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# Post-traumatic epilepsy in Cameroon: a retrospective study in a referral hospital

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## Abstract

**Background:** Post-traumatic epilepsy is defined as the onset of at least one seizure beyond the first week following a traumatic brain injury (TBI). High prevalence of TBI in our setting may contribute to the burden of epilepsy in adult population. This is a retrospective review of medical records of patients admitted from January 1st, 2010 to December 31st, 2019) at Douala General Hospital. We included patients aged  $\geq 18$  years with seizure onset at least one week after TBI. Incomplete files and previously known epilepsy were excluded. Data on sociodemography, clinical and para-clinical features, treatment and outcome were analysed using R software version 36.2.

**Results:** We finally included 65 patients with post-traumatic epilepsy among 993 medical records of epilepsy. The mean age was  $35.1 \pm 12.6$  years, with 64.6% of male. Road traffic accident was the main aetiology of brain trauma (78.5%), resulting in haemorrhagic contusions (21.5%), sub-dural haematoma (15.4%), and diffuse axonal lesions (15.4%) mainly. Seizure onset was within 2 years post-trauma in 73.8% of cases. Generalized tonic-clonic seizures were the commonest seizure's type. Electroencephalogram was abnormal in 81%, including 47% of focal discharges. Antiepileptic drugs were mainly sodium valproate, carbamazepine, and phenobarbital. Seizure freedom was obtained in 67.7% of cases.

**Conclusions:** Post-traumatic epilepsy is a heterogeneous, frequent and often disabling complication of traumatic brain injury. Road traffic accident is the main cause of brain trauma. It affects a young and active population. About half of cases presented GTCS. With antiepileptic drugs, more than two-thirds of patients become seizure-free.

**Keywords:** Post-traumatic epilepsy, Traumatic brain injury, Road traffic accident, Cameroon

## Background

The International League Against Epilepsy (ILAE) defines epilepsy as a chronic neurological disease characterized by the occurrence of at least two unprovoked or reflex epileptic seizures (ES) spaced more than 24 h apart, or one unprovoked seizure with a high probability ( $\geq 60\%$ ) of occurrence of another ES in the next ten years or the existence of an epileptic syndrome [1]. Traumatic brain

injury (TBI) is a physical shock on the skull, which may lead to lesions of the brain [2]. Post-traumatic epilepsy (PTE) is defined as the occurrence of at least one ES beyond the first week following a TBI. Early ES (immediate  $< 24$  h or delayed  $< 1$  week) are acute symptomatic events corresponding to the brain's response to the physical effects of TBI [3].

Incidence of PTE ranges from 30 to 40% in penetrating TBI versus 2% to 7% in the absence of penetrating trauma [4, 5]. Risk factors for PTE included Glasgow Coma Scale (GCS)  $< 10$ , early seizure, displaced skull fracture, focal neurological deficit, cortico-subcortical lesion on neuroimaging, and duration of coma  $> 5$  days [6–9]. Anti-seizure medications (ASMs) are widely used in PTE.

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However, in case of intolerance or resistance to ASMs, surgical treatment may help to reduce seizure frequency [10].

In Cameroon, TBI mainly affects young and active people [11]. Intracranial lesions such as sub-dural haematoma (SDH), epidural haematoma (EDH), diffuse axonal injury (DAI), contusions, craniocerebral wounds, increase the burden of PTE in adult population. Patients with PTE suffer double the consequence of physical trauma and psychological trauma associated with significant stigma associated with epilepsy in Cameroon. In this study, we aimed to determine characteristics of post-traumatic epilepsy (PTE) in a referral hospital of Cameroon.

**Materials and methods**

**Study design**

We retrospectively reviewed medical records of patients admitted or consulted from January 1st, 2010 to December 31st, 2019 for epilepsy in the neurology and neurosurgery units of the Douala General hospital (DGH). We included all adult cases ( $\geq 18$  years old) of post-traumatic epilepsy defined as onset of epileptic seizure at least one week after a traumatic brain injury (TBI). Incomplete file and previously known patients with epilepsy were excluded from this study. A file was considered incomplete when missing at least two of the following data (TBI mechanism, time to onset of the first seizure, neuroimaging of the trauma, type of ES).

