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How To Diagnose And Treat An Inflammatory Optic Neuropathy?

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Summary:
Inflammatory optic neuropathies (ION) or optic neuritis are one of the most common and feared neurological symptoms, as they can compromise the visual functional prognosis of our often young patients and those of childbearing age. This optic neuritis sometimes constitutes one of the early and even unique signs of an autoimmune inflammatory pathology.
All the interest is aroused to specify the clinical and evolutionary characteristics of this Optic Neuritis, and to perform radiological explorations (orbital, brain, and medullary imaging) and biological ones (study of cerebrospinal fluid and antibody assay), in order to move towards a possible inflammatory etiology, notably Multiple Sclerosis (MS), an Aqaporin 4 (AQP4) antibodies in optic Neuromyelitis (NMOSD with AQP4 antibodies), or a diseases with myelin oligodendrocyte glycoprotein antibodies (MOGAD).
The early demonstration of a positive diagnosis and the rapid precision of the corresponding etiology will allow instant, sometimes aggressive, management based on high-dose corticoid boluses and/or repeated sessions of plasma exchange (PE)., especially in the case of NMOSD to AQP4 antibodies or MOGAD. The proposal of an adapted background treatment remains inevitable, with the aim of improving the functional prognosis of this condition and preventing relapses and disability in the long term.
Keywords: *Optic neuritis/ Multiple sclerosis/ NMOSD with AQP4 antibodies/ MOGAD.*

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I. Introduction:

Inflammatory optic neuropathies (ION) or optical neuritis (ON) represent an important part of neurological symptoms, due to their frequency, their potential handicap on a young population and their activity.

With ON, the risk of revealing a more diffuse pathology of the central nervous system (CNS) remains very likely. Its etiologies are variable: in the presence of a first episode of NO, it may be difficult for the practitioner to predict whether it is an isolated event, or if he is facing an inflammatory pathology of the CNS, which will justify a rapid and adapted flare-up treatment and the early and aggressive one, for better prevention of disabling functional sequelae.

II. Make A Positive Diagnosis Of An ION:

The clinical arguments in favor:

- Acute to subacute installation
- Monocular or binocular decrease in visual acuity
- Pain with the mobilization of the eyeballs
- Color and contrast vision disorders
- Deficit of the visual field (scotoma or others)
- Relative Afferent Pupillary Deficit +++ (also called **the Marcus Gunn sign**): it is an enlargement of the pupil of the eye affected following the successive illumination of both eyes in the half-light. It is present in case of unilateral or bilateral asymmetric NO.
- Healthy papilla, œdematous or normal at the back of the eye
- Phenomenon of Uhthoff: it is an appearance and/or aggravation of visual symptoms following an increase in body temperature
- Pulfrich effect: It is an alteration of the perception of moving objects

The paraclinical arguments in favor:

- Elements for imaging the optic nerves: *in sequences T2, Flair, T1 without and with injection and in 3D- DIR*, which show an enlargement of the optic nerve, with a hypersignal taking or not taking contrast, and whose location and extent will be variable.
- Anomalies in ophthalmological examinations: notably Optical Coherence Tomography (OCT)
- Alterations of visual evoked potentials (VEP): useful especially in doubtful cases, and which show an extension of the latency of the P100 wave, indicating demyelinating damage to the optic nerve.

III. Identify The Etiology Of ION:

The case of 'Typical' ION = ON related to multiple sclerosis (MS)

- Age between 20 and 40 years
- Unilateral and subacute impairment (installed between 48 to 72 hours)
- BAV often moderate
- Almost constant peri-orbital pain (92% of cases)
- Dyschromatopsia, central scotoma
- The Marcus Gunn sign
- Normal eyeground in 2/3 of the cases
- Favorable evolution often regressive after a few days or even weeks.
- The imaging of the optic nerve: shows an unextended hypersignal interesting the anterior segment of the nerve, with contrast taking (Figure 1).

The diagnosis of definite MS is immediately established if the criteria for temporal and spatial dissemination are met (by bone marrow imaging and lumbar puncture in search of oligoclonal bands), after exclusion of other etiologies (inflammatory, immunological and negative serologies assessments).
If the criteria are not met, we will then talk about an isolated clinical syndrome with a progressive risk of MS.

The McDonald criteria of MS have evolved over time, the last update dates from the year 2024 (figure 2), and has added the topography of the optic nerve as a preferred location for this disease. Additional criteria were reported, radiological (highlighting at least 6 lesions with a sign of the central vein (SVC) and/or a lesion or more paramagnetic (PRL)) and biological (Kappa chain index greater than 6.1 in the CSF with or intrathecal synthesis of immunoglobulins G).

