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A COMPREHENSIVE REVIEW ON RECENT ADVANCEMENT IN ALLOPATHY AND AYURVEDA IN THE MANAGEMENT OF EPILEPSY

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ABSTRACT: Millions of people throughout the world suffer with epilepsy, a neurological illness that frequently results in seizures. Epilepsy has been treated using both allopathic and ayurvedic medicinal methods. Antiepileptic medications (AEDs) are used by allopathic medicine, commonly referred to as contemporary medicine, to manage seizures. Several AEDs have been created recently in allopathic medicine, such as Brivaracetam and Perampanel, which have better effectiveness and safety than earlier AEDs. **Background:** Approximately 1% of the global population suffers from epilepsy, a neurological illness marked by recurring seizures. Epilepsy is a difficult condition to manage despite improvements in medical technology, with up to 30% of patients still experiencing seizures while receiving antiepileptic medication (AEDs). **Conclusion:** Epilepsy is increasingly being treated with both allopathic and ayurvedic therapy. There are several ongoing clinical trials for the treatment of epilepsy using both allopathic and ayurvedic drugs. Because it is more practical for their busy lifestyles, has a quick mechanism of action, and is readily accessible, allopathy therapy is preferred by many patients.

INTRODUCTION:

Background: Recurrent seizures are the hallmark of the neurological condition known as epilepsy, which affects 1% of people worldwide. Epilepsy is a difficult condition to manage despite improvements in medical technology, with up to 30% of patients still experiencing seizures while receiving antiepileptic medication (AEDs).

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The main method of treating epilepsy has been allopathy, or conventional Western medicine, with an emphasis on controlling seizures with the aid of AEDs. AEDs alone may not always completely control seizures in patients, and these drugs can have serious adverse effects.

The Indian traditional medical system known as Ayurveda has been utilised for thousands of years to treat epilepsy. Ayurvedic therapy for epilepsy often combine dietary adjustments, lifestyle adjustments, herbal treatments, and physical therapies. Although the effectiveness of Ayurveda remedies for epilepsy is not well established, some studies have indicated that they may be useful in lowering the frequency and intensity of seizures as

well as enhancing patients' general quality of life. A thorough analysis of recent developments in epilepsy therapy in both allopathy and Ayurveda can offer a more nuanced knowledge of the present status of epilepsy treatment and point up prospective directions for further investigation. The most recent advancements in AEDs, including novel drug classes and drug delivery technologies, as well as developing non-pharmacological therapies like neuromodulation and gene therapy, can be covered in this study. The review can also look at the most recent studies on Ayurveda therapies for epilepsy, including clinical trials and the use of cutting-edge scientific methods to look at how Ayurvedic medicines work. A thorough analysis can provide light on the state of epilepsy therapy today and identify possible topics for future study and cross-system collaboration by looking at the most recent developments in both allopathy and Ayurveda.

Epilepsy: A brain disorder known as epilepsy is characterised by a inclination to have ongoing seizures as well as the neurobiologic, cognitive, psychological, and social ramifications of this illness. There must be at least one epileptic episode for epilepsy to be diagnosed. An epileptic seizure, which lasts only a few minutes, is caused by abnormally high or coordinated neuronal activity in the brain [1]. According to classification, epilepsy is a brain disease characterised by one or more among the following symptoms:

(1) At least two unprovoked (or reflex) seizures that occur more than 24 hours apart, (2) two unprovoked (or reflex) seizures that occur within ten years of one another, one unprovoked (or reflex) seizure and a likelihood of additional seizures equal to the general recurrence risk (at least 60%), or (3) the presence of an epilepsy syndrome [2].

Pathophysiology of Epilepsy: The complicated biochemistry of epilepsy and seizures explains the wide variety of unique seizure illnesses. As seen in **Fig. 1**, all epilepsies lead to an unbalanced synaptic drive between excitatory (through glutamatergic signalling) and inhibitory (by GABAergic signalling), which can result in seizure activity. A preliminary pharmacologic investigation suggests that GABAA-receptor antagonists and glutamate-receptor (NMDA, AMPA, kainate) agonists may be able to cause seizures in healthy animals [3].

