

# Reduced Hospital Incidence of Autoimmune Encephalitis During the COVID-19 Pandemic

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## Abstract

### Objectives

The aim of this study was to analyze changes in hospital incidence cases and disease severity of autoantibody-associated autoimmune encephalitis (AE) during the COVID-19 pandemic compared with the prepandemic period.

### Methods

A retrospective multicenter study analyzed data from 24 centers within the German Network for Research on Autoimmune Encephalitis (GENERATE). Patients with a new diagnosis of definite antibody-positive autoimmune encephalitis from 2017 to 2022 were included and divided into prepandemic (2017–2019) and pandemic (2020–2022) periods.

### Results

Among 392 patients, 227 were diagnosed before and 165 during the pandemic (mean 9.5 vs 6.9 per site,  $p = 0.04$ ). A reduction was observed in cases with antibodies to neuronal surface antigens (174 vs 122 cases; mean 7.3 vs 5.1 per site,  $p = 0.02$ ), while cases with antibodies against intracellular antigens remained stable ( $p = 0.40$ ). No differences were observed in disease severity, age, or sex distribution between periods.

### Discussion

This study provides clinical data on antibody-positive AE before and during the COVID-19 pandemic. The findings do not support the hypothesis that SARS-CoV-2 infection triggers autoantibody-associated AE or increases disease severity.

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### Supplementary Material

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Coinvestigators are listed online at [Neurology.org/NN](https://www.neurology.org/NN).

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## Introduction

Several reports suggested an increased risk of autoimmune encephalitis (AE) as an immunologic CNS consequence after SARS-CoV-2 infection.<sup>1-3</sup> Indeed, postinfectious peripheral nervous system autoimmune syndromes have been found to be associated with a preceding infection with SARS-CoV-2 or immunization with SARS-CoV-2 spike glycoprotein.<sup>4-6</sup> Other studies reported an increase in antibodies to known and potentially unknown neuronal surface (sAb) and intracellular (iAb) antigens in patients with COVID-19.<sup>7,8</sup> However, 2 large national referral laboratory-based studies of autoantibody prevalence in serum and CSF found no increase in sAb during the pandemic period.<sup>9,10</sup> Still, information on the clinical presentation of patients and disease severity is lacking, and it needs to be shown whether these laboratory findings actually reflect changes in de novo occurrence of clinically definite AE.

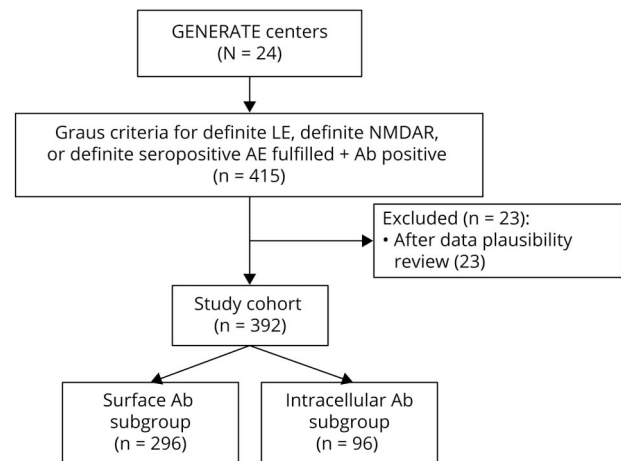
## Methods

We performed a retrospective multicenter, hospital-based registry study in 24 centers (23 in Germany [in 12/16 states], 1 in Austria) within the registry of the German Network for Research on Autoimmune Encephalitis (GENERATE). We analyzed a 6-year period from 2017 to 2022, divided into a pre-pandemic period (2017–2019) and a pandemic period (2020–2022), and determined overall and per-study-site case numbers.

Only patients fulfilling the Graus criteria<sup>11</sup> for definitive antibody-positive AE, including definite antibody-positive limbic encephalitis and definite NMDA-receptor encephalitis, were included in this study (Figure 1). Both inpatient and outpatient individuals were part of the study. Depending on the target antigen, patients were assigned to an sAb or iAb subgroup. Antibody analyses were performed according to the local procedures of each respective clinic. These typically comprised CSF analyses using cell-based assays for detection of antibodies to neuronal surface antigens and immunoblots for analysis of onconeurological antibodies. Further details on data acquisition, antibody classification, and patient selection are described in eMethods.

