JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

EPILEPSY AND ITS MANAGEMENT: A SYSTEMATIC REVIEW

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Abstract: Epilepsy, a neurological disorder characterized by recurrent epileptic seizures, affects millions of individuals worldwide and poses significant challenges to healthcare systems and affected individuals alike. Despite advancements in diagnosis and treatment, the burden of epilepsy remains high, particularly in developing countries where access to care is limited. This review explores the current understanding of epilepsy, including its classification, diagnostic evaluation, pharmacological and non-pharmacological management, FDA-approved drugs, challenges in management, unmet needs, and future directions. Key strategies to reduce the burden of epilepsy include minimizing diagnostic errors, improving access to diagnostic tools and treatments, preventing symptomatic epilepsies through early identification of comorbidities, addressing socioeconomic disparities, raising awareness about epilepsy-related mortality, quantifying epilepsy-related costs, and providing support for caregivers. Collaborative efforts are essential to address these challenges and improve outcomes for individuals living with epilepsy.

Index Term: Epilepsy, Epileptic, Seizures, antiepileptic drugs.

I.INTRODUCTION

Epilepsy is a neurological disorder characterized by a persistent tendency to experience epileptic seizures.[1] Epilepsy impacts approximately 50 million individuals worldwide,[2] with the highest prevalence observed among children and older adults. Those living with epilepsy experience increased risks of injury and premature mortality compared to the general population. Additionally, many individuals with epilepsy experience a diminished quality of life, even if their seizures are effectively controlled. [3-5] Individuals living with epilepsy face discrimination, social stigma, and misunderstanding,[6] adding to the burden of coping with a chronic and unpredictable condition that may compromise their autonomy in daily activities. Despite the potential for successful treatment in many cases, there exists a significant treatment gap, particularly in low- and middle-income countries,[7] where antiepileptic medications are often inaccessible or prohibitively expensive.[8]

Epilepsy is recognized as the most prevalent serious neurological disorder, affecting approximately 0.5% of the population. While the majority of individuals with epilepsy can achieve seizure freedom and lead normal lives through appropriate drug therapy, a significant proportion around 30% to 40% continue to experience seizures despite treatment with antiepileptic drugs (AEDs), whether used alone or in combination.[9] Epilepsy imposes a considerable impact on the quality of life for both affected individuals and their families. Following the introduction of bromide as an antiseizure medication in 1857, there has been a remarkable proliferation of therapies demonstrating clinical effectiveness in reducing the frequency and severity of seizures in individuals with epilepsy. These therapeutic interventions, collectively known as "antiepileptic drugs" (AEDs), have significantly expanded treatment options for managing epilepsy symptoms.[10]

Epilepsy can arise from a combination of genetic and acquired factors, with their interaction contributing to the condition in many cases.[11][12] Common acquired causes include severe brain trauma, stroke, tumors, and brain complications resulting from prior infections.[11] Approximately 60% of cases have an unknown cause.[13][14] Genetic,

congenital, or developmental conditions tend to be more prevalent among younger individuals, while brain tumours and strokes are more frequently observed in older populations.[13] Seizures may also manifest as a result of other health issues.[15] When seizures occur in close proximity to a specific cause, such as a stroke, head injury, toxic ingestion, or metabolic disturbance, they are categorized as acute symptomatic seizures. These fall under the broader classification of seizure-related disorders, distinct from epilepsy itself.[16][17]

II.TERMINOLOGY

2.1 Defination

An epileptic seizure is described by the International League against Epilepsy (ILAE) as "a transient display of signs and/or symptoms arising from abnormal, excessive, or synchronized neuronal activity within the brain." Epilepsy is conceptually depicted as a "continual vulnerability of the brain to generate epileptic seizures, resulting in neurobiological, cognitive, psychological, and social consequences."[1]

