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18F-fluorodeoxyglucose-positron emission tomography findings in patients with genetic frontotemporal dementia

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State of art: The current study aims to delineate brain regions with reduced glucose metabolism in patients with genetic frontotemporal dementia (FTD) caused by three common genes in comparison with cognitively normal controls.

Methods: (18)F- fluorodeoxyglucose (FDG)-positron emission tomography (PET) images were retrospectively obtained from 17 genetic symptomatic FTD patients (Mean age 60+6, 7 females). FDG-PET was collected at Lawrence Berkeley National Laboratory on a PET or PET-CT scanner as six 5-min frames acquired 30 min post tracer injection. Standard Uptake Value Ratio (SUVR)images were created for each patient using pons as a reference region. W-score (age-adjusted Z-score) maps were created for each patient's FDG SUVR using a group of neurologically unimpaired controls (N=74, Mean age 65+16, 41 females) as reference.

Result: On review of single-subject w-maps, all *MAPT* carriers showed hypometabolism in the anteromedial temporal lobes with frontal, dorsolateral parietal, and anterolateral temporal hypometabolism seen in lower frequency. Asymmetric hypometabolism in the frontoparietal region was seen in 75% of *GRN* patients. 40% of *C9orf72* patients showed hypometabolism in the posterior cingulate, parietal lobes, cerebellum, insula, thalamus, and caudate while 80% showed hypometabolism in frontotemporal areas. On group-level analysis, compared to controls, besides frontotemporal regions, hypometabolism was seen in the cerebellum and thalamus in *C9orf72* patients and parietal regions in *GRN* mutation. *MAPT* mutation carriers showed frontal and anteromedial temporal hypometabolism.

Conclusion: Parietal and cerebellar hypometabolism were suggestive of *C9orf72* repeat expansion. *GRN and MAPT* mutation carriers showed an asymmetric pattern of hypometabolism and anteromedial temporal lobe hypometabolism, respectively.

Conflicts of interest

None