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Autoimmune encephalitis. An overview of potential treatments and their current outlook

Encefalitis autoinmune. Una visión general de los posibles tratamientos y su perspectiva actual

Hidalgo Acosta Javier Aquiles[1*], Coello Ricaurte Víctor Félix[2], Mora González Milton Joel[3], Nagua Argudo Katryn Alexandra[1], Vera Vera Liliana Jazmin[4]

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[1] Universidad Espíritu Santo.
Faculty of Health Sciences.
Critical Care and Intensive Care.
Av. Samborondón 5, Samborondón 092301.
Guayaquil, Ecuador.

[2] Ministry of Public Health.
University Hospital of Guayaquil.
Calle 24A NW, Vía Perimetral.
Guayaquil, Ecuador.

[3] University of Guayaquil.
Faculty of Health Sciences.
Off. Delta s/n y Av. Kennedy.
Guayas, Ecuador.

[4] Ministry of Public Health.
Oramas Gonzales Maternity.
Cdla. Oramas González, Mz. 8, Solar 36.
Durán,Ecuador.

***Contact address**:

Universidad Espíritu Santo.
Faculty of Health Sciences.
Critical Care and Intensive Care.
Av. Samborondón 5, Samborondón 092301.
Guayaquil, Ecuador.
Phone: +593990733946.

Javier Aquiles Hidalgo Acosta
E-mail address: javhidalgo1985@gmail.com

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Abstract

Encephalitis refers to an inflammation of the brain secondary to an autoimmune response; it is characterized by being a serious neurological complication that presents various manifestations of a neuropsychiatric type. Hence, the objective of the research is to describe the current management of autoimmune encephalitis. The search terms consisted of antibody-mediated encephalitis, autoimmune diseases, nervous system, autoimmune encephalitis, in databases such as Pubmed, Mendeley, Wiley, Web of Science, Cochrane, Latindex and Google Scholar with articles published in the last 5 years and others for their interest from previous years. The results of clinical trials indicate that the current management of autoimmune encephalitis consists of the use of anti-convulsants as seizure prevention, immunotherapy with rituximab and cyclophosphamide, tumor resection in cases associated with tumors, plasma exchange, immunoadsorption, corticosteroids and the use of immuno-globulin. It is concluded that autoimmune encephalitis is a rare and multifactorial disease, whose diagnosis is based on the study of antibodies in cerebrospinal fluid in addition to imaging tests. The importance of this article lies in the need to recognize the neurological and psychiatric manifestations of autoimmune diseases for early action. The review recognizes advances in the treatment of autoimmune encephalitis, among the most important of which are immunotherapy and immunosuppressive therapy, which can modify the course of the disease.

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Resumen

Encefalitis se refiere a una inflamación del cerebro secundaria a una respuesta autoinmune, se caracteriza por ser una complicación neurológica grave que presenta manifestaciones diversas de tipo neuropsiquiátrico. De ahí que el objetivo de la investigación está orientado en describir el manejo actual de la encefalitis autoinmune. Los términos de búsqueda consistieron en encefalitis mediada por anticuerpos, enfermedades autoinmunes, sistema nervioso, encefalitis autoinmune, en bases de datos como Pubmed, Mendeley, Wiley, Web of Science, Cochrane, Latindex y Google Scholar con artículos publicados en los últimos 5 años y otros por su interés de años anteriores. Los resultados de los ensayos clínicos refieren que el manejo actual de la encefalitis autoinmune consiste en el uso de anticonvulsivantes como prevención de convulsiones, inmunoterapia con rituximab y ciclofosfamida, resección tumoral en casos asociados a tumores, recambio plasmático, inmunoadsorción, corticoides y uso de inmunoglobulina. Se concluye que la encefalitis autoinmune es una enfermedad rara y multifactorial, cuyo diagnóstico se fundamenta en el estudio de anticuerpos en líquido cefalorraquídeo además de pruebas de imágenes. La importancia del presente artículo radica en la necesidad reconocer las manifestaciones neurológicas y psiquiátricas de las enfermedades autoinmunes para una actuación temprana. La revisión reconoce los avances en el tratamiento de la encefalitis autoinmune entre los más importantes la inmunoterapia y la terapia inmunosupresora que puede modificar la evolución de la enfermedad.

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Introduction

Autoimmune encephalitis (AE) refers to inflammation of the brain secondary to an autoimmune response and is characterized as a serious neurological complication that presents various manifestations of a neuropsychiatric type[1,2].

There are patients with non-specific presentations, for example, a review determined that suicidal behavior in patients with systemic lupus erythematosus (SLE) was associated with neuropsychiatric SLE, which suggests a genetic origin due to haplotypes in chromosomal region 6p21.33, 6p21.22 that contain a combination of risk alleles that predispose to SLE and depression, this term encompasses neurological and psychiatric manifestations[3,4].

The neuropsychiatric manifestations of SLE are diverse and include: cognitive dysfunction, mood disorders, psychosis, cerebrovascular disease, demyelinating syndromes, and neuropathies. Symptoms can fluctuate, hence the importance of early recognition of AE, which will facilitate targeted treatment to reduce morbidity[5].

The differential diagnosis of seizures should include SLE encephalitis. In the presence of status epilepticus, secondary causes to autoimmune pathologies must be considered[6].

