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Managing Epilepsy: A Nursing Perspective and Future Advances-An Updated Review Article

[1]-Muhamad Abdulaziz B Alsaid,[2]-Humoud, Fathiah Saud A,[3]- Areej Abdullah Alruwaili,[4]- Aisha Abdow Hadadi,[5]-Zahra Mohammed Khobrani,[6]- Sulaiman Hamad Almogbel,[7]-MOhammed Mubarak Abdullah Al-Dosari,[8]-. Tagiah Jilany Ahmed Awaji,[9]-Neamah Ali Yahia Magarashi,[10]-Afnan Naseer Knadiry,[11]-Thamer Muteb Jeery Alsabhan,[12]-Nawal Mofadi Alenezi,[13]-Budur Thamer Alruwaili,[14]-Meshal Wanees Alruwily,[15]-Nader Hammad Aldawsari

1. Ksa, Ministry of Health, Second Health Cluster
2. Ksa, Ministry of Health, King Faisal Hospital
3. Ksa, Ministry of Health, Second Health Cluster School Health
4. Ksa, Ministry of Health, Imam Abdul Rahman Al-Faisal Hospital
5. Ksa, Ministry of Health, Imam Abdulrahman Al Faisal Hospital
6. Ksa, Ministry of Health, King Salman Hospital
7. Ksa, Ministry of Health, Wadi Al-Dawasir General Hospital
8. Ksa, Ministry of Health, Sabya General Hospital Jazan Health Cluster
9. Ksa, Ministry of Health, Al Ardha Primary Health Care
10. Ksa, Ministry of Health, King Salman Bin. Abdulaziz Hospital
11. Ksa, Ministry of Health, Riyadh
12. Ksa, Ministry of Health, Hafr Al-Batin Health Cluster
13. Ksa, Ministry of Health, Primary Health Care Center In Western Tarif
14. Ksa, Ministry of Health, Dental Art Installations
15. Ksa, Ministry of Health, Wadi Al-Dwasir General Hospital

Abstract:

Background: Epilepsy is a complex neurological disorder with diverse etiologies and clinical presentations. Over the years, significant advancements have been made in epilepsy classification, genetics, and drug treatment. Epilepsy management involves addressing the varied needs of individuals, including pharmacological interventions and specialized nursing care. Despite these advancements, challenges persist in optimizing treatment, particularly in resource-limited settings.

Aim: This review aims to update current knowledge on epilepsy management, focusing on advancements in epilepsy genetics, the role of epilepsy specialist nurses (ESNs), and developments in drug treatment, with an emphasis on novel anti-seizure medications (ASMs).

Methods: A comprehensive literature review was conducted, synthesizing recent research on epilepsy genetics, clinical advancements, and the role of ESNs. The review also explored the efficacy of newer ASMs, such as cenobamate, and the global challenges in epilepsy treatment, particularly in developing countries.

Results: Genetic testing has become essential in diagnosing epilepsy, particularly in refractory cases, with numerous single-gene disorders identified. ESNs have gained prominence in both developed and developing nations, improving patient outcomes through education, care coordination, and specialized support. Advances in drug treatment, including the use of lamotrigine and levetiracetam, have improved the management of focal and generalized epilepsy. Cenobamate, a novel ASM, has shown promising results in reducing seizure frequency and improving patient outcomes.

Conclusion: The field of epilepsy management has made significant strides in recent years, particularly in genetic understanding, nursing care, and drug development. However, disparities in treatment accessibility remain, especially in low-income regions. The growing role of ESNs and the introduction of novel ASMs like

cenobamate offer new opportunities for enhancing care and improving quality of life for individuals with epilepsy. Ongoing research and policy efforts are needed to address these disparities and optimize global epilepsy care.

Keywords: Epilepsy, epilepsy genetics, anti-seizure medications, epilepsy specialist nurses, cenobamate, drug treatment, epilepsy care, nursing, seizure management.

