

White matter alterations in narcolepsy children with cataplexy

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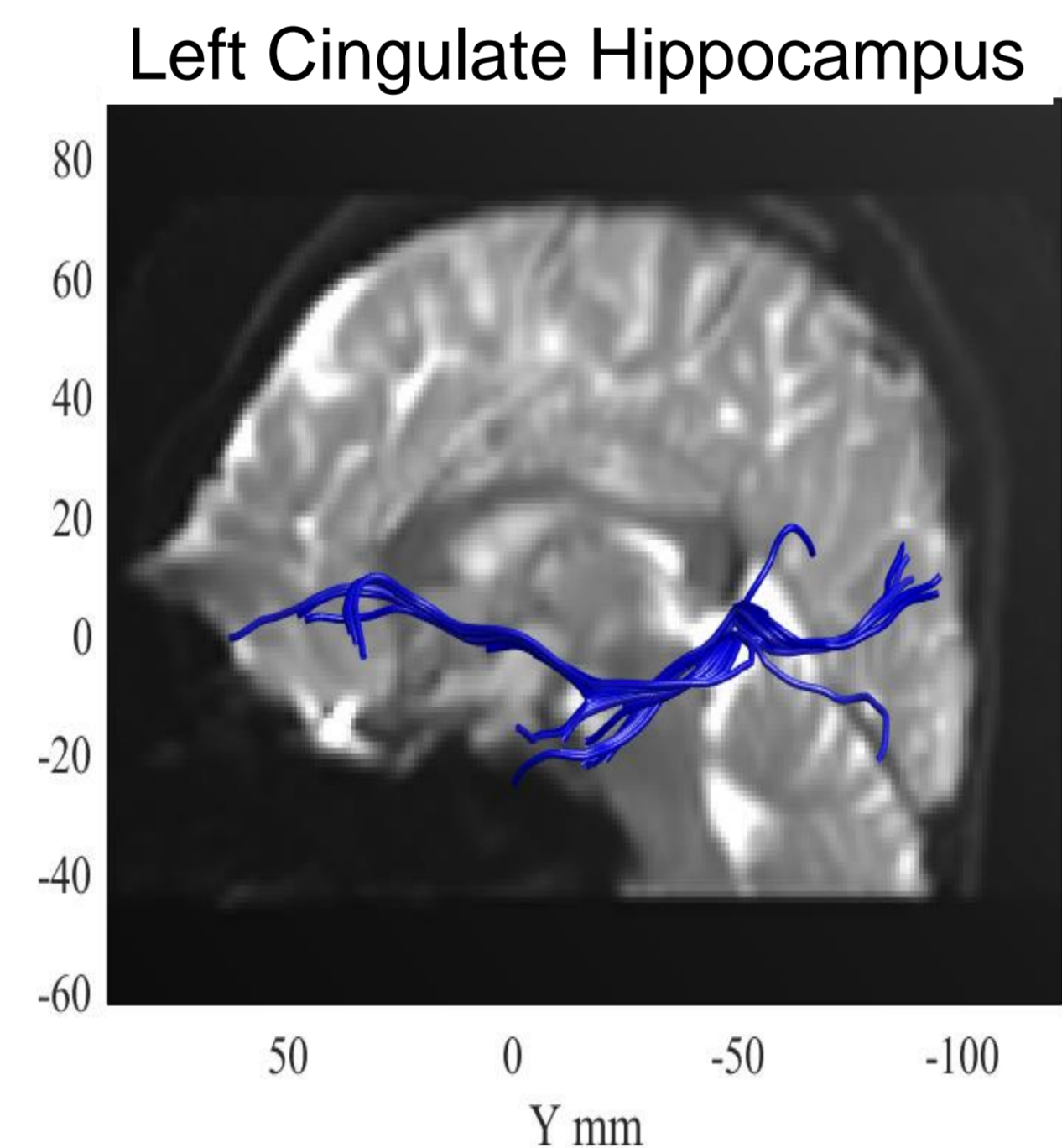
Narcolepsy type 1 is a chronic sleep-wake disease in children, often impairing mental health, social relationships, and education. Studies have found that the level of hypocretin in human narcolepsy with cataplexy is significantly reduced with hypocretin cells losing. Neuroimaging has been widely studied in brain volume and density, nerve fibers and brain function changes of patients. The results are controversial.

