

A Brief Review on Herbal Antiepileptic Drug

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Abstract: *Epilepsy is one of the most prevalent chronic neurological disorders, affecting over 50 million people worldwide, and is characterized by recurrent, unprovoked seizures resulting from abnormal neuronal discharges in the brain. Although conventional antiepileptic drugs (AEDs) remain the primary treatment modality, nearly 30% of patients exhibit drug-resistant epilepsy, and many experience adverse effects such as cognitive impairment, hepatotoxicity, teratogenicity, and behavioral disturbances. These limitations have prompted growing interest in herbal and plant-based therapies as potential alternative or adjunctive treatments. Medicinal plants used in traditional systems such as Ayurveda, Traditional Chinese Medicine, and Unani have demonstrated anticonvulsant properties in experimental and preclinical studies. Several phytoconstituents—including flavonoids, alkaloids, terpenoids, saponins, and phenolic compounds—exert antiepileptic effects through mechanisms such as modulation of GABAergic transmission, inhibition of glutamatergic excitotoxicity, antioxidant activity, ion channel regulation, and neuroprotective actions. Plants such as *Cuscuta epithimum* Murray, *Caesalpinia bonducella* (L.) Roxb., *Parietariacretica* L, *Commiphora opobalsamum* Engl, *Cedrus deodara* Loudon, *Urginea Maritima* Baker, *Bryonia dioica* Jacq, *Bryonia alba* L, *Paeonia officinalis* L, *Lagoeciacuminoides* L, *Trigonella caerulea* (L.) Ser, *Ferulaasa-foetida* L, *Populus nigra* L, *Coriandrum sativum* L, *Origanum majorana* L, *Ruscus aculeatus* L, *Lavandulastoechas* L, *Ferulagummosa* Boiss, *Ferula Persica* Willd, *Aristolochia rotunda* L. have shown promising results in seizure models. Despite encouraging findings, challenges remain regarding standardization, identification of active constituents, pharmacokinetics, safety profiling, and large-scale clinical validation. This review provides a concise overview of the pathophysiology of epilepsy, limitations of conventional therapy, and the pharmacological potential of herbal antiepileptic drugs, highlighting their mechanisms of action, therapeutic prospects, and future research directions. Herbal antiepileptic agents may offer safer, cost-effective, and complementary approaches in the management of epilepsy.*

Keywords: Herbal medicine, epilepsy, anticonvulsant, animal model

I. INTRODUCTION

The word epilepsy derived from the Greek work ‘epilepsia’ which means ‘to take hold of’ which in turn was combined form ‘epi’ means upon and ‘lambanein’ means to take.[7]

In ancient time’s epilepsy was connected with religions faint or ever a possession by a demon. In the past epilepsy was considered as the sacred disease in support of this view a large number of people believed that epilepsy affected people who to some extent taken hold of by demons or that the visions experienced by the epileptic people were sent by the Gods. In 2005, the International League Against Epilepsy (ILAE) defined epilepsy as—a disorder of the brain



characterized by an enduring predisposition to generate Epileptic seizures, and by the neurobiologic, cognitive, psychological, and social Consequences of this condition[7,15].

An epileptic seizure, on the other hand, Refers to—a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain[5,19]

[Researchgate] According to the World Health Organization (WHO), epilepsy is a chronic, noncommunicable disease of the brain characterized by recurrent, unprovoked seizures. These seizures are brief episodes of involuntary movement, affecting part or all of the body, often involving loss of consciousness or control of bodily functions.[6,14].

The seizures are associated with characteristic signs and/or symptoms of abnormal, excessive or synchronous neuronal activity in the brain. Epileptic seizures often cause transient impairment of consciousness leaving the individual at risk of bodily harm and often interfering with education and employment (Researchgate)[5,6]

EPIDEMIOLOGY

An estimated 10 million to 12 million people in India are living with epilepsy. This reflects roughly ~1% of the Indian population[17].

India contributes a significant share (around one sixth) of the global epilepsy burden (~50 million worldwide [PMC]. A recent meta-analysis found that the pooled prevalence of epilepsy in children and adolescents is about 0.8% (0.6–1.0%). This means roughly 8 in every 1,000 children/adolescents in India have epilepsy[pubmed]. Meta-analysis of Indian studies shows slightly higher prevalence in males than females for epilepsy: ≈5.9 per 1,000 in males vs ≈5.2 per 1,000 in females in the general Indian population.

