

## Epilepsy Unveiled: Advances in Understanding, Diagnosis, and Management

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### ABSTRACT

In addition to the numerous stress that characterize individuals today's fast-paced lives, the majority of people on the planet encounter various neurological problems. Epilepsy, one of the most common neurological disorders of the brain, affects over 50 million people worldwide. 90% of whom are from developing nations. Genetic factors, brain infections, strokes, tumors, and high body temperatures are all contributors to epilepsy. Along with the social stigma that brings discrimination against patients and even their families in the community, it also, creates a significant financial burden on the healthcare systems of different countries. People with epilepsy frequently experience extreme mental suffering, behavioral problems, and little to no social engagement. Numerous seizure types exist, as are numerous mechanisms by which the brain can trigger seizures. Neuronal hyperexcitability and neural circuit hypersynchrony are the two fundamental features of seizure genesis. Numerous mechanisms may disturb the equilibrium between excitation and inhibition, predisposing a specific or generalized region of the brain to hyperexcitability and hypersynchrony. The review will include the classification, background, epidemiology, etiology, pathophysiology, symptoms, diagnosis, and management of epilepsy.

**Key words:** Epilepsy, Seizure, Anti-epileptic Drug, Pathophysiology

Epilepsy is a widespread chronic neurological illness that affects up to 1% of the population, making it the second most prevalent serious neurologic disorder after stroke [1]. It affects around 50 million individuals worldwide, with 90% of those affected living in underdeveloped nations [2]. Epilepsy is defined by recurrent unprovoked 3-5 seizures, with substantial changes in the biology and consequences of seizures between the immature and mature brain [3]. It refers to a variety of seizures that vary in severity, appearance, cause, effect, and management. Epilepsy frequently causes brief impairments in consciousness, putting people in danger and interfering with schooling and jobs.

It has no age, gender, geographical, social class, or racial boundaries. Epilepsy is more common in young children and those over the age of 65, but it can develop at any age [4]. Seizure onset can be focal, generalized, or unknown, and can be classified based on awareness or motor or nonmotor aspects. Active epilepsy is defined by regular treatment with antiepileptic medications or the most recent seizure occurring within the last 5 years [5]. A condition known as Status Epilepticus (SE) is a prolonged or repeated seizure that can lead to long-term consequences, including neuronal injury or death. A new diagnostic classification for SE has been proposed [6]. Sudden unexpected death in epilepsy (SUDEP) is defined as a sudden, unexpected, witnessed or unwitnessed death in epileptic patients, with or without seizure evidence, and excluding established SE.

#### Access this article online

Received – 30<sup>th</sup> Oct 2023

Initial Review – 16<sup>th</sup> Nov 2023

Accepted – 07<sup>th</sup> Dec 2023



Quick Response Code

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Seizures cause SUDEP, which may include seizure-induced cardiorespiratory changes. The frequency of epilepsy is determined by its incidence, prevalence, and mortality, whereas the burden is determined by disability-adjusted life-years (DALYs), years of life lost, and years of living with disability [7]. Conventional treatment generally consists of anticonvulsant drugs, however even with the finest current treatments; more than 30% of persons with epilepsy do not have seizure control [8]. Therapy is symptomatic because existing medications reduce seizures, but there is no effective preventive or cure. Medication adherence is a big issue due to the long-term negative effects of many medicines [9].

**History of Epilepsy:** Epilepsy, originating from the Greek term "epilepsia," which means "to seize," has long been tied with religion and demon possession. Epilepsy was once thought to be a sacred sickness, with many believing that it affected those who were abducted by demons or that their visions were sent by the gods. Epilepsy was considered a demonic spirit attack in Hmong generations, although affected persons could become honored as shamans as a result of their explicit experiences [10].

