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EDITORIAL



Dear Colleagues,

For ten years, I have served as the Editor of Archives of Epilepsy (formerly Epilepsi). Today, I have reached the end of my term as editor.

Throughout this period, we had one single goal: to publish scientific studies in the field of epilepsy accurately and reliably, after a rigorous and impartial peer-review process. We worked together to preserve and improve the scientific quality of our journal.

In this process, I received the greatest support from my esteemed associate editors. I sincerely thank them for their dedication and meticulousness at every stage, from the evaluation of manuscripts to their preparation for publication.

I am also grateful to all our publishers who contributed to the continuation of our journal's publication life.

I would also like to thank the boards of directors and presidents of the Turkish Epilepsy Society for their support and cooperation with our journal.

I am handing over the duty to the new management with peace of mind. I believe they will carry the journal even further while preserving its scientific line. I wish the new management success.

I thank everyone who contributed during this ten year period.

With respect,
S. Naz Yeni, M.D., Prof.
Editor-in-Chief

Archives of Epilepsy

Review

From Dysbiosis to Seizure Control: The Gut-brain Axis as an Emerging Therapeutic Frontier in Epilepsy

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Abstract

Epilepsy is a chronic neurological disorder characterized by recurrent seizures resulting from abnormal and excessive neuronal activity. Although antiseizure medications (ASMs) remain the cornerstone of treatment, approximately one-third of patients develop drug-resistant epilepsy, highlighting the need for alternative therapeutic strategies. Increasing evidence suggests that gut microbiota play an important role in the pathophysiology of epilepsy through bidirectional communication along the gut-brain axis. Beyond cataloguing these associations, this review critically appraises the methodological quality and internal consistency of the existing evidence base, identifies recurring sources of contradiction across studies (sampling design, taxonomic resolution, confounding by diet, and ASM), and proposes an integrative framework in which dysbiosis is treated as a perturbation of a functionally interconnected gut-immune-neural network rather than an isolated biomarker. Experimental and clinical studies have demonstrated alterations in gut microbial composition in patients with epilepsy and in animal models, suggesting a potential disease-associated microbial signature. Moreover, interventions targeting gut microbiota, including ketogenic diet (KD), probiotics, prebiotics, antibiotics, and fecal microbiota transplantation (FMT), have shown promising anticonvulsant effects in preclinical studies, while clinical evidence remains limited and heterogeneous. Among these approaches, the KD currently has the strongest supporting evidence, whereas other microbiota-based therapies remain in the early stages of investigation. Despite substantial progress, causal relationships between alterations in the microbiota and epilepsy are not fully understood, and standardized clinical protocols are lacking. Rather than leaving this open-ended, we reach a graded conclusion: the KD and FMT are currently the only interventions supported by experiments that can distinguish causation from correlation and such evidence is confined to animal models; probiotic, prebiotic, short-chain fatty acid, and antibiotic data remain associative. Therefore, the priority for the field is not further cross-sectional taxonomic profiling-which cannot resolve causality by design-but rather longitudinal pre-diagnosis sampling, function-level multi-omics readouts, and donor- or strain-characterized interventional trials with prespecified microbiota endpoints. Until these are available, microbiota modulation should be positioned as an adjunctive, mechanistically stratified strategy under investigation for drug-resistant epilepsy, rather than as an established therapeutic option.

Keywords: Epilepsy, gut-brain axis, microbiota, microbiome, dysbiosis

INTRODUCTION

Epilepsy is a neurological disorder characterized by recurrent seizures resulting from abnormal brain activity. The International League Against Epilepsy defines an epileptic seizure as "a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain," with epilepsy diagnosed when there is an enduring predisposition to generate such seizures-established by two unprovoked seizures more than 24 hours apart, one unprovoked seizure with a high (>60%) probability of recurrence within 10 years, or an established epilepsy syndrome.[1] Epileptiform activity on electroencephalography and epileptogenic lesions on neuroimaging remain the two principal parameters for estimating recurrence risk.[2] Epilepsy is among the most common neurological disorders, with a pooled incidence of 61.4 per 100,000 person-years across international cohorts.[3] Antiseizure medications (ASMs)—a more accurate term than "antiepileptic drugs," since they suppress seizures rather than epileptogenesis itself—remain the cornerstone of treatment, but roughly one-third of patients develop drug-resistant epilepsy despite adequate ASM trials.[4] Beyond seizures themselves, drug-resistant epilepsy carries substantial burden through increased mortality risk, cognitive and psychiatric comorbidity, and reduced quality of life, which has driven sustained interest in adjunctive strategies that act through mechanisms distinct from conventional ASM pharmacology.[4] For drug-resistant patients who are not surgical candidates, the ketogenic diet (KD)—a high-fat, adequate-protein, low-carbohydrate regimen—is an established first-line therapy in glucose transporter-1 deficiency syndrome and an effective adjunct more broadly, reducing seizure frequency in both children and adults.[5-7] Its antiseizure mechanisms are incompletely understood but implicate altered energy metabolism, enhanced gamma-

aminobutyric acid (GABAergic) transmission, modulation of ion channels, and reduced oxidative stress and neuroinflammation.[8,9] Increasing attention has turned to the gut microbiota as a mediator of these effects: specific microbial taxa are required for the KD's full antiseizure efficacy in mice, acting through hippocampal GABA/glutamate balance and related metabolic pathways,[10] establishing the KD as both a treatment and a model system for studying microbiota-brain interactions.[11] A narrative review of nearly 200 studies has since consolidated this link, showing that the KD raises the *Bacteroidetes/Firmicutes* ratio and promotes short-chain fatty acid (SCFA) and neurotransmitter production, and proposing bacterial signatures as candidate biomarkers of KD responsiveness;[12] a 2026 pediatric study identified baseline microbiota signatures that distinguish KD responders from non-responders.[13]

Microorganisms—bacteria, yeasts, viruses—have long been known to coexist with the host across body sites.[14] The microbiota constitutes roughly 90% of the body's total cell population and harbors nearly 150-fold more genetic information than the human genome;[15,16] microbiome additionally encompasses collective genomes, metabolites, and environmental context.[17] Each healthy adult harbors over 1,000 gut bacterial species, dominated by *Bacteroidetes* and *Firmicutes*,[18] and unlike the largely stable host genome, this community is highly dynamic and environmentally responsive-an attractive therapeutic target.[19] Gut dysbiosis can alter intestinal permeability, metabolism, and immune signaling and has been implicated in gastrointestinal, autoimmune, and neuropsychiatric diseases,[14-20] which has motivated interest in probiotics, prebiotics, and fecal microbiota transplantation (FMT) as therapeutic strategies.

The Gut-brain Axis

The gut-brain axis is a bidirectional communication network linking the gut and its microbiota with the brain,[21] maintained not only by the vagus nerve and autonomic nervous system (ANS) but also by the enteric nervous system (ENS), immune system, and neuroendocrine signaling.[22] The ANS and ENS jointly regulate gastrointestinal motility, secretion, and local blood flow while relaying sensory information from the gut wall to the central nervous system (CNS), and the immune system contributes a further signaling layer: gut-associated lymphoid tissue continuously samples luminal microbial antigens and translates microbial composition into circulating cytokine and immune-cell signals capable of reaching the brain.[22] Enteroendocrine "neuropod" cells form synaptic connections with vagal neurons and transmit luminal signals using glutamate,[23] and gut microorganisms themselves activate vagal and enteric afferents directly.[22] Vagotomy studies underline the functional importance of this signaling: vagotomized patients show increased psychiatric morbidity,[24] and vagotomized rodents show reduced locomotor activity[25] and impaired neurogenesis,[26] consistent with the therapeutic use of vagus nerve stimulation in depression[27] and epilepsy[28]—implying that microbiota-mediated vagal modulation could affect either condition. The microbiota also produces and regulates neurotransmitters—tryptophan, serotonin, GABA, epinephrine, norepinephrine, dopamine.[19-29] The microbiome encodes glutamate decarboxylase, converting glutamate to GABA.[30] Microbially produced serotonin shapes ENS maturation.[31] SCFAs generated by microbial fermentation enter systemic circulation and influence CNS function,[32] and the microbiota is closely linked to the hypothalamic-pituitary-adrenal (HPA) axis:[22] germ-free (GF) animals show altered hormonal signaling and neurotransmission,[33,34] and GF mice show increased embryonic blood-brain barrier (BBB) permeability and reduced occludin expression that persists into adulthood unless microbiota is restored after birth.[35] More recent metabolomic work has identified altered tryptophan metabolism as a candidate mechanistic node linking gut microbial activity to seizure-related brain metabolism in pediatric epilepsy,[36] while an emerging conceptual framework proposes environmental factors such as heat stress as upstream modulators of this axis via microbial diversity and hydration-dependent seizure thresholds.[37]

These connections position the gut microbiota as a plausible contributor to the pathophysiology of neurological diseases, and correcting dysbiosis has emerged as a candidate therapeutic strategy not only in neurodevelopmental and neuropsychiatric disorders but also in Alzheimer's disease, Parkinson's disease, and epilepsy.

MAIN POINTS

- Epilepsy affects over 50 million people worldwide, and a substantial proportion of patients remain drug-resistant.
- Growing evidence indicates that the gut microbiota contributes to seizure susceptibility and to epileptogenesis via immune, metabolic, and neural pathways of the gut-brain axis.
- Beyond summarizing this literature, this review critically evaluates the internal consistency and causal strength of the evidence supporting each microbiota-targeted approach—ketogenic diets, probiotics, prebiotics, synbiotics, fecal microbiota transplantation and short-chain fatty acids—and proposes an integrative framework in which seizure susceptibility is governed by the functional output of the gut microbial community rather than any single taxon.
- This framework reconciles otherwise contradictory findings across studies and identifies the specific experimental designs needed to move the field from descriptive association toward mechanism-based, personalized microbiota therapeutics for epilepsy.

The Role of Microbiota in Epilepsy

Gastrointestinal symptoms are common in epilepsy, and epilepsy prevalence is elevated among patients with inflammatory bowel disease,[38] supporting a bidirectional gut-epilepsy relationship in which the microbiota appears central. Comparable alterations have been reported in experimental models:[39] in one rare study, dysbiosis-induced intestinal damage increased absence-seizure activity, and FMT-driven restoration of gut microbial composition partially normalized intestinal morphology and reduced seizure frequency,[40] suggesting that gut-derived inflammatory and metabolic signals may influence thalamocortical circuits in absence seizures-though mechanistic studies remain limited.

In population-based clinical studies, comparisons of epilepsy patients and healthy controls consistently report differences in gut microbiota composition,[41-44] but not the same differences: in a 14-vs.-30 comparison, controls showed higher diversity and *Bacteroidetes* dominance versus *Firmicutes* dominance in drug-resistant patients;[41] a larger study similarly found *Firmicutes* dominant only in the drug-resistant group, with seizure frequency correlating with microbial composition and *Bifidobacterium*/*Lactobacillus* lower in

more frequent seizures;[42] while a 55-vs.-46 cohort instead reported increased *Actinobacteria* and *Verrucomicrobia* and decreased *Proteobacteria*.[43] Taken together, these studies illustrate a recurring problem in the field rather than a converging signal. Smith et al.[41] and Peng et al.[42] reach opposite conclusions about which phylum dominates in drug-resistant epilepsy relative to controls, Gong et al.[43] implicates yet different phyla, and none of the three cohorts exceeds low tens to low hundreds of participants. Because ASM regimens, disease duration, dietary intake, and geographic cohorts were not consistently controlled or reported across these studies, phylum-level shifts attributed to epilepsy cannot be distinguished with confidence from shifts attributable to its treatment or from unrelated population-level variation in baseline microbiota composition. This inconsistency is a structural limitation of the descriptive literature as a whole: phylum-level taxonomy is a coarse readout that can mask convergent functional changes (e.g., reduced SCFA-producing capacity) occurring through divergent taxonomic routes, which is one reason cross-cohort taxonomic signatures have so far failed to replicate. Microbiome perturbations also track acute seizure states: patients with status epilepticus and new-onset refractory status epilepticus show markedly altered, more variable fecal microbiomes than chronic epilepsy patients, with shifts correlating with cytokine levels and evolving across the disease course.[45] Dysbiosis-driven neuroinflammation has also been implicated in comorbid depression in epilepsy via a proposed "gut-brain-emotion" network, with C-reactive protein-based subtyping suggested to individualize treatment,[46] and probiotic supplementation has been reported to improve seizure control and depressive symptoms together via shared HPA-axis and glutamatergic mechanisms.[47]

Because epilepsy pathogenesis is closely tied to neuroimmunity,[44] microbiome-associated pro-inflammatory states may plausibly contribute via the same microglia/astrocyte activation that drives epileptogenesis;[48] the gut microbiota shapes immune cell development generally—for example, specific *Clostridium* species induce colonic regulatory T-cells that constrain systemic inflammatory tone,[49] and the microbiota similarly regulates type-2 immune responses via retinoic acid-related orphan receptor γt-expressing T-cells[50]—and microglial differentiation specifically in an age- and sex-dependent manner,[51] with GF and antibiotic-treated animals showing microglial and immune deficits partially reversible by recolonization.[52] Mechanistically, barrier disruption may allow bacterial metabolites, lipopolysaccharide, and cytokines into circulation, damaging the BBB,[53] while reduced anti-inflammatory SCFA levels may compound this permeability and promote hippocampal/cortical apoptosis.[54] Directly relevant to seizures, intestinal inflammation increased convulsive activity and reduced ASM efficacy in a pentylenetetrazol (PTZ) mouse model, while butyrate reversed this via reduced inflammation and oxidative stress (Figure 1).[55]

Beyond inflammation, the microbiota directly shapes the excitation-inhibition balance disrupted in epilepsy:[56] *Lactobacillus* and *Bifidobacterium* produce GABA, *Escherichia* and *Bacillus* produce norepinephrine/dopamine,[44] and *Coprococcus*, *Ruminococcus*, and *Turicibacter* correlate with glutamate/glutamine levels;[57] FMT from KD-treated donors and KD-associated *Akkermansia*/*Parabacteroides* each raised hippocampal GABA/glutamate ratio and conferred seizure protection.[10] The microbiota also modulates hippocampal/amygdalar GABA and N-methyl-D-aspartate receptor expression[38] and brain-derived neurotrophic factor—a key regulator of excitatory-inhibitory transmission during epileptogenesis[58]—which is deficient in GF mouse hippocampus and cortex.[33] Collectively, these findings support a role for the gut-brain axis in epileptogenesis and seizure severity, though, as detailed above, this support is largely at the level of biological plausibility rather than confirmed causation in humans.

Microbiota-targeted Therapeutic Approaches for Epilepsy

Therapeutic interventions aimed at modulating the gut microbiota, correcting dysbiosis, and promoting a healthy gut-brain axis may offer hope for patients with drug-resistant epilepsy, despite current treatment options. A healthy microbiota may reduce seizure generation by restoring disrupted gut-brain axis mechanisms involved in epilepsy pathophysiology, while also potentially improving response to ASMs by supporting drug metabolism and absorption. Microbiota-targeted approaches include dietary interventions, antibiotics, probiotics, prebiotics, and FMT.

Diet

The KD is the most-studied dietary intervention in epilepsy, and evidence linking its antiseizure effects to the gut microbiota keeps accumulating: pediatric cohort studies report that KD reduces microbial diversity while shifting composition toward a *Bacteroidetes*-enriched, *Firmicutes*-depleted profile that correlates with clinical response[59,60] broadly consistent with the gnotobiotic mechanistic work described below[10]—though a study in 12 children with drug-resistant epilepsy instead found reduced beneficial bacteria and increased pathobiont *Escherichia coli* after KD.[61] This literature remains limited in scope, and the Olson et al.[10] and Lindefeldt et al.[61] findings are difficult to reconcile at face value: the former, in gnotobiotic mice, demonstrates specific microbiota-dependent seizure protection, while the latter, in children, reports an unfavorable microbial shift under the same intervention. Rather than a contradiction, this more plausibly reflects a mismatch in what is measured—a mechanistic, taxon-specific causal effect under controlled gnotobiotic conditions versus an uncontrolled clinical before-after comparison in which caloric restriction, fiber intake, and baseline microbiota are not held constant. This gap between causally validated preclinical mechanisms and associative human observations recurs throughout this literature and is, in our view, the central methodological fault line separating mechanistic plausibility from clinical proof.