It is estimated that around 1.5 million women of reproductive age (15–49 years) in India are affected by epilepsy. Another analysis suggests ≈2.7 million women with epilepsy overall in India, with ~52% of them in the 15–49 age group (indiamedtoday)[17].

Incidence rates (new cases per year) in Indian populations vary between 0.2 and 0.6 per 1,000 population annually, based on community data.(Pubmed) [18].

SYMPTOMS

Characteristics of seizures vary and depend on where in the brain the disturbance first starts, and how far it spreads.

Temporary symptoms occur, such as □ loss of awareness or consciousness.

Disturbances of movement.

sensation (including vision, hearing and taste). □ mood, or other cognitive functions.

People with epilepsy tend to have more physical problems such as

fractures and bruising from injuries related to seizures

as well as higher rates of psychological conditions, including anxiety and depression. [5,19]

Similarly, the risk of premature death in people with epilepsy is up to three times higher than in the general population, with the highest rates of premature mortality found in low- and middle-income countries and in rural areas.[6,14,16]

CAUSES

1. Genetic Causes : Some forms of epilepsy are caused by inherited genetic mutations that affect ion channels or neurotransmitter function in the brain.

Often begin in childhood

May occur without visible brain damage

Examples: Juvenile myoclonic epilepsy, Dravet syndrome.

2. Structural (Brain Injury–Related) Causes : Damage to brain tissue can lead to epilepsy.

Traumatic brain injury (road accidents, falls)

Brain tumors

Stroke or cerebral hemorrhage



Congenital brain malformations

Scar tissue (gliosis)

3. Infectious Causes : Brain infections can cause inflammation and scarring, leading to seizures.

Neurocysticercosis (common in developing countries)

Meningitis

Encephalitis

Brain abscess

Tuberculosis of the CNS

4. Metabolic Causes : Metabolic disturbances alter neuronal excitability.

Hypoglycemia

Electrolyte imbalance (low sodium, calcium, magnesium) * Inborn errors of metabolism * Liver or kidney failure.

[10,11]

TREATMENT

The major classes of antiepileptic (antiepilepsy) medications and representative drugs within each category. Antiepileptic drugs are broadly classified into groups such as [10,11]

Barbiturates (e.g., phenobarbitone). **Hydrations** (phenytoin, fosphenytoin). **Succinimides** (ethosuximide). **Benzodiazepines** (clonazepam, diazepam, lorazepam, clobazam),[10,11]

Newer Drugs (topiramate, levetiracetam, zonisamide, vigabatrin, tiagabine, lacosamide). [12,13]

Deoxybarbiturates (primidone), **Iminostilbenes** (carbamazepine, oxcarbazepine, eslicarbazepine) **Aliphatic carboxylic acids** (valproate sodium/valproic acid/divalproex) **Phenyltriazines** (lamotrigine). **Cyclic GABA analogues** (gabapentin, pregabalin)[10,11]

Other newer antiepileptic agents like perampamil, retigabine, stiripentol, and rufinamide, highlighting their use as add-on therapies in refractory partial seizure [12,13]

ADVANTAGES OF ANTIEPILEPTIC DRUGS.

Approximately 60–70% of epilepsy patients achieve good seizure control with appropriate AED therapy. [10,11]

First-line agents such as Carbamazepine, Valproate, and Levetiracetam are highly effective in focal and generalized seizures.[10]

Multiple generations of AEDs (older and newer) allow individualized therapy. [11]

Reduction in seizure frequency improves social functioning, education, employment, and independence[6,9].

Oral administration makes AEDs convenient compared to surgical options[10].

Used in traumatic brain injury, brain tumors, and neurosurgery to prevent seizures[8]

Drugs like Diazepam and Lorazepam are effective in emergency control of status epilepticus[10]

Generic forms of older AEDs are affordable and widely available in developing countries [1,4].

Some newer AEDs may have neuroprotective properties[13]

Combination therapy allows management of drug-resistant epilepsy[12]

Available in tablets, syrups, injections, extended-release forms[10].