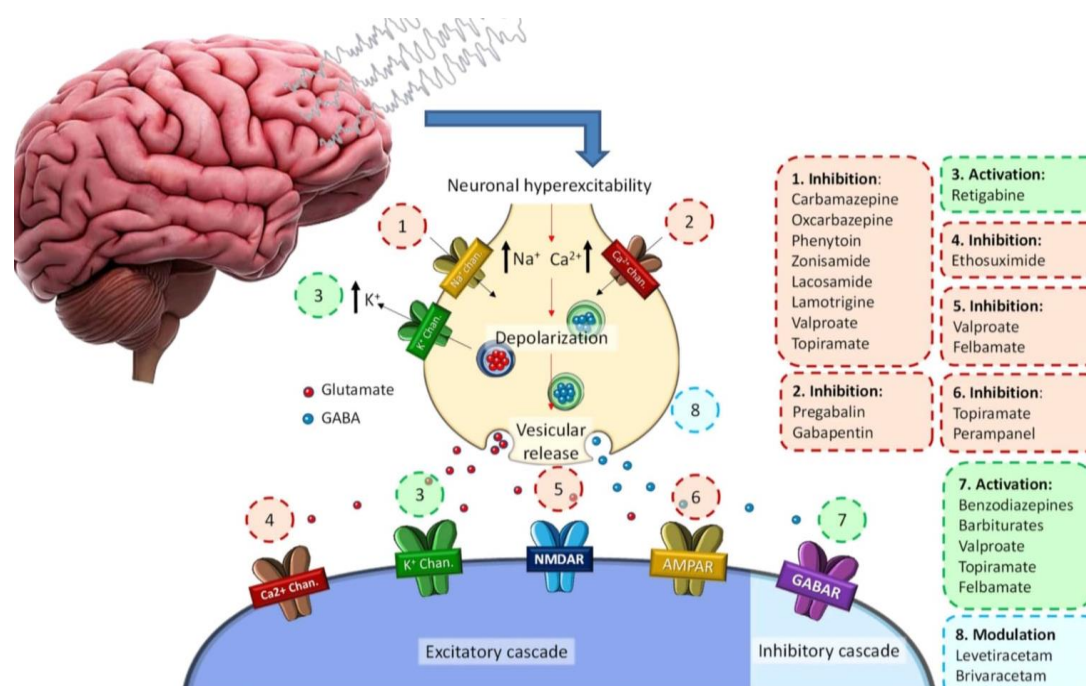
Epileptic people were scorned and even imprisoned in most societies. Jean-Martin Charcot noted that epileptic persons were mentally retarded and criminally insane in the Salpetriere, the birthplace of contemporary neuroscience. People in Tanzania believed epilepsy was caused by bad spirits, witchcraft, poisoning, or was communicable. Epilepsy was considered a god-given curse in Rome, known as *Morbus comitialis* [11]. Stigma continues to this day, but it is gradually decreasing in developed countries. Hippocrates predicted that it would not take much time to eradicate epilepsy as it is not divine.

**Epidemiology:** Epilepsy is a common neurological illness, affecting an estimated 55 lakhs individuals in India, 20 lakhs in the United States, and three lacks in the United Kingdom. In the United States, 120 persons out of every 100,000 have a seizure each year, with a recurrence rate ranging from 23% to 80%. The annual age-adjusted incidence of epilepsy is 44 per 100,000 persons. Every year, around 125,000 new cases are diagnosed, with 30% of those diagnosed being under the age of 18. The elderly have a high prevalence of epilepsy, which is now acknowledged. At least 10% of patients in long-term care facilities are on at least six antiepileptic medications. The National Sentinel Audit of Epilepsy-Related fatalities highlights the problem, indicating that 1,000 people die in the UK each year as a result of epilepsy, with 42% of these fatalities being avoidable [12].

**Causes of Epilepsy:** Epilepsy is a disorder in which the cause or severity of seizures is not directly connected. Some cases are genetic, while others are the result of brain traumas, strokes, infections, high fever, or malignancies [13]. Many incidences of epilepsy in young children are caused by heredity, but it can affect people of any age. Specific precipitants or triggers, such as reading, flashing lights, mental stress, sleep deprivation, heat stress, alcohol, and febrile sickness, are required for reflex epilepsy disorders [14]. The impact of these precipitants varies depending on the epileptic syndrome. In epileptic women, the menstrual cycle can also alter seizure recurrence patterns. The most prevalent causes of epilepsy in infants and early infancy are hypoxic-ischemic encephalopathy, CNS infections, trauma, congenital CNS abnormalities, and metabolic problems. CNS infections and trauma can cause febrile seizures in late infancy and early childhood. Cerebrovascular disease is the most common cause of death in the elderly, followed by CNS malignancies, head trauma, and degenerative disorders such as dementia [15].

