IMAGE OF THE MONTH



Tau aggregation following subcortical hemorrhage

Elena Jaeger¹ • Gérard N. Bischof^{1,4} • Oezguer A. Onur^{2,5} • Marc Schlamann³ • Alexander Drzezga^{1,4,6}

Received: 15 January 2024 / Accepted: 15 February 2024 / Published online: 4 March 2024 © The Author(s) 2024, corrected publication 2024

A 54-year-old male presented in the department of neurology with cognitive-amnestic 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-\u00e3-1-42 was within normal range. The patient was suspected to suffer from a corticobasal syndrome and a tau-PET/CT scan was performed. Tau-PET/CT imaging ([18F]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 [18F]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 [18F]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.



[⊠] Elena Jaeger elena.jaeger1@uk-koeln.de

Department of Nuclear Medicine, University of Cologne, University Hospital of Cologne, Kerpenerstraße 62, 50931 Cologne, Germany

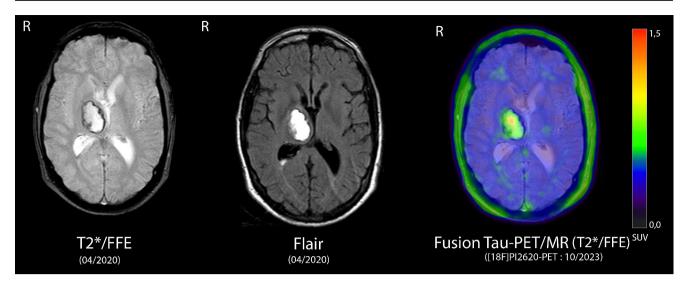
Department of Neurology, University of Cologne, University Hospital of Cologne, Cologne, Germany

Department of Diagnostic and Interventional Radiology, University of Cologne, University Hospital of Cologne, Cologne, Germany

Research Center Juelich, Institute for Neuroscience and Medicine II, Molecular Organization of the Brain, Juelich, Germany

⁵ Research Center Juelich, Institute for Neuroscience and Medicine III, Cognitive Neuroscience, Juelich, Germany

⁶ German Center for Neurodegenerative Diseases (DZNE), Bonn-Cologne, Germany



Acknowledgements AD, GNB and EJ are funded by the Deutsche Forschungsgemeinschaft - Project-ID 431549029 - SFB 1451. OAO was funded by the Marga and Walter Boll-Foundation.

Author contributions All authors contributed to the study conception and design. EJ, GB and AD drafted the manuscript and EJ, GB, OAO, MS and AD revised the manuscript. All authors read and approved the final manuscript.

Funding Open Access funding enabled and organized by Projekt DEAL. Drafting of the manuscript was supported by an educational grant from Lilly.

Declarations

Conflict of interest AD: Research support: Siemens Healthineers, Life Molecular Imaging, GE Healthcare, AVID Radiopharmaceuticals, Sofie, Eisai, Novartis/AAA, Ariceum Therapeutics. Speaker Honorary/Advisory Boards: Siemens Healthineers, Sanofi, GE Healthcare, Biogen, Novo Nordisk, Invicro, Novartis/AAA, Bayer VitalStock: Siemens Healthineers, Lantheus Holding, Structured therapeutics, ImmunoGen. Patents: Patent for 18 F-JK-PSMA- 7 (Patent No.: EP3765097A1; Date of patent: Jan. 20, 2021).

Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this

article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit http://creativecommons.org/licenses/by/4.0/.

References

- Dronse J, Fliessbach K, Bischof GN, von Reutern B, Faber J, Hammes J, et al. In vivo patterns of Tau Pathology, Amyloid-β Burden, and neuronal dysfunction in clinical variants of Alzheimer's Disease. J Alzheimers Dis. 2017;55:465–71. https://doi. org/10.3233/jad-160316.
- Katzdobler S, Nitschmann A, Barthel H, Bischof G, Beyer L, Marek K, et al. Additive value of [18F]PI-2620 perfusion imaging in progressive supranuclear palsy and corticobasal syndrome. Eur J Nucl Med Mol Imaging. 2023;50:423–34. https://doi. org/10.1007/s00259-022-05964-w.
- Hatsuta H, Takao M, Nogami A, Uchino A, Sumikura H, Takata T, et al. Tau and TDP-43 accumulation of the basal nucleus of meynert in individuals with cerebral lobar infarcts or hemorrhage.
 Acta Neuropathol Commun. 2019;7:49. https://doi.org/10.1186/s40478-019-0700-z.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