A group of neurons fire excessively synchronously and continuously during epileptic convulsions. Neuronal excitability is consistently elevated, and this is the one characteristic shared by all epileptic disorders. Many conditions, including trauma, oxygen deprivation, malignancies, infections, and metabolic disturbances, can cause abnormal cellular discharges. However, there are no obvious reasons found in about 50% of instances of epilepsy. There are some epilepsy types with known underlying causes and pathophysiological processes, such as those brought on by aberrant neuronal migration and monogenic epilepsies. Currently, knowledge about a few further types of epilepsy is lacking [4].

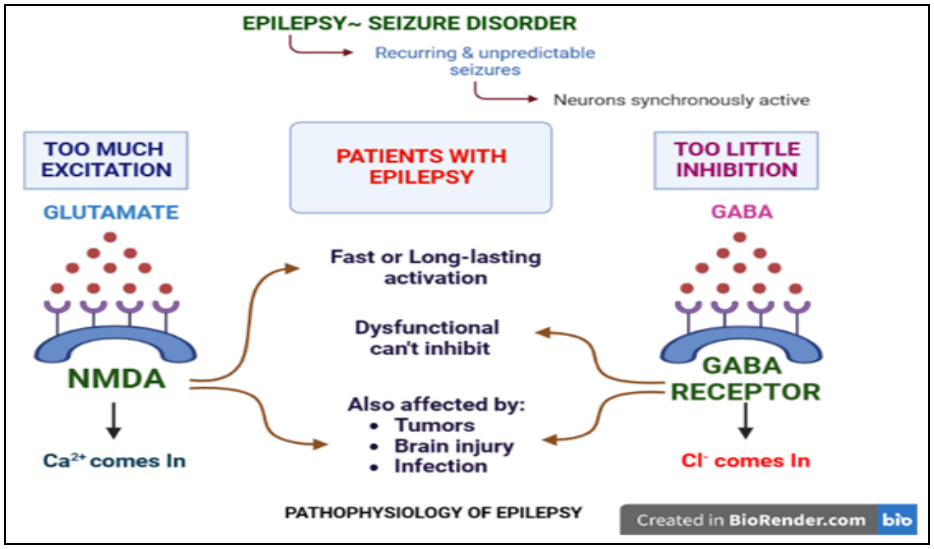

FIG. 1: PATHOPHYSIOLOGY OF EPILEPSY

The diagnosis, prognostic advice, and treatment of epilepsies are all aided by knowing the cause of an individual's epilepsy. In fact, determining the likelihood that a single seizure would recur and, thus, making the epilepsy diagnosis, may depend on the aetiology. It divides aetiologies into six groups, including structural, genetic, infectious, metabolic, immunological, and neurodegenerative (all of which are categorised by the International League against Epilepsy [ILAE]) (which we have considered separately because of its growing importance in epilepsy). Several aetiologies fit into more than one category since these divisions are not mutually exclusive. In fact, to varying degrees, hereditary factors are likely to play a part in a person's propensity for seizures if they have epilepsy [5].

Epilepsy in Ayurveda: The four Vedas, which are usually acknowledged to have been imparted to the sages by Brahma (the creator) around 6,000 years before the Christian period, are the main basis of ancient Indian Aryan culture and medicine. First millennium BC is the oldest. The term "Ayurveda" refers to the body of knowledge that helps one to comprehend the essence of life and, as a result, lengthen one's life (Ayu means "life" in Sanskrit, and Veda means "to know"). The Samhitas (encyclopaedic writings) of Caraka and Sushruta describe the majority of the Ayurvedic principles, but unhappily, they are not still preserved in their original form. These manuscripts, regarded to be the most authentic and well-known examples of the ancient Ayurveda, were initially written around 1,000 BC. The Charaka Samhita, a literature on the old Indian medical system, was written between 1,000 and 800 BC. It was authored by Agnivesa, Caraka, and Drdhabala. The Sushruta Samhita, one of the greatest pieces of its kind in Sanskrit literature, is crucial to comprehending surgery [6].