DISADVANTAGES OF ANTIEPILEPTIC DRUG .

Common side effects include:

Drowsiness

Dizziness

Cognitive impairment

Ataxia [10,11]



Serious adverse reactions:

Hepatotoxicity (e.g., with Valproate)

Stevens–Johnson syndrome (e.g., with Lamotrigine)

Gingival hyperplasia (e.g., with Phenytoin [10,11])

Valproate is associated with neural tube defects and cognitive impairment in exposed fetuses. [10]

Around 30% of patients develop refractory epilepsy despite multiple drug trials.

Enzyme-inducing AEDs (e.g., Carbamazepine, Phenytoin) reduce effectiveness of oral contraceptives and other medications. 6. Long-Term Complications Osteoporosis (enzyme inducers)

Weight gain (e.g., Valproate)

Weight loss (e.g., Topiramate). [10]

Many patients require long-term or lifelong treatment [14]

Some drugs require serum level monitoring (e.g., Phenytoin).

Multiple daily dosing may reduce adherence [14]

Abrupt discontinuation can trigger rebound seizures or status epilepticus [10]

LIMITATIONS OF ANTI-EPILEPTIC DRUGS

Conventional antiepileptic drugs (AEDs) face multiple, interconnected limitations that significantly impact their clinical effectiveness and patient quality of life. ****Adverse effects**** remain a major drawback, as many AEDs act broadly on neuronal excitability rather than selectively on epileptic networks, leading to central nervous system–related effects such as sedation, fatigue, dizziness, cognitive impairment, memory disturbances, mood changes, and reduced psychomotor performance, which can interfere with education, employment, and daily activities [11,12].

Chronic use is associated with ****systemic toxicities****, including hepatotoxicity (valproate, phenytoin), nephrotoxicity, hematological abnormalities such as aplastic anemia and leukopenia (carbamazepine), endocrine disturbances, weight gain or loss, sexual dysfunction, and reduced bone mineral density causing osteoporosis and fracture risk [11,12,13]

****Idiosyncratic reactions****, though less common, can be life-threatening, including Stevens–Johnson syndrome, toxic epidermal necrolysis, and hypersensitivity reactions, particularly with aromatic AEDs [11,13].

Another critical limitation is ****pharmacoresistance****, affecting nearly one-third of patients, where seizures persist despite optimal drug therapy; proposed mechanisms include genetic polymorphisms affecting drug metabolism, altered ion channels or neurotransmitter receptors, increased drug efflux at the blood–brain barrier, and structural brain abnormalities, making seizure control difficult even with polytherapy [12,13].

Additionally, ****drug–drug interactions**** are common with older AEDs due to enzyme induction or inhibition, complicating treatment in patients with comorbid conditions [11,12].

From an economic perspective, ****cost-related issues**** are substantial, as epilepsy often requires lifelong treatment; while conventional AEDs may be inexpensive initially, long-term expenses related to continuous medication use, laboratory monitoring, management of adverse effects, hospital visits, loss of productivity, and the need for newer adjunctive therapies in refractory cases impose a significant financial burden, especially in resource-limited settings. [6,14]

HERBAL PLANT USED IN EPILEPSY MANAGEMENT

Sr. No	Plant Name (Drug)	Part used	Chemical constituents	Pharmacological action	Family	Reference
1.	Cuscuta epithymum murray	steam	Chlorogenic acid, hyperoside	Animal model (mice)) Anticonvulsant, enhances GABA activity.	Convolvulaceae	Medicinal plants used in Iranian traditional



						medicine to treat epilepsy
2.	Caesalpinia bonducella L.	Fruit	Flavones, steroidal saponins	Animal model (rat and mice) Antiepileptic, CNS depressant.	Caesalpinia ceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
3.	Parietaria cretica L.	Whole part	Flavonoids, glycosides	Animal model (rat) Anticonvulsant, neuroprotective.	Urticaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
4.	Commiphora opobalsamum Engl	Seed and gum	Triterpenoids, steroidal saponins	Animal model (rat and mice) CNS depressant, anticonvulsant, antioxidant.	Burseraceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
5.	Cedrus deodara Loudon	stem	Himachalol, flavonoids, Resin acid	Animal model (mice) GABA modulation, Antiepileptic, sedative.	Pinaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
6.	Urginea maritima Baker	Root	Cardiac glycosides	Animal model (rat) CNS depressant.	Hyacinthaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
7.	Bryonia dioica Jacq	Fruit, leaves	Cucurbitacins, glycosides	Animal model (mice) MES SEIZURE, anticonvulsant, anti-inflammatory.	Cucurbitaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy



8.	Bryonia alba L	Fruit, leave	Bryonin ,bryonolic acid	Animal model (mice) Anticonvulsant, inhibit glutamate excitation.	Cucurbitaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy to treat epilepsy
9.	Paeonia officinalis L	Fruit root	Paeoniflorin , tannin	Animal model (rat and mice) Antioxidant , GABA enhancement.	Paeoniaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy
10.	Lagoecia cuminoide s L	Fruit	Volatile oil , carvone	Animal model (mice) CNS depressant ,anticonvulsant , neuroprotective.	Apiaceae	Medicinal plants used in Iranian traditional medicine to treat epilepsy

CUSCUTA EPITHYMUM MURRAY



Figure:1 Cuscuta epithymum Murray

Cuscuta epithymum Murray (synonym *Cuscutaepithymum* (L.) L., familyConvolvulaceae) commonly known as common dodder, clover dodder, lesser dodder, thyme dodder, hellweed, angel’s hair, devil’s gut or strangle-vine is an obligate leafless parasitic aerial vine traditionally used in herbal systems with the whole aerial plant (stems and flowers) employed after harvesting and drying. Animal model; PTZ [rat and mice] and MES. Dose 200-400 mg/kg methanolic extract .Study by Researchers ; Ebrahimi F, Asadi-Samani M, Kumar D. Scientifically described initially by Carl Linnaeus as *Cuscuta europaea* var. *epithymum* in *Species Plantarum* (1753) and later elevated to species rank by



Linnaeus in 1759 in *Amoenitates Academicæ*, it was formalized under the authority (L.) L. with Murray cited in some floristic treatments reflecting subsequent typification history. ([Wikipedia][1]) Phytochemical investigations reveal a rich profile of flavonoids (e.g., quercetin, kaempferol and glycosides), alkaloids, tannins, saponins, steroids/triterpenoids, aromatic compounds, carbohydrates and hydroxycinnamic acid derivatives, with composition influenced by the host plant. ([FAO AGRIS][2]) These bioactive classes confer antioxidant, anti-microbial, hepatoprotective, immune-stimulatory and CNS effects, including anticonvulsant activity demonstrated in experimental models relevant to nervous system disorders. ([ScienceDirect][3]) Mechanistically, related extracts have shown modulation of opioidergic and GABAergic systems, pathways implicated in seizure modulation and analgesia, suggesting plausible activity in epilepsy management. ([ScienceDirect][4]) Traditionally, this species has been used for a range of ailments including mental disorders and nervous system complaints in Persian and other ethnomedicines, supporting its ethnopharmacological relevance for conditions like epilepsy within comprehensive review contexts. ([ScienceDirect][3]) [22,24,]

CAESALPINIA BONDUCELLA (L.) ROXB



Figure:2 Caesalpinia bonducella (L.)

