITS MRI-RNFL

We observed a correlation between ORBITS MRI and RNFL at Kruskal-Wallis test (p=.037)

Fig. 13

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Conclusion

BRAIN MRI findings do not correlate with visual outcome, but with development of MS.

ORBITS MRI findings do not correlate with visual acuity, nor with visual outcome, but are correlated with RNFL at OCT.

MRI brain features and lesions number can predict the risk of MS conversion.
Personal information

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References