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Introduction:

At the time of the prior review [1], the International League Against Epilepsy (ILAE) had recently introduced an updated classification system for seizures and epilepsy [2, 3]. This classification acknowledges the intricate interplay of etiological factors that contribute to the emergence of various electroclinical syndromes. This complexity is elegantly depicted using a Venn-like diagram, which provides a practical framework for syndromic diagnosis. The concept of "epileptic encephalopathy" aptly captures conditions in which epilepsy itself is linked to functional decline, as exemplified by infantile spasms. Additionally, the removal of terms such as "simple" and "complex," previously used to denote retained or lost awareness, respectively, represents a logical refinement. Nevertheless, some newly introduced terminology, such as "focal to bilateral tonic-clonic" seizures replacing "secondarily generalized seizures," may face challenges in gaining widespread acceptance due to its linguistic complexity.

Advances in Epilepsy Genetics

The number of single-gene disorders associated with epilepsy has progressively increased, establishing genetic testing as a cornerstone in the evaluation of children with refractory epilepsy or epileptic encephalopathies. This approach is also well-defined for adults presenting specific electroclinical syndromes linked to single-gene mutations, such as autosomal dominant nocturnal frontal lobe epilepsy. However, its utility in the broader adult-onset epilepsy population remains less clear. For adults with significant intellectual disabilities, genetic panel testing has demonstrated a diagnostic yield of up to 23% [4, 5], which may rise to 47% when incorporating whole-genome sequencing and chromosomal analysis [6]. These findings underscore the potential therapeutic implications, as certain genetic diagnoses, such as sodium channelopathies, may contraindicate sodium channel-blocking antiseizure medications (ASMs). However, the diagnostic yield in routine practice may differ, given the selective nature of the study populations. Most identified mutations associated with epilepsy in the absence of broader neurodevelopmental disorders impact ion channels or neurotransmitter systems. Interestingly, some ion channel mutations are also linked to structural brain anomalies, such as polymicrogyria [7], suggesting their role in brain development and raising critical questions about the downstream effects of ion-channel-targeting drugs. Mutations in the mechanistic target of rapamycin (mTOR) pathway, such as those seen in tuberous sclerosis, are regionally expressed in forms of brain mosaicism. For instance, DEP domain-containing protein 5 (DEPDC5) mutations may result in focal cortical dysplasia and epilepsy [8]. The proposed two-hit hypothesis—combining somatic mutations with germline variants—provides insights into structural brain abnormalities associated with epilepsy [9]. DEPDC5 mutations also influence hippocampal potassium channel function, which can exacerbate seizures but may be reversed by specific inhibitors [10]. Research in animal models of mTOR mosaicism suggests that seizure activity originates from non-mutated neurons influenced by increased adenosine release from mutated cells, offering novel therapeutic targets [11].

A genome-wide association study involving 15,212 patients and 29,677 controls identified 16 loci and 21 biological candidate genes associated with epilepsy, with 11 genes implicated in generalized epilepsy syndromes [12]. The study attributed 32.1% of generalized epilepsy heritability and 9.2% of focal epilepsy heritability to identified single-nucleotide polymorphisms. Among these, genes such as pyridoxine 5′-phosphate oxidase (PNPO), linked to pyridoxine metabolism, have demonstrated relevance to both single-gene epilepsies and generalized epilepsies. Emerging research highlights the role of mutations in non-coding regulatory genome regions, particularly in neurodevelopmental disorders where exon

sequencing yields negative results [13]. For example, microRNAs, whose activity is modulated by neuronal activity, may influence potassium currents and seizure frequency [14]. These regulatory mechanisms are also implicated in responses to neuronal injuries such as stroke or traumatic brain injury, presenting opportunities for targeted oligonucleotide therapies to prevent epilepsy following such insults [15]. Epigenetic mutations, such as those seen in Rett syndrome, further underscore the complexity of genetic influences on epilepsy [16].

