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Utility of electroencephalography in toxin-induced seizures

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Abstract

Introduction: Toxin-induced seizures differ from seizures occurring in epilepsy and have a high rate of complications. Electroencephalography (EEG) is routinely obtained when there is concern for nonconvulsive status epilepticus (NCSE). The purpose of this study was to characterize the typical findings after toxin-induced seizures, assess the rate of epileptiform discharges and NCSE, and identify any changes in management resulting from EEG.

Methods: Patients older than 16 years who had an EEG during hospitalization for drug-induced seizure or seizure-like activity were included. We reviewed 10 years of data (2013–2022) across our hospital system (four community hospitals and one academic center). Patients with a history of seizures and those with cardiac arrest prior to EEG were excluded. The primary outcome was incidence of epileptiform discharges on EEG. The secondary outcome was number of antiseizure medications (ASM) added after EEG.

Results: A total of 256 encounters were screened with 83 patient encounters included. A total of 53% (44/83) of EEGs showed some degree of generalized slowing. A total of 2.4% (2/83) of cases had epileptiform activity on EEG. No cases of nonconvulsive status were identified. No ASM was started in the two cases where epileptiform discharges were identified.

Conclusions: During usual care of toxin-induced seizures, epileptiform discharges are uncommon.

KEYWORDS
antiseizure medications, electroencephalography, seizures

INTRODUCTION

Seizures and their potential progression to status epilepticus are a severe complication from medication and illicit drug overdoses. The seizures generated after an overdose in a patient with no history of seizures can be different than those that occur in patients with epilepsy. As opposed to an onset from foci within the central nervous system (CNS) that then generalize, toxin-induced seizures often occur secondary to diffuse CNS excitation. Mechanisms include, but are not limited to, inhibition of GABA signaling, histamine blockade, reuptake inhibition or release of biogenic amines from presynaptic termini, and sodium-channel blockade.[1,2] Drug-induced seizures have a high rate of complications with one study showing prolonged hospitalization, endotracheal intubation, anoxic brain injury, status epilepticus, or death in 60% of patients.[3] When seizures do occur, and especially in cases of suspected generalized convulsive status epilepticus (GCSE), electroencephalography (EEG) is obtained.[4,5] Case reports of epileptogenic overdoses have described abnormal findings that include burst suppression (a pattern of low voltage or no activity intermixed with shorter periods of high-amplitude electrical activity), decreased neuronal reactivity (low voltage on EEG), and increased or generalized theta activity.[6–8] While helpful, this case report data are limited. To our knowledge there is no summary of the typical findings after toxin-induced seizures. Furthermore, it is unclear whether nonconvulsive

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status epilepticus (NCSE) or epileptiform discharges occur in this setting and, if they do, with what frequency. For comparison, it is known that NCSE can occur in 32%–59% of postarrest patients and critically ill patients in the intensive care unit, making EEG an integral diagnostic tool in their care.[4] If a similar incidence of NCSE exists in patients with toxin-induced seizures, an EEG could guide treatment. Both the frequency of any epileptiform discharges and the overall utility of obtaining an EEG in these cases are important to these patient's dispositions and may affect the decision to transfer to a center for continuous EEG (cEEG). In this study we aim to characterize the typical findings after toxin-induced seizures, assess the rate of epileptiform discharges and NCSE, and identify any changes in management resulting from EEG.

METHODS

Study design

We conducted a retrospective, cross-sectional study of EEG findings in patients with toxin-induced seizures or seizure-like activity over 10 years (January 1, 2013–December 31, 2022). The study was approved by our institutional review board with a waiver of consent.

Study setting

Multicenter study in one health system encompassing four community hospitals and one academic hospital. The system contains 1952 licensed beds.

