Journal of Neurology (2024) 271:2926–2927
https://doi.org/10.1007/s00415-024-12238-y

CORRECTION

Correction to: Neurofilament light in plasma is a potential biomarker of central nervous system involvement in systemic lupus erythematosus

Maria Boge Lauvsnes[1] · Henrik Zetterberg[8,9,10,11] · Kaj Blennow[8,9] · Jan Terje Kvaløy[2,12] · Anne Bolette Tjensvoll[3] · Stian Maroni[4] · Mona K. Beyer[14,15] · Ole Jacob Greve[5] · Ingeborg Kvivik[2] · Guido Alves[3,6,13] · Lasse Gunnar Gøransson[7,16] · Erna Harboe[7] · Shunsei Hirohata[17] · Roald Omdal[1,16]

Published online: 23 March 2024

Correction to:
Journal of Neurology (2022) 269:3064–3074
https://doi.org/10.1007/s00415-021-10893-z

The original version of this article unfortunately contained a mistake. In Table 4, the second paragraph regarding SLEDAI, NP items, is wrong and should be excluded from the table. It was based on results from a preliminary regression analysis that were included in the table. In an old dataset four patients were wrongly marked to have neuropsychiatric manifestations with consequences for scoring in the SLEDAI. Later in the process this was double-checked against the original database, where none of the patients were scored with NP-items in the SLEDAI. Correct data have been used everywhere else in the manuscript, but we are sorry to say that it was overlooked in the table and in a reference to the table in the text.

In section "Multivariable regression analyses with both laboratory and clinical variables" paragraph which previously read:

Based on the results of initial regression analyses, we included creatinine, Q-albumin, and aPL in multivariable regression analyses. In the above-mentioned models, the

The original article can be found online at https://doi.org/10.1007/s00415-021-10893-z.

✉ Maria Boge Lauvsnes
mariaboge@hotmail.com

1 Department of Rheumatology, Stavanger University Hospital, Pb. 8100 Forus, 4068 Stavanger, Norway

2 Research Department, Stavanger University Hospital, Stavanger, Norway

3 Department of Neurology, Stavanger University Hospital, Stavanger, Norway

4 Clinical Neuropsychology Unit, Division of Psychiatry, Stavanger University Hospital, Stavanger, Norway

5 Department of Radiology, Stavanger University Hospital, Stavanger, Norway

6 The Norwegian Centre for Movement Disorders, Stavanger University Hospital, Stavanger, Norway

7 Department of Internal Medicine, Stavanger University Hospital, Stavanger, Norway

8 Clinical Neurochemistry Laboratory, Sahlgrenska University Hospital, Mölndal, Sweden

9 Department of Psychiatry and Neurochemistry, Institute of Neuroscience and Physiology, The Sahlgrenska Academy at the University of Gothenburg, Mölndal, Sweden

10 UK Dementia Research Institute at UCL, London, UK

11 Department of Neurodegenerative Disease, UCL Institute of Neurology, London, UK

12 Department of Mathematics and Physics, Bioscience and Environmental Engineering, University of Stavanger, Stavanger, Norway

13 Department of Chemistry, Bioscience and Environmental Engineering, University of Stavanger, Stavanger, Norway

14 Instiute of Clinical Medicine, University of Oslo, Oslo, Norway

15 Division of Radiology and Nuclear Medicine, Oslo University Hospital, Oslo, Norway

16 Department of Clinical Science, Faculty of Medicine, University of Bergen, Bergen, Norway

17 Department of Rheumatology and Infectious Diseases, Kitasato University School of Medicine, Sagamihara, Japan

SLEDAI NP items, motor function, and a history of seizures were found to exert an effect on plasma NfL concentrations (Table 4). However, SDI scores, lacunar infarcts, cognitive (all domains), or specifically memory or language dysfunction in multivariable regression analyses did not exert an effect on the plasma NfL concentrations (data not shown).

should have read:

Based on the results of initial regression analyses, we included creatinine, Q-albumin, and aPL in multivariable regression analyses. In the above-mentioned models, motor function, and a history of seizures were found to exert an effect on plasma NfL concentrations (Table 4). However, SDI scores, lacunar infarcts, cognitive (all domains), or specifically memory or language dysfunction in multivariable regression analyses did not exert an effect on the plasma NfL concentrations (data not shown).

The corrected Table 4 is given below.

Table 4 Associations between NfL plasma concentrations and laboratory and clinical variables in 67 patients with SLE

Variables in final model	β	CI	p value	adj. R^2
SLEDAI, all items				
Age	0.002	0.001 to 0.002	< 0.001	
Creatinine	0.009	0.004 to 0.013	< 0.001	
Q-albumin	0.01	− 0.036 to 0.064	0.58	
SLEDAI score	0.032	0.005 to 0.059	0.02	
				0.46
Motor function				
Age	0.001	< 0.001 to 0.002	0.01	
Creatinine	0.09	0.005 to 0.014	< 0.001	
Q-albumin	0.03	− 0.02 to 0.08	0.2	
Motor function	0.34	0.12 to 0.56	0.003	
				0.50
Seizure disorder				
Age	0.001	< 0.001 to 0.002	0.003	
Creatinine	0.006	0.001 to 0.011	0.02	
Q-albumin	0.056	0.002 to 0.11	0.04	
Seizure disorder	0.53	0.13 to 0.93	0.01	
				0.46

Linear multivariable regression models with log-transformed pNfL as the dependent variable. Anti-phospholipid antibodies were excluded from all final models

adj adjusted, *SLEDAI* systemic lupus erythematosus disease activity index