Basic Mechanism of Epilepsy:

Traumatic brain injury (TBI) represents a prevalent etiology of acquired epilepsy, with early post-traumatic seizures recognized as a significant risk factor for the subsequent development of epilepsy. Hemorrhagic trauma to the temporal lobe has been identified as a critical predictor of epileptogenesis [17]. Advanced imaging biomarkers, including tractography, resting-state MRI, and the detection of blood products, provide promising avenues for early intervention strategies [18]. Despite considerable efforts, serum biomarkers indicative of epileptogenesis have yielded limited progress [19]. In experimental models, a distinctive EEG pattern comprising rhythmic spikes with superimposed high-frequency oscillations has shown a partial correlation with the emergence of spontaneous seizures, offering a potential diagnostic tool [20].

TBI serves as a valuable framework for investigating epileptogenesis and its prevention [21]. Mechanistic studies focus on various pathways, including tau protein, amyloid precursor protein, iron accumulation, neurotransmitter activity, and t-type calcium channels, which are modulated by ethosuximide. However, the most robust models revolve around inflammation and the mammalian target of rapamycin (mTOR) pathway. A comprehensive study involving everolimus, used for managing tuberous sclerosis-associated seizures, demonstrated sustained seizure reduction over two years, suggesting a potential anti-epileptogenic effect of targeting the mTOR pathway [22, 23]. Inflammatory processes are increasingly implicated in epilepsy. Elevated levels of various cytokines have been documented in the plasma of individuals with epilepsy, irrespective of its etiology or severity [24]. Animal studies have shown that interleukin-1 (IL-1) expression surges within 40 minutes of status epilepticus onset, and the administration of an IL-1 receptor antagonist prevents diazepam habituation. Additionally, IL-1 receptor knockout mice exhibit reduced benzodiazepine resistance compared to wild-type counterparts, implicating IL-1 in the acute stages of status epilepticus [25]. Cyclooxygenases (COX), similarly upregulated in epilepsy models, may influence blood–brain barrier alterations and sustained seizure activity. Balloon cells associated with focal cortical dysplasia type 2b show an increase in immune molecules and mTOR pathway activation, providing a therapeutic rationale for repurposing COX inhibitors to manage and prevent epilepsy [26, 27]. Moreover, several other immunomodulatory strategies are currently under exploration [28].

The role of oxidative stress in epileptogenesis is gaining recognition. Nicotinamide adenine dinucleotide phosphate (NADPH) oxidase isoform 2, prevalent in microglia, neurons, and astrocytes, has been identified as a seizure trigger. Inhibitors targeting this enzyme have demonstrated efficacy in mitigating seizures in animal models. Glutamate-induced excitotoxicity, characterized by elevated hydrogen peroxide levels, correlates with epileptic activity and is attenuated by NADPH oxidase inhibitors [29]. Chronic hippocampal glucose deprivation also induces seizures in animals, suggesting metabolic stress contributes to epilepsy [30]. Advocates for oxidative stress mechanisms propose that interictal PET scan findings of focal hypometabolism may not merely be consequences but contributors to epilepsy. They argue that ketogenic diets, by promoting ketone metabolism over glucose utilization, may offset chronic glucose deprivation effects [31]. The interplay between oxidative stress and inflammation has been elucidated, with hydrogen peroxide activating genes responsible for pro-inflammatory cytokine synthesis and upregulating receptors like Toll-like and Nod-like receptors, which are implicated in epileptogenesis. Excessive glutamate receptor activation exacerbates free radical production, diminishing GABAergic inhibitory potentials and increasing neural excitability [32]. Cannabidiol's anticonvulsant effects may partly stem from its ability to mitigate oxidative stress and free radical formation. Additionally, phenserine tartrate, initially developed for Alzheimer's disease, has shown promise in reducing hippocampal and cortical cell loss in TBI models,

potentially through modulation of inflammation and oxidative stress. This highlights its potential utility in anti-epileptogenesis interventions [33].