Study participants

We included patients aged 16–80 years with a seizure secondary to drug toxicity or overdose. We screened patients using the SlicerDicer feature of EPIC (EPIC Systems Corporation) including the general concepts of "toxicity," "poisoning," and/or "overdose" (and any adjacent terms) with an EEG obtained on the same visit. Using the SlicerDicer feature, the entire patient record was searched for these key terms. Only patients with a seizure occurring in the setting of an acute overdose or drug toxicity were included. A medical toxicologist reviewed charts for inclusion. We screened all notes and the indication listed in the EEG for mention of seizure, status epilepticus, or seizure-like activity. Patients were excluded if there was no documented seizure or seizure-like activity. Additional exclusion criteria included patients with an EEG obtained after cardiac arrest, those with acute CNS pathology (subarachnoid hemorrhage, ischemic stroke, etc.), and those with a history of epilepsy.

Data abstraction and measurements

Data were extracted directly from the electronic medical record (EMR) using a standardized spreadsheet created during institutional review board submission. Initial data abstractors were not blinded to the study protocol. Abstraction of the following variables was performed separately from abstraction of EEG results. Data included patient demographics (patient age, gender, and race), laboratory results on presentation (glucose, creatinine, potassium, magnesium, bicarbonate, and lactate), and description of seizure and overdose (drug ingested, number of seizures, sedative medications used prior to EEG, and antiepileptics administered prior to EEG). We categorized seizures as being single, multiple, status epilepticus (if documented as such in the patient record), and partial (if seizure-like activity reported, but no loss of consciousness or generalized tonic–clonic activity described). We identified causative agents from triage, emergency department (ED), and admitting notes. If agent was unknown, then we recorded it as undetermined. Presentations after illicit drug ingestions were recorded as undetermined illicit ingestions with/without cocaine (charted as with cocaine if enzyme-multiplied drug immunoassay was positive for cocaine given this test's high specificity).

EEG reports in the EMR were used to determine whether a study was positive or negative depending on whether epileptiform discharges were identified. This was done for all screened patients. All EEGs were interpretated by a board-certified neurologist during admission and were not reinterpreted during the study. Both routine EEGs (recordings typically performed for 30 min) and cEEGs were included in the study. cEEGs in our health system have a technologist monitoring the tracing and a physician on call to read any changes in EEG waveform. We reviewed all cEEG reports during hospitalization to determine whether epileptiform discharges were present. A second reviewer blinded to the study objectives reviewed 20% of the documented EEG results in the EMR to verify data abstraction. If there was a discrepancy, reviewers reexamined the case and came to a final determination as to the EEG result. After subjects were found to meet inclusion criteria, EEG findings were further categorized. The clinical description of events during EEG recording was used to determine whether NCSE was present. For negative studies, we placed patients into three groups: normal, generalized slowing, or other (burst suppression, generalized rhythmic delta activity, cortical irritability/sharp waves, or other). For the secondary outcome, we recorded addition of any antiseizure medications (ASM) after EEG started as a proxy for change in management.

Outcomes

The primary outcome was incidence of epileptiform discharges on EEG. The secondary outcome was frequency and number of ASM added after EEG was obtained.

Data analysis

Given that there are no available prior studies for estimating incidence of epileptiform activity in our population of interest, we reported primarily descriptive outcomes (means and confidence intervals [CIs] or medians and interquartile ranges [IQRs] depending on the normality of data; *n* and percentage for categorical variables).

Listwise deletion was used for missing data. Inter-rater reliability was calculated based on raw agreement. A chi-square test was used to compare the rate of positive EEG in patients in the study to a larger cohort without excluding for lack of seizure (including patients for whom EEG was obtained due to toxic encephalopathy or coma).

RESULTS

Our initial screen of patients identified 256 potential cases with 83 patient encounters ultimately meeting inclusion criteria (Figure 1). There were 81 patients included, two of whom had repeat presentations. Generalized slowing was seen in 53% (44/83) of EEGs. Epileptiform activity was identified in 2.4% (2/83) of cases. No cases of NCSE were identified. No ASM was started in the two cases where epileptiform discharges were identified. Inter-rater reliability on review of EEG reports was 94%.

