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CLINICAL RESEARCH ARTICLE

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Comparison of neurological and psychiatric profiles of people with epilepsy based on the presence and timing of potentially psychologically traumatic experiences

Deniz Ertan[a,b], Nicolas Mezouar[a], Alexis Tarrada[b], Louis Maillard[b], Wissam El-Hage[c,d] and Coraline Hingray [b,e]

[a]Institut la Teppe, Tain l'Hermitage, France; [b]CHRU de Nancy, Département de Neurologie, Nancy, France; [c]CHU de Tours, Tours, France; [d]UMR 1253, iBraiN, Université de Tours, INSERM, Tours, France; [e]Pôle Hospitalo-Universitaire de Psychiatrie d'Adultes du Grand Nancy, Centre Psychothérapique de Nancy, Laxou, France

ABSTRACT

Objective: While psychological trauma in people with epilepsy (PWE) is a major issue, there is limited research on the interactions between such trauma and epilepsy. Therefore, our primary aim is to describe types and timing of potentially psychologically traumatic experiences (PPTE) in relation to epilepsy onset. Our secondary objective is to evaluate the impact of the timing of the PPTE on patients' psychiatric and neurological profiles.

Methods: We conducted an observational study involving 182 PWE, excluding patients with comorbid functional/dissociative seizures. All participants underwent a comprehensive psychiatric evaluation, including biographical, neurological, psychiatric, and traumatic data collection through a semi-structured clinical interview and standardized scales. We compared the neurological and psychiatric characteristics of three groups of patients: those without PPTE, those with PPTE occurring before the onset of epilepsy, and those with PPTE occurring after the onset of their epilepsy.

Results: Sixty-one patients (33.5%) reported having experienced PPTE before the onset of epilepsy, 65 patients (35.7%) reported having experienced PPTE after the onset of their epilepsy, and 56 patients (30.8%) had no history of PPTE neither before nor after the onset of epilepsy. The 'before' group had a significantly higher prevalence of epilepsy localized in the temporal lobe ($p = .043$). The 'after' group showed significantly more general psychiatric symptoms ($p = .026$), as well as more postictal mood and anxiety symptoms ($p = .014$). Additionally, the 'before' group reported a higher number of past traumatic experiences, with childhood traumatic experiences being more prevalent. According to our multinomial logistic regression model, higher temporal localization ($p = .028$) and fewer febrile seizures ($p = .030$) were significant predictors for the 'before' group.

Significance: This study highlights the potential impact of the timing of PPTE on patients' psychiatric and neurological profiles. It underscores the importance of systematically assessing psychiatric and posttraumatic comorbidities in PWE. The role of trauma in temporal epilepsy requires further investigation.

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HIGHLIGHTS

- The timing of potentially psychologically traumatic experiences (PPTE) may influence patients' psychiatric and neurological profiles.
- Patients with a history of PPTE tend to have higher rates of psychiatric comorbidities.
- Early exposure to PPTE may impact the temporal localization of epilepsy.

Comparación de perfiles neurológicos y psiquiátricos en personas con epilepsia según la presencia y temporalidad de experiencias potencialmente traumáticas

Objetivo: Aunque el trauma psicológico en personas con epilepsia (PCE) es un problema relevante, existe escasa investigación sobre la interacción entre dicho trauma y la epilepsia. Nuestro principal objetivo es describir los tipos y temporalidad de experiencias potencialmente traumáticas (EPT) en relación con el inicio de la epilepsia. El objetivo secundario es evaluar el impacto de la temporalidad de las EPT en los perfiles psiquiátricos y neurológicos de los pacientes.

Métodos: Se realizó un estudio observacional con 182 PCE, y excluyó a pacientes con comorbilidad de crisis funcionales/disociativas. Todos los participantes se sometieron a una evaluación psiquiátrica integral, incluyendo la recopilación de datos biográficos, neurológicos, psiquiátricos y traumáticos mediante una entrevista clínica semiestructurada y escalas estandarizadas. Se compararon las características neurológicas y psiquiátricas de tres grupos: pacientes sin EPT, con EPT previas al inicio de la epilepsia y con EPT posteriores al inicio de la epilepsia.

Resultados: Sesenta y un pacientes (33.5%) reportaron haber experimentado EPT antes del inicio de la epilepsia; 65 pacientes (35.7%) reportaron EPT posteriores al inicio de la

CONTACT Coraline Hingray c.hingray@chru-nancy.fr CHRU de Nancy, Département de Neurologie, 29 Av. du Maréchal de Lattre de Tassigny, 54000 Nancy, France

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epilepsia, y 56 pacientes (30.8%) no presentaron antecedentes de EPT ni antes ni después del inicio de la epilepsia. El grupo de 'antes' presentó una mayor prevalencia significativa de epilepsia localizada en el lóbulo temporal ($p = .043$). El grupo de 'después' mostró más síntomas psiquiátricos generales ($p = .026$) y síntomas postictales de ánimo y ansiedad ($p = .014$). Además, el grupo de 'antes' reportó un mayor número de experiencias traumáticas previas, siendo más frecuentes los traumas en la infancia. Según el modelo de regresión logística multinomial, la mayor localización temporal ($p = .028$) y un menor número de crisis febriles ($p = .030$) fueron predictores significativos del grupo 'antes'.
Importancia: Este estudio resalta el impacto potencial del momento de las EPT en los perfiles psiquiátricos y neurológicos de los pacientes. Subraya la importancia de evaluar sistemáticamente las comorbilidades psiquiátricas y postraumáticas en las PCE. Se requiere mayor investigación sobre el papel del trauma en la epilepsia temporal.

1. Introduction

There is a growing body of literature exploring the relationship between psychological trauma and epilepsy, revealing a complex and multidimensional link between the two. Psychological trauma encompasses direct or indirect exposure to threatening events that endanger an individual's life or integrity, such as serious injury, sexual emotional, or physical violence, unexpected loss of a loved one, or natural disasters (American Psychiatric Association, 2013; Crocq, 2003). Several studies have demonstrated an increased risk of developing epilepsy following psychological trauma (Chen et al., 2017). For instance, a nationwide longitudinal study found that patients with posttraumatic stress disorder (PTSD) had a 3.72 times higher risk of developing epilepsy (CI: 2.27–6.11) compared to the general population (Chen et al., 2017). Similarly, a study conducted after a life-threatening earthquake in Japan observed a significant increase in the number of patients with seizures during the weeks following the event (Shibahara et al., 2013). Additionally, research has shown a correlation between traumatic experiences and the onset of epilepsy with people with epilepsy (PWE) reporting a higher prevalence of potentially psychologically traumatic experiences (PPTE) compared to control groups (Soncin et al., 2021). Some of these PWE (18.6%) also reported a chronological link between PTSD symptoms and the onset of epilepsy (Soncin et al., 2021). Moreover, studies have highlighted a higher incidence of epilepsy among soldiers experiencing work-related stress (Moshe et al., 2008), further emphasizing the association between psychological trauma and epilepsy.

