Research Article

BIOMEDICAL Journal of Scientific & Technical Research

ISSN: 2574 -1241

DOI: 10.26717/BJSTR.2023.53.008376

Epilepsy; An Insight into Epileptogenic Potential of Infections and Antibiotics

Nadia Ilyas[1], Humaira Nawaz[2], Asim Gamaryani[3], Muhammad Hasnain[4], Zunaira Saeed[5], Afaq Ahmad[2], Syed Hamza Abbas[2], syed yawar syed[2], Inam ur Rahman Zafar[2], Nimrah Shah[2], Jawad Ali[2] and Shahzar Khan[2]*

[1]*Department of Microbiology, Abdul Wali khan University Mardan, Pakistan*

[2]*Department of Microbiology, Quaid-i-Azam University Islamabad, Pakistan*

[3]*School of Health and society, University of Wollongong, Australia*

[4]*Department of Pharmacy, University of Peshawar, Pakistan*

[5]*Institute of Molecular Biology and Biotechnology, University of Lahore, Pakistan*

***Corresponding author:** Shahzar Khan, Department of Microbiology, Quaid-i-Azam University Islamabad, Pakistan

ARTICLE INFO

Received: September 19, 2023

Published: October 12, 2023

Citation: Nadia Ilyas, Humaira Nawaz, Asim Gamaryani, Muhammad Hasnain, Zunaira Saeed, Afaq Ahmad, Syed Hamza Abbas, syed yawar syed, Inam ur Rahman Zafar, Nimrah Shah, Jawad Ali and Shahzar Khan. Epilepsy; An Insight into Epileptogenic Potential of Infections and Antibiotics. Biomed J Sci & Tech Res 53(2)-2023. BJSTR. MS.ID.008376.

ABSTRACT

Epilepsy is a neurological disorder characterized by recurrent seizures. In addition to genetic and idiopathic causes, certain drugs, infections, and viral illnesses have been associated with epileptogenic potential. This study aims to explore the epileptogenic potential of antibiotics, other medications, viral infections, bacterial infections, and parasitic infections. Antibiotics, such as fluoroquinolones and penicillin's, have been reported to have a low epileptogenic potential. However, some specific antibiotics, such as cephalosporin's and carbapenems, may carry a slightly higher risk of seizures in susceptible individuals. Certain medications, including antidepressants (e.g., selective serotonin reuptake inhibitors), antipsychotics (e.g., clozapine), and anti-malarial drugs (e.g., mefloquine), have been associated with an increased risk of seizures, particularly at higher doses or in predisposed individuals. Viral infections, such as herpes simplex virus, human immunodeficiency virus (HIV), and influenza, can induce seizures directly or as a result of associated encephalitis. In particular, herpes simplex virus encephalitis is a well-documented cause of seizures and epilepsy. Bacterial infections, including meningitis and brain abscesses caused by bacteria such as Streptococcus pneumoniae and Staphylococcus aureus, can lead to seizures due to the inflammatory response and neuronal damage in the brain. Parasitic infections, such as cerebral malaria caused by Plasmodium falciparum and neurocysticercosis caused by Taenia sodium, are associated with a high risk of seizures. The invasion of cerebral vasculature and inflammatory response in cerebral malaria, as well as the presence of cysts in neurocysticercosis, contribute to the epileptogenic potential of these parasitic infections. Understanding the epileptogenic potential of various antibiotics, medications, viral infections, bacterial infections, and parasitic infections is crucial for accurate diagnosis, appropriate treatment, and better management of epilepsy patients.

Abbreviations: HIV: Human Immunodeficiency Virus; HSV: Herpes Simplex Virus; GABA: Gamma-Aminobutyric Acid; ROS: Reactive Oxygen Species; INH: Antitubercular Medication Isoniazid; GM: Gut Microbiota; CNS: Central Nervous System; TLE: Temporal Lobe Epilepsy; HPeV: Human Parechovirus; ACE2: Angiotensin-Converting Enzyme 2; HHV-6: Human Herpesvirus 6; HAV: Hepatitis A Virus; BBB: Blood-Brain Barrier; TMEV: Theiler's Murine Encephalomyelitis Virus; EBV: Epstein-Barr Virus

Introduction

Seizures and epilepsy are complex neurological disorders that can have a profound impact on individuals' lives. Epilepsy, in particular, is a chronic condition characterized by recurrent seizures, which can cause seizures, convulsions, and loss of consciousness. There are many different factors that can contribute to the development of seizures and epilepsy, including genetics, traumatic brain injuries, and infections (Fisher, et al. [1,2]). In recent years, however, there has been growing concern about the potential role of antibiotics in the development of seizures and epilepsy. Antibiotics are among the most commonly prescribed medications worldwide, and they have been instrumental in reducing morbidity and mortality associated with infectious diseases (Ventola [3]). However, recent studies have suggested that antibiotics may have unintended adverse effects on neurological function, including the potential to increase the risk of seizures and epilepsy (Vezzani, et al. [4]). The mechanisms underlying this association are not yet fully understood, but may involve alterations in the gut microbiome, inflammation, and changes in neurotransmitter signaling (Morgan [5]).