The world's oldest medical system is called Ayurveda, which originates from ancient India and means "knowledge of life." The term "epilepsy" is abbreviated as apasmara, where "apa" stands for denial or absence and "smara" for recollection or consciousness. Apasmara Poorva Roopa was the name given to Aura after her identification. There were many different symptoms linked to auras that were noted. The subjective perception of sounds, the sense of darkness, the perception of delusion, and the dream-like state are all remarkable. An Apasmara episode is characterised by falling down, shaking of the hands, legs, and body, rolling of the eyes, clenching of the teeth, and lip foaming. There are four main forms of epilepsy based on the disruption of the doshas (humours) that control the physiological and physiochemical processes of the body. Apasmara is a serious chronic condition that is difficult to cure. There are several justifications. Therapy includes addressing the underlying reasons, altering one's diet, and avoiding potentially dangerous situations [7].

Ayurvedic Approach in Treatment of Epilepsy: With a heavy burden of illnesses, suicides, physical comorbidities, high expenses, and poor quality of life, A significant global health burden is posed by neuropsychiatric and neurodegenerative conditions such Alzheimer's, Parkinson's, schizophrenia, epilepsy, depression, and anxiety. Traditional medicines are now more popular than ever, and because they come from a natural source and have fewer side effects, academics like phytomedicines. Contrarily, conventional synthetic drugs have been associated with unfavourable but unavoidable side effects and low patient compliance. As a result, natural pharmacological techniques are increasingly regarded as a successful treatment for a variety of brain problems. Ayurveda provides a thorough method of therapy in addition to several nootropic herbs with multifaceted bioactivities in a range of diseases. There is a patchwork of information available on conventional Ayurveda treatment methods for different mental illnesses. [8]Ayurvedic treatment puts the patient first, not the illness. According to Ayurveda, different diseases might be brought on by the same sort of disturbance, whereas different types of disturbances or imbalances can bring about the same condition.

By clearing blocked heart and brain connections that may be caused by an excess of doshas or humours, ayurvedic medicine aims to treat epilepsy. To open the channels, purgatives and different combinations are used. Oral medicines created with oils and ghee (purified butter) are another option for treating ailments outside external oil treatments, massages, and baths. The medicine chosen for one epileptic patient might not be suitable for another, as was previously stated. The conventional therapies listed below are frequently

prescribed because patients have faith in alternative therapies, even though the majority, if not all, have not been clinically or scientifically proven to be effective in treating or aiding people with epilepsy. [9]. Comprehensive treatment for apasmara uses both pharmaceutical and nonpharmacological methods. The exact medicine combination is selected after a thorough assessment of the client depending on the disease and host variables. The disease' paroxysmal character was understood, and a number of treatments were encouraged both within and outside of ictuses. These include colonic medicine administration (basti), various forms of nasal medication administration (nasya), oleation (snehana) and sudation (svedana) treatments, cleansing (shodhana) modalities including emesis (vamana) and purgation (virechana), and oleation(snehana) and sudation (svedana) therapies. These are supplemented by the use of appropriate internal medications prepared in a range of pharmaceutical forms, such as decoctions (kashaya), powders (churna), fermented preparations (asava-arishta), pills (vati), and freshly prepared herb juices (svarasa), pastes (kalka), oil- or ghee-based formulations (sneha), among others. A few examples of the herbal remedies used are Aswagandharishtam, Saraswatachurna, Kalyanakaghrita, Dadhikaghrita, Kushmandaghrita, Panchagavyaghrita, MahaPanchagavyaghrita, Siddarthakaghrita, Tiktakaghrita, and Mahayogarajaguggulu. Manasamitravatakam, Mritasanjeevanigutika, Apasmarahara rasa, Apasmararirasayana, Bhootabhairava rasa, Smritisagara rasa, Chaturbhuja rasa, Chaturmukha rasa, Chintamanichaturmukha rasa, Tapyadilauha, Vatakulantaka rasa, Yogendra rasa are herbal/mineral formulations commonly prescribed for Apasmara as shown in the **Table 1** [10].