2.2 Classification

Numerous efforts have been undertaken to organize and categorize seizures and epilepsies.[18,19] In light of scientific progress over recent decades, the ILAE Commission on Classification and Terminology proposed revisions to nomenclature and methodology in 2010, introducing a flexible multidimensional framework.[20] Further refinements continue to emerge based on feedback from the epilepsy community.[21] The proposed changes involve replacing the term "partial" with "focal" to describe seizures originating within neuronal networks confined to one cerebral hemisphere. Additionally, focal seizures are no longer classified as simple or complex based solely on presumed alterations in consciousness level. Instead, a diagnosis of focal seizure is warranted when focal symptoms and signs are present, even if bilateral motor manifestations occur. Generalized seizures are believed to arise from bilaterally distributed cortical or cortical-subcortical networks that rapidly engage without a distinct focal point, potentially involving cortical and subcortical structures without necessarily affecting the entire cortex. While many syndromes may encompass both focal and generalized seizure types, efforts should be directed toward determining whether epilepsy stems from focal pathology, as this determination may carry implications for surgical interventions.[22]

An alternative proposal introduced in 2010 integrated the notion of brain networks connecting subcortical and cortical structures, as well as interconnections among different cortical areas. This framework allowed for the development of non-mutually exclusive etiological classifications, including genetic, structural, metabolic, and unknown factors.[23,24] Subsequently, ILAE Commissions introduced new classifications for seizures and epilepsies in 2017 (ILAE-EC). These classifications encompassed seizure types(table1), epilepsy types(fig1), and etiologies, aiming to provide a more comprehensive understanding of the diverse manifestations and underlying causes of epilepsy.[23,25,26,27] Common Genetic and Autoimmune Epilepsies.[28- 34]

Tabel 1. The 2017 International League Against Epilepsy classification of seizure Types

Focal onset	Generalised onset	Unknown onset
Motor onset • Automatism • Atonic • Clonic • Epileptic spasms • Hyperkinetic • Myoclonic • Tonic **Non-motor onset** • Autonomic • Behaviour arrest • Cognitive • Emotional • sensory	**Motor** • Tonic-clonic • Clonic • Myoclonic • Myoclonic-tonic-clonic • Myoclonic-atonic • Atonic • Epileptic spasms **Non-motor(absence)** • Typical • Atypical • Myoclonic • Eyelid	**Motor** • Tonic-clonic • Epilepteptic spasms **Non-motor** • Behaviour arrest **Unclassified**

Focal to bilateral tonic-clonic

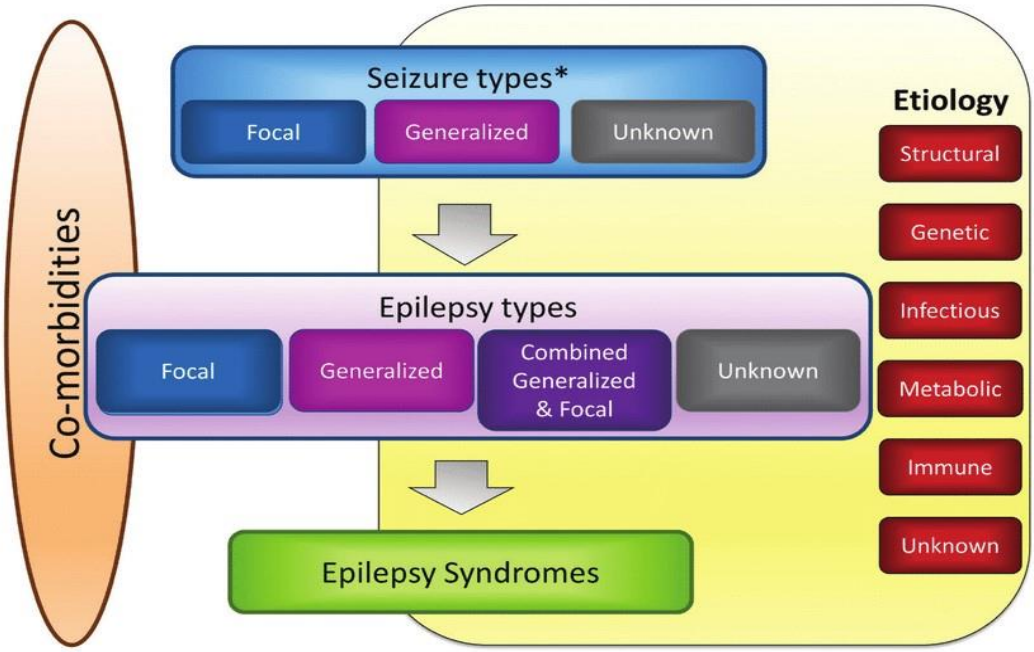

fig1.The 2017 International League Against Epilepsy classification of the epilepsie