However, it is important to weigh the potential benefits of these medications against the risk of seizures when prescribing them to patients. Viral infections have also long been recognized as potential triggers for seizures. Viruses such as herpes simplex virus (HSV), human herpesvirus 6have been associated with seizure activity in both children and adults. These viruses can directly infect the central nervous system and induce inflammation, leading to the development of seizures (Braun, et al. [6]). Bacterial infections, including meningitis and encephalitis are also known to be associated with an increased risk of seizures. The inflammatory response triggered by bacterial infections can disrupt the normal functioning of the brain and lead to seizure activity (Young [7]). Parasitic infections, such as neurocysticercosis and toxoplasmosis, have been implicated as potential causes of epilepsy. Parasites can directly invade the brain or elicit an immune response that can result in inflammation and seizures (Vezzani, et al. [8]). The epileptogenic potential of antibiotics, other drugs, viral infections, bacterial infections, and parasite infections is highlighted in this article (Figure 1).

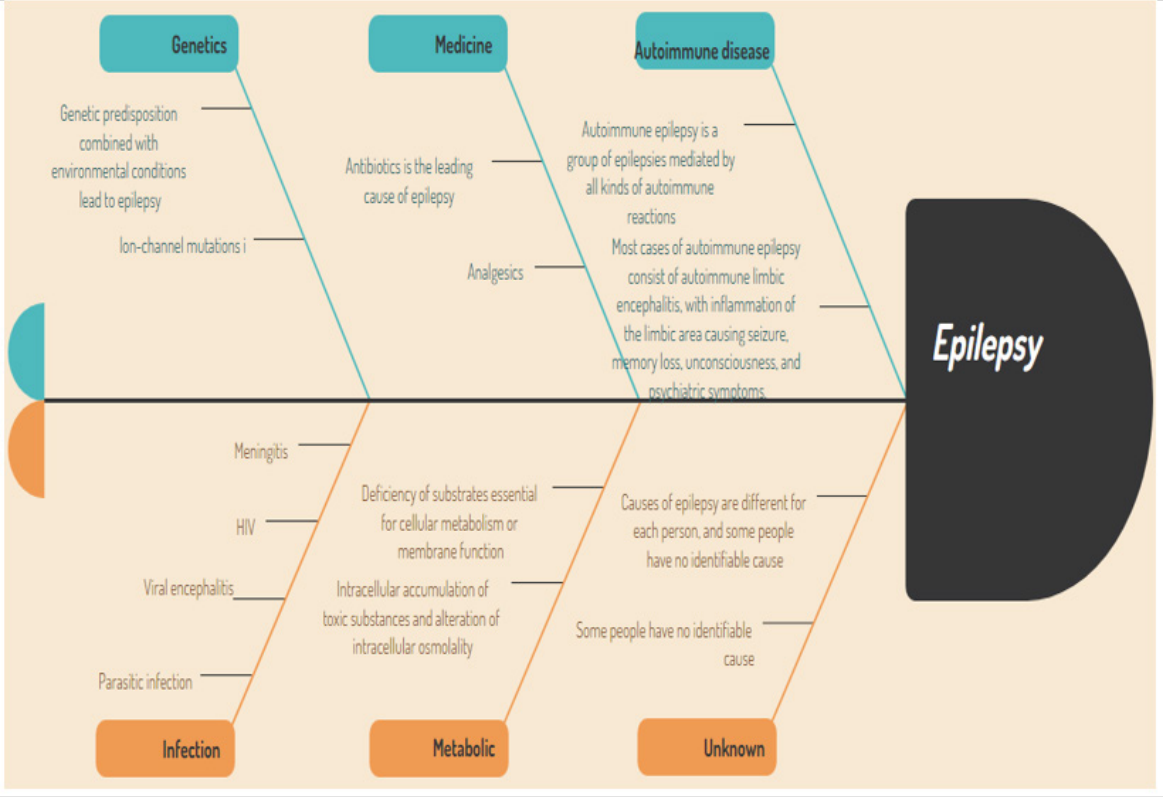

Figure 1: Representation of causative agents of Epilepsy through Fish Bone Diagram.

Antibiotic Induced Seizure and Its Mechanism

The exact mechanisms by which antibiotics can lead to seizures and epilepsy are not fully understood. However, some studies have suggested that antibiotics may interfere with normal brain function by affecting various biochemical pathways in the brain. Here are some examples:

Gabaergic Neurotransmission

The brain's major inhibitory neurotransmitter is gamma-aminobutyric acid (GABA), and its activity is crucial for regulating neuronal excitability and preventing seizures. Some antibiotics have been shown to interfere with GABAergic neurotransmission, potentially leading to increased neuronal excitability and seizures. For example, beta-lactam antibiotics like penicillins and cephalosporins have been shown to inhibit the activity of GABA transaminase, an enzyme that breaks down GABA in the brain. GABA levels may rise as a result, and at first this may have an anticonvulsant impact on the brain. However, over time, the increased levels of GABA may lead to downregulation of GABA receptors and increased neuronal excitability, potentially leading to seizures (Razavi [9]).

Glutamatergic Neurotransmission

The main excitatory neurotransmitter in the brain, glutamate, plays a crucial role in maintaining proper brain function. But excessive glutamate release or poor glutamate clearance can cause seizures and neuronal hyperexcitability. Some antibiotics, such as fluoroquinolones, have been shown to inhibit the activity of glutamate transporters, which are responsible for removing excess glutamate from the brain. This can lead to increased levels of extracellular glutamate and hyperexcitability of neurons, potentially leading to seizures (Takano, et al. [10]).