The scientific literature underscores the significant impact of adverse life events on brain development and the onset of chronic health issues, including heart problems, obesity, substance abuse, and chronic depression in adulthood. These factors contribute to an overall increase in morbidity and mortality rates (Anda et al., 2006). Furthermore, studies have explored the potential role of psychological distress and negative life events during childhood in the development of epilepsy, potentially increasing vulnerability to seizures (Koe et al., 2009; Novakova et al., 2013). Animal research has elucidated a notable association between hypothalamic–pituitary–adrenal (HPA) stress meditators and seizures (Novakova et al., 2013). Chronic stress has been linked to significant health issues and structural changes in the brain. For example, stress-induced morphological alterations in the hippocampus and amygdala have been demonstrated (Vyas et al., 2002). Of particular concern is the impact of stress and adverse experiences during early life on brain development (Anda et al., 2006). Such experiences can exacerbate epileptogenesis, rendering the brain more susceptible to seizures (van Campen et al., 2014). Moreover, some studies have emphasized the role of psychological trauma in shaping brain networks in PWE, particularly within the limbic system and amygdala networks (Lanteaume et al., 2012; Soncin et al., 2022). Experimental data in adult animals also provide a basis for the interactions between psychological trauma and epileptogenesis (Becker et al., 2015). Recently, a new concept of 'psychoepileptogenesis' was proposed to explain the interaction between the changes induced by stressful events and epileptogenesis (Jhaveri et al., 2023; Lanteaume et al., 2009; Soncin et al., 2021). These findings underscore the importance of early intervention and support to mitigate the long-term effects of stress on brain health and seizure susceptibility.

Indeed, research indicates that PWE tend to report a higher frequency of traumatic experiences compared to the general population (Díaz-Olavarrieta et al., 1999; Kwon et al., 2011). Additionally, PWE are at an increased risk of experiencing social stigma, which can have a detrimental effect on their quality of life (Hermann & Jacoby, 2009). Furthermore, studies have highlighted that PWE face a higher risk of suicide and are more susceptible to being victims of physical assaults (Kwon et al., 2011). Moreover, epileptic seizures themselves can be experienced as traumatic events. Some individuals may develop PTSD and other psychiatric comorbidities following seizure episodes (Chung & Allen, 2013; Ertan et al., 2024;

Labudda et al., 2018; Mariotti et al., 2021; Soncin et al., 2023). This underscores the multifaceted impact of epilepsy on psychological well-being and highlights the importance of addressing both the physical and mental health aspects of the condition in clinical management.

Some recent studies have advanced our understanding of PTSD in PWE (Ertan et al., 2024; Soncin et al., 2023). For instance, a recent study showed alterations in cognition and emotion linked to both epilepsy and PTSD (Soncin et al., 2023). The authors found that patients with epilepsy who also experienced PTSD had difficulties with emotional inhibition and exhibited attentional overcontrol.

While psychological trauma in PWE is a significant concern, there is a notable lack of research investigating the potential interactions between such trauma and epilepsy. Moreover, a history of psychological trauma in PWE is often overlooked in routine epilepsy clinics. To address this gap, our study had several aims. Firstly, we aimed to investigate the prevalence and types of exposure to PPTE in PWE. Secondly, we sought to explore the potential influence of the timing of PPTE on patients' psychiatric and neurological profiles. To achieve these aims, we compared the neurological and psychiatric characteristics of three groups of patients: those who experienced PPTE before the onset of epilepsy, those who experienced PPTE after the onset of epilepsy, and those with no history of PPTE. Additionally, we aimed to examine differences in traumatic characteristics between patients who experienced PPTE before and after the onset of epilepsy. This comprehensive approach should allow us to gain insights into the complex relationship between psychological trauma and epilepsy, and its implications for patient care.

2. Methods

2.1. Study sample

We prospectively recruited PWE from the inpatients units of two epileptology clinics, namely Nancy University Hospital, and La Teppe Institute, between 2021 and 2022. Additionally, data from patients included in our previous study (Mariotti et al., 2021) were incorporated into the present study. The diagnosis of epilepsy was established by experienced epileptologists in accordance with the International League Against Epilepsy (ILAE) criteria (Fisher et al., 2017). Patients with comorbid functional/dissociative seizures were excluded from the study. Moreover, individuals for whom the temporal relationship between traumatic events and the onset of epilepsy was unclear, such as those who could not recall whether the epilepsy or the traumatic event occurred first, were also excluded from the study ($n = 11$). This study protocol was reviewed and approved by the local ethical committee (reference: 2022PI33).

2.2. Data collection

Data were systematically collected through psychiatric evaluations conducted by psychologists or psychiatrists specialized in Epi-Psy (Hingray et al., 2021) at each of the health centers involved in the study. This comprehensive assessment encompassed the exploration of biographical, neurological, psychiatric, and traumatic data of the PWE through semi-structured clinical interviews and standardized scales. Biographical data collected included information on sex, age, marital status, education level, and profession. Neurological data comprised details such as age at onset of epilepsy, type of seizures, seizure semiology (including loss of consciousness, urinary or fecal incontinence, falls, and seizure-related injuries), seizure frequency, history of traumatic brain injury, history of birth complications, and history of encephalitis. Psychiatric data encompassed information pertaining to interictal disorders and peri-ictal symptoms experienced by patients.

General interictal psychiatric disorders (non-specific psychiatric disorders without temporal link with seizures). Psychiatric assessments for mood, anxiety, addiction, and eating disorders were conducted using a semi-structured clinical interview format guided by the Mini-International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998). To complement this assessment, we employed standardized scales. Specifically, we utilized the Neurological Disorders Depression Inventory for Epilepsy (NDDIE), a scale developed according to DSM-IV criteria, to evaluate depression symptoms in PWE (Micoulaud-Franchi et al., 2015; Mula et al., 2016). Additionally, the Generalized Anxiety Disorder (GAD-7) was administered to assess anxiety symptoms (Löwe et al., 2008; Micoulaud-Franchi et al., 2016). Furthermore, the Toronto Alexithymia Scale-20 (TAS-20) was utilized to assess the presence of alexithymia, a condition characterized by difficulties in identifying and describing emotions (Bagby et al., 1994).