Incident cases including subgroups by antibody type were aggregated at the study site level so that data within sites could be analyzed as dependent data between the 2 periods (using the paired *t* test and the nonparametric Wilcoxon signed-rank test for robustness). All other variables (age (metric variable)/disease severity (ordinal variable)/sex (nominal variable)) were available at the patient level and were analyzed as 2 independent groups (using the Wilcoxon–Mann-Whitney test for ordinal and metric variables and Chi-square tests for the nominal variable). All statistical analyses were performed using OriginPro (v.2020b).

**Figure 1** Flowchart Illustrating Patient Selection



Only patients fulfilling the Graus criteria for definitive antibody-positive autoimmune encephalitis (AE), including definite antibody-positive limbic encephalitis and definite NMDA-receptor encephalitis, with onset of autoimmune encephalitis between 2017 and 2022 were included in this study. Patients were required to have autoantibodies (Abs) typically associated with autoimmune encephalitis. A total of 23 patients were excluded after data plausibility review: 12 patients harbored only aquaporin-4 or MOG antibodies. They were excluded from the analysis to prevent selection bias because these patients are recorded within a different specialized registry in Germany. A total of 8 patients had isolated SOX1, isolated Yo, or isolated VGKC antibodies (without detection of LGI1 or CASPR2 antibodies). They were excluded from the analysis to avoid inclusion of unspecific antibody reactivity and isolated cerebellar affection. Three patients were duplicates.

## Standard Protocol Approvals, Registrations, and Patient Consents

The lead protocol (Ethikkommission Lübeck, University Lübeck, Germany, 13-162) was approved by all participating institutional review boards. Written informed consent was obtained from all enrolled patients or their legal representatives before inclusion into the GENERATE registry.