**Pathophysiology of Epilepsy:** Seizures are paroxysmal manifestations of the cerebral cortex caused by a transient imbalance in excitatory and inhibitory forces in the cortical neuron network. The seizure event is recognized in an unstable cell membrane or its surrounding cells, arising from either cortical or subcortical area's gray matter. A small number of neurons fire inappropriately at first, and normal membrane conductance and inhibitory synaptic current breakdown, as well as excess excitability, spread either locally to cause a focal seizure or more broadly to produce a generalized seizure. This onset spreads through physiologic pathways to include nearby and remote locations [16].

A failure in potassium conductance, a defect in voltage-activated ion channels, or a shortage in membrane ATPases involved in ion transport can all lead to neuronal membrane instability and a seizure. Certain neurotransmitters, such as glutamate, aspartate, acetylcholine, norepinephrine, histamine, corticotropin-releasing factor, purines, peptides, cytokines, and steroid hormones, increase neuronal excitability and propagation, whereas -amino butyric acid (GABA) and dopamine decrease neuronal excitability and propagation [18]. During a seizure, the demand for blood flow to the brain increases in order to remove CO<sub>2</sub> and bring substrate for neuronal metabolic activity. Some types of epilepsy may be connected to mutations in many genes.



**Figure 1: Pathophysiology of Epilepsy and Understanding Seizure Mechanisms [17]**

**Diagnosis:** A number of different tests have been developed to determine the epilepsy in an individual and its type. This may include a neurological exam that examines behavior, emotions, and mental function to diagnose and classify epilepsy. Blood tests look for evidence of infections, genetic disorders, or other seizure-related illnesses. Genetic testing, which is commonly performed in children but can also benefit some adults with epilepsy, can provide more information about the condition and therapy [19].

Some brain imaging tests and scans that detect changes are-

**Electroencephalogram (EEG) Monitoring:** Electron encephalograms are extremely helpful in the identification of many seizure disorders. Even if the EEG is normal in some people, they still have the clinical diagnosis of epilepsy. Many people who do not have epilepsy have atypical brain activity. Video monitoring is frequently used in conjunction with EEG to establish the type of seizures a person has [20].

**High-density EEG:** In this test, electrodes are put closer together than in a traditional EEG. High-density EEG may aid in pinpointing which parts of the brain are affected by seizures [21].

**Computerized tomography (CT) scan:** CT scans use X-rays to create cross-sectional images of the brain, which can help detect tumors, bleeding & cysts that cause epilepsy [22].

**Magnetic resonance imaging (MRI):** An MRI, like a CT scan, employs powerful magnets and radio waves to provide a precise image of the brain in order to detect future seizures, but it provides a more detailed view than a CT scan [23].

**Functional MRI (fMRI):** A functional MRI measures blood flow changes in brain parts, aiding in identifying critical functions like speech & movement before surgery, enabling surgeons to avoid these areas during the procedure [24].

**Positron emission tomography (PET):** PET scans use low-dose radioactive material injected into veins to visualize brain metabolic activity and detect changes, potentially identifying low metabolism areas as seizures [25].

**Single-photon emission computerized tomography (SPECT):** A SPECT test uses low-dose radioactive material to create a 3D map of blood flow during seizures, indicating seizure locations. SISCOM, or subtraction ictal SPECT connected to MRI, overlaps SPECT results with brain MRI results for more detailed results [26]. Other techniques, such as Statistical Parametric Mapping (SPM), Electrical Source Imaging (ESI), and Magnetoencephalography (MEG), can be used to detect seizures. SPM contrasts locations with increased blood flow during seizures versus those without seizures. For a more thorough picture of seizures, ESI projects EEG data onto an MRI. MEG detects magnetic fields generated by brain activity, resulting in more accurate results due to reduced

interference from the skull and surrounding tissue. These approaches, when combined, provide images of areas

impacted and not affected by seizures, allowing for a more complete knowledge of the brain's origins [27].