Table 2: Common Genetic and Autoimmune Epilepsies

	Syndrome	**Genes and loci**
CommonGenetic Epilepsies	Febrile Seizures	8q13-q21 (FEB1), 19p (FEB2), 2q23-q24(FEB3), 5q15-q15(FEB4), 6q22-q24(FEB5), 18p11(FEB6)
	Genetic epilepsy with febrile seizures plus	SCN1A, SCN2A, SCN1B, GABRD, GABRG2, PCDH19
	Severe myoclonic epilepsy of infancy and related syndromes	SCN1A, SCN2A, GABRG2
	West syndrome and early infantile epileptic	ARX, CDK15, STXBP1

	encephalopathy with suppression-burst	
	Malignant migrating partial seizures of infancy	KCNT1
	Other early onset epilepsy	PLCB1, PCDH19, KCTD7, BCKDK, SYN1, GRIN2B, GRIN2A, TNK2, KCNQ2
	Benign familial neonatal convulsions	KCNQ2, KCNQ3
	Benign familial neonatal-infantile seizures	SCN2A
	Benign familial infantile seizures	PRRT2, ATP1A2
	infantile myoclonic epilepsy	
	Juvenile myoclonic epilepsy	TBC1D24
	Childhood absence epilepsy	EFHC1, GABRA1
	Epilepsy + paroxysmal exercise-induced dyskinesia	GABRG2, GABRA1, SLC2A
	Familial Autosomal dominant nocturnal frontal lobe epilepsy	SLC2A1
	Familial lateral temporal lobe epilepsy	CHRNA4, CHRNA2, CHRNB2, KCNT1
		LGI1

	Familial focal epilepsy with variable foci	DEPDC5
Common Autoimmune Epilepsies	Anti-NMDA antibody, Anti-GABAB antibody, Anti-AMPA antibody, Anti-LGI1 antibody, Anti-CASPR2 antibody, Anti-contactin-2 antibody, Anti-GADantibody, Anti-VGKC antibody	

III. DIAGNOSTIC EVALUATION

The National Institute for Health and Care Excellence (NICE) in the UK offers valuable recommendations regarding the initial diagnosis and management of individuals suspected of experiencing seizures and epilepsy, alongside providing care pathways.[35] Similarly, the American Academy of Neurology (AAN) has developed guidelines for assessing and treating patients who present with their first seizure episode.[36,37] Multiple diagnostic tools are employed to identify and categorize seizure types and syndromes, as well as their causes. These include electroencephalogram (EEG), magnetic resonance imaging (MRI), positron emission tomography (PET), single photon emission computed tomography (SPECT), magnetoencephalogram (MEG), and neuropsychological testing. EEG is particularly crucial as it records the brain's electrical activity, although epileptiform discharges detected in EEG aren't exclusive to epilepsy. Confirmation of seizures through EEG is only possible if a seizure occurs during the test. While EEG sensitivity for epilepsy is around 50%, and specificity is high at 98-99%, conducting serial EEGs can increase sensitivity to 80-90%. Additionally, specific activation techniques like hyperventilation, photic stimulation, and sleep deprivation can enhance the test's sensitivity.[30] Neuroimaging studies are essential in evaluating seizures and epilepsy to determine both structural and functional causes. The current standard neuro-radiological imaging method involves using a 3T brain MRI with coronal or oblique-coronal images utilizing T1-weighted, T2-weighted sequences, and fluid-attenuated inversion-recovery (FLAIR). While 1.5T brain MRI can also pinpoint seizure origins, its sensitivity is lower compared to the higher field strength of 3T MRI.[38] Generally, the sensitivity of MRI for identifying intractable epilepsy ranges from 82% to 86%.[39,40] In certain cases, additional imaging modalities such as functional neuro-imaging are employed for selected patients. PET reveals regional variations in metabolic activity, while SPECT examines regional disparities in blood flow during seizures (ictal SPECT) and between seizures (interictal SPECT). MR Spectroscopy (MRS) is utilized to assess the biochemical composition of the imaged tissue,[41-48] aiding in distinguishing between tumor and gliosis in lesions. MEG and functional MRI offer supplementary insights by helping localize potential epileptogenic lesions and identifying adjacent areas of eloquent cortex.[49]