Epilepsy Specialist Nursing

Initially introduced in the United Kingdom, the role of epilepsy specialist nurses (ESNs) has now expanded globally. This position is highly versatile and increasingly focused on specific patient demographics, such as pediatric or adolescent populations, patients undergoing surgical interventions, and those requiring antenatal or perinatal care, or managing developmental and intellectual disabilities [34]. As of 2022, the UK's National Institute for Health and Care Excellence (NICE) has mandated that individuals with active epilepsy should have access to ESN reviews at least biannually, as well as following emergency admissions [35]. The establishment of the International League Against Epilepsy (ILAE) Nursing Section in November 2021 reflects the growing emphasis on standardizing education, enhancing nursing competencies, and fostering international collaboration to optimize nursing care for epilepsy patients [36].

Globally, disparities in epilepsy treatment are starting with the World Health Organization estimating a treatment gap of over 75% between developed and developing regions. Notably, 80% of people with epilepsy (PWE) reside in low-income countries, with 80–90% lacking access to anti-seizure medications (ASMs) in some areas [37, 38]. In 2008, a nurse training initiative was launched in Cameroon, inspired by similar projects in Kenya, Tanzania, and Malawi, to address epilepsy care in regions with a higher availability of nurses than physicians. This program significantly reduced seizure frequency, leading to wider incorporation of epilepsy education into nurse training across Africa and Central America. These efforts have yielded transformative outcomes, such as improved community awareness and policy changes, including enabling children with epilepsy to attend school [38, 39]. Such findings underline the potential of ESNs and targeted nursing education to enhance seizure management outcomes in resource-limited settings. A recent systematic review of ESNs in developed nations revealed limited evidence of changes in seizure frequency or severity but highlighted substantial qualitative improvements in care. These enhancements, such as evidence-based counseling, increased patient satisfaction, and reduced hospitalizations, may have a profound impact on quality of life, particularly for those who remain symptomatic [40-47]. ESN services are cost-effective, with innovations like telephone clinics and collaborative links with community teams ensuring accessibility, even during the COVID-19 pandemic [41, 48]. However, the heterogeneity of the epilepsy population and the absence of standardized ESN roles necessitate further research into long-term interventions and improved outcome measurement tools [34].

Drug Treatment of Common Epilepsies

The selection of first-line anti-seizure medications (ASMs) varies internationally, influenced by economic factors and local regulatory guidelines, such as those of the European Medicines Agency and the Food and Drug Administration. In the UK, the 2007 Standard and New Antiepileptic Drugs (SANAD) study demonstrated superior tolerability of lamotrigine for focal epilepsy compared to carbamazepine, leading to a shift in prescribing practices [49]. Subsequent data revealed that carbamazepine adversely affects lipid profiles and cardiovascular risk to a greater extent than oxcarbazepine or eslicarbazepine, prompting recommendations for the latter [50, 51]. For post-stroke epilepsy, lamotrigine and levetiracetam are preferred due to their lower vascular risk compared to valproate and carbamazepine [52].

Levetiracetam has emerged as a favored option in both inpatient and outpatient settings, owing to its ease of use, multiple administration routes, and favorable safety profile. However, SANAD II findings challenged its position, indicating lamotrigine's superiority in terms of adverse effects and remission timelines in focal epilepsy. While levetiracetam's adverse effects are often psychiatric, lamotrigine demonstrated better patient outcomes. Zonisamide showed lower efficacy in treatment retention and is not recommended as a first-line agent despite its licensing for monotherapy. In generalized or unclassifiable epilepsy, valproate remains more effective than levetiracetam, but its teratogenic risks necessitate cautious use [53, 54]. Cenobamate, a novel ASM approved for focal epilepsy, combines sodium channel blockade and