Laboratory values on presentation are included in Table 1. No patient had a glucose of <60 mg/dL or a sodium of <130 mmol/L on presentation. Most patients (60%) were managed at the academic medical center with the remainder at a community hospital. Eight patients were transferred with six (7.2%) transferred for cEEG and two (2.4%) for pediatric admission. The most common xenobiotics implicated were bupropion ($n=16$; 7/16 bupropion alone), diphenhydramine (9; 7/9 diphenhydramine alone), cocaine (7), fluoxetine (6), and citalopram/escitalopram (6; Table 2). Fifty-one patients (63%) were intubated at the time of EEG (Table 3). None were found to have nonconvulsive status or any persistent epileptiform activity on EEG. Two patients had brief seizure activity on initial reading from cEEG, which resolved early in the course of the recording. Fifty-three patients (62%) had an abnormal EEG, the majority of which showed generalized slowing of various degrees (91%; 48/53). Two patients had an EEG showing burst suppression and one had brief focal sharp waves. Four patients were started on a new ASM after EEG (Table 4). Two had a normal EEG and the other two had an EEG demonstrating generalized slowing. The rationale for adding an agent after EEG was unclear in three cases and in the other to treat both mood and prevent further seizures with valproic acid started.

Fifteen patients were diagnosed with clinical status epilepticus prior to EEG. EEG was obtained after intubation and initiation of continuous sedation in all cases. Thirteen (87%; 13/15) had cEEG performed. There was one case (6.7%; 1/15) of epileptiform discharges on EEG (Table 4). Four cases (27%; 4/15) of GCSE had no ASM given prior to EEG. No epileptiform discharges were identified in these cases.

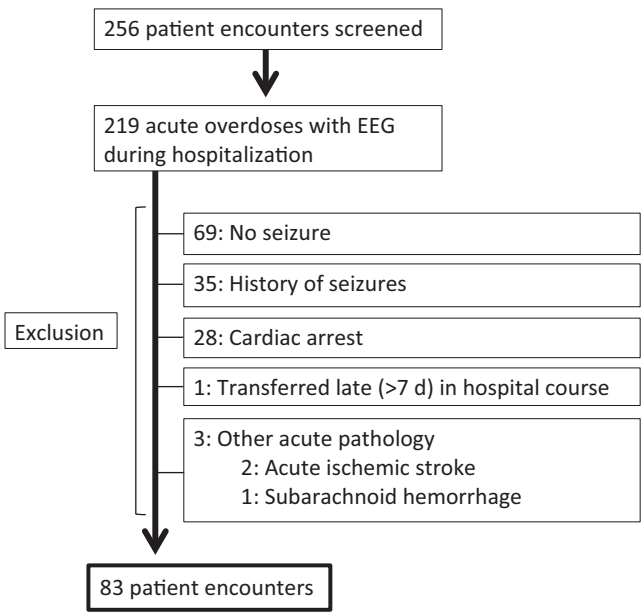

FIGURE 1 Diagram showing the count of initial patients screened, those who were excluded from the study, and then final amount included. EEG, electroencephalography.

DISCUSSION

We found that when an EEG is ordered after a suspected toxin-induced seizure, generalized slowing is most often seen, and seizure activity is rare. It occurs much less frequently than that seen in trials of encephalopathic patients in the intensive care unit.[4] A seizure secondary to an acute overdose or drug toxicity will either be preceded by or occur in the context of neuroexcitatory symptoms and, if present, often be self-limited. However, with certain ingestions, status epilepticus can occur. Apart from camphor, for example, rarely do toxin-induced seizures occur in the absence of overt neurotoxicity, toxidrome, or altered sensorium.[1,9] Treatment of the toxidrome mirrors what would be appropriate for treatment of seizures, namely $GABA_A$ agonists, and thus control of neuroexcitatory symptoms would likewise treat seizure. Since treatment is focused on controlling the underlying neuroexcitation until seizures and associated psychomotor agitation stop, it is unclear whether any EEG finding beyond NCSE would change the management of toxin-induced seizures. Performing a spot or cEEG requires a considerable commitment of provider time, can prolong inpatient hospital stays, and may require patient transfer between facilities.

