LETTER RE: COGNITIVE RESERVE IN FRONTOTEMPORAL DEGENERATION: NEUROANATOMIC AND NEUROPSYCHOLOGICAL EVIDENCE

Fiona Kumfor, Sydney, Australia; Cristian E. Leyton, Boston, MA; Olivier Piguet, Sydney, Australia: In the article by Placek et al., letter fluency was used as a proxy of executive function and as one of the tasks to assess cognitive reserve (CR). Letter fluency relies on multiple cognitive processes and, in such a diverse clinical sample, is unlikely to reflect a single neurocognitive process. These findings are therefore difficult to interpret.

Further, the results seem interpreted by conflating 2 distinct, but related, mechanisms: brain reserve (i.e., passive process where larger brains are less affected by damage than smaller brains) and CR (i.e., active use of compensatory processes to maintain function despite advancing neuropathology). Accordingly, larger brain volume appears supportive of greater brain reserve, whereas better letter fluency may reflect greater CR. These 2 mechanisms may help reconcile apparently contradictory findings in dementia, whereby higher CR is associated with shorter disease duration in Alzheimer disease (AD), but longer duration in frontotemporal dementia. It is plausible that, in AD, compensatory mechanisms can delay symptomatology, whereas, in frontotemporal dementia, brain reserve may be more important. Longitudinal studies that combine behavioral and neuroimaging data are likely to shed light on this important issue.


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LETTER RE: TUBERCULOUS OPTOCHIASMATIC ARACHNOIDITIS AND VISION LOSS

Hardeep S. Malhotra, Neeraj Kumar, Ravindra Kumar Garg, Lucknow, India: The NeuroImage by Drs. Chee and Dinkin included T1-weighted gadolinium contrast coronal images instead of T2-weighted sequences. The legend should be revised to include more details, such as having performed a shunt procedure, as evident from the image.1


AUTHOR RESPONSE: TUBERCULOUS OPTOCHIASMATIC ARACHNOIDITIS AND VISION LOSS

Marc J. Dinkin, Ru-Ik Chee, New York: We thank Malhotra et al. for pointing out that our NeuroImage was of T1 postcontrast coronal images, not T2, and apologize for the error.1 Malhotra et al. are also correct in observing a right-frontal ventriculoperitoneal shunt, which had been placed to treat hydrocephalus associated with tuberculous meningitis. Hydrocephalus is a common feature of this disease, occurring in up to 26% of adults and 90% of children,2,3 due to either an adhesive leptomeningitis in the basal cisterns (communicating) or obstruction of the aqueduct or fourth ventricle (noncommunicating). Shunting for tuberculous hydrocephalus demonstrated substantial clinical improvement in 7 children, 3 of whom had near complete recovery of vision after weeks of blindness, attributed to reduced compression on the ophthalmic arteries and optic apparatus by exudate and dilated ventricles.4


CORRECTION

Tuberculous optochiasmatic arachnoiditis and vision loss

In the NeuroImage “Tuberculous optochiasmatic arachnoiditis and vision loss” by R.I. Chee and M.J. Dinkin,1 the image should have been labeled “T1 postcontrast coronal images” rather than “T2 coronal MRI.” The authors regret the error.

REFERENCE


Author disclosures are available upon request (journal@neurology.org).

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Tuberculous optochiasmatic arachnoiditis and vision loss

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