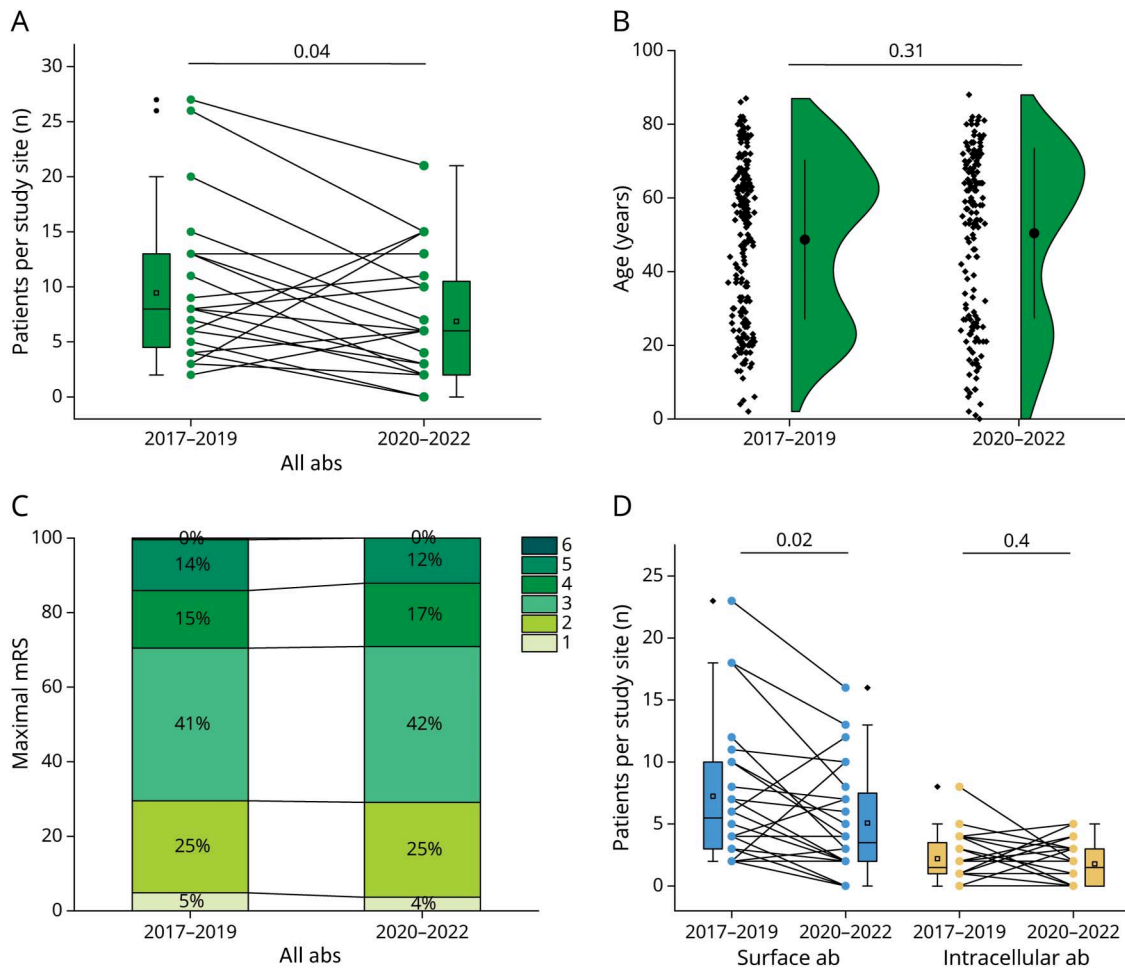
## Data Availability

The data that support the findings of this study are available from the corresponding author on reasonable request.

## Results

A total number of 392 patients (211 female, 181 male) were included in the analysis (Figure 1). The overall three-year number of de novo autoantibody-associated AE cases decreased during the pandemic period (2020–2022) compared with the pre-pandemic period (2017–2019), from 227 (9.5 cases/study site ([SD]: 6.9)) to 165 (6.9 cases/study site [5.7]) cases ( $p = 0.04$ , paired *t* test) (Figure 2a). The age distribution between the 2 periods was similar (median 55 years (IQR 27–66) and 58 years (IQR 27–69.5);  $p = 0.31$ , Wilcoxon–Mann-Whitney test) (Figure 2B). No evidence for differences in disease severity measured using the modified Rankin Scale (mRS) ( $p = 0.82$ , Wilcoxon–Mann-Whitney test) (Figure 2C) or sex distribution (55.9% vs 50.9% female,  $p = 0.32$ ,  $\chi^2$  test) was detected.

**Figure 2** Analysis of Autoimmune Encephalitis Hospital Incidence Cases Per Study Site, Age, and Disease Severity in the Prepandemic (2017–2019) and Pandemic (2020–2022) Periods



(A) Line series and boxplot graph depicting the number of all autoimmune encephalitis cases per study sites. Each dot represents data of a specific data set. The boxplots show aggregated data. (B) Violin plot showing age at onset of disease in all patients. Each dot represents one patient. (C) 100% stacked column chart illustrating the disease severity (maximal modified Rankin Scale [mRS] scores) of all patients with autoimmune encephalitis. (D) Line series and boxplot graphs showing the number of new autoimmune encephalitis cases per study site with antibodies against neuronal surface (blue) or intracellular antigens (yellow). Each dot represents data of a specific data set. The boxplots show aggregated data.

Analyzed by autoantibody group (total: 296 sAb, 96 iAb), we found a reduction in overall ( $n = 174$  vs  $n = 122$ ) and per-study-site hospital incidence cases in patients with AE with sAb (prepandemic period: 7.25 cases/study site [5.7]; pandemic period: 5.1 cases/study site [4.4],  $p = 0.02$ , paired  $t$  test), but not in those with iAb (overall  $n = 53$  vs  $n = 43$ ) (prepandemic period: 2.2 cases/study site [1.9]; pandemic period: 1.8 cases/study site [1.7],  $p = 0.40$ , paired  $t$  test) (Figure 2D).

Figure 3 displays the relative distribution of detected antibody specificities during the prepandemic and pandemic phases.

