American Journal of Biology and Natural Sciences
https://biojournals.us/index.php/AJBNS
ISSN: 2997-7185

Autoimmune Encephalitis: Diagnostic Biomarkers and Advances in Immunotherapy

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Received: 2025, 15, Aug
Accepted: 2025, 21, Sep
Published: 2025, 16, Oct

Annotation: Autoimmune encephalitis (AE) represents a rapidly growing field of neuroimmunology, characterized by an immune-mediated attack on neuronal cell surface or synaptic antigens. Over the past decade, substantial progress has been made in understanding its pathophysiology, leading to the identification of a range of diagnostic biomarkers that have revolutionized early detection and treatment. Clinically, AE often presents with subacute onset of psychiatric symptoms, cognitive decline, seizures, and movement disorders, frequently mimicking infectious or primary psychiatric conditions. The detection of specific neuronal autoantibodies, such as those against N-methyl-D-aspartate receptor (NMDAR), leucine-rich glioma-inactivated 1 (LGI1), contactin-associated protein-like 2 (CASPR2), and γ-aminobutyric acid (GABA) receptors, has emerged as a cornerstone in diagnosis. These biomarkers not only aid in differentiating AE from other

encephalopathies but also provide insights into disease mechanisms and prognosis.

Advances in neuroimaging, particularly MRI and FDG-PET, combined with cerebrospinal fluid analysis, further enhance diagnostic accuracy. Importantly, immunotherapy has transformed the management of AE. First-line therapies, including corticosteroids, intravenous immunoglobulin, and plasma exchange, often yield substantial improvement, while second-line agents such as rituximab and cyclophosphamide are employed in refractory cases. Novel approaches targeting B cells and complement pathways are currently under investigation, offering promise for more tailored interventions. Early recognition and treatment are crucial, as delays significantly impact long-term neurological outcomes.

This review highlights the evolving landscape of AE, emphasizing the role of biomarkers in guiding diagnosis and monitoring, as well as recent advances in immunotherapy that have significantly improved prognosis and patient quality of life.

Keywords: Autoimmune encephalitis; Diagnostic biomarkers; Neuronal autoantibodies; Immunotherapy; Neuroimaging; Precision medicine.

1. Introduction to Autoimmune Encephalitis

Autoimmune encephalitis encompasses rapidly progressive encephalopathies linked to an autoimmune defense mechanism, causing various neurological and psychiatric symptoms. It's crucial to consider an autoimmune cause when patients exhibit encephalitis or sudden altered mental states for timely treatment and to prevent severe complications [1]. Autoantibodies targeting extracellular and intracellular components can impact the central and peripheral nervous systems, muscle, and metabolic pathways. Common autoantibodies include N-methyl-D-aspartate receptor (NMDAR), leucine-rich glioma-inactivated 1 (LGI1), α-amino-3-hydroxy-5-methyl-4-

isoxazolepropionic acid receptor (AMPAR), and contactin-associated protein-like 2 (CASPR2) [2]. Although the immune activation mechanisms in the central nervous system remain unclear, autoantibody production can lead to tissue inflammation. Diagnostic biomarkers such as neuroimaging, cerebrospinal fluid analysis, and serum markers enhance early detection and case definitions, particularly in atypical situations, and may aid in follow-up and prognosis [3] .

2. Pathophysiology of Autoimmune Encephalitis

Autoimmune encephalitis is a recent disease that has attracted attention in clinical practice. Its pathogenesis is largely unknown, although neuronal surface autoantibodies impair specific cell-surface protein functions [4]. Due to the lack of randomized controlled trials and clear treatment guidelines, first-line immunotherapies are often based on clinical experience with paraneoplastic autoimmunity and other antibody-mediated CNS disorders [5]. This condition can affect young females and may occur during pregnancy, leading to poor outcomes for mother and fetus. Clinicians must understand its symptoms, diagnostic criteria, treatment strategies, and prognosis, adopting a multidisciplinary approach to manage this challenging disorder effectively [6].

