Data Article

Prognosis in autoimmune encephalitis: Database

James Broadley a,*, Udaya Seneviratne a,b, Paul Beech c,d, Katherine Buzzard e,f, Helmut Butzkueven a,e,f, Terence O’Brien a,f,g, Mastura Monifa f,g

a Department of Neuroscience, Monash University, Melbourne, Australia
b Department of Neuroscience, Monash Health, Melbourne, Australia
c Department of Radiology, Alfred Health, Melbourne, Australia
d Department of Radiology, Monash Health, Melbourne, Australia
e Department of Neurosciences, Eastern Health, Melbourne, Australia
f Department of Neurology, Melbourne Health, Melbourne, Australia
g Department of Neurology, Alfred Health, Melbourne, Australia

A R T I C L E   I N F O

Article history:
Received 20 October 2018
Received in revised form 1 November 2018
Accepted 2 November 2018
Available online 13 November 2018

A B S T R A C T

Autoimmune encephalitis is a rare and debilitating disease. An important question in clinical neurology is what factors may be correlated with outcomes in autoimmune encephalitis. There is observational data describing statistical analyses on such variables, but there are no review articles that collaborate and interpret this information. This data in brief article represents the data collection for such a review (Broadley et al., 2018).

Herein we summarize clinical information from 44 research articles, in particular pertaining to outcomes and prognostic variables.

© 2018 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

Specifications table

| Subject area | Neurology |
|--------------|-----------|
| More specific subject area | Autoimmune encephalitis |
| Type of data | Table |

DOI of original article: https://doi.org/10.1016/j.jaut.2018.10.014
* Corresponding author.
E-mail address: james.broadley@monash.edu (J. Broadley).
How data was acquired
Systematic database search

Data format
Filtered and analyzed

Experimental factors
Confirmed or suspected cases of autoimmune encephalitis with any antibody subtype

Experimental features
Search for relevant publications in major databases. Collection of data from included articles.

Data source location
Monash University, Wellington Rd, Clayton, VIC 3800, Australia

Data accessibility
Data included in article

Related research article
J. Broadley, U. Seneviratne, P. Beech, K. Buzzard, H. Butzkueven, T. O’Brien, M. Monif, Prognosticating autoimmune encephalitis: a systematic review, J. Autoimmun., (In Press 2018).

Value of the data
- This data provides a summary of relevant publications in the field, including antibody types, outcome measures, prognosis variables and the quality of this information
- Clinicians seeking to examine research articles for particular prognosis variables or in certain antibody subtypes can review this data to answer these questions easily
- This data provides summaries of demographics, typical symptomatology, rates of tumor association and routine investigation findings for key antibody types
- Clinicians can see rates of good and poor outcomes with respect to antibody subtype and the outcome measure of interest

1. Data

The data provided here is a summary of information in research articles describing prognosis in autoimmune encephalitis. This data includes entirely clinical information, with a focus on outcomes and prognosis variables. All causes of autoimmune encephalitis were included in the search, but most of the included articles described cases with antibodies directed to one of the cell surface antigens; NMDAR (N-methyl-D-aspartate receptor), VGKC (voltage-gated potassium channel) or GABAb (γ-aminobutyric acid receptor B). Other important abbreviations include MRI (magnetic resonance imaging), CSF (cerebrospinal fluid) and EEG (electroencephalogram). A glossary of terms is provided to aid the interpretation of the following data tables.

Table S1 provides a detailed description of all the research articles that met the inclusion criteria. For each publication we document the type, number of patients, antibody profiles, clinical syndromes, demographics, routine investigation findings, outcome measures, prognosis variables analyzed and the results of this analysis. There is also an objective assessment of quality of each publication, as listed in the final column.

Tables 1–4 summarize clinical information in the research articles divided into each major antibody group. This included cases of anti-NMDAR encephalitis (Table 1), anti-VGKC encephalitis (Table 2) and anti-GABAb encephalitis (Table 2), as well as cases with intracellular antibodies (Table 3). The information listed includes the rates of cardinal symptoms (cognitive impairment, seizure and psychosis), rates of underlying tumor diagnoses, percentage receiving immunotherapy, duration of follow-up and percentage to have good clinical outcomes for each article. Two publications were designed to examine cases without any of the aforementioned antibodies and therefore are only included in Supplementary Table S1 [37,45].
Table 1
Summary of anti-NMDAR encephalitis cases.