Specific Interictal psychiatric disorders (psychiatric disorders specifically associated with epilepsy without temporal link with seizures). We evaluated the presence of interictal dysphoric disorder based on Blumer's criteria (Blumer et al., 2004). This evaluation involved a careful examination of symptoms and criteria outlined by Blumer to identify this specific psychiatric condition in PWE. The Blumer criteria require the presence of at least three out of the following eight symptoms: depressed mood, asthenia,

atypical pain, insomnia, fear and anxiety, irritability, euphoric mood, and instability of mood, within the past 12 months, resulting in dysfunction in the daily lives of individuals. Furthermore, we investigated the presence of anticipatory anxiety, which refers to the fear or significant distress associated with the anticipation of having a seizure. Additionally, we explored avoidance behaviors related to anticipatory anxiety, which may manifest as efforts to avoid situations or activities perceived as potential triggers for seizures (Hingray et al., 2019). To further assess anxiety specific to epilepsy, we utilized the Epilepsy Anxiety Survey Instrument (EASI), both in its short (8 items) and long versions (18 items). This self-report scale was specifically developed to comprehensively evaluate anxiety symptoms and their impact on PWE (Scott et al., 2019). Both short and long version of EASI showed excellent validity and reliability scores. The area under curve of the short version was respectively .89 and the Cronbach α was .91. For the long version, the Cronbach α was .94 and test-rest score was .77 (Scott et al., 2019).

Peri-Ictal Symptoms (specific psychiatric symptoms temporally associated with seizures). We thoroughly explored the presence of peri-ictal mood and anxiety symptoms, encompassing pre-ictal (several hours to 3 days before the seizure), ictal (during the seizure) and post-ictal (several hours to 3 days after the seizure) symptoms (Mula et al., 2010). There is no validated scale to evaluate peri-ictal mood and anxiety changes. Therefore, we asked the patients if they experienced any mood changes, unusual worries or uncommon stress or fear before, during or after the seizure, lasting from several hours to three days.

2.3. Quality of life

Quality of life (QoL) of PWE was assessed through QoL in epilepsy inventory (QOLIE-31), a 31 items self-assessment scale (Amruth et al., 2014). We obtained a subscore for each of the 7 subscales: worry about seizures, general QoL, emotional well-being, feeling of energy/vitality, memory/cognitive impairment, adverse effects due to drug treatment, social functioning. A good internal consistency was demonstrated for the QOLIE-31 overall scale (Cronbach's $\alpha = .93$) and for the subscales (Cronbach's $\alpha > .70$), along with good reliability for the total scale (test-retest = .89) and for the subscales (test-retest ranging from .68 to .84) (Amruth et al., 2014).

2.4. Psychotraumatic data

To assess psychotraumatic experiences comprehensively, we employed a combination of clinical psychiatric interviews and standardized scales. Life Event Checklist for DSM-5 (LEC-5), a self-report measure, was used to assess traumatic life events, supplemented by the Childhood Traumatic Experiences (CTQ-28), a self-administered questionnaire designed to identify various forms of childhood abuse (Weathers et al., 2013; Paquette et al., 2004). Furthermore, we evaluated the presence of PTSD using a clinical interview format guided by Mini-International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998), and measured the severity of PTSD symptoms using the PCL-5, a self-administered questionnaire specifically designed to gauge the intensity of PTSD symptoms (Ashbaugh et al., 2016). We also evaluated the presence of dissociative symptoms using the Dissociative Experience Scale (DES), a self-questionnaire developed to quantify the level of dissociation experienced by individuals (Bernstein & Putnam, 1986).

2.5. Statistical analysis

First, descriptive statistics were calculated for the collected data. Second, for continue variables, we performed univariate ANOVA or non-parametric Kruskal–Wallis tests (regarding the results of the homogeneity of variance test and test of normality) to compare the demographic, neurological and psychiatric characteristics of patients with PPTE before the onset of epilepsy, patients with PPTE after the onset of epilepsy and patients with no traumatic history (group 'before' vs group 'after' vs group 'no PPTE'). Third, for categorical variables, we performed Chi-square test or Fisher exact test to test the difference between the expected frequencies and the observed frequencies. We performed t Student or Mann–Whitney U non-parametric tests for continuous variables, to compare the psychotraumatic profiles of patients with PPTE before the onset of epilepsy and the patients with PPTE after the onset of epilepsy. Again, for categorical variables, we performed Chi-square test or Fisher exact Test. Finally, the effect sizes were calculated through Cohen's d; eta squared and Cramer's V for the variables with a significant p value $< .05$. For Cramer's V, the values $< .20$ were considered to have small effect size, values between .20 and .40 were considered to have medium effect size and the values $> .40$ were considered to have large effect size. For Eta Squared, the values .01 were considered to have small effect size; the values .06 were considered to have medium effect and the values .14 were considered to have large effect size. For Cohen's d, the values .50 were considered to have small effect size; the values between .50-.80 were considered to have medium effect size and the values $> .80$ were considered to have large effect size. Descriptive and statistical analyses were performed using SPSS 24.0 software (IBM). Since our study is exploratory in nature, we did not perform Bonferroni correction (Armstrong, 2014; Perneger, 1998).

Finally, we performed multinomial logistic regression analyses to examine the variables associated with different trauma groups. The results are presented as odds ratios (OR) with 95% confidence intervals (CI) and *p*-values. We included the following variables in the analysis: age of onset of epilepsy, febrile seizures, temporal localization, NDDIE scores, GAD-7 scores, and the presence of at least one psychiatric disorder.

3. Results

3.1. Prevalence and type of potentially psychologically traumatic experiences

We enrolled 182 PWE (96 female; 52.7%), with a mean age of 33.9 years (SD = 12.02). The majority of our patients had focal drug-resistant epilepsy. Among them, 61 (33.5%) reported experiencing PPTE before the onset of epilepsy, 65 (35.7%) reported trauma after the onset of epilepsy, and 56 (30.8%) had no history of PPTE. Traumatic experiences were reported to have occurred during childhood only (26.9%, $n = 49$), adulthood only (24.2%, $n = 44$), during both childhood and adulthood (17%, $n = 31$), or repeatedly (44%, $n = 80$). Specific types of trauma included sexual trauma (15.9%, $n = 29$), physical trauma (24.7%, $n = 45$), emotional trauma (34.1%, $n = 62$), and traumatic experiences related to sudden loss of a close relative or friend (24.2%, $n = 44$). Furthermore, PTSD was prevalent in our study population, with 23.6% ($n = 43$) currently meeting criteria for PTSD, and 22% ($n = 40$) reporting a history of PTSD.

3.2. Demographic characteristics: comparison of the three groups

We found that demographic characteristics did not significantly differ between the groups classified based on the timing of PPTE (i.e. group 'before,' group 'after,' and group 'no PPTE'). These findings are detailed in Supplementary Material, Table S1.