GABA modulation. Its extended half-life allows for once-daily dosing. Clinical trials reported seizure reduction in over half of patients and freedom from seizures in up to 21% within 12 weeks. Retention rates were high, with nearly 80% of long-term users continuing treatment beyond six years [55, 56]. Comparative analyses suggest cenobamate's efficacy surpasses other third-generation ASMs, although brivaracetam and lacosamide exhibit better tolerability profiles. Further head-to-head trials are needed for definitive conclusions [57, 58]. The most common side effects of cenobamate include somnolence, dizziness, fatigue, and headache. Given its extensive hepatic metabolism and renal excretion, clinicians must exercise caution in prescribing it to patients with renal or hepatic impairment. Additionally, its potential interactions with ASMs like clobazam, phenytoin, and phenobarbital warrant careful review. Initial trials reported rare cases of drug reaction with eosinophilia and systemic symptoms (DRESS), but these were mitigated in later studies through slower titration schedules [59].

Brivaracetam, initially licensed as adjunctive therapy for focal epilepsy in 2016, has subsequently obtained FDA approval for use as monotherapy. In the European Union, however, it remains approved solely as an adjunctive treatment, and the UK National Institute for Health and Care Excellence (NICE) designates it as a second-line adjunctive option for focal-onset seizures. Although its precise mechanism of action is not fully elucidated, brivaracetam is believed to modulate neurotransmitter release by binding to synaptic vesicle protein 2A (SV2A), akin to levetiracetam. A meta-analysis of six randomized, double-blind, placebo-controlled, multicenter trials involving 2,399 patients revealed a risk ratio for seizure freedom of 4.74 in favor of brivaracetam compared to placebo [60]. Pooled data from these studies further demonstrated its favorable long-term tolerability profile, with common adverse events including dizziness, somnolence, fatigue, and irritability. The relative risk of discontinuation due to treatment-emergent adverse events (TEAEs) was 1.58, with psychiatric side effects observed less frequently than with levetiracetam. Notably, many patients transitioning from levetiracetam to brivaracetam reported alleviation of behavioral issues [60, 61]. While the drug shows potential safety in individuals with significant psychiatric comorbidities, the lack of sensitive tools to evaluate mood in the analyzed studies warrants cautious interpretation. Interestingly, brivaracetam may benefit patients unresponsive to levetiracetam, despite similarities in their mechanisms of action [62]. Current evidence suggests limited efficacy for brivaracetam in generalized epilepsies, a domain for which it lacks regulatory approval [63]. Although promising as an alternative to levetiracetam, findings from the SANAD II study underscore the necessity of rigorous, evidence-based assessments, highlighting the need for direct comparative trials to validate these observations. Perampanel has undergone extensive evaluation in observational studies for generalized epilepsy, demonstrating efficacy against absence seizures, myoclonic seizures, and tonic–clonic seizures. Its therapeutic effects, typically achieved with doses ranging from 4–8 mg, are sustained over the long term [64, 65]. Similarly, lacosamide has exhibited enduring benefits in generalized epilepsy [66, 67]. However, consistent with other sodium channel blockers, it has been reported to exacerbate myoclonus in some cases [68]. The potential inclusion of these anti-seizure medications (ASMs) in future trials, such as SANAD III, offers an exciting prospect for further exploration of their comparative efficacy and safety profiles.

Less Common Epilepsies: Drug Treatment:

