Depressive Disorder in Epilepsy: An Evidence-Based Case Report

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Abstract

Introduction: *Depression affects approximately 22.9% of people with epilepsy (PWE), substantially higher than the general population. This comorbidity is mediated by complex neurobiological mechanisms including neuroinflammation, GABA-glutamate imbalance, and hypothalamic-pituitary-adrenal (HPA) axis dysregulation, and is further exacerbated by psychosocial stigma.*
Case Presentation*: We report the case of a 19-year-old woman with epilepsy diagnosed at age 6 who had been treated with phenytoin for 12 years. She developed depressive symptoms following prolonged bullying and social stigma, presenting with persistent sadness, anhedonia, self-harm behaviors, and subsequent psychotic features. Initial management included sertraline and aripiprazole. A drug-drug interactions between sertraline and phenytoin resulted in ataxia and gait imbalance, necessitating a switch in antiepileptic therapy to levetiracetam, carbamazepine, and clobazam.*
Methods: *A systematic literature review using PubMed, Cochrane, and Google Scholar databases, focusing on studies published between 2018 amd 2023 evaluating antidepressant treatment in PWE.*
Result: *Selective serotonin reuptake inhibitors (SSRIs) are recommended as first-line treatment for depression in epilepsy (recommendation level B). Venlafaxine reduced Hamilton Depression Rating Scale (HAMD) scores by 7.59 points compared with no treatment (RR 3.25; 95%CI 1.19–8.90; NNT = 4). Paroxetine versus doxepin demonstrated an RR of 1.16 (95%CI 0.88–1.52; NNT = 9), while amitriptyline versus nomifensine showed an RR of 0.55 (95%CI 0.28–1.06; NNT = 3). SSRIs (sertraline or citalopram) reduced Beck Depression Inventory (BDI) scores by 4.90 points compared with cognitive behavioral therapy.*
Conclusion: *SSRIs are effective to reduce depressive symptoms in PWE, with venlafaxine representing a viable alternative for non-responders. However, clinicians should carefully consider potential drug-drug interactions, particularly CYP450 enzyme inhibition, which may increase antiepileptic drug levels and require dose adjustments and close monitoring.*

Keywords: *Epilepsy, Depression, SSRI, Antidepressants, Comorbidity, Drug-drug interactions.*

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Laporan Kasus Berbasis Bukti: Depresi pada Epilepsi

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Abstrak

Pendahuluan: *Depresi terjadi pada 22,9% penderita epilepsy, jauh lebih tinggi dibandingkan populasi umum. Kondisi ini melibatkan mekanisme neurobiologis kompleks, termasuk neuroinflamasi, disfungsi GABA–glutamat, dan disregulasi sumbu HPA, serta diperberat oleh stigma psikososial.*
Presentasi Kasus: *Seorang perempuan 19 tahun dengan epilepsi sejak usia 6 tahun telah mendapat terapi fenitoin selama 12 tahun. Pasien mengalami depresi akibat perundungan kronis dan stigma sosial, dengan gejala sedih menetap, anhedonia, perilaku melukai diri, dan kemudian muncul gejala psikotik. Terapi awal yang diberikan berupa sertralin dan aripiprazol. Interaksi obat sertralin dan fenitoin menyebabkan ataksia dan gangguan keseimbangan sehingga terapi antiepilepsi diganti menjadi levetiracetam, karbamazepin, dan klobazam.*
Hasil: *Selective serotonin reuptake inhibitor (SSRI) direkomendasikan sebagai terapi lini pertama untuk depresi pada epilepsi (tingkat rekomendasi B). Venlafaxin dilaporkan menurunkan skor Hamilton Depression Rating Scale (HAMD) rata-rata 7,59 poin dibanding tanpa terapi (RR 3,25; IK95% 1,19–8,90; NNT = 4). Paroxetin dibanding doxepin menunjukkan RR 1,16 (IK95% 0,88–1,52; NNT = 9), sementara amitriptilin dibanding nomifensin menunjukkan RR 0,55 (IK95% 0,28–1,06; NNT = 3). SSRI (sertralin atau citalopram) mampu menurunkan skor Beck Depression Inventory (BDI) rata-rata 4,90 poin dibanding terapi kognitif perilaku.*
Kesimpulan: *SSRI efektif menurunkan gejala depresi pada penderita epilepsi, dengan venlafaxin sebagai alternatif pada pasien yang tidak responsif. Namun, potensi interaksi obat, khususnya inhibisi enzim CYP450 yang dapat meningkatkan kadar obat antiepilepsi, perlu diperhatikan melalui penyesuaian dosis dan pemantauan ketat.*