Various Ayurvedic Formulations for the Treatment of Epilepsy:

TABLE 1: DIFFERENT TYPES OF AYURVEDIC FORMULATIONS FOR THE TREATMENT OF EPILEPSY

S. no.	Name of Formulation	Type of Formulation	Functions
1	Saraswat	Churna	Nootropic and cognitive booster [11]
2	Panchagavya	Ghrit	It regulates the frequency and duration of seizures.[12]
3	Kushmanda	Ghrit	It is a nootropic that is advised for improving memory and other cognitive processes.[13]
4	Brahmi	Ghrit	exhibited improved alleviation in attack duration and hindered higher mental functioning [14]
5	Manasyadi	Kwatha	Used in episodes of hysteria.[15]
6	Aswagandharista	Aasav-arista	Anxiolytic impact of GABA-mimetic action [16]
7	Saraswatarista	Aasav-arista	Antipsychotic medication that promotes intelligence.[17]
8	Smritisagara rasa	Rasausadhi	Stress is reduced through improving intelligence.[18]
9	UnmadaGajakesari	Rasausadhi	GABAergic activation as well as extra antioxidant activity [19]
10	Apasmarari rasa	Rasausadhi	Act as an anti-convulsant medication [20]
11	Tantupashana	Rasausadhi	a therapy option for generalised tonic-clonic seizures [21]
12	KsheerabalaTaila	Taila	It lowers oxidative stress. [22]
13	Shatavari	Taila	It has anticonvulsant properties.[15]

Allopathy:

Pharmacological Approach on Epilepsy: The phrase allopathy was created by German physician Samuel Hahnemann out of two Greek concepts:"allos," that means the opposite or the other, and "pathos," which means agony. Delivering a therapeutic substance that has the illness's opposite effect is the foundation of allopathic therapy [23, 24]. The foundation of the allopathic medical system is a set of procedures, tools, and tests that have been approved by science. Pharmaceuticals that are synthetic, semi-synthetic, and augmented and that have been proven to be effective, safe, and of high quality are used to treat illnesses and ailments. It focuses on the ailment that has an impact on the body, with health being defined as the absence of disease. Pharmaceuticals that have been used, approved, and commercialised by several international and local regulatory organisations are utilised in the medical system.

It might be viewed as a form of symptomatic treatment [25]. The use of foxglove plant extract to treat dropsy is regarded to have been the first use of allopathic medicine, or modern therapeutics, in the 1250s (congestive heart failure).

After that, improvements in manufacturing, quality assurance, diagnosis, and therapy supported the development of the allopathic system. Recent changes to the law and regulatory approval have given processes and drug regulatory matters top priority. Improvements in system quality, safety, and effectiveness as a consequence [26]. Voltage-dependent ion channel modulation, GABA activation, and glutamate receptor inhibition are the most often employed methods by AEDs with licences to treat epilepsy. Many effective routes have been identified, some of which are potential targets for treatment of neurodegenerative diseases. Many third-generation anti-epileptic drugs are readily available. With a focus on the mTOR pathway, blood-brain barrier breakdown, and inflammatory pathways, several research that back up current epilepsy treatment methods have been presented [27].

Primidone, ethosuximide, phenobarbital, and phenytoin are only a few of the antiepileptic medications that were created during the start of the 20th century. More than 20 antiepileptic medications are now being used in clinical settings, and more than 10 separate classes of antiepileptic medications were created in the 1990s [28].

General Concept of Antiepileptic Drugs: Antiepileptic medication mechanisms have previously been classified into four categories:1) Phenytoin, carbamazepine, lamotrigine, oxcarbazepine, ethosuximide, and zonisamide control Na+, Ca2+, and K+ voltage-dependent ion channels (2) GABA potentiation (phenobarbital, benzodiazepines, vigabatrin, and tiagabine), (3) several methods of action (sodium valproate, gabapentin, felbamate, and topiramate, among others), and (4) a further mechanism of action (levetiracetam) [29].

Antiepileptic medications that have recently been produced function in a variety of unique ways that boost their effectiveness while lowering adverse effects. Since there are several antiepileptic medications available, selecting an effective therapy necessitates expanding our understanding of how to use these drugs depending on the various types of seizures in order to provide tailored care for epilepsy patients. As shown in **Table 2**, antiepileptic drug selection is based on the clinical features of seizure types, electroencephalogram results, epileptic syndrome, and medication stability [30].

