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REVIEW ARTICLE

Temporal Lobe Epilepsy and Autoimmune Disorder Associations and Mechanisms: A Scoping Review

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ABSTRACT: Temporal Lobe Epilepsy (TLE) is a common form of epilepsy defined by seizures that specifically arise from the temporal lobe, an area of the brain vital for emotional processing and memory. TLE can be associated with several autoimmune conditions through underlying mechanisms, including Systemic Lupus Erythematosus, Hemolytic Anemia, and Type II Diabetes Mellitus (T2DM). This scoping review explores possible pathways and associations between the conditions. Examining inflammatory markers alongside stress-related pathways, including the hypothalamic-pituitary-adrenal (HPA) Axis, helps formulate these correlations. SLE and TLE are suggested to have a bidirectional relationship due to shared inflammatory processes and heightened expression of specific cytokines (e.g., IL-18). Evidence suggests that Hemolytic Anemia shares a relationship with epilepsy through the use of antiseizure medication, which can trigger further immune responses, ultimately resulting in hemolysis. T2DM is associated with TLE through heightened stress responses. Elevated cortisol levels and increased HPA axis activity are common mechanisms underlying both. Given the evidence, treatment implications include exploring complementary and alternative medicine to reduce the risk of developing these autoimmune disorders. Future studies should further explore the pathways behind hemolytic anemia and TLE to understand these comorbidities.

KEYWORDS: Cellular and Molecular Biology, Neurobiology, Epilepsy, Autoimmune.

Introduction

Epilepsy is a neurological condition defined by the presence of two or more seizures occurring within 24 hours, leaving lasting effects on both caregivers and those living with the condition. This condition is characterized by increased electrical activity in the brain, which can lead to seizures. In the United States, epilepsy impacts the lives of 3.4 million people.[1]

Epilepsy has been posited to be associated with a wide range of causes, one of which is chronic stress. Stress is associated with seizure occurrence and frequency,[2] as well as with Temporal Lobe Epilepsy (TLE). Heightened activity in brain regions such as the amygdala and hippocampus is implicated in TLE, and this heightened activity is one pathway through which seizures occur. TLE can subsequently lead to temporal lobe degeneration and dysfunction, particularly in the amygdala and hippocampus.[3] The bidirectional relationship between stress and seizures may be partially due to social stigma and fears of having a seizure in epileptic patients. Seizures occur when the balance between inhibitory and excitatory neurotransmitters is disturbed, leading to hyperexcitability, which is typically expressed as a seizure.[4] Seizures can also occur through an imbalance of ions in an action potential, a process that allows neurons to communicate.[4]

The hypothalamic-pituitary-adrenal (HPA) axis directly contributes to exacerbating stress responses, including increased release of glucocorticoids such as cortisol (with the assistance of adrenocorticotropic hormone and corticotropin-releasing hormone).[5] The limbic system, which regulates emotional processing, is directly involved in HPA axis *inhibition*, where the hippocampus antagonizes responses while the amygdala induces this glucocorticoid secretion.[6] Exacerbated levels of cortisol and glucocorticoids can result in hippocampal dendritic retraction, a form of reversible plasticity. Additionally, it can cause hippocampal damage, impairing memory and emotion, and reducing neurogenesis.[7] Therefore, this becomes a possible pathway for a seizure to occur. Increased neuronal activity in brain regions (such as the hippocampus) can cause inflammation during prolonged periods of stress.[8] Inflammation is the body's immune response to stimuli and external stressors it perceives as potentially dangerous or harmful.[8] This can have systemic effects, leading to inflammation in the entire body.

To understand epilepsy, stress, and autoimmune comorbidities, it is necessary to understand the bidirectional relationship between seizures and inflammation. When individuals experience seizures, pro-inflammatory cytokines and other mediators released by neurons and glial cells are activated, leading to inflammation.[9] Similarly, pre-existing inflammation can elevate these same mediators, disrupting the brain's excitatory/inhibitory balance and causing seizures.