| Paper | Patients | % Seizure | % Cognitive change | % Psychosis | % Tumour | %Received IT | No followed | Median follow-up (months) | %Good outcome |
|-------|----------|-----------|-------------------|-------------|----------|-------------|-------------|--------------------------|---------------|
| Byun et al. [9] | 11 | 54.5 | 54.5 | 72.7 | 12.5 | 72.7 | 11 | 3 | 63.6 |
| Byun et al. [10] | 17 | 100 | 52.9 | 76.5 | 0 | 100 | 17 | 6 | 76.5 |
| Chi et al. [12] | 96 | 80.2 | NR | 90.6 | 13.5 | 95.8 | 96 | 24.5 | 88.5 |
| Constantinescu et al. [13] | 4 | 100 | 75 | 75 | 25 | 100 | 4 | 12 | 75 |
| Dalmau et al. [15] | 100 | 76 | 23 | 77 | 59.2 | 92 | 100 | 17 | 75 |
| de Montmollin [16] | 77 | 81.6 | NR | NR | 47.4 | 97.4 | 76 | 6 | 56.6 |
| Duan et al. [17] | 28 | NR | NR | NR | 25 | 96.4 | 28 | 6 | 89.3 |
| Dubey (Journal of Neuroimmunology) [18] | 16 | 75 | NR | NR | 12.5 | Unclear | 16 | NA | 62.5 |
| Dubey et al. [19] | 7 | 100 | NR | NR | 28.6 | 100 | 7 | Unclear | 42.9 |
| Dubey et al. [20] | 6 | 100 | NR | NR | NR | Unclear | NA | NA | Unclear |
| Finke et al. [21] | 40 | 77.5 | NR | NR | NR | Unclear | NA | NA | Unclear |
| Gablondo et al. [24] | 25 | NR | NR | NR | 20 | 84 | 25 | 20 | 80 |
| Gresa-Arribas et al. [25] | 45 | NR | NR | NR | 38.8 | Unclear | NA | 26 | Unclear |
| Harutyunyan et al. [26] | 3 | NR | NR | NR | 0 | NR | Unclear | NA | NA | Unclear |
| Iizuka et al. [27] | 15 | 93.3 | NR | 80 | 33.3 | 86.7 | 15 | 68 | 86.7 |
| Irani et al. [28] | 44 | 81.8 | 90.9 | 77.3 | 20.5 | 79.5 | 44 | 16 | 70.4 |
| Jang et al [31] | 15 | NR | NR | NR | NR | 100 | 15 | 1 | 46.7 |
| Lee et al 2016 (Neurology) [33] | 27 | NR | NR | NR | NR | 18.5 | 100 | NA | NA | Unclear |
| Lee et al 2016 (Neurotherapeutics) [34] | 26 | NR | NR | NR | NR | 23.1 | 100 | NA | NA | Unclear |
| Leyboldt et al. [35] | 167 | NR | NR | NR | NR | 100^ | 137 | 8 | 58.4 |
| Lim et al. [36] | 32 | 50 | 34.4 | 68.6 | 27.3 | Incomplete | 21 | 4 | 63.6 |
| Quek et al. [39] | 1 | 100 | 0 | 0 | 0 | 100 | 1 | 5 | 100 |
| Titulaer et al. (Lancet Neurology) [42] | 577 | NR | NR | NR | 39.5 | 92.2^ | 501 | 6 | 78.6 |
| Titulaer et al. (Neurology) [43] | 31 | 48.4 | 100 | NR | 22.6 | Incomplete | 29 | 24 | 72.4 |
| Wang et al. [46] | 43 | 86 | NR | 95.3 | 2.3 | 83.7 | 38 | 4 | 71.1 |
| Wang et al. [47] | 51 | 84.3 | 31.4 | 90.2 | 7.8 | 88.2 | 51 | 12 | 80 |
| Zhang et al. [48] | 62 | 74.2 | 8.1 | 43.5 | 8.1 | 100 | 62 | 6 | 87.1 |
| Mean/cumulative | 1566 | 81.27 | 47.02 | 70.56 | 22.07 | 93.13 | 1294 | 13.76 | 72.61 |

^ Only reported immunotherapy use in the patients that had follow-up.
Table 2
Summary of anti-VGKC encephalitis cases.

| Paper | Patients | % Seizure | % Cognitive change | % Psychosis | % Tumour | % received IT | No followed | Median follow-up (months) | %Good outcome |
|-------|----------|-----------|--------------------|-------------|----------|--------------|-------------|---------------------------|---------------|
| Arino et al. [5] | 76 | 88.2 | 100 | 30.3 | 6.6 | 100 | 48 | 24 | 70.8 |
| Aurangzeb et al. [6] | 16 | 100 | 93.8 | NR | NR | Unclear | 16 | 24 | 81.3 |
| Bataller et al. [7] | 5 | NR | NR | NR | 20 | 80 | 5 | Unclear | 100 |
| Butler et al. [8] | 19 | 73.7 | 100 | 31.6 | NR | 100 | 17 | Unclear | 70.6 |
| Byun et al. [10] | 17 | 100 | 52.9 | 47.1 | 0 | 100 | 17 | 6 | 82.4 |
| Constantinescu et al. [13] | 1 | 100 | 0 | 0 | 0 | 100 | 1 | 12 | 100 |
| Dubey et al. (Journal of Neuroimmunology) [18] | 9 | 88.9 | NR | NR | 44.4 | Unclear | 9 | Unclear | 44.4 |
| Dubey et al. (Seizure) [19] | 8 | 100 | NR | NR | 37.5 | 100 | 8 | Unclear | 62.5 |
| Dubey et al. [20] | 18 | 100 | NR | NR | NR | Unclear | NA | NA | Unclear |
| Finke et al. [22] | 30 | 93.3 | 100 | NR | 10 | 96.7 | 30 | 23.3 | 80 |
| Flanagan et al. [23] | 11 | 54.5 | 100 | 36.4 | NR | 100 | 11 | 22 | 90.9 |
| Harutyunyan et al. [26] | 6 | NR | NR | NR | NR | Unclear | NA | NA | Unclear |
| Irani et al. [29] | 26 | 34.5 | NR | NR | 41.4 | Unclear | 26 | unclear | 65.4 |
| Irani et al. [30] | 10 | 100 | 80 | NR | 10 | 100 | 10 | 18 | 100 |
| Jang et al. [31] | 15 | NR | NR | NR | 100 | NA | NA | NA | Unclear |
| Lee et al. (Neurology) [33] | 3 | NR | NR | NR | 100 | NA | NA | NA | Unclear |
| Lee et al. (Neurotherapeutics) [34] | 3 | NR | NR | NR | 100 | NA | NA | NA | Unclear |
| Malter et al. [38] | 10 | 100 | 90 | NR | 0 | 100 | 9 | 17 | 100 |
| Quek et al. [39] | 18 | 100 | 61.1 | 16.7 | 16.7 | 88.9 | 18 | 100 | 100 |
| Shin et al. [40] | 14 | 100 | 85.7 | 0 | 7.1 | 100 | 12 | 4.5 | 91.7 |
| Thompson et al. [41] | 103 | 100 | 78.6 | 12.6 | 7.8 | 95.1 | NA | NA | Unclear |
| Toledano et al. [44] | 12 | 100 | NR | NR | NR | 100 | 12 | 22.5 | 100 |
| Mean/cumulative | 430 | 90.18 | 78.51 | 21.84 | 13.73 | 97.69 | 249 | 17.33 | 83.75 |
Table 3
Summary of anti-GABAb encephalitis cases.