TABLE 1 Laboratory results obtained on presentation to the hospital.

	Median/means ($n=83$)	95% CI/IQR
Glucose (70–100 mg/dL)[a]	115 ($n=83$)	96–154
Sodium (136–145 mmol/L)[b]	140 ($n=82$)	139–141
Creatinine (0.6–1.2 mg/dL)[b]	1.0 ($n=82$)	0.8–1.2
Bicarbonate (95–110 mEq/L)[b]	20 ($n=82$)	18–22
Potassium (3.4–4.7 meq/L)[b]	4.1 ($n=82$)	3.9–4.3
Magnesium (1.3–2.2 mg/dL)[b]	2.1 ($n=53$)	2.0–2.2
Lactate (0.5–2.2 mmol/L)[a]	3.2 ($n=40$)	1.6–5.4

[a]Medians and IQRs for nonparametric continuous variables.

[b]Means and 95% CIs for parametric data.

TABLE 2 Reported ingestions for each overdose in study.

Single agent	Polysubstance ingestions
Bupropion (7)**	Undetermined (10)*
Diphenhydramine (7)**	+ Ketamine, baclofen, cyclobenzaprine, flurbiprofen, gabapentin, lidocaine
Tramadol (2)	+ Diphenhydramine, zolpidem
Cocaine (2)*	+ Bupropion, dextromethorphan, fluoxetine, trazodone, metaxalone, alcohol*
Ivermectin	+ Cocaine, heroin, alprazolam
Lidocaine*	+ Fluoxetine, trazodone, hydroxyzine
Olanzapine	+ Duloxetine, citalopram, olanzapine
Nortriptyline*	+ Venlafaxine, mixed amphetamine salts, clonazepam
Ibuprofen	+ Oxycodone-acetaminophen, THC
25I-NBOMe*	+ Cocaine, etizolam
Alcohol	+ 1,3-DMAA, caffeine
Dextromethorphan	+ Citalopram, benzonatate
Fluoxetine	+ Citalopram, benzonatate
Baclofen	+ Fluoxetine, trazodone, bupropion
Acetylsalicylate	+ Bupropion, aripiprazole*
Venlafaxine	+ Lamotrigine, clonazepam, risperidone
Doxylamine	+ Acetaminophen, unknown coformulation
Zolpidem	+ Dextromethorphan, THC
2,5-Dimethoxy-4-chloramphetamine	+ Diphenhydramine, ibuprofen
Amphetamine salts	+ Bupropion, acetaminophen
Escitalopram	+ Lamotrigine, methylphenidate, venlafaxine, aripiprazole*†
	+ Tramadol, carisoprodol
	+ Bupropion, diltiazem, hydroxyzine, venlafaxine, cetirizine, hydrochlorothiazide
	+ Fluoxetine, bupropion
	+ Amitriptyline, oxycontin, zolpidem
	+ Undetermined illicit overdose, cocaine
	+ Alprazolam, quetiapine*
	+ Clonazepam, phentermine
	+ Baclofen, gabapentin‡
	+ Bupropion, trazodone‡
	+ Escitalopram, lamotrigine
	+ Lithium, bupropion, fluoxetine, naproxen, hydroxyzine
	+ Bupropion, cocaine*
	+ Escitalopram, bupropion*
	+ Cocaine, phencyclidine

Notes: Table broken into two columns: reported single agent ingestion and those where patients had overdosed on multiple substances or the xenobiotics were not able to be determined. Multiple instances of an overdose are indicated in parentheses (*n*). Specific cases have also been highlighted: *status epilepticus (one for each case), †epileptiform activity on EEG, and ‡cases with burst suppression.