3.3. Neurological and epilepsy characteristics: comparison of the three groups

Regarding epilepsy characteristics (Table 1), we observed significant differences among the groups. Specifically, we found a significant difference in the age at onset of epilepsy ($p < .001$). Those with PPTE occurring after the onset of epilepsy had significantly earlier onset of epilepsy compared to the 'before' group (12.01 ± 10.05 vs 20.9 ± 10.05; H(2) = 25.58, $p < .001$, $\eta^2 = .16$). We also noted a significantly higher proportion of temporal localization in the 'before' group compared to the 'after' group (78.3% vs 57.1%; $\chi^2(2) = 6.29$, $p = .043$, V = .118). Conversely, the 'before' group exhibited a significantly lower proportion of febrile seizures compared to the other groups (3.3% vs 20% vs 17.9%; $\chi^2(2) = 8.58$, $p = .014$, V = .217). Moreover, we observed a higher proportion of patients experiencing falls during seizures in the 'after' group compared to those with no traumatic experience (64.1% vs 38.2%; $\chi^2(2) = 7.96$ $p = .019$, V = 210).

3.4. Psychiatric characteristics: comparison of the three groups

We found a lower proportion of patients with psychiatric comorbidity in the 'no PPTE' group compared to the other groups (42.9% vs 66.7% vs 54.6%; $\chi^2(2) = 8.29$, $p = .016$, V = .214).

Additionally, the 'before' group exhibited a more frequent history of psychological/psychiatric follow-up (63.9% vs 41.1%; $\chi^2(2) = 6.67$ $p = .036$, V = .191), and had a significantly higher proportion of family history of psychiatric disorder, compared to the 'no PPTE' group (42.6% vs 10.7%; $\chi^2(2) = 15.14$, $p < .001$, V = .288) (Table 2).

We did not find significant differences in alexithymia scores between the three groups.

The 'before' group demonstrated a significantly higher NDDIE score compared to the 'no PPTE' group (10.98 ± 4.81 vs 9.19 ± 4.19; H(1) = 29.25, $p = .002$), along with a higher prevalence of history of eating disorders (18% vs 3.6%, $\chi^2(2) = 6.25$, $p = .017$, V = .185). Conversely, the 'no PPTE' group reported a significantly better quality of life compared to the group 'before' (70.59 ± 20.26 vs 60.38 ± 20.21, H(1) = 22.21, $p = .021$) (Table 2).

Lastly, the 'after' group exhibited higher anxiety scores on the GAD-7 compared to the 'no PPTE' group (6.67 ± 5.4 vs 4.29 ± 4.7; H(1) = 24.148, $p = .009$) (Table 2).

Our analysis of psychiatric characteristics linked to epilepsy revealed several notable findings among the different groups. Firstly, the 'no PPTE' group reported a lower prevalence of post-ictal mood symptoms compared to the 'before' group (3.6% vs 14.8%; $\chi^2(1) = 4.28$ $p = .038$, V = .191) and to the 'after' group (3.6% vs 20%; $\chi^2(1) = 7.47$, $p = .011$, V = .249). Conversely, the 'after' group had a significantly higher prevalence of post-ictal anxiety symptoms compared to the 'before' group (20% vs 4.9%; $\chi^2(1) = 6.45$, $p = .015$, V = .226).

We observed significant differences in scores on the EASI, both the original (11.82 ± 12.58 vs 15.38 ± 11.87 vs 6.07 ± 8.57, H(2) = 7.635, $p = .022$, $\eta^2 = 0.93$) and short versions (6.33 ± 5.2 vs 8.19 ± 5.2 vs 3 ± 4.45, H(2) = 10.52, $p = .005$, $\eta^2 = .124$) 'among the groups'. *Post hoc* analysis revealed a significant difference between 'after' group and the 'no PPTE' group in terms of EASI scores (short version), with *p*-values of .008 (Table 3).

Table 1. Epilepsy characteristics.

	PPTE before onset of epilepsy $n = 61$ (33.5%)	PPTE after the onset of epilepsy $n = 65$ (35.7%)	No PPTE $n = 56$ (30.8%)	p value	Effect size
Neurological History					
Febrile seizures, *n* (%)	2 (3.3%)[x,y]	13 (20%)	10 (17.9%)	**.014**[c]	.217[v]
Birth Complication, *n* (%)	6 (10%)	10 (15.4%)	9 (16.1%)	.575[c]	
Head Injury, *n* (%)	12 (19.7%)	7 (10.8%)	8 (14.3%)	.369[c]	
Encephalitis, *n* (%)	4 (6.6%)	3 (4.6%)	5 (8.9%)	.654[f]	
Age at the onset of epilepsy, years (SD)	20.9 (9.96)[x]	12.01 (10.05)	16.87 (13.79)	**<.001**[k]	**.016**[e]
Treatment History					
Number of antiseizure medication, *n* (%)	2.28 (1.2)	2.49 (1.13)	2.39 (1.09)	.347[k]	
Type of seizure, *n* (%)					
Focal seizure	57 (93.4%)	57 (87.7%)	51 (91.1%)	.537[c]	
Generalized seizure	4 (6.6%)	8 (12.3%)	5 (8.9%)	.537[c]	
Secondarily generalized seizure	34 (55.7%)	36 (55.4%)	26 (46.4%)	.523[c]	
Multifocal	17 (28.3%)	15 (23.4%)	15 (27.3%)	.808[c]	
Localization, *n* (%)					
Temporal	47 (78.3%)[x]	36 (57.1%)	36 (65.5%)	**.043**[c]	.188[v]
Frontal	18 (30%)	20 (31.7%)	14 (25.5%)	.745[c]	
Occipital	2 (3.3%)	4 (6.3%)	3 (2.8%)	.765[f]	
Parietal	4 (6.7%)	9 (14.3%)	9 (16.4%)	.244[c]	
Insular	7 (11.7%)	10 (15.9%)	5 (9.1%)	.525[c]	
Lateralization, *n* (%)				.690[c]	
Left	30 (50.8%)	35 (55.6%)	29 (53.7%)		
Right	17 (28.8%)	21 (33.3%)	15 (27.8%)		
Bilateral	12 (20.3%)	7 (11.1%)	10 (18.5%)		
Frequency of seizures		%)	35 (63.6%)	.272[c]	
Frequency of focal seizures, mean (SD)	29.12 (116.18)	15.46 (43.38)	10.22 (10.3)	.562[k]	
Frequency of generalized seizures, mean (SD)	1.09 (2.21)	4.15 (16.23)	3.39 (13.37)	.479[k]	
Semiology					
Loss of consciousness during a seizure, *n* (%)	44 (72.1%)	50 (76.9%)	35 (63.6%)	.272[c]	
• Score ICI Total, mean (SD)	4.88 (6.31)	4 (5.28)	3.63 (5.72)	.718[k]	
• Score ICI Nature, mean (SD)	4.26 (7.02)	4.33 (5.92)	3.94 (6.33)	.817[k]	
• Score ICI Content, mean (SD)	2.52 (3.91)	3.03 (4.77)	1.89 (4.54)	.360[k]	
Urine or feces loss during a seizure, *n* (%)	21 (34.4%)	17 (26.2%)	12 (21.8%)	.300[c]	
Fall during a seizure, *n* (%)	31 (50.8%)	41 (64.1%)[z]	21 (38.2%)	**.019**[c]	.210[v]
Injuries during a seizure, *n* (%)	27 (44.3%)	34 (53.1%)	23 (41.8%)	.420[c]	

[c]Chi-square; [k]Kruskal-Wallis; [f]Fisher Exact; [v]Cramer's V; [e]Eta Squared; x: before vs after; y: before vs no PPTE; ICI: Ictal Consciousness Inventory.