**Data collection**

We carefully reviewed patients’ data in archives, outpatient registers, and through call. Data collected included: socio-demographic, comorbidities and risk factors of seizures, clinical description of seizures, neuroimaging and EEG, treatment and outcome.

**Statistical analysis**

We carried out a descriptive analysis of the variables using R software version 36.2. Categorical variables were expressed as frequencies with percentage. Continuous variables were presented as mean with standard deviation (SD). The Chi-square test was used to look for associations between our variables. The results were considered significant for a *p*-value  $< 0.05$ .

**Results**

Among the 993 medical records of patients with epilepsy, we found 81 cases with post-traumatic epilepsy (PTE). We finally included 65 patients (we excluded 5 incomplete files and 11 patients aged less than 18 years old). Male represented 64.6% of this population, the mean age (SD) was  $35.1 \pm 12.6$  years old, and 18 to 28 years was the

main age group (Table 1). The history of traumatic brain injury (TBI) was relevant for initial loss of consciousness (70%), early seizures (27.7%). In this study, road traffic accident (RTA) was the TBI aetiology in 78.5% of cases. TBI was classified as severe (GCS  $< 8$ ) in 61.5% of patients (Table 2). The frequency of seizures onset decreased over time: 43% during the first year to 7.7% between 6 and 10 years after the trauma. Generalized tonic-clonic seizures (GTCS) were the commonest seizure’s type. 50.8% of patients had a biannual seizure frequency (Table 3). The brain imaging was performed either at the time of TBI ( $n = 51$ ) or after the diagnosis of PTE ( $n = 14$ ). The commonest lesions at the time of TBI were: haemorrhagic contusion (21.5%), diffuse axonal injury (15.4%), and sub-dural haematoma (15.4%); see Table 4. EEG was done in all cases resulting in abnormal findings in 81.5%. Anti-seizure medications (ASMs) were prescribed in all patients. Sodium valproate and carbamazepine were the frequent ASMs used. 13.8% of cases received a combination of two ASMs. No patient underwent surgical procedure for epilepsy management. Seizure frequencies drop after initiation of ASMs, with 67.7% of seizure freedom (Table 5). The mean duration time for seizure freedom was  $2.5 \pm 1.2$  years. No patient died in this population.

**Discussion**

This retrospective study conducted in a referral hospital in Cameroon, aimed to describe different features of post-traumatic epilepsy (PTE) for the first time.

**Table 1** General characteristics of patients

Variable	<i>n</i>	%
Gender		
Male	42	64.6
Female	23	35.4
Age groups		
18–28	26	40
28–38	13	20
38–48	14	21.5
48–58	8	12.3
> 58	4	6.4
Job		
Students	22	33.8
Taxi-bike riders	16	24.6
Office employees	11	19.9
Informal workers	10	15.9
Housewives	5	7.7
Alcohol consumption	26	40
Family history of epilepsy	6	9.2

**Table 2** Features of the traumatic brain injury leading to epilepsy

Variables	n	%
Mechanism of injury		
Road traffic accidents	51	78.5
Domestic accidents	9	13.8
Assaults	5	7.7
Initial loss of consciousness	46	70.8
Early seizures	18	27.7
GCS* on admission		
< 8 (severe TBI†)	40	61.5
8–12 (moderate TBI†)	11	16.9
> 12 (mild TBI†)	14	21.5
Focal neurological signs		
Motor deficit	10	15.4
Amnesia	7	10.8
Aphasia	4	6.2
Brain lesions		
Haemorrhagic contusions	14	21.5
Diffuse axonal injury	10	15.4
Sub-dural haematoma	10	15.4
Depressed skull fractures	4	6.2
Penetrating cranioencephalic injury	2	3.1
Cerebral oedema	4	6.2
Sub-arachnoid haemorrhage	1	1.5