Inflammation is correlated with, and is a predictor of, autoimmune disorders, given that inflammation is often a marker of overactive immune responses.[9] Some of the most common autoimmune conditions that are comorbid with epilepsy are Diabetes Mellitus, Systemic Lupus Erythematosus, and Hemolytic Anemia; these autoimmune conditions have both direct and indirect correlations with epilepsy. Systemic Lupus Erythematosus (SLE) is an autoimmune, multisystemic condition that primarily affects the body's connective tissue and nearly every organ system (skin, lungs, heart, etc.), affecting

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approximately 200,000 people in the USA.10 SLE causes inflammation in these body parts due to the immune system attacking its own cells. The production of autoantibodies and tissue injury impacts the likelihood of developing SLE, through environmental and maladaptive health behaviors that can also contribute to SLE development, such as smoking, sunlight, and certain medications.[11] Diabetes Mellitus (also known as Type II Diabetes) is characterized by hyperglycemia due to reduced insulin production; the prevalence of T2DM is about 11.6% of the US population.[12] Hemolytic Anemia is identified as a life-threatening and chronic condition caused by the destruction of premature red blood cells. Immune-mediated hemolysis can occur intravascularly, extravascularly, or in the reticuloendothelial system. Systemic inflammation can occur due to autoantibody production following the response to hemolytic anemia. It is novel to consider the potential comorbidities of epilepsy with these life-threatening conditions to find potential routes for prevention.

Taken together, while existing literature has shown associations between epilepsy and autoimmune disorders, there is a need to understand the mechanisms between these conditions. This scoping review aims to provide a synopsis of potential mechanisms by addressing the following aims:

1. We examined the associations between temporal lobe epilepsy and autoimmune disorders (DM, Lupus, HA).

2. We explore autoimmune processes/mechanisms underlying the potential relationships between epilepsy and common comorbid autoimmune disorders (DM, Lupus, HA).

Method

This scoping review searched Google Scholar and PubMed for relevant manuscripts.

The following keywords were used to search: ["Epilepsy" OR "seizure" AND "Neuroimmune" OR "autoimmune disorders" OR "diabetes" OR "lupus" AND "mechanisms"], as well as the search ["Epilepsy" OR "seizure" AND "autoimmune" OR "neuroimmune" OR "diabetes" OR "lupus" AND "development" OR "severity" AND "preventative" OR "health behaviors" OR "diet" or "exercise"]. Studies were included if they were primarily observational or review studies examining epilepsy and at least one autoimmune disorder. We excluded studies that used animal samples and included only those involving human subjects. Studies were not excluded based on publication date or participant age. We included 12 studies in our review (see Table 1 for study details). We examined the overall relationship between epilepsy and autoimmune disorders, then identified relevant themes and mechanisms from the literature. All results presented in the manuscript are results from existing experimental studies and reviews.

Discussion

We examine the mechanisms between epilepsy and three common comorbid autoimmune disorders, SLE, HA, and T2DM, to better understand their causes and therefore future treatment (see Table 1 for study descriptions).

Systemic Lupus Erythematosus:

Systemic Lupus Erythematosus (SLE) has a bidirectional correlation with epilepsy.[13] One subcategory of SLE, neuropsychiatric SLE, is particularly associated with seizures as a symptom. In this population, previous studies have identified specific antibodies, antiphospholipid antibodies, which are associated with epilepsy.