## Discussion

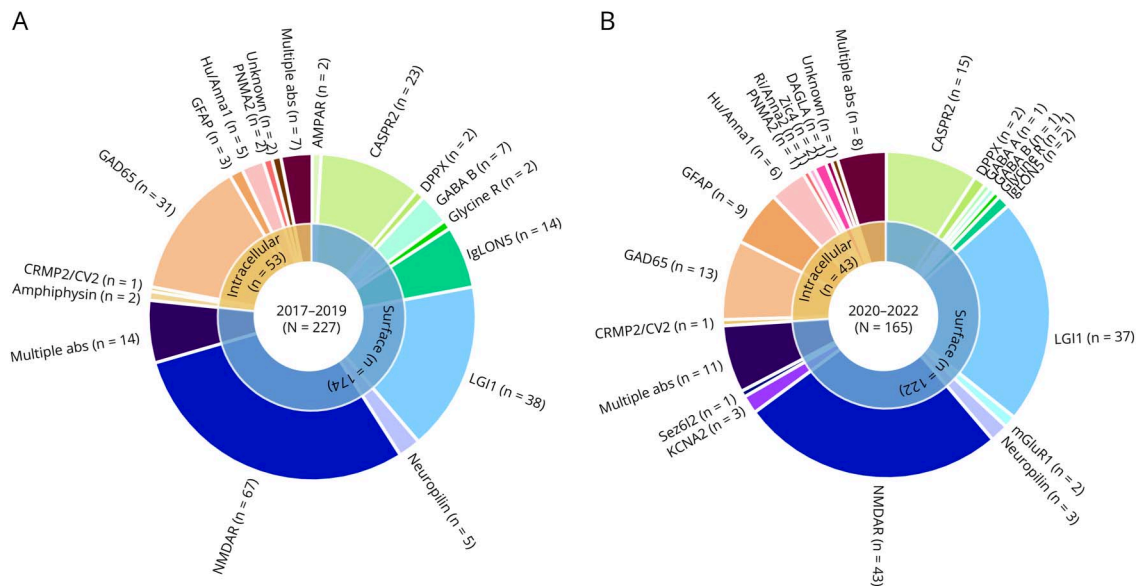
In our multicenter, hospital-based registry analysis, we found a decrease in the number of new AE cases during the COVID-19 pandemic period (2020–2022) compared with

a prepandemic control period, which was mainly caused by a reduction in AE cases associated with sAb.

The lack of evidence for an overall increase in new hospital cases of AE during the COVID-19 pandemic is in line with 2 laboratory-based antibody-frequency studies from Spain and the United Kingdom,<sup>9,10</sup> with clinical evidence provided by our study. Although viral triggers such as preceding CNS infections with herpes simplex virus are well known to trigger AE (in particular, NMDA-receptor encephalitis<sup>12</sup>), our findings collectively argue against the hypothesis that SARS-CoV-2 infection and SARS-CoV-2 vaccination, which was extensively performed during the pandemic, are potent triggers for the development of clinically relevant autoimmune CNS disease associated with antineuronal antibodies.

We furthermore observed a reduction in new hospital cases of definitive AE with sAb during the pandemic period.

**Figure 3** Antibody Findings in the Prepandemic (2017–2019) and Pandemic (2020–2022) Periods



Donut pie chart illustrating the relative proportion of antibodies detected in 227 patients with autoimmune encephalitis during the prepandemic phase (A) and 165 patients with autoimmune encephalitis during the pandemic phase (B). If multiple antibodies were detected, the respective coexisting antibodies are listed in eTable 1.

Several factors might have contributed to this change. Because hospital and outpatient referrals were restricted during the pandemic, patients with a more chronic disease course and milder disease symptoms might have been underrecognized because of more local patient management, leading to a selection or referral bias. Of interest, the number of patients with IgLON5 disease, who more often show a slow disease onset and chronic disease course, decreased during the pandemic (14/174, 8% vs 2/122, 1%). This might also be a possible explanation for the findings of the laboratory-based study on autoantibody prevalence in the United Kingdom, which reported a decreased detection rate of LGI1 antibodies during the national lockdown in 2020. It is possible that patients with more subtle presentations, such as only faciobrachial dystonic seizures, were not tested for LGI1 antibodies and thus underdiagnosed during this period. In the Spanish cohort of autoantibody-positive biosamples, the overall sAb prevalence was unchanged in a more extended observation time of two-year pandemic (2020–2021) compared with a prepandemic three-year period.

Although Spain, Germany, and the United Kingdom had different durations and intensities of lockdown periods, a cumulative “social distancing measure index” calculated by the University of Oxford did not show relevant differences up until the end of 2022.<sup>13</sup> Of interest, although the absolute number is lower, new hospital cases of encephalitis with iAb did not decrease in our study. It is important to emphasize that our analysis focused exclusively on patients with definitive antibody-positive AE. Seronegative and probable AE

cases were excluded owing to their clinical heterogeneity, which renders them less well characterized.

The potential selection bias due to fewer referrals of patients with AE to specialized centers might have led to the overall reduction in cases during the pandemic. However, we did not observe a shift toward higher maximal mRS values during the pandemic phase. It is also possible that during the challenges in the early phase of the pandemic, some patients with AE were missed and not entered in the GENERATE registry. However, because we observed no changes in the number of patients with AE with iAb within the observation periods, we assume no major change in patient recruitment in these well-established specialized study centers throughout the pandemic. In addition, we used the full three-year period (2020–2022) for the pandemic episode to reduce potential effects of disturbances in clinical procedures during the early pandemic phase.

