Editorial

Diagnostic and therapeutic neuroradiology of neurodegenerative diseases

As we have seen over the last few years, thanks to parallel advances in both imaging and therapeutic possibilities in the area of stroke. It is therefore the moment to ask ourselves if and how this success model can be replicated in an area where despite many advances, treatment is still insufficient. The diseases that come to mind are those of the neurodegenerative kind, be they neuro-immunological or neurogenetic in origin. When we consider the stroke model, over the last two decades, we had seen progress in imaging with advances in both CT, MRI and angiography [1,2], that finally led to developing an approach to the disease where imaging was deeply imbedded in the management not just for diagnostic purposes but also for therapy [3,4]. This may not be applicable to all situations but to some degree, we observe that the use of imaging as a marker for any of these diseases remain very important. In stroke, we have improved the visualization with CT and MRI of the acute ischemic lesion, while at the same time surprisingly neglecting the clot [5]. However, our understanding of the hemodynamics that underlie ischemia have improved thanks to the translational use of the new imaging techniques. For a disease such as MS thanks to imaging, new concepts of diagnosis based on imaging have emerged with even the emergence of a concept of the radiologically isolated syndrome [6], i.e., cases in which the disease is purely made evident on the basis of imaging findings, even in sometimes the absence of clinical deficits. The domain of the dementias remains more problematic, because, while we dispose of clearly diagnostic markers for the established disease, early markers are necessary in order to obtain early treatment and possibly optimal outcomes. Indeed, using MR techniques we can assess macroscopic changes: atrophy but we can see microscopic changes with diffusion-weighted and diffusion tensor imaging [7–9], perfusion techniques such as arterial spin labeling [10], susceptibility-weighted imaging [11], in addition to further markers such as brain perfusion and spectroscopy. Until now, these markers that seem to show an active disease have failed to have their final impact since very often they have not been used at disease onset and also require advanced post-processing [12]. While this can probably only be done in a first phase in a patient population that is at risk and not nation-wide, some kind of screening programs must clearly be implemented in order to detect the disease earlier. While this also creates a need for a more easy to use diagnostic paradigm as is often the case in stroke, where very often the need to "go ahead" can be done by just looking at the unenhanced CT images where one sees early changes and the hyperdense clot, need that may be in inherent contradiction with the use of more complex techniques mentioned above. However, in the difference with stroke, as far as we know there is no time window for initiating treatment yet. As in stroke, it is probably by adapting to a more target-adjusted therapy that we can succeed, i.e. treating what we at the moment see on the images. Thus we need as in stroke, early imaging derived markers together with clearly focused treatment targets in order to finally vanquish these diseases.

Disclosure of interest

The author declares that he has no competing interest.

References

[1] Lövblad KO, Altrichter S, Viallon M, Sztajzel R, Delavelle J, Maria I, et al. Neuro-imaging of cerebral ischemic stroke. J Neuroradiol 2008;35:197–209.

[2] Mendes Pereira V, Yilmaz H, Pellaton A, Slater LA, Krings T, Lovblad KO. Current status of mechanical thrombectomy for acute stroke treatment. J Neuroradiol 2015;42(1):12–20.

[3] Jovin TG, Chamorro A, Cobo E, de Miquel MA, Molina CA, Rovira A, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke. N Engl J Med 2015;372(24):2296–306.

[4] Nogueira RG, Jadhav AP, Haussen DC, Bonafe A, Budzik RF, Bhuva P, et al. Thrombectomy 6 to 24 hours after stroke with a mismatch between deficit and infarct. N Engl J Med 2018;378(1):11–21.

[5] Bouchez L, Lovblad KO, Kulcsar Z. Pretherapeutic characterization of the clot in acute stroke. J Neuroradiol 2016;43(3):163–6.

[6] Lebrun C. The radiologically isolated syndrome. Rev Neurol 2015;171(10): 698–706.

[7] Haller S, Missonnier P, Herrmann FR, Richiardi J, Rodriguez C, Deiber-Ibanez MP, et al. Individual classification of MCI subtypes by support vector machine analysis of white matter derived from diffusion tensor imaging (DTI). AJNR Am J Neuroradiol 2013;34(2):283–91.

[8] Haller S, Badoud S, Nguyen D, Garibotto V, Lovblad KO, Burkhard PR. Individual detection of Parkinson disease patients using support vector machine analysis of diffusion tensor imaging data: initial results. AJNR Am J Neuroradiol 2012;33(11):2123–8.

[9] Goujon A, Mejdoubi M, Purcell Y, Banydeen R, Colombani S, Arrigo A. Can MRI water apparent diffusion coefficient (ADC) value discriminate between idiopathic normal pressure hydrocephalus, Alzheimer's disease and subcortical vascular dementia? J Neuroradiol 2018;45(1):15–22.

[10] Xekardaki A, Rodriguez C, Montandon ML, Herrmann F, Zekry D, Lovblad KO, et al. ASL predicts cognitive deterioration in healthy elderly individuals. Radiology 2015;274(2):490–9.

[11] Haller S, Bartsch A, Nguyen D, Rodriguez C, Emch J, Gold G, et al. Susceptibility weighted imaging (SWI) assessment of cerebral microbleeds and iron deposition in mild cognitive impairment. Radiology 2010;257(3):764–73.

[12] Haller S, Lovblad KO, Giannakopoulos P. Principles of classification analyses in mild cognitive impairment (MCI) and Alzheimer disease. J Alzheimers Dis 2011;26(0):389–94.

Karl Olof Lovblad
Division of Neuroradiology, Geneva University Hospitals, 4, rue Gabrielle-Perret-Gentil, 1211 Geneva 14, Switzerland
E-mail address: olof.lovblad@hcuge.ch

Available online 5 September 2018