\* Glasgow Coma Scale; †traumatic brain injury

**Table 3** Description of seizures in patients

Variables	n	%
Time to seizure onset (months)		
≤ 3	10	15.4
4–6	14	21.5
7–12	4	6.1
13–24	20	30.8
25–60	12	18.5
61–120	5	7.7
Seizure's type		
GTCS*	31	47.6
Focal	22	33.8
FwSG†	12	18.5
Seizure's frequency		
Biannual	33	50.8
Monthly	28	43.1
Weekly	4	6.2

\* Generalized tonic-clonic seizures; †focal seizure with secondary generalization

**Table 4** Neuroimaging findings after the traumatic brain injury and after the onset of post-traumatic epilepsy

Variables	N = 65	(%)
At the time of trauma		
Haemorrhagic contusion	14	21.5
Diffuse axonal injury	10	15.4
Sub-dural haematoma	10	15.4
Depressed skull fracture	4	6.2
Cerebral oedema	4	6.2
Penetrating cranioencephalic injury	2	3.1
Sub-arachnoid haemorrhage	1	1.5
Normal	9	13.8
After epilepsy onset		
Gliososis	4	6.2
Sequelae of cerebral infarction	3	4.6
Cortical atrophy	1	1.5
Porencephalic cyst	1	1.5
Non-specific hyperintensities	1	1.5
Normal	4	6.2
Site of lesions		
Frontal	25	38.5
Parietal	12	18.5
Temporal	7	10.8
Occipital	2	3.1

**Table 5** Anti-seizure medication and seizure outcome

Variables	n	%
Post-traumatic epilepsy prevention		
Phenobarbital	2	3.1
Antiepileptic drugs		
Monotherapy	56	86.2
Sodium valproate	32	49.2
Carbamazepine	15	23.1
Phenobarbital	9	13.8
Bitherapy	9	13.8
Sodium valproate + phenobarbital	2	3.1
Sodium valproate + clonazepam	2	3.1
Carbamazepine + phenobarbital	2	3.1
Carbamazepine + clonazepam	2	3.1
Sodium valproate + carbamazepine	1	1.5
Seizure frequency after ASMs* initiation		
Biannual	2	3.1
Annual	19	29.2
Seizure freedom	44	67.7

\* Anti-seizure medications

In about 6% of patients with epilepsy, traumatic brain injury (TBI) is thought to be the cause [12]. In this study, patients with PTE represented 8.1% of the patients

followed up for epilepsy in our institution. The male predominance found in this study is consistent with the findings of Ogunrin et al. in Nigeria and Zhao et al. in China

[13, 14]. This concordance of results could be explained by the fact that men are more active and carry out more risky activities exposing them to TBI. Two patients out of five were within the 18 to 28 age group. These results are similar to those found in China by Zhao et al. with a mean age of 40.07 years and the most represented age group varying between 20 and 29 years [14]. Similar findings were reported by Ogunrin et al. found also a mean age of 38.6 years this agreement of the results is explained by the fact that the young population is more active and consequently more at risk of undergoing TBI [13]. In Cameroon, young people are mainly involved in motorcycle taxi with very few regulations. This contributes to the burden of trauma in general, and TBI in particular.

In this study, road traffic accidents (RTA) were the TBI mechanism in more than three-quarters of cases. This is consistent with other studies which reported RTA as the main mechanism of TBI in different countries [15–17]. TBI was classified as severe in close to two-thirds of cases. Severe TBI has been found to be associated with PTE [18]. Similar findings were also reported by other studies [15, 19]. The time to the first seizure, which corresponds to the duration of epileptogenesis, varies depending on the individual. In this study, more than two patients out of five underwent their first seizures within one year following the TBI, and close to three-quarters of cases within the first 2 years. These results are similar to other studies who reported 80% of patients with first seizure during the first 2 years following the TBI [13, 15, 20, 21].

In this study, close to half of cases presented a generalized tonic–clonic seizures (GTCS). Other seizures types included focal seizures, and focal seizures with secondary generalization. Similar results were reported by Haltiner et al. with 51% of GCTS, 33% of focal seizures, and 17% focal seizures with secondary generalization [22]. Englander et al. found a higher frequency of GTCS [23]. However, our findings were different from the results of da Silva et al. who described 54.6% of focal seizure, and 37.6% of GTCS in a military population [24]. The focal of seizure could be overshadowed by the spectacular presentation of GTCS. Families of patients tend to report secondary generalization more frequently. In addition, the mechanisms of TBI in the military population are different from RTA.