Kata Kunci: *Epilepsi, Depresi, SSRI, Antidepresan, Komorbiditas, Interaksi obat*

Introduction

Epilepsy is a prevalent neurological disorder affecting individuals worldwide. In 2021, approximately 51.7 million people, equivalent to 0.7% of the global population, were living with epilepsy. Notably, one-third of individuals with epilepsy also experience comorbid psychiatric conditions. Neuroimaging studies in patients with primary psychiatric disorders, such as depression and schizophrenia, have demonstrated abnormalities in brain networks that overlap with implicated in temporal lobe epilepsy (TLE), particularly within the amygdala and hippocampi.[1] Additionally, treatment with anti-seizure medications may presipitate or exacerbate psychiatric symptoms.[2]

Depression is one of the most prevalent psychiatric comorbidity associated with epilepsy. The global prevalence of depression among people with epilepsy (PWE) is substantially higher than that in the general population, estimated at 22,9%.[3,4] Epilepsy and depression share several biological mechanisms, including neuroinflammation processes that disrupts serotonin metabolism and dysregulate the hypothalamus-pituitary-adrenal (HPA) axis, leading to elevated corticosteroid levels.[3] Both conditions are also linked to disturbances in GABAergic and glutamatergic neurotransmission, contributing to seizure and

depressive symptoms, respectively.[5] Both epilepsy and depression are also associated with disturbed GABA and glutamate neurotransmitters, resulting in seizure and depressive symptoms, respectively.[6,7] Furthermore, PWE frequently experience limited social support, which may heighten stress levels. Reduced social support and elevated stress haven been identified as significant predictors of depression in this population.[8]

Although depression is highly prevalent among PWE, information regarding optimal treatment strategies remains limited. Current treatment recommendations are largely based on clinical consensus rather than robust empirical data.[9] Moreover, the available evidence on the effectiveness of antidepressants for managing depression in PWE is insufficient and and highly variable.[10]

Case Presentation

A 19-year-old woman had been diagnosed with epilepsy at 6 years of age. She had been treated with phenytoin for approximately 12 years, during which her seizures occurred 1-2 times per year. Her longstanding history of epilepsy led to persistent bullying beginning in early elementary school.

In January 2022, she presented with persistent sadness triggered by bullying and accusations from classmates regarding an alleged debt. She cried daily, refused to attend school, and engaged in self-injurious behavior by intentionally hitting her head against the wall when distressed. Her symptoms worsened after her brother left for an Islamic boarding school, leaving her feeling increasingly lonely. She questioned why she was born and why she had epilepsy. Additional symptoms included loss of appetite, anergia, and frequent drowsiness. She denied suicidal ideation and hallucinations at that time. She was evaluated in the psychiatric clinic and started on sertraline.

By July 2022, she began experiencing auditory hallucinations commanding her to stay in her room becaise someone allegedly intended to kill her. She also reported visual hallucinations of cockroaches and snakes under her bed. Her psychiatrist then added aripiprazole to her medication.

In October 2022, aripiprazole was replaced with risperidone due to lack of improvement in her positive symptoms. Two days after starting risperidone, she developed marked lethargy and unsteadiness, resulting in an inability to walk without assistance. Risperidone was discontinued, and aripiprazole was reintroduced. Because of relapsing seizures, valproic acid was added alongside phenytoin.