SCFAs (acetate, propionate, butyrate)—fermentation products with established roles in gut-brain communication[62]—cross the BBB, modulate neuroinflammation, regulate microglial activation, and influence neurotransmitter systems.[19,63,64] Butyrate, the most studied, is both an epithelial energy source and a histone deacetylase inhibitor with anti-inflammatory, neuroprotective effects,[65] reducing seizure severity in PTZ models[66] and suppressing neuroinflammation in traumatic brain injury epileptogenesis model.[67] Prebiotic/synbiotic interventions that raise endogenous SCFA levels similarly reduce seizure susceptibility and neuroinflammation preclinically,[39,68] and propionate reduced epileptiform discharge frequency *ex vivo* in pediatric cortical tissue[69]—but randomized clinical trials of SCFA supplementation in epilepsy are still lacking. This is changing: a 2026 murine drug-resistant-epilepsy study found systemic SCFA supplementation disease-modifying—reducing

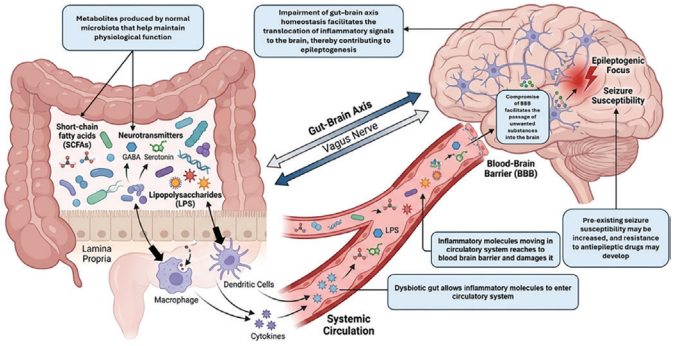

Figure 1. Proposed gut microbiota-brain interactions in epilepsy. The gut microbiota influences brain excitability through immune, metabolic, and neural pathways. Dysbiosis may promote neuroinflammation and alter neuroactive metabolite production, contributing to seizure susceptibility. Bidirectional interactions suggest that both epilepsy and its treatments can modify microbial composition. Targeting the gut microbiota represents a potential therapeutic avenue in epilepsy
GABA: Gamma-aminobutyric acid

seizure progression and clustering and improving cognition, with corroborating changes in human epilepsy brain tissue[70]—and a 2026 meta-analysis of preclinical models found SCFAs and SCFA-producing probiotics consistently reduced seizure susceptibility across chemoconvulsant and kindling paradigms.[71]

Antibiotics

Few studies address antibiotic use in epilepsy. A retrospective study of six drug-resistant patients found certain antibiotics reduced seizure frequency, implicating gut-microbiota-mediated modulation.[72] The relationship may also run in the opposite direction: some ASMs themselves possess intrinsic antimicrobial activity against common gut bacterial species *in vitro*, raising the possibility that long-term ASM use could itself reshape the gut microbiota independent of any dietary or antibiotic co-exposure, a confound rarely addressed in the clinical microbiota literature.[73] Yet penicillins, cephalosporins, and carbapenems can themselves provoke seizures, particularly with renal dysfunction:[74,75] cephalosporins via GABAergic inhibition and carbapenems via inhibitory neurotransmitter interference.[76,77] Antibiotics also disrupt both beneficial and pathogenic taxa, contributing to dysbiosis; further work is needed to determine how different agents modulate the microbiota and, in turn, seizure mechanisms. These dual effects highlight the complexity of interpreting antibiotic-related neurological outcomes. A 2025 mechanistic review proposed a unifying hypothesis: antibiotics affect seizure activity primarily through microbiota-dependent gut-brain modulation rather than direct neurotoxicity alone, with net effect depending on spectrum, dose, and which taxa are depleted or spared.[78]

Probiotics and Prebiotics

Probiotics are defined by Food and Agriculture Organization of the United Nations/World Health Organization as "live microorganisms which, when administered in adequate amounts, confer a health benefit on the host,"[79] typically *Lactobacillus* and *Bifidobacterium* formulations thought to correct dysbiosis and regulate immune/inflammatory responses via mechanisms overlapping with those described above, including GABA production, SCFA generation, and modulation of gut-associated immune signaling;[80] prebiotics are non-digestible substrates such as inulin and fructooligosaccharides that selectively promote such growth,[81] with reduced intake—linked to Western-style diets—associated with obesity, metabolic syndrome, and inflammatory disease.[19]

Preclinically, several multi-strain *Lactobacillus*/*Bifidobacterium* mixtures attenuated PTZ-kindling progression and seizure severity in Wistar rats,[82-84] and synbiotic/probiotic-prebiotic combinations reduced seizure frequency and cognitive deficits in kainic acid and PTZ models via reduced inflammation and oxidative stress and increased SCFA production.[39,85] However, a similar three-strain combination produced no independent reduction in seizure duration or severity in a different PTZ paradigm, its only measurable benefit being potentiation of co-administered diazepam.[86] In a post-traumatic epilepsy (PTE) model, a 7-strain probiotic with or without prebiotic inulin enhanced lacosamide's seizure control and improved locomotor, anxiety-like, cognitive, and pain-threshold comorbidities.[87] This preclinical heterogeneity is informative rather than incidental because strain identity, dose, duration, seizure model, and timing relative to kindling all vary simultaneously across these studies; the field cannot currently distinguish genuine strain- or model-specific biology from underpowered single-study effects—a standardization problem rather than a mechanistic one—which limits claims that probiotics constitute a uniform therapeutic class rather than a set of strain-specific interventions whose active principle remains unidentified.

Clinical data remain sparser: propionate reduced epileptiform discharge frequency *ex vivo* in pediatric temporal-lobe tissue,[88] and 28.9% of drug-resistant patients receiving a four-month probiotic mixture achieved ≥50% seizure reduction, largely sustained four months post-discontinuation.[89] This evidence has since been consolidated in a 2025 meta-analysis of six randomised controlled trials (656 patients) showing probiotics plus ASMs improved response rates and reduced seizure frequency/duration versus ASMs alone without excess adverse events-though the authors stressed persistent trial heterogeneity and the need for larger, multicenter studies;[90] a companion review similarly found convergent but insufficiently standardized signals across probiotic and KD studies,[91] and a 2025 canine drug-resistant-epilepsy pilot extended these behavioral and seizure benefits across species.[92]

Fecal Microbiota Transplantation

FMT transfers processed donor fecal material into a recipient's gut via oral capsule or endoscopy that can reduce pathogen colonization and enhance host resistance through immune regulation and restoration of colonization resistance against pathobionts,[93] with established efficacy in ulcerative colitis and *Clostridioides difficile* infection, the two indications for which controlled trial data and regulatory experience are most mature.[94,95] In GF mice, FMT from KD-fed donors or KD-associated organisms raised seizure threshold,[10] and clinically, FMT fully suppressed seizures for 20 months post-ASM discontinuation in a patient with Crohn's disease and 17 years of epilepsy[96]—though long-term safety and efficacy data remain absent. In a PTE rat model, pre-existing microbial composition and SCFA profile—rather than injury-induced dysbiosis—predicted PTE risk,[97] and in a follow-up study, FMT from PTE-prone donors conferred mild-to-moderate pro-epileptic effects on recipients, confirming that transferred microbiota actively modulates susceptibility.[98]

Read together, this literature makes a stronger causal case than the cross-sectional taxonomic studies described earlier: donor microbiota transfers either protection[10] or susceptibility,[98] the clearest evidence available that gut composition is not merely a passive marker of epilepsy. Yet the entire human clinical basis for this intervention is a single case report;[96] no controlled human FMT trial in epilepsy exists, epilepsy-specific donor-selection and safety standards are undefined, and the same paradigm that shows FMT protective with one donor shows it pro-epileptic with another—indicating that clinical translatability depends entirely on donor characteristics not yet defined. This directional dependence is arguably the strongest argument in this review that microbiota-targeted therapy cannot be a one-size-fits-all intervention and must instead be developed as a donor- or strain-characterized, mechanistically stratified approach. Consistent with this, microbiota-driven intestinal dysfunction contributes to seizure susceptibility in an absence-epilepsy model, where FMT-based restoration attenuated seizure activity,[40] and a 2026 study showing SCFA supplementation attenuates post-traumatic brain injury neurologic injury via a gut-brain-microglia axis further supports microbiota-targeted strategies in post-traumatic epileptogenesis.[99]

Toward an Integrative Framework: From Association to Mechanism

The preceding sections deliberately traced each intervention class separately; however, when taken in isolation, taxonomic association studies, dietary trials, antibiotic case series, probiotic experiments, and FMT studies each tell an incomplete story. Read as a single body of evidence, they converge on a more specific claim than "the microbiota is altered in epilepsy": they indicate that seizure susceptibility is governed by the functional output of the gut microbial community (SCFA production, GABA/glutamate precursor supply, barrier integrity, vagal and immune signaling) rather than by any single taxon or phylum ratio. This reframing has direct explanatory power for two puzzles raised above. First, it explains why taxonomic signatures fail to replicate across cohorts:[41-43] different bacterial assemblages can converge on similar functional outputs, so taxonomy is a noisy proxy for the mechanistically relevant variable. Second, it explains the directional dependence of FMT outcomes:[10-98] whether a donor community is protective or pro-epileptic should depend on its net functional output, not simply on which phyla it contains. Under this view, dysbiosis in epilepsy is best modeled as a perturbation of an interconnected gut-immune-neural regulatory network-analogous to a loss of ecological stability in a microbial community, in which the system's net functional state, not its species list, determines whether it buffers or amplifies neuronal hyperexcitability. When evaluated against this framework, the KD and FMT provide the field's only two genuinely causal demonstrations (via gnotobiotic reconstitution and donor transfer, respectively) that a defined microbial functional state is sufficient to shift the seizure threshold; probiotic, prebiotic, SCFA, and antibiotic studies remain correlational or only partially mechanistic by comparison. Table 1 summarizes this stratification.

DISCUSSION

Weighed against the causal-versus-associative distinction established above, the evidence in this field does not support treating "the gut microbiota is altered in epilepsy" as a settled premise from which therapeutic conclusions can be drawn; only the KD and FMT literatures currently supply experiments capable of distinguishing cause from correlation, and even these are causal only in animal models. When stated at the level that the evidence can bear, three claims are supportable. First, altered gut microbial composition is reproducibly detected in patients with epilepsy and in animal models, even though the specific taxa involved differ between cohorts.[42,44] Second, studies involving microbiota interventions collectively indicate that modulation of the gut microbiota can influence seizure susceptibility, epileptogenesis, and several epilepsy-associated comorbidities.[10,89,97] Third, these observations support the concept that the gut microbiota are not merely altered as a consequence of epilepsy but may actively participate in disease mechanisms through the gut-brain axis, although in humans, this remains an inference from biological plausibility rather than a demonstrated causal relationship.

Several biological pathways have been proposed to explain how gut microorganisms may affect epileptic processes. Dysbiosis may increase intestinal permeability and facilitate the systemic dissemination of bacterial metabolites, lipopolysaccharides, and pro-inflammatory cytokines, ultimately contributing to BBB dysfunction and neuroinflammation.[19] Since neuroinflammation is increasingly recognized as a critical component of epileptogenesis, microbiota-induced immune activation may represent an important mechanistic link between gut dysbiosis and seizure generation. In parallel, gut microorganisms are capable of producing or modulating neurotransmitters and neuroactive compounds, including GABA, serotonin, dopamine, norepinephrine, and glutamate-related metabolites.[19] These molecules may influence neuronal excitability and contribute to the disruption of the excitation-inhibition balance that characterizes epilepsy. Moreover, microbial metabolites such as SCFAs appear to regulate microglial activation, oxidative stress, neurotrophic signaling, and BBB integrity, further supporting the biological plausibility of microbiota-mediated effects on seizure susceptibility.[62] Among currently available microbiota-targeted interventions, the KD possesses the strongest evidence base. In contrast, evidence supporting probiotics, prebiotics, synbiotics, SCFA supplementation, and FMT remains promising but comparatively limited, with most available data originating from experimental studies and relatively few clinical investigations. Most studies evaluating these approaches have been conducted in experimental models, while human studies are characterized by small sample sizes, heterogeneous patient populations, and considerable variability in treatment protocols. Consequently, the clinical applicability of many microbiota-targeted therapies remains uncertain. A further, often-overlooked source of heterogeneity is demographic and geographic variation: gut microbiota composition differs substantially by age, sex, diet, and geographic region, independent of epilepsy status, yet few of the studies reviewed above matched cases and controls on these variables or replicated findings across cohorts from different countries. Until microbiota studies in epilepsy routinely control for this baseline population-level variability, it will remain difficult to distinguish disease-specific signals from ordinary demographic noise. Despite the rapid expansion of research in this field, several important knowledge gaps remain. First, no specific microbial signature has yet been universally identified as a reliable biomarker for epilepsy diagnosis, prognosis, or treatment response. Different studies have reported distinct alterations in microbial taxa, and reproducibility across populations remains limited. Second, the direction of causality remains unclear. While dysbiosis may contribute to epileptogenesis,

Table 1. Overview of microbiota-targeted therapeutic strategies investigated in epilepsy

Therapeutic strategy	Approximate number of published studies*	Evidence level	Overall findings	Main proposed mechanisms	Current limitations
Ketogenic diet	>20 preclinical, >15 clinical	High	Consistently reduces seizure frequency, particularly in drug-resistant epilepsy. Several studies suggest that part of its antiseizure efficacy is microbiota-dependent.	Altered gut microbial composition, increased GABA/glutamate ratio, reduced neuroinflammation, metabolic reprogramming	Mechanisms remain incompletely understood; long-term microbiota consequences remain controversial
Probiotics	~15-20 preclinical, 3-5 clinical	Moderate	Most animal studies report reduced seizure susceptibility, delayed seizure onset, and improved cognitive outcomes. Limited clinical studies suggest seizure reduction in a subset of patients.	Restoration of dysbiosis, SCFA production, immunomodulation, neurotransmitter regulation	Small sample sizes and lack of standardized probiotic formulations
Prebiotics	~10 preclinical, very few clinical	Low-moderate	Generally associated with reduced neuroinflammation and improved seizure-related outcomes in animal models.	Increased SCFA production, enhancement of beneficial bacterial populations, improved gut barrier function	Lack of epilepsy-specific clinical trials
Synbiotics (probiotic+prebiotic)	~5-10 preclinical, very limited clinical	Low-moderate	Often show greater efficacy than individual probiotic or prebiotic interventions in experimental studies.	Synergistic microbiota modulation, enhanced SCFA production, anti-inflammatory effects	Clinical evidence remains scarce
Short-chain fatty acids (SCFAs; butyrate, propionate, acetate)	~10-15 preclinical, very limited human evidence	Low-moderate	Generally demonstrate anticonvulsant, neuroprotective, and anti-inflammatory properties in experimental epilepsy models.	HDAC inhibition, blood-brain barrier stabilization, microglial regulation, oxidative stress reduction	Few direct supplementation studies and lack of controlled clinical trials
Antibiotics	<10 studies	Low	Some studies report reduced seizure frequency, whereas others highlight seizure-provoking effects of specific antibiotics.	Microbiota depletion and restructuring of gut microbial communities	Conflicting findings; potential adverse neurological effects
Fecal microbiota transplantation	~10 preclinical, 1-3 clinical reports/case studies	Low	Animal studies support a causal role of microbiota in seizure susceptibility. Human evidence remains limited but promising.	Global restoration of microbial ecosystem, immunomodulation, metabolite transfer	Lack of randomized controlled trials; donor selection and safety concerns
Postbiotics/microbial metabolites	Emerging field (~5-10 studies)	Low	Early evidence suggests beneficial effects on neuronal excitability and neuroinflammation.	Direct action of microbial metabolites on neuronal and immune pathways	Insufficient clinical evidence
Combined microbiota-targeted therapies with antiseizure drugs	Very limited (<5 studies)	Low	Preliminary studies indicate that microbiota-targeted interventions may enhance antiseizure drug efficacy and improve comorbidities.	Improved drug metabolism, anti-inflammatory effects, modulation of gut-brain signaling	Requires validation in larger experimental and clinical studies