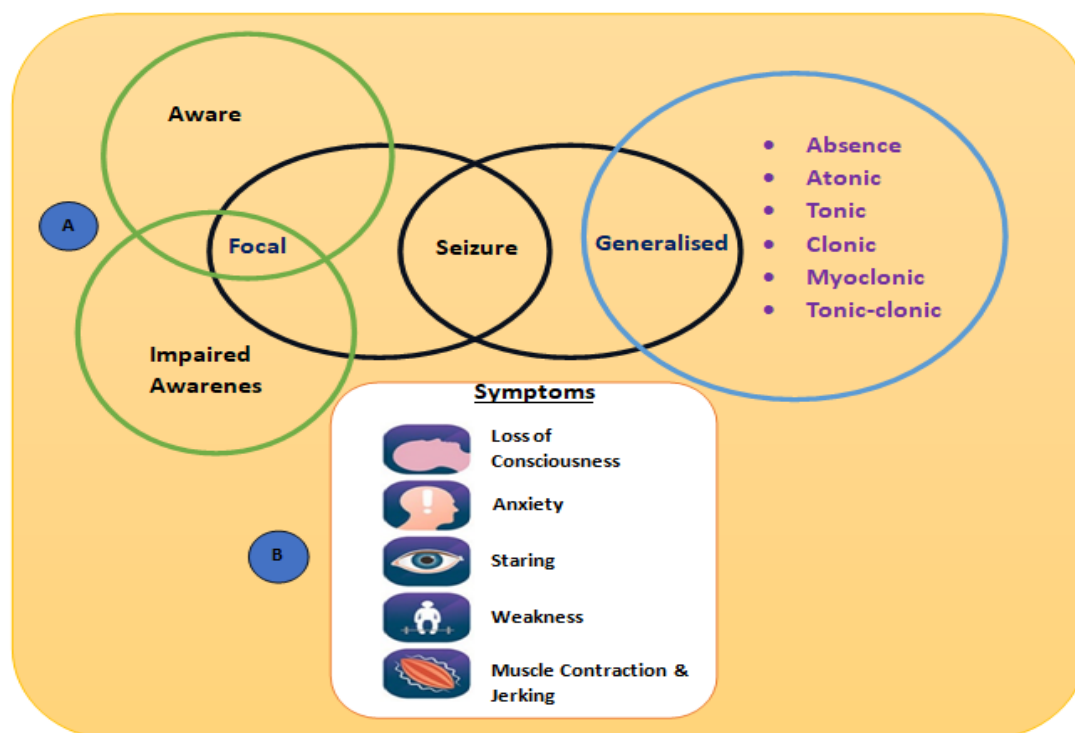


Figure 2: A) Types of Seizure B) Symptoms of Seizure [28].

Table 1: Classification of Drugs use in therapy of epilepsy [29]

| AED                     | Class of Drug   | Mechanism of Action   | Uses   | Advantages   | Disadvantages   |
|-------------------------|-----------------|---|--|--|---|
| Pregabalin              | Anticonvulsants | inhibiting calcium influx and subsequent release of excitatory neurotransmitters.   | Treat epilepsy, anxiety and neuropathic pain.                | <b>Add on efficacy:</b> partial onset seizures, no drug interactions. No hypersensitivity skin reactions. Work also in neuropathic pain and generalized anxiety disorders. | Weight gain. Increased side effect risk in patients with low glomerular filtration rate requires a lower dose. Seizure aggravation in idiopathic generalized epilepsy (absence or myoclonic seizures).  |
| Phenobarbital/primidone | Barbiturate     | Prolonged and frequency of GABA mediated chloride channel opening. Blockade of AMPA receptors.  | Anti-seizure, Anti-epileptic, treat insomnia, treat anxiety. | <b>Efficacy:</b> partial-onset seizures and generalized myoclonic seizures. Rash is uncommon. Phenobarbital is widely available and inexpensive parenteral formulation.    | Drug interactions(may lower the efficacy of concomitant medications metabolized by the P450 hepatic enzyme system). Sedation, cognitive slowing, arthralgia   |
| Phenytoin               | Anticonvulsants | Voltage gated sodium channel blocker, keep maintain the sodium channel's inactive condition and extending the neuronal refractory period. | Anti-epileptic.  | <b>Efficacy:</b> partial-onset seizures. Long accumulated experience. Can be loaded orally or intravenously (intravenous phenytoin or the prodrug fosphenytoin).           | Drug interaction (through P450 enzyme induction and extensive protein binding). Nonlinear pharmacokinetics (small changes in dose or bioavailability may produce large fluctuations in level). Ataxia, rash. Seizures aggravation in idiopathic generalized epilepsy (absence or myoclonic seizures). |