Inflammation

Finally, some studies have suggested that antibiotics may induce neuroinflammation, which can contribute to neuronal damage and seizures. For example, fluoroquinolones have been shown to activate microglia, the immune cells in the brain, leading to the production of pro-inflammatory cytokines and reactive oxygen species (ROS) (Bhattacharyya, et al. [11]).

Epileptogenic Potential of Cell Wall Synthesis Inhibitors

Penicillins, cephalosporins, carbapenems, and monobactams are examples of antibiotics that prevent the formation of bacterial cell walls, which are essential for bacterial survival and reproduction. Nevertheless, there is mounting evidence that some antibiotics may have negative neurological consequences, such as epilepsy and seizures. The disturbance of GABAergic neurotransmission, which is essential for controlling neuronal excitability and avoiding seizures, is the basis for the link between cell wall synthesis inhibitors and epilepsy. Seizures may result from beta-lactam medicines such cefepime and piperacillin-tazobactam that raise GABA levels in the brain. Additionally, glutamatergic neurotransmission, which is similarly connected to epilepsy, may be impacted by cell wall synthesis inhibitors. Some antibiotics, notably carbapenems, block glutamate transporters, increasing extracellular glutamate levels and causing neuronal hyperexcitability, which may lead to seizure (Razavi, et al. [9,10,12-15]).

Macrolide

It is not fully understood how macrolide antibiotics cause seizures. Although macrolides may cause neurological adverse effects such disorientation and dizziness, epileptic seizures are extremely uncommon and not frequently recorded. There have been a few isolated case reports of macrolides possibly causing seizures in some people, however these reports are anecdotal and do not prove a definitive cause-and-effect link. It is believed that there is a minimal overall risk of getting epilepsy or having seizures as a result of macrolides. A healthcare practitioner should be consulted for specific guidance on the use of macrolide antibiotics and any possible dangers (Carranco [16,17]).

Fluoroquinolones

Fluoroquinolones, commonly used to treat infections, have been associated with CNS side effects, including seizures. Underlying risk factors, such as a history of epilepsy, renal or hepatic failure, and drug interactions, increase the susceptibility to quinolone-induced seizures. The mechanism may involve GABA receptor stimulation and activation of the NMDA receptor. Drug interactions with NSAIDs and electrolyte imbalances can potentiate the proconvulsive activity of fluoroquinolones. Cases of quinolone-induced seizures have been reported, and in some instances, substituting the treatment resolved the seizures (Green [18,19]).

Isoniazid

Antitubercular medication isoniazid (INH) has the potential to be hazardous and even therapeutic dosages can result in seizures. Inhibiting pyridoxal-5 phosphate, a required cofactor for glutamic acid decarboxylase's enzymatic activity, prevents GABA production. This causes GABA levels to drop and increases seizure susceptibility. Further reducing GABA concentrations is pyridoxine depletion brought on by INH. INH poisoning can cause seizures that are severe and even lead to status epilepticus. These seizures can be controlled with pyridoxine treatment. Therefore, even when INH is administered at therapeutic levels, it is important to take pyridoxine supplementation into account and rule out the possibility that pyridoxine shortage is the root of seizures (Thomsen [20-22]).

Safe Antibiotics in Epilepsy

Research on tetracycline-class drugs, including minocycline, doxycycline, and tetracycline, discovered possible anti-seizure properties. These antibiotics may have neuroprotective qualities due to their anti-apoptotic and anti-inflammatory actions. In vivo testing revealed that these antibiotics protected against partial seizures in seizure

tests, highlighting their potential as supplementary medicines for epilepsy therapy (Wang [23]).

Current Evidence on the Association of Antibiotics and Seizure

Several studies have connected antibiotics to increased seizures and epilepsy. (Wang [23]) discovered that exposure to broad-spectrum antibiotics and specific antibiotic families, such as cephalosporins, fluoroquinolones, and macrolides, was linked to an elevated incidence of epilepsy. The processes behind this relationship are not entirely known, however they may entail disturbance of the gut flora, neurotoxicity, and immunological responses. Some antibiotics modify the gut flora, influencing the central nervous system, while others have direct neurotoxic effects. Antibiotics, according to current research, may increase the incidence of seizures and epilepsy, especially in children and adolescents (Etminan [24]; Tzeng, et al. [2015]; Micallef [25]).

Human Gut Microbiota (GM) and Epilepsy

The human gut microbiota (GM) regulates metabolism and the immunological response of the host. Firmicutes and Bacteroidetes are the most abundant phylum, accounting for more than 90% of the GM. Neuropsychiatric and neurodegenerative illnesses may be linked to changes in GM composition. However, the link between GM and epilepsy remains unknown. Only a few population-based studies have found changes in the variety and composition of GM between epilepsy patients and healthy controls. GM analysis has been used to distinguish epileptic patients from healthy people and to distinguish between drug-resistant and drug-sensitive epilepsy. To understand the intricate interaction between GM and epilepsy, more research with bigger sample numbers and controlled factors is required (Thijs, et al; Beghi, et al. [2015]; Beghi, et al. [2019]; Sheng, et al. [2018]; Engel, et al. 7///2018; Kobow, et al. 2018; Kwan, et al. 2010; De Caro, et al. 2019; López González, et al. 2015).