Mechanism of Action of AEDS:

TABLE 2: MECHANISM OF ACTION OF AEDS

S. no.	Drugs	Mechanism of Action
1	Clobazam	Binds to and stimulates GABAA receptors, resulting in a greater frequency of Cl-channel opening. [31]
2	Carbamazepine	Voltage-gated sodium channel inhibition.[32]
3	Ethosuximide	T-type calcium channel inhibition[31]
4	Phenobarbital	Synaptic inhibition via an action on GABAA.[33]
5	Phenytoin	Reduce high-frequency repeated action potential firing by increasing sodium-channel inactivation.[34]
6	Gabapentine	Limiting calcium influx and consequent excitatory neurotransmitter release[35]
7	Lamotrigine	Neuronal voltage-activated sodium channels are blocked.[36]
8	Levetiracetam	reducing the release of calcium from intraneuronal storage, blocking the function of GABA and glycine-gated current negative modulators, and preventing excessive synchronised activity between neurons. [37]
9	Felbamate	Inhibits excitatory postsynaptic potentials while increasing intracellular Ca2+ levels.[31]
10	Vigabatrin	GABA-transaminase (GABA-T) inhibitor that is irreversible and raises the amount of GABA[38]

MATERIALS AND METHODS:

Data Sources and Descriptions: For this review, a comprehensive search of the literature on controlled clinical trials conducted by pharmaceutical companies (product monographs of the drugs), observational studies, case reports, academic or peer-reviewed articles (prospective and retrospective studies), as well as experimental trials conducted, was conducted. The Pub Med and Google Scholar search engines were used to find relevant papers using phrases like Ayurveda, epilepsy, seizures, allopathy, antiepileptic, or

anticonvulsant medications, first, second, and third generation antiepileptic pharmaceuticals. The individual names of 05 first generation and 05 second generation AEDs are also included in the search. There was no requirement for ethics committee approval because this work was based on a thorough literature search.

Determining Recent Advancements in both Systems:

Ayurvedic:

Extracts of Cannabis Used as Anticonvulsants: When we conducted extensive investigation, we discovered numerous intriguing plants in the ayurvedic system, including cannabis and its extracts. Pioneering studies conducted in the late 1970s and early 1980s suggested that cannabidiol (CBD)-enriched cannabis extracts for epilepsy may have some, if not any, benefit. Tetrahydrocannabinol (THC) and cannabido-dioecious CBD, the two main physiologically active cannabinoids, have more convincing preclinical evidence that they have anticonvulsant properties. There aren't any reliable clinical statistics on epilepsy sufferers, though [39, 40].

Epidiolex® a Pure CBD for Epilepsy: Cannabidiol (CBD), a component of medical marijuana, is the source of the medication Epidiolex. FDA-approved cannabidiol for prescription is Epidiolex. It has been shown that CBD medication is effective in treating seizures that are very severe or difficult to treat. In animal models, CBD that has been purified has shown to be effective against certain types of seizures and epilepsy [41]. Adenosine-mediated signalling and intracellular Ca2+ may both be affected by cannabidiol (CBD), according to researchers at the University of California, Berkeley [42]. The first randomised controlled trials for Lennox-Gastaut syndrome and Dravet syndrome used the medication Epidiolex, and those results were reported in 2017 and 2018, respectively [43]. CBD was given FDA approval in June 2018 as an additional antiepileptic medication for adults with Lennox-Gastaut syndrome or Dravet syndrome.

Mentat® an Herbal Formulation for Epilepsy: Mentat is a remedy that incorporates several important Ayurveda elements. A Mentat® randomised controlled study was conducted in 31 adult patients with epilepsy. Some of the plants used in mentat syrup include *Bacopa monnieri, Nardostachys jatamansi, Centella asiatica, Acorus calamus,* and *Prunus amygdalus* [44, 45]. Convulsions can be treated using the useful herb Nardostachys jatamansi. It enhances intelligence and memory while also drastically reducing seizure frequency [46, 47]. A neurotropic anticonvulsant drug is *Centella asiatica*. Children who struggle with behaviour difficulties behave much better as a result [48]. The plant acoruscalamus improves cognitive function and helps cure epilepsy [49]. It has been discovered that *Prunus amygdalus* improves cognitive functions [50].