Neuroimaging (brain CT scan or MRI) allows after a TBI to visualize the type and extent of brain damage. After epileptogenesis, neuroimaging contributes to rule out other causes, and to demonstrate TBI sequelae. In this study, the commonest traumatic lesions were brain contusions, diffuse axonal injuries (DAI), and epidural haematoma (EDH). Studies conducted in Africa reported mainly depressed skull fracture, and EDH [13, 15]. In

these latest studies depressed skull fracture were associated with contusions. In any type of epilepsy, EEG is very useful not only for diagnosis, but also the monitoring of patients. This was demonstrated in this study where we found abnormal EEG findings in more than four patients out of five (81.5% of cases). These EEG abnormalities were: generalized spikes-and-waves discharges (41.6%), frontal spikes-and-waves discharges (30.8%), temporal spikes-and-waves discharges (6.2%), and focal slow wave activity (3%).

The choice of ASMs in PTE depends on the availability, seizure type, and tolerance and drugs interactions [25, 26]. In a resource limited setting like Cameroon, the panel of ASMs available is narrow. Based on their availability, commonest ASMs are sodium valproate, carbamazepine, phenobarbital, and diazepam. More than half of patients were on monotherapy with either sodium valproate, carbamazepine, or phenobarbital. For Ogunrin et al. patients received mainly phenytoin, carbamazepine, and sodium valproate as molecules [13]. Right after a moderate to severe TBI, patients can be placed on ASMs in order to prevent the progression of epileptogenesis to late seizures and epilepsy [27]. Phenytoin is the ASMs with most evidence in seizure prevention [28]. In this study, two patients received seizure prevention medication with phenobarbital. Phenytoin is almost not available in our setting. The use of ASMs in prophylaxis is still controversial. According to Pingue et al., the use of ASMs was associated with a poor rehabilitation outcome, independently of the onset of epilepsy during treatment [29].

The use of ASMs in this study showed some benefits with more than two-thirds of cases with seizures freedom after a mean treatment duration of 2.5 years. For other patients, there was a reduction in seizure frequency. In Nigeria, Ogunrin et al. found 87% of patient's seizures free after 3 months of ASMs and 100% after 6 months [13]. In this latest study, phenytoin was frequently prescribed. This demonstrates the effectiveness of phenytoin in seizure control in patients with PTE. When ASMs do not significantly reduce seizures, epilepsy surgery and vagus nerve stimulation may improve seizures control [16, 30]. In this study, no patients underwent surgery, which is not available in Cameroon.

## Conclusion

In Cameroon, post-traumatic epilepsy affects mainly young adults of the male gender. Road traffic accident are by the far, the leading mechanism of injury, leading to severe traumatic brain injury in about two-thirds of cases. Contusions and epidural haematoma were the commonest brain lesions on imaging. Generalized tonic–clonic seizures are the most frequent clinical seizure's type. Despite the limited choice in antiseizure medications, we achieve a seizure

freedom in about two-thirds of patients after 2.5 years of follow-up. Further studies need to be done to assess the long-term outcome and other clinical features of post-traumatic epilepsy, such as neuropsychological consequence and dissociative seizures.

#### Abbreviations

ILAE: International League Against Epilepsy; ES: Epileptic seizures; TBI: Traumatic brain injury; PTE: Post-traumatic epilepsy; GCS: Glasgow Coma Scale; ASM: Anti-seizure medications; SDH: Sub-dural haematoma; EDH: Epidural haematoma; DAI: Diffuse axonal injury; DGH: Douala General Hospital; EEG: Electroencephalogram; SD: Standard deviation; RTA: Road traffic accident; GTCS: Generalized tonic-clonic seizures.