*: Approximate numbers are based on the currently available epilepsy-microbiota literature, including experimental, translational, and clinical studies published up to 2025, HDAC: Histone deacetylase, GABA: Gamma-aminobutyric acid

seizures themselves, ASMs, dietary modifications, and lifestyle factors may also alter gut microbial composition.[73,100] Distinguishing cause from consequence, therefore, represents a major challenge, and it is largely unaddressed by the cross-sectional designs reviewed above: a single-timepoint comparison between patients and controls cannot, in principle, distinguish a microbiota that causes seizures from one that is reshaped by them or by their treatment. Resolving this would require that designs already validated in the FMT literature discussed above—longitudinal pre-diagnosis sampling in at-risk cohorts combined with GF or antibiotic-depleted rodent recolonization from human-derived material—be extended systematically across intervention classes rather than remain confined to isolated proof-of-concept studies at present. Third, most current studies rely primarily on taxonomic analyses, whereas the functional consequences of microbiome alterations may be more relevant than changes in microbial abundance alone. Future investigations integrating multi-omics (metagenomics, metatranscriptomics, metabolomics, and proteomics) may provide a more comprehensive understanding of the functional microbiome in epilepsy. Consistent with this outlook, recent literature has begun to advocate for multi-omics integration coupled with artificial intelligence-driven analytical approaches, together with clinically actionable inflammatory subtyping strategies, as tools to move the field from descriptive taxonomic associations toward individualized, mechanism-based microbiota therapeutics for epilepsy and its neuropsychiatric comorbidities.[46]

Another important limitation is the lack of standardization among microbiota-targeted interventions. Probiotic studies utilize different bacterial strains, dosages, treatment durations, and formulations, making direct comparisons difficult. Similarly, optimal donor selection criteria, transplantation protocols, and long-term safety profiles for FMT remain poorly defined. The development of standardized protocols and consensus guidelines will be essential before these approaches can be incorporated into routine clinical practice. The relationship between microbiota-targeted therapies and ASMs also warrants further investigation. Emerging evidence suggests that the gut microbiota may influence drug metabolism, bioavailability, and therapeutic responsiveness. Conversely, ASMs may themselves alter microbial composition and function. Understanding these bidirectional interactions may facilitate the development of personalized therapeutic strategies that combine conventional pharmacological treatments with microbiota-directed interventions. Such approaches may be particularly relevant for patients with drug-resistant epilepsy, for whom treatment options remain limited. Future research should prioritize large-scale, multicenter, longitudinal clinical studies to validate microbiota-related biomarkers and identify microbial signatures associated with specific epilepsy syndromes. Mechanistic studies investigating the role of microbial metabolites, immune pathways, neurotransmitter systems, and gut barrier integrity will be equally important. In addition, advances in precision medicine may ultimately enable individualized microbiota-based therapies tailored to each patient's microbial profile, disease characteristics, and treatment response. The growing interest in postbiotics, engineered probiotics, targeted microbial metabolites, and next-generation microbiome therapeutics may further expand the therapeutic landscape of epilepsy in the coming years.

To convert these gaps into a concrete agenda instead of a general call for more research, we propose five priorities, ordered by their capacity to change clinical practice rather than by feasibility:

(1) Replace cross-sectional case-control profiling with longitudinal, pre-diagnosis sampling. Cohorts at known risk of epileptogenesis—traumatic brain injury, febrile status epilepticus, autoimmune encephalitis—should be sampled serially before and after the first seizure, because only this design can establish temporal precedence between dysbiosis and epilepsy.

(2) Report function, not taxonomy, as the primary endpoint. Metagenomic, metatranscriptomic, and metabolomic measures of SCFA production, GABA/glutamate precursor supply, and barrier integrity should be prespecified as primary outcomes, with taxonomic composition reported as a secondary outcome. This follows directly from the framework proposed above and represents the most plausible route to obtaining findings that replicate across cohorts.

(3) Treat ASM, diet, age, sex, and geography as prespecified covariates rather than as post-hoc explanations. At a minimum, studies should report ASM regimen and duration, dietary intake, and recruitment region, and should match or statistically adjust for these factors; cohorts that cannot do so should be labelled hypothesis-generating.

(4) Characterize the donors and strains before scaling up interventions. Because FMT is protective or pro-epileptic depending on the donor,[10,98] epilepsy-specific donor-selection criteria based on functional microbial output—not infectious-disease safety alone—must precede any controlled human FMT trial. Probiotic trials should likewise specify strain identity, dose, and duration, rather than treating probiotics as a single therapeutic class.

(5) Design the first clinical trials to be adjunctive, stratified, and biomarker-anchored. The most realistic near-term protocol is an add-on trial for drug-resistant epilepsy, in which patients are stratified by a prespecified baseline functional microbiota profile, the intervention is a defined-strain consortium or an SCFA preparation, and both seizure outcomes and the intended microbial mechanism are measured. Such a design would test the mechanism and the therapy simultaneously and generate evidence that the current literature cannot provide.

Until at least the first three of these are routinely implemented, further descriptive microbiota surveys in epilepsy are unlikely to advance either mechanistic understanding or clinical care.

The message of this review is deliberately narrow rather than broadly optimistic. Seizure susceptibility is modulated by the functional output of the gut microbial community; this has been demonstrated causally only in animal models through KD reconstitution and donor microbiota transfer, and no microbiota-targeted intervention currently meets the evidentiary standard required for routine clinical use in epilepsy. The appropriate clinical position today is therefore adjunctive and investigational, and the appropriate research response is the prioritized agenda set out above rather than continued descriptive profiling. Future large-scale, mechanistically oriented, and longitudinal studies will be crucial for determining whether modulation of the gut microbiota can be translated into safe, effective, and personalized therapeutic strategies for patients with epilepsy. Practical translation will also require attention to issues rarely discussed in the preclinical literature reviewed above: regulatory pathways for live-biotherapeutic and FMT products differ substantially from those for small-molecule ASMs; standardized safety and donor-screening protocols specific to epilepsy do not yet exist; and demonstrating cost-effectiveness relative to established treatments will require pragmatic trials rather than by further mechanistic studies alone. Addressing these translational—not only biological—gaps will determine whether microbiota-targeted approaches reach clinical practice.

Footnotes

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Archives of Epilepsy

Original Article

Electroclinical Characteristics of Status Epilepticus and Clustered Seizures Observed During Video-EEG Monitoring

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Abstract

Objective: Video-electroencephalography (EEG) monitoring (VEM) has critical importance in epilepsy for exact diagnosis, differential diagnosis, and determination of surgical candidacy. However, reduction of antiseizure medications (ASMs) may trigger status epilepticus (SE) and clustered seizures, creating a safety risk. In this study, the frequency, electroclinical characteristics, acute treatment approaches, intubation requirements, and long-term outcomes of SE (convulsive/non-convulsive) and clustered seizures, as observed during VEM, were evaluated.

Methods: In a retrospective cohort study, 34 adult patients who developed SE or clustered seizures during VEM between 1994 and 2025 and who had complete emergency intervention records were analyzed. Demographic data, epilepsy type, provocation methods, EEG findings, seizure patterns, acute treatment strategies, and long-term management approaches were recorded.

Results: A total of 34 patients, 17 of whom were female, were included (mean age: 27.9±7.1 years). The most common type of epilepsy was temporal lobe epilepsy (n=13; 38.2%). SE or clustered seizures occurred around day 3 (range, 1-9) of hospitalization and followed an average ASM reduction of 60.0% in 70.0% of patients. The most frequent pattern was clustered seizures (41.2%), followed by postconvulsive non-convulsive SE (29.4%), isolated convulsive SE (20.6%), and isolated non-convulsive SE (8.8%). Seizure patterns showed a statistically significant difference according to electroclinical epilepsy localization; clustered seizure events were more frequently observed in extratemporal epilepsies, whereas postconvulsive non-convulsive SE was more common in temporal lobe epilepsy ($p<0.05$). SE patterns were more frequently associated with the presence of structural lesions on magnetic resonance imaging ($p<0.05$). During acute treatment, benzodiazepines and intravenous levetiracetam were sufficient for most patients; only 2 patients (5.9%) required intubation. During long-term follow-up, 52.9% of patients underwent surgery or neuromodulation.

Conclusion: SE and clustered seizures that develop early, and those that occur following marked ASM reduction, can be safely controlled with appropriate acute management. The association between seizure patterns and electroclinical localization of epilepsy indicates that data obtained during VEM play a major role in surgical and neuromodulatory decision-making and thereby improve the clinical value of VEM.

Keywords: Electroencephalography, epilepsy, status epilepticus, seizure, clustered, patient safety

INTRODUCTION

During video-electroencephalography (EEG) monitoring (VEM), controlled reduction of antiseizure medications (ASMs), hyperventilation, sleep deprivation, and photic stimulation are frequently used to capture ictal events.[1] However, these interventions may increase the risk of status epilepticus (SE) and clustered seizures, particularly in patients with drug-resistant epilepsy, leading to patient safety concerns.[2,3] SE and clustered seizures may cause morbidity and mortality due to trauma, aspiration, hypoxia, and cardiorespiratory events.[4]

The contemporary classification of SE, based on defined time thresholds (t1/t2), has standardized diagnostic and therapeutic approaches and enabled rapid intervention algorithms in clinical practice.[5] However, non-convulsive SE (NCSE), particularly following convulsive seizures, may present with minimal clinical findings and prolonged confusion, making it easily overlooked. Continuous EEG monitoring during VEM is critical for detecting NCSE and preventing permanent neurological injury.[6]

Studies conducted in international centers have demonstrated that properly structured VEM protocols, including 24-hour supervision, seizure safety precautions, early benzodiazepine administration, and stepwise intravenous treatment can maintain serious complication rates at very low levels.[7] In contrast, aggressive use of activation methods or the presence of inexperienced teams are known to increase the risk of complications. Therefore, it is important for centers to present their own safety data and update their protocols in light of scientific evidence.

The objective of this study is to retrospectively examine, among events that developed during VEM hospitalization at the study center, the frequency, electroclinical characteristics, emergency treatment approaches, intubation requirements, and long-term outcomes of surgical or neuromodulatory treatment for convulsive SE, postconvulsive NCSE, isolated NCSE, and seizure clusters.

METHODS

Study Design and Population

In this single-center retrospective observational study, patients aged 18 years and older who were monitored in the VEM unit between 1994 and 2025 and developed SE or clustered seizures during the monitoring period were included.

Clinical and EEG data were obtained from the hospital information management system, the video-EEG archive, and the patient files, and recorded in a standardized data form, excluding personal identifiers. The collected variables included age, sex, epilepsy duration and syndrome, seizure type, ASMs and reduction rates, triggering factors (sleep deprivation, hyperventilation, photic stimulation, medication reduction), interictal and ictal EEG findings, day of occurrence during hospitalization, status/seizure type, ictal semiology and EEG pattern, acute treatment approach, intubation requirement, brain magnetic resonance imaging (MRI) findings, clinical response and discharge status, long-term treatment plan (medical follow-up, epilepsy surgery and/or vagus nerve stimulation), and duration of follow-up.

ASM tapering was performed in a patient-specific manner, taking into account seizure frequency, epilepsy type, and safety considerations. Dose reductions were implemented gradually during the first days of hospitalization. GABAergic ASMs (such as benzodiazepines and barbiturates) were reduced with greater caution because of their potential to lower the seizure threshold and to cause withdrawal-related seizure exacerbation.

Epilepsy types were classified as focal (temporal and extratemporal-frontal, parietal, or occipital, if clearly localized) or generalized. In focal epilepsies, cases in which the epileptogenic zone was considered to predominantly involve the temporal lobe, with extension to adjacent regions, were categorized as temporal-plus epilepsy.

SE was evaluated according to the International League Against Epilepsy 2015 definition and time thresholds; NCSE was confirmed based on the Salzburg criteria and continuous EEG findings.[5,8]

MAIN POINTS

- Status epilepticus and clustered seizures that develop during video-electroencephalography monitoring most commonly occur around the third day of hospitalization and following reduction of antiseizure medication.
- Acute management with benzodiazepines and intravenous antiseizure medications allows successful control of most cases without the need for intubation.
- Seizure patterns vary with electroclinical localization; clustered seizures predominate in extratemporal epilepsies, whereas postconvulsive non-convulsive status epilepticus is more frequently observed in temporal lobe epilepsy.

Clustered seizures were defined as recurrent, brief seizure episodes occurring within a period of less than four hours.[6]

Approval from the Hacettepe University Health Sciences Research Ethics Committee was obtained for the study, and the research was conducted in accordance with the principles of the Declaration of Helsinki (decision no: SBA 25/972, date: 01.12.2025).

Statistical Analysis

Statistical analyses were performed using SPSS software (IBM Corp., version 25). Categorical variables were presented as numbers and percentages, and continuous variables were presented as mean ± standard deviation or median (minimum-maximum), as appropriate. The Kruskal-Wallis test was used for between-group comparisons, and exact tests were used for categorical variables. A p-value <0.05 was considered statistically significant.

RESULTS

Patient Characteristics

Among 2339 patients admitted for VEM between 1994 and 2025, a total of 34 adult patients who developed SE or clustered seizures and had complete emergency intervention records were analyzed; these included 17 females and 17 males.

The mean age of the 34 patients was 27.9±7.1 years, and the mean epilepsy duration was 18.0±9.0 years. Patients were using a median of 3 ASMs (range, 1-7) prior to admission.

In the electroclinical classification of the study population (n=34), temporal lobe epilepsy was the most frequent diagnosis (n=13; 38.2%). Extratemporal epilepsy was identified in 16 patients (47.1%), including frontal lobe epilepsy (n=4, 11.8%) and parietal lobe epilepsy (n=2, 5.9%). In addition, temporal-plus epilepsy was observed in 4 patients (11.8%), and generalized epilepsy was identified in 1 patient (2.9%).

Epileptogenic lesions on brain MRI were present in the majority of patients (76.5%; n=26), and hippocampal sclerosis and cortical dysplasia were the most common findings.

The clinical and electrophysiological characteristics of the patients are summarized in Supplementary Table 1.

Characteristics of Status Epilepticus and Seizure, Clustered Events

The distribution of seizure patterns was as follows: clustered seizures in 41.2% (n=14), postconvulsive NCSE in 29.4% (n=10), convulsive SE in 20.6% (n=7), and isolated NCSE in 8.8% (n=3) (Figure 1).

SE or clustered seizures developed at a median of 3 days (range, 1-9) during VEM hospitalization. In 74.0% of patients, ASM dose reduction had been performed at the time of event onset, and in these patients, the mean reduction rate was 60.0% (range, 16.7-87.5%).

The most frequently reduced ASMs included levetiracetam, valproate, carbamazepine, and lamotrigine. GABAergic medications (benzodiazepines and phenobarbital) were generally

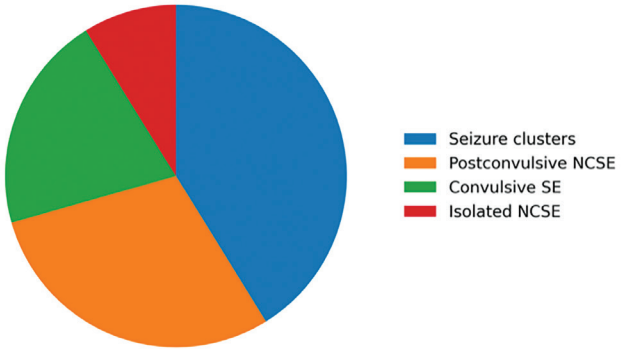

Figure 1. Distribution of seizure patterns. The figure shows the percentage distribution of seizure patterns observed during video-EEG monitoring in the study population. Seizure clusters were the most frequently observed pattern, followed by post-ictal NCSE, convulsive SE, and isolated NCSE
EEG: Electroencephalography, SE: Status epilepticus, NCSE: Non-convulsive SE

tapered more cautiously and often later during monitoring than other ASMs.

In the acute management of SE or clustered seizures, intravenous benzodiazepines (diazepam) were used as first-line treatment, followed by intravenous levetiracetam as second-line therapy in most cases. In selected patients, intravenous valproate, phenytoin, or lacosamide was administered. Only 2 patients (5.9%) required intubation and midazolam infusion due to refractory events. No severe complications were observed.

