LETTER TO THE EDITOR

MRI to predict severe tissue damage in inflammatory lesions in animal models of multiple sclerosis

Bruno Brochet,1,2 Vincent Dousset,3 Mathilde Deloire,1 Claudine Boiziau1 and Klaus G. Petry1

1EA 2966, Neurobiology of myelin diseases, Bordeaux Neuroscience Institute, University Victor Segalen (Bordeaux 2), 2Department of Neurology, Hôpital Pellegrin, Centre Hospitalier Universitaire and 3Department of Neuroradiology, Hôpital Pellegrin, Centre Hospitalier Universitaire, 33076 Bordeaux cedex, France

Correspondence to: Prof. Bruno Brochet
E-mail: bruno.brochet@chu-bordeaux.fr
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Sir, To correlate magnetic resonance imaging (MRI) patterns with tissue destruction is interesting, but to correlate MR patterns with ongoing inflammation to predict the fate of a lesion is of great importance in clinical practice of Multiple sclerosis (MS). The paper of Nessler et al. (2007) reporting early and predictive MRI changes in a mouse model of MS is therefore of great interest. The paper is part of ongoing efforts by many laboratories devoted to identify MRI patterns that allow predicting evolution of MS lesions. The main presented finding is that reduced signal intensity in both T1- and T2-weighted images is associated with more severe tissue destruction and a higher density of inflammatory cells. The presented work completes previously published data demonstrating that grading severity of inflammatory lesions in a relapsing rat model of MS by MRI monitoring of macrophage infiltration with ultra small super paramagnetic iron oxide nanoparticles (USPIO) may help to predict the forthcoming severity of tissue destruction in the lesions (Brochet et al., 2006). This method was recently applied in MS revealing that about 30% of the lesions enhanced with Gadolinium(Gd)-DTPA, a marker of increased permeability of the blood-brain-barrier are negative for USPIO (Dousset et al., 2006), confirming results obtained in EAE rat models (Dousset et al., 1999). Interestingly, Nestler et al. demonstrated that MRI with Gd-DTPA correlated with Ig deposits, and activation of astrocytes and microglia. However, Gd-DTPA enhanced lesions were not associated with the more severe pattern of tissue damage, i.e. loss of axons and myelin, or inflammatory cell infiltrates. These observations confirm that Gd-DTPA is not useful to evaluate in vivo tissue destruction and to predict the fate of an inflammatory lesion. The various experimentally established MRI parameters offer new perspectives in evaluating preclinical and clinical therapeutic strategies. We consider that this information might interest the readers of Brain as a complement to that provided in the article by Nessler et al.

References