| Paper | Patients | % Seizure | % Cognitive change | % Psychosis | % Tumour | % Received IT | No Followed | Median follow-up (months) | % Good outcome |
|-------|----------|-----------|--------------------|-------------|----------|---------------|-------------|--------------------------|---------------|
| Byun et al. [10] | 3 | 100 | NR | NR | 66.7 | 100 | 3 | 6 | 66.7 |
| Chen et al. [11] | 11 | 100 | 90.9 | 27.3 | 27.3 | 100 | 11 | 8.6 | 63.6 |
| Constantinescu et al. [13] | 1 | 100 | 0 | 100 | 0 | 100 | 1 | 12 | 100 |
| Dubey et al. (Journal of Neuroimmunology) [18] | 3 | 66.6 | NR | NR | 33.3 | Unclear | 3 | Unclear | 66.7 |
| Dubey et al. (Seizure) [19] | 2 | 100 | NR | NR | 50 | 100 | 2 | Unclear | 50 |
| Harutyunyan et al. [26] | 2 | NR | NR | NR | Unclear | NA | NA | NA | Unclear |
| Jang et al. [31] | 1 | NR | NR | NR | 100 | NA | NA | NA | Unclear |
| Lancaster et al. [32] | 15 | 100 | 100 | 26.7 | 46.7 | 73.3 | 14 | 6 | 57.1 |
| Mean/cumulative | 38 | 94.43 | 63.63 | 51.33 | 37.33 | 95.55 | 34 | 8.15 | 67.35 |
### Table 4
Summary of encephalitis cases with intracellular antibodies.

| Paper | Patients | Antibody types | % Seizure | % Cognitive change | % Psychosis | % Tumour | % Received IT | No followed | Median follow-up (months) | %Good outcome |
|-------|----------|----------------|-----------|--------------------|-------------|----------|---------------|-------------|---------------------------|--------------|
| Bataller et al. [7] | 14 | 7 Hu; 6 Ma2; 1 atypical | NR | NR | NR | 100 | 100 | 12 | Unclear | 25 |
| Byun et al. [10] | 4 | 2 Ma2 (also had Ta); 1 Yo; 1 amphiphysin | 100 | NR | NR | 50 | 100 | 4 | 6 | 75 |
| Constantinescu et al. [13] | 2 | 2 Ma2 | 100 | 50 | 50 | 0 | 100 | 2 | 12 | 100 |
| Dalmau et al. [14] | 38 | 38 Ma2 (15 also had Ma1) | 31.6 | 68.4 | NR | 89.5 | 50 | 33 | 30 | 33.3 |
| Dubey et al. (Journal of Neuroimmunology) [18] | 1 | 1 Hu | 0 | NR | NR | unclear | 1 | Unclear | 0 |
| Dubey et al. [20] | 4 | 2 Hu; 1 CV2; 1 Ri | 100 | NR | NR | NR | Unclear | NA | NA | Unclear |
| Flanagan et al. [23] | 2 | 1 Hu; 1 amphiphysin | 50 | 100 | 50 | 100 | 100 | 2 | 34 | 50 |
| Harutyunyan et al. [26] | 4 | 1 Hu; 1 Yo; 1 Ma2 (also Ma1); 1 CV2 | NR | NR | NR | NR | Unclear | NA | NA | Unclear |
| Lee et al. (Neurology) [33] | 15 | 4 Ma2 (also Ta); 2 Yo; 2 Hu; 7 amphiphysin | 40 | NR | NR | 20 | 100 | 15 | Unclear | 60 |
| Lee et al. (Neurotherapeutics) [34] | 2 | 2 amphiphysin | NR | NR | NR | 0 | 100 | NA | NA | Unclear |
| Quek et al. [39] | 3 | 2 CV2; 1 Ma2 (also had Ma1) | 100 | 66.7 | 0 | 33.3 | 100 | 3 | Unclear | 66.7 |
| Toledano et al. [44] | 1 | 1 Ma2 (also had Ma1) | 100 | NR | NR | NR | 100 | 1 | 32 | 0 |
| Mean/cumulative | 90 | | 69.07 | 71.28 | 33.33 | 43.64 | 94.44 | 73 | 22.8 | 45.56 |
2. Experimental design, materials and methods

