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Integrated Strategies in Epilepsy Management: Bridging Pharmacological and Non-pharmacological Therapies

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Abstract

Epilepsy is characterised by unprovoked seizures arising from disproportionate firing of brain neurons, accompanied by brief lapses of sudden muscular contractions with or without the loss of consciousness. It is among the most prevalent neurological diseases in children and adults. Etiology ranges from premature birth, fever, infections such as meningitis, maternal complications, structural brain abnormalities, and several others. Epilepsy is primarily managed by two methods: i) Pharmacological management and ii) Non-Pharmacological management. Epilepsy is managed non-pharmacologically through various approaches, including first aid, acupuncture, deep brain stimulation, artificial intelligence, neurostimulation, ketogenic diets, etc. Conversely, pharmacological management by administering anti-epileptic drugs as first-line therapy, evaluation of epigenetic parameters of the disorder and intervention of the mTOR pathway. Conditions such as drug-resistant epilepsy is treated by dispensing conventional AEDs or by either of the non-pharmacological management methods.

Keywords: Seizures, meningitis, neurostimulation, ketogenic diets, epigenetics, mTOR pathway, anti-epileptic drugs.

1. Introduction

Epilepsy is a most prevalent neurologic disorder globally (Sireesha V, Nikshitha M 2025). Epilepsy is distinguished by dysrhythmia in the cerebral cortex. It occurs as short episodes

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(seizures) of dissipation of consciousness with or without convulsions, a sensory or psychiatric phenomenon (Tripathi KD 2016). Epilepsy may be caused by genetic issues, head trauma, stroke, brain infection, meningitis or any other cause (Ester Maria de Almeida Costa, Brenda Martins da Silva 2023).

Epilepsy is estimated to affect 50 million people universally. Studies have shown that epilepsy prevails between 4 to 10 per 1000 people; hence, it is one of the most common neurodegenerative disorders (Chen Z, Brodie MJ 2023). Epilepsy accounts to tremendous incidence rate and death due to seizures (Aayushi Umesh Soni, Yash Sunilkumar Lodha 2025).

A person is concluded to be epileptic only when more than two unstimulated seizures occur whose cause is not related to any other health conditions, such as fever, etc. Seizures are of various types, which are represented by behavioral changes and electrophysiological disturbances (Bazil CW, Morrell MJ, 2005). It is a transient epileptic event which indicates improper disruption of brain function (Goldenberg MM 2010). International League Against Epilepsy (ILAE) classifies epilepsy into the following categories:

1.1 Partial seizures

Further classified into two types: i) seizures with loss of consciousness and ii) seizures without loss of consciousness. These may further develop into generalized seizures.

1.2 Simple partial seizures

Occurs without the loss of consciousness and subsequent realization of the event that took place. The focus for such seizures is the primary motor cortex. Sensations of these seizures include tingling, heat and cold, and pins and needles.

1.3 Complex partial seizures

Includes partial loss of consciousness with slight awareness of what happened and inability of the patient to respond. Consists of a peculiar feeling arising from the stomach to the head or a sensation of smell, light, etc.

1.4 Generalised seizures

Complete loss of consciousness and lack of peculiar feeling are characteristics of these seizures. Generalised tonic-clonic seizures also known as GTCS, are the most prevalent. Include petit-mal seizures, myoclonic seizures, tonic seizures and clonic seizures (Aayushi Umesh Soni, Yash Sunilkumar Lodha 2025).

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Although the cause of the epilepsy is unknown, by possessing knowledge about its pathophysiology, the underlying mechanism has been concluded in recent decades. The disparity between the excitation caused by glutamatergic signaling and inhibition which arises due to the GABAnergic signaling of the activity of neurons which has a vital function in its pathophysiology. The prospective ways of treating epilepsy are various but are majorly managed by the use of antiepileptic drugs, whose mechanism of action is classified into the following three categories: 1) augmentation of inhibitory transmission, 2) reduction in excitatory transmission and 3) obstruction of voltage-gated ion channels (Domingo Sánchez, Jorge Gómez-Carpintero, 2024). Table 1 refers to the classification of anti-epileptic drugs. The various other approaches for the treatment of epilepsy include the use of brivaracetam, artificial intelligence, deep brain stimulation therapy, medical cannabis, ketamine and several others.