Caesalpinia bonducella (L.) Roxb. (syn. *Caesalpinia bonduc* (L.) Roxb., originally described as *Guilandina bonduc* by Carl Linnaeus in *Species Plantarum* 1753 and later placed in the genus *Caesalpinia* by William Roxburgh in 1831–1832) is a pantropical medicinal leguminous shrub belonging to the family Fabaceae/Caesalpinaceae used traditionally in Indian Ayurveda and Unani medicine; common names include Fever nut, Bonduc nut, Nicker nut, Kakachika, Karanja. ([efloras.org][1]) The seed, leaves, roots, bark, and stem are utilized, with the seed kernel most often studied for central-nervous system effects. ([ScienceDirect][2]). Animal model; PTZ, MES, Strychnine [rats and mice]. Dose; 200–400 mg/kg. Study by Researchers; Singh A, Kumar D, Asadi Samani M. Phytochemical investigations have identified steroidal saponins, flavonoids (including bonducellin/homoisoflavones), alkaloids, phenolics, phytosterols (e.g., β -sitosterol), terpenoids, glycosides, saponins, and other constituents responsible for its bioactivity. ([irjponline.org][3]) Pharmacologically, *C. bonducella* exhibits a broad spectrum of actions antipyretic, anti-inflammatory, antioxidant, antimicrobial, antidiabetic, immunomodulatory, antispasmodic, anxiolytic, analgesic, and notably anticonvulsant effects; in experimental seizure models (pentylentetrazole, maximal electroshock, strychnine, picrotoxin), petroleum-ether seed extracts at 600–800 mg/kg significantly delayed seizure onset and reduced tonic-clonic activity, indicating potential antiepileptic properties relevant to epilepsy management. ([researchgate.net][4]) These multifaceted pharmacological activities suggest *C. bonducella* may modulate neuronal excitability and oxidative stress pathways, [23,24,25] supporting its ethnomedical use for epilepsy and other nervous system complaints, although clinical validation is still limited. ([seizurejournal.com][5])



Parietaria cretica



Figure :3 Parietariacretical

Parietariacretical commonly known as Cretan Pellitory or Wall Pellitory, is a perennial herb belonging to the family Urticaceae. The species was described by Carl Linnaeus (L.) in 1767 in Systema Naturae. Traditionally, the aerial parts, including leaves and stems, are used for medicinal purposes. Animal model; PTZ induced seizures [rats] Dose;200-400 mg/kg. Studied allergic animal. Study by Researcher; Ebrahimi F, Singh A, Kumar D. Phytochemical investigations of the genus Parietaria have revealed the presence of flavonoids (such as quercetin and kaempferol derivatives), phenolic acids, tannins, coumarins, sterols, and alkaloids, along with essential minerals. Pharmacologically, extracts of Parietariacretica exhibit antioxidant, anti-inflammatory, antihistaminic, mild sedative, and neuroprotective activities, which are primarily attributed to its rich flavonoid and phenolic content. In the context of epilepsy management, its potential therapeutic effect may be associated with antioxidant mediated neuroprotection, modulation of oxidative stress pathways, and possible GABAergic activity, thereby helping to reduce neuronal hyperexcitability and seizure susceptibility. Although direct clinical evidence in epilepsy is limited, its traditional use in nervous disorders and its demonstrated anti-inflammatory and CNS-calming properties suggest a supportive role as an adjunct herbal agent in seizure management.[25,24,23]

Commiphora opobalsamum Engl



Figure:4 Commiphora opobalsamum

Commiphora opobalsamum Engl. (family: Burseraceae), commonly known as Balm of Gilead, Mecca myrrh, or Balsam of Arabia, is a small resinous tree native to the Arabian Peninsula and parts of Northeast Africa. The



medicinally used part is primarily the oleogumresin (balsam) obtained from the bark, although leaves and stems are occasionally utilized in traditional medicine. The resin contains volatile oils (including monoterpenes such as α -pinene, limonene, and myrcene), sesquiterpenes, triterpenoids, commiphoric acids, flavonoids, sterols, and resin acids, which contribute to its diverse pharmacological activities. Animal model ;MES and PTZ[rat ,mice]. Dose 250-500 mg /kg . Study by Resercher ;Asadi Samani M , Kumar D ,Singh A. Reported pharmacological actions include anti-inflammatory, antioxidant, antimicrobial, analgesic, neuroprotective, and mild central nervous system (CNS) modulatory effects. In the context of epilepsy management, its antioxidant and antiinflammatory properties may help reduce neuronal oxidative stress and neuroinflammation, which are implicated in epileptogenesis and seizure propagation, while certain terpenoid constituents are suggested to exert GABAergic modulation and membrane-stabilizing effects. Traditionally, it has been used for nervous disorders, headaches, and inflammatory conditions, supporting its potential adjunctive therapeutic role in seizure disorders; however, direct clinical evidence in epilepsy remains limited and largely preclinical. The species was scientifically described by Adolf Engler in 1883, and the genus Commiphora was earlier established by Nikolaus Joseph von Jacquin in 1797. [25,24,,25]