Long-term Outcomes

During long-term follow-up, 16 of the 34 patients (47.1%) were managed with medical treatment alone. Anterior temporal lobectomy ± amygdalohippocampectomy was performed in 6 patients (17.6%), and vagus nerve stimulation was implanted in 6 patients (17.6%).

Lesionectomy was performed in 3 patients (8.8%), and vagus nerve stimulation was planned for 3 patients (8.8%) who were awaiting surgery. In 1 patient (2.9%), anterior temporal lobectomy with amygdalohippocampectomy and vagus nerve stimulation were performed; in 1 patient (2.9%), hemispherotomy with amygdalohippocampectomy was performed; and in 1 patient (2.9%), anterior callosotomy was performed. In addition, 1 patient (2.9%) was presented to the surgical council for consideration of vagus nerve stimulation or lesionectomy.

Distribution of Seizure Patterns According to Electroclinical Epilepsy Groups

The distribution of seizure patterns noted during VEM across electroclinical epilepsy groups is shown in Figure 2. The distribution of seizure patterns differed significantly between epilepsy groups ($p<0.05$).

The frequency of clustered seizures was significantly higher in extratemporal epilepsies than in temporal lobe epilepsy (Fisher's exact test, $p<0.05$). In contrast, the rate of SE was significantly higher in temporal lobe epilepsy; this difference was particularly

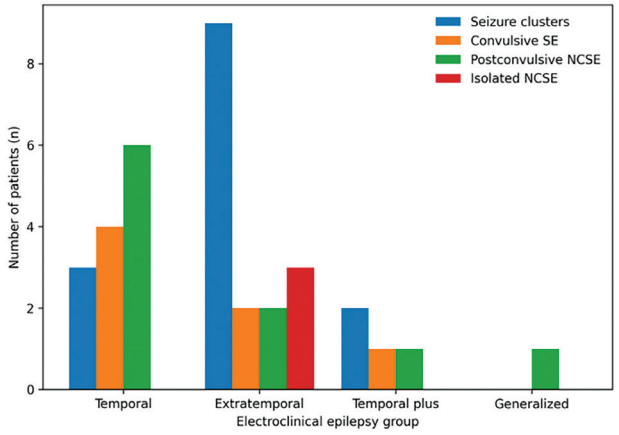

Figure 2. Distribution of seizure patterns according to electroclinical epilepsy groups. This figure shows the distribution of seizure patterns observed during video-EEG monitoring across electroclinical epilepsy groups (temporal, extratemporal, temporal plus, and generalized epilepsy). Seizure clusters were more frequent in extratemporal epilepsies, whereas post-ictal NCSE was more commonly observed in temporal lobe epilepsy
EEG: Electroencephalography, SE: Status epilepticus, NCSE: Non-convulsive SE

prominent for postconvulsive NCSE (Fisher's exact test, $p<0.05$). In the temporal-plus epilepsy group, a heterogeneous distribution was observed, with seizure, clustered, and SE patterns present.

Evaluation of MRI findings revealed a statistically significant association between seizure patterns and the presence of structural lesions ($p<0.05$). Structural lesions were more frequent in SE patterns. In contrast, no statistically significant differences were found between epilepsy groups or between seizure patterns with respect to age, epilepsy duration, day of event onset during VEM, percentage reduction in ASM, EEG propagation patterns, or long-term treatment decisions($p>0.05$).

DISCUSSION

In this study, it was demonstrated that SE or seizure clusters developed in 1.45% of patients monitored during VEM. This rate was lower than those reported in the literature.[9] The gradual tapering and withdrawal of ASMs during hospitalization may have contributed to this finding. Indeed, consistent with our results, previous studies have shown that centers employing slow and controlled ASM reduction protocols, continuous EEG monitoring, and predefined rescue treatment algorithms report lower rates of serious complications and SE during VEM.[10,11]

Seizure clusters and SE in our patients were most frequently observed on the third day of hospitalization, following a reduction in the ASM dose. Previous studies focusing on the timing of seizures and related complications during VEM have similarly reported that most events occur within the first few days of monitoring, with this period generally ranging between 1 and 4 days across centers.[11] These differences are thought to be closely related to center-specific ASM tapering protocols, the speed of medication withdrawal, and the provocation methods used during VEM.[12,13] Indeed, the literature emphasizes that ASM reduction lowers the seizure threshold of epileptic networks, facilitating seizure clustering and potentially creating a substrate for the

development of SE. In this respect, our findings are consistent with previous studies reporting seizure clustering and SE that are related to ASM tapering during VEM.[14]

In addition, seizure clusters were predominant in extratemporal epilepsies, whereas SE—particularly convulsive-onset NCSE—was more frequently observed in temporal lobe epilepsy. The literature reports that in extratemporal epilepsies, especially frontal lobe epilepsy, seizure clusters characterized by brief but frequently recurring seizures, often with nocturnal features, are prominent.[15] In contrast, seizures in temporal lobe epilepsy may have longer durations, and the tendency of limbic networks to sustain epileptic activity has been emphasized as a risk factor for the development of SE.[16,17] Taken together, these findings suggest that seizure patterns are closely associated not only with provocation strategies applied during VEM but also with the electroclinical localization of epilepsy.

In our study, seizure clusters were the most frequently observed pattern (41.2%). The literature has shown that seizure clusters may constitute a threshold for the development of SE.[18] From this perspective, early intervention in seizure clusters is of critical importance. Accordingly, all patients with seizure clusters in our cohort received oral or intravenous medication loading immediately following seizure clustering. In addition, no serious traumatic injuries or cardiac arrest occurred in any patient in the study group. The absence of progression to SE in patients with seizure clusters and the lack of traumatic complications are consistent with literature findings indicating that continuous observation and rapid intervention strategies significantly improve the safety profile during VEM.[6,18] The fact that only two patients required intubation under a benzodiazepine-based rapid intervention strategy further supports the effectiveness of structured safety protocols.

This study also demonstrated a significant association between seizure patterns and the presence of structural lesions on MRI. The higher frequency of structural lesions in SE patterns suggests that epileptic networks may become more easily sustained on a background of structural damage. This finding is consistent with previous studies reporting a higher risk of SE in structural epilepsies.[5]

An important methodological consideration in this study is the heterogeneity of ASM tapering strategies. ASM reduction was individualized for each patient to reflect real-world clinical practice in VEM units; however, this variability limits the ability to standardize the effects of ASM withdrawal on seizure occurrence and may introduce bias into the dynamics of provoked seizures. In particular, withdrawal of GABAergic agents such as benzodiazepines may significantly reduce inhibitory tone and lower the seizure threshold, thereby facilitating seizure clustering or SE. Previous studies have emphasized that both the type of ASM and the rate of withdrawal may influence seizure risk during VEM.[13,14]

Study Limitations

In addition, the retrospective design of this study entails a risk of information loss due to incomplete clinical records, and the relatively small sample size from a single center may limit the generalizability of the findings. Therefore, prospective, multicenter studies with standardized protocols and larger patient cohorts are required to confirm these results.

CONCLUSION

Due to the inherent nature of investigation and monitoring during VEM, the risk of seizure clusters or SE is not negligible. Early intervention strategies, predefined and tailored to the patient's clinical and EEG characteristics, are crucial for reducing morbidity and mortality.

Ethics

Ethics Committee Approval: Approval from the Hacettepe University Health Sciences Research Ethics Committee was obtained for the study, and the research was conducted in accordance with the principles of the Declaration of Helsinki (decision no: SBA 25/972, date: 01.12.2025).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: F.İ.T., Concept: F.İ.T., Design: F.İ.T., Data Collection or Processing: M.M.Ç., Analysis or Interpretation: M.M.Ç., F.İ.T., Literature Search: M.M.Ç., Writing: M.M.Ç., F.İ.T.

Conflict of Interest: No conflict of interest was declared by the authors.

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Supplementary Table: https://d2v96fxpocvxx.cloudfront.net/488590dc-488d-47e2-b626-b249fd4be6fc/content-images/6994d384-22db-4ba8-86c0-a395ad8bdf3c.pdf

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Archives of Epilepsy

Original Article

Examination of the Effects of Protective Factors for Psychological Resilience on the Personal Impact of Epilepsy in Patients with Epilepsy

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Abstract

Objective: This study aims to examination of the effects of protective factors for psychological resilience on the personal impact of epilepsy in patients with epilepsy.

Methods: This descriptive cross-sectional study involved 107 individuals who attended the Neurology Clinic of Adıyaman University Training and Research Hospital and met the inclusion criteria and agreed to participate. Data were collected using the Personal Identification Form, the Protective Factors for Resilience Scale (PFRS), and the Personal Impact of Epilepsy Scale (PIES).

Results: The mean PFRS score was 77.24±12.90, and the mean PIES score was 34.31±15.62. Weak to moderate negative correlations were found between PFRS total and subscale scores and PIES total and subscale scores ($p<0.05$). Regression analyses revealed that both models were statistically significant [PFRS: $F(21, 85)=3.603$, $p<0.001$, $R^2=0.340$; PIES: $F(21, 85)=3.192$, $p<0.001$, $R^2=0.303$). Brain trauma-related etiology was a significant predictor, being negatively associated with PFRS scores ($\beta=-0.215$, $p=0.018$) and positively associated with PIES scores ($\beta=0.219$, $p=0.018$).

Conclusion: The findings demonstrate an inverse relationship between protective factors for psychological resilience and the perceived impact of epilepsy. Strengthening resilience through psychosocial support, particularly involving family and social environments, may help reduce the burden of epilepsy.

Keywords: Epilepsy, psychological resilience, protective factors

INTRODUCTION

Around the world, approximately 50 million people have epilepsy, which is characterized by recurrent seizures and a chronic course. Epilepsy is a common neurological disorder worldwide. The estimated rate for active epilepsy in the general population is between 4 and 10 per 1000 people. Epilepsy affects approximately 5 million people worldwide every year. It is most common in low- and middle-income countries, where around 80% of people with the condition live.[1] People with epilepsy also have a lifestyle associated with isolation in social life, dependency in behavior, low marriage rates, reduced potential for employment, and low quality of life.[2] Studies have found that higher psychological resilience is significantly associated with a better quality of life.[3-5] Psychological resilience, which plays an essential role for patients with epilepsy, encompasses various processes that are crucial for understanding health and disease and for supporting recovery and healing. These processes encompass biological, psychological, social, and spiritual factors and mechanisms that regulate the relationship between the disease, on the one hand, and a positive, favorable, or desired outcome, on the other.[6] Resilience is the use of mental processes to protect oneself from the adverse effects of stress. While these mental processes contribute to resilience, coping strategies are realized through psychological protective factors. Protective factors are personal, familial, and social factors.[7] Individuals may encounter numerous challenging and stressful life events throughout their lives. While some individuals are negatively affected by stressful and traumatic situations, others can quickly overcome the mental state caused by such negative experiences and return to their everyday lives thanks to resilience.[8] Although previous studies have examined psychological resilience and quality of life in individuals with epilepsy,

the specific role of protective factors that underlie resilience in shaping the personal impact of epilepsy remains insufficiently explored. In particular, there is a lack of integrative studies that simultaneously evaluate resilience-related protective mechanisms and the multidimensional impact of disease.[3,4] Therefore, this study was conducted to investigate, within a comprehensive framework, how protective factors for psychological resilience relate to the perceived personal burden of epilepsy.

METHODS

Research Design

This cross-sectional descriptive study was designed to investigate the effects of protective factors for psychological resilience on the personal impact of epilepsy on patients.

Population and Sample of the Study

This study included 107 patients who attended the Epilepsy Polyclinic of Adıyaman University Training and Research Hospital between December 2024 and February 2025, met the research criteria, and volunteered to participate. Power analysis was conducted using G*Power 3.9.1. The minimum number of patients with epilepsy required to find a statistically significant medium effect size (ρ=0.37) in determining the relationship between protective factors for psychological resilience in patients with epilepsy and the epilepsy's personal impact was defined as 52 (α=0.05; 1-β=0.80).[9] A total of 107 patients who met the inclusion criteria were enrolled at the Neurology Polyclinic of Adıyaman University Training and Research Hospital between December 2024 and March 2025. Data were collected face-to-face, and the data collection sessions lasted 20-25 minutes on average.

Inclusion criteria are: (1) a diagnosis of epilepsy for at least 6 months; (2) age of at least 18 years; (3) no diagnosed psychiatric disease; (4) no mental insufficiency and no communication problems; and (5) voluntary agreement to participate in the research. Exclusion criteria are: (1) having been diagnosed with a psychiatric disorder and (2) having communication problems.

Data Collection Tools

The "Person Identification Form", "Protective Factors for Resilience Scale (PFRS)", and "Personal Impact of Epilepsy Scale (PIES)" were used to collect data.

Person Identification Form: This information form consists of 11 questions, including sociodemographic characteristics and disease-related information, and was developed based on the literature.[10,11]

MAIN POINTS

- Weak to moderate negative correlations were found between the total score and subscale mean scores of the Protective Factors for Resilience Scale and the total score and subscale mean scores of the Personal Impact of Epilepsy Scale.
- Educational level, employment status, epilepsy etiology, and the presence of comorbidities are the main factors influencing protective factors for resilience.
- Employment status and epilepsy etiology are the main factors influencing the personal impact of epilepsy.

Protective Factors for Resilience Scale (PFRS): A measurement tool designed to assess protective factors that influence psychological resilience. The scale, developed by Harms et al.,[12] consists of 15 items and three subscales: personal resources, social resources (peers), and social resources (family). The Turkish validity and reliability study was conducted by Tanko et al.[13] There are no reverse-scored items in the scale. The scale, which has a 7-point Likert structure, is scored between "strongly disagree (1)" and "strongly agree (7)", and a high score indicates a high level of resilience. High scores on the scale indicate a high level of protective factors for psychological resilience.[13] In the study by Özer et al.,[14] the Cronbach's alpha coefficient for the scale was 0.93. In this study, the Cronbach's alpha value for the scale was 0.84.

Personal Impact of Epilepsy Scale (PIES): The scale, developed by Fisher et al.,[15] was used to measure the effect of epilepsy on patients with epilepsy. Its validity and reliability in Turkish were studied by Bahcecioğlu Turan et al.[16] This scale contains 25 items and three subscales. The items on the scale are evaluated using a 5-point Likert scale ranging from 0 to 4. An increase in the score indicates a more severe negative effect of epilepsy. The subscales are: seizures (items 1-9), side effects (items 10-18), and comorbidities (items 19-25). In general, scores between 0 and 25 are considered very low, 25 and 49 are low, 50 and 69 are medium, 70 and 89 are high, and 90 and 100 are very high.[15,16] In the study of Bahcecioğlu Turan et al.[16] the Cronbach's alpha of the scale was 0.95. In this study, the Cronbach's alpha of the scale was 0.84.

Ethics

The study was conducted permission (approval no: HRÜ/24.21.52, date: 30.12.2024) from the Harran University Clinical Research Ethics Committee, and after obtaining written consent from the institution. The study was conducted in accordance with the Declaration of Helsinki, which establishes ethical standards for research involving human subjects. Before data collection began, the study was explained orally to the volunteers and written consent forms were collected.

Statistical Analysis

Analyses were performed using SPSS (version 22; IBM Corp., Armonk, NY, USA). The data were analyzed using descriptive statistics, including the mean, standard deviation, count, and percentage. The Kolmogorov-Smirnov test was used to determine whether the data conformed to a normal distribution. Since the data were not normally distributed, Spearman's correlation was used. Multiple linear regression was performed to examine whether various independent variables affected the total average scores on the PFRS and PIES. The significance level was set at $p<0.05$.

RESULTS

Of the individuals participating in the study, 58.9% were male, 64.5% were married, 53.3% were high school graduates, 45.8% were workers, 50.5% had income equal to their expenses, 49.5% had focal seizures, 58.9% had epilepsy of unknown etiology, and 72.9% had no other illnesses. The average age of the patients with epilepsy was 32.91±14.61 years, and the time from diagnosis was 5.96±5.09 years (Table 1).

