## Advances in neuromyelitis optica

Avanços em neuromielite óptica

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Neuromyelitis optica (NMO) is considered a demyelinating disorder, predominantly affecting the optic nerve and the spinal cord. However the disease concept has undergone changes over the past decade. In the neurological field, NMO is no longer considered as being a multiple sclerosis (MS) subtype, being currently diagnosed as a disease itself, with a differentiated clinical course, besides different prognosis and treatment as compared with MS. In what regards the immunological aspect, the discovery of the central nervous system water channels (aquaporin) involvement by an autoantibody called anti-AQP4 was a key factor leading to the differentiation of the disease. The attack by autoimmune complexes to areas containing water channels, particularly those adjacent to ventricular regions, in the optic nerves and around the central channel of the spinal cord, causes necrosis in such regions, leading to more severe lesions which are more difficult to heal than MS lesions.

All such discoveries were essential for the evolvement of radiological evaluation in NMO. The involvement of the brain in NMO has been increasingly investigated. Previously, the criteria for diagnosis of NMO established that cranial magnetic resonance imaging (MRI) should be normal. Currently, such criteria include the presence of lesions at cranial MRI which do not meet the imaging criteria for MS. At MRI, intracranial lesions are poorly specific, but are frequently found, affecting up to 90% of the cases. However, about 8–10% of the patients present more severe brain lesions, with typical images from affected regions which are rich in APQ4 channels, such as those around the third ventricle, the periaqueductal gray matter, hypothalamus and periventricular region<sup>(1)</sup>.

However, the investigation of brain involvement in NMO by means of more advanced MRI techniques is still poorly developed. The concept of apparently normal white matter (ANWM) that is well established in MS, corresponding to the presence of normal signal intensity on white matter areas at conventional MRI sequences, however with significant inflammatory infiltration as histopathologically analyzed, is also currently being defined in NMO. The study with diffusion tensor imaging - a diffusion-based technique that allows analyzing the white matter microstructure integrity - has demonstrated to be useful in this process. A recent study published by the journal **Radiology** in April/2012 approached this subject<sup>(2)</sup>. By means of measurements of fractional anisotrophy (FA), radial diffusion (RD) and axial diffusion (AD), one can infer that diffuse microstructural changes occur in the apparently normal brain white matter tracts, going beyond the corticospinal tracts and optic radiations. Additionally, such study suggests that such changes are predominantly related to demyelination (RD-related FA change). In NMO, there has always been a tendency to consider that such loss of matter integrity was related to Wallerian degeneration secondary to distal lesions affecting the spinal cord and optic nerves. However, such study has demonstrated that the extent of the microscopic damage, otherwise "invisible" at conventional MRI techniques, is far greater and that, therefore, large areas of the radiate crown and particularly of the corpus callosum are affected.

In patients with NMO, the utilization of conventional and advanced MRI techniques in association with novel histopathological techniques has been useful to clarify the disease physiopathology, besides allowing a more appropriate classification of the disease within the spectrum of demyelinating disorders.

## REFERENCES

- Pires CE, Silva CM, Lopes FC, et al. Brain MRI abnormalities in Brazilian patients with neuromyelitis optica. J Clin Neurosci. 2012; 19:969–74.
- 2. Rueda Lopes FC, Doring T, Martins C, et al. The role of demyelination in neuromyelitis optica damage: diffusion-tensor MR imaging study. Radiology. 2012;263:235–42.

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