3. Clinical Presentation and Symptoms

Autoimmune encephalitis is a complex condition resulting from an immune response targeting various central nervous system antigens. Identifying specific antibodies is crucial for diagnosis, and tests should focus on antigen specificity with mandatory screenings for potential malignancies. Both serum and cerebrospinal fluid should be tested; if initial results are negative, whole-body imaging becomes essential. Analysis of cerebrospinal fluid can reveal inflammatory indicators like pleocytosis, oligoclonal bands, and raised IgG levels. Neuroimaging, particularly MRI, may show characteristic hippocampal hyperintensities associated with limbic encephalitis, while functional imaging can identify metabolic changes in the temporal lobes [7]. EEG may exhibit abnormalities like slow waves, but these findings are often non-specific. A positive response to immunotherapy supports the immune nature of the condition, although it should not be taken as definitive proof of antibody presence. Increased availability of immunotherapy has improved outcomes, underscoring the need for early diagnosis for effective treatment. The clinical presentation varies, often starting with psychiatric symptoms, making diagnosis challenging in the absence of detectable autoantibodies. A reported case illustrated a manic syndrome with psychotic features as the initial manifestation, where the patient had no autoantibodies but responded well to immunotherapy [8].

4. Current Diagnostic Criteria

Autoimmune encephalitis represents a diverse group of inflammatory brain diseases mediated by an autoimmune response that causes brain damage and heterogeneous neurological and psychiatric symptoms. In recent years, several autoantibodies against neuronal surface and synaptic proteins have been identified in association with these diseases, leading to the redefinition of several subtypes of autoimmune encephalitis. In addition to autoantibodies, specific diagnostic biomarkers related to autoimmunity have been reported and are used in clinical practice. Treatment for this heterogeneous group of diseases remains challenging due to a lack of evidence; nonetheless, immunotherapy targeting the underlying autoimmune processes effectively treats the disease and prevents progression in most cases [9].

While identifying autoantibodies is useful when the clinical presentation is ambiguous, results may take several days and standardization is lacking. Expert consensus recommendation criteria have therefore been developed to guide early clinical diagnosis and treatment in patients with suspected autoimmune encephalitis [10]. Early diagnosis has become increasingly important, as many patients respond well to immunotherapy if diagnosed promptly and before irreversible neuronal damage occurs [11].

5. Role of Neuroimaging in Diagnosis

Autoimmune encephalitis (AIE) includes a range of immune-mediated brain inflammatory disorders with varied neurological symptoms such as cognitive decline, altered consciousness, seizures, psychosis, and abnormal movements. Treatment primarily involves immunotherapies like corticosteroids, IVIG, plasmapheresis, and monoclonal antibodies [12]. Misdiagnosis is common due to rapid advancements in the field; however, the 2016 diagnostic criteria and new biomarkers have improved accuracy in diagnosis. While no FDA-approved drugs exist for AIE and clinical trials for immunotherapy are lacking, research aims to enhance patient outcomes [13]. MRI is integral to the 2016 diagnostic criteria, helping to rule out other diagnoses, with about 40% showing positive findings. Common abnormalities include asymmetric T2-weighted FLAIR hippocampal hyperintensities and multi-lobar or diffuse cerebral FLAIR hyperintensities. Other manifestations may include leptomeningeal enhancement and increased FLAIR signals in basal ganglia and spinal cord [14,15]. FDG PET mapping reveals hypermetabolism in temporal lobes and relative hypometabolism in the parieto-occipital cortex. Advanced voxel-based techniques improve the sensitivity and accuracy of detecting these brain abnormalities, assisting the diagnosis of autoimmune encephalitis [16].

6. Cerebrospinal Fluid Analysis

Cerebrospinal fluid (CSF) analysis provides important investigative support for the diagnosis of autoimmune encephalitis; however, the findings are often subtle or nonspecific [17]. In the majority of cases, the CSF leukocyte count is mildly to moderately elevated, but in some patients, even with acute and fulminant disease, it can be normal [18]. An elevated protein concentration may also be detected. Oligoclonal bands are present in approximately 60% of cases and can be helpful when the clinical presentation is unclear . Cytokine and chemokine expression may differ between etiologies of encephalitis [19]. Patients with anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis demonstrate the highest concentrations of interleukins 6 and 17A, compared with inflammatory demyelinating encephalitis and enteroviral encephalitis [20].