3.5. Traumatic characteristics: comparison of the three groups

For traumatic characteristics (Table 4), we compared the groups 'after' and 'before' to elucidate differences between individuals who reported having experienced psychological trauma before versus after the onset of epilepsy. The 'before' group had a significantly higher prevalence of multiple traumas compared to the 'after' group (45.9% vs 24.6%; $\chi^2(1) = 6.27$, $p = .012$, V = .223). We also found a higher prevalence of a history of PTSD in the 'before' group (42.6% vs 21.5%; $\chi^2(1) = 6.45$, $p = .011$, V = .226). The group 'before' reported significantly earlier PPTE compared to the 'after' group (12.21 vs 20.58 years; U = 1917.00, z = 4.393, $p < .001$, $d = .92$). Additionally, the 'before' group had a higher number of PPTE compared to the 'after' group (2.3 vs 1.32; U = 1323.0, z = −3.387, $p < .001$, $d = .48$). Regarding types of traumatic experiences, the 'before' group exhibited a higher prevalence of sexual and physical traumas compared to the 'after' group (respectively; 31.1% vs 15.4%; $\chi^2(1) = 4.4$, $p = .036$, V = .187; 50.8% vs 21.5%; $\chi^2(1) = 11.75$, $p < .001$, V = .305). The 'before' group reported significantly higher prevalence of childhood traumatic experiences across all types of traumatic experiences. Scores on the CTQ-28 were significantly higher in the 'before' group, particularly in emotional abuse (9.43 vs 6.98; U = 1035.0, z = −2.44, $p = .015$, $d = .57$), physical abuse (7.26 vs 5.57; U = 1247.0, z = −3.858, $p < .001$, $d = .47$), and sexual abuse scores (7.43 vs 5.91; U = 1107.0, z = −2.456, $p = .014$, $d = .014$). However, there were no significant differences between the groups in terms of emotional or physical neglect scores. Also, the groups did not differ regarding PCL-5 scores. Our results did not show any significant difference between the 'before' and 'after' groups regarding DES total and sub-scores.

Our multinomial analysis revealed significant predictors for the 'before' and 'after' trauma groups, as detailed in the provided table (Table 5). The late onset of epilepsy (= .001), less febrile seizures (= .030) and higher temporal localization (= .028) were significant for the 'before' group. The results also showed a trend towards statistical significance for febrile seizures between 'before' and 'no trauma' group, with febrile seizures being less prevalent in the 'before' group ($p = .064$).

4. Discussion

The main objective of this study was to investigate the prevalence and types of PPTE in PWE. Additionally,

Table 2. Psychiatric comorbidities non-specific to epilepsy.

	PPTE before onset of epilepsy $n = 61$ (33.5%)	PPTE after the onset of epilepsy $n = 65$ (35.7%)	No PPTE $n = 56$ (30.8%)	p value	Effect size
Psychiatric History					
At least one psychiatric comorbidity, *n* (%)	40 (66.7%)[y]	42 (64.6%)[z]	24 (42.9%)	**.016**[c]	.214[v]
History of psychiatric follow-up, *n* (%)	39 (63.9%)[y]	38 (58.5%)	23 (41.1%)	**.036**[c]	.191[v]
History of hospitalization in psychiatry, *n* (%)	4 (6.6%)	8 (12.3%)	3 (5.4%)	.390[f]	
History of family psychiatric disorder, *n* (%)	26 (42.6%)[y]	17 (26.2%)	6 (10.7%)	**<.001**[c]	.288[v]
History of suicide attempt, *n* (%)	12 (19.7%)	11 (16.9%)	4 (7.1%)	.137[c]	
Psychiatric Medication					
Antidepressant, *n* (%)	8 (13.1%)	9 (13.8%)	7 (12.5%)	.976[c]	
Antipsychotic, *n* (%)	5 (8.2%)	1 (1.5%)	1 (1.8%)	.159[f]	
Anxiolytic, *n* (%)	7 (11.5%)	10 (15.4%)	3 (5.4%)	.211[c]	
General Psychiatric Disorders					
Mood Disorders					
• Current, *n* (%)	12 (19.7%)[y]	20 (30.8%)[z]	6 (10.7%)	**.025**[c]	.202[v]
• History, *n* (%)	29 (47.5%)[y]	34 (52.3%)[z]	11 (19.6%)	**<.001**[c]	.288[v]
• NDDIE, mean (SD)	12.13 (5.16)[y]	10.98 (4.81)	9.19 (4.19)	**.008**[k]	.057[e]
Anxiety Disorders					
• Current, *n* (%)	19 (31.1%)[y]	24 (36.9%)[z]	10 (17.9%)	.064[c]	
• History, *n* (%)	24 (39.3%)[y]	29 (44.6%)[z]	8 (14.3%)	**<.001**[c]	.276[v]
• GAD-7, mean (SD)	6.51 (5.4)	6.67 (5.4)[z]	4.29 (4.7)	**.018**[k]	.040[e]
Eating disorder					
• Current eating disorder, *n* (%)	3 (4.9%)	6 (9.2%)	1 (1.8%)	.233[f]	
• History of eating disorder, *n* (%)	11 (18%)[y]	7 (10.8%)	2 (3.6%)	**.044**[c]	.185[v]
Addiction disorder					
• Current addiction disorder, *n* (%)	21 (34.4%)	19 (29.2%)	13 (23.2%)	.802[c]	
• History of addiction disorder, *n* (%)	26 (42.6%)	22 (33.8%)	15 (26.8%)	.411[c]	
TAS-20, n (%)					
Alexithymia (according TAS score > 61)	12 (22.2%)	15 (27.3%)	4 (9.8%)	.104[c]	
Quality of Life, mean (SD)					
Overall quality	60.38 (20.21)[y]	61.4 (19.19)	70.59 (20.26)	**.023**[k]	.048[e]
Emotional well-being	63.7 (21.22)	64.7 (19.2)	70.05 (18.64)	.214[k]	
Energy/fatigue	49 (18.96)	47.59 (18.19)	49.13 (19.55)	.897[a]	
Cognitive functioning	55.75 (21.2)	61.94 (22.08)	56.44 (26.08)	.421[k]	
Medication effects	53.55 (30.17)	55.19 (29.41)	60.49 (28.29)	.472[k]	
Social functioning	56.28 (26.05)	58.29 (26.94)	62.4 (21.91)	.567[k]	
Seizure worry	54.06 (28.57)	57.4 (26.04)	58.07 (25.25)	.790[k]	

[c]Chi-square; [k]Kruskal-Wallis; [f]Fisher Exact; [v]Cramer's V; [e]Eta Squared: = 0.01; [y]: before vs no PPTE; [z] = after vs no PPTE; GAD-7: Generalized Anxiety Disorder; NDDIE: Neurological Disorders Depression Inventory in Epilepsy; TAS-20: Toronto Alexithymia Scale.

we aimed to explore the potential relationship between the timing of PPTE relative to the onset of epilepsy and the psychiatric and neurological profiles of patients.