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#### Author contributions

All persons listed as authors in the manuscript have made substantial contribution. MM: conceptualization, study design, statistical analysis, writing—original draft, writing—review and editing. DGM: conceptualization, study design, statistical analysis, writing—original draft, writing—review and editing. FAS: study design, data collection, statistical analysis, writing—original draft, discussion. MAM, EGBL, CN, JD, IE, VVS: writing—review and editing. VCE: conception, study design, writing—review and editing. YNM: conception, study design, writing—review and editing, supervision. All authors read and approved the final manuscript.

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#### Availability of data and materials

Data can be made available upon reasonable request.

#### Declarations

##### Ethics approval and consent to participate

This study was submitted and approved by the Institutional Ethical Committee for human research of University of Douala (N° 2166 CEI-UDo/07/2020/T) signed on the 1<sup>st</sup> July, 2020.

##### Consent for publication

All authors were informed and gave their consent for the submission of this paper.

##### Competing interests

The authors have no conflicts of interest to declare.

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#### References

- Fisher RS, Acevedo C, Arzimanoglou A, Bogacz A, Cross JH, Elger CE, et al. ILAE official report: a practical clinical definition of epilepsy. *Epilepsia*. 2014;55:475–82.
- Larousse É. Archive Larousse : Larousse Médical—traumatisme crânien [en ligne]. [Consulté le 12 déc. 2019]. Disponible sur: <http://www.larousse.fr/archives/medical/page/1023>.
- Lowenstein DH. Epilepsy after head injury: an overview. *Epilepsia*. 2009;50:4–9.
- Weiss GH, Feeney DM, Caveness WF, Dillon LD, Kistler JP, Mohr JP, Rish BL. Prognostic factors for the occurrence of posttraumatic epilepsy. *Arch Neurol*. 1983;40:7–10.
- Annegers JF, Grabow JD, Groover RV, Laws ER, Elveback LR, Kurland LT. Seizures after head trauma: a population study. *Neurology*. 1980;30:683–683.
- Toussaint-Thorin M, Watier L, Laurent-Vannier A, Bourgeois M, Meyer P, Chevignard M. Incidence et facteurs de risque de survenue d'une épilepsie dans les suites d'un traumatisme crânien sévère chez l'enfant : résultats d'une étude prospective (cohorte TGE). *Ann Phys Rehabil Med*. 2013;56: e297.
- Asikainen I, Kaste M, Sarna S. Early and late posttraumatic seizures in traumatic brain injury rehabilitation patients: brain injury factors causing late seizures and influence of seizures on long-term outcome. *Epilepsia*. 1999;40:584–9.
- Arrouf L, Arrouf C. Facteurs de risque d'épilepsie post-traumatique. *Neurochirurgie*. 2010;56:533.
- Baulac M. Épilepsie post-traumatique : aspects cliniques, facteurs de risques et essais thérapeutiques. *Revue Neurologique*. 2012;168:A176–7.
- Zimmermann LL, Martin RM, Girgis F. Treatment options for posttraumatic epilepsy. *Curr Opin Neurol*. 2017;30:580–6.
- Motah M, Sende charlotte N, Beyiha G, Priso EB, Nguemgne CM, Fotsin JG. Prise en charge des traumatismes crâniens isolés à l'hôpital général de douala. *health sciences and diseases [en ligne]*. 2013 [consulté le 18 déc 2019];12(3). Disponible sur: <https://www.hsd-fmsb.org/index.php/hsd/article/view/122>.
- Temkin NR. Preventing and treating posttraumatic seizures: the human experience. *Epilepsia*. 2009;50(Suppl 2):10–3. <https://doi.org/10.1111/j.1528-1167.2008.02005.x>.
- Ogunrin O, Adeyekun A. Profile of post-traumatic epilepsy in Benin City, Nigeria. *West Afr J Med*. 2011;29(3):153–7.
- Zhao Y, Wu H, Wang X, Li J, Zhang S. Clinical epidemiology of posttraumatic epilepsy in a group of Chinese patients. *Seizure*. 2012;21:322–6.
- Essanhaji A, Kissani N, Kadiri B. Épilepsie post traumatique sur traumatismes crâniens graves, analyse rétrospective d'une série de 40 cas à Marakech Maroc. *Afr Middle East Epilepsy J [en ligne]*. 2015 [consulté le 12 déc 2019];4(1). Disponible sur: <https://revues.imist.ma/index.php?journal=AMEEJ&page=article&op=view&path%5B%5D=3684>.
- Gupta PK, Sayed N, Ding K, Agostini MA, Van Ness PC, Yablon S, Madden C, Mickey B, D'Ambrosio R, Diaz-Arrastia R. Subtypes of post-traumatic epilepsy: clinical, electrophysiological, and imaging features. *J Neurotrauma*. 2014;31:1439–43.
- Richard I, François C, Louis F, de la Grève IM, Perrouin-Verbe B, Mathé J. Épilepsie post-traumatique: analyse rétrospective d'une série de 90 traumatismes crâniens graves. *Ann Readapt Med Phys*. 1998;41:409–15.
- Xu T, Yu X, Ou S, Liu X, Yuan J, Huang H, et al. Risk factors for posttraumatic epilepsy: a systematic review and meta-analysis. *Epilepsy Behav*. 2017;67:1–6.
- Ferguson PL, Smith GM, Wannamaker BB, Thurman DJ, Pickelsimer EE, Selassie AW. A population-based study of risk of epilepsy after hospitalization for traumatic brain injury: risk of posttraumatic epilepsy. *Epilepsia*. 2010;51(5):891–8.
- Courjon J. A longitudinal electro-clinical study of 80 cases of post-traumatic epilepsy observed from the time of the original trauma. *Epilepsia*. 1970;11:29–36.
- Caveness WF, Meirinsky AM, Rish BL, Mohr JP, Kistler JP, Dillon JD, Weiss GH. The nature of posttraumatic epilepsy. *J Neurosurg*. 1979;50(5):545–53.
- Haltiner AM, Temkin NR, Dikmen SS. Risk of seizure recurrence after the first late posttraumatic seizure. *Arch Phys Med Rehabil*. 1997;78(8):835–40.
- Englander J, Bushnik T, Duong TT, Cifu DX, Zafonte R, Wright J, Hughes R, Bergman W. Analyzing risk factors for late posttraumatic seizures:

- a prospective, multicenter investigation. *Arch Phys Med Rehabil.* 2003;84(3):365–73.
24. da Silva AM, Nunes B, Vaz AR, Mendonça D. Posttraumatic Epilepsy in Civilians: Clinical and Electroencephalographic Studies. In: da Silva AM, Melo AR, Loew F, editors. *Neurotraumatology: progress and perspectives.* Acta Neurochirurgica, vol 55. Springer, Vienna; 1992. [https://doi.org/10.1007/978-3-7091-9233-7\\_16](https://doi.org/10.1007/978-3-7091-9233-7_16).
  25. Pritchard PB. (2016). Neuropharmacology of antiepileptic drugs. In Lisak RP, Truong DD, Carroll WM, Bhidayasiri R, editors *International Neurology.* <https://doi.org/10.1002/9781118777329.ch36>
  26. Sills GJ, Rogawski MA. Mechanisms of action of currently used antiseizure drugs. *Neuropharmacology.* 2020;15(168): 107966. <https://doi.org/10.1016/j.neuropharm.2020.107966>.
  27. Szaflarski JP, Nazzari Y, Dreier LE. Post-traumatic epilepsy: current and emerging treatment options. *Neuropsychiatr Dis Treat.* 2014;10:1469–77.
  28. Candy N, Tsimiklis C, Poonnoose S, Trivedi R. The use of antiepileptic medication in early post traumatic seizure prophylaxis at a single institution. *J Clin Neurosci.* 2019;69:198–205. <https://doi.org/10.1016/j.jocn.2019.07.066>.
  29. Pingue V, Mele C, Nardone A. Post traumatic seizures and antiepileptic therapy as predictors of the functional outcome in patients with traumatic brain injury. *Sci Rep.* 2021;11:4708. <https://doi.org/10.1038/s41598-021-84203-y>.
  30. Hakimian S, Kershenovich A, Miller JW, et al. Long-term outcome of extratemporal resection in posttraumatic epilepsy. *Neurosurg Focus.* 2012;32:1–3. <https://doi.org/10.3171/2012.1.FOCUS11329>.

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