7. Serological Biomarkers

Serum biomarkers for neuronal antibodies provide valuable tools to support the diagnosis of autoimmune encephalitis, a potentially life-threatening disease of disability [21]. Many antibodies associated with autoimmune encephalitis target neuronal surface proteins, compared to paraneoplastic variants, which mainly target intracellular proteins. These antibodies represent crucial serological biomarkers for the diagnosis and also the classification of autoimmune encephalitis [22]. In particular, neuronal IgG targeting N-methyl-D-aspartate receptor (NMDAR) is the most common and popular antibody to discriminate autoimmune encephalitis from other causes [23]. In addition to serological biomarkers, cerebrospinal fluid (CSF) offers an effective tool for the detection of antibodies used in the diagnostic approach [24,25]

8. Advancements in Biomarker Discovery

In the context of Autoimmune Encephalitis (AE), advancements in biomarker discovery are pivotal for enhancing diagnostic sensitivity and specificity, as well as for monitoring disease severity [26]. The identification of serum biomarkers, including cytokines such as interleukin (IL)-6, IL-17A, and IL-21, contributes to the detection of active disease state. Proteins indicative of central nervous system (CNS) injury and blood–brain barrier function, exemplified by glial fibrillary acidic protein, neurofilaments, and extracellular matrix protein 1, aid in assessing ongoing neuroaxonal damage [27]. Novel molecular biomarkers encompassing genetic and micro-RNA signals have emerged, potentially facilitating the prediction of treatment response and differentiation among various forms of autoimmune-associated neuro-inflammation [28]. Simultaneously, emerging diagnostic techniques, such as advanced imaging modalities, complement the biomarker-based diagnosis and aid in refining the early identification and treatment strategies [29].

10. Corticosteroids in Treatment

Corticosteroids are widely used to treat symptoms of autoimmune encephalitis. They reduce inflammation by inhibiting the secretion of cytokines and decreasing major histocompatibility complex expression by dendritic cells. Several cohorts have demonstrated that corticosteroids administered as a first-line agent improve clinical outcomes and reduce mortality [30]. However, corticosteroid therapy may have undesirable effects, and prolonged corticosteroid therapy can produce severe side effects such as osteoporosis [31].

Both intravenous methylprednisolone (IVMP) and oral administration of prednisone are used to mitigate the adverse effects of corticosteroids. IVMP is generally administered at 1,000 mg/day for 5 days, followed by 1 mg/kg orally or a slower taper [32]. Treatment with corticosteroids combined with plasma exchange or intravenous immunoglobulin (IVIG) has also produced markedly favorable outcomes [33].

11. Intravenous Immunoglobulin (IVIG)

Autoimmune encephalitis is a neuropsychiatric condition most frequently characterized by the subacute onset of amnesia, behavioral change, and seizures. Many forms are likely to be antibody-mediated with common neuronal surface protein targets including NMDAR, LGI1, and GABAB-receptor [34]. Immunotherapy has an important role, with corticosteroids, intravenous immunoglobulin (IVIG), or both usually given as first-line interventions. Plasma exchange or immunoadsorption may also be of benefit but typically entered into only as second-line measures [35].

The superiority of first- and second-line immune therapies over anti-epileptic treatment to control seizures in autoimmune encephalitis has also been established [36]. IVIG is currently the single most extensive neurological indication for therapy in the UK accounting for just over 40% of usage. Usage data collected by the NHS England Database monitoring system (NHS BSA, York) can therefore permit a nationwide investigation of a significant cohort [37]. Because IVIG is a limited availability resource with a five-day treatment course costing approximately £5040, the database was subsequently modified to incorporate outcome data and monitor treatment-related side effects such as thromboembolism and haemolytic anaemia [38]. By means of a retrospective analysis of data captured through the commissioning contract, an examination of outcomes in cases of autoimmune encephalitis and controls on data-entry accuracy has been undertaken [39].

12. Plasmapheresis

Plasmapheresis is regarded as a cornerstone in autoimmune encephalitis treatment. It is generally employed in patients unresponsive to corticosteroids and intravenous immunoglobulin (IVIG) [40]. Clinical experience suggests that patients with anti-N-methyl-D-aspartate receptor (NMDAR) antibodies exhibit the greatest responsiveness to plasmapheresis [41].