4.1. Prevalence of trauma & comparison with general population

Our findings revealed that approximately 70% of our population had reported experiencing at least one PPTE in their lifetime, a prevalence consistent with that observed in the general population (Benjet et al., 2016). Indeed, studies have indicated that around 70% of individuals report experiencing at least one traumatic event during their lives (Benjet et al., 2016). Regarding specific types of trauma, research based on nationwide surveys conducted in the United States has shown that 8–12% of individuals were survivors of childhood sexual abuse before the age of 18, while 9–19% reported being physically assaulted during their childhood (Saunders & Adams, 2014). A meta-analysis has indicated that 27% of individuals reported being survivors of childhood emotional abuse (Stoltenborgh et al., 2012). Our findings were consistent with these prevalence rates, with 11% reporting childhood sexual abuse, 16.4% reporting childhood physical abuse, and 26.4% reporting childhood emotional abuse. It is also important to highlight that some epilepsy-specific studies showed a higher incidence of psychological trauma in drug-resistant epilepsy patients (78%) compared to a healthy control group (52%) (Soncin et al., 2021).

4.2. The time of trauma and epilepsy characteristics

4.2.1. Age of onset of epilepsy

We found some differences in the neurological profiles among the three groups of patients. First of all, patients who reported having PPTE after the onset of epilepsy exhibited a significantly earlier onset of seizures. This finding suggests that epilepsy might have commenced during early stages of life, potentially during the first few years, for this group. Consequently, it is less likely for these individuals to have experienced and reported a PPTE before the onset of their seizures, thus explaining this significant difference. Possibly, there might be a phenomenon of childhood amnesia, contributing to the reduced likelihood of recalling traumatic events occurring before the onset of epilepsy in this group.

Table 3. Epilepsy specific psychiatric characteristics.

	PPTE before onset of epilepsy $n = 61$ (33.5%)	PPTE after the onset of epilepsy $n = 65$ (35.7%)	No PPTE $n = 56$ (30.8%)	p value	Effect size
Peri-ictal Psychiatric symptoms (temporally associated with seizures)					
Mood disorder					
• Pre-ictal, *n* (%)	6 (9.8%)	5 (7.7%)	2 (3.6%)	.421[f]	
• Ictal mood symptoms, *n* (%)	3 (4.9%)	0 (0)	0 (0)	.064[f]	
• Post ictal mood symptoms, *n* (%)	9 (14.8%)[y]	13 (20%)[z]	2 (3.6%)	**.026**[c]	.200[v]
Anxiety disorder					
• Pre-ictal anxiety symptoms, *n* (%)	18 (29.5%)	13 (20%)	10 (17.9%)	.267[c]	
• Ictal anxiety symptoms, *n* (%)	19 (31.1%)	15 (23.1%)	12 (21.4%)	.423[c]	
• Post-ictal anxiety symptoms, *n* (%)	3 (4.9%)[x]	13 (20%)[z]	4 (7.1%)	**.014**[c]	.217[v]
Interictal Psychiatric symptoms (without temporal link with seizures)					
Interictal dysphoric disorder, *n* (%)	12 (19.7%)	14 (21.5%)	4 (7.1%)	.074[c]	
Anticipatory anxiety of a seizure, *n* (%)	27 (45%)	27 (42.2%)	17 (31.5%)	.303[c]	
EASI, mean (SD)	11.82 (12.58)	15.38 (11.87)[z]	6.07 (8.57)	**.022**[k]	.093[e]
EASI (short version), mean (SD)	6.33 (5.2)	8.19 (5.2)[z]	3 (4.45)	**.005**[k]	.124[e]
Behavior of limitation or avoidance, *n* (%)	19 (31.1%)	19 (29.2%)	11 (19.6%)	.327[c]	
Pharmaco-induced disorders					
Pharmaco-induced mood disorder, *n* (%)	21 (34.4%)	18 (27.7%)	12 (21.4%)	.294[c]	
Pharmaco-induce anxiety, *n* (%)	3 (4.9%)	8 (12.3%)	1 (1.8%)	.064[f]	

[c]Chi-square; [k]Kruskal-Wallis; [f]Fisher Exact; [v]Cramer's V; [e]Eta Squared; [x]: before vs after; [y]: before vs no PPTE; [z] = after vs no PPTE.

Table 4. Traumatic characteristics of patients with trauma before vs after the onset of epilepsy.

	PPTE before onset of epilepsy $n = 61$ (48.4%)	PPTE after the onset of epilepsy $n = 65$ (51.6%)	p value	Effect size
Trauma History				
Age of trauma, years, mean (SD)	12.21 (6.35)	20.58 (11.26)	**<.001**[m]	.92[d]
Number of traumas, mean (SD)	2.3 (2.68)	1.32 (1.03)	**<.001**[m]	.48[d]
Multiple traumas, *n* (%)	28 (45.9%)	16 (24.6%)	**.012**[c]	.223[v]
Temporality between onset of epilepsy and onset of trauma, years, mean (SD)	8.28 (7.63)	11.29 (9.52)	.52[m]	
Overall trauma				
Sexual, *n* (%)	19 (31.1%)	10 (15.4%)	**.036**[c]	.187[v]
Physical, *n* (%)	31 (50.8%)	14 (21.5%)	**<.001**[c]	**.**305[v]
Emotional, *n* (%)	35 (57.4%)	27 (41.5%)	.076[c]	
Loss of close relative, *n* (%)	23 (37.7%)	21 (32.3%)	.525[c]	
Traumatic seizure, *n* (%)	27 (45%)	38 (58.5%)	.132[c]	
Trauma during childhood				
Sexual, *n* (%)	14 (23%)	6 (9.2%)	**.035**[c]	.188[v]
Physical, *n* (%)	23 (37.7%)	7 (10.8%)	**<.001**[c]	.316[v]
Emotional, *n* (%)	31 (50.8%)	17 (26.1%)	**.004**[c]	.254[v]
Loss of close relative, *n* (%)	14 (23%)	5 (7.7%)	**.017**[c]	.213[v]
Trauma during adulthood				
Sexual, *n* (%)	5 (8.2%)	4 (6.2%)	.656[f]	
Physical, *n* (%)	13 (21.3%)	10 (15.4%)	.389[c]	
Emotional, *n* (%)	15 (24.6%)	12 (18.5%)	.402[c]	
Loss of close relative, *n* (%)	13 (21.3%)	17 (26.2%)	.524[c]	
PTSD				
Current PTSD, *n* (%)	22 (36.1%)	21 (32.3%)	.657[c]	
History of PTSD, *n* (%)	26 (42.6%)	14 (21.5%)	**.011**[c]	.226[v]
PCL-5				
Total, mean (SD)	17.19 (17.68)	11.58 (15.76)	.073[m]	
Re-experiencing, mean (SD)	4.65 (5.16)	3.05 (4.67)	.094[m]	
Avoidance, mean (SD)	2.92 (4.69)	1.9 (3.36)	.282[m]	
Hyperarousal, mean (SD)	5.19 (6.29)	3.62 (4.83)	.131[m]	
Negative alteration in cognition and mood, mean (SD)	4.53 (6.33)	2.95 (5.29)	.102[m]	
CTQ Score				
Physical neglect, mean (SD)	7.28 (3.31)	6.42 (2.42)	.273[m]	
Emotional abuse, mean (SD)	9.43 (5.27)	6.98 (3.01)	**.015**[m]	.57[d]
Physical abuse, mean score (SD) mean (SD)	7.26 (4.38)	5.57 (2.5)	**<.001**[m]	.47[d]
Emotional neglect, mean (SD)	10.23 (4.88)	8.67 (4.06)	.113[m]	
Sexual abuse, mean (SD)	7.43 (4.84)	5.91 (3.61)	**.014**[m]	.35[d]
DES				
Total, mean (SD)	14.88 (16.36)	15.05 (12.4)	.548[m]	
Depersonalization and derealization, mean (SD)	13.68 (19.35)	11.43 (11)	.850[m]	
Dissociative amnesia, mean (SD)	12.57 (15.87)	11.55 (12.71)	.663[m]	
Absorption, mean (SD)	17.92 (16.99)	21.34 (17.64)	.247[m]	