Relevant publications were identified by searching abstracts in MEDLINE, Embase, PsychInfo and PubMed databases from their inception to 30/04/2018. Search terms including autoimmune encephalitis, autoimmune antibody subtypes, outcome and prognosis were combined with Boolean operators (see Table 1 in Ref [1]). Furthermore, the reference lists of included publications were also examined to identify additional articles undetected in the initial search.

Research articles were eligible for this review if they were original research on patients diagnosed with autoimmune encephalitis that provided a statistical analysis of factors that correlated with the patient outcome. Publications were included based on cases with features of encephalitis that were suspected or confirmed to have an autoimmune cause. Publication focusing on other antibody associated CNS or non-CNS syndromes, such as paraneoplastic cerebellar degeneration, stiff person syndrome, isolated myelitis or paraneoplastic neuropathy were excluded. In addition, articles that reported such cases in a wider cohort of patients with encephalomyelitis, where the statistics could not be isolated for cases of encephalitis only, were also excluded. Inclusion criteria were English language publications and the availability of full text. Animal publication, grey literature and case series reporting less than 10 patients were excluded. Data performed solely in children were also excluded. Finally, articles where autoimmune encephalitis was a subset of a larger publication on encephalitis due to multiple aetiologies were excluded.

Each publication underwent a detailed review, during which the following details were extracted: number of patients, antibody subset, clinical syndrome, age, sex, abnormal investigation findings, outcome measures, factors tested for outcome correlation and publication results (Table S1). Papers that tested early magnetic resonance imaging (MRI) findings and/or cerebrospinal fluid (CSF) characteristics as possible markers for prognosis were noted. The CSF parameters considered applicable were those identified by routine testing, such as protein, glucose, white cell count, or differential cell counts. The MRI abnormalities recorded were those likely due to encephalitis, or any MRI abnormality if these details were not specified. Articles listed as having “Multiple” antibodies include both seropositive and seronegative cases unless stated otherwise. All ages are reported in years. Average age is given as what was reported in paper (mean, median or mode). Ranges were also as reported in paper and given to nearest whole number (actual range, upper/lower quartiles or standard deviation). Where both were included in the data, only the actual range was recorded. Abnormal CSF is reported based on the presence of either elevated protein or leukocytosis on initial CSF analysis. Abnormal MRI is reported based on the presence of one or more of T2/FLAIR changes, contrast enhancement, or leptomeningeal enhancement on initial MRI. Abnormal EEG is reported based on the presence of epileptiform discharges, inter-ictal spikes, focal slowing or extreme delta brush pattern on EEG in initial admission. Numbers in brackets in the EEG column are inclusive of patients with any electrographic abnormality (including generalised slowing). In articles that did not include this level of detail, we took their definition of abnormal CSF/MRI/EEG. Percentages are taken in relation to the reported numbers of participants to have had each test. In circumstances where both values (eg elevated CSF protein and white cell count) were reported but not the number of participants that had one or the other, we took the highest single value. Relationships are based on highest statistical test performed; multivariate regression analysis if available, univariate analysis if not. Therefore, significant relationship on univariate analysis, subsequently disproven on multivariate analysis is not listed here as a correlation.

For the most common antibody subtypes details were obtained on seizure frequency, cognitive impairment, psychosis, underlying neoplasia, immunotherapy, follow-up timeframes and reported outcomes (Tables 1–4). Reported rates of immunotherapy usage were in the original cohort unless otherwise stated. In papers where there was loss to follow-up, this data describes outcomes in the most complete group of patients, even if that follow-up was significantly shorter than the longest follow-up. This was meant to reduce the error in the collection and reporting of data. This data used the outcomes that were defined as good or favorable by the authors of each research article, or where patients were left with no or only mild deficits when no definition was given. For articles with an adequate description of relevant outcome data, this data includes the patients’ median follow-up timeframe, the numbers of patients with follow-up information and the proportion to have a positive
outcome. Data was excluded if any information was unclear or incomplete, in particular noting if it was unclear for the antibody subset of interest. A breakdown of constituent antibodies was documented in articles that included intracellular autoantibodies. Using the information obtained from the articles, this dataset includes the calculated average rates of seizure presentations, cognitive impairment, tumor diagnoses and immunotherapy usage as well as the average rates of favorable outcomes for the major antibody groups. This information describes seizure of any type, including faciobrachial dystonic seizures. Symptoms listed were based on initial symptoms only, not symptoms at relapse. Notably some publications report cognition or psychiatric features together; this data was excluded. Percentage tumor reflects the proportion of diagnosis from patients that were imaged for tumor. The last row in each table represents mean values, except in columns of patient numbers which are cumulative.