Overall, our data do not support an increase in AE cases during the COVID-19 pandemic but instead may indicate a reduction in hospital incidence cases of AE with sAb. Further studies should test the occurrence of AE during the COVID-19 pandemic in other countries at a larger scale.

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J. Wickel: drafting/revision of the manuscript for content, including medical writing for content; major role in the

acquisition of data; study concept or design; analysis or interpretation of data. H.-Y. Chung: major role in the acquisition of data; study concept or design; analysis or interpretation of data. F.F. Konen: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. R. Rössling: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. A. Bertolini: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. A. Kraft: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. K. Siebenbrodt: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. S. Bittner: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. A. Juranek: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. M. Brokbals: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. S. Räuber: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. J. Klausewitz: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. L.K. Pfeffer: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. A. Scherag: drafting/revision of the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data. T. Menge: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. A. Finke: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. K. Doppler: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. C. Urbanek: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. C. Bien: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. T. Seifert-Held: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. F. Hoffmann: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. K.-P. Wandinger: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. S.C. Tauber: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. M. Süße: drafting/revision of the manuscript for content, including medical writing for content; major role in the acquisition of data. J. Lewerenz: drafting/revision of the manuscript for content, including medical writing for

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## Disclosure

J. Wickel reports speaker honoraria from Argenx and Alexion and travel compensations from Neuraxpharm. F.F. Konen received travel compensations from Alexion, Argenx, Merck, Takeda, and Novartis. A. Bertolini reports travel and congress fees from Octapharma and honoraria from Horizon for serving on advisory boards. S. Bittner received honoraria from Biogen Idec, Bristol-Myers Squibb, Merck Healthcare, Hexal, Novartis, Sanofi-Genzyme, Roche, and Teva. S. Räuber received travel grants from Merck Healthcare Germany GmbH, Bristol-Myers Squibb, and Alexion Pharmaceuticals. She served on a scientific advisory board of Merck Healthcare Germany GmbH and received speaker honoraria from Roche and Merck Healthcare Germany GmbH. Her research was supported by Novartis, Stiftung zur Förderung junger Neurowissenschaftler, and Else Kröner-Fresenius-Stiftung. L.K. Pfeffer received travel grants from Merck Healthcare Germany GmbH. T. Menge received personal fees from Biogen, BMS, Novartis, Teva, Roche, and Merck Serono, as well as nonfinancial support from Biogen, Merck Serono, and Roche. K. Doppler received honoraria from Grifols, Roche, Takeda, and Argenx. S.C. Tauber served on the

scientific advisory board for Roche and Merck and received travel and speaker honoraria from Novartis, Teva, Merck, Roche, Amgen, and Biogen. K. Rostasy received consultant fees for Operetta II study/Roche and honoraria from Horizon, UCB, and Euroimmun. K.-W. Sühs received honoraria for lectures or travel reimbursements from Biogen, Merck, Bavarian Nordic and Bristol-Myers Squibb, Mylan, Novartis, Roche, and Viartis, as well as research support from Bristol-Myers Squibb. T. Kämpfel received speaker honoraria and/or personal fees for advisory boards from Novartis Pharma, Merck, Roche Pharma, Alexion/AstraZeneca, Horizon, Chugai, and Biogen. F. Thaler reports speaker honoraria from Alexion Pharmaceuticals. F. Leyboldt received speaker honoraria from Grifols, Teva, Biogen, Bayer, Roche, Novartis, and Fresenius; received travel funding from Merck, Grifols, and Bayer; and served on advisory boards for Roche, Biogen, Argenx, and Alexion. C. Geis received travel support from Alexion and honoraria from Alexion, Argenx, Sobi, AstraZeneca, and Roche. None of the reported disclosures is related to this work. C. Bien, H.-Y. Chung, J. Klausewitz, A. Kraft, H. Prüss, A. Scherag, T. Seifert-Held, K. Siebenbrodt, C. Urbanek, and K.-P. Wandinger declare that they have no conflict of interest related to this study. Go to [Neurology.org/NN](https://www.neurology.org/NN) for full disclosures.

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Coinvestigators are listed online at [Neurology.org/NN](https://www.neurology.org/NN).

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