Microbiota–Gut–Brain Axis and Epilepsy

The microbiota-gut-brain axis is an important system that regulates the development and control of a wide range of physiological and pathological processes, including epilepsy. The gut microbiota, a complex collection of bacteria found in the gastrointestinal tract, has an impact on the central nervous system and can have a major impact on brain function and behaviour. Atypical gut microbiota composition is linked to epilepsy-promoting metabolites and inflammatory factors, which disturb the equilibrium of GABA and glutamate neurotransmission. This process can be triggered by chronic stress. A healthy gut microbiota, on the other hand, can create anti-inflammatory and neuroprotective metabolites such as short-chain fatty acids and serotonin. Understanding this dynamic has the potential to lead to the development of innovative treatment techniques, such as targeting the gut microbiota with therapies such as probiotics, prebiotics, or dietary changes (Ding, et al. 2019) (Figure 2).

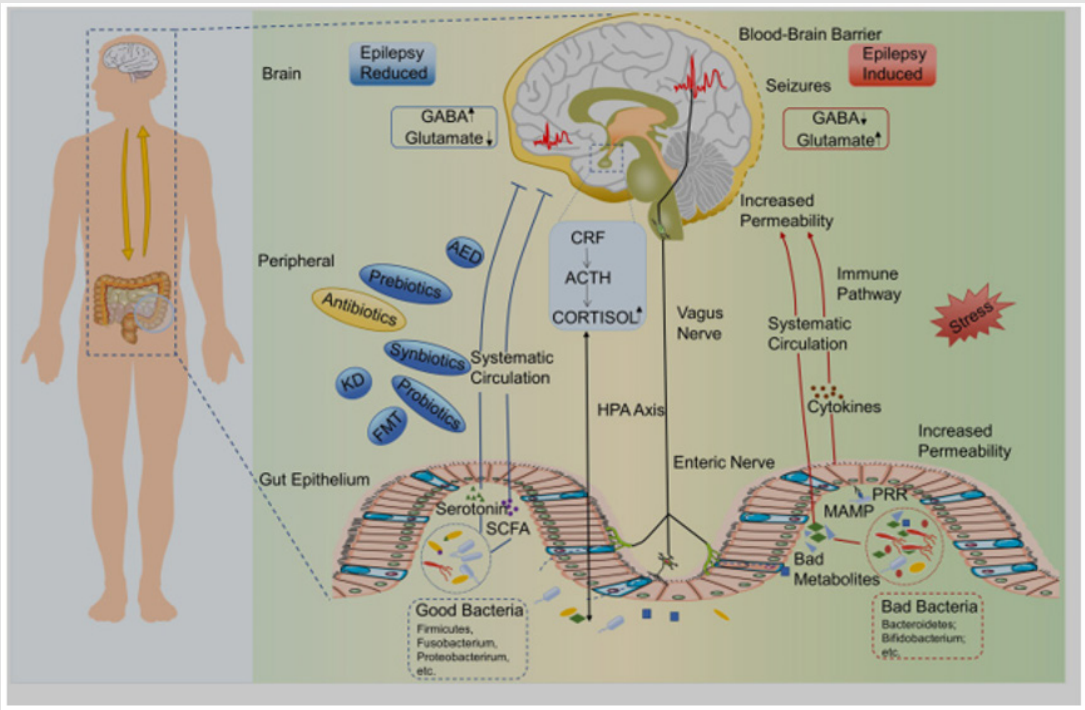

Figure 2: The microbiota-gut-brain axis influences the creation of epilepsy-promoting metabolites and inflammatory factors by gut microbiota, whereas healthy gut microbiota generates beneficial ones, affecting the brain's response (Ding, et al. 2019).

Correlation of Viral Infections and Epilepsy

Viruses can enter the central nervous system (CNS) via peripheral or neuronal pathways. They have the ability to multiply in mononuclear cells, brain endothelial cells, and peripheral neurons. Some viruses are capable of crossing the blood-brain barrier and infecting new neurons. Encephalitis is distinct from aseptic meningitis since it is caused by viral replication within the CNS. Inflammation arises because of direct neuro infection, the production of proinflammatory cytokines, and the activation of innate and adaptive immune responses. This can result in immunopathology and further harm (Kirkpatrick [26,27]).

HIV and Epilepsy Correlation

In patients with advanced HIV infection or AIDS, seizures and epilepsy are common and more prevalent than in the general population. These seizures are typically generalized and can be caused by opportunistic infections, metabolic imbalances, and interactions between antiepileptic and antiviral drugs. However, there is a lack of specific guidelines for the use of antiepileptic drugs in HIV-infected individuals.