Table 1. Sociodemographic and disease-related characteristics of the patients with epilepsy

Characteristics		
	n=107	%
Gender		
Female	44	41.1
Male	63	58.9
Marital status		
Single	38	35.5
Married	69	64.5
Educational level		
Primary school	18	16.8
High school	57	53.3
Associate degree	23	21.5
Bachelor's degree or higher	9	8.4
Occupation		
Civil servant	12	11.2
Worker	49	45.8
Retiree	9	8.4
Self-employed	13	12.1
Housewife	10	9.3
Unemployed	14	13.1
Economic level		
Income is less than the expenses	46	43
Income is equal to the expenses	54	50.5
Income is more than the expenses	7	6.5
Seizure types		
Focal	53	49.5
Focal onset bilateral	19	17.8
Generalized tonic-clonic seizure		
Generalized	23	21.5
Myoclonic	12	11.2
Epilepsy etiologies		
Cerebrovascular disease	13	12.1
Brain trauma	13	12.1
Brain tumor	8	7.5
Other	10	9.3
Unknown	63	58.9
Comorbidity		
Present	29	27.1
Absent	78	72.9
	X̄±SD	
Age	32.91±14.61	
Duration of the diagnosis (years)	5.96±5.09	
Total	107	100

X̄: Mean, SD: Standard deviation

The PFRS average score was 77.24±12.90. When the PFRS subscales' average scores were examined, the average scores for personal resources, social resources-peers, and social resources-family were 25.95±6.76, 25.31±5.16, and 25.97±4.73, respectively. The PIES average score was 34.31±15.62. The average scores for the PIES subscales were 12.14±6.62 for seizures, 10.49±4.92 for side effects, and 11.67±6.97 for comorbidities (Table 2).

The correlation analysis revealed a weak positive relationship between age and duration of diagnosis ($p<0.05$). A weak negative correlation was found between the duration of diagnosis and the mean total PFRS score and the PIES side effects subscale ($p<0.05$) (Table 3).

A weak negative correlation was observed between the mean score of the personal resources subscale of the PFRS and the mean scores of the comorbidities subscale and total scores of the PIES ($p<0.05$). A weak negative correlation was found between the PFRS social resources-peers subscale and the mean scores of all PIES subscales, and a moderate negative correlation was found with the total mean score of PIES ($p<0.05$). A weak negative correlation was found between the PFRS social resources-family subscale and the PIES seizures, side effects subscales, and comorbidities; a moderate negative correlation was found with the total mean score of the PIES ($p>0.05$). The PFRS total mean score was found to have a weak negative correlation with the seizures and side effects subscales of the PIES, and a moderate negative correlation with the comorbidities subscale and total mean score of the PIES ($p<0.05$) (Table 3).

To assess multicollinearity in the model, the variance inflation factor (VIF) was examined. VIF values for all independent variables are close to 1, and none exceed 10, indicating that there is no multicollinearity in the model and that the analysis results are reliable (Table 4). A multiple regression analysis estimating the relationships between gender, marital status, educational level, occupation, economic level, seizure types, epilepsy etiologies, comorbidities, and the PFRS total average score indicated that the model was statistically significant overall [$F(21, 85)=3.603$, $p<0.001$, $R^2=0.340$]. This indicates that the independent variables explained 34.0% of the variance in the PIES total average score. To assess multicollinearity in the model, the VIF was examined. VIF values for all independent variables are close to 1, and none exceed 10, indicating no multicollinearity in the model and supporting the reliability of the analysis results (Table 4).

Table 2. The PFRS, PIES, and their subscales' mean scores

Scales			
	Min	**Max**	**X̄±SD**
PFRS			
Personal resources	13	75	25.95±6.76
Social resources-peers	7	35	25.31±5.16
Social resources-family	15	35	25.97±4.73
Total mean score	47	115	77.24±12.90
PIES			
Seizures	0	29	12.14±6.62
Side effects	0	29	10.49±4.92
Comorbidities	0	23	11.67±6.97
Total mean score	0	71	34.31±15.62

X̄: Mean, SD: Standard deviation, Min: Minimum, Max: Maximum, PFRS: Protective Factors for Resilience Scale, PIES: Personal Impact of Epilepsy Scale

According to the regression analysis results, the mean PFRS scores of patients with epilepsy who had a high school education were significantly higher than those of patients with epilepsy who had a primary education (β=0.523, p<0.001). The average PFRS scores of the patients with epilepsy who were civil servants were significantly higher than those of the unemployed patients with epilepsy (β=0.311, p=0.008). Patients with epilepsy whose etiology was brain trauma had significantly lower average PFRS scores than patients with epilepsy whose etiology was unknown (β=-0.215, p=0.018). Patients with epilepsy and comorbid non-epileptic diseases had significantly lower PFRS mean scores than those without such diseases (β=-0.386, p<0.001). The remaining independent variables did not significantly affect the average scores obtained on PIES (p>0.05) (Table 4).

The selection of independent variables for the regression models was guided by both theoretical considerations and the literature, aiming to capture demographic, clinical, and psychosocial factors potentially associated with resilience and the personal impact of epilepsy. Given the multidimensional nature of these constructs, a relatively comprehensive set of variables was included to avoid omitting relevant predictors. The literature emphasizes that the psychosocial effects of epilepsy on individuals are significantly determined by individual characteristics and clinical factors related to the disease.[4,17,18] Multiple regression analysis estimating the relationships between gender, marital status, educational level, occupation, economic level, seizure types, epilepsy etiologies, comorbidities, and the PIES total average score showed that the model was statistically significant [F(21, 85)=3.192, p<0.001, R^2=0.303). This means that the independent variables explained 30.3% of the variance in the PIES total average score. To assess multicollinearity in the model, the VIF was examined. The VIF values for all independent variables are close to 1 and none exceed 10, indicating that there is no multicollinearity in the model and that the analysis results are reliable (Table 5).

According to the regression analysis results, patients with epilepsy who were employed received significantly higher average scores on the PIES than those who were unemployed (β=0.362, p=0.014). Retired patients with epilepsy had significantly higher PIES scores than unemployed patients with epilepsy (β=0.382, p<0.001). Patients with epilepsy whose etiology was brain trauma received significantly higher PIES scores than patients with epilepsy whose etiology was unknown (β=0.219, p=0.018). The remaining independent variables did not significantly affect the average scores obtained from the PIES (p>0.05) (Table 5).

DISCUSSION

In chronic diseases, resilience refers to an individual's ability to maintain adequate functioning while coping with adverse conditions and to adapt successfully by employing skillful behavioral patterns. Psychological protective factors are characteristics and variables that buffer, enhance, or modify individuals' responses to stressors.[7]

Table 3. Relationships between age, duration of the diagnosis, and scales

Variables		PFRS					
		Age	Duration of the diagnosis	Personal resources	Social resources-peers	Social resources-family	Total
Age	r	1.000	**0.377****	-0.009	-0.023	-0.111	-0.047
	p		**0.000**	0.925	0.811	0.256	0.628
Duration of the diagnosis	r	**0.377****	1.000	-0.084	-0.069	-0.038	**-0.212***
	p	**0.000**		0.389	0.483	0.697	**0.028**
PIES seizures	r	0.004	-0.169	-0.160	**-0.350****	**-0.412****	**-0.375****
	p	0.971	0.082	0.099	**0.000**	**0.000**	**0.000**
Side effects	r	-0.098	**-0.291***	-0.148	**-0.310****	**-0.489****	**-0.381****
	p	0.316	**0.002**	0.128	**0.001**	**0.000**	**0.000**
Comorbidities	r	-0.044	-0.143	**-0.204***	**-0.394****	**-0.374****	**-0.402****
	p	0.650	0.141	**0.035**	**0.000**	**0.000**	**0.000**
Total	r	-0.050	-0.182	**-0.206***	**-0.422****	**-0.496****	**-0.458****
	p	0.609	0.061	**0.033**	**0.000**	**0.000**	**0.000**

*: p<0.05, **: p<0.001, Spearman correlation analysis, PFRS: Protective Factors for Resilience Scale, PIES: Personal Impact of Epilepsy Scale

Table 4. Multiple linear regression analysis on the effects of independent variables on the PFRS

	B	Std. error	Beta	t	Sig.	95% CI	
						Lower	Upper
(Constant)	75.923	4.657		16.303	<0.001	66.664	85.183
Educational level (high school)	13.481	3.847	0.523	3.504	**<0.001**	5.832	21.129
Occupation (civil servant)	12.654	4.661	0.311	2.715	**0.008**	3.388	21.920
Epileptogenic disease (brain trauma)	-8.442	3.484	-0.215	-2.423	**0.018**	-15.370	-1.514
Comorbidity (present)	-11.171	2.705	-0.386	-4.129	**<0.001**	-16.550	-5.792

Note: The expressions in parentheses indicate the categories whose effects are compared with the reference category. CI: Confidence interval, Std: Standard, Sig.: Significance, PFRS: Protective Factors for Resilience Scale

Table 5. Multiple linear regression analysis on the effects of independent variables on the PIES

	B	Std. error	Beta	t	Sig.	95% CI	
						Lower	Upper
(Constant)	18.317	5.796		3.161	0.002	6.794	29.841
Occupation (worker)	11.298	4.522	0.362	2.498	**0.014**	2.307	20.288
Occupation (retiree)	21.397	6.482	0.382	3.301	**0.001**	8.510	34.285
Epileptogenic disease (brain trauma)	10.441	4.336	0.219	2.408	**0.018**	-2.510	14.432

Note: The expressions in parentheses indicate the categories whose effects are compared with the reference category. CI: Confidence interval, Std: Standard, Sig.: Significance, PIES: Personal Impact of Epilepsy Scale

In this study, the total average score for PFRS was 77.24±12.90. When the PFRS subscales were examined, the average scores were 25.95±6.76 for personal resources, 25.31±5.16 for social resources-peers, and 25.97±4.73 for social resources-family. Epilepsy, a chronic neurological disorder characterized by seizures, negatively affects the daily lives of individuals with the condition. Seizures, adverse effects of antiepileptic drugs, and comorbidities are among the most significant factors contributing to this impact.[4]

In the present study, the PIES total average score was 34.31±15.62, while the PIES subscale averages were 12.14±6.62 for seizures, 10.49±4.92 for side effects, and 11.67±6.97 for comorbidities. Considering the highest possible total and subscale scores, these results suggest that the protective factors for psychological resilience among patients with epilepsy were moderate, and that the personal impact of epilepsy was likewise moderate. In a study by Bahcecioglu Turan et al.,[16] similar results were reported for the comorbidities subscale, whereas lower scores were observed in other domains. In contrast, Reyes-Miranda et al.[19] reported substantially higher PIES total and subscale scores in a veteran population. These findings indicate that the level of personal impact experienced by patients with epilepsy can vary considerably depending on demographic, clinical, and contextual factors such as disease duration, treatment adherence, comorbidities, and social support. Furthermore, consistent with previous literature, stronger social and family support systems appear to play a buffering role by reducing the negative personal impact of chronic diseases like epilepsy and by enhancing psychological resilience.[20,21]

The correlation analysis conducted in this study revealed a weak positive relationship between age and duration of diagnosis. A weak negative correlation was observed between the duration of diagnosis and the total PFRS score and the side effects subscale of the PIES. Similarly, a study conducted by Reyes-Miranda et al.[19] on veterans with epilepsy reported that total and subscale scores on the PIES were significantly lower in older individuals. In the present study, a significant negative relationship was also found between the PFRS total and subscale scores and the PIES total and subscale scores. A review of the literature reveals that Dias and Cadime[22] a positive relationship between psychological resilience and supportive environmental factors. In the context of epilepsy, peer support and individual psychological characteristics are emphasized as playing an essential role in disease management and the individual's adaptation process.[23] From a neuropsychological perspective, resilience in epilepsy may involve cognitive flexibility, executive functioning, and emotional regulation processes that enable individuals to better manage uncertainty and stress.[24]

Various studies of chronic neurological diseases have shown that strong social networks and effective coping mechanisms buffer the adverse effects of these diseases. For example, Elliott et al.[20] showed that perceived social support and psychological resilience are significant predictors of quality of life in adult patients with epilepsy; low levels of social support are associated with higher seizure-related stress and lower treatment adherence. Similarly, Lin et al.[25] reported that resilience mediates the relationship between social support and psychological well-being in individuals with chronic illness. Taken together, these findings indicate that protective factors associated with psychological resilience—particularly family and peer support—play a critical role in reducing the adverse effects of epilepsy in individuals with epilepsy. Therefore, psychosocial interventions focused on strengthening coping strategies, increasing family involvement, and supporting social connectedness are thought to significantly improve the overall well-being and disease management of patients with epilepsy.

The regression analysis conducted in this study revealed that seizure characteristics were not significantly associated with psychological resilience (PFRS) or the personal impact of epilepsy (PIES). Some studies in the literature report that seizure control is associated with higher psychological resilience and that the personal impact of epilepsy is related to seizure status, seizure type, seizure frequency, and seizure history in the past year.[3,16,19,26] However, as in this study, research conducted by Tombini et al.[4] also determined that seizure-related clinical characteristics did not significantly affect resilience. This finding suggests that adaptation to epilepsy may depend not only on clinical features but also on psychosocial resources, coping strategies, and environmental support systems.

In this study, mean scores on both the PFRS and PIES were significantly affected in individuals with a history of brain injury. Patients with epilepsy and brain trauma had low mean PFRS scores and high mean PIES scores. This finding suggests that epilepsy associated with brain trauma may be linked to lower resilience and a higher personal burden of disease due to both its negative effects on neurological function and its psychosocial consequences. In line with existing literature, our findings suggest that neurological deficits following traumatic brain injury may impair cognitive flexibility, thereby simultaneously lowering resilience scores and intensifying the perceived burden of the disease.[27-29]

Comorbidities in epilepsy are essential factors that worsen both the clinical course and the psychological burden of the disease. Keezer et al.[30] reported that comorbidities accompanying epilepsy have a significant negative impact on disease burden, treatment compliance, and quality of life. In the present study, 27.1% of patients with epilepsy had at least one additional disease. This finding indicates that comorbidities associated with epilepsy

impose an additional burden on individuals and may be linked to lower levels of psychological resilience.

Study Limitations

This study is limited by its cross-sectional design, single-center setting, and, consequently, its small sample size. However, given the relatively high number of independent variables in the regression models compared with the sample size, certain methodological limitations should be acknowledged. Therefore, the findings of this study should be interpreted with caution, particularly regarding the predictive relationships identified in the regression analyses. Future studies with larger sample sizes and more parsimonious models are needed to confirm these associations. This study cannot be generalized to all patients with epilepsy because of its small sample size and the heterogeneity of psychological resilience factors. Despite these limitations, it can serve as a valuable resource for future studies. Future research may benefit from employing longitudinal designs and model validation techniques to better assess the stability and generalizability of these findings.

CONCLUSION

This study revealed that protective factors for psychological resilience in patients with epilepsy were moderate, and the disease's impact on these patients was low. In this study, the mean scores of PFRS and its subscales were negatively correlated with those of PIES and its subscales. This result shows that the protective factors of psychological resilience are significantly related to individuals' subjective experience of the burden of epilepsy. Strengthening resilience through psychosocial support, particularly involving family and social environments, may help reduce the burden of epilepsy. In addition, it was observed that patients with epilepsy due to brain trauma have lower psychological resilience and greater personal impact of epilepsy compared to patients with epilepsy of unknown etiology. Based on this result, it has been clearly shown that patients whose epilepsy has a traumatic etiology constitute the most vulnerable group with respect to psychological resilience and that this group requires specialized psychosocial support programs.

Ethics

Ethics Committee Approval: The study was conducted permission (approval no: HRÜ/24.21.52, date: 30.12.2024) from the Harran University Clinical Research Ethics Committee, and after obtaining written consent from the institution.

Informed Consent: Before data collection began, the study was explained orally to the volunteers and written consent forms were collected.

Footnotes

Authorship Contributions

Concept: R.G., M.K., Design: R.G., M.K., İ.Ç., T.G.D., Data Collection or Processing: R.G., M.K., İ.Ç., Analysis or Interpretation: R.G., İ.Ç., Literature Search: R.G., M.K., T.G.D., Writing: R.G., M.K., İ.Ç., T.G.D.