IV. MANAGEMENT

4.1 Pharmacological Management

The main approach for treating provoked seizures involves addressing the underlying cause, such as correcting metabolic imbalances or treating infections.[30] Epilepsy, characterized by two unprovoked seizures occurring more than 24 hours apart, typically necessitates pharmacological intervention to prevent further seizures. Since the introduction of the first anticonvulsant, bromide, in 1857, a plethora of antiepileptic medications have been developed and prescribed. Presently, there are 29 different antiepileptic drugs available in the United States. Certain medications like benzodiazepines, lamotrigine, levetiracetam, topiramate, valproic acid, and zonisamide offer broad spectrum coverage, effectively treating both primary generalized and focal onset seizures. Others, such as carbamazepine and oxcarbazepine, demonstrate better efficacy in focal onset seizures. Only a handful of medications have level A evidence supporting their efficacy across various types of epilepsy and epilepsy syndromes, while most have lower levels of evidence. A recent review conducted by the International League Against Epilepsy on the efficacy and effectiveness of antiepileptic drugs as initial monotherapy found level A evidence supporting the use of levetiracetam, zonisamide, carbamazepine, and phenytoin in adult patients with partial onset seizures. Oxcarbazepine is the only medication with

level A evidence for children with partial onset seizures. Valproic acid and ethosuximide also demonstrate level A efficacy and effectiveness in children with absence seizures. However, other types of primary generalized epilepsy lack clear level A evidence, although there are multiple medications supported by level C and D evidence.[47]

When neurologists make decisions about which medications to prescribe for seizures, they take into account several factors including the evidence of effectiveness and efficacy, the classification of seizures, potential side effects, any existing comorbid conditions, as well as the patient's age and gender. For instance, valproic acid has been associated with a significant increase in the risk of major fetal malformations in women of childbearing age, while lamotrigine and levetiracetam are considered safer alternatives. Long-term use of antiepileptic drugs can lead to bone weakness. Certain medications that induce hepatic enzymes may have more drug interactions, potentially posing challenges in patients requiring anticoagulation, anti-tumoral, or anti-HIV treatments. Some antiepileptic drugs can also affect mood. Levetiracetam, for example, carries a higher risk of causing irritability, depression, and other mood disturbances, whereas lamotrigine and valproic acid may have mood-stabilizing effects. Additionally, specific medications such as topiramate and valproic acid have been found to be effective in treating migraine headaches.[30,49,50]

Despite the availability of numerous antiepileptic medications, approximately one third of patients with epilepsy continue to experience medically refractory seizures. Mohanraj and Brodie conducted a review of retrospective data focusing on the responses of adolescent and adult patients to sequential antiepileptic medication treatments in Scotland. They found that the overall response rates with the first, second, and third treatment schedules were 50.4%, 10.7%, and 2.7%, respectively. Moreover, only 0.8% of patients responded optimally to further drug trials. The response to the initial medication is a significant predictor of future seizure control.[51]

4.2 Nonpharmacoligical Managemaent

Medically intractable or refractory epilepsy is characterized by the inability to achieve seizure control despite adequate trials of two tolerated and appropriately chosen antiepileptic medication regimens with sufficient doses.[52]For patients with intractable epilepsy, alternative non-pharmacological treatments can be explored, including epilepsy surgery, neurostimulation therapy, and dietary interventions such as the ketogenic diet. Epilepsy surgery options may encompass focal resective surgery, multiple subpial transections, anterior corpus callosotomy, or hemispherectomy.[30] Neurostimulation therapies comprise interventions such as vagal nerve stimulation (VNS), responsive neurostimulation (RNS), and other investigational neurostimulation modalities. These approaches involve the use of electrical stimulation to modulate neural activity and potentially reduce seizure frequency in patients with epilepsy.[30,41]