|            |   |   |   |   |  |
|------------|---|---|---|---|--|
| Rufinamide | Anticonvulsants                                 | It is a triazole derivative antiepileptic which prolong the inactive state of voltage gated sodium channel.   | Control Seizure.  | <b>Efficacy:</b> Lennox-Gastaut syndrome.   | Drug interactions (clearance decreased by valproate and increased by enzyme inducers, may reduce efficacy of oral contraceptives).   |
| Tiagabine  | Anticonvulsants                                 | Inhibit the reuptake of GABA into presynaptic neurons and increase the amount of GABA to postsynaptic neurons.                                      | Treat partial seizure in epilepsy.                        | <b>Efficacy:</b> partial-onset seizures. Does not affect other AEDs. Relatively favorable cognitive profile   | Only indicated as adjunctive therapy. Requires a slow titration, given three or four times daily. Can cause nonconvulsive status epilepticus or encephalopathy that resembles nonconvulsive status epilepticus, even in the absence of prior epilepsy. |
| Topiramate | Second generation antiepileptic drug.           | It blocks voltage gated sodium channels. Increasing GABA activity and inhibit glutamate activity. Inhibition of kinetically evoked currents.        | Manage and treat epilepsy and migraine.                   | <b>Efficacy:</b> partial-onset seizures and generalized seizures. Rash is uncommon. Efficacy against migraine. Weight loss.   | <b>Weight loss:</b> aphasia and cognitive impairment, nephrolithiasis, metabolic acidosis, hypohidrosis. Requires slow titration rate because of adverse cognitive effects   |
| Valproate  | Anticonvulsants                                 | Blocks voltage gated ion channel and increase the inhibitory neurotransmission.   | Treat epilepsy and bipolar disorder.                      | Wide spectrum of efficacy against partial-onset seizures and generalized seizures. No hypersensitivity skin reactions. Work for bipolar disorder and migraine. Intravenous preparation. | <b>Weight gain:</b> encephalopathy, tremor, Parkinsonian syndrome. Teratogenicity and permanent adverse cognitive outcomes in fetus. Drug interaction (due to inhibition of P450 enzymes and extensive protein binding).                               |
| Vigabatrin | Anti-epileptic/Anticonvulsants                  | Inhibit GABA degraded enzyme GABA-Transaminase, increase GABA concentration in the brain  | Treat refractory complex partial seizure.                 | Add-on efficacy for partial-onset seizures and West syndrome. No interactions.  | Concentric visual field defects, irreversible.   |
| Zonisamide | Sulfonamide anti-epileptic drug/Anticonvulsants | Block voltage sensitive Sodium channel and T-type Calcium channel. Enhancement of GABAergic transmission and inhibition of glutamatergic transmiss. | Treat partial onset seizure in the treatment of epilepsy. | <b>Efficacy:</b> partial-onset seizures; generalized-onset seizures (evidence not rigorous). Long half-life (allows once daily dosing). Weight loss                                     | Weight loss, aphasia and cognitive impairment, nephrolithiasis, metabolic acidosis; anhidrosis in children (fever).  |

**Blood Biomarkers in Epilepsy:** Biochemical marker advancements can identify brain pathology, increasing hope in epilepsy. Although connectivity/resting state imaging and gadgets such as smartwatches and implantable EEG can detect epilepsy, their cost and impracticality limit their usage in larger patient populations. Blood testing may be a more scalable method of diagnosing epilepsy and seizure burden. Pilot studies have shown feasibility, and trials with acute brain illnesses with a high risk of epileptogenesis could be used as well. Biomarkers of disease activity, such as NT-proBNP or HBA1c, may help in illness management and intervention [30].

**Neuronal/brain biomarkers:** Neuronal/brain biomarkers like S100B, NSE, GFAP, NfL, Tau, UCHL-1, and MMP-9

are crucial for identifying epileptic pathophysiological changes. S100B, primarily expressed in astrocytes, is linked to poststroke epilepsy. GFAP, NSE, NfL, and UCHL-1 are linked to neurodegenerative illnesses. Elevated serum concentrations in epilepsy patients are detected in plasma. Further research is needed to determine their clinical use and role in epilepsy [31].

**Neuroinflammatory biomarkers:** Neuroinflammatory indicators, such as cytokines like IL-1, IL-2, and IL-4, are potential biomarkers for epilepsy. Elevated interleukins (IL) and plasma IL-6 levels are associated with long-term seizures. Individuals with epilepsy have higher levels of IFNs, TNFs, CCL17, and other neuroinflammatory mediators. These factors contribute to inflammation and seizure development [32].

**Epilepsy and Oral Contraceptives:** Antiepileptic medications such as carbamazepine, phenytoin, barbiturates, topiramate, and Oxcarbazepine can result in hormonal contraception failure, requiring patients to use a higher estrogen-containing oral contraceptive (50 mg/mg/day) [33].