In February 2023, she developed imbalance and ataxia and was hospitalized for 12 days. After discharge, she was able to walk with assistance. Reducing phenytoin to once daily led to symptomatic improvement. She continued to feel sad about her illness, although psychotic symptoms had resolved. Her psychiatrist subsequently discontinued aripiprazole.

However, decreasing the phenytoin dose again resulted in seizure relapse, prompting referral for further management. Neurologists initiated a taper of both phenytoin and valproic acid and replaced them with levetiracetam, carbamazepine, and clobazam. The prolonged use of phenytoin was suspected to have contributed to her lethargy and gait disturbances.

In June 2023, she developed status epilepticus and presented to the emergency department with seven seizures in a single day. Clobazam and carbamazepine were administered to control her seizures. Following complete discontinuation of both phenytoin and valproic acid, her balance improved, and she regained the ability to walk independently. She continued treatment with levetiracetam, carbamazepine, and clobazam.

After finishing high school, her depressive symptoms resolved; however, she continued to struggle with loneliness due to limited social connections. Seizures remained well controlled with her current antiepileptic medications. She continued sertraline for her depressive syptoms and reported ongoing difficulty forming relationships due to past bullying experience. Written informed consent for publication of this case was obtained from the patient and a family member.

Clinical Problems

The clinical problem is formed using the PICO method follows:
What is the drug of choice recommended for patients with depressive features in epilepsy?

- P (Patient) : Patient with epilepsy and depression.
- I (Intervention) : Antidepressant for epilepsy with depressive features.
- C (Comparison) : -
- O (Outcome) : >50% reduction in depressive symptoms.

Methods

We conducted electronic searches for PubMed, Cochrane, and Google Scholar for this review. The searches were performed from July to September 2023. Eligible studies included full-text articles, systematic review, meta-analyses, randomized controlled trials published within the preceding five years.

Results

Two studies were included: clinical practice recommendations by the International League Against Epilepsy (ILAE) Task Force (2021) and a systematic review by Maguire, et al. (2021) were included. The ILAE recommendations, developed under the Commission on Psychiatry, were based on systematic review of treatment for depression in epilepsy and supplemented by clinical consensus. Despite the high prevalance of depression in epilepsy, evidence-based data remain limited, resulting in most recommendations being consensus-based. Nevertheless, these guidelines provide a general framework for

Table 1. Search Strategy

Search Engine	Keywords	Results
Pubmed	((epilepsy[Title/Abstract]) AND (treatment[Title/Abstract])) AND (depression[Title/Abstract])	73
Cochrane	MeSH descriptor: [Epilepsy] explode all trees AND MeSH descriptor: [Depression] explode all trees AND MeSH descriptor: [Psychotropic Drugs] explode all trees	1
Google Scholar	"epilepsy" AND "depression" AND "treatment"	46.800