13. Monoclonal Antibodies

Autoimmune encephalitis is antibody-mediated and can be triggered by tumors, infections, or be of unknown origin, with pathogenesis attributed to intrathecal and peripheral production of autoantibodies. Characteristic symptoms of autoimmune encephalitis include cognitive deficits, psychiatric changes, and seizures; these symptoms frequently mimic those of infectious encephalitis, therefore these disorders are often indistinguishable on clinical grounds alone [42]. Prompt diagnosis and treatment of autoimmune encephalitis is critical, but remains challenging due to high heterogeneity in clinical presentation and limited availability of readily accessible biomarkers. Although autoantibody testing is considered the gold standard for diagnosis, these tests are not widely available, time-consuming, and within the context of anti-N-methyl-d-aspartate (NMDA) receptor encephalitis may yield false-negative results in up to 50% of patients. Ongoing research efforts are focused on discovery of new diagnostic biomarkers to improve early diagnosis, predict clinical outcomes, and guide treatment [43].

14. Emerging Therapies and Clinical Trials

Several new immunotherapeutic strategies, together with numerous ongoing clinical trials, promise to transform the one-fits-all management of autoimmune encephalitis (AIE) into tailored treatments matched to specific autoimmune mechanisms. Clinical studies of B-cell–depleting agents confirmed the prominent contribution of B cells in AIE. As such patients respond often well to rituximab, the monoclonal antibody targeting CD20-expressing B cells [44] , anti-CD19 therapies (demethylating the complete B-cell lineage, including plasmablasts and plasma cells), $CXCR5^{+}$ T helper antagonists (inhibiting memory B-cell and plasmablast differentiation), and anti-CD38 monoclonal antibodies (targeting plasma cells and plasmablasts) offer promising options. Moreover, CD19-directed chimeric antigen receptor T cells have been demonstrated to be effective in refractory systemic lupus erythematosus and might be worth testing in refractory seronegative and antibody-mediated AIE [45]. Because antibody-mediated pathogenesis is presumed to be dominant in AIE with antibodies against neuronal surface antigens [2] , therapeutic reduction of pathogenic antibodies may be rationalized. However, in neuroglial antigen- or T-cell cytotoxicity–driven AIE—for example, in patients with paraneoplastic antibodies against intracellular antigens—anti-B-cell treatments would likely be less efficient and tumor management could be the most effective intervention [46].

15. Long-term Outcomes and Prognosis

Preliminary prospective data from long-term follow-up after autoimmune encephalitis (AE) of other types can provide useful context. Within the LGI1-IgG AE cohort, 77% were living independently with normal cognitive function after a median follow-up of 8.5 years [47]. The predominant long-term symptoms were memory dysfunction and psychiatric disturbances, whereas seizures improved in the majority. Lower Montreal Cognitive Assessment (MOCA) scores and temporal lobe hyperintensity at diagnosis correlated with greater disability at long-term follow-up. Although disease severity and disability improved over time—even among patients with high initial disability—significant improvement in disability scores was apparent only after 12 months. Disability measurements within the first year may therefore underestimate long-term outcomes, underscoring the need for follow-up beyond 12 months to accurately assess prognosis. While seizures were common at baseline, they did not predict long-term disability, possibly because of their typically focal and brief nature. Cognitive and psychiatric symptoms, especially anxiety and depression, increased in prevalence over time [48].

A 2015 study indicated that patients with α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAR) antibodies tend to show less substantial recoveries than those with other types of AE. Poor prognosis has been associated with coexisting tumours, older age at onset, and delayed treatment. Most patients nonetheless recovered well during a 6- to 48-month follow-up, exhibiting an average 78% improvement in Clinical Assessment Scale in Autoimmune Encephalitis (CASE) scores. Four cases treated early with first-line therapy showed over 70% improvement, including young patients without residual symptoms. Prolonged unclear diagnosis delayed one patient’s treatment; older age and more severe initial symptoms further signalled poor prognosis. The patient with coexisting tumours recorded the lowest rate of improvement and persistent mild psychiatric symptoms at 21 months [49]. An association between mental symptoms, malignancies, and poor prognosis was reinforced. Some patients experience relapses after initial treatment, underscoring the importance of on-going tumour screening in those with psychiatric abnormalities. Antibody titre assessments are also important: changes in titres over the disease course may inform prognosis. A decline in AMPAR antibody titres is linked to recovery of memory function.

