Correspondence

Herpes Zoster and Stroke: Implications for Therapy and Vaccination

TO THE EDITOR—Langan et al [1] present convincing evidence that acute herpes zoster infection is associated with an increased risk of stroke in the following 6 months, that this risk is increased in herpes zoster ophthalmicus (HZO), and that antiviral therapy is partially protective. The use of the self-controlled case series method in their study increases its validity, as does the fact that their findings echo those of previous, less rigorous cohort studies. The accompanying commentary reviews the evidence that the increased risk of stroke after zoster may be due to direct infection and viral replication within arterial walls, with subsequent vascular damage and disruption of vascular flow resulting in cerebral ischaemia or haemorrhage [2].

Antiviral therapy for zoster has long been known to reduce morbidity through reduction in rash and acute pain, and also in incidence and severity of postherpetic neuralgia (PHN) [3]. In the United Kingdom, therapy is currently recommended up to 72 hours after onset of rash, and only later than this in patients at increased risk of severe or complicated infection, as its benefit if started after 72 hours has not been clearly demonstrated [4]. In Langan and colleagues’ study, patients were defined as having had antiviral therapy if this was prescribed in the 2 weeks after their zoster diagnosis, and no data are provided on actual timing of therapy within this period. Because the time course of vascular infection and subsequent anatomical changes may differ from the effect of the virus on nerves and skin, it is possible that late initiation of therapy may provide additional benefit in stroke risk reduction compared with effects on local symptoms and PHN, and further research in this area would be of major benefit in guiding national recommendations on timing of therapy.

Of the 11,997 patients who had first-ever zoster and first-ever stroke or transient ischemic attack (TIA) during the study period, nearly half had had TIAs and were excluded from the analysis. Although a diagnosis of TIA may have been less robust than a stroke diagnosis using this methodology, it would have been useful to provide data on this group, because if we assume that TIAs and strokes in this context have similar pathogenesis, the observed effect of zoster on risk of cerebrovascular accident as a whole would have been even greater. Numbers of patients in the HZO group were very small, limiting the conclusions that can be drawn, and inclusion of patients with TIAs as their first episode may have added statistical power.

Finally, there are implications for zoster vaccination [5]. Current UK policy is to vaccinate all individuals between 70 and 79 years of age, delivering vaccine to 1 age cohort per year to ensure adequate vaccine supplies [6]. In the future, there may be a role for vaccination of younger individuals, or possibly using a process based on stratification of risk of cerebrovascular disease. Ultimately, avoidance of childhood varicella infection through universal vaccination may have the greatest impact on stroke and other acute vascular events in later life.

Note

Potential conflicts of interest. Author certifies no potential conflicts of interest.

The author has submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

Ann L. N. Chapman
Department of Infectious Diseases, Monklands Hospital, Airdrie, United Kingdom

References


Correspondence: Ann L. N. Chapman, BM BCh, FRCP, DTM&H, MSc, PhD, MD, Department of Infectious Diseases, Monklands Hospital, Airdrie ML6 0JS, United Kingdom (ann.chapman2@nhhs.net).

Clinical Infectious Diseases 2014;59(9):1185
© The Author 2014. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.
DOI: 10.1093/cid/ciu560