For patients with medically resistant seizures, epilepsy surgery is typically considered if they possess a discernible seizure focus amenable to removal. An effective and secure alternative for individuals with focal onset epilepsy is represented by this treatment modality.[53-62] The occurrence of major complications arising from epilepsy surgery and subdural electrode evaluation is reported to be below 7%, with enduring permanent deficits manifesting in less than 2% of instances.[63-69]The evaluation process for epilepsy surgery usually begins with extended video-electroencephalogram (EEG) monitoring to verify the diagnosis and type of focal onset epilepsy, determine the seizure type and onset zone, and assess the disabling effects of ictal behavior.[53,70] Additionally, various neuro-radiological imaging techniques are employed to further pinpoint structural or functional epileptogenic lesions. Numerous studies have indicated that the prognosis of epilepsy surgery varies based on the etiology and location of the epileptogenic zone.[71-75]Radiographically detectable epileptogenic lesions offer valuable insights into the etiology and localization of epilepsy, thereby furnishing prognostic details for focal epilepsy surgery. The presence of a structural lesion on MRI typically signifies a favorable prognosis for epilepsy surgery, with 60-90% of patients achieving freedom from disabling seizures.[48,72] Temporal lobe epilepsy, particularly mesial temporal sclerosis (MTS), is the most prevalent form of epilepsy.[76]

MTS, identifiable on MRI by hippocampal atrophy and increased signal, is acknowledged to be medically resistant but tends to respond well to anterior temporal lobectomy, resulting in superior postoperative outcomes compared to other forms of temporal lobe epilepsy.[61,72,74] However, when MRI fails to detect a potential epileptogenic lesion, the likelihood of an excellent surgical outcome diminishes significantly, ranging from 20-65%.[53,54] This challenge may stem from the difficulty in localizing and excising the epileptogenic zone.[71] To enhance the radiographic detection of epileptogenic lesions, advanced imaging techniques such as 7T MRI, volumetric analysis, Diffusion Tensor Imaging (DTI), arterial spin labeling, and PET have been explored.[77-80] Nevertheless, it remains

unclear whether such techniques will enhance outcomes. Precise identification of the seizure origin is crucial for the effectiveness of surgical removal.[53,75] In cases where non-invasive tests yield inconclusive or conflicting results regarding potential seizure sites and regions of critical brain function, the placement of subdural grid electrodes (SDGE) and depth electrodes (DE) for invasive EEG monitoring becomes necessary.[69]

V. FDA APPROVED DRUGS (TABLE,3[81],4[82],5[83])

Table 3:First line antiepileptic drug

S.no	Drug	Dose (mg/day)	Mechanism of action	Adverse effect
01	Carbamazepine	200-400	Blocks voltage-gated sodium channels,reducing neuronal excitibility	dizziness, drowsiness, nausea, vomiting, headache, and rash. Rarely, it can cause serious skin reactions like Stevens-Johnson syndrome or toxic epidermal necrolysis.
02	Valproic acid	10-15	Increase GABA levels and may also inhibit voltage-gated sodium channels and T-type calcium channels.	drowsiness, dizziness, nausea, vomiting, diarrhea, and weight gain. Rarely, it can cause liver toxicity or pancreatitis.
03	Lamotrigine	25	Inhibits voltage-gated sodium channels, stabilizing neuronal membranes.	dizziness, drowsiness, headache, nausea, vomiting, and rash. Serious rashes such as Stevens-Johnson syndrome or toxic
04	Phenytoin	100	Blocks voltage-gated sodium channels, reducing neuronal excitability	dizziness, drowsiness, nausea, vomiting, gingival hyperplasia, and skin rash. Long-term use can lead to adverse effects on bone health and connective tissues.
05	Etosuximidie	250	Blocks T-type calcium channels, reducing neuronal excitability in thalamic neurons.	drowsiness, dizziness, headache, nausea, vomiting, and gastrointestinal disturbances.