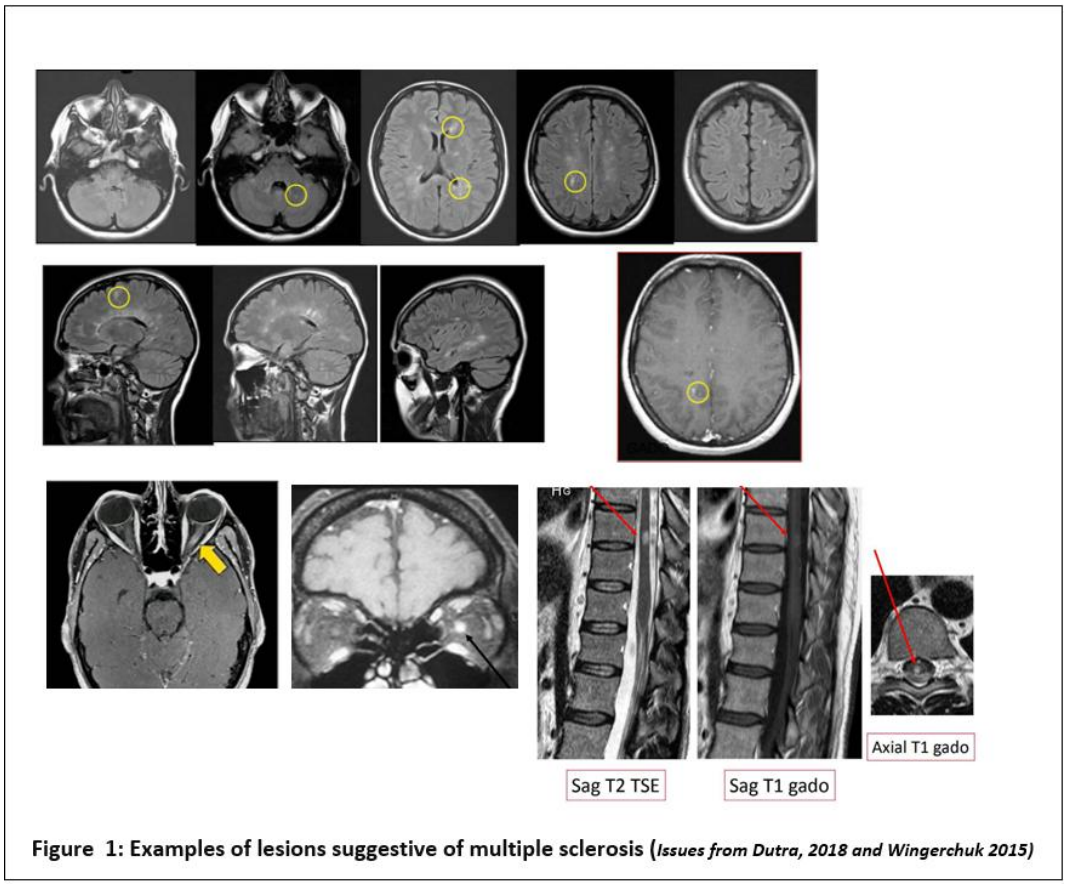

Figure 1: Examples of lesions suggestive of multiple sclerosis (*Issues from Dutra, 2018 and Wingerchuk 2015*)

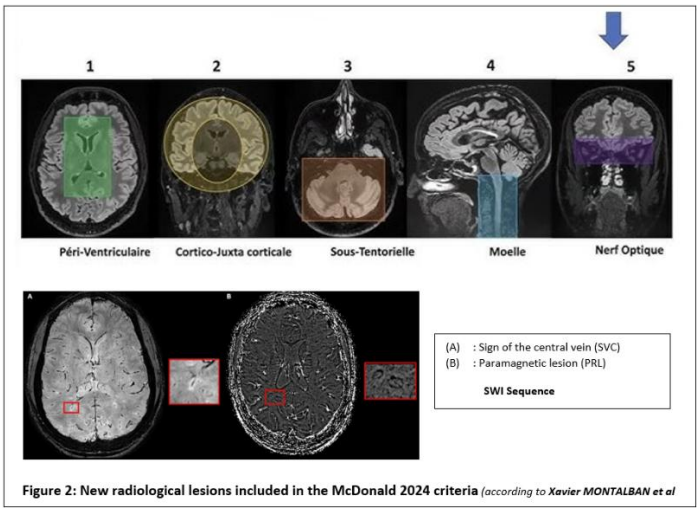

Figure 2: New radiological lesions included in the McDonald 2024 criteria *(according to **Xavier MONTALBAN et al***

The case of 'Atypical' ION = related to NMO spectrum disorders (NMOSD) or MOG antibodies disorders (MOGAD)

In the NMOSD with AQP4 antibodies:

- Age of occurrence over 40 years
- Installation over more than 2 weeks
- severe, deep and often bilateral decreased visual acuity
- Painless +++
- Little or no recovery beyond 3 weeks
- The papillitis can be seen in the back of one's eye
- Imaging of the optic nerves: shows extensive involvement exceeding 50% of the nerve length, and preferentially affecting the posterior segments up to the chiasm and the optic tract (Figure 3).
- Extensive clinical and/or radiological bone marrow involvement may be associated with it (myelitis exceeding 3 metameres with a *Bright spotty lesions appearance)*
- Frequent pleocytosis with rare BOCs
- The seropositivity of antibodies against Aquaporin 4 (against ONM) confirms the diagnosis.

