



# Tau aggregation following subcortical hemorrhage

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A 54-year-old male presented in the department of neurology with cognitive-amnesic deficits and episodes of dystonic spasms in the left hand. In 2020, the patient had a right basal ganglia hemorrhage accompanying a paresis of the left hand. Due to the paresis, he was not able to define the exact symptom onset of the dystonic spasms. Cerebrospinal fluid (CSF) diagnostics (05/2023) showed an increase in total tau as well as phospho-tau. Amyloid- $\beta$ -1-42 was within normal range. The patient was suspected to suffer from a cortico-basal syndrome and a tau-PET/CT scan was performed. Tau-PET/CT imaging ( $[^{18}\text{F}]\text{PI-2620}$ , 0–75 min. p. i.) revealed a higher radiotracer-uptake mainly in the right thalamus extending towards the right striatum/globus pallidus compared to the left side. No further tau-retention was detected. High spatial correspondence between the location of the intracerebral hemorrhage and the increased  $[^{18}\text{F}]\text{PI-2620}$ -PET uptake was observed.

Tau-PET is employed for the detection of neurodegenerative tauopathies, particularly Alzheimer's disease (AD). In AD, predominantly cortical distribution patterns are observed [1]. For second-generation tau-PET tracers like  $[^{18}\text{F}]\text{PI-2620}$ , potential diagnostic value to detect non-AD tauopathies such as progressive supranuclear palsy (PSP) or corticobasal degeneration (CBD) has been discussed [2]. In these disorders, basal ganglia uptake of the tracer is expected. However, post-mortem analyses have shown that tau-deposition can also occur after ischemic or hemorrhagic events [3]. This suggests that the tracer uptake observed in the current case, although specifically indicating tau-pathology may be the consequence of tissue damage following the intracerebral hemorrhage rather than of a neurodegenerative disease.

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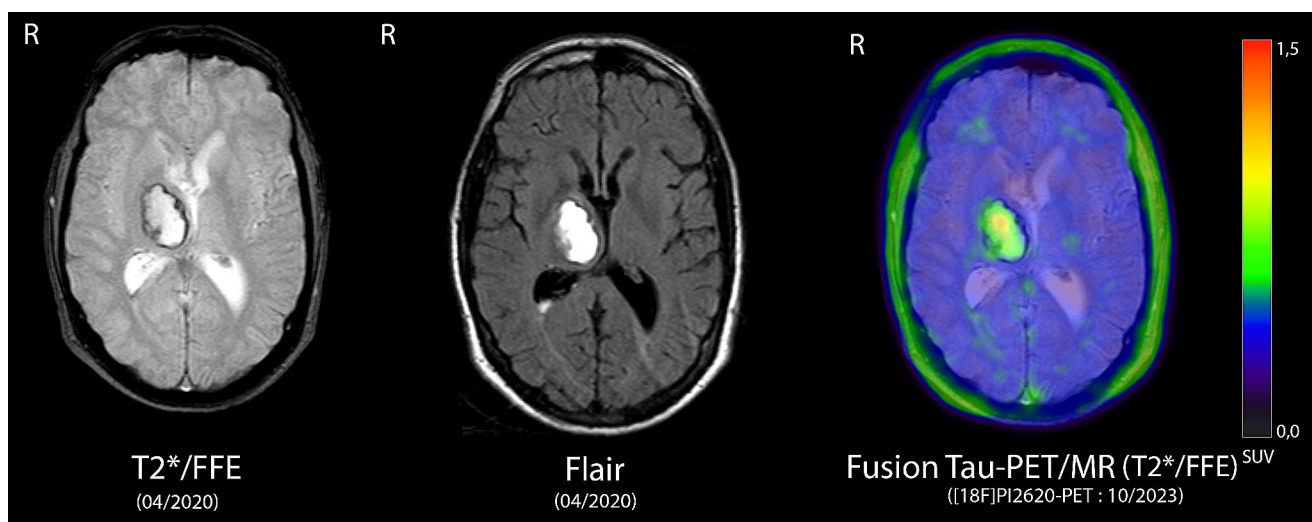
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## Declarations

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