Editors’ note: Epidemiology of NMOSD in Sweden from 1987 to 2013: A nationwide population-based study

In their retrospective observational cohort study of 294 patients with neuromyelitis optica spectrum disorder (NMOSD), Drs. Jonsson et al. summarized the rising incidence rate of this condition in Sweden between 1987 and 2013. The investigators postulate that the higher incidence rate—0.79 per 1,000,000 person-years from 0.30 per 1,000,000 person-years—may have been attributed, in part, to heightened awareness of the disease and greater availability of MRI and NMO antibody testing. These incidence estimates mirror what have been reported in other predominantly Caucasian cohorts. In response, Drs. Papp et al. reiterate that although incidence and prevalence estimates are similar between nations of similar ethnic profiles, Southern Denmark has reported some of the highest prevalence (4.4 per 100,000 person-years) and incidence rates (4.0 per 1,000,000 person-years). Drs. Jonsson et al. comment that the higher estimates reported by Asgari et al. from Southern Denmark may be partially explained by the prospective nature of their study and the fact that patients from that study were evaluated for NMOSD even if optic neuritis was the presenting symptom. Based on the collective results from these studies, it is likely that NMOSD remains an underrecognized condition, and physicians should have a lower threshold to evaluate these patients with serum antibody testing and MRI, given the treatment considerations that are specific to NMOSD compared with MS. A correction to figure 5 was published in the September 10, 2019, issue of Neurology.

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Reader response: Epidemiology of NMOSD in Sweden from 1987 to 2013: A nationwide population-based study

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We read the nationwide Swedish epidemiologic study of neuromyelitis optica spectrum disorder (NMOSD) published by Jonsson et al.1 with great interest. The study revealed similar prevalence (1.04 per 100,000 people [CI: 0.85–1.26]) and incidence (0.79 per 1,000,000 person-years [CI: 0.55–1.03]) of NMOSD to the prevalence (1.09 per 100,000 people [CI: 0.81–1.44]) and incidence (0.70 per 1,000,000 person-years [CI: 0.46–1.02]) estimates of the recent nationwide Danish study2 and also to the incidence study of NMOSD from the Region of Central Denmark (incidence: 0.12 per 1,000,000 person-years).3 These studies from Scandinavia showed similar data to each other and also to other white populations, and they contrast with the results of the study from the region of Southern Denmark,4 reporting the highest prevalence (4.4 per 100,000 people [CI: 3.1–5.7]) and incidence (4.0 per 1,000,000 person-years [CI: 3.0–5.4]) so far in a predominantly Caucasian population.
We would also like to bring attention to figure 5 (Comparison of incidence and prevalence in Sweden with other regions) concerning the incidence estimates. The incidence of NMOSD in Australia and New Zealand published by Bukhari et al. is 0.37 per 1,000,000 person-years (CI: 0.35–0.39) and not 3.7 per 1,000,000 person-years (CI: 3.5–3.9), as it is incorrectly shown in figure 5.

Incidence (A) and prevalence (B) of neuromyelitis optica/neuromyelitis optica spectrum disorder in other regions from available publications. Horizontal error bars represent the mean surrounded by a 95% CI.


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Editors’ note: Low hemoglobin and hematoma expansion after intracerebral hemorrhage

To corroborate previous reports that lower hemoglobin levels may be associated with hematoma expansion (HE) and poor functional outcomes after intracerebral hemorrhage (ICH), Drs. Roh et al. queried their single-center prospective ICH registry of 256 patients. After adjustment for known predictors of HE, each 1 g/dL fall in hemoglobin levels was associated with a 20% higher odds of HE and 24% higher odds of severe disability (modified Rankin Scale score 4–6) at 3 months. Mediation analysis demonstrated that HE contributed to the poor long-term outcomes seen in patients with lower admission hemoglobin levels. Yu et al. address important unmeasured confounders—notably, coagulopathy—which could have also contributed to adverse radiographic and clinical outcomes. However, as Dr. Roh responds, patients in this study were excluded if there was evidence of coagulopathy secondary to systemic disease. Furthermore, the investigators adjusted for the use of therapeutic anticoagulation, irrespective of coagulation studies. Ultimately, both groups agree that these results warrant replication in further studies.

James E. Siegler III, MD, and Steven Galetta, MD
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Reader response: Low hemoglobin and hematoma expansion after intracerebral hemorrhage

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With interest and appreciation, I read the article by Roh et al.,1 which suggested that low hemoglobin levels could be related to hematoma expansion in intracerebral hemorrhage. Although previous studies have shown anemia is associated with worse outcomes in these patients,2,3 the study by Roh et al. is the first one showing that hematoma expansion mediates this association. However, some issues should be noted.

The time from onset to baseline CT was relatively long (median 5.6 hours), which could influence the identification of hematoma expansion. Moreover, some important results of laboratory coagulation testing, such as prothrombin time and international normalized ratio, were not included in multivariate logistic regression, which could make the results not robust enough. Therefore, their findings still need to be confirmed by further studies.

Author response: Low hemoglobin and hematoma expansion after intracerebral hemorrhage

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The comments posited by Yu et al.1 appropriately highlight limitations of single-center observational data and the importance of external replication for our findings. While adjusting for laboratory coagulation testing is important to consider, it is worth noting that we excluded patients with laboratory evidence of coagulopathy because of systemic medical disease. In addition, we adjusted for anticoagulation use, which should account for prothrombin time/international normalized ratio elevations due to a medication effect. Subsequently, it would be unlikely that the addition of laboratory coagulation markers in a multivariable model would affect the conclusions seen in our cohort.

If our findings are replicated, the need to study the mechanism(s) behind the findings is important. Given that underlying medical illness can be associated with lower hemoglobin levels, it is plausible that the relationship of lower hemoglobin levels with hematoma expansion and poor outcomes seen in our cohort is driven by the underlying disease rather than the hemoglobin level itself. Translational studies may be the only way to disentangle this potentially confounding factor; it is important to consider this factor before advocating for liberal red blood cell transfusion in intracerebral hemorrhage, as there are known complications associated with unnecessary red blood cell transfusions.


CORRECTION

Vascular safety of erenumab for migraine prevention

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In the article "Vascular safety of erenumab for migraine prevention" by Kudrow et al.,1 Dr. Pascual's affiliation should have been listed as the University of Cantabria. The authors regret the error.

Reference

Vascular safety of erenumab for migraine prevention

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