Table 1: Classification of Anti-epileptic drugs (Mukhopadhyay, Hirak & Kandar 2012).

Chemical Class	Examples of antiepileptic drug
Barbiturates	Phenobarbitone, Mephobarbitone, Primidone
Hydantoins	Phonations, Mephenytoin
Iminostilbene	Carbamazepine
Oxazolidinedione	Trimethadione (Troxidone)
Succinimide	Ethosuximide
Aliphatic Carboxylic acid	Valproic acid (Sodium valproate)
Benzodiazepines	Clonazepam, Diazepam
Acetyl urea	Phenacemide
Newer drugs	Progabide, Vigabatrin, Gabapentin Lamotrigine, Felbamate, Topiramate, Tiagabine
Miscellaneous	Acetazolamide, Dexamphetamine

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2. Management of Epilepsy

The management of Epilepsy can be broadly understood under the categories of Nonpharmacological management and pharmacological management.

2.1 Nonpharmacological management

It includes first aid, ketogenic diet, vagal nerve stimulation, surgery, stress management, relaxation techniques, acupuncture, yoga, Ayurveda, massage therapy and others.

2.1.1 Ketogenic diet (KD)

It is the method of choice in patients with intractable epilepsy, especially in epilepsy encephalopathies (Saxena VS, Nadkarni VV 2011). The ketogenic diet, which includes a diet containing large amounts of fat, less carbohydrate content, and a sufficient amount of protein, was initially introduced in the 1920s for the treatment of epilepsy and provides energy through ketone bodies. The three major ketone bodies include a) β-hydroxybutyrate (BHB), ii) acetoacetate (ACA), and iii) acetone.

The ketogenic diet is majorly of four types:

I. Classic KD: this included fat and carbohydrate in the ratio of 4:1, which can be altered based on requirements to 3:1.
II. Medium chain triglyceride (MCTD): it is an altered Atkins diet which involved large production of ketone bodies and is associated with high fat content of up to 60%.
III. Modified Atkins diet (MAD): contains 65% fat content, 25% protein content and 10% carbohydrate content.
IV. Low glycemic index treatment (LGIT): it is similar to the Atkins diet but with a low glycemic content.

Efficacy of KD

It has not only been effective in the treatment of epilepsy for children but for adults as well. KD shows its efficacy in the treatment of epilepsy related syndromes. Several studies have suggested that KD is closely related to the rate of seizures (Imdad K, Abualait T, 2022).

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2.1.2 Deep brain stimulation

This method is majorly used for treating intractable epilepsy. The potency of DBS is based on the underlying mechanism, which ranges from molecular and cellular processes to wide-scale network modulation.

In molecular and cellular events, it produces a functional lesion which is marked by hyperpolarization of neurons nearer to the stimulating electrode, which in turn reduces the sudden burst firing as well as the local generation of discharges. It has been observed that it also induces long-term neuroplastic changes (Shon YM, Park HR, 2025).

Network level modulation includes activity not only on the site of stimulation but also alters the dynamics of interconnected regions in the brain. In addition to this, CM and hippocampus (hip-DBS) exhibit similar network like effects. Stimulation of CM has been shown to alter the thalamo-cortical circuits, which causes large-scale desynchronisation of activity. Whereas the stimulation of the hippocampus caused direct inhibition of the primary seizure and simultaneously altered the hippocampal and cortical interactions (Shon YM, Park HR, 2025).

2.1.3 Neurostimulation

This is a methodology which includes sending electrical signals to the neurons with the help of a specially designed gadget. The three types of neurostimulation comprise: stimulation of the vagal nerve, brain-responsive neurostimulation and neuromodulation, also known as deep brain stimulation (Sireesha V, Nikshitha M 2025).

2.1.4 Artificial Intelligence in Epilepsy

Artificial intelligence can be defined as the utilization of various aspects of human intellect as computer algorithms to aid machines in solving problems naturally.

