Correspondence

Cerebellar and thalamic degeneration in spinocerebellar ataxia type 10

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We read with great interest the manuscript published by Hernandez-Castillo et al. entitled “extensive cerebellar and thalamic degeneration in spinocerebellar ataxia type 10” [1]. The authors studied 18 Mexican patients with genetically confirmed spinocerebellar ataxia type 10 (SCA10) and 18 healthy controls matched for age and gender [1]. Whole-brain Tract-Based Spatial statistics (TBSS) and Voxel-Based Morphometry (VBM) were used to evaluate white and grey matter degeneration. The TBSS analysis demonstrated white matter atrophy in the cerebellum of the SCA10 patients. VBM analysis showed extensive grey matter degeneration in the cerebellum, brainstem, thalamus, and putamen [1]. Additionally, degeneration of the thalamus was significantly associated with the presence of seizures [1]. Recently, our group just finished a very similar study entitled “Volumetric MRI changes in spinocerebellar ataxias (SCA3 and SCA10) patients” [2]. All Brazilian patients and individuals (18 of them with SCA 10, 19 with SCA3 and 18 from the control group) were studied using the software FreeSurfer 5.3 which allows surface-based morphometry, and a cross-sectional evaluation [2]. The volumetric changes were much more prevalent in the SCA3 than SCA10 patients. On the other hand, in SCA10 patients, cerebellum and pallidum showed significant volume reduction, but thalamus was totally normal. It should be considered that all patients with SCA10 in this study had no epilepsy [2]. These findings reinforce the results of Hernandez-Castillo et al. study, emphasizing the role of the thalamus in the origin of seizures in patients with SCA10 [1,2]. Nuysts et al. studied the structural brain abnormalities in genetic generalized epilepsies (GGE), using a quantitative magnetic resonance imaging techniques, through a systematic review and meta-analysis. The authors observed significant structural differences between GGE and healthy controls, with volume reduction in whole brain, thalamus, and putamen, among others structures [3].

Author’s role

W.O.A analyzed the data and drafted the manuscript. A.T.M., C.H. F. C. and M.G.F. helped with analysis and revised the manuscript. A.E.O. and A.C. helped with the data analysis and the MRI studies. S.R. and T.A. helped with data analysis and genetic studies. H.A.G.T. helped with analysis and revised the manuscript.

Declaration of competing interest

All authors report no conflict of interest.

References


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