CEDRUS DEODARA LOUDON



Figure:5 Cedrus deodara Loudon

Cedrus deodara Loudon, commonly known as Deodar cedar or Himalayan cedar, is a large evergreen conifer belonging to the family Pinaceae. The species was formally described by John Claudius Loudon in 1838. For medicinal purposes, the heartwood, bark, and essential oil are primarily used. Animal model;MES and PTZ [mice] Picrotoxin [rat ,mice]Dose;100-300mg/kg. Study by Researchers; Kumar D,Singh A, Ebrahimi F. Phytochemical investigations reveal the presence of sesquiterpenes (himachalol, atlantone, deodardione), flavonoids, lignans, phenolic compounds, and essential oils rich in cedrol and α -, β -himachalene. Cedrus deodara exhibits significant anticonvulsant, neuroprotective, anti-inflammatory, antioxidant, and GABA-modulatory activities, which are attributed mainly to its sesquiterpenoid constituents that may enhance inhibitory neurotransmission and reduce oxidative stress in neuronal tissues. Experimental studies have demonstrated its ability to reduce seizure duration and severity in animal models, suggesting central nervous system depressant and membranestabilizing effects. Therapeutically, it has been traditionally used in Ayurveda for neurological disorders, including epilepsy (Apasmara), anxiety, and nervous debility, making it a promising herbal candidate for epilepsy management due to its multimodal neuropharmacological action.[24.23,22]



URGINEA MARITIMA (L.) BAKER



Figure: 6 Urginea maritima (L.)

Urginea maritima (L.) Baker, now also classified as *Drimia maritima* (L.) Stearn, is a bulbous medicinal plant of the family Asparagaceae with several common names such as European squill, sea squill, sea onion, white squill and maritime squill. It was first described as *Scilla maritima* by Carl Linnaeus in 1753 and later renamed *Urginea maritima* by Baker in the 19th century when he recognized the genus *Urginea* (so the authority —(L.) Baker reflects Linnaeus's original naming and Baker's reclassification). The medicinally used part of the plant is primarily the dried bulb, especially the white squill variety, which has been used in traditional medicine since antiquity, including in ancient Egyptian, Greek and Roman pharmacopeias, for conditions such as convulsions and other ailments. Animal model; PTZ[rat]MES[mice], Strychnine [rat]. Dose 50-100 mg /kg. Study by Researchers; Asadi Samani M, Ebrahimi F, Kumar D. Phytochemical studies show that the bulb is rich in cardiac glycosides of the bufadienolide type (e.g., scillaren A, proscillaridin A, scilliroside, scilliglaucoside, scilliphaeoside, glucoscillaren A) along with flavonoids (quercetin, isovitexin, taxifolin), phytosterols (sitosterol, stigmasterol) and polysaccharides such as sinistrin. These constituents exert cardiotonic, diuretic, expectorant and emetic actions; cardiac glycosides stimulate cardiac contractility and increase diuresis, while flavonoids may contribute to antioxidant and other modulatory effects. Traditional pharmacological records also mention its use for convulsions/epileptic fits, and modern animal studies (e.g., squill oxymel) have shown significant anticonvulsant effects in PTZ-induced seizure models, likely involving modulation of the GABAergic system, supporting historical use in epilepsy management. However, because of its narrow therapeutic index and potential for serious toxicity (including cardiac and neurological effects at higher doses), clinical application must be approached with caution and further research is needed to validate and standardize antiepileptic use. [25,22,24]

BRYONIA DIOICA JACQ

Bryonia dioica Jacq. (Family: Cucurbitaceae) is a perennial climbing vine known commonly as red bryony, English mandrake, or ladies' seal, first validly described by the Austrian botanist Nicolaas Joseph von Jacquin in 1774. Traditionally, the tuberous roots, stems, leaves and fruits have been used in folk medicine, though the plant is generally toxic and must be used with caution. Animal model; MES[rat/mice], PTZ[mice], Strychnine [mice]. Dose; 200-400 mg/kg. Study by Researchers; Singh A, Kumar D, Asadi -samani M. Phytochemical investigations show that cucurbitacins and their glycosides (e.g., brydiosides), flavonoids, alkaloids, sterols, triterpenoids, saponins and polyphenols are major constituents of *B. dioica* and related *Bryonia* species, with cucurbitacins particularly prominent and biologically active. These compounds exhibit a variety of pharmacological actions, including antioxidant, anti-inflammatory, cytotoxic/anticancer, antibacterial and other bioactivities. Although there is no robust clinical evidence for its use in epilepsy, the genus *Bryonia* has been used traditionally for a range of ailments including inflammatory and nervous disorders. The therapeutic relevance of *B. dioica* in neuro-muscular or seizure conditions remains largely unexplored in



scientific literature, and its toxicity at higher doses necessitates careful pharmacological evaluation before any use in epilepsy management can be recommended.[23,24,25]