06	Phenobarbital	1-3	Enhances the effects of GABA by increasing the duration of chloride channel opening.	dizziness, confusion, respiratory depression, and dependence with long-term use.
07	Gabapentin	300	Binds to the α2δ subunit of voltage-gated calcium channels, reducing calcium influx and inhibiting excitatory neurotransmitter release.	dizziness, drowsiness, ataxia, fatigue, and peripheral edema.
08	Pregabalin	150	Binds to the α2δ subunit of voltage-gated calcium channels, reducing calcium influx and inhibiting excitatory neurotransmitter release.	dizziness, drowsiness, dry mouth, weight gain, and peripheral edema.
09	Levetiracetam	500	Binds to synaptic vesicle protein SV2A, modulating neurotransmitter release.	dizziness, drowsiness, irritability, mood changes, and fatigue.
10	oxcarbazepine	300	Blocks voltage-gated sodium channels, reducing neuronal excitability.	dizziness, drowsiness, headache, nausea, vomiting, and hyponatremia.

Table 4:Second line antiepileptic drug

S.no	Drug	Dose (mg/day)	Mechanism of action	Adverse effect
01	Topiramate	25	Enhances the activity of GABA, inhibits glutamate receptors, and may also inhibit voltage-gated sodium channels.	cognitive impairment, somnolence, dizziness, weight loss, and paresthesia.
02	Clonazepam	0.5	Enhances the effects of GABA by increasing the duration of chloride channel opening.	drowsiness, sedation, confusion, and ataxia.
03	Zonisamide	100	Blocks voltage-gated sodium channels and T-type calcium channels, reducing neuronal excitability.	drowsiness, dizziness, loss of appetite, weight loss, and kidney stones.
04	Lacosamide	50	Enhances the slow inactivation of voltage-gated sodium channels.	dizziness, headache, nausea, and double vision.
05	Rufinamide	400-800	Prolongs the inactive state of voltage-gated sodium channels.	dizziness, drowsiness,

				nausea, and vomiting.
06	Perampanel	2	Antagonizes AMPA-type glutamate receptors, reducing excitatory neurotransmission.	dizziness, somnolence, irritability, and aggression.
07	Eslicarbazepine	400-800	Blocks voltage-gated sodium channels, reducing neuronal excitability.	dizziness, drowsiness, headache, and nausea.
08	Gabapentin enacarbil	600	Prodrug of gabapentin, metabolized to gabapentin in the body.	dizziness, drowsiness, headache, and nausea.
09	Ezogabine	50	Opens voltage-gated potassium channels, stabilizing neuronal membranes.	dizziness, somnolence, fatigue, and confusion.
10	Brivaracetam	50	Binds selectively to synaptic vesicle protein SV2A, modulating neurotransmitter release.	dizziness, headache, and fatigue.

Table 5: Third line antiepileptic drug

S.no	Drug	Dose (mg/day)	Mechanism of action	Adverse effect
01	Cannabidiol	2.5	Mechanism not fully understood; may involve modulation of cannabinoid receptors and GABAergic neurotransmission.	Somnolence, sedation, decreased appetite, diarrhea, and elevated liver enzymes.
02	Stiripentol	50	Mechanism not fully understood; may potentiate GABAergic neurotransmission.	Somnolence, decreased appetite, weight loss, and abdominal pain.
03	Fenfluramine	0.1	Mechanism not fully understood; may involve modulation of serotonin receptors and neurotransmitter release.	Decreased appetite, fatigue, somnolence, and diarrhea
04	Vigabatrin	50	Irreversibly inhibits GABA transaminase, increasing GABA levels.	Drowsiness, fatigue, dizziness, and visual field defects.
05	Clobazam	5	Enhances the effects of GABA by increasing the duration of chloride channel opening.	Drowsiness, sedation, drooling, and irritability.
06	Rufinamide	10-20	Prolongs the inactive state of voltage-gated sodium channels.	Dizziness, drowsiness,

				nausea, and vomiting.
07	Diazepam rectal gel	0.2-0.5	Enhances the effects of GABA by increasing the duration of chloride channel opening.	Drowsiness, fatigue, and respiratory depression.
08	Pidotimod	400-800	Modulates the immune system, reducing inflammation that may contribute to epilepsy.	Gastrointestinal disturbances, headache, and allergic reactions.
09	Sultiame	15-20	Mechanism not fully understood; may involve modulation of neurotransmitter release.	Dizziness, nausea, and vomiting.
10	Methsuximide	15-30	Blocks T-type calcium channels, reducing neuronal excitability.	Dizziness, headache, and gastrointestinal disturbances.