Human Herpesvirus 6 (HHV-6)

Human herpesvirus 6 (HHV-6) is a common virus that infects most people during childhood and can establish dormant infections in the central nervous system. Specifically, HHV-6B, a subtype of the virus, has been found to be associated with neurological diseases, particularly epilepsy. In cases of temporal lobe epilepsy (TLE), HHV-6B has been detected in the brains of affected individuals who experience recurrent febrile seizures and hippocampal sclerosis. However, the exact mechanisms through which HHV-6B contributes to the development of TLE are not yet fully understood. It is believed that the virus can directly harm neurons, trigger immune responses, disrupt normal neural circuitry, and promote the proliferation of glial cells. Certain cytokines and signaling pathways, including IL-17A, NF-κB, TGF-β, MAPK, and phospholipase A2, appear to be involved in the pathological processes of TLE. Further research is needed to uncover the precise mechanisms underlying the relationship between HHV-6B and epilepsy and to identify potential biomarkers that can aid in identifying specific patient groups for targeted anti-inflammatory or immunomodulatory treatments (Sellner [28]; Sellner, et al. 2008).

Picornaviruses

Various picornaviruses, including rhinoviruses, enteroviruses, and parechovirus, have been associated with encephalitis and seizures. These neurotropic viruses can infect the peripheral nerve, breach the blood-brain barrier, and invade the central nervous system. Human rhinovirus (HRV) infections have been linked to a higher incidence of seizures, particularly in individuals with underlying neurological abnormalities. Cytokines, such as interleukin-6 and interleukin-1, may play a role in HRV-induced seizures. Similarly, human parechovirus (HPeV) infections have been reported to cause encephalitis with seizure outbreaks, often accompanied by white matter lesions (Britton [29]). Coxsackieviruses and enteroviruses, on the other hand, may contribute to myocarditis and have been associated with seizures in some cases (Muehlenbachs [30]). Hepatitis A virus (HAV) infection has been implicated in seizure activity, as evidenced by the detection of HAV RNA in the cerebrospinal fluid of an infected patient (Lee [31]). In animal models, Theiler's murine encephalomyelitis virus (TMEV) has been shown to induce transient and chronic seizures, along with cognitive impairments and hippocampal cell death. The infiltration of monocytes and granulocytes, as well as the role of cytokines, further contribute to the pathogenesis of seizures in TMEV-infected animals (Bijalwan [32]). These findings highlight the diverse mechanisms by which picornaviruses can induce encephalitis and seizures, emphasizing the need for further research and understanding in this area (Anastasina [33]).

Influenza

Influenza is a highly infectious respiratory virus that kills millions of people each year, with children being especially vulnerable. High temperature, gastrointestinal problems, and neurological consequences such as seizures and encephalopathy are also possible. Seizure history, genetic susceptibility, and pre-existing neurological disorders are all risk factors. Children under the age of five are particularly vulnerable in tropical nations such as Thailand (Chen, et al. [34-36]).

COVID-19

In COVID-19, like other beta-coronaviruses, the virus has the ability to invade the nervous system and cause neurological symptoms (Huang [37]). The angiotensin-converting enzyme 2 (ACE2) receptor serves as the entry point for the virus into human cells, and while primarily found in the brainstem for regulating cardiovascular and respiratory functions, the virus can also enter the brain through the olfactory tract. Once in the central nervous system, the virus triggers reactive astrogliosis and activates microglia, leading to a cascade of inflammatory responses (Wu, et al. [38]). The release of pro-inflammatory cytokines, such as TNF-α, IL-6, and IL-1β, along with the influx of calcium ions and disruption of glutamate-GABA balance, contribute to neuronal hyper-excitability, apoptosis, and chronic inflammation (Huang [37]). The breakdown of the blood-brain barrier (BBB) allows the entry of peripheral cytokines, further exacerbating the inflammatory response. Additionally, fever and hyperthermia associated with COVID-19 can lead to BBB disruption and the release of inflammatory mediators, including cytokines, further increasing neuronal excitability (Samuelsson [39]). Coagulation abnormalities observed in COVID-19 patients, such as DIC, can also contribute to neurological complications. Stroke, as a consequence of vascular damage and blood clotting, can result in post-ischemic seizures and contribute to the development of epilepsy (Postnikova, et al. [40]). The disruption of the BBB, along with the release of cytokines and glutamate, plays

a role in the initiation and progression of epilepsy after stroke. The interplay between inflammation, coagulation, and neuronal hyper-excitability in COVID-19-associated neurological complications underscores the need for further research and understanding to effectively manage and prevent these complications (Tufan [41-47]).

Epstein-Barr Virus (EBV)

EBV is a herpesvirus that causes infectious mononucleosis as well as epilepsy. It has been connected to neurologic problems such as encephalitis, which can result in seizures. The precise processes behind EBV triggers and epilepsy development are unknown. EBV is thought to infiltrate the brain, causing inflammation and neuronal damage. Understanding this link is critical for the early identification, prevention, and management of EBV-related epilepsy, as well as the development of specific therapeutic strategies (Ebell et al., 2016; Cheng, et al. [44]); Doja et al. 2006; Narula et al. 2020).

Flavivirus

Astrocytes, a critical component of the central nervous system, play a role in the immune response to viral infections such as TBEV, WNV, ZIKV, and JEV. In a healthy CNS, these specialized cells maintain homeostasis and synaptic function. When a virus causes encephalitis, astrocytes undergo reactive astrogliosis, a pathogenic feature of CNS structural lesions. Astrogliosis can cause changes in glioneuronal communication as well as seizures. Astrocytes emit pro-inflammatory cytokines and chemokines during neuroinflammation, increasing neuronal excitability and leading to seizures. Flavivirus infections cause morphological and functional alterations in astrocytes, disruption of the blood-brain barrier, and neuroinflammation. Astrocytes also produce vascular endothelial growth factor, interleukin-6, and MMPs, which may contribute to BBB disruption and neuroinflammatory reactions (Potokar [48-50]).