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Determining the Level of Knowledge and Awareness of the Term Sudden Unexpected Death in Epilepsy Among Healthcare Professionals

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Abstract

Objective: Sudden and unexpected death is a serious condition seen in patients with epilepsy. This study aimed to determine the level of awareness and knowledge of the term "sudden unexpected death in epilepsy (SUDEP)" among healthcare professionals.

Methods: The study was conducted with 376 healthcare professionals working in a university hospital in Türkiye between May-July 2025. Data were collected through face-to-face interviews using a data collection form that the researchers developed based on a literature review.

Results: The majority of participants were physicians and nurses. The majority of healthcare professionals provided care/treatment support to epilepsy patients. Only 12.2% of participants were aware of the term SUDEP, while 8.5% stated that it meant "SUDEP patients." Participants indicated that they heard this term most often at a symposium or conference and during their undergraduate or postgraduate training. A statistically significant difference was found between the preference for providing information to patients about SUDEP and the average age and occupational groups of individuals ($p<0.001$; $p=0.031$).

Conclusion: Although healthcare professionals have a high rate of providing care/treatment to epilepsy patients, their awareness and understanding of SUDEP are quite low. It is recommended that the curriculum be revised in undergraduate and postgraduate training to increase awareness and understanding of this term among healthcare professionals, that the literature be enriched with case studies, and that sessions or topics on epilepsy and its complications be included in congresses, symposia, and conferences.

Keywords: Epilepsy, sudden unexpected death in epilepsy, awareness, nursing

INTRODUCTION

Patients with epilepsy are considered a population at high risk for premature mortality. Deaths associated with epilepsy may arise from a wide range of causes, both related and unrelated to epilepsy. The literature identifies seizure-related injuries, motor vehicle or bicycle accidents, adverse effects of antiepileptic drugs, aspiration pneumonia, drowning, and status epilepticus among the causes of epilepsy-related mortality.[1] Sudden unexpected death in epilepsy (SUDEP) is a significant cause of mortality among patients with the disorder. SUDEP is defined as a sudden, unexpected death in individuals with epilepsy that is not attributable to trauma, drowning, status epilepticus, or other known causes, but is often associated with evidence of a recent seizure.[2,3] Death may occur suddenly and unexpectedly, either witnessed or unwitnessed. The absence of anatomical or toxicological findings that could explain the cause of death on postmortem examination is also a key feature. Reports indicate that, in a large proportion of cases, patients are found prone in bed and deaths are most often unwitnessed. The prone position at death is considered indicative of a recent seizure followed by the development of respiratory failure.[4] The incidence of SUDEP has been reported as 0.81% in the general population and 1.16% among individuals diagnosed with epilepsy, ranking as the second leading cause of epilepsy-related mortality after stroke.[5,6] In Denmark, the incidence of SUDEP in patients aged 18-49 is 1.21 per 1000 people/year.[7]

Several studies have demonstrated that the level of awareness of SUDEP, which represents a critical risk factor for patients with epilepsy and their families, is lower than expected among healthcare professionals.[8-10] However, healthcare professionals' knowledge of the significance of SUDEP, its risk factors, and preventive strategies is essential for providing effective counseling to patients and their families and enabling the modification and management of potentially preventable risks.[11] The most important components of SUDEP counseling include an adequate level of knowledge, effective communication skills, and a multidisciplinary approach. Comprehensive epilepsy care should be delivered through collaboration among neurologists, epilepsy nurses, primary healthcare providers, and other relevant healthcare professionals.[12] From a broader perspective, professional expertise and nursing care play a central role in reducing the incidence of SUDEP and in ensuring safer living conditions for patients. A review of the literature indicates that most studies have focused on evaluating awareness of SUDEP among patients with epilepsy and their relatives,[13-16] whereas studies examining the awareness of healthcare professionals regarding this issue are limited.[8-10] In this context, this study was designed to assess knowledge and awareness of SUDEP among healthcare professionals.

METHODS

Study Desing

This study was conducted using a cross-sectional, descriptive design. Data were collected through face-to-face interviews with 376 healthcare professionals working at a university hospital in Türkiye between May and July 2025. Based on a literature review, the researchers developed a data collection form consisting of 13 questions (1, 4, 6, 9, 10-13). Inclusion criteria were: being 18 years of age or older and working as a physician, nurse, paramedic, etc., at the hospital where the study was planned. Exclusion criteria were having serious visual, reading, or writing problems, or refusing to consent to participate in the study. The first section covered sociodemographic characteristics and the second section addressed SUDEP-related information. On average, it took 10 minutes to complete the questionnaire. The sample size was calculated using G*Power version 3.1 software, based on a 5% margin of error (95% confidence level), 95% statistical power, and a standardized effect size of 0.33. The power analysis indicated that a minimum of 120 participants was required. To further increase the study's statistical power, 376 healthcare professionals were included.

Statistical Analysis

The collected data were analysed using SPSS version 20. The Kolmogorov-Smirnov test was used to assess the distribution of continuous variables. Descriptive statistics were presented as the mean and standard deviation. The Mann-Whitney U test and chi-squared test were used to compare groups. A p-value of less than 0.05 was considered statistically significant.

Approval was obtained from the Balıkesir University Health Sciences Non-Interventional Research Ethics Committee (approval no: 2025/204, date: 16.05.2025), and institutional permission was obtained from Balıkesir University Health Practice and Research Hospital. Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki.

MAIN POINTS

- Patients with epilepsy are at risk of sudden unexpected death in (SUDEP).
- Healthcare professionals need information on SUDEP in epilepsy.
- There is no consensus on how to educate patients about SUDEP.
- There is a need for guidelines regarding patient education on SUDEP.

RESULTS

The mean age of the participants was 38.21±12.6 years, and 52.9% were male. The majority of participants were physicians and nurses. Most participants were working in intensive care units (39.4%), followed by internal medicine departments (19.7%) and emergency departments (11.7%). The mean duration of professional experience was 8.77±8.5 years (Table 1).

When asked whether they had ever provided treatment or care for patients with epilepsy, 65.7% of participants reported that they had. Only 12.2% of participants stated that they were familiar with the term SUDEP, and 8.5% correctly defined it as "SUDEP patients." Participants reported that they most commonly encountered the term SUDEP at a symposium or conference (6.4%) and during undergraduate or postgraduate education (2.9%). When asked about their knowledge of SUDEP risk factors, 91.2% of participants reported no knowledge, whereas 5.8% identified nocturnal and frequent seizures as risk factors.

Regarding providing information about SUDEP to patients with epilepsy, 78.4% of participants stated that patients should be informed. The main reasons cited were that providing information would increase patients' or caregivers' attentiveness to the disease (43.6%), and that receiving such information is a legal right of patients (21.0%) (Table 2).

When examining the association between participants' sociodemographic characteristics (age, sex, professional group, and years of professional experience) and their awareness of SUDEP, no statistically significant differences were observed with respect to having known of the term SUDEP. However, a statistically significant difference in the preference for providing SUDEP-related information was found according to mean age ($p<0.001$). In addition, a significant association was observed between professional group and the preference for providing information about SUDEP ($\chi^2=8.905$; $p=0.031$) (Table 3).

A statistically significant difference was found in healthcare professionals' attitudes toward providing information about SUDEP. Further analysis showed that physicians' attitudes towards providing information (41.2%) were significantly higher than those of midwives (10.5%) ($p<0.05$). Upon examination (adjusted residual 2.6), physicians' tendency to provide information was above the expected value, whereas midwives' opposition to providing information (34%) was higher than the overall average (21.8%), showing a trend contrasting with that of physicians. Nurses and paramedics occupied an intermediate position between the two extreme attitudes (Table 3).

Table 1. Sociodemographic characteristics of participants and their opinions regarding SUDEP

		Mean ± SD	min-max
Age (years)		38.21±12.6	22-65
Years of professional experience (years)		8.77±8.5	1-44
		n	%
Sex	Female	177	47.1
	Male	199	52.9
Health professional	Physician	142	37.8
	Nurse	112	29.8
	EMT/paramedic	75	19.9
	Midwife	47	12.5
Deparment	Intensive care units (neonatal, CVS, anesthesia, coronary)	148	39.4
	Internal medicine units (cardiology, pulmonology, PMR, neurology, infectious diseases)	101	26.9
	Emergency department	74	19.7
	Surgical units (ENT, urology, orthopedics, operating room, obstetrics, gynecology, plastic surgery)	44	11.7
	Other (medical biochemistry, radiology, pediatrics)	9	2.4
Providing care/treatment to a patient diagnosed with epilepsy	Yes	247	65.7
	No	129	34.3
Awareness of the term “SUDEP”	Yes	46	12.2
	No	330	87.8
Source of information about SUDEP	I have no information	337	89.6
	Symposium/conference	24	6.4
	Undergraduate/specialization training	11	2.9
	Academic article	2	0.5
	Social media	1	0.3
	Patient/treatment experience	1	0.3
What does the term “SUDEP” mean to you?	I have no information.	338	89.9
	Sudden unexpected death in epilepsy patients.	32	8.5
	Recurrent seizures.	5	1.3
	Defecation/mixing during seizures.	1	0.3
What do you think are the risk factors for “SUDEP”?	I don’t know	343	91.2
	History of nocturnal seizures	11	2.9
	Frequent seizures (more than 13 seizures per year)	11	2.9
	Medication/treatment non-compliance	7	1.9
	Other*	4	1.2
Total		376	100

*Use of more than three antiepileptic drugs, male gender, seizure onset age between 0-15 years, and epilepsy diagnosis more than 15 years ago. CVS: Cardiovascular surgery, PMR Physical medicine and rehabilitation, ENT: Ear nose throat, EMT: Emergency medical technician, SUDEP: Sudden unexpected death in epilepsy, min: Minimum, max: Maximum, SD: Standard deviation

Table 2. Participants' opinions on providing information about SUDEP

Question	Response	n	%
Should patients with epilepsy be informed about SUDEP? (n=376)	Yes, information should be provided.	294	78.4
	No, information should not be provided.	82	21.6
Epilepsy patients should be informed about SUDEP because; …… (n=299)	Patients/caregivers would be more attentive to treatment and care.	164	43.6
	It is the patient's legal right to be informed about the consequences of the disease.	79	21.0
	Patients/caregivers should be informed about risks at the time of diagnosis.	56	14.9
Epilepsy patients should not be informed about SUDEP because; ... (n=78)	It can cause unnecessary fear/anxiety in the patient.	35	44.9
	It can increase the frequency of seizures in the patient.	21	26.9
	It can cause non-compliance with medication and treatment in the patient/relative.	18	23.1
	It can prevent the patient from coping with their illness.	4	5.1

SUDEP: Sudden unexpected death in epilepsy

Table 3. Association between participants' demographic and professional characteristics and SUDEP awareness and preference for informing patients (n=376)

Variable	Heard of SUDEP (mean ± SD)	Not heard of SUDEP (mean ± SD)	p-value	Provide information (mean ± SD)	Do not provide information (mean ± SD)	p-value
Age (year)*	39.57±12.32	38.02±12.7	0.38	36.86±12.10	43.06±13.53	<0.001
Professional experience (years)*	9.53±8	8.66±8.5	0.34	8.04±7.64	11.40±10.75	0.084
Sex**						
Female	22 (12.4)	155 (87.6)	0.96	136 (46.3)	41 (50.0)	0.548
Male	25 (12.6)	174 (87.4)		158 (53.7)	41 (50.0)	
Health professional**						
Nurse	20 (17.9)	92 (82.1)	0.14	86 (29.3)	26 (31.7)	0.031
Physician	16 (11.3)	126 (88.7)		121 (41.2)[a]	21 (25.6)	
EMT/paramedic	5 (6.7)	70 (93.3)		56 (19.0)	19 (23.2)	
Midwife	6 (12.8)	41 (87.2)		31 (10.5)[b]	16 (19.5)	

Note: Values in the same column with different superscripts ([a], [b]) are significantly different according to Bonferroni-corrected column proportion comparisons (p<0.05). *Mann-Whitney U test, **Chi-square test, EMT: Emergency medical technician, SUDEP: Sudden unexpected death in epilepsy, SD: Standard deviation

DISCUSSION

This study found that, although most healthcare professionals care for patients with epilepsy, their awareness of and understanding of SUDEP is limited. Only a small proportion of participants were familiar with the term and could correctly identify its risk factors. These results are consistent with those of previous studies which have reported generally low levels of SUDEP awareness among healthcare professionals.[8-10,17]

In the present study, 78.4% of healthcare professionals stated that patients with epilepsy should be informed about SUDEP. International guidelines similarly recommend that SUDEP counseling should be provided at the time of diagnosis or during a consultation when the patient is deemed ready to receive such information.[18-21] In this study, physicians were most likely to provide information about SUDEP. In the United Kingdom, nurses are reported to be the professional group most frequently discussing SUDEP with patients. This is because the United Kingdom has teams of nurses who specialise in epilepsy, and they play an important role in SUDEP counselling. In Norway and Türkiye, however, SUDEP counselling is considered the responsibility of physicians. It is thought that the high tendency of physicians to provide information on SUDEP in this study reflects this result.[19]

A study conducted in Germany revealed that 27% of patients had never heard of SUDEP; and one-third stated that they had not been informed by their physicians about any risk factors, morbidity, or mortality causes.[16] In one study involving 1123 neurologists from 27 countries, 41.5% were more hesitant to discuss SUDEP (surgical and clinical epilepsy-related education). Experts specializing in epilepsy, however, showed a higher level of awareness regarding the provision of information. This research shows that epilepsy is a specialized field requiring advanced knowledge and expertise. Providing care to epilepsy patients by specialists working in this field will closely affect both the disease and patient compliance.[22]

Whether or not to disclose information about SUDEP to patients remains a topic of ongoing debate.[11] In the present study, 21.6% of healthcare professionals opted not to provide information about SUDEP. Previous studies have also reported that healthcare professionals may be reluctant to discuss SUDEP due to concerns that such discussions could cause patient distress, anxiety, anger, fear or a decline in quality of life.[23-25] However, studies conducted with patients have shown that, while receiving information about SUDEP may initially cause discomfort, patients generally want to be informed about this risk.[24,26] Contrary to these views, it has been reported that early information does not increase depressive symptoms or anxiety. Furthermore, it has been added that epilepsy patients who are satisfied with receiving information do not experience negative effects on their overall health, quality of life, stigma, or seizure anxiety.[27]

This study found that healthcare professionals who preferred not to inform patients about SUDEP were older on average. Consistent with this finding, other studies have demonstrated that older physicians tend to avoid discussing SUDEP. Multivariate analyses have indicated that factors such as educational level, clinical experience and involvement in the treatment of patients with epilepsy also influence attitudes towards SUDEP counselling, in addition to age.[23] The literature suggests that reluctance to engage in SUDEP counselling is associated with clinical seniority and concerns about inducing anxiety in patients.[11,23] It has been suggested that insufficient experience and educational needs may contribute to hesitation in discussing epilepsy-related risks.[12] Conversely, younger healthcare professionals may be more inclined to provide information about SUDEP, as they are more likely to have received training through updated curricula and programmes, and to be familiar with recent guidelines and research findings.

It is thought that midwives' tendency to care for more female patients and to see fewer patients with epilepsy may have influenced their information-giving tendencies. Physicians, on the other hand, may be more willing to provide information regarding medical diagnoses as part of their professional duties. The negative tendency of midwives not to provide information and the positive tendency of physicians to provide information may both stem from this.

Study Limitations

The research was conducted at a single center and within a limited time frame.

CONCLUSION

The findings of this study demonstrate that although healthcare professionals are highly involved in the care and treatment of patients with epilepsy, their awareness and understanding of SUDEP remain limited. This gap indicates a critical unmet need for knowledge that may directly affect patient safety. To address this deficiency, it is recommended that SUDEP-related content be incorporated into undergraduate and postgraduate curricula, that interactive and problem-based learning approaches be implemented, and that continuing professional development programs supported by seminars and online training be expanded. In addition, increasing the number of SUDEP-focused awareness sessions at scientific meetings and establishing standardized national guidelines for clinical practice are strongly recommended. Collectively, these strategies can enhance healthcare professionals' knowledge and awareness, thereby reducing preventable risks for patients with epilepsy and significantly improving the quality and safety of care.