Physiopathologically, intracellular and extracellular autoantibodies against receptors in the neuronal nucleus are of great importance, among these we have: anti-neuronal nuclear antibodies (ANNA) important in the development of syndromes such as limbic encephalitis or encephalomyelitis, these autoantibodies target and attack proteins, DNA, RNA, altering the neuronal nucleic acid-protein complexes that are of great importance because they regulate the biological activities that the neuronal nucleic acid and protein complexes neThey are responsible for antibody-mediated AE and complex neurological manifestations[7]. Intracellular neuronal autoantibodies include: ANNA-1, anti-Yo (PCA-1) responsible for paraneoplastic cerebellar degeneration (PCD), anti-Ri (ANNA-2) causing opsoclonus, myoclonus, and brainstem encephalitis. Anti-CV2 (CRMP5) causes encephalomyelitis, motor neuropathy, sensory neuropathy and cerebellar ataxia, anti-amphiphysin is an autoantibody that causes stiff person syndrome, PCD and Lambert-Eaton syndrome, anti-Tr (PCA-Tr) is also associated with PCD, anti-Ma2 (PMNA2) has been identified in limbic encephalitis and mesoencephalic encephalitis. All these antibodies are associated with neurological pathology and various brain syndromes mediated by intracellular antibodies[8,9].

There is a wide variety of extracellular immunoglobulins responsible for autoimmune encephalitis, including: anti-protein inactivated by leucine-rich glioma type 1 (LGI1), dipeptidyl-peptidase-like antiprotein (DPPX), antibody against contactin-associated protein-2 (CASPR2), anti-N-methyl D-aspartate receptor antibody (NMDAr), anti-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (AMPAr), anti-acid receptor gamma-aminobutyric (GABABr), anti-metabotropic glutamate receptor subtype 5 (m-GluR5), voltage-gated anti-calcium channels (VGCC/CCVD). All of these may be responsible for the neurological manifestations of autoimmune diseases or paraneoplastic syndromes whose main manifestation is neuropsychiatric, and it is necessary to perform complete studies of the autoimmune profile in serum and cerebrospinal fluid for early diagnosis[10].

Among the types of encephalitis is autoimmune limbic encephalitis (ALE) associated with SLE, a rare presentation that occurs when inflammation exclu-

sively affects limbic and extralimbic structures such as the cerebellum, thalamus and trunk. Striatal dominant lupus encephalitis, characterized by affecting the striatum, affects young women with generalized immune dysregulation with multisystemic involvement, being considered a complex form of neuropsychiatric SLE (LESNP), being necessary brain magnetic resonance studies that will allow to observe changes in the signals in T2 and FLAIR at the level of the basal ganglia. There is also a very serious type known as SLE-associated encephalitis of the brainstem, a rare pathology, whose diagnosis is based on symptoms; cerebrospinal fluid (CSF) analysis. The autoimmune profile in CSF is a key tool for diagnosis, revealing the presence of antinuclear antibodies such as: anti-Ro or SSA antibodies, anti-La/SSB or antinuclear antibodies (ANA), manifesting with high CSF values associated with low complement (C3 and C4) in the blood in patients with lupus activity, which if accompanied by neuropsychiatric manifestation confirms the diagnosis of neurolupus. Magnetic resonance imaging (MRI) shows changes characterized by hyperintensity in T2 and decreased diffusion in FLAIR at the trunk level. Management consists of treatment with corticosteroids, immunosuppressants, and hydroxychloroquine[11-14].

Lupus encephalitis is a disorder that predominantly affects women with an average age of 42 ± 14 years, corresponding to 83 % of cases. Its clinical debut is mainly characterized by the presence of seizures and neuropsychiatric alterations. A review of 44 studies investigated 17 865 patients with early-onset SLE and 2970 with late-onset SLE, seizures and psychosis were more common than in patients with early-onset SLE, and neuropathy was more common in late-onset cases[15].

Diagnosis is clinical and imaging, including tomography, MRI of the brain and abdomen, electroencephalogram, serological analysis, and CSF analysis, the latter being the gold standard for evaluating the presence of antibodies.

Current treatment of lupus encephalitis includes corticosteroids as the first line, mainly prednisone and methylprednisolone, followed by cyclophosphamide at induction doses of 750 mg m^{-2} every 4 weeks for 24 weeks or 500 mg biweekly for 12 weeks, synthetic azathioprine DMARDs (2 mg/kg daily), biological therapies, plasmapheresis, intravenous immunoglobulin, autologous stem cell transplantation, and combination therapies[16]. Pulse glucocorticoids and intravenous cyclophosphamide are the first line of treatment when encephalitis is suspected, and are used in severe cases due to the risk of neurological complications[17].