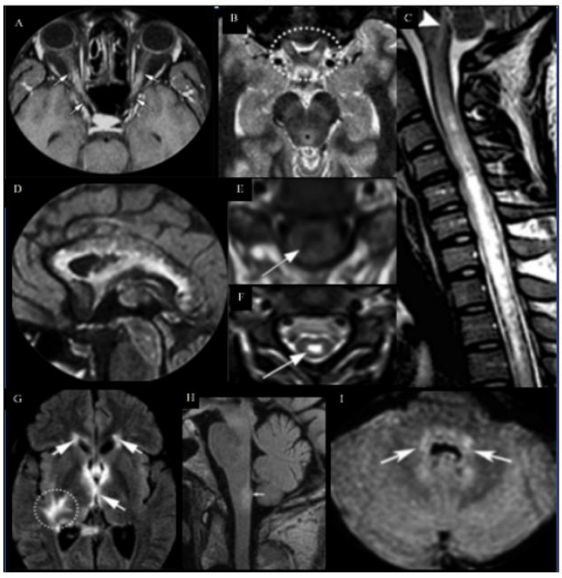

Figure 3: Examples of lesions suggestive of NMOSD with Antibodies against Aquaporin 4

***A.** Posterior T1 hypersignals of the 2 optic nerves (arrows) after gadolinium injection. **B.**chiasmatic involvement in T2 hypersignal (dotted lines). **C.** MATLE cervico-thoracic with bulbar extension (T2). **D.** Hypersignal T2 of the ependymal face of the corpus callosum. **E & F** : Central spinal cord injury in hyposignal T1 (E) and hypersignal T2, called 'bright spotty lesion' (F). **G.** Periventricular FLAIR hypersignals (arrows) and right cortico-spinal bundle (in dotted lines). **H.** Affected the Area Postrema (posterior part of the spinal bulb). **I.** Hypersignal FLAIR around the 4th ventricle.*

Issues from Dutra, 2018 and Wingerchuk 2015

In the MOGAD:

- Occurring in adults, sometimes the child
- Severe decreased visual acuity, frequently bilateral
- Very painful +++, sometimes with prodromal headaches, or even signs suggestive of intracranial hypertension syndrome (ICH)
- Papilledema sometimes very voluminous at the eyeground
- The objective orbital imaging is an extensive, rather anterior approach, with respect for the optic chiasm. The presence of a peri-**neural enhancement is very characteristic of this disease** (Figure 4).
- Brain damage in the form of acute disseminated encephalomyelitis (ADEM), that of the basal ganglia or the brainstem, are highly suggestive of the diagnosis. As well as an involvement combining extensive and non-extensive myelitis of low lumbar location.
- Frequent pleocytosis with rare BOCs in the immunological analysis of the LCS
- Seropositivity to antibodies against Myelin Oligodendrocyte Glycoprotein (MOG) confirms the diagnosis.