Cannabidiol (CBD), in combination with clobazam (CLB), has been approved for managing Dravet Syndrome, Lennox–Gastaut Syndrome (LGS), and, more recently, focal seizures linked to tuberous sclerosis complex (TSC), supported by randomized clinical trials [69, 70, 71]. In Dravet Syndrome, 45.4% of patients receiving adjunctive CBD achieved at least a 50% reduction in convulsive seizure frequency compared to 26.6% in the placebo group. In cases without concurrent CLB use, the response rate was 27.5%. The augmented efficacy of the CBD-CLB combination is likely due to their bidirectional pharmacokinetic interactions, where CBD increases the active metabolite of clobazam by two- to fourfold, while CLB elevates the active metabolite of CBD by 1.5-fold. For TSC-related focal seizures, CBD at 20 mg achieved a 50% reduction in seizures in 41% of patients compared to 17% in the placebo group, and CBD at 25 mg achieved this reduction in 49% versus 27% for placebo. Common adverse effects include diarrhea, fatigue, somnolence, and appetite suppression. CBD's therapeutic mechanism involves low affinity for cannabinoid receptors, with additional actions such as suppression of transient receptor potential vanilloid 1 (TRPV1)

receptors, which play roles in capsaicin-mediated pain and neuroinflammatory processes in the central nervous system. This includes reducing astrocyte migration and pro-inflammatory cytokine release (tumor necrosis factor, IL-1β, IL-6, and inducible nitric oxide synthase), potentially influencing epileptogenesis [72]. Furthermore, CBD targets T-type calcium channels and voltage-gated potassium channels, critical in seizure modulation and epileptogenesis [73, 74].

Fenfluramine, initially approved as a weight-loss medication, was withdrawn in 1997 due to associations with cardiac valvulopathy. However, it gained approval in 2020 as a second-line adjunctive treatment for Dravet Syndrome in the US and EU and in 2022 in the UK. Two industry-funded, randomized, double-blind, placebo-controlled trials demonstrated significant efficacy. In one trial, patients not receiving stiripentol experienced a 62.3% reduction in convulsive seizures with higher-dose fenfluramine, while a lower dose achieved a 32.7% reduction compared to placebo [75]. In the second trial, where patients received stiripentol, lower-dose fenfluramine reduced convulsive seizures by 54% compared to placebo [76]. Common adverse events include decreased appetite, pyrexia, fatigue, and diarrhea. In LGS, randomized trials revealed significant reductions in harmful drop seizures, prompting FDA approval and discussions regarding its use in Europe and the UK [77]. Long-term follow-up studies indicate no evidence of valvular heart disease or pulmonary artery hypertension over several months [78, 79]. No trials have explored fenfluramine's efficacy in more common epilepsies.

Carisbamate, a sodium channel-targeting alkyl-carbamate like cenobamate, has received orphan designation for LGS and infantile spasms, although trials in focal epilepsy have demonstrated only limited benefits [80, 81]. Ganaxolone, a synthetic analogue of the neurosteroid allopregnanolone, functions as a positive modulator of GABA-A receptors. In 2022, it was FDA-approved for seizures associated with cyclin-dependent kinase-like 5 (CDKL5) deficiency disorder (CDD) in individuals aged two years or older. A multinational Phase III trial demonstrated its efficacy, with a median reduction in major motor seizure frequency of 30.7% over 28 days [82]. This trial represents the first to assess the safety and efficacy of antiseizure medication in CDD-associated refractory epilepsy. However, trials in common refractory focal epilepsy have yielded modest or non-significant outcomes [83, 84]. Phase III trials are underway to assess oral ganaxolone for TSC-related epilepsy, and an intravenous formulation has shown promise for refractory status epilepticus [85].

Teratogenicity

Recent updates on teratogenic risks associated with antiseizure medications (ASMs) have been reported through epilepsy pregnancy registers and position statements, including those from the International League Against Epilepsy (ILAE) [86,87,88,89]. Evidence consolidates earlier findings, indicating that levetiracetam poses a low risk for major fetal malformations. Similarly, lamotrigine demonstrates a dose-dependent risk, with doses below 325 mg/day categorized as low risk, while oxcarbazepine likely carries a comparably low risk. In contrast, carbamazepine shows dose-dependent teratogenicity, with risks below 700 mg/day deemed lower. The Australian epilepsy register highlights a specific association of carbamazepine with urogenital malformations. Additionally, topiramate has emerged as a notable cause of fetal malformations and intrauterine growth retardation, while valproate remains a major concern due to its teratogenic potential, though its use in pregnancy is significantly declining [90]. Analysis from these registers suggests that the teratogenic risk is more significantly influenced by the specific ASM prescribed than by the number of ASMs administered. A genetic predisposition may also be contributory, as mothers with a child affected by ASM-related malformations are more likely to have subsequent children similarly impacted [87]. For most ASMs, the paucity of robust safety data necessitates their classification as potentially unsafe during pregnancy, with the possible exception of clonazepam, for which emerging evidence is relatively reassuring.