The manifestations of the presence of autoantibodies will depend on the receptors and the affected areas, for example, those that affect NMDA receptors, against the GluN1 subunit, they are mainly directed to the hippocampus and the prefrontal cortex of the brain, causing lesions in neurons, generating excessive release of glutamate and dopamine, causing a variety of neurological symptoms such as changes in personality, hallucinations, aggressiveness, catatonia, epilepsy, ataxia, chorea, dysautonomia, alterations of consciousness, paranoia, delusions, acute psychotic episode to coma. Cases of tumor-associated AE have been detected, so in half of the cases they have been related to the presence of ovarian teratoma, prostate cancer, breast cancer, neuroblastoma, lymphoma, thymoma and other paraneoplastic syndromes, highlighting the importance of a complete investigation. In a study conducted on 108 female patients with anti-N-methyl-D-aspartate receptor encephalitis, 26.9 % had pathologically confirmed ovarian teratoma. This disorder has been associated with cases with postpartum psychosis and related to

other tumors such as neuroendocrine tumors, mediastinal teratoma, testicular teratoma, and small cell carcinoma of the lung, a study on its diagnosis in positive cases, the detection of anti-NMDAR antibodies was more frequent: 24.6 % (adult serum), 53.1 % (children's serum), 39.7 % (adult CSF), and 88.1% (children's CSF)[18,19].

Treatment of anti-NMDA antibody encephalitis includes immunotherapy with corticosteroids, intravenous immunoglobulin, plasmapheresis, cyclophosphamide, and rituximab. Surgical treatment includes oophorectomy and cystectomy followed by immunotherapy, which has been shown to improve the resolution of symptoms[18].

With these arguments, the objective is to describe the characteristics of AE and the possible treatments with its current perspective.

Material and methods

A structured review was carried out, for the research, two structured research questions focused on the management of AE were generated: What are the notable advances, especially in the use of biological therapies? What is the current treatment of lupus encephalitis?, Search terms consisted of Antibody-mediated encephalitis, autoimmune diseases, nervous system, AE, in databases such as Pubmed, Mendeley, Wiley, Web of Science, Cochrane, Latindex, and Google Scholar with published articles on the research topic. Observational studies, randomised studies, systematic reviews and meta-analyses, case presentations related to the topic were chosen to assess the number of available studies. Clinical characteristics and positive or negative treatment outcomes were used as the primary product, and all review authors contributed freely, proposing, executing or presenting results. The resources were randomly selected according to the PRISMA 2020 statement, without directing the investigation or search.

Eligibility criteria. Through the development of inclusion and exclusion criteria, the research was grouped for synthesis.

Inclusion criteria. i) Articles published in the AE Journals. ii) Articles published on the clinical characteristics of AE. iii) Articles on advances in the management of AEs. iv) Articles on types of AEs.

Exclusion criteria. i) Unpublished articles. ii) Articles that do not address the topic of research with other types of encephalitis. iii) Articles with results not available.

Sources of information. Published articles on AE were reviewed using information from databases such as PubMed, Mendeley, Wiley, Web of Science, Cochrane, Latindex, and Google Scholar.

Search strategy. As a search strategy, manual randomization focused on the AE research topic was used, deleting articles that did not address the specific topic, Boolean operators and search words were: Antibody-mediated encephalitis, autoimmune diseases, nervous system, AE, lupus encephalitis treatment. The terms used to connect the search in the consulted databases allowed it to be restricted to these specific terms in Spanish and English.

Study selection process. Articles that met the objective and met the inclusion criteria were selected.

Data extraction process. Manual stratified randomization was used for data extraction according to the date of the research, the types of studies and the intervention used. To ensure transparency, the results

were extracted and analyzed together by a working group to avoid selection and randomization biases.

Assessment of risk of bias in publication. The studies used different tools and metrics, as well as rare disease bias due to the small sample size and the need to include case studies.

Measures of effect. The p-value was used to evaluate statistical inference; other measures evaluated were relative risk, confidence interval, absolute risk, odds ratio, median.

Assessing the certainty of the evidence. Randomized and non-randomized population-based studies and publications on AD were obtained. The evidence was derived from highly relevant research. We assessed the certainty of the evidence using the GRADE (Grading of Recommendations Assessment, Development and Evaluation) methodology.