Included articles were reviewed independently by two authors (JB & US), and classified based on the publication design. Where discrepancy was found, consensus agreement was reached. The articles were objectively assessed for quality, using the Newcastle-Ottawa Scale for cohort and case-control studies[2], an adapted version of the Newcastle-Ottawa Scale for cross-sectional studies[3], and the quality assessment tool for case series proposed by Moga et al[4]. Each research article was classified as having good, fair or poor quality based on the scoring of these quality assessment tools (see Table 2 in Ref [1]).

**Transparency document. Supporting information**

Transparency data associated with this article can be found in the online version at https://doi.org/10.1016/j.dib.2018.11.020.

**Appendix A. Supporting information**

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.dib.2018.11.020.

**References**

[1] J. Broadley, U. Seneviratne, P. Beech, K. Buzzard, H. Butzkueven, T. O’Brien, M. Monif, Prognosticating autoimmune encephalitis: a systematic review, J. Autoimmun. (2018) (in press).

[2] S.B. Wells, G. O’Connell, D. Peterson, J. Welch, V. Losos, M. Tugwell, P. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses Accessed at http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp (July 2018).

[3] R. Herzog, M.J. Alvarez-Pasquin, C. Diaz, J.L. Del Barrio, J.M. Estrada, A. Gil, Are healthcare workers’ intentions to vaccinate related to their knowledge, beliefs and attitudes? A systematic review, BMC Public Health 13 (2013) 154.

[4] G.B. Moga, C. Schopflocher, D. Harstall C, Development of a Quality Appraisal Tool for Case Series Studies using a Modified Delphi Technique, 2012. Accessed at https://www.ihe.ca/publications/development-of-a-quality-appraisal-tool-for-case-series-studies-using-a-modified-delphi-technique(July 2018).

[5] H. Arino, T. Ar mangue, M. Petit-Pedrol, L. Sabater, E. Martinez-Hernandez, M. Hara, E. Lancaster, A. Saiz, J. Dalmau, F. Graus, Anti-LGI1-associated cognitive impairment: presentation and long-term outcome, Neurology 87 (2016) 759–765.

[6] S. Aurangzeb, M. Symmonds, R.K. Knight, R. Kennett, T. Wehner, S.R. Irani, LGI1-antibody encephalitis is characterised by frequent, multifocal clinical and subclinical seizures, Seizure 50 (2017) 14–17.

[7] L. Bataller, K.A. Kleopa, G.F. Wu, J.E. Rossi, M.R. Rosenfeld, J. Dalmau, Autoimmune limbic encephalitis in 39 patients: immunophenotypes and outcomes, J. Neurol. Neurosurg. Psychiatry 78 (2007) 381–385.

[8] C.R. Butler, T.D. Miller, M.S. Kaur, I.W. Baker, G.D. Boothroyd, N.A. Illman, C.R. Rosenthal, A. Vincent, C.J. Buckley, Persistent anterograde amnesia following limbic encephalitis associated with antibodies to the voltage-gated potassium channel complex, J. Neurol. Neurosurg. Psychiatry 85 (2014) 387–391.

[9] J.I. Byun, S.T. Lee, J. Moon, K.H. Jung, J.W. Shin, J.S. Sunwoo, J.A. Lim, Y.W. Shin, T.J. Kim, K.J. Lee, K.I. Park, K.Y. Jung, S.K. Lee, K. Chu, Cardiac sympathetic dysfunction in anti-NMDA receptor encephalitis, Auton. Neurosci. 193 (2015) 142–146.

[10] J.I. Byun, S.T. Lee, K.H. Jung, J.S. Sunwoo, J. Moon, J.A. Lim, D.Y. Lee, Y.W. Shin, T.J. Kim, K.J. Lee, W.J. Lee, H.S. Lee, J. Jun, D. Y. Kim, M.Y. Kim, H. Kim, H.Y. Kim, H.I. Suh, Y. Lee, D.W. Kim, J.H. Jeong, W.C. Choi, D.W. Bae, J.W. Shin, D. Jeon, K.I. Park, K. Y. Jung, K. Chu, S.K. Lee, Effect of immunotherapy on seizure outcome in patients with autoimmune encephalitis: a prospective observational registry study, PLoS One 11 (2016) e0146455.
[11] X. Chen, F. Liu, J.M. Li, X.Q. Xie, Q. Wang, D. Zhou, H. Shang, Encephalitis with antibodies against the GABAB receptor: seizures as the most common presentation at admission, Neuro. Res. 39 (2017) 973–980.

[12] X. Chi, W. Wang, C. Huang, M. Wu, L. Zhang, J. Li, D. Zhou, Risk factors for mortality in patients with anti-NMDA receptor encephalitis, Acta Neurol. Scand. 136 (2017) 298–304.

