Reversible Diffusion-Weighted Imaging High Intensity Signal in Wilson Disease

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Contributions:
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Jiting Zhu: Major role in the acquisition of data; Study concept or design

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A 41-year-old woman presented bradykinesia, tremor and dysarthria 2 years ago and progressively developed to cognitive decline after being treated for Parkinson’s disease.
The dystonia progressed and eventually she developed akinesia, was unable to speak or walk. After referred to our hospital, she was diagnosed with Wilson disease (WD) based on the presence of Kayser-Fleischer rings, hypoceruloplasminemia and ATP7B pathogenic variants (c.2804C>T/p.T935M, c.2975C>T/p.P992L). Neuroimaging revealed abnormalities in the bilateral basal ganglia, thalamus and brain stem on T2 sequences and hyper intensity along the corticomedullary junction on diffusion-weighted imaging (DWI; Figure 1A). DWI high intensity in the corticomedullary junction is the typical feature of neuronal intranuclear inclusion disease (NIID). The subsequent genetic test ruled out NOTCH2NLC mutation and intranuclear inclusions were not detected in skin biopsy sample. After treatment with chelating agents and zinc, she made a partial recovery and the DWI high intensity dramatically disappeared (Figure 1B).
Figure 1: Reversible DWI high intensity signal in Wilson Disease.

Figure legend:

(A) The initial brain magnetic resonance image (MRI) shows typical lesions on T2 sequences and hyper intensity along the corticomedullary junction on DWI. (B) DWI high intensity along the corticomedullary junction disappeared on the following MRI after treatment.
References

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