Given the low incidence of epileptiform activity found in our patient population, and the mechanism of drug-induced seizures just discussed, it is unlikely that the routine ordering of EEGs would be beneficial in the management of overdose patients and transfer for cEEG may be unwarranted in many cases. While several studies have examined critically ill populations, prior studies have not focused on this question specifically. A prior study by Yigit et al.[10] had suggested that EEGs provide useful diagnostic information when obtained from the ED, but any abnormality in the EEG was judged as a positive finding and utility was inferred

TABLE 3 Patient demographics, EEG results, timing, and type, and clinical characteristics of seizures and ASM treatment.

Patient characteristics	Total ($N = 83$)
Age (years), mean (95% CI)	33 (29–47)
Sex, M:F (% female/total)	40:43 (52)
Intubated	51 (61)
EEG results	
Positive	
Brief epileptiform discharges	2 (2.4)
Nonconvulsive status	0 (0)
Negative	
Normal	30 (36)
Burst suppression	2 (2.4)
Generalized slowing	48 (58)
Cortical irritability/sharp	1 (1.2)
ASM	
None	36 (43)
Phenytoin	4 (5)
Levetiracetam	40 (48)
Multiple	3 (4)
Seizure or seizure-like activity	
Status epilepticus	15 (18)
Partial/myoclonus	14 (17)
Single	36 (43)
Multiple	18 (22)
EEG characteristics	
cEEG	44 (53)
Obtained in <24 h	53 (64)

Note: Data are reported as *n* (%) unless otherwise specified.
Abbreviations: ASM, antiseizure medications; cEEG, continuous electroencephalography; EEG, electroencephalography.

from a higher proportion of admitted patients having an abnormal EEG. However, assessment of whether a patient was admitted due to their EEG result was not done. Praline et al.[11] showed that physicians self-report a change in management after EEG in 46.6% of cases; however, the primary benefit was in cases of suspected subtle status epilepticus that were not toxin-induced. How management changed was also not demonstrated. We attempted to use an objective finding (addition of ASM) that occurred after EEG as a proxy for a change in management. In our cohort, an initial or additional ASM other than a benzodiazepine or propofol was started in <5% of cases, and in those, the EEG findings did not spur on this change in management. In both cases where seizure activity was seen on cEEG, clinical correlates were present: a back-arching episode with periodic agitation in the first and eye fluttering in the second. The first patient was not on continuous sedation at the time of epileptiform activity and the second was on 10 μg/kg/h propofol. Given clinical seizure activity, a bolus of

TABLE 4 Primary and secondary outcomes per subcategory.

		Seizure category				EEG		EEG within 24 h		Intubated	
Category	Variable	Status epilepticus ($n = 15$)	Multiple generalized ($n = 18$)	Single generalized ($n = 36$)	Partial ($n = 14$)	Continuous ($n = 44$)	Routine ($n = 39$)	Y ($n = 53$)	N ($n = 30$)	Y ($n = 51$)	N ($n = 32$)
EEG results	Positive	1	0	1	0	2	0	2	0	2	0
	Generalized slowing	13	6	18	11	36	12	34	14	38	10
	Normal	1	10	17	2	4	26	14	16	4	22
	Other	0	1	1	1	2	1	3	0	2	0
ASM after EEG	Y	2	36	4	2	2	2	2	2	4	0
	N	0	12	26	1	22	37	51	28	47	32

Note: Number of encounters where EEG findings were identified and where an ASM was started after EEG are listed per subcategory.
Abbreviations: ASM, antiseizure medications; EEG, electroencephalography.

intravenous midazolam was given in the first case and the propofol infusion was increased in the second. These interventions led to resolution of epileptiform discharges on EEG in both.

For patients presenting with a single seizure that were not intubated, EEG was normal with no generalized slowing or other abnormal findings in 71% (15/21) and in those who were not intubated presenting with more than one seizure, but no diagnosis of status epilepticus, EEG was normal in 100% (6/6; Table 4). Although we believe an EEG may not be necessary in most cases of toxin-induced seizures with management instead focused on adequate sedation and control of neuroexcitatory manifestations of toxicity if present, nonintubated patients would be one area where reduction in testing could be focused.