[13] R. Constantinescu, D. Krysl, F. Bergquist, K. Andre, C. Malmestrom, F. Asztely, M. Axelsson, E.B. Menachem, K. Blennow, L. Rosengren, H. Zetterberg, Cerebrospinal fluid markers of neuronal and glial cell damage to monitor disease activity and predict long-term outcome in patients with autoimmune encephalitis, J. Neurolm. 23 (2016) 796–806.

[14] J. Dalmau, F. Graus, A. Villarejo, J.B. Posner, D. Blumenthal, B. Thiessen, A. Saiz, P. Meneses, M.R. Rosenfeld, Clinical analysis of anti-Ma2-associated encephalitis, Brain 127 (2004) 1831–1844.

[15] J. Dalmau, A.J. Gleichman, E.G. Hughes, J.E. Rossi, X. Peng, M. Lai, S.K. Dessnain, M.R. Rosenfeld, R. Balice-Gordon, D.R. Lynch, Anti-NMDA-receptor encephalitis: case series and analysis of the effects of antibodies, Lancet Neurol. 7 (2008) 1091–1098.

[16] E. de Montmollin, S. Demonet, N. Brule, M. Conrad, F. Dailler, N. Llolera, J.C. Naveilou, C. Schwebel, M. Alves, M. Cour, N. Engrand, J.M. Tommelier, E. Maury, S. Ruckly, G. Picard, V. Rogemond, E. Magalhaes, T. Sharshar, J.F. Timsit, J. Homorat, R. Sonveille, E.S.Gd dagger, Anti-N-methyl-d-aspartate receptor encephalitis in adult patients requiring intensive care, Am. J. Respir. Crit. Care Med. 195 (2017) 491–498.

[17] B.C. Duan, W.C. Weng, K.L. Lin, L.C. Wong, S.T. Li, M.H. Hsu, J.J. Lin, P.C. Fan, M.I. Lin, N.C. Chiu, Y.C. Lin, H.S. Wang, K.L. Hung, W.T. Lee, Variations of movement disorders in anti-N-methyl-D-aspartate receptor encephalitis: a nationwide study in Taiwan, Medicine 95 (2016) e4365.

[18] D. Dubey, A. Sawhney, B. Greenberg, A. Lowden, W. Warnack, P. Khemani, O. Stuve, S. Verno, The spectrum of autoimmune encephalopathies, J. Immunol. 287 (2015) 93–97.

[19] D. Dubey, N. Samudra, P. Gupta, M. Agostini, K. Ding, P.C. Van Ness, S. Verno, R. Hays, Retrospective case series of the clinical features, management and outcomes of patients with autoimmune encephalitis, Seizure 29 (2015) 143–147.

[20] D. Dubey, J. Singh, J.W. Britton, S.J. Pittock, E.P. Flanagan, V.A. Lennon, J.M. Tillema, E. Wirrell, C. Shin, E. So, G.D. Cascino, D. Wingerchuk, M.T. Hoerth, J.J. Shih, K.C. Nickels, A. McKeon, Autoimmune epilepsy, Epilepsia 58 (2017) 1181–1189.

[21] C. Finke, U.A. Kopp, A. Pakjert, J.R. Behrens, F. Leyboldt, J.T. Wuerfel, C.J. Ploner, H. Pruss, F. Paul, Structural hippocampal damage following anti-N-methyl-D-aspartate receptor encephalitis, Biol. Psychiatry 79 (2016) 727–734.

[22] C. Finke, H. Pruss, J. Heine, S. Reuter, U.A. Kopp, F. Wegner, F. Then Bergh, S. Koch, O. Jansen, T. Munte, G. Deuschl, K. Kurepet, W. Stocker, K.F. Wandinger, F. Paul, T. Bartsch, Evaluation of cognitive deficits and structural hippocampal damage with leucine-rich, glioma-inactivated 1 antibodies, JAMA Neurol. 74 (2017) 50–59.

[23] E.P. Flanagan, A. McKeon, V.A. Lennon, B.F. Boeve, M.R. Trenerry, K.M. Tan, D.A. Drubach, K.A. Josephs, J.W. Britton, J. W. Lee, S.T. Lee, J.I. Byun, J.S. Sunwoo, T.J. Kim, J.A. Lim, J. Moon, H.S. Lee, Y.W. Shin, K.J. Lee, K.Y. Chung, S.K. Lee, High albumin level is a predictor of favorable outcome in patients with paraneoplastic encephalitis, JAMA Neurol. 13 (2014) 167–177.

[24] G. Harutyunyan, L. Hauer, M.W. Dunser, T. Moser, S. Piaggi, M. Leitinger, H.F. Novak, W. Aichhorn, E. Trinka, J. Sellner, Risk factors for intensive care unit admission in patients with autoimmune encephalitis, Front. Immunol. 8 (2017) 835.

[25] T. Izuka, J. Kaneko, N. Tominaga, H. Someko, M. Nakamura, D. Ishima, E. Kitamura, R. Masuda, E. Oguni, T. Yanagisawa, N. Kanazawa, J. Dalmou, K. Nishiyama, Association of progressive cerebellar atrophy with long-term outcome in patients with anti-n-methyl-D-aspartate receptor encephalitis, JAMA Neurol. 73 (2016) 706–713.