Valproate exposure in utero carries a major congenital malformation (MCM) risk of 10.9% at doses exceeding 1000 mg daily [91]. It is strongly associated with a spectrum of neurodevelopmental impairments, including significant IQ reductions and a heightened incidence of autism spectrum disorders. Meta-analyses underscore these findings, but individual risks are modulated by dose and genetic factors.

Reassuringly, carbamazepine, oxcarbazepine, lamotrigine, and levetiracetam show fewer adverse neurodevelopmental outcomes. However, the ILAE emphasizes that data remain insufficient even for these ASMs, and no reliable safety data exist for other drugs [88]. Experimental studies underscore valproate's teratogenicity, particularly through epigenetic mechanisms. Animal models have demonstrated that valproate induces histone hyperacetylation, methylation, and DNA demethylation by inhibiting histone deacetylase, leading to altered gene expression and congenital malformations. Additionally, rodent studies suggest the potential transgenerational transmission of autism spectrum disorder-related traits. While human extrapolation remains tentative, preliminary questionnaire-based studies indicate transgenerational effects in humans, with 23% of offspring experiencing malformations and 44% facing neurodevelopmental challenges. Valproate has also been implicated in male fertility issues, potentially influencing sperm quality and thereby impacting fetal development, though direct evidence is currently unavailable.

Heightened regulatory scrutiny has emerged regarding valproate use in pregnancy. Since 2018, regulations in the UK and European Union mandate pregnancy prevention programs for women prescribed valproate. In December 2022, the UK's Medicines and Healthcare products Regulatory Authority (MHRA) proposed further restrictions, recommending against valproate initiation in individuals under 55 years unless no viable alternatives exist. These recommendations extend to male patients due to potential implications for offspring, although corresponding guidelines in the EU and the US remain unchanged. Despite these concerns, valproate's therapeutic efficacy, particularly in generalized epilepsy, remains unparalleled, as reaffirmed by the SANAD II study [54]. This underscores the complexity of its clinical use. While minimizing fetal exposure is imperative, it is equally crucial to address the risk of mortality in young adults with uncontrolled epilepsy who may benefit from valproate. Responsible prescribing practices and collaborative regulatory frameworks are essential to balance these competing priorities effectively.

Conclusion:

Managing epilepsy continues to be a dynamic challenge, with progress across multiple domains—genetics, nursing, and pharmacology. The expanded understanding of the genetic underpinnings of epilepsy has opened new avenues for diagnosis and treatment. Genetic testing, once primarily focused on children with refractory epilepsy, is now increasingly applied to adults, particularly those with intellectual disabilities or specific electroclinical syndromes. While the genetic landscape of epilepsy is still being explored, it is clear that single-gene mutations affecting ion channels and neurotransmitter systems play a significant role in epilepsy pathophysiology. Furthermore, the identification of genetic mutations, such as those related to the mTOR pathway, offers novel therapeutic targets, potentially altering the way epilepsy is treated in the future. In the realm of nursing, the role of epilepsy specialist nurses (ESNs) has grown substantially. The establishment of ESNs has been pivotal in improving patient outcomes, particularly in regions with limited access to physicians. ESNs provide specialized care, education, and counseling, which have led to improvements in quality of life, even though the impact on seizure frequency remains variable. The success of nurse-led initiatives in countries like Cameroon and Kenya underscores the potential for ESNs to bridge the treatment gap in resource-poor settings, contributing to better seizure management and societal integration for individuals with epilepsy. On the pharmacological front, the development of new anti-seizure medications (ASMs) has brought more options to the table, offering better tolerability and efficacy. Drugs such as cenobamate are showing promise in treating focal epilepsy, with trials demonstrating significant seizure reduction and favorable retention rates. However, the use of such drugs must be closely monitored, especially in patients with coexisting conditions. Despite these advancements, challenges persist, especially in low-income countries, where access to medications and specialized care remains a significant issue. Continued efforts in research, education, and policy development are essential to address these gaps and ensure equitable epilepsy care worldwide.