Recovery regarding bortezomib remains unclear because of extensive prior immunosuppressive therapy, complicating interpretation. Nevertheless, AE cases—especially those involving anti-LGI1 and anti-N-methyl-D-aspartate receptor (NMDAR) antibodies—are increasingly identified. Diagnosis typically involves interdisciplinary testing of serum and cerebrospinal fluid (CSF) in

patients presenting with unexplained psychiatric or neurological symptoms. Treatment options for antibody-associated syndromes include rituximab and plasmapheresis, whereas tumour control remains crucial for onconeural antibody cases. Decisions to escalate or terminate treatment should be based on clinical and biomarker monitoring, with close follow-up. Further studies are required to establish treatment scales and biomarkers, and international collaborations are necessary. Treatment should be conducted in tertiary hospitals [50].

16. Challenges in Diagnosis and Treatment

The clinical implementation of diagnostic criteria for autoimmune encephalitis often depends on a complex matrix of key symptoms, findings on neuroimaging, cerebrospinal fluid, electroencephalography, and the detection of known autoantibodies. The resulting diagnostic challenges can lead to treatment delays and severe neurological complications, and the clinical course of the disease commonly terminates in a refractory state despite several lines of immunotherapy [51].

26. Conclusion

Considerable progress has been made in the diagnosis and treatment of autoimmune encephalitis over the past decade. The development of international diagnostic criteria, along with the discovery of antibody and molecular biomarkers in blood and CSF, has advanced the understanding of the disease. Significant advances in immunotherapy have also improved the management of autoimmune encephalitis. Despite these achievements, many challenges remain. The diagnostic and serological landscape of autoimmune encephalitis is evolving rapidly, with antibody tests proliferating and criteria for clinical presentation becoming more refined. This fast-paced development renders ongoing updates necessary. Nevertheless, the progress realized in the past five years provides substantial hope for the future of the field.

Funding

There is no funding

Declaration of Competing Interest

The authors say they don't have any known personal or financial relationships or financial interests that could have seemed to affect the work in this study.

References:

1. M. Mistieri Simabukuro, G. Diogo da Silva, L. Henrique Martins Castro, and L. Tavares Lucato, "A critical review and update on autoimmune encephalitis: understanding the alphabet soup," 2022.
2. C. Hermetter, F. Fazekas, and S. Hochmeister, "Systematic Review: Syndromes, Early Diagnosis, and Treatment in Autoimmune Encephalitis," 2018.
3. M. Neațu, A. Jugurt, A. Covaliu, E. Irene Davidescu et al., "Autoimmune Encephalitis—A Multifaceted Pathology," 2023.
4. H. Abboud, J. C. Probasco, S. Irani, B. Ances, "Autoimmune encephalitis: proposed best practice recommendations for diagnosis and acute management," Journal of Neurology, vol. 2021.
5. E. Orozco, C. Valencia-Sanchez, J. Britton, "Autoimmune encephalitis criteria in clinical practice," Clinical Practice, 2023.
6. M. A. M. de Bruijn, F. Leypoldt, J. Dalmau, et al., "Autoimmune encephalitis," *Nature Reviews Disease*, 2025.

