



Imaging of the Visual Pathway in Optic Neuritis

Optic Neuritis (NMO, Devic syndrome) is a rare autoimmune inflammatory disease of the Central Nervous System (CNS) that is clinically characterized by primarily severe damage to the optic nerve and spinal cord. For a long time, NMO was considered a variant of Multiple Sclerosis (MS). However, recent mounting evidence points to a distinct pathogenesis. An important step was the discovery of a highly specific biomarker for NMO, the so-called NMO-IgG, whose target antigen turned out to be Aqua Porin-4 (AQP4) of the most abundant water channel in the body central nervous system. Since then, various tests for the detection of anti-AQP4 antibodies have been developed, which facilitate the differentiation of NMO from clinically relevant MS. Anti-AQP4 antibodies are detectable in 60-90% of NMO patients with a specificity of 91-100%. Contrary to previous belief, NMO is now considered a relapsing disease in 80-90% of patients. With the discovery of AQP4 antibodies, the clinical spectrum of NMO has expanded and now AQP4-Positive Persistent Transverse Myelitis (LETM) and AQP4-positive Recurrent Optic Neuritis are considered is part of the NMO Spectrum Disorders (NMOSD).

KEYWORDS: Diagnostic Imaging • Medical Imaging • Visual Pathway • Optic Neuritis

Introduction

Multiple sclerosis studies have shown that neuritis and neuro degeneration can cause damage to different parts of the optic tract, including inflammation in the retina, neuro degeneration of the retina. Retinal nerve axonal degeneration as well as pathological changes in the optic tract, lateral nuclei, radiating optic nerve, and visual cortex. The purpose of this article is to provide an overview of recent research findings on similar or different involvement of the visual pathway in NMO pathology as assessed by Magnetic Resonance Imaging (MRI) and Optical Coherence Tomography (OCT) [1].

From a clinical point of view, abnormal brain MRI was until recently considered an argument against the diagnosis of NMO. However, several publications in recent years have shown that in NMO, various brain lesions may be present at the onset or during the course of the disease. A high proportion of NMO patients develop nonspecific brain lesions after diagnosis (60% in Pittock series, 85% in Li China series), and 10% in the series of 60 brain injury patients met diagnostic criteria for MS. An additional 10% had brain damage in peri cortical regions (eg. hypothalamus, peritubal brain stem) that were rich in AQP4. Unlike MS, widespread and diffuse white matter abnormalities have been described by various groups. Recently, large edematous scar tissue lesions were also reported in 4 out of 22 NMO patients [2].

Discussion

Other magnetic resonance imaging techniques that are not routinely performed in clinical practice are Magnetization Transfer Imaging (MTI), Diffusion Tensor Imaging (DTI), and Magnetic Resonance Spectroscopy (MRS). MTI is a technique that analyzes the energy transfer from matrix bound water to the hydrogen nuclei of free water and thus allows the integrity of the anatomical structure to be assessed. DTI measures the direction-dependent diffusion limit of water, and the resulting data can be calculated to describe fibrous structures, such as optical radiation. SRM differs from other imaging techniques because it does not provide structural information but rather provides information about ongoing metabolic processes by measuring concentrations of metabolites that are representative of the metabolism. Energy, integrity of cell membranes, integrity of axons or neurons, and others. These methods show normal-appearing gray matter lesions, but no lesions or only minimal abnormalities in seemingly normal white matter. In contrast, a recent DTI study by Yu and colleagues reported increased cortical band diffusion capacity and visual radiation but not the corpus callosum and rim in NMO patients compared with controls, and the authors concluded that aberrant diffusion was limited to connectivity regions to the optic nerves and spinal cord, thus arguing for axonal degeneration secondary to optic nerve and spinal cord injury [3, 4].

Pfueller Caspar*

Neuro Cure Clinical Research Center,
Charité Universitäts, Medizin Berlin,
Berlin, Germany

*Author for correspondence
pfueller.caspar@charite23.de

Received 1-May-2023, Manuscript
No. fmim-23-92005; Editor assigned:
3-May-2023, Pre QC No. fmim-23-
92005(PQ); Reviewed 17-May-2023,
QC No. fmim-23-92005; Revised:
24-May-2023, Manuscript No.
fmim-23-92005(R); Published:
30-May-2023; DOI: 10.37532/1755-
5191.2023.15(3).44-46

■ Visual Pathway: Optic Nerve and Optical Radiation

Magnetic resonance imaging studies aimed at visualizing optic nerve damage in NMO are rare. Lee and associates reported thickening of the optic nerve sheath in 16 of 33 patients, all of whom had recurrent ON symptoms. Wang and colleagues found optic nerve intensity in 6 out of 10 patients with optic nerve MRI available. Optic nerve enhancement with intravenous gadolinium was detected in 4 patients undergoing MRI within 6 weeks of the onset of acute ON. Similar observations were made in a comparative study. However, as similar findings have been described in optic neuritis in MS, it is doubtful whether the aforementioned findings are NMO-specific or instead just optic neuritis, regardless of the underlying condition. Lin et al. reported the ability to distinguish NMO from MS based on DTI data. However, no pathway-specific analysis was performed here. The only MRI study to date that has evaluated the regenerative portion of the visual pathway in NMO by DTI has reported an increase in the diffusivity of optical radiation. To our knowledge, there are no larger studies on MT imaging of the visual pathway in NMO patients [5, 6].