[c]Chi-square; [k]Kruskal-Wallis; [f]Fisher Exact; [m]Mann Whitney U; [v]Cramer's V; [d]Cohen's *d*. PCL-5: Posttraumatic stress disorder Checklist version DSM-5; CTQ: The Childhood Trauma Questionnaire; DES: Dissociative Experiences Scale.

Table 5. Regression analysis.

Time of trauma	OR adjusted (95% CI)	*p* value
Group Before (base) vs Group After		
Age at the onset of epilepsy	0.935 (0.898–0.974)	**.001**
Febrile seizure	6.091 (1.193–31.107)	**.030**
Temporal localization	0.359 (0.144–0.893)	**.028**
NDDIE Score	0.947 (0.847–1.060)	.346
GAD-7 Score	1.061 (0.960–1.174)	.246
At least one psychiatric comorbidity	0.910 (0.343–2.414)	.85
vs Group No PPTE		
Age at the onset of epilepsy	0.986 (0.954–1.019)	.386
Febrile seizure	4.890 (0.912–26.213)	.064
Temporal localization	0.588 (0.226–1.530)	.276
NDDIE Score	0.916 (0.808–1.038)	.167
GAD-7 Score	0.980 (0.871–1.103)	.734
At least one psychiatric comorbidity	0.544 (0.215–1.381)	.20

4.2.2. Temporal localization

Another interesting finding from our study was the difference in temporal localization of epilepsy among the groups. Specifically, we observed a significantly higher prevalence of temporal localization in patients who reported having experienced psychological trauma before the onset of epilepsy. Surprisingly, we also found a significantly lower history of febrile seizures in this group compared to others, despite febrile seizures being commonly associated with temporal epilepsy (Cendes et al., 1993). These results also supported by our regression analysis which showed that temporal localization was a significant predictor of increased likelihood, while febrile seizures were a significant predictor of reduced likelihood for the 'before' group. This unexpected result prompts us to consider alternative etiological explanations for temporal localization in this group, beyond febrile seizures. These findings compel us to question the role of trauma in the localization of epilepsy and therefore its potential impact on epileptogenesis. Both human and animal studies have demonstrated the profound effects of early life stress on brain development, leading to structural, functional, and neurobiological alterations in regions associated with the temporal lobe. Stress was shown to be the only risk factor, which increases both incidence, and frequency of epilepsy (Novakova et al., 2013). Chronic stress and trauma exposure have been shown to induce neuroanatomical changes, such as gray matter reduction in the anterior cingulate cortex, ventromedial prefrontal cortex, left temporal pole/middle temporal gyrus, and left hippocampus in individuals with PTSD compared to those without (Kühn & Gallinat, 2013). Moreover, individuals experiencing chronic stress exhibit modifications in the hippocampus and amygdala (Vyas et al., 2002). The role of psychological trauma in temporal lobe epilepsy can be examined through the concept of 'psychoepileptogenesis' (Jhaveri et al., 2023; Soncin et al., 2021). A recent prospective multicentric study showed that only populations with a vulnerable predisposition to stress, characterized by low level of brain-derived neurotrophic factor (BDNF), develop higher sensitivity to epileptogenesis (McGonigal et al., 2023).

4.2.3. Childhood traumas

It is crucial to emphasize that patients who reported having experienced traumatic events before the onset of their seizures reported a significantly higher frequency of multiple traumas, an earlier onset of traumatic experiences, and a greater prevalence of childhood traumatic experiences compared to those who reported having experienced trauma after the onset of epilepsy. Specifically, individuals with trauma before the onset of epilepsy reported significantly more instances of sexual and physical trauma, as well as significantly higher emotional, physical, and sexual abuse scores on the CTQ-28. Some studies have indicated that exposure to traumatic experiences during childhood is associated with an increased risk of experiencing multiple types of traumatic events (Park et al., 2014). It is conceivable that the severity of traumatic events in this group may be greater. Also, prolonged activation of stress hormones during early stages of life, such as exposure to repeated traumatic events, can lead to more pronounced changes in brain plasticity (Kessler et al., 2010; Murphy et al., 2022). This prolonged stress during early life may result in more permanent alterations in brain morphology and the HPA axis (van Campen et al., 2014), potentially increasing the risk of epileptogenesis and rendering the brain more susceptible to developing seizures (van Campen et al., 2014). While these findings require replication, they suggest the possibility that early traumas could heighten the risk of developing temporal lobe epilepsy specifically.

A review of the literature has highlighted the different effects of stress across the lifespan (Lupien et al., 2009). Chronic stress impacts the brain, behavior, and cognition differently depending on the timing of exposure: prenatal, childhood, adolescence, adulthood, and aging (Lupien et al., 2009). For instance, exposure to repeated stress during the prenatal period is associated with depression, anxiety symptoms, and learning impairments. Individuals who experienced stress early in life typically exhibit lower levels of glucocorticoids. Changes in brain and cognition caused by chronic stress are more easily reversible when the stress is experienced in adulthood compared to early life. Stress during aging is associated with cognitive problems such as memory impairment (Lupien et al., 2009).