Ethics

Ethics Committee Approval: Approval was obtained from the Balıkesir University Health Sciences Non-Interventional Research Ethics Committee (approval no: 2025/204, date: 16.05.2025), and institutional permission was obtained from Balıkesir University Health Practice and Research Hospital.

Informed Consent: Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Footnotes

Authorship Contributions

Surgical and Medical Practices: B.C.K., T.Ç., B.F., Concept: B.C.K., T.Ç., B.F., Design: B.C.K., T.Ç., B.F., Data Collection or Processing: B.F., Analysis or Interpretation: B.C.K., T.Ç., Literature Search: B.C.K., T.Ç., Writing: B.C.K., T.Ç., B.F.

Conflict of Interest: No conflict of interest was declared by the authors.

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Archives of Epilepsy

Original Article

Knowledge, Attitudes, and Referral Practices Regarding Epilepsy Surgery Among Neurologists and Neurosurgeons in Türkiye: A Cross-sectional Survey

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Abstract

Objective: For patients with drug-resistant epilepsy, surgery can be life-changing. Yet referrals often come too late. This study aimed to assess the knowledge, attitudes, and referral practices of neurologists and neurosurgeons in Türkiye regarding epilepsy surgery in routine clinical care.

Methods: We ran a cross-sectional survey from June 1 to August 1, 2022. The questionnaire included a nine-item factual test drawn from publicly available board exam sources and six Likert-scale questions capturing attitudes. We also asked about referral volume, institutional setting, training background, and any previous collaboration with dedicated epilepsy centers. A total of 154 clinicians volunteered and were sent the survey link, of whom 116 responded. After discarding eight incomplete forms, we analyzed data from 108 specialists-72 neurologists and 36 neurosurgeons.

Results: The mean knowledge score was 6.31±1.34 out of 9. Neurologists came out ahead of neurosurgeons (6.49±1.35 vs. 5.94±1.26; p=0.044). Scores varied significantly by type of institution, with academic centers and training and research hospitals scoring the highest (p=0.003). We observed the largest knowledge gaps regarding the risk of serious complications (35.2% answered correctly), surgical candidacy when an magnetic resonance imaging is negative (37.0% answered correctly), and age-related eligibility rules (47.2% answered correctly). Having worked with an epilepsy center before was linked to a meaningfully better score (6.78±1.24 vs. 6.07±1.34; p=0.008). Of concern, about one in three respondents had not referred a single patient for surgery in the previous year, and only 12.0% had referred anyone within one year of the appearance of the indication. Knowledge score was not significantly associated with annual referral volume (Spearman ρ=0.14; p=0.151) or usual referral delay (ρ=-0.10; p=0.280).

Conclusion: The findings point to clear knowledge gaps and a pattern of delayed referral. Targeted education regarding qualification criteria for surgery, realistic complication risks, and the ideal timing of referral—alongside closer, sustained ties with specialized epilepsy centers—could facilitate earlier evaluation of patients.

Keywords: Drug-resistant epilepsy, epilepsy surgery, knowledge, neurologists, neurosurgeons, referral

INTRODUCTION

Drug-resistant epilepsy (DRE) affects about one out of every three people diagnosed with epilepsy. It brings with it not just continuing seizures but also physical injuries, a lower quality of life, cognitive and psychosocial decline, higher death rates, and significantly increased healthcare costs.[1,2] For patients who go through a proper surgical evaluation, the procedure can stop seizures completely and bring functional gains that are rarely, if ever, achieved by simply adjusting medication.[3-6] Yet despite strong evidence supporting surgery, referral for evaluation remains strikingly low, and when it does happen, it's often far too late.[4,7]

The timeliness of referral largely depends on the treating physician's ability to recognize medication failure and to initiate the next step. Guidelines from the International League Against Epilepsy recommend that patients should be sent for surgical evaluation as soon as drug resistance is established regardless of age, seizure type, brain imaging findings, or other medical conditions.[3] Even so, surveys of clinicians in countries like Canada, Italy, and the United States keep turning up the same problems: confusion about what "two drugs failed" really means, exaggerated fears of surgical risk, overly narrow ideas about who is a good candidate, and various bottlenecks in the referral system.[8-13]

Information from Türkiye has been limited, particularly regarding studies that include adult neurology and neurosurgery specialists. One recent study with pediatricians and pediatric residents found considerable uncertainty about when and where to refer, as well as ongoing safety worries.[14] Neurologists and neurosurgeons work closely together along the epilepsy care pathway, but very few studies have examined their knowledge and how they coordinate referrals.

With this in mind, we set out to map knowledge, attitudes, and referral practices regarding epilepsy surgery among neurologists and neurosurgeons working in Türkiye. We also explored how these measures varied by subspecialty, institution type, and prior involvement in dedicated epilepsy surgery programs.

METHODS

Study Design and Participant Selection

Data for this cross-sectional survey were collected between June 1 and August 1, 2022, via national WhatsApp groups connecting neurologists and neurosurgeons in Türkiye. Specifically, we circulated the study invitation within two professional WhatsApp groups created for the 2020 national congresses: one for neurologists attending the Turkish Neurological Society congress and one for neurosurgeons attending the Turkish Neurosurgical Society Congress. Overall, 154 specialists agreed to participate and received the link to the online survey.

To check for duplicate submissions, the survey requested each physician's full name and official registration number. A review of these entry pairs revealed no overlap, and all identifying information was subsequently removed before creating the final analytical dataset. Out of the 154 invited physicians, 116 submitted their responses. Excluding eight incomplete questionnaires, 108 fully answered forms remained, yielding a 70.1% effective completion rate (Figure 1).

To be eligible, respondents had to be board-certified -neurologists or neurosurgeons actively practicing in Türkiye. We grouped their workplaces into four categories: university medical centers, training and research hospitals, public state clinics, and private medical facilities. All fully completed questionnaires received within the data collection-period were included in the analysis; no formal power calculation was performed a priori to determine a target sample size.

MAIN POINTS

- This study identified moderate knowledge of epilepsy surgery among neurologists and neurosurgeons in Türkiye, with the greatest gaps involving serious complication risk, magnetic resonance imaging-negative candidacy, and age eligibility.
- Knowledge scores were higher among physicians working in academic institutions and among those who had previously collaborated with an epilepsy surgery center.
- Nearly one-third of participants had made no surgical referral in the previous year, and only 12.0% reported referral within one year.
- Targeted education and stronger referral networks may help reduce delays in surgical evaluation.

Survey Instrumentation and Endpoint Metrics

The questionnaire was put together by a panel of three neurologists and split into five thematic blocks:

1. Practitioner background and clinical profile.

2. A set of nine factual questions that tested core knowledge.

3. Six items meant to explore personal attitudes and clinical impressions.

4. Direct questions about current referral behavior.

5. Previous contact with epilepsy surgery educational content and care pathways.

The knowledge questions were adapted from publicly available board-certification prep materials. They covered key points: -the formal definition of DRE; the efficacy of surgery; the candidacy of patients with a normal magnetic resonance imaging (MRI); the diagnostic role of video-electroencephalography (EEG) monitoring; optimal timing for surgery; complication rates; the relevance of generalized epilepsy; cognitive changes after surgery; and age-based candidacy rules-.

For each knowledge item, respondents could pick "true," "false," or "unsure." Correct answers scored one point; wrong answers and "unsure" responses received zero points. That gave each person a total knowledge score ranging from 0 and 9. The full questionnaire and the scoring guide are provided in Supplementary Material 1. The original primary knowledge scoring rule was retained.

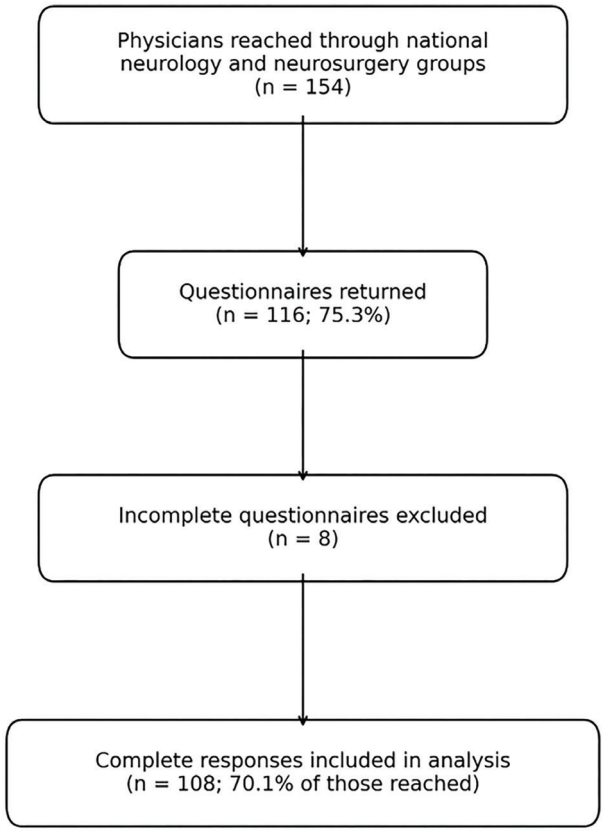

Figure 1. Participant flow through recruitment and analysis

To distinguish incorrect beliefs from acknowledged uncertainty, incorrect and "unsure" responses were summarized separately for each item (Supplementary Table 1).

The six attitude items were answered on a five-point Likert scale. Because these prompts addressed separate but related ideas—support for early referral, perceived bottlenecks, danger estimates, how participants rated their own knowledge, expected quality-of-life improvements, and geographic access to specialized care—we treated the overall sum (ranging from 6 to 30) as an exploratory composite endorsement index, rather than a formally validated psychometric instrument. Higher totals indicated stronger overall agreement with the combined statements. Because the items were not designed as a single favorable-versus-unfavorable attitude scale, no reverse-scoring was applied; the sum was interpreted only as an exploratory index of overall statement endorsement.

We also recorded several practice-related measures: the number of surgical referrals made during the previous year; the typical delay before a referral was initiated; weekly epilepsy patient volume; perceived geographic accessibility of specialized surgical centers; any formal training in epilepsy management; and prior collaboration with a dedicated surgical program.

Statistical Analysis

We summarized categorical variables using frequencies and percentages, while continuous measures were reported as means with standard deviations (± SD). We used Welch's independent-samples t-tests to compare knowledge scores between neurologists and neurosurgeons and between those with and without prior collaboration with an epilepsy surgery center. Differences across the four institutional categories were assessed with a one-way analysis of variance (ANOVA). Descriptive statistics captured item-level accuracy and referral patterns. All tests were two-tailed, and we considered p-values below 0.05 statistically significant. Analyses were performed with IBM SPSS Statistics (IBM Corp., Armonk, NY, USA). A post-hoc sensitivity analysis showed that, with our final specialty breakdown (72 vs. 36), the study had 80% power to detect a standardized mean difference of about 0.58 at a two-tailed alpha of 0.05. Institutional variance homogeneity was verified using Levene's test prior to running the primary ANOVA, after which post-hoc pairwise group differences were evaluated through Tukey's honestly significant difference (HSD). To measure the magnitude of the specialty-based effect, we derived Cohen's d.

Table 1. Participant characteristics and clinical exposure

Variable	Category	n	%
Specialty	Neurology	72	66.7
	Neurosurgery	36	33.3
Institution	University hospital	33	30.6
	Training and research hospital	33	30.6
	State hospital	23	21.3
	Private hospital	19	17.6
Prior collaboration with an epilepsy surgery center	Yes	36	33.3
	No	72	66.7
Formal epilepsy surgery education	Yes	42	38.9
	No	66	61.1

Non-parametric Spearman's rank correlations were used to assess how knowledge scores and the exploratory endorsement index correlated with ordinal referral volume and timing. Response options (correct, incorrect, unsure) were cross-tabulated for each knowledge item, and an exploratory chi-square analysis tested potential links between reported access barriers and clinicians' referral volume or latency.

Ethical Approval

The study protocol was approved by the Adıyaman University Non-Interventional Clinical Research Ethics Committee on March 15, 2022 (approval no: 2022/3-14). The work was conducted in accordance with the Declaration of Helsinki. Voluntary participation was emphasized throughout, with survey completion implying informed consent. Participant names and medical license numbers were collected solely for de-duplication and subsequently removed from the file prior to statistical analysis.

RESULTS

Participant Demographics and Backgrounds

The final sample included 108 physicians: 72 neurologists (66.7%) and 36 neurosurgeons (33.3%). University medical centers and training/research hospitals each contributed 30.6% of the cohort. Thirty-six participants (33.3%) reported a history of collaborative work with an epilepsy surgery center, and 42 (38.9%) reported receiving dedicated training in epilepsy surgery (Table 1).

Evaluated Knowledge and Clinical Perspectives

Across the whole group, the average knowledge score was 6.31±1.34 out of a possible 9, with a median of 6.5. The composite endorsement index on the six Likert-scale items came to 21.36±2.61 out of 30, though we emphasize that this aggregate was used only for description—it is not a validated, one-dimensional scale.

The areas where understanding was strongest included post-surgical cognitive changes (91.7% correct), the role of generalized epilepsy in candidacy (88.0%), the diagnostic value of video-EEG (88.0%), and the definition of DRE (86.1%). In contrast, the most striking knowledge gaps comprised three misconceptions: that serious complication rates typically exceed 10% (35.2% correct), that patients with normal MRI scans are not surgical candidates (37.0% correct), and that epilepsy surgery is restricted to children (47.2% correct) (Table 2 and Figure 2). When non-correct responses were analyzed separately, the MRI-negative surgical candidacy item had 38 incorrect responses (35.2%) and 30 unsure responses (27.8%), while the serious-complication item had 41 incorrect responses (38.0%) and 29 unsure responses (26.9%). Full item-level distributions are shown in Supplementary Table 1.

Neurologists scored significantly higher than neurosurgeons (6.49±1.35 vs. 5.94±1.26; p=0.044). Knowledge also differed markedly by institutional setting (p=0.003). The highest scores came from university hospitals (6.70±1.21) and training/research hospitals (6.67±1.43), followed by state hospitals (5.83±1.19) and private clinics (5.58±1.17) (Table 3 and Figure 3). The specialty difference was modest in magnitude (Cohen's d=0.41). Levene's test did not indicate unequal variances across institution groups

(p=0.375). Tukey HSD showed that knowledge scores were higher in university hospitals than in private hospitals (mean difference =1.12; p=0.015) and in training/research hospitals than in private hospitals (mean difference =1.09; p=0.019); the remaining pairwise comparisons were not significant.

Finally, physicians who had worked together with an epilepsy surgery center achieved notably higher knowledge scores than those without such experience (6.78±1.24 vs. 6.07±1.34; p=0.008) (Table 3 and Figure 4).

Referral Practices and Care Accessibility

An analysis of actual referral activity over the preceding year showed that 32 physicians (29.6%) did not send a single patient for surgical evaluation. A larger group of 42 clinicians (38.9%) referred one to two patients, while 27 (25.0%) referred three to five patients. Only 7 respondents (6.5%) facilitated more than five referrals.

Timing also varied widely. Only 13 participants (12.0%) reported to us that they usually make a referral within the first year after identifying drug resistance. The majority waited longer: 42 (38.9%) were referred after one to three years, 39 (36.1%) after three to five years, and 14 (13.0%) after more than five years. Moreover, a substantial proportion of the sample, 45 respondents (41.7%), reported difficulty accessing specialized epilepsy surgery centers (Table 4). Knowledge score was not significantly associated with annual referral volume (Spearman ρ=0.139; p=0.151) or with usual referral timing (ρ=-0.105; p=0.280). The exploratory endorsement index was not associated with the knowledge score (ρ=-0.057; p=0.557) or with referral timing (ρ=-0.069; p=0.480), but showed a weak positive association with annual referral volume (ρ=0.233; p=0.015). Perceived difficulty of access was not significantly associated with referral volume (χ^2=2.20; p=0.531) or with referral timing (χ^2=5.25; p=0.154).