XI. CHALLENGES IN EPILEPSY MANAGEMENT

- Managing epilepsy presents several challenges to both patients and healthcare professionals. Some of the key challenges include:

Seizure Control: Achieving optimal seizure control can be challenging, as seizures can vary in frequency, severity, and type among individuals. Finding the right medication(s) and dosage(s) that effectively control seizures while minimizing side effects can require trial and error.[84]

Medication Side Effects: Many antiepileptic drugs (AEDs) can cause side effects ranging from mild to severe. Common side effects include dizziness, drowsiness, cognitive impairment, weight gain, and mood changes. Managing these side effects while maintaining seizure control is crucial.[85]

Drug Interactions: AEDs can interact with other medications, potentially affecting their efficacy or increasing the risk of side effects. Healthcare professionals must carefully consider drug interactions when prescribing AEDs, especially in patients with multiple comorbidities or taking multiple medications.[85]

Psychosocial Impact: Epilepsy can have a significant psychosocial impact on individuals, affecting their quality of life, self-esteem, relationships, education, employment, and social activities. Stigma associated with epilepsy may also lead to social isolation and discrimination.[86]

Cognitive and Behavioral Challenges: Epilepsy can affect cognitive function and behavior, particularly in individuals with poorly controlled seizures or certain types of epilepsy. Cognitive impairments, memory difficulties, attention deficits, and mood disorders are common challenges that may require additional support and intervention.[86]

Comorbidities: Epilepsy often coexists with other medical conditions such as depression, anxiety, migraine, autism spectrum disorder, intellectual disabilities, and developmental delays. Managing these comorbidities alongside epilepsy can be complex and may require a multidisciplinary approach.[84]

Treatment Adherence: Adherence to medication regimens is crucial for seizure control, but it can be challenging for some patients due to factors such as medication side effects, forgetfulness, socioeconomic barriers, and misconceptions about epilepsy and its treatment.[86]

Access to Healthcare Services: Access to specialized epilepsy care, including neurologists, epileptologists, diagnostic tests (e.g., EEG, MRI), and epilepsy surgery, may be limited in certain geographic areas or healthcare systems. This can delay diagnosis, treatment initiation, and optimization of care.[86]

Safety Concerns: Epilepsy increases the risk of accidents and injuries, especially during seizures. Ensuring safety measures at home, work, and in the community is essential to prevent accidents, falls, burns, and other injuries.[86]

Reproductive Health and Pregnancy: Managing epilepsy during pregnancy requires careful consideration of medication safety, potential teratogenic effects, seizure control, and maternal-fetal health. Women with epilepsy may require specialized care to optimize pregnancy outcomes while minimizing risks to themselves and their babies.[88]

VII. UNMET NEEDS AND FUTURE DIRECTIONS

The primary step to alleviate the impact of epilepsy is minimizing diagnostic errors, which are prevalent in both developed and developing countries. Adequate diagnostic tools and educational resources should be accessible in developing nations. Improving diagnostic accuracy for paroxysmal events through well-defined algorithms is crucial to avoid misdiagnosis.[89] Misdiagnosis not only includes missing epilepsy but also incorrectly labeling other paroxysmal events as epilepsy, leading to stigma and unnecessary drug treatment. The use of incorrect treatments contributes to the disease burden. Eliminating seizures is essential to reducing epilepsy burden, particularly in developing countries where the treatment gap is significant. Providing appropriate medication to individuals in these regions can address this unmet need effectively.[90]

Preventing symptomatic epilepsies through early identification of comorbidities is another critical intervention. Addressing comorbidities can prevent complications, improve quality of life, and reduce healthcare costs. Social deprivation is associated with epilepsy risk factors and mortality, emphasizing the importance of addressing socioeconomic disparities.[91] The increasing elderly population in developing countries necessitates early detection and prevention of epileptogenic conditions. Despite increasing awareness, epilepsy-related mortality remains underestimated and inadequately addressed. Efforts to promote education about epilepsy-related mortality, including Sudden Unexpected Death in Epilepsy (SUDEP), are essential.[92]Quantifying epilepsy-related costs and disentangling them from underlying conditions requires well-designed prospective studies. The economic burden of epilepsy, particularly out-of-pocket costs in countries with limited resources, needs further investigation. Caregiver burden is a significant concern, highlighting the need for positive family support, educational resources, support groups, and psychotherapy. Collaborative efforts are crucial to address these unmet needs and improve outcomes for individuals with epilepsy and their families.[93]