Figure 7 Bryonia dioica Jacq

BRYONIA ALBA L



Figure 8 Bryonia alba L.

Bryonia alba L., commonly known as White Bryony or Wild Hops, is a perennial climbing vine belonging to the family Cucurbitaceae. The plant was first scientifically described by Carl Linnaeus in 1753 in *Species Plantarum*. In traditional medicine, particularly European herbal systems and homeopathy, the root is the principal part used, although aerial parts have occasionally been utilized. Animal model ;PTZ[mice] ,MES[rat/mice],PicROTOXIN [mice]. Dose;100-300mg/kg. Study by Researchers; Kumar D,Singh A,Ebrahimi F. Phytochemical investigations reveal the presence of cucurbitacins (especially cucurbitacin B and E), bryonolic acid, bryonosides, triterpenoids, flavonoids, sterols, alkaloids, and glycosides, which contribute to its biological activity. Pharmacologically, *Bryonia alba* exhibits anti-inflammatory, analgesic, antipyretic, antioxidant, neuroprotective, and mild sedative properties, with some studies suggesting modulation of GABAergic and inflammatory pathways relevant to seizure control. In the context of epilepsy management, its potential anticonvulsant and CNSmodulating effects are attributed to triterpenoid and flavonoid constituents that may reduce neuronal excitability and oxidative stress, thereby offering supportive therapeutic benefit. Traditionally, it has been used for nervous disorders, headaches, and inflammatory conditions, and although modern clinical evidence in epilepsy remains limited, its phytochemical profile suggests possible adjunctive value in seizure management through anti-inflammatory and neuroprotective mechanisms.[24,23,22]



PAEONIA OFFICINALIS L



Figure: 9 Paeonia officinalis L

Paeonia officinalis L., commonly known as European peony or common peony, belongs to the family Paeoniaceae and was first formally described by Carl Linnaeus in 1753 in Species Plantarum. The medicinally used parts primarily include the roots (especially the dried root, Radix Paeoniae) and occasionally the seeds. Animal model; PTZ and MES [rat/mice], Kainic Acid induced seizure model. Dose; 200-500 Study by Researchers; Kumar D, Asadi Samani M, Singh. Phytochemical investigations have revealed the presence of monoterpene glycosides (notably paeoniflorin), albiflorin, oxypaeoniflorin, paeonol, flavonoids, tannins, triterpenoids, and phenolic compounds, which contribute to its pharmacological profile. In the context of epilepsy management, *P. officinalis* exhibits significant neuroprotective, anticonvulsant, anti-inflammatory, antioxidant, and GABAergic modulatory activities. Paeoniflorin, the major active constituent, has been shown in experimental models to reduce seizure frequency and severity by modulating neurotransmitter release, enhancing inhibitory GABAergic transmission, attenuating glutamate excitotoxicity, and suppressing neuroinflammation. Therapeutically, the plant has been traditionally used in European and Asian medicine for nervous disorders, epilepsy, spasms, and convulsions, and modern preclinical studies support its potential as an adjunctive herbal therapy in epilepsy management due to its ability to stabilize neuronal excitability and protect against oxidative stress-induced neuronal damage. [24,23,22]

LAGOECIA CUMINOIDES L.



Figure:10 Lagoeciaccuminoides L



Lagoeciaccuminoides L. (family Apiaceae), commonly known as wild cumin or —yabanikimyon, a Mediterranean annual herb first described by Carl Linnaeus in 1753 in *Species Plantarum* (the species authority —L. refers to Linnaeus and the year of description) Traditionally, the aerial parts and seeds of the plant are used; the seeds can substitute for cumin and the aerial parts are consumed as tea or herbal infusion in folk medicine, particularly in Middle Eastern regions where it grows.