A Mentat® Syrup Placebo-Controlled Clinical Trial: While treating febrile convulsions, mentat syrup may be an effective medication. Youngsters who consumed Mentat syrup behaved better than the placebo group did. In contrast to all the feverish children taking a placebo, 36% of the children using Mentat syrups suffered a febrile episode but no convulsions at the conclusion of the research [51].

Allopathic:

Cenobamate for Treatment-Resistant Focal Seizures: Cenobamate is one of the most recently created antiseizure drugs (ASMs) for treating focal onset seizures in people over the age of 18. The recommended daily dosage of 200 mg should be progressively raised from the first dose of 12.5 mg [52].

Clinical Trials of Cenobamate: The USFDA authorised cenobamate in light of the promising results of two substantial phase II randomised, double-blind clinical studies comprising 659 people [53, 54]. According to the risk-of-bias evaluation approach, both trials showed a low risk of attrition bias, performance bias, detection bias, and selection bias [56]. Cenobamate's effectiveness as a complement to anti-seizure drugs was assessed in a significant phase II randomised, double-blind, placebo-controlled multicenter study (NCT01397968) for drug-resistant focal seizures. Participants in the study were both male and female, with ages ranging from 18 to 65 [55]. Cenobamate appears to be a potential therapeutic option for partial-onset seizures that are resistant to medication, according to the results of these studies; nonetheless, there are still a number of

treatment-related side effects and drug-drug interactions that require care [56].

Third Generation AEDs: Pharma companies have created and licenced about 20 novel third-generation AEDs. Based on how they impact neurotransmission in the brain, anticonvulsant medications are selected. Several medications specifically increase the GABAA receptor-mediated response or reduce excitatory neurotransmission [57, 58, 59].

Ganaxolone a New Third Generation AED: Synthetic neuroactive steroid ganaxolone (ZTALMY®; Marinus Pharmaceuticals) modulates the GABAA receptor complex in a positive allosteric manner. In March 2022, the medication received its initial approval in the US for the management of CDD-related seizures [60].

Advancement on Molecular Level:

Gabaa Receptor Pharmacology: There are many subunit classes that may be created from the five subunits that make up GABA (A) receptors, which are ligand-gated chloride channels. Since there are 19 distinct subunits, there are several GABA (A) receptor subtypes with various subunit compositions, regional, cellular, and subcellular distributions, and pharmacology. Most of these receptors have two, two, and one two subunits. Many drugs with clinical and pharmacological relevance target GABA(A) receptors, including benzodiazepines, barbiturates, neuroactive steroids, anaesthetics, and convulsants.

Benzodiazepines interact with the extracellular (+) 2(-) interface, whereas GABA acts at the two extracellular (+) (-) interfaces of GABA (A) receptors. However, anaesthetics, neuroactive hormones, and barbiturates appear to interact with the solvent-accessible regions of the transmembrane domain. The GABA (A) receptor subtypes with 22, 32, or 52 subunits are preferentially binded by a number of benzodiazepine site ligands. This demonstrates that these receptors' many subunit types offer the benzodiazepine binding site enough structural variation to allow for a range of interactions with ligands that bind to the benzodiazepine binding site. Recently, it was discovered that the (+) (-) interface has a brand-new drug binding site.

The kind of receptor subunit present has a substantial influence on how this binding site operates, despite the fact that it is comparable to the benzodiazepine binding site at the (+) 2(-) interface. Drugs that interact with this binding site do not directly activate the GABA (A) receptors; rather, they control them allosterically. The potential benefit of this drug is emphasised. It targets a far wider variety of receptor subtypes than benzodiazepines do [61].

Cannabinoids: Chemicals called cannabinoids are present in the cannabis plant. Tetrahydrocannabinol, a phytocannabinoid, is the main psychoactive component of cannabis (THC). Cannabidiol (CBD), another essential component of the plant, is also present [62].