Results

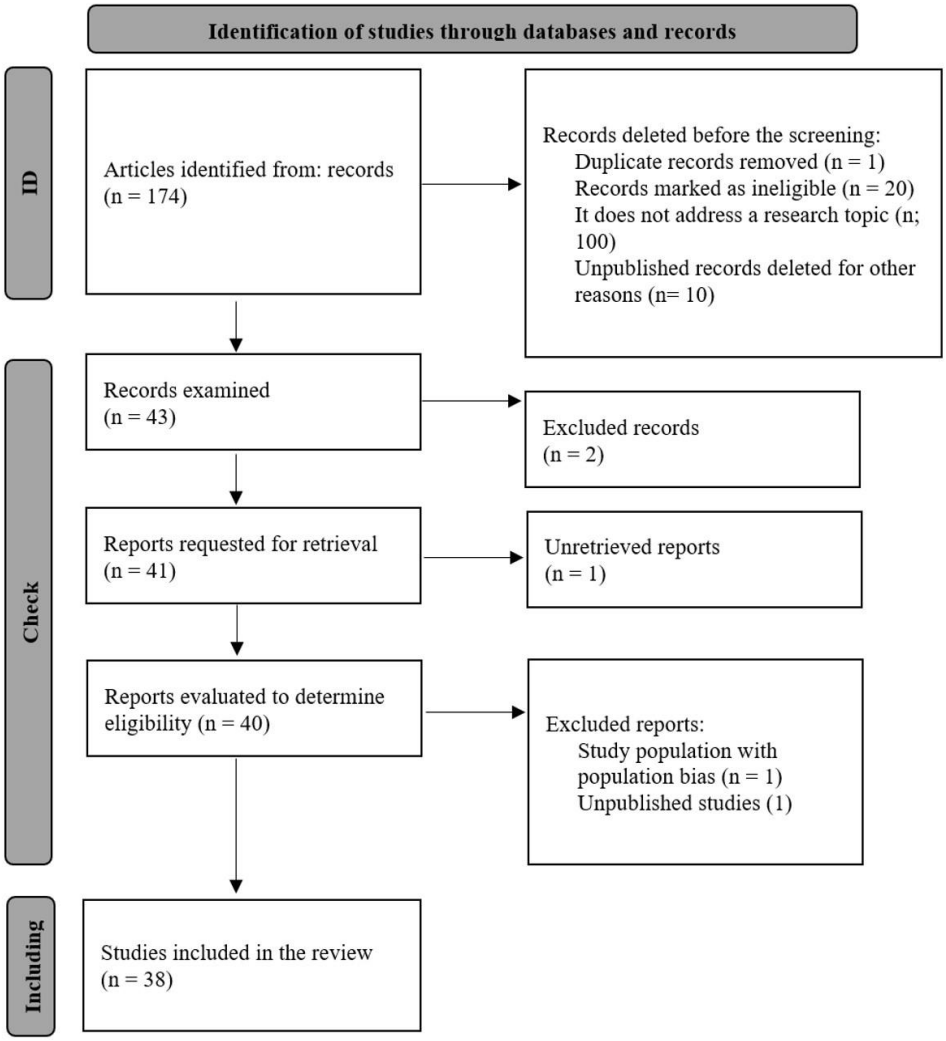


Figure 1 PRISMA diagram describing the inclusion/exclusion process

Table 1 Clinical characteristics of lupus encephalitis, possible autoimmune encephalitis and its first-line treatment

Reference	Clinical Features of Lupus Encephalitis, Possible AD	Results	Treatment
Raventhiranathan et al.[20]	Striatal dominant lupus encephalitis. 1 case reported: an 18-year-old male recently diagnosed with SLE was admitted to the emergency room due to fever, auditory and visual hallucinations, ataxia, headache, diarrhea, epigastric abdominal pain and bilateral flank pain for three days.	2 weeks after treatment, resolution of T2 FLAIR hyperintensity was observed in the bilateral basal ganglia.	Methylprednisolone 1 g daily for 5 days, plasmapheresis.
Robles-Monroy et al.[Art. 22]	Characteristics of the Possible AE: Previous infection 35.5% Headache 41.9 % Fever 41.9 % Psychotic symptoms 74.2 % Cognitive alterations 90.3 % Epileptic seizures 64.5 % Status epilepticus 19.4 % Catatonia 67.7 % Abnormal movements 71% Dysautonomia 58.1 % Diagnosis of tumor 6.5%.	58% of patients had a good prognosis, 42% had a poor prognosis, and mortality was 6.4%.	Boluses of methylprednisolone 88.8 - 100% received treatment and plasmapheresis 66.7-92.3%
Tsuchiya et al.[23]	Limbic encephalitis in a 46-year-old female patient with SLE, presenting with seizures, sensory aphasia, and pseudobulbar affect.	Elevated anti-GluRs titers with brain imaging at the midbrain level on MRI and monophoton emission computed tomography, restriction of water diffusion around the left thalamus, increased blood flow in the left temporal lobe and supramarginal gyrus. The images disappeared after treatment.	Pulses of methylprednisolone followed by high-dose prednisolone and intravenous cyclophosphamide.
Li et al.[24]	Neuropsychiatric lupus erythematosus with neurogenic pulmonary edema and limbic encephalitis due to anti-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor **(AMPAR)** in a 21-year-old woman with SLE with manifestations of generalized tonic-clonic seizures, headache and dyspnea.	The patient improved significantly after methylprednisolone pulse therapy and antiepileptic treatment.	Methylprednisolone 200 mg/day with gradual reduction to maintenance doses of 40 mg/day, 1.2 g/day of sodium valproate, and 1.0 g/day of levetiracetam seizure remission was obtained.
Chen et al.[25]	4 patients diagnosed with leucine-rich glioma 1 **(anti-LGI1)** inactivated encephalitis in patients with SLE, signs of limbic encephalitis, including short-term memory deficits, seizures, and psychiatric disorders. Two cases also presented seizures and faciobrachial dystonics.	Significant improvement in three patients, with no relapses over a follow-up period of 33 to 60 months, 1 patient had mortality from pneumonia.	Treatment with corticosteroids, intravenous immunoglobulin and mycophenolate mofetil as immunosuppressant.

The first-line treatment to be used when lupus or autoimmune encephalitis is suspected

Selection of studies. For the development of the research, randomized clinical trials, non-randomized observational studies, case presentations, reviews and meta-analyses were identified and selected for the analysis of data and studies obtained from the search terms.

The results of the search and selection processes were represented by a PRISMA 2020 flowchart (Figure 1).

The results of clinical trials show that PET is an advance in diagnosis that allows us to visualize that the affected areas are multiple, which are not visualized in images such as CT or MRI, treatment with immunotherapy has to be early in cases of failure of other treatments.

In addition, immunosorbent therapy will allow for greater generalization of therapy with the results of the two more lasting due to recurrences that occur after treatment.