Machine learning (ML) is an affiliation of artificial intelligence which studies the computer systems which learn through experience devoid of explicit instructions utilizing various programming languages to code algorithms.

Creation of an automated computerized system for epilepsy diagnosis is of utmost importance. The procedure includes extracting entropy attributes from EEG recordings. Various ML techniques are developed, such as the fuzzy Sugano classifier, support vector networks,

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classification and regression trees also known as decision tree, and others. Aids in the identification of newborn seizures by recognizing the type and starting point. 27 studies have shown the utilization of ML in the diagnosis of epilepsy by employing several algorithms (Al-Breiki A, Al-Sinani S, 2025).

2.1.5 Acupuncture and epilepsy

This ancient method is being recognized by patients suffering from epilepsy to be an aid in controlling seizures, often used as a synonym in Chinese medicine, which also includes modification of diet, exercises and herbal remedies (Imdad K, Abualait T 2022). Essentially used for the treatment of refractory epilepsy. Number of clinical studies have proved that acupuncture provides beneficial effects on various types of epilepsy such as febrile convulsion, absence seizure, generalized tonic-clonic seizures. Generalized therapeutic benefits comprise improvement of encephalogram (i.e., reduction of spike wave, etc.) and relieving epileptic syndromes, alleviating severity of status epilepticus (Chen S, Wang S 2014).

2.2 Pharmacological management

This includes treatment of epilepsy by investigating epigenetics, AEDs, newer AEDs, intervention of mTOR pathway and others.

2.2.1 Epigenetics in epilepsy

In simpler terms, epigenetics deals with the study of changes in living organisms due to the modification of gene expressions rather than the alteration of genetic code itself. Studying the function of epigenetic modifications that occur in the duration of the disease development is an upcoming concept in research. Knowledge of the epigenetic modifications aids in the evolution of therapies for the prevention of this disease. Henceforth, epileptogenesis shall be reviewed as an essential channel in the evolution of therapies in cases of augmentation in the frequency and severity of epilepsy. Modification of histones is one of the major channels of epigenetics that possesses significant capacity to alter the gene expression by exhibiting its additional consequences in a corresponding way. The role of nucleosome proteins or histone protein is to act as pillar to the quaternary as well as tertiary structure of DNA. Hence, interruption in these pivotal epigenetic regulators causes diseases such as autism, epilepsy etc. The histone modification responsible for epilepsy is lysine acetylation. HDACs (Class 1 histone deacetylases) are liable to remove the acetyl groups from histone terminal tails; therefore, such

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epigenome alterations are considered to be implied for the treatment of various diseases such as epilepsy (Ghosh S, Sinha JK 2021).

2.2.2 Treatment of pharmaco-resistant epilepsy

Also known as intractable epilepsy, refractory epilepsy, uncontrolled epilepsy or pharmaco-resistant epilepsy. It is defined as the misfiring of more than two adequate trials of a physiologically tolerated, selected AED regimen, appositely used, that are dispensed as monotherapies or with another composition to achieve alleviation from the symptoms of seizures.

The postulated mechanisms for the underlying cause of drug-resistant epilepsy are the following: i) alterations in the drug targets, ii) inability of the drug to reach their target, iii) actual target missed by drugs. These can't be treated with conventional AEDs.

Hence, they are treated by non-conventional forms of AEDs used as an addition or in combination with other drugs (Ghosh S, Sinha JK 2021).

Treatment should be initiated with the use of 1st line AEDs. Determination of the appropriate AED is based on the type of epileptic seizure. Other important factors include biological half-life, efficacy, tolerability, drug interactions and several others. (Singh, Rajveer 2020). A list of first-line AEDs used in therapy is given in Table 2.

Table 2. First-line AEDs for refractory epilepsy therapy (Singh, Rajveer 2020).