VIII. FINDINGS:EPILEPSY AND ITS MANAGEMENT:

Table 6.Findings epilepsy and its management

S.no	Name of researcher/year	Title	Findings	Ref.no
01	Edward K Avila et al/2024	Brain tumor-related epilepsy management: A Society for Neuro-oncology (SNO) consensus review on current management.	Review highlights the essential aspects of diagnosis and treatment of TRE with ASMs, surgery, chemotherapy, and radiotherapy while indicating areas of uncertainty. Future studies should consider the use of a standardized method of seizure tracking and incorporating seizure outcomes as a primary endpoint of tumor treatment trials.	94
02	Piccenna L et al/2023	Management of epilepsy in older adults: A critical review by the	Future treatment studies should use greater homogeneity in the inclusion criteria to allow for clearer findings that can be comparable with other	95

		ILAE Task Force on Epilepsy in the elderly	studies to build the existing treatment evidence base.	
03	Biondi A et al/2022	Noninvasive mobile EEG as a tool for seizure monitoring and management: A systematic review	Review supports the potential clinical value of noninvasive mobile EEG systems and their advantages in terms of time, technical support, cost, usability, and reliability when applied to seizure detection and management.	96
04	Balestrini S et al/2021	The aetiologies of epilepsy	The introduction contains information suitable for level 1 competency (entry level), whilst the subsequent sections contain information aimed at level 2 competency (proficiency level) as part of the new ILAE competency-based curriculum.	12
05	Belayneh Z et al/2020	A systematic review and meta-analysis of anti-epileptic medication non-adherence among people with epilepsy in Ethiopia	Found that there is a high burden of anti-epileptic medication non-adherence among people with epilepsy in Ethiopia. This demonstrates a need for clinicians to give more attention for the monitoring and evaluation of anti-epileptic medication adherence in the health care service.	97
06	Sharma S et al/2019.	Management of psychogenic non-epileptic seizures: a multidisciplinary approach	Safety of anti-epileptic drug therapy in adults with a focus in newly diagnosed patients. Areas covered include the most commonly experienced adverse drug effects, as well as those with the highest impacts on drug tolerability, quality of life, morbidity and mortality.	98
07	Smith G et al/2018	Measurement in pediatric epilepsy self-management: A critical review.	Results highlight several key modifiable cognitive and behavioral targets for skills development: adherence, self-efficacy for seizure management, attitudes toward epilepsy, and family variables	99
08	Thurman DJ et al/2017	The burden of premature mortality of epilepsy in high-income countries: A systematic	Epilepsy-associated premature mortality imposes a significant public health burden, and many of the	05

		review from the Mortality Task Force of the International League Against Epilepsy	specific causes of death are potentially preventable	
09	Ferlazzo E et al/2016	Challenges in the pharmacological management of epilepsy and its causes in the elderly	Evidences from double-blind and open-label studies indicate lamotrigine, levetir acetam and controlled-release carbamazepine as first line treatment in late-onset epilepsy.	87
10	Edward K et al/2015	An integrative review of the benefits of self-management interventions for adults with epilepsy	Self-management interventions were delivered in diverse formats, and the inclusion of this type of intervention should be part of the comprehensive care for people living with epilepsy.	100

IX. CONCLUSION

Epilepsy remains a significant public health concern globally, with substantial impacts on individuals, families, and healthcare systems. While advancements have been made in the diagnosis and treatment of epilepsy, numerous challenges persist, particularly in resource-limited settings. Addressing these challenges requires a multifaceted approach, including improving access to diagnostic tools and treatments, raising awareness about epilepsy, reducing socioeconomic disparities, and providing support for caregivers. Collaborative efforts among healthcare professionals, policymakers, researchers, and advocacy groups are essential to reduce the burden of epilepsy and improve outcomes for affected individuals and their families.

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