Table 2 Advances in the management of autoimmune encephalitis diagnosis, rescue treatment and current immunotherapy, results of randomized clinical trials

Reference	Population	Treatment	Results	Level of evidence
Boedecker et al.[Art. 26]	32 patients, 19 were treated with plasmapheresis and 13 patients with immunosorbent (IA).	Immunoadsorption (AI) and plasma exchange in the treatment of autoimmune neurological diseases.	Significant reduction of the pro-inflammatory cytokines IL-12, IL-17, IL-6, INF-γ and TNF-alpha during treatment with AI, diagnosis of AEs treated with therapy (n:3) (limbic encephalitis, anti-NMDA receptor encephalitis, cerebellar syndrome with anti-GAD antibodies), the antibody reduction time was longer in AI P < 0.05.	High
Lee et al.[27]	23 patients they were treated with IV Ig, and 18 patients were treated according to the study protocol plus salvage immunotherapy when the patient deteriorated before day 8 or showed no improvement on day 8.	Intravenous immunoglobulin in AE (0.4 g/kg/day) for 5 days.	Rescue immunotherapy included steroids (n = 2), rituximab (n = 5), steroids followed by rituximab (n = 3), tocilizumab (n = 1), and rituximab followed by tocilizumab (n = 1). (77.3-77.8 %) there was significant improvement in both groups, particularly at day 8 (P 0.004), rescue immunotherapy after 8 days of evolution was not significant	High
Bai and aal.[Art. 28]	42 patients with acute/subacute seropositive AE and 45 healthy controls were performed	Brain imaging of positron emission tomography (PET) with 18F-fluorodeoxyglucose (18F-FDG).	We identified 15 areas that showed statistically significant changes in standardized absorption among AE patients compared to controls (p<0.05).	High

Currently the pillars of treatment of this consist of: 1) anticonvulsant therapy because they are present in 90 %, their treatment is relevant because it can occur unexpectedly, benzodiazepines, levetiracetam 30 to 60 mg kg^{-1} body weight are used as the first option in benzodiazepine-refractory status epilepticus, valproic acid is also included at a dose of 40 mg kg^{-1} maximum 3000 mg administered in 10 minutes, 2) immunotherapy with rituximab and cyclophosphamide/tumor resection in cases associated with tumors such as encephalitis due to anti-NMDA antibodies and 3) plasma exchange, immunoadsorption, corticosteroids and immunoglobulin consist of the initial base treatment.

Discussion

The diagnosis of lupus encephalitis is based on a history of SLE, positive antibodies and neurological manifestations with positive MRI studies; The main treatment includes corticosteroid pulses and plasmapheresis, with the aim of reducing the immune response and removing circulating autoantibodies through plasma exchange therapy[20,21].

A study conducted by Robles-Monroy et al. on the clinical characteristics and prognosis in 9046 patients admitted to the emergency room, 2.2 % were diagnosed with encephalitis as a probable autoimmune cause; at discharge, 0.3 % met diagnostic criteria for probable AD (PAD), 19 patients had anti-NMDAR encephalitis and 12 with negative PAD and anti-NMDAr antibodies[22].

Currently, the pillars of AD treatment consist of: 1) glucocorticoids[23], 2) anticonvulsant therapy, because they are present in 90%, their treatment is relevant because it can occur unexpectedly, benzodiazepines, levetiracetam 30 to 60 mg/kg body weight are used as the first option in benzodiazepine-refractory status epilepticus, valproic acid is also included at a dose of

40 mg/kg maximum 3000 mg administered in 10 min[24], 3) immunotherapy with rituximab and cyclophosphamide, 4) tumor resection in cases associated with tumors such as encephalitis due to anti-NMDA antibodies, and 5) plasma exchange, immunoadsorption, corticosteroids, and immunoglobulin [25-28], these treatments are considered the fundamental pillars of AD management because they modify the course of the disease and improve the prognosis[29].

As a last resort, biological therapy with rituximab is the most commonly used, being effective and safe in neuropsychiatric SLE. As part of lupus activity, there are investigational treatments such as Belimumab and Anifrolumab[30-32].

A meta-analysis on the treatment of AEs with rituximab reports that 72.2 % of patients obtained results (95% CI: 66.3-77.4 %) with an improvement in the modified Rankin scale, since it should be considered as a second line in cases of not responding to other treatments or presenting relapses, after treatment with corticosteroids. cyclophosphamide, immunoglobulin and plasmapheresis.

Third-line treatment includes immunosuppressive drugs [33,34].

Pediatric encephalitis has significant morbidity and mortality; however, 50% of cases are unexplained. The genetics of the host influence the development of encephalitis; however, the variants that contribute are little known[35].

Both genetic and environmental factors contribute to the development of the disease, with triggers such as exposure to ultraviolet light, infections, and hormonal changes affecting genetically susceptible individuals. The FDA has only approved two targeted biologic drugs for the treatment of SLE[36].

As a differential diagnosis, it is important to recognize toxoplasmic encephalitis, a fatal disease without treatment; previous case reports note that most SLE patients who developed toxoplasmic encephalitis were misdiagnosed and treated as neuropsychiatric SLE, leading to unfavorable outcomes[37].

In other cases, severe neuroinvasive disease (i.e., meningitis, encephalitis, and acute flaccid paralysis) was also observed in 1 % of people infected with West Nile virus (WNV), a mosquito-borne flavivirus, and constitutes an emerging public health problem in Europe, especially in older and immunocompromised people, whose clinical picture should be considered within the AD differential[38].