Primary generalized tonic-clonic	Partial	Absence	Atypical absence, myoclonic, atonic
Valproic acid Lamotrigine Levetiracetam	Carbamazepine Phenytoin Lamotrigine Oxcarbazepine Valproic acid	Valproic acid Ethosuximide	Valproic acid Lamotrigine

First-line AEDs are useful in the treatment of approximately 60% of epilepsy patients. The remaining patients are majorly subjected to polytherapy. In such instances of drug-resistant epilepsy, rational polytherapy is widely used, whose concept is based on the use of AEDs with different mechanisms in combination.

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Other methods of treatment include non-drug therapy, vagus nerve stimulation, surgery, dietary therapies, cannabidiol and several others (Singh, Rajveer 2020).

2.2.3 Anti-epileptic drugs

There are more than 20 AEDs available that are being used for the treatment of epilepsy. Their pharmacological mechanism is classified into the following three: 1) augmentation of inhibitory transmission, 2) reduction in excitatory transmission and 3) obstruction of voltage-gated ion channels (Domingo Sánchez, Jorge Gómez-Carpintero 2024). An appropriate drug is chosen based on the type of seizure, such as generalized tonic-clonic seizures, absence seizures, etc., the age of the patient, and ADRs. Adverse drug reactions are the most important profile to be considered for the treatment of early-onset seizures, such as Dravet syndrome, whose therapy requires the administration of two or more AEDs in combination, hence, augmenting the risk of ADRs (De Bellis M, Rubino EM 2025).

The common ADRs include:

i) Neurological ADRs: Ataxia, dysarthria, diplopia (coordination disorders), sedation, asthenia, dizziness, tremors, cognitive impairment, mood alterations, seizure aggravation, precipitation of state of malaise.
ii) ADRs related to other organs: osteoporosis, increased tendency of bone fractures, obesity, cardiovascular diseases (arrhythmia, cardiac arrest), skin rashes, nausea, vomiting, abdominal pain, hyperammonemic encephalopathy (serum ammonia level above 80 mcg/dL), genetic abnormalities, and hepatotoxicity.
iii) Chronic ADRs: Hirsutism, gingival hyperplasia, Dupuytren's contraction, hypovitaminosis D, hormonal disorders, dyslipidemia, pigmentation of skin, lips and nails.
iv) Teratogenesis (De Bellis M, Rubino EM 2025).

Therapy of other types of epilepsy is initiated by the utilization of AEDs, which are best administered with sodium valproate for treating symptomatic myoclonic jerks and grand-mal seizures also known as generalized tonic-clonic seizures. Primary treatment of complex partial convulsions is proceeded by the administration of carbamazepine and oxcarbazepine with valproate is being recommended (Sireesha V, Nikshitha M 2025). A few examples of AEDs are enlisted in Table 1.

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2.2.4 New antiepileptic drugs

Their pharmacological mechanism is exhibited by blocking Na^+ or Ca^{2+} channels or through GABAergic transmission. The newer generation of AEDs have several other minor sites for action along with the major sites. Studies have shown that LEV, PER, and LCM have unique binding sites and profiles (Hanaya R, Arita K 2016). The various drugs and their targets have been depicted in Table 3.

Table 3. Major pharmacological targets of AEDs (Goldenberg MM, 2010).

Drug	Action site	
	Target	Type of cell
Phenytoin	Na^+ channel	Excitatory neuron
Carbamazepine	Na^+ channel	Excitatory neuron
Lamotrigine	Na^+ channel	Excitatory neuron
Lacosamide	Na^+ channel	Excitatory neuron
Zonisamide	Na^+ channel	Excitatory neuron
Rufinamide	Na^+ channel	Excitatory neuron
Valproate, divalproex	Na^+ channel	Excitatory neuron
Retigabine	K^+ channel	Excitatory neuron
Gabapentin	Ca^{2+} channel	Excitatory neuron
Pregabalin	Ca^{2+} channel	Excitatory neuron
Ethosuximide	Ca^{2+} channel	Postsynaptic neuron
Vigabatrin	GABA-T	Glial cell
Benzodiazepines	$GABA^A$ receptor	Postsynaptic neuron
Barbiturates	$GABA^A$ receptor	Postsynaptic neuron
Topiramate	$GABA^A$ receptor	Postsynaptic neuron
Stiripentol	$GABA^A$ receptor	Postsynaptic neuron
Ganaxolone	$GABA^A$ receptor	Postsynaptic neuron
Felbamate	NMDA receptor	Postsynaptic neuron
Perempanel	AMPA receptor	Postsynaptic neuron
Tiagabine	GAT-1	Inhibitory neuron
Levetiracetam	SV2	Excitatory neuron
Brivaracetam	SV2	Excitatory neuron