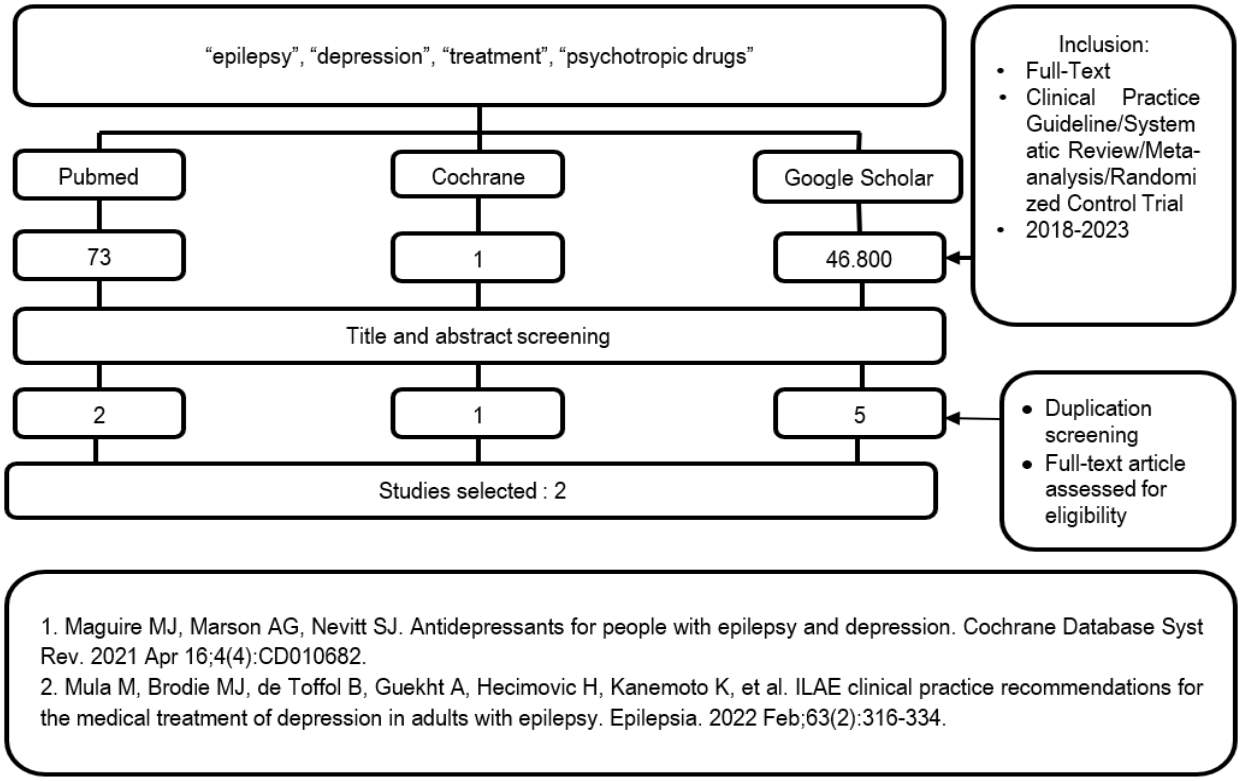

Figure 1. Study Flow Diagram

Study selection was performed using the following keywords. Study selection was conducted through title and abstract screening, resulting in 2 studies from PubMed, 1 study from the Cochrane Library, and 5 studies from Google Scholar. After duplicate removal and full-text assessment for eligibility, 2 studies were included in the final analysis.

healthcare professionals managing depression in epilepsy. Clinical evidence was classified into six categories according to the World Federation of Societies of Biological Psychiatry (WFSBP) guidelines; in the absence of external evidence, recommendations were designated as clinical consensus (CONS) and classified as unknown (U).

In PWE with depressive symptoms, SSRIs are the first-line pharmacological treatment, while psychological interventions may be considered as initial therapy (recommendation level: WFSBP = 1; ILAE = B). In patient who do not respond to SSRIs, switching to venlafaxine may be considered (recommendation level: WFSBP = 3; ILAE = C). Combined psychological and pharmacological treatment is recommended for patients with moderate to severe depression, partial response to antidepressant; or poor adherence to medication (recommendation level: WFSBP = 3; ILAE = C). Cognitive behavioral therapy has the strongest evidence among psychological interventions for treating depression in epilepsy, whereas evidence for other modalities remains limited. Seizure incidence following SSRI treatment did not differ from placebo in a pooled analysis of Phase II-III psychotropic drug trials approved by the US Food and Drug Administration between 1985 and 2004.

Owing to their complex metabolism, antidepressants may cause pharmacokinetic interactions with antiepileptic drugs. SSRIs can induce drug-drug interactions through inhibition of cytochrome-P450 isoenzymes, necessitating dose adjustment in some cases (recommendation level: WFSBP = CONS; ILAE = U CONS). Fluoxetine, fluvoxamine, and sertraline inhibit CYP2C9 and may increase phenytoin concentrations. However, sertraline-related CYP2C9 inhibition has been observed only at the maximum recommended dose (200 mg/day) and is generally considered clinically insignificant.

