Response to Professor Kawada

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We thank Professor Kawada for his interest in our study regarding ongoing impairments following transient ischaemic attack (TIA).[1] We are particularly encouraged that he recognised the need to improve care for TIA patients presenting with signs of fatigue, psychological impairment or cognitive impairment.

The study of impairments post-TIA is currently under-researched. Indeed, the findings of our systematic review,[2] which aimed to establish prevalence of impairments post-TIA/minor stroke, were inconclusive because the quality of the included studies was not high. Our review identified the limitations of existing research and our current study was conducted in response to this gap in the literature. We addressed these limitations through subsequently conducting a retrospective cohort study which has a large sample size, included matched controls and adjusted for confounding variables and presence of impairments pre-TIA. We believe this robust study design provides reliable estimates to measure the association between TIA and subsequent impairments. However, future research is required to determine the incidence, prevalence and natural history of impairments post-TIA.

In response to Professor Kawada's second comment regarding the possibility of impaired cognitive function being present prior to TIA, for this reason we purposefully adjusted for presence of cognitive impairment prior to TIA in our main analysis. Furthermore, we conducted an exploratory analysis which excluded people with cognitive impairment recorded prior to TIA. In this exploratory analysis the association between TIA and cognitive impairment remained; indeed, the adjusted hazard ratio actually increased from 1.45 (95% CI, 1.28–1.65) to 1.54 (95% CI, 1.35–1.77). However, as we discussed in the limitations, it is possible that cognitive impairment prior to TIA may have been undetected or not recorded. We agree that caution also should be paid regarding inclusion of patients with dementia; however, only 4% and 3.4% of TIA patients and controls, respectively, had dementia recorded.

The mechanism of residual impairments post-TIA is unknown and in our discussion we hypothesised three potential explanations: (i) presence of cerebral infarction, i.e. minor stroke misdiagnosed; (ii) undetected microinfarcts; and (iii) psychological impact of the event. An alternative hypothesis could be that cognitive impairment is caused by vascular risk factors which predispose TIA. However, a systematic review by Blom et al. (2013) found inconsistent results for the association between vascular risk factors and cognitive decline in patients with dementia.[3] We agree that the mechanism of cognitive impairment necessitates further research and this information may facilitate the development of an intervention to improve cognition in TIA patients.

References

[1] Turner GM, Calvert M, Feltham MG, Ryan R, Marshall T. Ongoing impairments following transient ischaemic attack: retrospective cohort study. Eur J Neurol. 2016; doi: 10.1111/ene.13088

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[3] Blom K, Emmelot-Vonk MH, Koek HL. The influence of vascular risk factors on cognitive decline in patients with dementia: a systematic review. Maturitas. 2013;76(2):113-7.