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The newer AEDs are classified as anti-epileptic, anticonvulsant and anti-epileptogenic drugs.

Brivaracetam

A 21st-century AED that has been frequently administered in the treatment of epilepsy in patients older than 16 years. It is affiliated with the racetam family, similar to levetiracetam, and has a high affinity for the synaptic vesicle protein 2A ligand (SV2A ligand). However, it binds to a distinct conformational location of the SV2A ligand. It is an approved drug for the monotherapy of focal onset seizures.

Pharmacokinetics: Brivaracetam is an orally administered drug, rapidly absorbed from the intestine, and has 100% bioavailability. Illustrates linear kinetics with the plasma concentration increasing in proportion to the dose administered.

Metabolism: extensively undergoes metabolism in the liver, principally through hydrolysis of the ethanamide group, resulting in the production of a metabolite of carboxylic acid. The latter step includes oxygenation by CYP450 (CYP) 2C9. The secondary metabolism includes fatty acid oxidation of the propyl side chain by CYP2C19, which results in a hydroxylated metabolite.

Excretion: 90% excreted by kidneys within two days of administration, 8.6% of it is eliminated as unmetabolised BRV through urine.

Efficacy: mainly exhibited as a supplementary therapy for focal seizures, tonic-clonic seizures and Progressive Myoclonus Epilepsy type 1 (EPM1) (Hwang H, Kim WJ 2025).

2.2.5. Treatment of Status Epilepticus

Status epilepticus is defined as a dangerous neurological crisis that requires immediate diagnosis and management to prohibit significant mortality. Initially, status epilepticus was defined as a seizure that extends for more than 30 minutes. The guidelines of the Neurocritical Care Society changed this duration to 5 minutes or more than 5 minutes of seizure activity.

Status epilepticus can be characterized with the production of convulsions or without convulsions, focal motor, myoclonic, or refractory.

The etiology of status epilepticus includes meningitis, encephalitis, metabolic abnormalities, brain injury, drug withdrawal syndromes, hypoxemia and several others (Wylie T, Sandhu DS 2023).

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Treatment

Is addressed with the simultaneous regulation of airway and blood circulation along with the administration of AEDs.

Benzodiazepines are the first-line AEDs that are used as an emergency control in the therapy of status epilepticus (Wylie T, Sandhu DS 2023).

Lorazepam is the choice of drug due to its rapid onset of action, administered at a dose of 0.1mg/kg IV, ensuring administration of less than a 2mg dose per minute. The second choice of drug in case of non-availability of lorazepam is diazepam 0.15-5 mg/kg IV per minute. Multiple doses are dispensed when seizures are not resolved by the first dose. Benzodiazepines are also administered through the intramuscular, rectal, nasal or buccal routes if venous access is not available (Wylie T, Sandhu DS 2023).

As one-third of status seizures are refractory upon administration of benzodiazepines, monotherapy with benzodiazepines yields no results (Yuki Kishihara, Hideto Yasuda, 2024). Hence, second-line AEDs are administered simultaneously with benzodiazepines. A list of second-line AEDs is given in Table 4.