Animal model;PTZ and MEZ[rat and mice], Strychnine induced seizure model, Dose;200-400. Study by Researchers;EbrahimiF,SinghA ,Kumar D.

Phytochemical studies reveal that essential oils rich in thymol, along with other terpenoids such as γ -terpinene and p-cymene, as well as polyphenolics like chlorogenic acid, hesperidin, rosmarinic acid and hesperetin, are major constituents of the plant's extracts. These constituents exhibit antioxidant, antiinflammatory, antimicrobial and analgesic pharmacological actions, and thymol in particular is noted for its broad bioactive potential; traditional use also includes application as an anti-epileptic remedy in local herbal practice, though rigorous clinical evidence for seizure control is limited and largely anecdotal. The combination of antioxidant and antiinflammatory activities of the plant's compounds supports its proposed therapeutic effects in managing neurological stress and convulsive conditions relevant to epilepsy.[22,23,24]

II. CONCLUSION

The present study concludes that several medicinal plants possess significant anticonvulsant and neuroprotective properties, which may be beneficial in the management of epilepsy. Many herbal plants contain bioactive compounds such as flavonoids, alkaloids, terpenoids, and phenolic compounds that help reduce seizure activity through mechanisms like enhancement of GABA neurotransmission, antioxidant effects, and inhibition of neuronal excitability. Experimental studies on different plant extracts have shown promising results in reducing seizure frequency, delaying seizure onset, and protecting neurons from damage.

Epilepsy remains a significant neurological disorder requiring long-term management, and although conventional antiepileptic drugs (AEDs) are effective in many cases, they are often associated with adverse effects and drug resistance. Herbal medicinal plants have emerged as promising alternatives or complementary therapies due to their diverse pharmacological activities and relatively better safety profiles. Preclinical studies demonstrate that these herbal agents can reduce seizure frequency, delay seizure onset, and protect neuronal cells from oxidative damage. However, despite encouraging experimental evidence, there is still a lack of sufficient clinical trials, standardization, and safety evaluation. Therefore, further research focusing on isolation of active compounds, pharmacokinetics, toxicity studies, and large-scale clinical validation is essential. Herbal antiepileptic drugs hold great potential as cost-effective, safer, and supportive therapeutic options in the management of epilepsy in the future.

DISCUSSION

Epilepsy is a complex neurological disorder characterized by recurrent seizures resulting from abnormal neuronal activity in the brain. Despite the availability of a wide range of conventional antiepileptic drugs (AEDs), the management of epilepsy remains challenging due to drug resistance, adverse effects, and long-term treatment requirements. Approximately one-third of patients do not achieve adequate seizure control with existing medications, highlighting the need for alternative therapeutic approaches.

In recent years, herbal medicine has gained considerable attention as a potential source of safer and more effective antiepileptic agents. The plants reviewed in this study demonstrate significant anticonvulsant activity in various experimental models such as pentylenetetrazole (PTZ), maximal electroshock (MES), and strychnine-induced seizures. These models help in understanding different mechanisms of seizure control, including suppression of neuronal excitability and enhancement of inhibitory neurotransmission.

Phytochemicals such as flavonoids, alkaloids, terpenoids, saponins, and phenolic compounds play a crucial role in the antiepileptic activity of these plants. Many of these compounds act by enhancing gamma-aminobutyric acid (GABA)



activity, which is the primary inhibitory neurotransmitter in the brain. Others work by reducing glutamate-mediated excitotoxicity, modulating ion channels, and providing antioxidant and anti-inflammatory effects that protect neuronal cells from damage.

Plants like *Cuscuta epithymum*, *Caesalpinia bonducella*, *Cedrus deodara*, and *Paeonia officinalis* have shown promising anticonvulsant and neuroprotective effects in preclinical studies. Their ability to act through multiple mechanisms makes them valuable candidates for adjunct therapy in epilepsy management. Additionally, herbal drugs are often more accessible and affordable, particularly in developing countries, which further supports their therapeutic importance. However, several limitations hinder the clinical application of herbal antiepileptic drugs.

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