Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

Fernando Spina Tensini, MD, PhD, 1*
Léo Coutinho, MD, 1 and
Hélio Afonso Ghizoni Teive, MD, PhD 1.2

1 Movement Disorders Unit, Neurology Service, Internal Medicine
Department, Hospital de Clínicas, Federal University of Paraná,
Curitiba, Brazil, and 2 Neurological Diseases Group, Graduate
Program of Internal Medicine, Internal Medicine Department,
Hospital de Clínicas, Federal University of Paraná, Curitiba, Brazil

References

- Turski CA, Turski GN, Faber J, Teipel SJ, Holz FG, Klockgether T, et al. Microvascular breakdown due to retinal neurodegeneration in ataxias. Mov Disord 2022;37:437–438.
- Spina Tensini F, Sato MT, Shiokawa N, Ashizawa T, Teive HAG. A comparative optical coherence tomography study of spinocerebellar ataxia types 3 and 10. Cerebellum [Internet] 2017;16(4):797–801.
- 3. Jacobi H, Bauer P, Giunti P, Labrum R, Sweeney MG, Charles P, et al. The natural history of spinocerebellar ataxia type 1, 2, 3, and 6: a 2-year follow-up study. Neurology 2011;77(11):1035–1041.
- Manrique RK, Noval S, Aguilar-Amat MJ, Arpa J, Rosa I, Contreras I. Ophthalmic features of spinocerebellar ataxia type 7. J Neuro Ophthalmol [Internet] 2009;29(3):174–179. https://www.amjcaserep.com/abstract/index/idArt/932279
- Pablo LE, Garcia-Martin E, Gazulla J, Larrosa JM, Ferreras A, Santorelli FM, et al. Retinal nerve fiber hypertrophy in ataxia of Charlevoix-Saguenay patients. Mol Vis 2010;2011(17):1871– 1876.

Reply to: "Microvascular Breakdown Due to Retinal Neurodegeneration in Ataxias"

We thank Dr. Tensini and colleagues¹ for their interest in and appreciation of our study describing concurrent retinal microvascular and structural changes in degenerative ataxias

© 2022 The Authors. *Movement Disorders* published by Wiley Periodicals LLC on behalf of International Parkinson and Movement Disorder Society

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs License, which permits use and distribution in any medium, provided the original work is properly cited, the use is non-commercial and no modifications or adaptations are made.

*Correspondence to: Dr. Christopher A. Turski, Department of Ophthalmology, University of Kentucky, 110 Conn Terrace, Suite 550, Lexington, KY 40508, USA; E-mail: christopher.turski@uky.edu

Relevant conflicts of interest/financial disclosures: Nothing to report.

Author roles may be found in the online version of this article.

Received: 19 December 2021; Accepted: 21 December 2021

Published online 10 January 2022 in Wiley Online Library (wileyonlinelibrary.com). DOI: 10.1002/mds.28916

shown by optical coherence tomography (OCT) angiography (OCT-A) and OCT.²

In the previous study, Tensini et al.³ compared diseasespecific effects on retinal morphology in spinocerebellar ataxia types 3 and 10 using OCT. In our study, we used OCT in parallel with OCT-A to assess alterations of retinal microvasculature and morphology simultaneously. We studied a mixed population of patients with spinocerebellar ataxia types 1, 2, 3, and 6, with Friedreich's ataxia, and with multiple system atrophy of cerebellar type. Our study showed changes in retinal vessel density in the superficial vascular complex primarily involving the radial peripapillary capillary network, the capillary density inside the optic nerve head, and the nasal region of the macular superficial vascular plexus in most patients with ataxia across all studied diseases.2 The limited size of each disease group did not allow for the detailed assessment of disease-specific alterations. Nevertheless, we fully agree with Dr. Tensini and coworkers¹ that disease-specific changes might be expected for retinal microvasculature, because they have been found for retinal morphology. In our ongoing studies, we are attempting to define such specific microvascular abnormalities in single genetically determined ataxia entities.

We would like to thank Dr. Tensini and colleagues¹ for their greatly considered comments and would like to emphasize the view that adding retinal phenotyping could potentially open a new field of research toward exploring degenerative ataxias from a different perspective.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable institutional request.

¹Department of Ophthalmology, University of Kentucky, Lexington, KY, USA, ²German Center for Neurodegenerative Diseases (DZNE), Bonn, Germany, ³Department of Ophthalmology, Duke University, Durham, NC, USA, ⁴Department of Neurology, University of Bonn, Bonn, Germany, ⁵Department of Psychosomatic Medicine, University of Rostock, Rostock, Germany, ⁶German Center for Neurodegenerative Diseases (DZNE), Rostock, Germany, and
⁷Department of Ophthalmology, University of Bonn, Bonn, Germany

References

- Tensini FS, Coutinho L, Ghizoni Teive HA. Reply to: microvascular breakdown due to retinal neurodegeneration in ataxias. Mov Disord 2022;37:438–438.
- Turski CA, Turski GN, Faber J, et al. Microvascular breakdown due to retinal neurodegeneration in ataxias. Mov Disord 2022;37: 437–438.
- Tensini FS, Sato MT, Shiokawa N, Ashizawa T, Teive HAG. A comparative optical coherence tomography study of spinocerebellar ataxia types 3 and 10. Cerebellum 2017;16: 797–801