Table 4. Second line Anti-epileptic drugs (Yoshihiro Sugiura, Masahiro Iguchi, 2018)

Partial seizure	Tonic clonic seizure, clonic seizure	Absence seizure	Myoclonic seizure	Status epilepticus	Tonic seizure, Atonic seizure
Phenytoin Clobazam, Clonazepam, Phenobarbital Gabapentin Lacosamide	Topiramate Zonisamide Perampanel	Lamotrigine	Topiramate Piracetam Clobazam	Fosphenytoin Lacosamide Levetiracetam Phenytoin Phenobarbital Valproate	Levetiracetam Lamotrigine

The management approach includes the use of phenytoin, phenobarbital, fosphenytoin (20 mg/kg), levetiracetam (40 to 60 mg/kg over 15 minutes), valproic acid (30 mg/kg to 10 mg/kg/minute), midazolam, and thiopental as the choice of drugs. For the treatment of refractory status epilepticus to be effective, the AEDs should be infused continuously (Wylie T, Sandhu DS 2023).

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2.2.6. Drug therapy in women of reproductive age

15 million women of reproductive age are diagnosed with epilepsy worldwide. Effective seizure management is provided by the administration of AEDs. Valproate is an extensively used ASM (anti-seizure medication) at around 38.4%, followed by levetiracetam (35.7%), lamotrigine, carbamazepine, topiramate, oxcarbazepine, clonazepam, lacosamide and gabapentin, according to the data collected in 2019 (Wójcik K, Kruk M 2024).

However, the major drawback of these drugs is the teratogenic effect that they exhibit. Several observational studies cited that valproate has the highest risk of producing congenital abnormalities and deformation in infants exposed to the drug in the uterus. Furthermore, it also causes autism, cognitive and behavioral teratogenicity, and loss of cognitive function. Hence, according to the proposal of the European Medicines Agency (EMA), valproate should not be administered to women of reproductive age unless there are no alternate measures available (Wójcik K, Kruk M 2024).

Due to the teratogenic effect and other side effects, the use of valproate and carbamazepine decreased to 27.6% and 17%, respectively, in 2022.

In contrast, the use of levetiracetam, lamotrigine and oxcarbazepine monotherapies has significantly increased. Therapy with AEDs possessing a safer profile is commenced either as a monotherapy or polytherapy.

Major therapy for childbearing women is provided via ASM monotherapy rather than VPA (valproic acid) (Wójcik K, Kruk M 2024).

Polytherapy is based on the concept of administering ADEs possessing different or similar mechanisms in combination. Polytherapy is less preferable, as it causes a decreased inhibition of seizures compared to monotherapy in both focal and primary generalized seizures. An increased rate of foetal malformations was observed in polytherapy that includes valproate and topiramate in drug combinations. Whereas lamotrigine and levetiracetam, when administered in combination, have exhibited convulsion control and foetal safety.

Therefore, polytherapy in pregnancy can be effectively used by avoiding valproate and topiramate in the combinations (Vajda FJ, O'Brien TJ 2018).

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2.2.7. Intervention of mTOR pathway

It is one of the contemporary approaches in epilepsy treatment. This is called as the mammalian target of rapamycin pathway, which is accountable for the regulation of cell division, cell proliferation, cell specialization, cell maturation, and cell commitment and metabolism. Several researches have indicated that the disruption of mTOR leads to the occurrence of a symptomatic epilepsy. Hence, the intervention of this pathway can aid in the discovery of antiepileptic drugs (Ghosh S, Sinha JK 2021).

Conflict of interest

None

3. Conclusion

Epilepsy is a serious nervous system disorder that affects 50 million population globally. It is distinguished by disturbances of the cerebral cortex. The etiology of epilepsy may range from genetic factors to infections of the vital organs. Factors such as age, medical conditions, genetic predisposition, and prior neurological history may aggravate the risk of seizures. Treatment of epilepsy is primarily achieved by using anti-epileptic drugs that have three mechanisms of action. The type of drug and its appropriate dose are administered based on the type of seizures, age and other conditions of the patient. Management of epilepsy comprises both pharmacological and non-pharmacological treatment. With this module of treatment, developing therapy for drug-resistant epilepsy becomes facile. The various side effects of AEDs can be overcome by using nonpharmacological methods, including artificial intelligence, ketogenic diets, and others. Management of epilepsy has become easier over the decades due to the advancement of technology, especially in the case of neurostimulation. Therefore, the remedy for epilepsy is effectively carried out by both methods.

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