One proposed pathway through which SLE leads to epilepsy is through neural mechanisms. SLE is a systemic autoimmune disease that affects multiple organs and organ systems and can damage the immune system, potentially leading to neuronal injury. Seizures have also been proposed as a secondary pro-inflammatory process in the condition.[14] These injuries to the body's nervous system can cause cortical thrombosis (blood clots) in the meninges of the brain. The blood clots prevent the brain from receiving adequate oxygen, blood, and other vital nutrients. Then, the lack of these nutrients causes an increased risk of seizures and, in turn, epilepsy.[15]

In addition to SLE increasing the risk of epilepsy, evidence has been found to suggest epilepsy has an impact on the symptoms/presence of SLE by way of antiseizure medications (ASMs). Antiseizure medications have been found to increase pro-inflammatory cytokines, specifically IL-18.[16] IL-18 is a significant marker for astrocyte activity in the brain. These proinflammatory markers signify an active nervous system due to the astrocyte's role in synaptogenesis, the process of forming new connections known as synapses in the brain, and transmission.[17] Higher levels of inflammation (IL-18) are associated with SLE.[18]

Despite the suggestions of epilepsy and SLE impacting one another, limited evidence and research are examining the mechanisms behind these bidirectional comorbid disorders. The mechanisms of how the body's immune system causes neuronal damage remain uncertain. Additionally, there is limited research suggesting the correlation between anti-seizure medications causing increased IL-18 and IL-18, then increasing the risk of SLE.

Hemolytic Anemia:

In addition to SLE, HA, and epilepsy, they represent a similar correlation to suggest comorbidity; they are connected through anti-seizure medication (ASM) pathways. One review in particular, by Padda and colleagues,[19] examined the role of ASMs in the association between epilepsy and anemia. Taking antiseizure medications increases the likelihood of developing hemolytic anemia. ASMs bind to the cell membrane via a non-haptene reaction, leading to the production of antibodies that coat the cell.[20] This coating results in the activation of a system directly related to hemolysis (the breakdown of red blood cells) or activation of antibody-dependent-cellular-cytotoxicity; these cells are considered to be natural killer cells.[19,21] The medication will similarly bind to the red blood cell (erythrocyte) membrane in heptane reactions within ASM. The immune system will eventually recognize the ASM as a foreign substance, leading to B-cell-mediated hemolysis via the antibodies produced.[19]

Furthermore, another predicted correlation between seizure medications/epilepsy and hemolytic anemia is the process of potential oxidative injury. Metabolic processes in anti-seizure medications can render red blood cell membranes susceptible to lipid peroxidation. The body's cell membranes are made of primary lipids, and the peroxidation process induces the deterioration of these macromolecules. This can cause certain cases of hemolysis.[19]

However, conflicting literature has been proposed by Padda and colleagues,[19] where anti-seizure medication such as oxcarbazepine has been shown to induce hemolytic anemia in the case of a 75-year-old male. On the contrary, the literature suggests that oxidative injury primarily induces hemolysis, which is inconsistent with pathways implicated in hemolytic anemia and epilepsy. The potentially overlapping mechanisms suggest the need for further research regarding specific pathways.

Type II Diabetes Mellitus:

Epilepsy has been found to have a one-way correlation with Type II Diabetes Mellitus (T2DM). One mechanism suggesting this comorbidity is the hypothalamic arousal in patients with epilepsy. It has been reported that epileptic patients generally have higher levels of cortisol in their bodies due to neurotransmitter alterations and stress from seizures.[22] The hypothalamic-pituitary axis, or HPA Axis, is very active in producing cortisol in the body.[5] Elevated cortisol induces the release of stored glucose, leading to persistently high blood sugar (hyperglycemia). It is vital to remember that T2DM is caused by a deficiency of pancreatic insulin secretion, leading to elevated glucose levels.[23]

Physiological stress has been suggested to exacerbate and increase the risk of autoimmune disease as well. Epilepsy has a direct impact on the body's immune system, as previously mentioned. In particular, the development of epilepsy is associated with elevated levels of IL-1β, IL-6, and tumor necrosis factor-alpha (TNF), which are active immune system markers.[24] Each of these inflammatory markers is also elevated in T2DM.25

The existence of mental health stigma is indirectly correlated with the presence of diabetes. Epileptic patients are less likely to exercise and take care of their physical well-being in fear of having seizures; to prevent themselves from a risky situation, they limit themselves in all social aspects of life. This lack of long-term exercise can simultaneously induce obesity and lead to weight and blood sugar issues. Obesity can lead to hyperglycemia because the body is in a suboptimal physical condition.