The current management of AE consists of the use of anticonvulsants as seizure prevention, immunotherapy with rituximab and cyclophosphamide, tumor resection in tumor-associated cases, plasma exchange, immunoadsorption, corticosteroids, and the use of immunoglobulin.

Conclusion

The importance of this article lies in the need to recognize the neurological and psychiatric manifestations of autoimmune diseases for early action; the review recognizes the advances in the treatment of AE among the most important immunotherapy and immunosuppressive therapy that can modify the evolution of the disease.

Corticosteroids are the mainstay of treatment for the vast majority of AE registries. As a second line of treatment: plasmapheresis, immunoadsorption and immunoglobulins.

An important achievement is to recognize that AE is part of the initial manifestation of paraneoplastic syndromes due to antibodies from existing tumors, since its timely diagnosis is crucial.

The research provides an overview of encephalitis associated with autoimmunity, being a rare disease, whose late diagnosis and treatment would expose a

catastrophic situation for the patient who suffers from it.

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Conflicts of interest

The authors declare that there are no conflicts of interest in the present research.

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Ethical considerations

The resources were chosen randomly according to the PRISMA 2020 declaration, without directing the research or search, they were analyzed together to avoid manipulation of the data, guaranteeing their availability and reproducibility, compliance with the ethical aspects of a systematic review.

Limitations in research

The limitation was dependent on the sample size in the studies due to the infrequent presentation of the pathology investigated, a greater number of studies with a larger sample size are needed to collect more data on some treatments under investigation, so future lines of research on the subject are suggested.

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All participants as authors of the review contributed to the research freely to propose, execute or present the results of the research and agree that the article will be published.

Contribution of the authors in the article

Javier Hidalgo, conceptualization and resources. *Víctor Coello*, methodology. *Milton Mora*, software and formal analysis. *Javier Hidalgo*, data curation. *Katryn Nagua*, writing the original draft. *Javier Hidalgo*, revision and editing of the original draft. *Liliana Vera*, visualization. *Víctor Coello*, research. *Javier Hidalgo*, supervision. *Milton Mora*, project management. *Katryn Nagua*, writing the final draft. *Javier Hidalgo*, revision of the final manuscript. *Liliana Vera*, final reception. *Javier Hidalgo*, acquisition of funds

Use of artificial intelligence

No artificial intelligence was used in the development of the manuscript.

Literature Cited

1. Venkatesan A, Tunkel AR, Bloch KC, Lauring AS, Sejvar J, Bitnun A, et al. Case definitions, diagnostic algorithms, and priorities in encephalitis: consensus statement of the international encephalitis consortium. Clin Infect Dis 2013;57(8):1114-28. DOI: https://doi.org/10.1093/cid/cit458. PMID: 23861361; PMCID: PMC 3783060.
2. Ahmed HS, D'Souza L, M S V, Sache MS. Neurological manifestations and complications of Kikuchi-Fujimoto disease: A comprehensive systematic review. Clin Neurol Neurosurg 2025;251: 108818. DOI: https://doi.org/10.1016/j.clineuro.2025.108818. PMID: 40056750.

3. Parperis K, Kyriakou A, Voskarides K, Chatzittofis A. Suicidal behavior in patients with systematic lupus erythematosus: Systematic literature review and genetic linkage disequilibrium analysis. Semin Arthritis Rheum 2022;54:151997. DOI: https://doi.org/10.1016/j. semarthrit.2022.151997. PMID: 35344734.
4. Voskarides K, Giannopoulou N, Eid R, Parperis K, Chatzittofis A. Genome-wide association studies reveal shared genetic haplotypes of autoimmune rheumatic and endocrine diseases with psychiatric disorders. Brain Behav 2023;13(4): e2955. DOI: https://doi.org/1002/brb3.2955. PMID: 36924079; PMCID: PMC1009 7071.
5. Jayasinghe M, Rashidi F, Gadelmawla AF, Pitton Rissardo J, Rashidi M, Elendu CC, et al. Neurological manifestations of systemic lupus erythematosus: A comprehensive review. Cureus 2025; 17(2):e79569. DOI: https://doi.org/10.7759 /cureus.79569. PMID: 40151747; PMCID: PMC1 19475 00.
6. Benavente E, Paira S, Roverano S, Saredo G. Encephalitis and status epilepticus in a patient with systemic lupus erythematosus. Reumatol Clin 2013;9(2):123-7. DOI: https://doi.org/10.1016/j. reuma.2012.05.007. PMID: 22766430.
7. Abbatemarco JR, Yan C, Kunchok A, Rae-Grant A. Antibody-mediated autoimmune encephalitis: A practical approach. Cleve Clin J Med 2021;88 (8):459-71. DOI: https://doi.org/10.3949/ccjm.88 a.20122. PMID: 34341030.
8. Carrasco Á, Alarcón I, González C, Graus F. Identification and clinical utility of antineuronal antibodies. Immunology 2014;33(4):128-136. DOI: https://doi.org/10.1016/j.inmuno.2014.04.0 03
9. Herrada-de la Cruz D, Bustamante-Canales LAA, Silva-Carrasco CP, Faúndez-Silva RA. Autoimmune encephalitis associated with anti-LGI1 antibodies: review of its clinical, diagnostic and therapeutic aspects. Rev Méd Clín Las Condes 2025; 36(3):192-7. DOI: https://doi.org/10.1016/j.rmclc. 2025. 06.004
10. Guasp M, Dalmau J. Autoimmune encephalitis. Med Clin North Am 2025;109(2):443-61. DOI: https://doi.org/10.1016/j.mcna.2024.09.001. PMI D: 39893022.
11. Khan MN, Fatima M, Mubashir M, Ali SMS. A case report of brainstem encephalitis associated with systemic lupus erythematosus in a young female. J Med Surg Public Health 2024;4:100145. DOI: https://doi.org/10.1016/j.glmedi.2024.1001 45
12. Monahan RC, Beaart-van de Voorde LJJ, Fronczek R, de Bresser J, Eikenboom J, Kloppen-burg M, et al. Clinical outcome in patients with suspected inflammatory neuropsychiatric lupus treated with immunosuppression: an observational cohort study. Lupus Sci Med 2023;10(1):e000850. DOI: https://doi.org/10.1136/lupus-202 2-000850. PMID: 36737098; PMCID: PMC9900068.
13. Chávez-Martínez EA, Martínez-Cabrera D, Padilla-Estrada CS, Hernández-Martínez HE, Ochoa-Madrigal MG. Neurolupus as a manifestation of first psychotic episode case report. Rev Fac Med 2023;66(3):27-34. DOI: https://doi.org/10.22201/ fm.24484865e.2023.66.3.03
14. Soo TH, Suppiah S, Zamri MF. The intersection of autoimmunity and neurology: insights from a case of striatal lupus encephalitis. Cureus 2024; 16(9):e68743. DOI: https://doi.org/10.7759/cureus.68743 | PMID: 39371814; PMCID: PMC114548 29.