[26] S.R. Irani, K. Bera, P. Waters, L. Zuliani, S. Koh, M.S. Zandi, M.A. Friese, I. Galea, D.M. Bullmull, D. Reesom, B. Lang, C. Finke, L. Rosengren, H. Zetterberg, Cerebrospinal fluid markers of neuronal and glial cell damage in patients with autoimmune encephalitis, Neurol. 10 (2014) 157.

[27] S.R. Irani, P. Pettingill, K.A. Kleopa, N. Schiza, P. Waters, C. Mazia, L. Zuliani, O. Watanabe, B. Lang, C. Buckley, A. Vincent, Morvan syndrome: clinical and serological observations in 29 cases, Ann. Neurol. 72 (2012) 241–255.

[28] S.R. Irani, C.J. Stagg, J.M. Schott, C.R. Rosenthal, S.A. Schneider, P. Pettingill, P. Pettingill, P. Waters, A. Thomas, N.L. Voets, M. J. Cardoso, D.M. Cash, E.N. Manning, B. Lang, S.J. Smith, A. Vincent, M.R. Johnson, Faciobrachial dystonic seizures: the influence of immunotherapy on seizure control and prevention of cognitive impairment in a broadening phenotype, Brain 136 (2013) 3151–3162.

[29] V. Jang, S.T. Lee, T.J. Kim, J.S. Jun, J. Moon, K.H. Jung, K.I. Park, K. Chu, S.K. Lee, High albumin level is a predictor of favorable response to immunotherapy in autoimmune encephalitis, Sci. Rep. 8 (2018) 1012.

[30] D. Lancaster, M. Lai, X. Peng, E. Hughes, R. Constantinescu, J. Raizer, D. Friedman, M.B. Skeen, W. Grisold, A. Kimura, Y. Jang, S.T. Lee, T.J. Kim, J.S. Jun, J. Moon, K.I. Park, K. Chu, Anti-N-methyl-d-aspartate receptor encephalitis: a retrospective cohort study, Neurotherapeutics 13 (2016) 1647–1658.

[31] W.T. Lee, Variations of movement disorders in anti-N-methyl-D-aspartate receptor encephalitis: a nationwide study in Taiwan, Medicine 95 (2016) e4365.

[32] L. Rosengren, H. Zetterberg, Cerebrospinal fluid markers of neuronal and glial cell damage to monitor disease activity and predict long-term outcome in patients with autoimmune encephalitis, J. Neurol. 23 (2016) 796–806.

[33] S.R. Irani, P. Pettingill, K.A. Kleopa, N. Schiza, P. Waters, C. Mazia, L. Zuliani, O. Watanabe, B. Lang, C. Buckley, A. Vincent, Morvan syndrome: clinical and serological observations in 29 cases, Ann. Neurol. 72 (2012) 241–255.

[34] S.R. Irani, C.J. Stagg, J.M. Schott, C.R. Rosenthal, S.A. Schneider, P. Pettingill, P. Pettingill, P. Waters, A. Thomas, N.L. Voets, M. J. Cardoso, D.M. Cash, E.N. Manning, B. Lang, S.J. Smith, A. Vincent, M.R. Johnson, Faciobrachial dystonic seizures: the influence of immunotherapy on seizure control and prevention of cognitive impairment in a broadening phenotype, Brain 136 (2013) 3151–3162.

[35] V. Jang, S.T. Lee, T.J. Kim, J.S. Jun, J. Moon, K.H. Jung, K.I. Park, K. Chu, S.K. Lee, High albumin level is a predictor of favorable response to immunotherapy in autoimmune encephalitis, Sci. Rep. 8 (2018) 1012.

[36] D. Lancaster, M. Lai, X. Peng, E. Hughes, R. Constantinescu, J. Raizer, D. Friedman, M.B. Skeen, W. Grisold, A. Kimura, Y. Jang, S.T. Lee, T.J. Kim, J.S. Jun, J. Moon, K.I. Park, K. Chu, Anti-N-methyl-d-aspartate receptor encephalitis: a retrospective cohort study, Neurotherapeutics 13 (2016) 1647–1658.

[37] W.T. Lee, Variations of movement disorders in anti-N-methyl-D-aspartate receptor encephalitis: a nationwide study in Taiwan, Medicine 95 (2016) e4365.
[37] S. Litmeier, H. Pruss, E. Witsch, J. Witsch, Initial serum thyroid peroxidase antibodies and long-term outcomes in SREAT, Acta Neurol. Scand. 134 (2016) 452–457.

[38] M.P. Malter, C. Helmstaedter, H. Urbach, A. Vincent, C.G. Bien, Antibodies to glutamic acid decarboxylase define a form of limbic encephalitis, Ann. Neurol. 67 (2010) 470–478.

[39] A.M. Quek, J.W. Britton, A. McKeon, E. So, V.A. Lennon, C. Shin, C. Klein, R.E. Watson Jr., A.L. Kotsenas, T.D. Lagerlund, G. D. Cascino, G.A. Worrell, E.C. Wirrell, K.C. Nickels, A.J. Aksamit, K.H. Noe, S.J. Pittock, Autoimmune encephalitis: clinical characteristics and response to immunotherapy, Arch. Neurol. 69 (2012) 582–593.