We cannot rule out that treatment with ASMs or continuous sedative infusions helped limit the number of positive tests. Nevertheless, the goal of our study was to assess whether an EEG should be ordered and not whether the EEG would show epileptiform discharges in the absence of treatment. Even in studies where epileptiform discharges were found in over 20% of patients, ASMs were given prior to EEG performance as patients were being treated for status epilepticus.[10] In our study, ASMs had been given prior to EEG in 57% of cases and continuous sedation started in 51%. Levetiracetam was the ASM most often used prior to EEG. A study by Lee et al.[12] suggests a benefit of levetiracetam in drug-provoked seizures. However, nearly half of patients in this study had a history of seizures thus differentiating it from our patient population. Ultimately, our data cannot be used to determine if levetiracetam or other ASM are helpful in treating seizures from drug toxicity.

The rate of positive EEG findings was 1.9% (3/152) if patients who did not have a witnessed seizure (e.g., EEG obtained for persistent encephalopathy or coma after overdose) were added to the patients studied (EEG after seizure or seizure-like activity). This was similar to the rate found in our study (2.4% vs. 1.9%, $p=0.83$). Though we had not planned to address patients without clinically apparent seizures, this finding suggests, but would need to be confirmed with additional investigations, that the rate of epileptiform activity on EEG in any patient after drug toxicity without history of epilepsy is low.

LIMITATIONS

This is a retrospective study and is limited by the accuracy of the information recorded in the EMR. It is also potentially biased by the accuracy of the data abstracted by the reviewers. We attempted to extract only data present in the chart and not to reinterpret the findings. There was a single discordance in EEG results. This initial discordance was due to missed epileptiform activity by the second reviewer on initial report on Day 1 of cEEG. All reports in the cohort were reanalyzed after review and no additional positive cases were found. Though we wanted to describe the spectrum of EEG findings after toxin-induced seizures as well as the potential presence of NCSE, we also wanted to identify if EEGs would change a patient's management and thus did not reinterpret the EEG. We instead used neurologists' interpretation at the time of the study as this is what would impact the management of the patient during hospitalization. We cannot exclude missed epileptiform activity on EEG. It is also possible that the seizure-like activities reported were not actual seizures but rather myoclonus; however, as this was the reason for the utilization of EEG, it was appropriate to include as a reflection of the current clinical practice. The timing of EEGs may have also impacted the results. Earlier studies may be more likely to identify epileptiform activity. In this study, the majority of EEGs were able to be obtained within 24 h though not in the ED. In a system where rapid EEG performance was to be achieved, it is possible the spectrum of finding would be different.

The secondary goal of the study in assessing the benefit of EEGs in patients with toxin-induced seizure is incompletely assessed in a retrospective study and additional prospective and randomized assortment would more definitively answer this question. We also understand that the decision to order an EEG may be beneficial in patients not studied in this cohort. The total number of patients was not large or comprehensive enough to account for all potential overdoses and it is possible that EEG may be useful in cases of xenobiotics not covered in this study. Additionally, patients with an EEG post–cardiac arrest or with a history of seizures were excluded.

CONCLUSIONS

In our patient population, an electroencephalography obtained after a toxin-induced seizure was likely to show generalized slowing and rarely led to the addition of an antiseizure medications. No cases of nonconvulsive status were identified.

AUTHOR CONTRIBUTIONS

Alexander M. Sidlak and Brian Schultz conceived and designed the trial and completed and revised the IRB application. Alexander M. Sidlak and Brent Dibble performed data collection and statistical analysis. Alexander M. Sidlak drafted the manuscript. Alexander M. Sidlak and Brent Dibble revised and reviewed the manuscript. All authors approve of the final publication.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Complete deidentified data sets are available from date of publication to 3 years from that date. Contact Dr. Alex Sidlak at alex.sidlak@inova.org.

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