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الملخص:

الخلفية: الصرع هو اضطراب عصبي معقد يتمتع بأسباب وعروض سريرية متنوعة. على مر السنين، تم إحراز تقدم كبير في تصنيف الصرع، وعلم الوراثة، وعلاج الأدوية. تشمل إدارة الصرع تلبية احتياجات الأفراد المختلفة، بما في ذلك التدخلات الدوائية والرعاية التمريضية المتخصصة. وعلى الرغم من هذه التقدمات، لا تزال هناك تحديات قائمة في تحسين العلاج، خاصة في المناطق ذات الموارد المحدودة.

الهدف: يهدف هذا المقال إلى تحديث المعرفة الحالية بشأن إدارة الصرع، مع التركيز على التقدمات في علم وراثة الصرع، ودور ممرضين متخصصين في الصرع(ESNs) ، والتطورات في علاج الأدوية، مع التركيز على الأدوية المضادة للنوبات الحديثة.

الطرق: تم إجراء مراجعة شاملة للأدبيات، حيث تم تجميع الأبحاث الحديثة حول علم وراثة الصرع، والتطورات السريرية، ودور ممرضي الصرع المتخصصين. كما استكشفت المراجعة فعالية الأدوية المضادة للنوبات الأحدث، مثل سينوباميت، والتحديات العالمية في علاج الصرع، خاصة في الدول النامية.

النتائج: أصبح اختبار الوراثة أمرًا أساسيًا في تشخيص الصرع، لا سيما في الحالات المقاومة للعلاج، مع تحديد العديد من الاضطرابات الجينية الفردية. لقد حصل ممرضو الصرع المتخصصون على مكانة بارزة في كل من البلدان المتقدمة والنامية، حيث حسّنوا نتائج المرضى من خلال التعليم، وتنسيق الرعاية، والدعم المتخصص. وقد حسّنت التقدمات في علاج الأدوية، بما في ذلك استخدام لاموتريجين وليفيتيراسيتام، من إدارة الصرع البؤري والمعمم. أظهر سينوباميت، وهو دواء مضاد للنوبات جديد، نتائج واعدة في تقليل تكرار النوبات وتحسين نتائج المرضى.

الاستنتاج: أحرزت إدارة الصرع تقدمًا كبيرًا في السنوات الأخيرة، خاصة في فهم الجينات، ورعاية التمريض، وتطوير الأدوية. ومع ذلك، لا تزال هناك تفاوتات في الوصول إلى العلاج، خاصة في المناطق ذات الدخل المنخفض. يقدم الدور المتزايد لممرضين الصرع المتخصصين وظهور أدوية جديدة مثل سينوباميت فرصًا جديدة لتحسين الرعاية وتعزيز جودة الحياة للأفراد المصابين بالصرع. هناك حاجة إلى أبحاث وجهود سياسة مستمرة لمعالجة هذه الفجوات وتحسين الرعاية العالمية للصرع.

الكلمات المفتاحية: الصرع، علم وراثة الصرع، الأدوية المضادة للنوبات، ممرضين متخصصين في الصرع، سينوباميت، علاج الأدوية، رعاية الصرع، التمريض، إدارة النوبات.