Parasitic Diseases and their Correlation with Epilepsy

Parasitic diseases have been strongly associated with the burden of epilepsy, particularly in low- and middle-income countries. Neurocysticercosis, caused by the larval stage of the tapeworm Taenia solium, is responsible for a significant proportion of epilepsy cases in many parts of Asia, Latin America, and sub-Saharan Africa. Other parasites, both microparasites (such as Plasmodium, Toxoplasma, and Trypanosoma species) and macroparasites (mostly helminths like Toxocara, Onchocerca, Paragonimus, and Schistosoma species), have also been linked to epilepsy. The mechanisms through which these parasites contribute to epileptogenesis may vary. Microparasites can directly invade the brain due to their small size, while macroparasites may rely on the neurotropic properties of their eggs or larvae, or other indirect mechanisms, to predispose individuals to epilepsy. Although some parasites are more extensively studied, there are still others that have received less attention or lack conclusive evidence of brain involvement. It is worth noting that the risk of epilepsy may extend beyond acute brain involvement, as even latent or asymptomatic infections with parasites like Toxoplasma and Toxocara can significantly increase the overall epilepsy burden due to their widespread exposure. Understanding the underlying mechanisms of epileptogenesis in parasitic diseases is crucial for developing interventions aimed at preventing and managing epilepsy associated with these infections (Mazumder [51]).

Conclusion

Epilepsy is a complicated neurological condition with several origins, including hereditary, idiopathic, and acquired causes. Certain medicines, infections, and viral disorders have been linked to epileptogenesis. The epileptogenic potential of antibiotics, viral infections, bacterial infections, and parasite infections was investigated in this article. Although most antibiotics have a low epileptogenic potential, some specific antibiotics, such as cephalosporins and carbapenems, may carry a slightly higher risk of seizures in susceptible individuals [52-65]. Certain medications, such as antidepressants, antipsychotics, and anti-malarial drugs, have been associated with an increased risk of seizures, particularly at higher doses or in predisposed individuals. Viral infections, bacterial infections, and parasitic infections can induce seizures directly or as a result of associated encephalitis or inflammatory response. Understanding the epileptogenic potential of various factors is crucial for accurate diagnosis, appropriate treatment, and better management of epilepsy patients. By identifying and addressing potential epileptogenic factors, clinicians can help to reduce the risk of seizures and improve the quality of life for people with epilepsy.

References

1. Robert S Fisher, Carlos Acevedo, Alexis Arzimanoglou, Alicia Bogacz, J Helen Cross, et al. (2014) ILAE official report a practical clinical definition of epilepsy. Epilepsia 55(4): 475-482.
2. Jones NC, Nguyen TT, Corcoran ME, Velísek L (2016) Convulsant activity of antibiotic trimethoprim sulfamethoxazole alone and with antiepileptics in mice. Epilepsy research 126: 18-23.
3. Ventola (2015) The antibiotic resistance crisis part 1causes and threats. Pharmacy and Therapeutics 40(4): 277-283.
4. Annamaria Vezzani, Robert S Fujinami, H Steve White, Pierre-Marie Preux, Ingmar Blümcke, et al. (2019) Infections inflammation and epilepsy. Acta neuropathologica 137(6): 769-778.
5. MorganDJ, Okeke IN, LaxminarayanR, Perencevich EN, Weisenberg S (2019) Non-prescription antimicrobial use worldwide a systematic review. The Lancet Infectious Diseases 11(9): 692-791.
6. Braun DK, Dominguez G, Pellett PE (1997) Human herpesvirus 6. Clinical microbiology reviews 10(3): 521-567.
7. Young GB (2016) Encephalopathy of infection and systemic inflammation. Journal of Clinical Neurophysiology 30(5): 454-461.
8. VezzaniA, Fujinami RS, White HS, Preux PM, Blümcke, et al. (2016) Infections, inflammation and epilepsy. Acta neuropathologica 131: 211-234.