4.3. The time of trauma and psychopathological profile

Regarding psychiatric profiles, our study revealed that patients who had not reported having experienced traumatic events reported significantly lower rates of psychiatric comorbidity and better overall quality of

life compared to those with traumatic experiences. Put simply, individuals who had reported having experienced traumatic events before and after the onset of epilepsy exhibited significantly higher rates of psychological comorbidities compared to those with no history of trauma. Indeed, existing research has consistently demonstrated that individuals exposed to PPTE are more likely to develop psychiatric disorders compared to those with no trauma history (Copeland et al., 2018; Kessler et al., 2010; McLaughlin et al., 2010). It is also noteworthy that the scientific literature has highlighted a bidirectional relationship between psychiatric comorbidities and psychopathological consequences of traumatic experiences (Lockwood & Forbes, 2014). In other words, traumatic experiences have been identified as a risk factor for the development of psychiatric disorders, while psychiatric disorders themselves are recognized as predisposing factors for the development of PTSD (Sayed et al., 2015).

Our findings revealed that the group who reported having experienced traumatic events before the onset of epilepsy exhibited a lower prevalence of anxiety and mood symptoms compared to the group who reported having experienced trauma after the onset of epilepsy. Furthermore, patients in the 'before' group had earlier traumatic experiences, compared to those in the 'after' group (age of trauma; 12.21 vs 20.58 years). Some studies have highlighted the phenomenon of spontaneous remission of posttraumatic symptoms and their consequences over time (Chapman et al., 2012; Morina et al., 2014). Research indicates that the median time for PTSD remission is around 14 years (Chapman et al., 2012). It is plausible that some patients who experienced traumatic events before the onset of epilepsy may have undergone spontaneous remission of posttraumatic symptoms over time. It is conceivable that individuals who experienced early life events may have had more opportunities to seek help from mental health specialists. Studies have shown that there is often an 11-year delay between the onset of first mental disorder and the initial treatment contact (Wang et al., 2004). In our 'before' group, where PPTE occurred before the onset of epilepsy, patients may have had the opportunity to benefit from psychological treatment for a longer duration. It is also important to highlight that PWE frequently report cognitive impairments, such as deficits in memory, attention, and executive functions (Khalife et al., 2022; Novak et al., 2022). There are several factors that can influence cognition in PWE. Early onset of epilepsy, seizure characteristics such as temporal localization and drug resistance, frequency of seizures, and treatments are some of those factors. On the other hand, PTSD is also associated with reduced cognitive functioning in memory, attention, executive function, and emotional regulation difficulties (Quinones et al., 2020). Our results showed that those who reported trauma after the onset of seizure had significantly earlier onset of epilepsy compared to those who reported trauma before the onset of epilepsy. Whereas before group had significantly earlier trauma compared to the after group. These are important age differences which might possibly influence some neurobiological and cognitive aspects.

Our study also identified a significantly higher prevalence of post-ictal anxiety and mood symptoms in patients who reported having experienced psychological trauma after the onset of epilepsy. Previous research has indicated that individuals with depression and anxiety are more likely to experience post-ictal psychiatric disorders, and the severity of these disorders tends to worsen during the post-ictal period (Kanner et al., 2004). This suggests a possible bi-directional link between peri-ictal and interictal psychiatric disorders. Moreover, patients in the 'after' group reported significantly higher incidence of falls during their seizures. It is plausible that the physiological consequences of seizures may be more severe in this group, thereby increasing the likelihood of experiencing distress during post ictal period. These findings underscore the complex interplay between seizure characteristics, psychological trauma, and post-ictal psychiatric symptoms, highlighting the importance of comprehensive assessment and management of psychiatric comorbidities in PWE.

5. Limitations

Our study has several limitations that warrant consideration. Firstly, it is a cross-sectional study without prospective follow-up, which makes it challenging to establish a causal relationship between trauma and epilepsy. Additionally, there may be a potential recall bias regarding the traumatic event data reported by our patients. Therefore, caution must be exercised when interpreting our results.

In our study, we collected data on trauma exposure using self-reported measures. A recent study emphasized that the association between exposure to early adversity and the presence of mental disorders later in life may be more related to subjective reporting of trauma than to objective exposure (Danese & Widom, 2023). The authors evaluated childhood trauma exposure using a self-questionnaire and corroborated participants' claims of traumatic events with reports from objective evidence provided by authorities. In our study, it is possible that epilepsy, by altering cognitive processing, may influence the cognitive evaluation of the environment and amplify the subjective reporting of trauma. Furthermore, our study sample comprised patients referred to specific epileptology clinics, and the majority of them had focal drug-resistant epilepsy. As a result, our findings

may not be generalizable to all PWE, particularly those with different seizure types or treatment responses. Furthermore, while our study shows a high prevalence of temporal localization in patients reported having experienced psychological trauma before the onset of their seizures, it is important to interpret these findings with caution due to the observational nature of the study, the *p*-value being close to .05, and the weak measure of association (v = 0.188). Future research should explore potential confounding variables that could be involved in this relationship between psychological trauma and temporal epilepsy.

Studies showed that women are at higher risk of developing PTSD than men and experience different types of trauma, the former being more exposed to sexual trauma. Moreover, traumatic stress affects different areas of the brains of boys and girls at different ages (Olff, 2017). In this current study, we did not investigate the sex differences. It would be interesting to further explore the male/female predominance depending on the epilepsy syndrome and the psychotraumatic factors.

These limitations highlight the need for further longitudinal studies with larger and more diverse patient populations to validate our findings and explore the complex relationship between trauma and epilepsy more comprehensively. Additionally, employing standardized measures to assess traumatic experiences and accounting for potential confounding variables could enhance the robustness of future research in this area.

6. Conclusion

Our study underscores the significant neurological and psychiatric ramifications of traumatic events in PWE. This study also sheds light on the potential impact of the timing of psychological trauma on patients' psychiatric and neurological profiles. This highlights the importance of systematically assessing psychiatric and psychotraumatic comorbidities in PWE to ensure comprehensive care. Our findings emphasize the necessity of a multidisciplinary approach in the management of epilepsy and its associated comorbidities. By integrating expertise from various disciplines, including neurology, psychiatry, and psychology, we can better address the complex needs of individuals with epilepsy and optimize their overall well-being. Furthermore, our study underscores the importance of further investigating the relationship between psychological trauma and temporal epilepsy. By gaining a deeper understanding of this relationship, we can develop more targeted interventions and support strategies to mitigate the impact of trauma on epilepsy outcomes.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The data that support findings of this study are available on request from the corresponding author (CH). The data are not publicly available due their containing information that could compromise the privacy of research participants.

ORCID

Coraline Hingray http://orcid.org/0000-0002-7665-3310

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