Sedative, anxiolytic, and anticonvulsant effects of CBD have led to suggestions that it be used to treat paediatric epilepsies such Dravet syndrome [63]. The main cannabinoid receptor has a low affinity for CBD, similar to GABA PAMs, and it has an activity profile that causes anxiolytic and anticonvulsant effects [64]. Endocannabinoids are substances the body produces that stimulate cannabinoid receptors (CB1, CB2) [65, 66]. Several substances that fall under this category are 2-Arachidonoylglycerol (2-AG), 2-Arachidonoyl glyceryl ether, N-Arachidonoyl dopamine (NADA), Arachidonoylethanolamide (AEA), and Lys phosphatidylinositol (LPI) (LPI) [67].

As positive modulators of GABAAR subtypes, they have also been discovered [68]. According to research using recombinant receptors, 2-AG boosts GABAAR activity at low, non-saturating GABA dosages while decreases activity at high, saturating GABA concentrations. Therefore, the control of GABA inhibition dictates the effect of endocannabinoids on GABAAR [69].

Levetiracetam: The anticonvulsant properties of LEV are mediated by several novel pathways, according to research. Levetiracetam has been demonstrated to impact GABA turnover in the striatum and reduce levels of taurine, a low affinity GABAA receptor agonist, in the hippocampus while having no effect on other amino acids [70]. The quantity of glutamate-mediated excitatory transmission presynaptically was decreased as a

result of Zn2+-induced reduction of GABAA-mediated presynaptic inhibition, which was reversed by LEV [71]. The 12 transmembrane integral protein synaptic vesicle protein 2 (SV2) is present at all synaptic locations. They come in 3 varieties: 2A, 2B, and 2C. SV2A is the most common brain isoform, followed by the brain-specific 2B and the less important 2C. It has been suggested that SV2 proteins act as vesicle components like Ca2+ or ATP transporters [72]. Therefore, SV2A ligands may prevent seizures by influencing synaptic release pathways. SV2 has been identified as LEV's most likely target. The autoradiographic distribution of the LEV-binding site in the brain and the immunocytochemical distribution of SV2A are found to be identical. [73]. SV2A is the LEV binding location in the brain, according to several studies [74, 75]. Consequently, the interaction between LEV and SV2A is one of the primary mechanisms behind LEV's anti-epileptic activity.

In the past, several medications for the treatment or symptomatic alleviation of epilepsy have been developed. The following are some of them; see **Table 3** below.

TABLE 3: LIST OF DRUGS UNDER DEVELOPMENT FOR EPILEPSY

S. no.	Brand Name	Generic Name	Chemical Name	Recent Advancement	Reference
1	Briviact	Brivaracetam	(BRI) {(2S)-2-[(4R)-2-oxo-4-propylpyrrolidinyl]-butanamide}	Brivaracetam is now being thoroughly evaluated in adult patients with partial-onset seizures[76].	Steinhoff et al. Therapeutic Advances in Neurological Disorders 12 (2019)
2	Comfyde	Carisbamate (CBM)	(S-2-O-carbamoyl-1-o-chlorophenyl-ethanol)	undertaking clinical trials to assess the long-term efficacy, safety, and tolerability of the drug as an adjunctive treatment for patients with partial-onset seizures. [77]	Lu, Chuansen et al. The Cochrane database of systematic reviews vol. 12,12 CD012121. 6 Dec. 2021
3	Cerebyx	Fosphenytoin (FPHT)	(Disodium phosphate ester of 5,5-diphenylhydantoin)	Clinical trials evaluate the effectiveness of FPHT for treating individuals with non-convulsive status epilepticus and recurring malignant gliomas. [78]	Husain et al. Annals of neurology 83.6 (2018)
4	Aptiom	Eslicarbazepin eacetate (ESL)	[(S)-(–)-10-acetoxy-10,11-dihydro-5H-dibenz [b, f] azepine-5-carboxamide]	Clinical study is examining the effectiveness, safety, and tolerability of ESL in the treatment of bipolar disorder patients experiencing manic episodes. [79]	Soares-da-Silva et al. Pharmacology Research & Perspectives 3.2 (2015)
5	Ztalmy	Ganaxolone (GNX)	(3α-hydroxy-3β-methyl-5α-pregnan-20-one)	Both persons with uncontrolled partial-onset seizures and infants with infantile spasms are being studied in clinical trials with GNX. [80]	Lattanzi et al. Expert review of neurotherapeutics 21.11 (2021)
6	Vimpat	Lacosamide (LCM)	[(R)-2-acetamido-N-benzyl-3-methoxypropionamide]	Clinical trials are investigating LCM as a supplementary therapy for those who have partial seizures and status epilepticus. [81]	Strzelczyk et al. Epilepsia vol. 58,6 (2017)
7	Banzel	Rufinamide (RUF)	{1-[(2,6-difluorophenyl) methyl]-1H-1,2,3-triazole 4-carboxamide}	RUF is being studied in patients with generalised anxiety disorder, refractory partial seizures, and to assess how well the treatment works in patients with drug-resistant epilepsy when compared to a ketogenic diet. [82]	Resnick et al. Epileptic Disord 13.Suppl 1 (2011)
8	Xafinact	Safinamide	(SAF) {(S)-(+)-2-4-[(3-fluorobenzyloxy) benzylamino] propanamide methane sulfonate salt}	In addition to levodopa or a single dopamine agonist, safinamide is currently being studied in individuals with early idiopathic Parkinson's disease. [83]	Wasan et al. Brain Research Bulletin 168 (2021)
9	Fycompa	Perampanel	2-(2-oxo-1-phenyl-5-	Clinical research and empirical	Chinvarun, Yotin et al.