OCT optical coherence tomography is a non-invasive and reproducible technique to study unmyelinated retinal axons with high spatial resolution *in vivo* and to quantify thickness of the Peri Aapical Nerve Fiber Layer (RNFL), fovea, and macula. In MS patients, OCT has been consistently shown to detect RNFL thinning most likely due to diffuse retinal axonal damage occurring at least in part independently of prior attack or ON current. In this context, OCT could prove to be a valuable tool for detecting and monitoring axonal damage in MS and other inflammatory CNS conditions such as NMO. Several groups have studied damage of frontal visual pathways in NMO by OCT compared with healthy controls or patients with MS. The first published study of OCT in NMO by de Seze and colleagues reported a significant reduction in mean RNFL thickness in NMO patients compared with healthy controls (77.9 μm vs. 102.3 μm) and good correlation between OCT results and visual acuity and visual acuity discussed potential lags. Interestingly, among patients at high risk for NMO (recurrent LETM ON), only those with recurrent ON had such a severely reduced mean RNFL thickness (74.2 μm) compared with those with recurrent ON. subjects with recurrent myelitis that did not differ from controls in mean RNFL thickness (101.8 μm vs 102.3 μm). Another recent OCT

study by Naismith and colleagues compared RNFL measurements in NMO patients with MS. Consistent with clinical experience of more severe loss of visual function in NMO than in MS after ON, RNFL thickness was significantly thinner in NMO patients than in MS after ON, giving found deeper axonal loss in the optic nerve in NMO. It has been observed that who estimated that the first episode of ON caused an additional 24 μm reduction in RNFL thickness in NMO compared with in recurrent MS. In addition, macular volume was also significantly reduced in NMO ON eyes compared with both MS and healthy controls. Interestingly, the eyes of the LETM subgroup and unaffected NMO eyes were no different from those of the control group. The difference in RNFL thickness between ON and non-ON eyes in NMO patients was much larger than in MS patients (34.3 μm vs 9.6 μm). This may suggest that retinal axon damage in NMO is primarily associated with ON attacks, whereas in MS RNFL thinning has been reported albeit to a lesser extent also in eyes are not ON. Other hypothetical explanations could be the more frequent occurrence of subclinical optic neuritis in MS compared with NMO, or that MS lesions in the interferometer or optic pathway lead to bipolar RNFL involvement side [7, 8].

Conclusion

There is serious discussion about the importance of OCT for axonal injury monitoring. On the one hand, the validity of past OCT measurements is limited by the instrument-specific measurement variations of time-domain tomographers within the suspected effect range, for example, for changes in the RNFLT over time. It is expected that the new spectral domain OCT devices will provide improved spatial resolution and better retest reliability in the future and thus help to provide a more accurate description of damage to with the visual pathway and its pathophysiological correlation while lying down. On the other hand, there is increasing evidence that OCT measurements not only reflect axonal damage but can also be affected by endophthalmitis or retinal neuron degeneration, as demonstrated described in a recent neuropathology study of postmortem postmortem analysis of MS brain tissue [9, 10].

Acknowledgement

None

Conflict of Interest

None

References

1. de Carvalho RM, Mazzer N, Barbieri CH. Analysis of the reliability and reproducibility of goniometry compared to hand photogrammetry. *Acta Ortop Bras.* 20, 139-49 (2012).
2. Naylor JM, Ko V, Adie S *et al.* Validity and reliability of using photography for measuring knee range of motion: a methodological study. *BMC Musculoskelet Disord.* 12, 77 (2011).
3. Li MK, Howard DP, King R. A picture tells a thousand words smartphone-based secure clinical image transfer improves compliance in open fracture management. *Injury.* 50, 1284-7 (2019).
4. Kazemi T, Lee KC, Bercovitch L. Just a quick pic: Ethics of medical photography. *J Am Acad Dermatol.* 80, 1172-4 (2019).
5. Archibald DJ, Carlson ML, Friedman O. Pitfalls of nonstandardized photography. *Facial Plast Surg Clin North Am.* 18, 253-66 (2010).
6. de Meijer PP, Karlsson J, LaPrade RF *et al.* A guideline to medical photography: a perspective on digital photography in an orthopaedic setting. *Knee Surg Sports Traumatol Arthrosc.* 20, 2606-11 (2012).
7. Uzun M, Bulbul M, Toker S *et al.* Medical photography: principles for orthopedics. *J Orthop Surg Res.* 9, 23 (2014).
8. Kim SH, Sobez LM, Spiro JE *et al.* Structured reporting has the potential to reduce reporting times of dual-energy x-ray absorptiometry exams. *BMC Musculoskelet Disord.* 21, 248 (2020).
9. Körber A, Rietkotter J, Grabbe S *et al.* Three-dimensional documentation of wound healing: first results of a new objective method for measurement. *J Dtsch Dermatol Ges.* 4, 848-54 (2006).
10. Wang S, Zhang Q, Huang W *et al.* A new smart mobile system for chronic wound care management. *IEEE Access.* 6, 52355-65 (2018).