[40] Y.W. Shin, S.T. Lee, J.W. Shin, J. Moon, J.A. Lim, J.J. Byun, T.J. Kim, K.J. Lee, Y.S. Kim, K.I. Park, K.H. Jung, S.K. Lee, K. Chu, VGKC-complex/LGI1-antibody encephalitis: clinical manifestations and response to immunotherapy, J. Neuroimmunol. 265 (2013) 75–81.

[41] J. Thompson, M. Bi, A.G. Murchison, M. Makuch, C.G. Bien, K. Chu, P. Farooque, J.M. Gelfand, M.D. Geschwind, L.J. Hirsch, E. Somerville, B. Lang, A. Vincent, M.J. Leite, K.H. Noe, S.J. Pittock, Treatment and prognostic factors for long-term outcome in patients with faciobrachial dystonic seizures, Brain 141 (2018) 348–356.

[42] M.J. Titulaer, L. McCracken, I. Gabilondo, T. Armand, C. Glaser, T. Iizuka, L.S. Honig, S.M. Benseler, I. Kawachi, E. Martinez-Hernandez, E. Aguilar, N. Gresa-Arribas, N. Ryan-Florance, A. Torrents, A. Saiz, M.R. Rosenfeld, R. Balice-Gordon, F. Graus, J. Dalmau, Late-onset anti-NMDA receptor encephalitis: clinical characteristics and response to immunotherapy, Acta Neurol. Scand. 135 (2017) 134–141.

[43] M.J. Titulaer, L. McCracken, I. Gabilondo, T. Iizuka, I. Kawachi, I. Bataller, A. Torrens, M.R. Rosenfeld, R. Balice-Gordon, F. Graus, J. Dalmau, Late-onset anti-NMDA receptor encephalitis, Neurology 81 (2013) 1058–1063.

[44] M. Toledano, J.W. Britton, A. McKeon, C. Shin, V.A. Lennon, A.M. Quek, E. So, G.A. Worrell, G.D. Cascino, C.J. Klein, T. D. Lagerlund, E.C. Wirrell, K.C. Nickels, S.J. Pittock, Utility of an immunotherapy trial in evaluating patients with presumed autoimmune epilepsy, Neurology 82 (2014) 1578–1586.

[45] B. von Rhein, J. Wagner, G. Widman, M.P. Malter, C.E. Elger, C. Helmstaedter, Suspected antibody negative autoimmune limbic encephalitis: outcome of immunotherapy, Acta Neurol. Scand. 135 (2017) 134–141.

[46] R. Wang, H.Z. Guan, H.T. Ren, W. Wang, Z. Hong, D. Zhou, CSF findings in patients with anti-N-methyl-D-aspartate receptor-encephalitis, Seizure 29 (2015) 137–142.

[47] W. Wang, J.M. Li, F.Y. Hu, R. Wang, Z. Hong, L. He, D. Zhou, Anti-NMDA receptor encephalitis: clinical characteristics, predictors of outcome and the knowledge gap in southwest China, Eur. J. Neurol. 23 (2016) 621–629.

[48] Y. Zhang, G. Liu, M.D. Jiang, L.P. Li, Y.Y. Su, Analysis of electroencephalogram characteristics of anti-NMDA receptor encephalitis patients in China, Clin. Neurophysiol. 128 (2017) 1227–1233.

Glossary of terms

AED: anti-epileptic drug,
AMPA: α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid,
CASPR2: contractin-associated protein-like 2,
CPS: cognitive performance score,
CRP: C-reactive protein,
CS: corticosteroid,
CSF: cerebrospinal fluid,
CT: computed tomography,
EEG: electroencephalogram,
FBDS: faciobrachial dystonic seizure,
GABAB: γ-aminobutyric acid receptor B,
GAD: glutamic acid decarboxylase,
GCS: Glasgow coma scale,
GFR: glomerular filtration rate,
ICU: intensive care unit,
IgG: immunoglobulin type-G,
IVlg: intravenous immunoglobulin,
IT: immunotherapy,
LFT: liver function test,
LGI1: leucine-rich glioma inactivated protein 1,
MMSE: mini-mental state examination,
MOCA: Montreal Cognitive Assessment,
MRI: magnetic resonance imaging of the brain,
MRS: modified Rankin Score,
MTL: medial temporal lobe,
NA: not applicable,
NMDA: N-methyl-D-aspartate,
NR: not reported,
PET: positron emission tomography,
TPO: thyroid peroxidase,
VGKC: voltage-gated potassium channel,
WBC: white blood cell.
Author/s:
Broadley, J; Seneviratne, U; Beech, P; Buzzard, K; Butzkueven, H; O'Brien, T; Monif, M

Title:
Prognosis in autoimmune encephalitis: Database

Date:
2018-12-01

Citation:
Broadley, J., Seneviratne, U., Beech, P., Buzzard, K., Butzkueven, H., O'Brien, T. & Monif, M. (2018). Prognosis in autoimmune encephalitis: Database. DATA IN BRIEF, 21, pp.2694-2703. https://doi.org/10.1016/j.dib.2018.11.020.

Persistent Link:
http://hdl.handle.net/11343/253374

File Description:
Published version

License:
CC BY