Conflicts in Literature:

The reviews we examined regarding hemolytic anemia had limited information on the biological processes and evidence of comorbidity to epilepsy in comparison to SLE and T2DM. There was not much information regarding the mechanisms behind epilepsy and HA, and only very specific processes were mentioned. Similarly, the presence of epilepsy does not have a direct impact on the likelihood of developing T2DM but shows indirect effects on T2DM development via inflammatory pathways.[26]

Clinical Implications:

Understanding the comorbidity patterns, rates, and relationships between epilepsy and these common autoimmune conditions is vital in determining how to take appropriate precautions for patients. As suggested in the results, SLE and T2DM are correlated with epilepsy through inflammatory processes[27] and this can be considered by both physicians and patients moving forward, should they wish to target inflammation. As a practice to reduce immune inflammation, physicians can encourage their epileptic patients to limit their consumption of alcohol,[28] a substance known for its detrimental impacts on the liver. Additionally, SLE can be triggered by excessive sunlight; to compensate, regular vitamin D supplementation can be taken.[29] Additionally, clinicians may wish to screen for inflammatory markers among those with TLE that are associated with these autoimmune disorders, especially as elevations in some of these autoimmune markers (e.g., IL-1β, IL-6) are associated with medication resistance.[30]

Most striking is the limited research on HA and epilepsy for a high-risk group. Hemolytic anemia can cause death in children and put a mother's life at risk; understanding the consequences and side effects that anti-seizure medications can cause during pregnancy is necessary for the mother to know, so they can decide to proceed with medications or use alternative medications for mothers who are epileptic. Conveying this information to patients will help set a new standard for how epileptic mothers can maintain safety and health for both themselves and their children. Some individuals may opt for other validated non-pharmaceutical integrative medicine approaches to epilepsy treatments, such as mind-body movement practices,[31] herbal supplements,[32] and a controlled ketogenic diet,[31] all of which may allow pregnant mothers to be more cautious with ASMs before giving birth. This, in turn, can set a standard for mothers to follow, not only regarding their medications but also regarding additional protocols that may be implemented. To avoid excessive inflammation caused by anti-seizure medications, there is evidence that therapies such as medical cannabis, integrative medicine, and dieting may be helpful.[33]

Strengths:

This study focuses on gathering and organizing information on comorbidity risk factors for epilepsy that are currently understudied. Epilepsy is a condition associated with several life-threatening comorbidities, and medical practitioners need to take the correct precautions to avoid the development of autoimmune conditions and vice versa. We address the commonalities and differences in mechanisms between common autoimmune comorbidities (T2DM, SLE, and HA) and epilepsy.

Limitations:

This study is not without limitations; several limitations arose among the studies we included. Our results on hemo-

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lytic anemia were primarily derived from a single study, as the literature is limited on the association between HA and epilepsy. Furthermore, this singular study addressed all anemias and their relationship to epilepsy rather than focusing on HA specifically. This study relies on theories and case studies rather than experimental studies. This is a limitation because case studies examine only specific patients. This can lead to accidental bias because each patient has a unique background, which can influence the presence of either condition; it does not necessarily imply that a comorbidity is present. Accurate generalizations cannot be made from larger samples without accounting for external factors.

Future Directions:

This review opens up several questions for future discussion. More experimental studies concerning hemolytic anemia and epilepsy should be conducted to help identify the true underlying pathways of HA and epilepsy beyond the ASM pathway (inflammation or alternative mechanisms could be revealed). Treatment studies should be conducted in the future to provide experimental data on pregnant mothers prescribed ASMs and provide information beyond theoretical correlations. Furthermore, experimental data should be a focus of future studies on integrative medicine practices (e.g., nutrition and stress-reduction practices such as meditation/yoga) in relation to epilepsy and autoimmune disease comorbidities. With copious amounts of data, more accurate conclusions can be drawn about specific comorbidity pathways and how to prevent them through alternative practices.