Antidepressants treatment should be continued for at least 6 months after remission of a first depressive episode. In patients with a history of prolonged or recurrent episodes, treatment should be extended to at least 9 months and maintained longer if residual symptoms persist. Gradual tapering over 1-4 weeks is recommended to minimize discontinuation symptoms; rather than abrupt withdrawal (recommendation level: WFSBP = CONS; ILAE = U CONS).

A systematic review by Maguire, et al (2021) evaluated the efficacy and safety of antidepressants for treatment of depression in epilepsy. The review included four randomized controlled trials (RCTs) and six non-randomized studies of interventions (NRSIs), comprising a total of 626 participants with epilepsy and depressive symptoms treated with antidepressants. Participants of any age, with any epilepsy type, were eligible if depression was diagnosed using standardized criteria. Treatment response was assessed using validated outcome measures and compared with baseline. Across the ten included studies, six comparisons were available for analysis.

Of the six comparison groups, three assessed a ≥50% reduction in depressive symptoms. Compared with no treatment, venlafaxine demonstrated a significant reduction in depressive symptoms (RR 3.25; 95% CI 1.19–8.90; number needed to treat [NNT] = 4). In contrast, paroxetine compared with doxepin showed a non-significant effect (RR 1.16; 95% CI 0.88–1.52; NNT = 9), while amitriptyline compared with nomifensine yielded an RR of 0.55 (95% CI 0.28–1.06; NNT = 3).

Improvement in depressive symptoms was also assessed using mean depression scores. Venlafaxine reduced the mean Hamilton Depression Rating Scale (HAMD) score by 7.59 points compared with no intervention. SSRIs (sertraline or citalopram) resulted in a mean Beck Depression Inventory (BDI) score that was 4.90 points lower than that observed with cognitive behavioral therapy.

Despite the observed efficacy, evidence supporting the effectiveness of antidepressants for treating depression in epilepsy remains limited. The certainty of evidence was rated as moderate to very low, owing to limited outcome data and a high risk of bias. Substantial heterogeneity was observed, with ≥50% improvement in depressive symptoms ranging from 43% to 82% in RCTs and from 24% to 97% in non-randomized studies. Nevertheless, available evidence indicates that SSRIs reduce depression severity in patients with epilepsy. In cases of inadequate response to SSRIs, switching to venlafaxine may be considered, as venlafaxine demonstrated a greater than 50% improvement in depressive symptoms compared with no treatment.

Discussion

Depression is a major psychiatric comorbidity associated with epilepsy. Its prevalence in PWE is higher than that in the general population, with a global estimate of 22.9%.[3,4] his increased prevalence is attributed to shared biological mechanisms and psychosocial fac-

Table 2. Effectiveness of Antidepressants for Treatment of Depression

Comparison		RR from > 50% reduction in depressive symptoms (95% CI)	NNT	Mean Depression Score	Certainity of the Evidence (GRADE)
Paroxetine	Doxepine	1.16 (0.88 to 1.52)	8.849 ≈ 9	The mean HAMD score was lower 0.65 points in Paroxetine group	Moderate
Amitriptyline	Nomifensine	0.55 (0.28 to 1.06)	2.824 ≈ 3	n/a	Low
Venlafaxine	No treatment	3.25 (1.19 to 8.90)	3.55 ≈ 4	The mean HAMD score was lower 7.59 points in Venlafaxine group	Low
Sertraline	CBT	n/a	n/a	The mean BDI score was lower 0.50 points in Sertraline group	Moderate to low
Citalopram (before)	Citalopram (after)	n/a	n/a	SMD in HAMD score was 1.17 lower, indicating improved outcomes	Low to very low
SSRI (Sertraline /citalopram)	CBT	n/a	n/a	The mean BDI score was lower 4.90 points in SSRI group	Very low

BDI, Beck depression inventory; CBT, cognitive behavior therapy; HAM-D, Hamilton rating scale for depression; NNT, number needed to treat; RR, relative risk; SSRI, selective serotonin reuptake inhibitor; 95%CI, 95% confidence of intervals.

tors.