9. Razavi B, Arshad, RafiqM (2015) An update on the mechanisms underlying the neurotoxicity induced by amphotericin B. Frontiers in microbiology 6: 1412.
10. T Takano, JH Lin, G Arcuino, Q Gao, J Yang, et al. (2014) Glutamate release promotes growth of malignant gliomas. Nature medicine 7(9): 1010-1015.
11. Bhattacharyya, Darpo B, Polli JW, Wang Y, Smolskis, et al. (2016) Safety assessment of fluoroquinolones focus on psychiatric neurological and dermatological reactions. Annals of Pharmacotherapy 50(8): 666-677.
12. Bhat S, AcharyaS, Nagarathna S (2016) Cefepime-induced status epilepticus. Indian journal of critical care medicine peer-reviewed, official publication of Indian Society of Critical Care Medicine 20(9): 559-561.
13. Hwang SH, Kim SY, Kim HJ, Lee SH (2019) Status epilepticus due to piperacillin-tazobactam treatment. BMC neurology 19(1): 181.
14. Li J, Pu, TangG, ZhangY (2015) Seizures and status epilepticus associated with imipenem-cilastatin a case report and literature review. BMC pharmacology toxicology 16: 8.
15. Glickman ME, Gupta M, Lio J (2012) Meropenem-induced status epilepticus. The Journal of emergency medicine 43(1): e21-e24.
16. Carranco E, Kareus J, CoS, Peak V, Al-Rajeh S (1985) Carbamazepine toxicity induced by concurrent erythromycin therapy. Arch Neurol 42(2): 187-188.
17. KeranenT, JolkkonenJ, Jensen PK, Menge GP, AnderssonP (1992) Absence of interaction between oxcarbazepine and erythromycin. Acta Neurol Scand 86(2): 120-123.
18. Green MA, Halliwell RF (1997) Selective antagonism of the GABA(A) receptor by ciprofloxacin and biphenylacetic acid. Br J Pharmacol 122(3): 584-590.
19. Tsutomi Y, Matsubayashi K, Akahane K (1994) Quantitation of GABAA receptor inhibition required for quinoloneinduced convulsions in mice. J Antimicrob Chemother 34(5): 737-746.
20. Thomsen MS, Groes L, Agerso H, Kruse T (1998) Lack of pharmacokinetic interaction between tiagabine and erythromycin. J Clin Pharmacol 38(11): 1051-1056.
21. Uzman S, Uludag Yanaral T, Toptas M, Koc A, Tas A, et al. (2013) Acute isoniazid intoxication: An uncommon cause of convulsion coma and acidosis. Tuberk Toraks 61(1): 50-53.
22. Puri MM, Kumar L, Vishwakarma PD, Behera D (2012) Seizures with single therapeutic dose of isoniazid. Indian J Tuberc 59(2): 100-102.
23. Wang J, Zhou, He F, Ruan Z (2021) Antibiotic exposure and the risk of epilepsy a systematic review and meta-analysis. Seizure 85: 139-146.
24. Mahyar Etminan, JamesM Brophy, Ali Samii (2012) Oral fluoroquinolone use and risk of peripheral neuropathy a pharmacoepidemiologic study. Neurology 79(22): 2294-2299.
25. Micallef C, Farrugia C, FenechA (2017) Antimicrobial stewardship in daily practice managing an essential resource. Frontiers in public health 5: 240.
26. Kirkpatrick M, Finbar Ocallaghan (2022) Epilepsy and cannabis: so near yet so far. Developmental Medicine & Child Neurology 64(2): 162-167.
27. Annamaria Vezzani, Robert S Fujinami, H Steve White, Pierre-Marie Preux, Ingmar Blümcke (2016) Infections, inflammation and epilepsy. Acta neuropathologica 131: 211-234.
28. Sellner, TrinkaE (2012) Seizures and epilepsy in herpes simplex virus encephalitis current concepts and future directions of pathogenesis and management. Journal of neurology 259(10): 2019-2030.
29. Philip N Britton, Cheryl A Jones, Kristine Macartney, Allen C Cheng (2018) Parechovirus an important emerging infection in young infants. Medical Journal of Australia 208(8): 365-369.
30. Muehlenbachs A, Bhatnagar, Zaki SR (2015) Tissue tropism pathology and pathogenesis of enterovirus infection. The Journal of pathology 235(2): 217-228.
31. Lee J, Kang K, Park M, Kwon, Kim BK (2011) Encephalitis associated with acute hepatitis a. Journal of Epilepsy Research 1(1): 27-28.
32. Bijalwan M (2017) Pathogenesis of Theiler's Murine Encephalomyelitis Virus (TMEV) in an Experimental Model of Epilepsy (Doctoral dissertation).
33. Anastasina M, Domanska A, Palm K, Butcher S (2017) Human picornaviruses associated with neurological diseases and their neutralization by antibodies. Journal of General Virology 98(6): 1145-1158.
34. Chen Q, Li P, LiS, Xiao W, Yang S, et al. (2020) Brain complications with influenza infection in children. Journal of Behavioral and Brain Science 10(3): 129-152.
35. Lochindarat S, Bunnag T (2009) Clinical presentations of pandemic 2009 influenza A (H1N1) virus infection in hospitalized Thai children. J Med Assoc Thai 94(Suppl 3): S107- S112.
36. Mastrolia MV, Rubino C, Resti M, Trapani S, Galli L (2019) Characteristics and outcome of influenza-associated encephalopathy encephalitis among children in a tertiary pediatric hospital in Italy. BMC Infect Dis 19(1): 1012.
37. Hwang SH, Kim SY, Kim HJ, Lee SH (2019) Status epilepticus due to piperacillin-tazobactam treatment. BMC neurology 19(1): 181.
38. Wu Y, et al. (2020) Nervous system involvement after infection with COVID-19 and other coronaviruses. Brain Behavior and Immunity.
39. Anne-Maj Samuelsson, Eva Jennische, Hans-Arne Hansson, Agneta Holmäng (2006) Prenatal exposure to interleukin-6 results in inflammatory neurodegeneration in hippocampus with NMDA/GABAA dysregulation and impaired spatial learning. American Journal of Physiology-Regulatory Integrative and Comparative Physiology. 290(5): R1345-R1356.
40. Postnikova T, et al. (2017) Status epilepticus impairs synaptic plasticity in rat hippocampus and is followed by changes in expression of NMDA receptors. Biochemistry (Moscow) 82(3): 282-290.
41. Abdurrahman Tufan, Aslihan Avanoğlu Güler, Marco Matucci-Cerinic (2020) COVID-19 immune system response hyperinflammation and repurposing antirheumatic drugs. Turkish Journal of Medical Sciences 50(SI-1): 620-632.
42. Rana A, Musto AE (2018) The role of inflammation in the development of epilepsy. Journal of neuroinflammation 15(1): 144.
43. Alyu F, Dikmen M (2017) Inflammatory aspects of epileptogenesis contribution of molecular inflammatory mechanisms. Acta neuropsychiatrica 29(1): 1-16.
44. B Viviani, S Bartesaghi, F Gardoni, A Vezzani, MM Behrens, et al. (2003) Interleukin-1β enhances NMDA receptor-mediated intracellular calcium increase through activation of the Src family of kinases. Journal of Neuroscience 23(25): 8692-8700.
45. Cristina Roseti, TY Postnikova, OE Zubareva, AA Kovalenko, KK Kim, et al. (2017) Status epilepticus impairs synaptic plasticity in rat hippocampus and is followed by changes in expression of NMDA receptors. Biochemistry (Moscow) 82(3): 282-290.
46. Stellwagen D, Malenka RC (2006) Synaptic scaling mediated by glial TNF-α. Nature 440(7087): 1054-1059.
47. Galic MA, Riazi K, Pittman QJ (2012) Cytokines and brain excitability. Frontiers in neuroendocrinology 33(1): 116-125.