Table 1: Summary of Included Studies Examining the Correlation Between TLE and Autoimmune Conditions (SLE, HA, T2DM).

Author, Year	Title	Number of Participants/number of studies (N)	Population	Key findings/Mechanisms	Type of Study (Review, observational, experimental)
Observational/Experimental Research Studies					
Chaudhry *et al.*[20]	Oxcarbazepine-Induced Hemolytic Anemia in a Geriatric Patient	75-year-old male case report	One patient	Oxcarbazepine (an antiepileptic drug) showed life-threatening effects on the patient, showing evidence of hemolysis	Case Report
Favilli, F., Anzilotti, C., Martinelli, L., Quattroni, P., De Martino, S., Pratesi, F., ... & Migliorini, P.[18]	IL-18 Activity in Systemic Lupus Erythematosus	n=40 individuals with SLE and n=17 healthy controls	Individuals with active SLE, inactive SLE, and healthy controls	IL-18 may act as a mechanism between stress and SLE.	Observational
Hopia, L., Andersson, M., Svenungsson, E., Khademi, M., Piehl, F., & Tomson, T.[15]	Epilepsy in systemic lupus erythematosus: prevalence and risk factors	n=440 SLE patients	SLE patients	SLE Pathways to epilepsy	Observational
Li, C. C., Chang, C. C., Cherng, Y. G., Lin, C. S., Yeh, C. C., Chang, Y. C., ... & Liao, C. C.[22]	Risk and outcomes of diabetes in patients with epilepsy	n=2854 patients with newly diagnosed epilepsy n=22,832 people without epilepsy n=92,438 patients with T2DM	Epileptic patients	Increased cortisol and interfered HPA axis in T2DM can result in an elevated risk of epilepsy.	Observational
Mochol, M., Taubøll, E., Aukrust, P., Ueland, T., Andreassen, O. A., & Svalheim, S.[17]	Serum Markers of Neuronal Damage and Astrocyte Activity in Patients with Chronic Epilepsy: Elevated Levels of Glial Fibrillary Acidic Protein	n=119 epilepsy patients and n=80 healthy controls	Epileptic patients (with glial damage analysis)	Antiseizure medications have been found to increase pro-inflammatory cytokines, specifically IL-18. IL-18 is a significant marker for astrocyte activity in the brain.	(Cross-Sectional, Observational)
Sinha, S., Patil, S. A., Jayalekshmy, V., & Satishchandra, P.[26]	Do cytokines have any role in epilepsy?	n= 16 epilepsy patients	Individuals with epilepsy	Cytokines released during inflammation affect the risk of epilepsy.	Observational
Tsai, J. D., Lin, C. L., Lin, C. C., Sung, F. C., & Lue, K. H.[14]	Risk of epilepsy in patients with systemic lupus erythematosus – a retrospective cohort study	32,301	Individuals with SLE from 1997 to 2010	SLE plays a role in affecting both the central and peripheral nervous system, which can correlate this condition with epilepsy	Retrospective Cohort Study
Review Studies					
Aarli, J. A.[16]	Epilepsy and the Immune System	N/A	General adult population	Inflammation is a mechanism of Systemic Lupus Erythematosus (SLE) and epilepsy	Review
DeFronzo, R. A., Ferrannini, E., Groop, L., Henry, R. R., Herman, W. H., Holst,	Type 2 Diabetes Mellitus	N/A	Individuals with Type 2 Diabetes Mellitus	Definition of T2DM and physiological and social implications	Review
Padda, J., Khalid, K., Syam, M., Kakani, V., Tonpouwo, G. K., Dhakal, R., ... & Jean-Charles, G.[19]	Association of Anemia With Epilepsy and Antiepileptic Drugs	N/A	Individuals with epilepsy and anemia	Epilepsy and hemolytic anemia share common pathways via anti-seizure medication pathways	Review
Rodríguez-Hernández, A., Ortiz-Orendain, J., Álvarez-Palazuelos, L. E., González-López, L., Gámez-Nava, J. I., & Zavala-Cerna, M. G.[13]	Seizures in systemic lupus erythematosus: A scoping review	N/A	General	Epilepsy and SLE are bidirectional in terms of inflammatory risk pathways	Review
Valencia, I.[27]	Epilepsy in Systemic Autoimmune Disorders	N/A	Epileptic patients and patients with autoimmune conditions	SLE is an autoimmune inflammatory disorder affecting multiple organs, including the brain and peripheral nervous system.	Review
Zaccara, G., Franciotta, D. and Perucca, E.[21]	Idiosyncratic Adverse Reactions to Antiepileptic Drugs	N/A	General Population	Mechanisms and risks of anti-seizure medication via activation of natural killer cells	Review