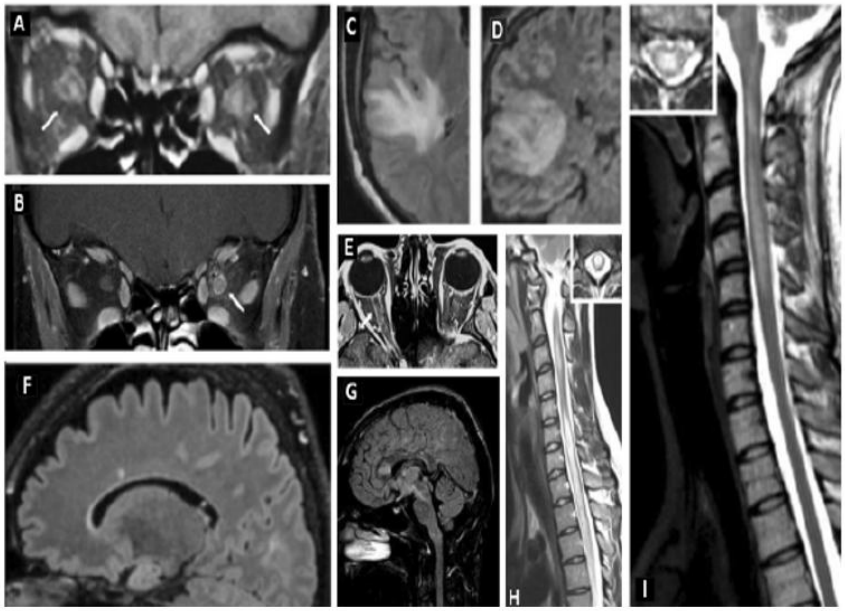

Figure 4: Examples of lesions suggestive of Mogopathies *(From Jarius, 2016)*

A & B. Coronal T1 sections showing an increase in the intraorbital portion of the optic nerves associated with an increase in the sheath of the optic nerve and the perineural area in a subject with NO. *C & D. Axial (C) and coronal (D) T2 section showing a* ***wide and*** *confluent lesion in the temporal lobe. E. Axial T1 section showing a gadolinium lift along the intraorbital portion of the right optic nerve.* ***F.*** *Sagittal FLAIR Cup showing corpus callosum lesion G.* ***Sagittal FLAIR Cup showing corpus callosum lesions and diencephalic lesion extending to the protubérance.*** *H. Sagittal medullary section T2 revealing a pan-medullary MATLE associated with edema of the spinal cord. Note the coexistence of a cervical MATC and a dorsal cervico MATLE.* ***I.*** *Sagittal medullary section T2 showing an extended hypersignal from the prominence to C5.*

ION of system diseases:

- Can be typical or atypical
- Associated with other neurological and extra-neurological signs +++ (dermatological, articular, cardiac, renal signs ...)
- Specific assessments allow for orientation towards etiology (biological and other)
- Among these diseases: sarcoidosis, systemic lupus erythematosus, Behçet's disease, Gougerot Sjogren syndrome...

IV. Eliminate Differential Diagnostics:

Several pathologies can be responsible for optic neuropathy and whose origin will vary. These diseases differ according to the mode of installation, the patients' field and by other clinical and paraclinical elements suggestive (Table 1).

Table 1: Different causes of optic neuropathies

Evocative signs	Diagnosis
Acute installation, brutal BAV, unilateral, **painless**; CV: déficit altitudinal; FO: papillary pallor, papilledema and/or hemorrhagic lesions; ERG: pathological	Anterior ischemic optic neuropathy
Same signs **+ Pain+++** and age over 50	**Horton's disease**
Subacute installation, severe papilloedema or macular star, febrile context	**Infectious:** Bartonellose, Lyme, tuberculosis, herpes, HIV, malaria, toxoplasmosis, cryptococcosis...
Subacute set-up, bilateral BAV with tetraparesis (NMOSD-mimick), corticoid aggravation and **hyperlactatorachial pain at the lumbar puncture**	**Biotinidase-deficient neuropathy**
Chronic installation	**Tumor**: meningioma or optic nerve glioma, pseudoinflammatory tumor by deposition of IgG 4 **Deficiency**: B12, B9, B1 deficiencies, Copper deficiency **Toxic**: Alcohol and tobacco poisoning, Methanol, Ethambutol, Amiodarone, PDE5 inhibitors, ITK **Genetics**: ON of Leber, dominant optical atrophies

V. Treat The ION:

An Emergency treatment:

- Bolus of Methyl prednisolone 1 gram per day for 3 to 5 days intravenously, in case of typical ION.
- Mega bolus of corticosteroids ranging from 1 to 2 grams per day for 5 days, in case of atypical ION.
- Plasma exchanges (PE) either in the first intention, or in the absence of a response at the end of the 3rd corticoid bolus period. The most severe cases concern the ON of NMOSD and MOGAD. 5 to 7 sessions are recommended (1 day/2). The early onset of PE is directly correlated with good functional recovery.

A basic treatment:

- Away from the flare-up: discuss a background treatment for MS, the choice of which will depend essentially on the age, form and activity of the disease and the desire for pregnancy in women.
- For NMOSD: a treatment, against a B-lymphocytes, an interleukin 6 or against a complement, is recommended to prevent relapses that can be harmful.
- For MOGAD: no consensus on the choice of treatment. A relay orally with corticoids followed by immunosuppressants remains frequent.

VI. Conclusion:

In front of a decrease in visual acuity from acute to subacute installation:

- Affirm an ION, by researching the evocative clinical and paraclinical elements
- Think about a MS in case of typical ION, with a better recovery prognosis
- Think rather of a NMOSD or a MOGAD in case of atypical ION, often with an unsatisfactory remission and more serious sequelae.
- Interest in rather early and aggressive emergency treatment, ranging from corticoid bolus to PE, depending on the etiology.

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