Table 2. Correct-response rates for knowledge items

Item	Knowledge domain	Correct, n (%)
Q4	Definition of drug-resistant epilepsy	93 (86.1)
Q5	Expected seizure-free rate	80 (74.1)
Q6	MRI-negative surgical candidacy	40 (37.0)
Q7	Role of video-electroencephalography	95 (88.0)
Q8	Timing of surgical evaluation	90 (83.3)
Q9	Serious complication risk	38 (35.2)
Q10	Generalized epilepsy candidacy	95 (88.0)
Q11	Postoperative cognitive change	99 (91.7)
Q12	Age eligibility	51 (47.2)

Percentages are based on 108 complete responses. Item labels summarize the tested domain and do not reproduce the questionnaire wording verbatim

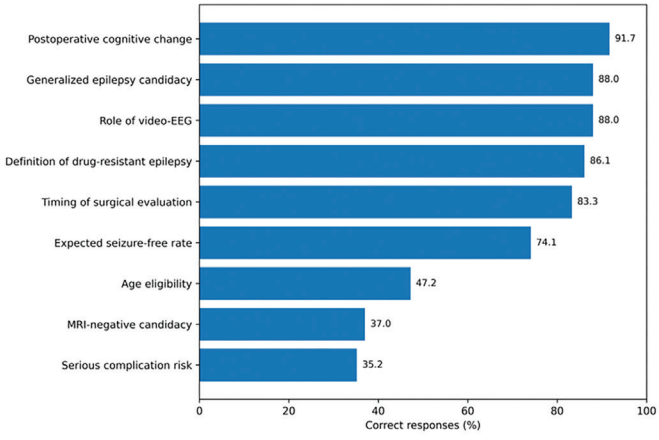

Figure 2. Correct-response rates for the nine epilepsy surgery knowledge items
EEG: Electroencephalography, MRI: Magnetic resonance imaging

DISCUSSION

This cross-sectional study demonstrates modest factual knowledge alongside consistently delayed and often very limited referral practices among neurologists and neurosurgeons across Türkiye. The most significant knowledge gaps centered on inflated fears about surgical complications, poor recognition that patients with a normal MRI can still be surgical candidates, and confusion about age boundaries for eligibility. Nevertheless, knowledge scores failed to correlate significantly with either the frequency or delay

Table 3. Subgroup comparisons of knowledge score

Comparison	Group	n	Mean ± SD	p-value
Specialty	Neurology	72	6.49±1.35	0.044
	Neurosurgery	36	5.94±1.26	
Institution	University hospital	33	6.70±1.21	0.003
	Training and research hospital	33	6.67±1.43	
	State hospital	23	5.83±1.19	
	Private hospital	19	5.58±1.17	
Prior collaboration	Yes	36	6.78±1.24	0.008
	No	72	6.07±1.34	

Welch independent-samples t-tests were used for two-group comparisons; one-way analysis of variance was used for institution categories. Post-hoc Tukey HSD: university vs. private, p=0.015; training/research vs. private, p=0.019; all other institution pairwise comparisons, p>0.05
SD: Standard deviation, HSD: Honestly significant difference

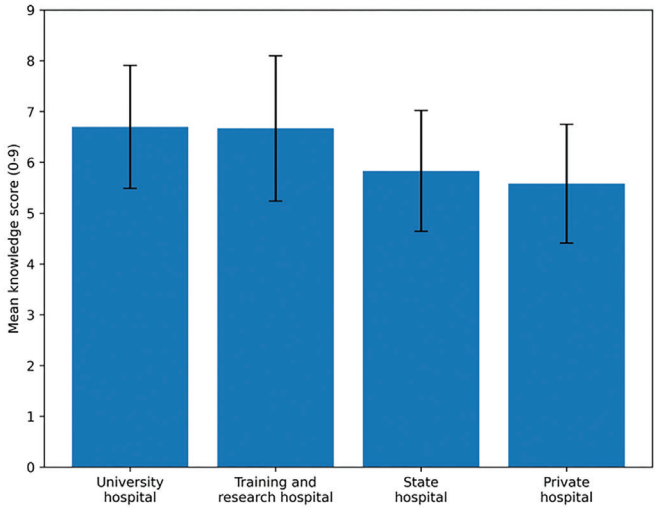

Figure 3. Mean knowledge scores by institutional setting. Error bars indicate standard deviations

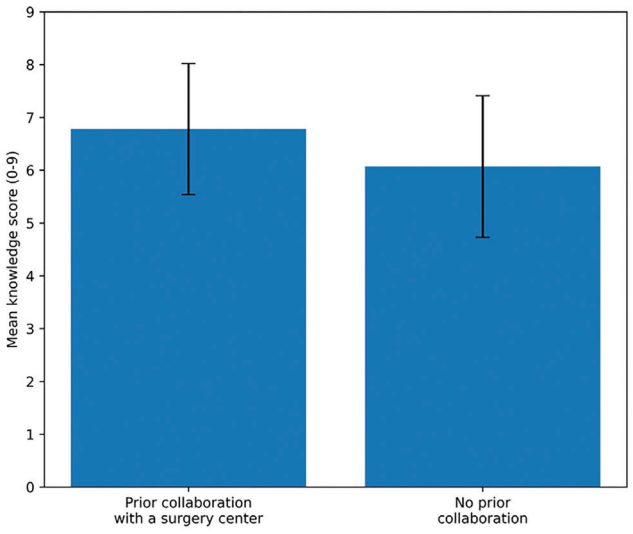

Figure 4. Mean knowledge scores according to prior collaboration with an epilepsy surgery center. Error bars indicate standard deviations

Table 4. Referral practices and perceived access

Variable	Category	n	%
Referrals in the previous year	None	32	29.6
	1-2 patients	42	38.9
	3-5 patients	27	25.0
	>5 patients	7	6.5
Usual time before referral	<1 year	13	12.0
	1-3 years	42	38.9
	3-5 years	39	36.1
	>5 years	14	13.0
Perceived access to surgery centers	Difficult	45	41.7
	Not difficult/other	63	58.3

of referrals. Thus, while knowledge gaps and delayed transfers co-occur, treating this overlap as proof that limited knowledge directly drives referral patterns would be unwarranted. The Likert-scale items we included provide context regarding institutional barriers and personal attitudes, but they do not constitute a validated psychometric instrument; therefore, that portion of the data should be regarded as exploratory.

The difference in knowledge scores between neurologists and neurosurgeons, although statistically significant, was modest (Cohen's d=0.41). That's not particularly surprising. Neurologists manage multiple drug trials sequentially, so they observe the transition to DRE unfolding in real time. Neurosurgeons, by contrast, often become involved later, after medical options have been exhausted. This gap should not considered a shortcoming specific to a single specialty. Both groups are central to patient selection, pre-surgical counseling, and the initial referral step, and the overall numbers suggest that there is considerable scope for educational improvement for both groups.

Much more striking was the divide associated with the institutional setting. Physicians working in university hospitals and tertiary training/research centers consistently outperformed colleagues based in public state hospitals or private clinics. This institutional disparity may reflect tertiary and academic settings offering routine exposure to video-EEG monitoring, presurgical terminology, and multidisciplinary case rounds, as well as access to dedicated epilepsy surgery units. Unmeasured in the present study, these operational variables should be regarded as plausible hypotheses that require future evaluation rather than as confirmed mechanisms.

These item-by-item results track closely with what has been reported internationally. A nationwide Canadian study found that many neurologists were still applying arbitrary seizure-frequency thresholds or had trouble accurately identifying the two-drug criterion for DRE.[8] An Italian survey similarly showed widespread misunderstandings and a tendency to overestimate surgical risks.[11] In work focusing on the New York metropolitan area, almost all neurologists viewed surgery as safe, yet only 56.9% actually followed guidelines by referring after two failed medication trials.[12] A national survey from Pakistan documented even sharper under-referral and a general hesitation to bring up surgical options with patients.[13] Closer to home, a recent Turkish pediatric survey echoed these messages, pointing to broad uncertainty around safety and timing.[14] Taken together, the literature indicates a consistent finding: endorsement of surgery in principle rarely translates into timely clinical action. Recent work reinforces the multifactorial nature of referral barriers. Hill et al.[15] identified knowledge, self-efficacy, practice environment, and coordination with epilepsy centers as relevant factors in neurologists' referral decisions. A systematic review similarly found recurring barriers involving knowledge, attitudes, communication, and referral coordination.[16] A nationwide Mexican survey reported gaps in DRE criteria and awareness of referral centers,[17] while Tokumoto et al.[18] demonstrated substantial facility-level differences in epilepsy care and referral in Japan.

The behavioral data from our study only reinforce this persistent implementation gap. Roughly a third of respondents had not referred a single patient in the previous year; only 12.0% managed a referral within a 12-month window; two-thirds had never worked directly with an epilepsy surgery facility. Past collaboration with a specialized epilepsy unit was associated with higher knowledge performance; however, determining whether collaboration drives knowledge or whether highly knowledgeable clinicians actively seek such partnerships remains beyond the scope of a cross-sectional study.

Structural accessibility warrants separate scrutiny, given that 41.7% of respondents identified difficulties accessing dedicated surgical facilities. While exploratory chi-square analyses showed no significant association between access hurdles and referral volume or timing, this high prevalence indicates that structural barriers to access should be considered alongside knowledge gaps. Consequently, overcoming these barriers requires pairing professional education with structural enhancements such as regional care pathways, telehealth options, and standardized inter-center communication channels.

For these reasons, future educational efforts need to focus on concrete decision-making thresholds rather than solely on broad awareness. The failure of two properly chosen and tolerated antiseizure medications should trigger an immediate referral for

a full surgical evaluation no delays.[2,3] Curricula should explicitly demystify non-lesional MRI-negative epilepsy, provide realistic complication rates, clarify that adult and older candidates can be suitable, and stress that a referral for an assessment is not the same thing as a final commitment to operate. Structured clinical pathways, teleconsultation, and regular joint case conferences would substantially benefit state and private hospitals that currently lack routine contact with specialized surgical centers.

Study Limitations

When interpreting the outcomes of this research, several limitations must be acknowledged. First, because the survey relied on voluntary enrollment and was disseminated via professional WhatsApp networks, selection bias is plausible, meaning that the study cohort may not perfectly mirror the entire population of neurosurgeons and neurologists operating across Türkiye. Second, geographic data were not collected, which precludes a definitive assessment of regional representation. Additionally, the aggregate sample size was relatively small for detailed subgroup analyses. Furthermore, the objective knowledge questionnaire was compiled and adapted from open-source board-certification study materials rather than an established psychometric tool; consequently, formal evaluations of test reliability and item discrimination indices were omitted. Similarly, the six Likert-scale statements were intended for exploratory analysis, addressed several distinct themes, and were not formally validated as an attitude scale. Additionally, data concerning patient referral behaviors depended entirely on self-reports rather than on verification through official clinical databases or hospital registries. Finally, the cross-sectional design precludes causal inference; clinicians with greater knowledge may simply be more proactive in seeking collaborative links with epilepsy surgical facilities. A notable constraint is that reliable census data covering active national specialists are missing; consequently, determining what percentage of the broader pool of neurologists and neurosurgeons our sample constitutes is not feasible.

Conversely, this study features notable methodological advantages. It successfully captured and combined the distinct professional viewpoints of both neurosurgeons and neurologists, measured clinical comprehension at the individual-question level, evaluated the role of institutional settings and care delivery environments, and obtained complete, usable data from 108 practitioners. The strong internal coherence of these observations, paired with their close parallels to findings from diverse international medical frameworks, underscores their utility for shaping future educational programs and structural system improvements.

CONCLUSION

Medical specialists in neurology and neurosurgery throughout Türkiye demonstrate a moderate grasp of factual data regarding epilepsy surgery, yet actual patient referrals continue to be subject to extensive delays. The primary knowledge gaps involve overestimating the risk of complications, failing to recognize the eligibility of individuals with normal MRI scans, and misunderstanding age boundaries. Affiliation with university or tertiary teaching hospitals, alongside a history of working in tandem with specialized epilepsy surgery centers, is closely linked to higher knowledge performance. Because knowledge score itself was not significantly associated with referral volume or timing, future efforts should address both educational gaps and structural barriers to access rather than assuming a single mechanism for delayed referral.

Ethics

Ethics Committee Approval: The study protocol was approved by the Adıyaman University Non-Interventional Clinical Research Ethics Committee on March 15, 2022 (approval no: 2022/3-14).

Informed Consent: Voluntary participation was emphasized throughout, with survey completion implying informed consent.

Acknowledgments

Generative artificial intelligence tools were used for language editing, assistance with statistical analysis, manuscript formatting, preparation of tables, and creation of data visualizations and figures based on the study results. Artificial intelligence was not used to generate, modify, or fabricate study data.

Footnotes

Authorship Contributions

Concept: A.B.E., A.Z.A.T., Design: A.B.E., A.Z.A.T., Data Collection or Processing: A.B.E., Analysis or Interpretation: A.B.E., A.Z.A.T., Literature Search: A.B.E., A.Z.A.T., Writing: A.B.E., A.Z.A.T.

Conflict of Interest: No conflict of interest was declared by the authors.

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Supplementary Material 1 Link: https://d2v96fxpocvxx.cloudfront.net/688d2d00-d207-464d-89b6-73f393f4f50c/content-images/a8af6765-17d4-4efc-91b3-056db748e295.pdf

Supplementary Table 1 Link: https://d2v96fxpocvxx.cloudfront.net/688d2d00-d207-464d-89b6-73f393f4f50c/content-images/546b31a9-4b5d-460a-ab31-6ec735d07fdd.pdf

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Turkish Translation of the 2025 Updated Classification of Epileptic Seizures

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Keywords: Epilepsy, taxonomy, consciousness, semiology, terminology

Dear Editor,

The International League Against Epilepsy (ILAE) has updated its 2017 seizure classification to establish a common language in clinical practice and standardize seizure descriptions. The new classification, published by Beniczky et al.[1] in 2025, retains the previous framework while introducing important changes to seizure classification and description. As the Turkish Epilepsy Society, we prepared Turkish translations of the updated classification materials to make them accessible to healthcare professionals in our country. The Turkish translations of Table 1 and Figures 1-2 have been published on the ILAE's official website in the section entitled "updated classification of epileptic seizures-2025" alongside translations into other languages. These translations enable Turkish-speaking healthcare professionals to access the classification's structure and its basic and expanded versions in Turkish.

Before finalizing the new classification, the ILAE invited clinicians to evaluate the proposed update and incorporated their feedback into the development process. In our 2024 article, we discussed this process and the main features of the proposal, informed colleagues about the proposed changes, and emphasized their importance for epilepsy practice.[2] We also outlined the fundamental approach of the proposed classification and the anticipated changes to be discussed. With the classification finalized in 2025, the updated approach to seizure classification and description has been established.[1] Preparing the Turkish translations continues our efforts to inform colleagues about these developments.

The four main categories have been retained: focal seizures, unknown (whether focal or generalized), generalized seizures, and unclassified seizures. A major change is the replacement of "awareness" with "consciousness" as a classifier. Consciousness is operationally defined as both awareness, assessed through recall, and responsiveness, assessed using verbal and motor tasks. Impairment of either component warrants classification as a seizure with impaired consciousness. The distinction between "motor" and "non-motor" has also been replaced by an approach based on the presence or absence of observable manifestations. The new classification distinguishes "classifiers," which determine seizure type, from "descriptors," which detail clinical features. The basic version provides a simpler classification for clinical use, whereas the expanded version allows semiological features to be listed chronologically. Generalized negative myoclonic seizures, absent from the previous classification, have been added to the expanded version. The classification also recognizes that epileptic spasms may occur across different onset categories. Whereas the previous classification listed 63 seizure types, the new approach defines 21 seizure types, with other seizure features expressed through descriptors, thereby enhancing flexibility and clinical applicability.

Making these Turkish translations available will support the integration of current scientific knowledge into clinical practice and promote consistent terminology in epilepsy care. We hope this work will benefit neurologists, child neurologists, and all healthcare professionals working in epilepsy.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.B.Ç., S.B., G.K., Concept: M.B.Ç., S.B., G.K., Design: M.B.Ç., S.B., G.K., Data Collection or Processing: M.B.Ç., S.B., G.K., Analysis or Interpretation: M.B.Ç., S.B., G.K., Literature Search: M.B.Ç., S.B., G.K., Writing: M.B.Ç., S.B., G.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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2026 Reviewer Index

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