



A comment on hypertension and the risk of poststroke epilepsy after primary intracerebral haemorrhage

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Provenance: This is an invited article commissioned by Section Editor Dr. Rui Liu (Department of Neurology, Jinling Hospital, Nanjing University School of Medicine, Nanjing, China).

Response to: Zelano J. A new study investigates the long-term risks of poststroke epilepsy after primary intracerebral haemorrhage. *AME Med J* 2017;2:137.

Received: 01 December 2017; Accepted: 09 December 2017; Published: 29 December 2017.

doi: 10.21037/amj.2017.12.06

View this article at: <http://dx.doi.org/10.21037/amj.2017.12.06>

We would like to thank Dr. Zelano for his accurate coverage of our recent study (1). Concerning the definition of what is considered an early seizure after intracerebral haemorrhage (ICH), we would like to point out that no recommendation or explicit consensus existed when we began collecting our data (2,3). At that time several different cut-off points for differentiating between early and late seizures were used in the field, the most frequent ones being at 7 and 14 days. We chose the 14-day time frame for early seizures partly to make our results more comparable with those released at the time, e.g., by Bladin *et al.* and Cervoni *et al.* (4,5). This heterogeneity in definitions has been a significant problem to date, and we recommend all future studies to follow the recently established guidelines to ameliorate comparability (6).

In our study, we observed hypertension to predict lower incidence of later poststroke epilepsy. To our knowledge, this kind of observation has not been made before. In our article we speculated the possible confounding effects of advice on alcohol consumption, cavernous malformations and cerebral amyloid angiopathy. Since patients with hypertension should be more likely to receive information in primary health care on the harmful effects of alcohol, it could be that alcohol use and abuse are less likely in this group compared to those without hypertension. Only 65 of our 615 patients reported alcohol abuse, but we did not collect more specific data on alcohol use (1).

Since our patients underwent imaging only after the

ICH, we cannot rule out the possibility of bleeding due to cavernous malformations, since the haemorrhage can destroy the site of the lesion making them sometimes very challenging to detect afterwards. Furthermore, no evidence exists of correlation between hypertension and the risk of ICH-associated with cavernous malformations, leaving open the possibility that bleedings due to cavernomas were more frequent in the non-hypertensive group of subcortical bleedings compared to the group with hypertension (7). Cavernous malformations are also a known independent risk factor for epilepsy, so it is hypothetically possible that an ICH due to a cavernoma is more epileptogenic than a truly primary hemorrhage. However, there is no data to support this theory, and therefore it is purely speculation.

Cerebral amyloid angiopathy is another possible structural confounding factor in our study. It cannot be diagnosed with CT alone, and since only a small minority of our patients underwent MRI, it is possible that we missed some patients with the named condition.

To conclude, we agree with Dr. Zelano in his statement that further studies are needed to replicate our unexpected finding regarding hypertension and the risk of post stroke epilepsy after primary ICH, before we can evaluate if it is a phenomenon that also applies outside our study cohort. We encourage further research on the subject and anticipate new results to answer the questions that still remain in this field.

Acknowledgements

None.

Footnote

Conflicts of Interest: S Juvela is an associate editor for *the European Journal of Neurology* and a member of the editorial board of *Stroke*. P Saloheimo is an associate editor for *the European Journal of Neurology* and reports Orion stock ownership. The other authors have no conflicts of interest to declare.

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doi: 10.21037/amj.2017.12.06

Cite this article as: Lahti AM, Juvela S, Saloheimo P, Tetri S. A comment on hypertension and the risk of poststroke epilepsy after primary intracerebral haemorrhage. *AME Med J* 2017;2:180.