Epilepsy and depression exhibit a complex bidirectional relationship mediated by shared neurobiological mechanisms. Both conditions are associated with increased hippocampal interleukin-1β signaling and elevated pro-inflammatory mediators, including transforming growth factor-β and cyclooxygenase-2, leading to chronic neuroinflammation and reduced serotonergic neurotransmission.[3,11] Dysregulation of GABAergic and glutamatergic signaling contributes to seizure activity and depressive symptoms, respectively. Structural brain changes, including atrophy of the frontal and temporal lobes and reduced serotonin binding in these regions, have been reported in both disorders.[12] Chronic epilepsy is further linked to neurodegeneration and hypothalamic–pituitary–adrenal axis dysregulation, resulting in elevated corticosteroid levels associated with depression.[3]

The patient was diagnosed with epilepsy at 6 years of age and experienced stigma and bullying from schoolmates beginning in early elementary school. These experiences contributed to persistent sadness, low self-esteem, and existential questioning related to living with epilepsy. Psychosocial factors are major contributors to depression in PWE, and multiple such factors were evident in this patient. Depressive mood and low self-esteem were present prior to the onset of a depressive disorder, consistent with evidence linking mood-related constructs to depression.[5]

After living with epilepsy for 12 years, prolonged exposure to stigma adversely affected the patient's quality of life and contributed to the development of psychiatric symptoms.[7,8] Children with epilepsy are frequently subjected to bullying and often exhibit impairments in theory of mind and social competence, predisposing them to social isolation and loneliness.[14] In this patient, limited social support and increased psychosocial stress—both established predictors of depression in PWE—were evident.[8]

Her depression was treated with sertraline. Biological mechanisms play a central role in depression associated with epilepsy; chronic epilepsy induces neuroinflammation, leading to reduced serotonergic neurotransmission.[3] Antidepressant therapy should therefore be guided by the complex biological interplay between neuroinflammation in epilepsy and serotonin deficiency in depression. Evidence indicates that SSRI treatment reduces mean BDI scores by 4.90 points compared with CBT alone.[10]

Nevertheless, CBT remains a relevant psychological intervention for patients with epilepsy and depression, particularly in addressing psychosocial contributors to depressive symptoms.[9] A biopsychosocial approach should be adopted to optimize treatment outcomes and improve quality of life in people with epilepsy.

The patient developed imbalance and ataxia in February 2023, possibly related to pharmacokinetic interactions between antidepressants and AEDs. SSRIs inhibit CYP2C9, potentially increasing phenytoin concentrations. Although sertraline-associated CYP2C9 inhibition is generally considered clinically insignificant and has been reported only at the maximum recommended dose (200 mg/day),[9,12,15] clinically relevant interactions have been described.

A case report by Nathan (2017) documented an increase in free phenytoin levels from 0.8 µg/mL to 4.9 µg/mL in a patient with well-controlled seizures on phenytoin who received sertraline 100 mg/day for depression, accompanied by lethargy and confusion. Symptoms resolved after phenytoin discontinuation and gradual tapering of sertraline.[16] In the present case, the neurologist switched AED therapy from phenytoin and valproic acid to levetiracetam, carbamazepine, and clobazam, resulting in resolution of imbalance and ataxia.

Despite these considerations, SSRIs remain effective and well-studied treatments for depression in PWE. In cases of inadequate response, switching to venlafaxine may be considered. However, evidence on pharmacological management of depression in PWE remains limited, highlighting the need for further studies to inform best clinical practice.[9]

Conclusion

SSRI reduce depression symptoms in PWE, and venlafaxine may be considered in patients with limited or no response to SSRI. However, owing to their complex metabolism, antidepressants may cause pharmacokinetic interactions with antiepileptic drugs; therefore, dose adjustment should be considered. To date, evidence supporting the use of antidepressants for treating depression in patients with epilepsy remains limited, and current clinical practice recommendations are largely based on expert consensus. To inform more robust treatment guidelines, large-scale RCTs across diverse populations of people with epilepsy are needed.