**Pregnancy and Epilepsy:** Pregnancy epilepsy can lead to difficulties for both the mother and the fetus, increasing the chance of spontaneous abortion and stillbirth. To avoid anoxiosis and metabolic abnormalities, it is critical to manage the illness effectively and cure it before pregnancy. Minor seizures should not be eliminated, and patients should take folic acid and vitamin K supplements orally. Some antiepileptic medicines can interfere with folic acid metabolism, resulting in neural tube abnormalities. Hepatic enzyme-inducing medications can also cause postpartum bleeding by lowering the mother's vitamin K levels [34].

**Breastfeeding:** Antiepileptics are normally present in low amounts in breast milk, making breastfeeding safe when administered in standard doses. However, benzodiazepines and barbiturates, which are found in high concentrations in breast milk, might cause newborn drowsiness, posing a risk to breastfeeding [35].

**Epilepsy in Children:** Children with epilepsy who have fits are treated similarly to adults, but they may react differently and be unpleasant. If febrile convulsions occur, a significant epilepsy medicine may be administered consistently until the kid reaches the age of five. Because prolonged therapy may interfere with cognitive development, the medicine is discontinued [36].

**Future Directions in Research and Treatment:** Epilepsy research is evolving towards preventive and curative strategies, emphasizing a shift from symptom control. Recent breakthroughs involve identifying mutated genes in inherited epilepsy, characterizing brain networks at the molecular level, enhancing seizure origin imaging, and advancing quantitative EEG analysis for seizure prediction. Therapeutic advancements focus on new molecular targets, EEG-tailored drug delivery, gene/cell therapy, and innovative surgical/non-ablative approaches [37].

Integrated methods, like combining imaging with electrophysiology, play a central role in localizing epilepsy development and improving prediction and treatment. Speculative approaches explore preventing epileptogenesis through cellular phenotype modulation, investigating protective factors, and understanding the fate of

endogenous neural stem cells in the mature CNS. Novel pharmacotherapies, biosensor-coupled delivery systems, gene/cell therapy, and progressive surgical methods represent emerging directions for epilepsy therapy. These advancements collectively contribute to a comprehensive understanding and treatment of epilepsy, providing a foundation for further research and breakthroughs [38].

**Challenges in new drug development:** Epilepsy research and treatment encounter multifaceted challenges, including the diverse origins of the condition, hindrances in identifying reliable biomarkers for early diagnosis, and the emergence of drug resistance in some patients. The complex interplay of epilepsy with comorbidities necessitates a holistic patient care approach, while the incomplete understanding of underlying mechanisms impedes the development of targeted therapies [39]. Societal stigma surrounding epilepsy and its impact on individuals' quality of life constitute significant hurdles. Furthermore, global discrepancies in accessibility to specialized epilepsy care underscore the need for more equitable healthcare distribution. Emerging research pointing to neuroinflammation and immune system involvement adds another layer of complexity, calling for further exploration and potential anti-inflammatory strategies [40].

Addressing these challenges requires collaborative efforts involving diverse stakeholders, an increase in funding to support comprehensive research endeavors, and a multidisciplinary approach to advance both the understanding and treatment of epilepsy. The integration of knowledge from various fields, coupled with innovative strategies, is pivotal for overcoming these challenges and improving outcomes for individuals affected by epilepsy.

## CONCLUSION

Epilepsy is a complex condition that affects people of all ages, with a special preference for children and the elderly. Its causes range from monogenic to cortical acquired diseases and its severity varies from patient to patient. Despite different treatments, public health surveys reveal that many people have a low quality of life as a result of the impact of recurring seizures on everyday activities. Thus, the selection of an anticonvulsant medication is mostly based on its efficacy for specific types of seizures and epilepsy. Despite adequate seizure control, a considerable proportion of epilepsy patients have intractable or drug-resistant epilepsy, necessitating the development of novel medications with better side effects and tolerance profiles, even at the expense of efficacy, when compared to existing antiepileptic therapies.

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**How to cite this article:** Shrijita Sikdar, Udit Dutta, Ayan Ghosh, Suparna Mondal, Krisnendu Das. *Epilepsy Unveiled: Advances in Understanding, Diagnosis, and Management*. *Indian J Pharm Drug Studies*. 2023; 2(4):148-155.

*Funding: None*

*Conflict of Interest: None Stated*