48. Maja Potokar, Jernej Jorgačevski, Robert Zorec (2019) Astrocytes in Flavivirus Infections. Int J Mol Sci 20(3): 691.

49. Zheng M, Li S, Hogan RE, Yang M (2020) Arbovirus and seizures. Acta Epileptol 2: 17.

50. Ashraf U, Ding Z, Deng S, Ye J, Cao S, et al. (2021) Pathogenicity and virulence of Japanese encephalitis virus: neuroinflammation and neuronal cell damage. Virulence 12(1): 968-980.

51. Mazumder R, Lee JK (2022) Epileptogenesis in common parasitic infections. Current Neurology and Neuroscience Reports 22(4): 285-291.

52. Wang XuePing, Wang HaiJiao, Zhu LiNa, Da Xu, Liu Ling (2019) Antibiotic use and the risk of epilepsy A systematic review and meta-analysis. Epilepsy research 98(30): e16402.

53. Wang XuePing, Wang HaiJiao, Zhu LiNa, Da Xu, Liu Ling (2021) Antibiotic exposure and the risk of epilepsy A systematic review and meta-analysis.

54. T Takano, JH Lin, G Arcuino, Q Gao, J Yang, et al. (2017) Antimicrobial stewardship in daily practice: managing an essential resource. Frontiers in public health 25(5): 241-245.

55. Nian-Sheng Tzeng, Chi-Hsiang Chung, Fu-Huang Lin, Chien-Ping Chiang, Chin-Bin Yeh, et al. (2018) Anti-herpetic medications and reduced risk of dementia in patients with herpes simplex virus infections-a nationwide population-based Cohort Study in Taiwan. Neurotherapeutics 15(2): 417-429.

56. Ventola (2015) The antibiotic resistance crisis part 1causes and threats. Pharmacy and Therapeutics 40(4): 277-283.

57. Bichler EK, Elder CC, García PS (2017) Clarithromycin increases neuronal excitability in CA3 pyramidal neurons through a reduction in GABAergic signaling. Journal of neurophysiology 117(1): 93-103.

58. Young GB (2016) Encephalopathy of infection and systemic inflammation. Journal of Clinical Neurophysiology 30(5): 454-461.

59. Mingrui Zheng, Shichuo Li, R Edward Hogan, Meihua Yang (2020). Arbovirus and seizures. Acta Epileptol 2: 17.

60. Chaolin Huang, Yeming Wang, Xingwang Li, Lili Ren, Jianping Zhao, et al. (2020) Clinical features of patients infected with novel coronavirus in Wuhan China. The lancet 395(10223): 497-506.

61. Sanchez Valiente S (1995) Myoclonic encephalopathy induced by diclofenac treatment. Revista de Neurología 23(124): 1226-1227.

62. Sellner, Trinka (2012) Seizures and epilepsy in herpes simplex virus encephalitis current concepts and future directions of pathogenesis and management. Journal of neurology 259(10): 2019-2030.

63. Misra UK, Tan CT, Kalita J (2008) Viral encephalitis and epilepsy. Epilepsia 6: 13-18.

64. Wei Y, Wang J, Xia Y, Liu, Huang, et al. (2020) Antibiotic exposure and risk of epilepsy a systematic review and meta-analysis. Epilepsy research 163: 106327.

65. Nian-Sheng Tzeng, Chi-Hsiang Chung, Fu-Huang Lin, Chien-Ping Chiang, Chin-Bin Yeh, et al. (2015) Anti-herpetic medications and reduced risk of dementia in patients with herpes simplex virus infections a nationwide population-based cohort study in Taiwan. Neurotherapeutics 15(2): 412-429.

ISSN: 2574-1241

DOI: 10.26717/BJSTR.2023.53.008376

Shahzar Khan. Biomed J Sci & Tech Res