Declaration of Conflicting Interests

The authors declare no conflicts of interest with respect to the research, authorship, and publication of this article.

References

1. Feigin VL, Vos T, Nair BS, Hay SI, Abate YH, Abd Al Magied AHA, et al. Global, regional, and national burden of epilepsy, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Public Health. 2025 Mar;10(3):e203–27.
2. Mula M, Kanner AM, Jetté N, Sander JW. Psychiatric comorbidities in people with epilepsy. Neurol Clin Pract. 2021 Apr;11(2):e112–20.
3. Singh T, Goel RK. Epilepsy associated depression: an update on current scenario, suggested mechanisms, and opportunities. Neurochem Res. 2021 Jun;46(6):1305–21.
4. Scott AJ, Sharpe L, Hunt C, Gandy M. Anxiety and depressive disorders in people with epilepsy: A meta-analysis. Epilepsia. 2017;58(6):973–82.
5. Hermann BP, Seidenberg M, Bell B. Psychiatric comorbidity in chronic epilepsy: identification, consequences, and treatment of major depression. Epilepsia. 2000;41(s2):S31–41.
6. Temple J, Fisher P, Davies C, Millar C, Gemma Cherry M. Psychosocial factors associated with anxiety and depression in adolescents with epilepsy: a systematic review. Epilepsy Behav. 2023 Dec;149:109522.
7. Mula M, Sander JW. Psychosocial aspects of epilepsy: a wider approach. BJPsych Open. 2016 Jul;2(4):270–4.
8. Gandy M, Sharpe L, Perry KN. Psychosocial predictors of depression and anxiety in patients with epilepsy: a systematic review. J Affect Disord. 2012 Nov;140(3):222–32.
9. Mula M, Brodie MJ, De Toffol B, Guekht A, Hecimovic H, Kanemoto K, et al. ILAE clinical practice recommendations for the medical treatment of depression in adults with epilepsy. Epilepsia. 2022 Feb;63(2):316–34.
10. Maguire MJ, Marson AG, Nevitt SJ. Antidepressants for people with epilepsy and depression. Cochrane Database Syst Rev. 2021 Apr 16;4(4):CD010682.
11. Liu GY, Fu FJ, Chou YX, Ye MS, Ouyang

YL, Yan MD, et al. Frontiers and hotspots in comorbid epilepsy and depression: a bibliometric analysis from 2003 to 2023. Front Neurol. 2024;15:1413582.
12. Miziak B, Czuczwar SJ, Pluta R. Comorbid epilepsy and depression—pharmacokinetic and pharmacodynamic drug interactions. Front Pharmacol. 2022 Oct 7;13:988716.
13. Batchelor R, Taylor MD. Young adults with epilepsy: Relationships between psychosocial variables and anxiety, depression, and suicidality. Epilepsy Behav. 2021 May;118:107911.
14. Stewart E, Catroppa C, Gonzalez L, Gill D, Webster R, Lawson J, et al. Theory of mind and social competence in children and adolescents with temporal lobe epilepsy. Neuropsychology. 2019 Oct;33(7):986–95.
15. Bousman CA, Stevenson JM, Ramsey LB, Sangkuhl K, Hicks JK, Strawn JR, et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2D6 , CYP2C19 , CYP2B6 , SLC6A4 , and HTR2A genotypes and serotonin reuptake inhibitor antidepressants. Clin Pharmacol Ther. 2023 Jul;114(1):51–68.
16. Nathan KT, Hopkins Gil HA, DiLoreto SE, Ta NA, Caprio TV. Sertraline and phenytoin drug interaction in a geriatric patient. Ann Longterm Care. 2017;25(5):46-8.