15. Pamuk ON, Raza AA, Hasni S. Neuropsychiatric lupus in late- and early-onset systemic lupus erythematosus patients: a systematic review and meta-analysis. Rheumatology (Oxford) 2024;63(1): 8-15. DOI: https://doi.org/10.1093/rheumatology/kead297. PMID: 37341643; PMCID: PMC 10765 162.
16. Papachristos DA, Oon S, Hanly JG, Nikpour M. Management of inflammatory neurologic and psychiatric manifestations of systemic lupus erythematosus: A systematic review. Semin Arthritis Rheum 2021;51(1):49-71. DOI: https://doi.org/10.1016/j.semarthrit.2020.12.004. PMID: 33360230
17. Liu Y, Tu Z, Zhang X, Du K, Xie Z, Lin Z. Pathogenesis and treatment of neuropsychiatric systemic lupus erythematosus: A review. Front Cell Dev Biol 2022;10:998328. DOI: https://doi.org /10.3389/fcell.2022.998328. PMID: 36133921 ; PMCID: PMC9484581.
18. Nguyen L, Wang C. Anti-NMDA receptor autoim mune encephalitis: diagnosis and management strategies. Int J Gen Med 2023;16:7-21. DOI: https://doi.org/10.2147/IJGM.S397429. PMID: 36628 299; PMCID: PMC9826635.
19. Dai Y, Zhang J, Ren H, Zhou X, Chen J, Cui L, et al. Surgical outcomes in patients with anti-N-methyl D-aspartate receptor encephalitis with ovarian teratoma. Am J Obstet Gynecol 2019;221(5):485. e1-485.e10. DOI: https://doi.org/10.1016/j.ajog.2019.05.026. PMID: 31128109.
20. Raventhiranathan N, Hussien AR, Mirchia K, Swarnkar A, Mangla R. Striatal dominant lupus encephalitis-Is it vasculitis or an autoimmune process? Literature review & new case report with vessel wall imaging. Radiol Case Rep 2022;17(4): 1205-10. DOI: https://doi.org/10.1016/j.radcr.2022.01.002. PMID: 35169429; PMCID: PMC8829497.
21. Ercan E, Ingo C, Tritanon O, Magro-Checa C, Smith A, Smith S, et al. A multimodal MRI approach to identify and characterize microstructural brain changes in neuropsychiatric systemic lupus erythematosus. Neuroimage Clin 2015;8:337-44. DOI: https://doi.org/10.1016/j.nicl.2015. 05.002. PMID: 26106559; PMCID: PMC44742 80.
22. Robles-Monroy PC, Martínez-Ángeles V, Ramírez-Bermúdez J, Violante-Villanueva A, Salas-Alvarado L, García X, et al. Clinical features and functional prognosis in patients with possible autoimmune encephalitis in a neurological emergency department. Rev Neurol 2025;80(1):36202. DOI: https://doi.org/10.31083/RN36202. PMID: 40084651; PMCID: PMC11907703.
23. Tsuchiya H, Iwasaki Y, Shoda H, Takahashi Y, Fujio K. Limbic encephalitis in a patient with systemic lupus erythematosus successfully treated with high-dose glucocorticoids and intravenous cyclophosphamide therapy: the potential pathogennicity of anti-glutamate receptor antibodies. Mod Rheumatol Case Rep 2021;5(2):250-3. DOI: https://doi.org/10.1080/24725625.2021.1876340. PMID: 33441041.
24. Li R, Wang Y, Wu X, Wang J, Wei W, Li X. Neuropsychiatric lupus erythematosus with neurogenic pulmonary edema and anti-α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor limbic encephalitis: a case report. BMC Neurol 2022;22(1):222. DOI: https://doi.org/10.1186/s12883-022-02747-6. PMID: 35710378; PMCID: PMC9203256.
25. Chen S, Ren H, Fan S, Zhang S, Li M, Guan H. Autoimmune encephalitis associated with anti-

LGI1 antibody: a potential cause of neuropsychiatric systemic lupus erythematosus. Lupus Sci Med 2025;12(1):e001429. DOI: https://doi.org/10.1136/lupus-2024-001429. PMID: 39965877; PMCID: PMC11836784.