OBJECTIVES

In this study, diffusion tensor imaging (DTI) was used to describe the abnormal structural brain regions in children with narcolepsy type 1, and to explore the correlation between brain lesions and clinical features. It help us to further understand the neuropathological mechanism of narcolepsy and to screen possible imaging markers for the diagnosis of narcolepsy in its early term.

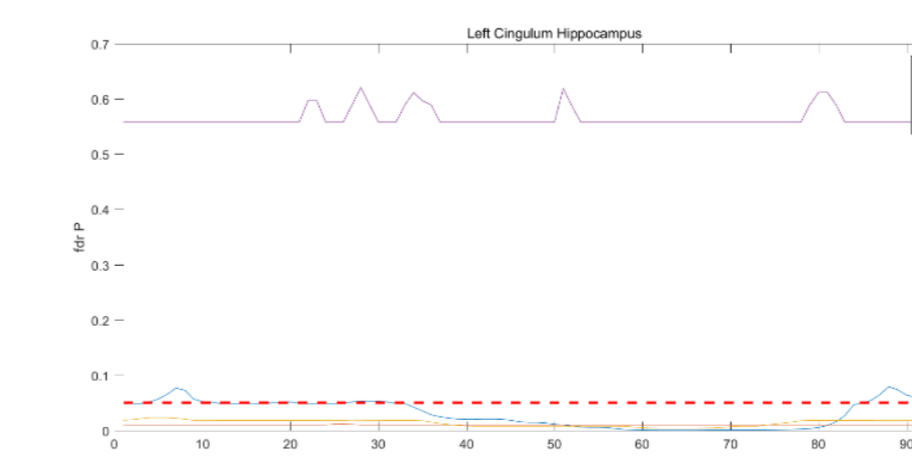
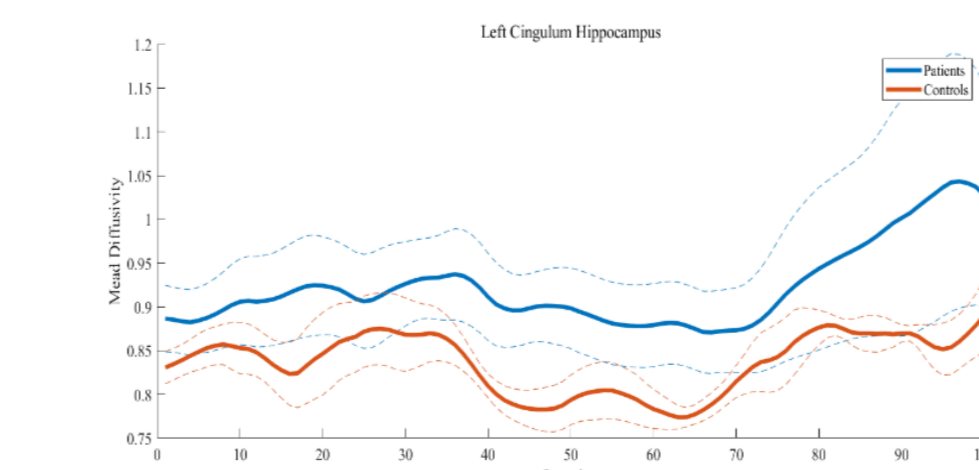
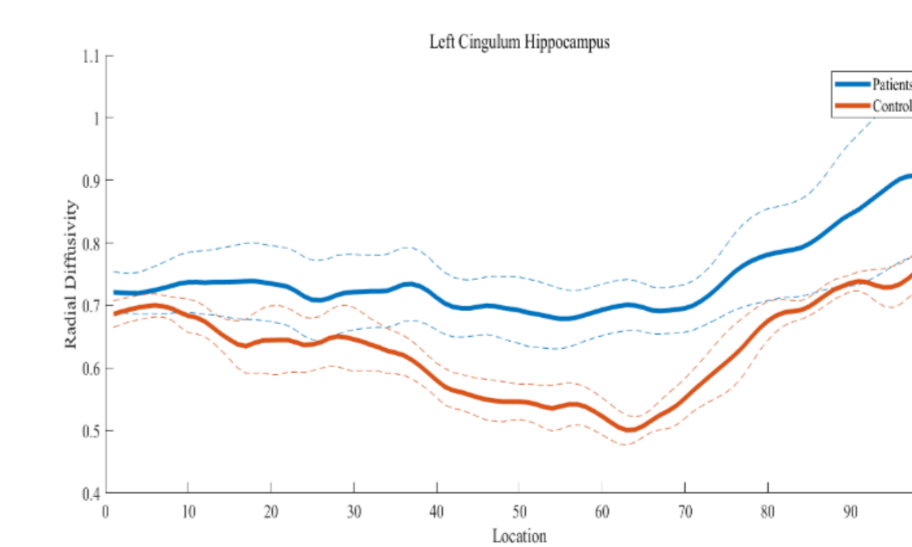
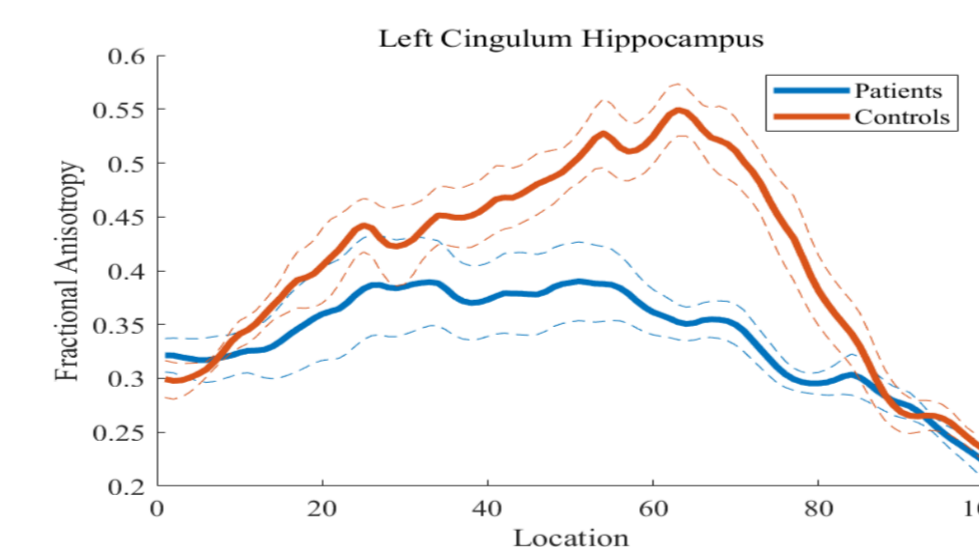
METHODS

We enrolled 30 children with narcolepsy type 1 and 26 age- and sex-matched healthy controls. Collecting clinical data, scanning diffusion tensor imaging, analyzing imaging data by automated fiber quantification (AFQ) were performed on all participants. The correlation between the abnormal brain areas of children with type 1 narcolepsy and clinical data was analyzed.



RESULTS

Automated fiber quantification (AFQ) analysis revealed widespread white matter disturbances, particularly myelin abnormalities, including decreased fractional anisotropy (FA) of the left corticospinal tract, decreased FA and increased radial/mean diffusivity (RD/MD) of the left cingulate hippocampus, increased RD and MD in corpus callosum and bilateral corticospinal tracts, and increased RD in the left uncinate fasciculus. Anxiety, grumpiness, sleep latency and REM sleep latency in children with narcolepsy type 1 were positively correlated with myelin damage of left cingulate hippocampus.



CONCLUSIONS

Children with narcolepsy type 1 have extensive white matter fiber damages mainly of myelin sheath. Such structural changes may underlie attention deficits, learning/memory impairments, and broader cognitive dysfunction frequently observed in this patient population.

REFERENCES

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