
We thank Reale et al1 for their interest in our study,2 and for describing their interesting case and suggestion that the absence of a diffusion-weighted imaging (DWI) lesion in a third of our patients with stroke was because of a shorter duration of ischemia.

Disappearing DWI lesions are well documented to be associated with short duration and lesser severity of ischemic insult in animal models.3 Similarly, in patients, milder strokes are less likely to produce a DWI lesion than severe strokes.4 Other factors influence DWI lesion visibility. The DWI lesion lasts longer in white than in gray matter, and the DWI lesion disappears more quickly in mild than in severe stroke,5 presumably reflecting the degree of tissue damage.

In our cohort of patients with a nondisabling stroke referred to by Reale et al,1 it was not possible to estimate the duration of ischemia precisely. None of our patients underwent angiography or clot retrieval and few were thrombolysed because of the mildness of the stroke or delayed presentation. However, we did record whether the stroke symptoms had resolved by the time of magnetic resonance imaging scan: 148 of 188 patients with a lesion on the DWI sequence at presentation and 46 of 77 patients without a lesion on the DWI sequence still had ongoing neurological symptoms when the magnetic resonance imaging scan was performed (P=0.06). Strokes were slightly more severe in patients with a DWI lesion (median National Institutes of Health Stroke Scale [NIHSS] 2; interquartile range, 1–2–4) than without a DWI lesion (median NIHSS 2; interquartile range, 1–2; P=0.03) but there was no difference in time to scanning or prevalence of vascular risk factors between the patients with and without a DWI lesion. Therefore, we think it is unlikely that the patients without a DWI lesion simply had a shorter duration of ischemia.

We did note that the patients with a DWI lesion were more likely to have a higher burden of white matter hyperintensities despite being the same age, which might suggest that patients with a DWI lesion were more vulnerable to showing ischemic change than those without a DWI lesion as suggested in our article. Other studies have shown that patients with preexisting brain vascular disease are more likely to have larger infarcts, more infarct growth and worse outcomes6 than those without previous lesions. Clearly, further research is required to evaluate the role of brain vulnerability in determining the response to ischemic stroke.

Sources of Funding

The Wellcome Trust funded the original study.

Disclosures

None.

Stephen D.J. Makin, MB ChB, MRCP
Joanna M. Wardlaw, MD

Centre for Clinical Brain Sciences, University of Edinburgh
Edinburgh, United Kingdom


Stephen D.J. Makin and Joanna M. Wardlaw

Stroke. 2016;47:e54; originally published online January 19, 2016; doi: 10.1161/STROKEAHA.115.012258

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2016 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/47/3/e54

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/