26. Boedecker SC, Luessi F, Engel S, Kraus D, Klimpke P, Holtz S, et al. Immunoadsorption and plasma exchange-efficient treatment options for neurological autoimmune diseases. J Clin Apher 2022;37(1):70-81. DOI: https://doi.org/10.1002/jca.21953. PMID: 34904748.
27. Lee ST, Lee HS, Lee WJ, Cha HA, Kim SH, Shin SY, et al. The safety and efficacy of intravenous immunoglobulin in autoimmune encephalitis. Ann Clin Transl Neurol 2022;9(5):610-21. DOI: https://doi.org/10.1002/acn3.51540. PMID: 35315247; PMCID: PMC9082384.
28. Bai S, Zhang C, Yao X, Shao H, Huang G, Liu J, et al. A novel classification model based on cerebral 18F-FDG uptake pattern facilitates the diagnosis of acute/subacute seropositive autoimmune encephalitis. J Neuroradiol 2023;50(5):492-501. DOI: https://doi.org/10.1016/j.neurad.2023.05.001. PMID: 37142216.
29. Macher S, Bsteh G, Pataraia E, Berger T, Höftberger R, Rommer PS. The three pillars in treating antibody-mediated encephalitis. Wien Klin Wochenschr 2024;136(1-2):13-24. DOI: https://doi.org/10.1007/s00508-023-02214-3. PMID: 37278857; PMCID: PMC10776469.
30. Wang M, Wang Z, Zhang S, Wu Y, Zhang L, Zhao J, et al. Progress in the pathogenesis and treatment of neuropsychiatric systemic lupus erythematosus. J Clin Med 2022;11(17):4955. DOI: https://doi.org/10.3390/jcm11174955
31. Morand EF, Furie R, Tanaka Y, Bruce IN, Askanase AD, Richez C, et al. Trial of anifrolumab in active systemic lupus erythematosus. N Engl J Med 2020;382(3):211-21. DOI: https://doi.org/10.1056/NEJMoa1912196. PMID: 31851795.
32. Manzi S, Sánchez-Guerrero J, Merrill JT, Furie R, Gladman D, Navarra SV, et al. Effects of belimumab, a B lymphocyte stimulator-specific inhibitor, on disease activity across multiple organ domains in patients with systemic lupus erythematosus: combined results from two phase III trials. Ann Rheum Dis 2012;71(11):1833-8. DOI: https://doi.org/10.1136/annrheumdis-2011-200831. PMID: 22550315; PMCID: PMC3465857.
33. Nepal G, Shing YK, Yadav JK, Rehrig JH, Ojha R, Huang DY, et al. Efficacy and safety of rituximab in autoimmune encephalitis: A meta-analysis. Acta Neurol Scand 2020;142(5):449-59. DOI: https://doi.org/10.1111/ane.13291. PMID: 32484900.
34. Halliday A, Duncan A, Cheung M, Boston RC, Apiwattanakul M, Camacho X, et al. Second-line immunotherapy and functional outcomes in autoimmune encephalitis: A systematic review and individual patient data meta-analysis. Epilepsia 2022;63(9):2214-24. DOI: https://doi.org/10.1111/epi.17327. PMID: 35700069; PMCID: PMC9796249.
35. Malik D, Simon DW, Thakkar K, Rajan DS, Kernan KF. Genetic variation in genes of inborn errors of immunity in children with unexplained encephalitis. Genes Immun 2022;23:235-9. DOI: https://doi.org/10.1038/s41435-022-00185-5
36. Polis B, Cuda CM, Putterman C. Animal models of neuropsychiatric systemic lupus erythematosus: decipherring the complexity and guiding therapeutic development. Autoimmunity 2024;57(1):2330 387. DOI: https://doi.org/10.1080/0891693

4.2024 .2330387. PMID: 38555866; PMCID: PMC12069 686.

37. Furuya H, Ikeda K, Iida K, Suzuki K, Furuta S, Tamachi T, et al. Disseminated toxoplasmosis with atypical symptoms which developed with exacerbation of systemic lupus erythematosus. Lupus 2019;28(1):133-6. DOI: https://doi.org/10.1177/0961203318815583. PMID: 30486727.

38. Schiuma M, Pezzati L, Ballone E, Borghi B, Osio M, Mattavelli D, et al. Case report: a fatal case of West Nile Virus meningoencephalomyelitis in a woman with systemic lupus erythematosus initially misdiagnosed as SARS-CoV-2 infection. Am J Trop Med Hyg 2021;104(5):1716-8. DOI: https://doi.org/10.4269/ajtmh.21-0041. PMID: 33782207; PMCID: PMC8103457.

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