The study table reports on demographic details, method, and key findings of each study.
Note: IL=interleukin; SLE= Systemic Lupus Erythamotosus; HA= Hemolytic Anemia; HPA=Hypothalamic-Pituitary-Adrenal; T2DM=Type 2 Diabetes Mellitus

Conclusion

This study aimed to explore the comorbidities between epilepsy and common autoimmune conditions. Regarding our first research question (*Is there an association between temporal lobe epilepsy and autoimmune disorders?*), our hypothesis was partially supported. First, there was a bidirectional relationship between epilepsy and SLE. Further, we see that epilepsy is predictive of T2DM through both neuro-immune and behavioral pathways. Lastly, epilepsy was only associated with hemolytic anemia when accounting for the use of anti-seizure medications rather than a direct association. We saw particularly strong associations between diabetes and epilepsy, with 19-25% of individuals with diabetes experiencing seizures.[34]

Regarding our second research question (*What are the autoimmune processes/mechanisms underlying the potential relationships between epilepsy and common comorbid autoimmune disorders?*), our *hypothesis that inflammation would be a mechanism linking* these conditions was partially supported. Studies show SLE does lead to an increased rate of seizures or epilepsy by way of increased inflammation. In particular, the inflammation found in SLE includes many of the same antibodies found in epilepsy. Seizures are more common among those with SLE-induced inflammation, where medication can give rise to the risk of seizures.

Regarding T2DM, studies show that epilepsy may increase the risk of T2DM by way of elevated HPA axis activity, increased cortisol, and increased glucose regulation. Epilepsy also directly affects the immune system, particularly the inflammatory cytokine IL-6, which is involved in T2DM. Overall, proinflammatory cytokines appear to play a role in how epilepsy is associated with DM.

Regarding HA, our hypothesis that inflammation would be a direct mechanism was not as strongly supported. The mech-

anism between HA and TLE is through medication-induced effects, including hemolysis. There is less robust evidence suggesting alternative mechanisms through which hemolytic anemia could impact epilepsy. Both SLE and T2DM influenced the presence of epilepsy through inflammatory processes, but that was not the case with HA. HA occurs through damage to blood plasma (intravascularly and extravascularly) and to the spleen/liver, and anti-seizure medications follow these same pathways.[35]

Final Conclusions:

Overall, this review aimed to examine the association between epilepsy and autoimmune conditions, as well as the proposed mechanisms linking them. The findings illustrated that epilepsy is associated with comorbidities such as systemic lupus erythematosus, hemolytic anemia, and diabetes mellitus. Inflammation through anti-seizure medications and physiological stress from seizures are key factors in the relationship between these comorbidities. In the future, more research should be conducted on stress, inflammation, and hemolytic pathways.

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