			pyridin-2-ylpyridin-3-yl) benzonitrile	data have shown the benefits of starting perampanel therapy at low dosages and titrating gradually. [84]	Therapeutics and clinical risk management vol. 17 739-746. 21 Jul. 2021
10	Topamac	Topiramate (TPM)	[(1R,2S,6S,9R)-4,4,11,11-tetramethyl-3,5,7,10,12-pentaoxatricyclo [7.3.0.02,6] dodecan-6-yl] methyl sulfamate	Trials have demonstrated that topiramate is effective in treating juvenile myoclonic epilepsy (JME)[85].	Liu, Jia et al. The Cochrane database of systematic reviews vol. 11,11 CD010008. 24 Nov. 2021

CONCLUSION: Epilepsy is increasingly being treated with both allopathic and ayurvedic therapy. There are several ongoing clinical trials for the treatment of epilepsy using both allopathic and ayurvedic drugs. Because it is more practical for their busy lifestyles, has a quick mechanism of action, and is readily accessible, allopathy therapy is preferred by many patients. Similar to how some people choose for Ayurveda therapy because of its affordability, lack of adverse effects on the body, and use of natural ingredients in ayurvedic medicine. Several compounds are now undergoing clinical trials and may show potential for pharmacotherapies in the future.

To determine which treatment, allopathy or ayurveda, is best and safest for the patient, more study is needed. Allopathy is significantly superior and more effective than ayurveda for suppressing symptoms, but ayurveda takes longer to have an effect, according to our analysis of both allopathic and ayurvedic medications in this study. One of the main shortcomings of ayurvedic medicine, which we also learned about throughout our inquiry, is the lack of clinical evidence. Hence, combining the two approaches can be more beneficial and secure for the patient.

DISCUSSION: Epileptic seizures, which are frequent, irregular disruptions of regular brain activity, are signs of epilepsy, a disorder of the brain. Epilepsy is a collection of illnesses that represent underlying brain dysfunction brought on by a variety of conditions, not a single disease entity. There are several seizure illnesses because the biochemistry underlying epilepsy and seizures is complicated. All epilepsies have an abnormal balance of excitatory and inhibitory drive at the synapse level, which might influence seizure activity. Any of the several forms of partial seizures might be the clinical manifestations of partial epilepsies, which affect 60% of people with epilepsy. Cortical lesions, tumours, anomalies in development, or acute cortical injury from trauma or stroke can all result in partial epilepsies. Ayurveda and Allopathy both have a significant impact on this disease. In order to treat this chronic disease, both must be taken into consideration. The history of Ayurveda and its specific treatments for this illness are distinct. Ayurveda is not regarded as standard care even though it has some advantages because there aren't as many clinically proven drugs available in it as there are in allopathy for the treatment or management of this disease. Similar to allopathy, there are some evidence-based medications, but they either do not work at the molecular level or have a variety of side effects. As a result, we have come to the conclusion that both conditions require further molecular research in order to develop effective medications or treatments in the future.

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