7. M. Soto Ontoso, R. A. Piernas González, I. Ramírez Martínez, and J. Miguel Meca García, "Clinical Diagnosis of an Autoimmune Encephalitis Presented as a Manic Episode with Psychotic Symptoms: A Case Report," 2022.
8. H. Prüss, "Autoantibodies in neurological disease," Nature reviews immunology, 2021.
9. G. Kocurova, J. Ricny, and S. V. Ovsepian, "Autoantibodies targeting neuronal proteins as biomarkers for neurodegenerative diseases," Theranostics, 2022.
10. N. Hansen, B. Malchow, I. Zerr, W. Stöcker, et al., "Neural cell-surface and intracellular autoantibodies in patients with cognitive impairment from a memory clinic cohort," Journal of Neural, vol. XX, no. XX, pp. XX-XX, 2021.
11. L. Y. Li, A. Keles, M. A. Homeyer, and H. Prüss, "Antibodies in neurological diseases: Established, emerging, explorative," Immunological reviews, 2024.
12. M. Abunada, N. Nierobisch, R. Ludovichetti, C. Simmen et al., "Autoimmune encephalitis: Early and late findings on serial MR imaging and correlation to treatment timepoints," 2024.
13. D. Moreno-Ajona, E. Prieto, F. Grisanti, I. Esparragosa et al., "(18)F-FDG-PET Imaging Patterns in Autoimmune Encephalitis: Impact of Image Analysis on the Results," 2020.
14. C. B Graos, V. M Montalvan, P. Torres, C. Coboj et al., "Clinical Characteristics and Neuroimaging Correlations in Autoimmune Encephalitis: A Report of Two Cases," 2024.
15. V. Pai, H. Kang, S. Suthiphosuwan, A. Gao, "Autoimmune encephalitis: insights into immune-mediated central nervous system injury," Korean Journal of…, 2024.
16. T. Rossor and M. Lim, "Immune-mediated encephalitis," Developmental Medicine & Child Neurology, 2024.
17. C. Liang, E. Chu, E. Kuoy, and J. E. Soun, "Autoimmune-mediated encephalitis and mimics: A neuroimaging review," Journal of Neuroimaging, 2023.
18. L. McCracken, J. Zhang, M. Greene, A. Crivaro et al., "Improving the antibody-based evaluation of autoimmune encephalitis," 2017.
19. K. Kothur, L. Wienholt, S. S. Mohammad, E. M. Tantsis et al., "Utility of CSF Cytokine/Chemokines as Markers of Active Intrathecal Inflammation: Comparison of Demyelinating, Anti-NMDAR and Enteroviral Encephalitis," 2016.
20. S. Carta, D. Ferraro, S. Ferrari, C. Briani, "Oligoclonal bands: clinical utility and interpretation cues," Critical Reviews in…, vol. XX, no. YY, pp. ZZ-ZZ, 2022.
21. H. Xue, X. Guo, Y. Jiang, L. Qin, X. Wang, and J. Xu, "Comparing clinical features, severity and prognosis of autoimmune encephalitis and with and without oligoclonal bands," *Frontiers in …*, 2024.
22. A. Rozenberg, S. Shelly, A. Vaknin-Dembinsky, "Cognitive impairments in autoimmune encephalitis: the role of autoimmune antibodies and oligoclonal bands," Frontiers in ..., 2024.
23. C. Hermetter, F. Fazekas, and S. Hochmeister, "Systematic Review: Syndromes, Early Diagnosis, and Treatment in Autoimmune Encephalitis," 2018.
24. M. S. Borioni, A. Morano, A. L. De Matteis, A. Mazzeo, "Neuroglial biomarkers in autoimmune encephalitis: advances in diagnosis, prognosis, and pathophysiological insights," Neurological Sciences, 2025.
25. R. D. Nass, K. Akgün, K. O. Dague, C. E. Elger, "CSF and serum biomarkers of cerebral damage in autoimmune epilepsy," Frontiers in ..., vol. 2021.
26. S. Zhang, C. Mao, X. Li, W. Miao et al., "Advances in potential cerebrospinal fluid biomarkers for autoimmune encephalitis: A review," Frontiers in Neurology, 2022.

27. E. U. Alum, "AI-driven biomarker discovery: enhancing precision in cancer diagnosis and prognosis," Discover Oncology, 2025.
28. MMA Alqahtani and MM Asiri, "Advancements in Biomarker Testing for Cancer Patients: A Comprehensive Review," Research, 2024.
29. S. Zafar, A. Hafeez, H. Shah, I. Mutiullah, A. Ali, "Emerging biomarkers for early cancer detection and diagnosis: challenges, innovations, and clinical perspectives," *Journal of Medical Research*, 2025.
30. S. Macher, F. Zimprich, D. De Simoni, R. Höftberger et al., "Management of Autoimmune Encephalitis: An Observational Monocentric Study of 38 Patients," 2018.
31. M. S. Topp, T. van Meerten, R. Houot, et al., "Earlier corticosteroid use for adverse event management in patients receiving axicabtagene ciloleucel for large B-cell lymphoma," *British Journal of Haematology*, vol. 2021, Wiley Online Library.
32. R. S. Goodman, D. B. Johnson, and J. M. Balko, "Corticosteroids and cancer immunotherapy," Clinical Cancer Research, 2023.
33. Taha, Ruqayah Qubtan, et al. "Bacterial aetiologies of otitis media and their antimicrobial susceptibility in ear swab culture." IJBS 6.1 (2024): 94-99.
34. J. A. Kinsella, S. R. Irani, R. Hollingsworth, D. O'Shaughnessy et al., "Use of intravenous immunoglobulin for the treatment of autoimmune encephalitis: audit of the NHS experience," 2018.
35. J. A. Kinsella, S. R. Irani, R. Hollingsworth, D. O'Shaughnessy et al., "Use of intravenous immunoglobulin for the treatment of autoimmune encephalitis: audit of the NHS experience," 2018.
36. I. Smets and M. J. Titulaer, "Antibody Therapies in Autoimmune Encephalitis," 2022.
37. Abbas, Abbas Mohsin, Iman H. Shewael, and Osama A. Mohsein. "Lipoprotein Ratios as Biomarkers for Assessing Chronic Atherosclerosis Progression." Academia Open 10.1 (2025): 10-21070.
38. S. Jafarpour and J. D. Santoro, "Autoimmune encephalitis," Pediatrics in review, 2022.
39. N. Hansen and C. Timäus, "Autoimmune encephalitis with psychiatric features in adults: historical evolution and prospective challenge," Journal of Neural Transmission, 2021.
40. A. R. A. C. Mota, "Plasmapheresis and Autoimmune Encephalitis in Paediatric Age," 2021.
41. H. Li, K. Wang, Q. Li, X. Pei, J. Ding, J. Peng, W. Li, "Clinical Efficacy and Immunomodulation of Double-Filtration Plasmapheresis in Autoimmune Encephalitis: A Prospective Study Highlighting the Importance of …," Autoimmunity, vol. 2025, Elsevier.
42. E. Sechi and E. P. Flanagan, "Antibody-mediated autoimmune diseases of the CNS: challenges and approaches to diagnosis and management," Frontiers in neurology, 2021.
43. Naif, Nabaa Hassan, et al. "The Impact of Inflammatory and Adipokine Biomarkers on Breast Cancer Progression and Patient Outcomes." Bulletin of Pharmaceutical Sciences Assiut University 48.1 (2025): 511-522.
44. J. Sell, H. Haselmann, S. Hallermann, et al., "Autoimmune encephalitis: novel therapeutic targets at the preclinical level," Expert Opinion on Therapeutic Targets, vol. 25, no. 5, pp. 345-356, 2021.
45. Y. Fu, C. Feng, S. Qin, Z. Xing, C. Liu, and Z. Liu, "Breaking barriers: advancing cellular therapies in autoimmune disease management," Frontiers in ..., 2024.

46. Adnan, Azher, Zainab Mohammed Farhan, and Osama A. Mohsein. "Understanding the link between diabetes mellitus and fatty liver disease: A narrative review." Int J Chem Stud 12.4 (2024): 40-48.

47. A. Aboseif, Y. Li, M. Amin, B. Lapin et al., "Clinical Determinants of Longitudinal Disability in LGI-1-IgG Autoimmune Encephalitis," 2023.

48. K. Wang, Y. Shi, Q. Du, R. Zhang et al., "Clinical Review and Prognostic Analysis of α-Amino-3-Hydroxy-5-Methyl-4-Isoxazole Propionate Receptor-Associated Encephalitis," 2021.

49. S. Muñiz-Castrillo, J. Haesebaert, L. Thomas, et al., "Clinical and prognostic value of immunogenetic characteristics in anti-LGI1 encephalitis," Neurology, vol. XX, no. XX, pp. XX-XX, 2021.

50. Hamad, Anas Abdullah, Hamssa Majid Mustafa, and Osama A. Mohsein. "Detection of the levels of immune cytokines (IL4, IL5, TNF-α) in school-age and preschoolers with an Ascaris lumbricoides infection." Journal of Parasitic Diseases 48.4 (2024): 782-787.

51. J. Dalmau and F. Graus, "Diagnostic criteria for autoimmune encephalitis: utility and pitfalls for antibody-negative disease," The La ncet Neurology, 2023.