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FULL-LENGTH ORIGINAL RESEARCH

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Long-term electro-clinical profile of sudden cardiac arrest survivors

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Abstract

Objective: Recent research has explored the use of continuous EEG (cEEG) monitoring for prognostication of spontaneous cardiac arrest (SCA). However, there is limited literature on the long-term (post-hospital discharge) electrographic findings among SCA survivors and their clinical correlates. Our study aims to fill this critical knowledge gap.

Methods: We retrospectively used our EEG database to identify adults (≥18 years) with SCA history who underwent an outpatient laboratory-based EEG between 01/01/2011 and 12/31/2018. After electronic medical records (EMR) review, patients with epilepsy history and unclear/poorly documented SCA history were excluded. Outpatient EEGs were reviewed by authors. Acute EEG findings were extracted from the EEG database and EMR. In addition, we extracted data on acute and long-term neuroimaging findings (CT/MRI), post-SCA seizures, and anti-seizure medications (ASM) status. Descriptive analysis and Fisher's exact test were performed.

Results: We included 32 SCA survivors (50% women; mean age = 52.1 ± 13.6 years) in the study. During a median clinical follow-up of 28.2 months, 3 patients suffered only clinical seizures, 3 only chronic post-hypoxic myoclonus, and 5 had both [11 (34.4%) in total]. Interictal epileptiform discharges (IEDs) were noted in one-third of the patients, which localized to vertex and frontocentral regions in all but one patient. Five (15.6%) of them did not suffer a clinical seizure despite the presence of EAs. Patients who developed epilepsy were significantly more likely to have abnormal neuroimaging findings [10/11 (90.9%)] during the follow-up compared to the rest of the patients [OR = 25 (95% CI 2.6–>100, P = .002)]. Half of the study cohort was taking ASM at the last follow-up.

Significance: Our small study reveals a signature location of IEDs in SCA survivors. Neuroimaging abnormalities seem to be a better indicator of epilepsy development, while EEG may reveal markers of potential epileptogenicity in the absence of clinical seizures. Future, larger studies are needed to confirm our findings.

KEYWORDS
anti-epileptic drugs, cardiac arrest, EEG, epileptogenesis, seizures

1 | INTRODUCTION

Cardiac arrest is a major public health problem. There are 356,000 out-of-hospital spontaneous cardiac arrests (SCA) annually in the United States, nearly 90% of them fatal.[1] In the last decade, a large amount of research has explored the use of continuous EEG (cEEG) monitoring for acute prognostication after SCA.[2,3] It has led to an improved understanding of acute electro-clinical changes after SCA, including the diagnosis of nonconvulsive seizures or status epilepticus (NCS/NCSE) and identifying electrographical patterns associated with poor prognosis.[2,3] However, there is limited literature on the long-term (post-hospital discharge) electrographic findings among SCA survivors and their clinical correlates. Our study aims to fill this critical knowledge gap.

Key Points

- We analyzed the electro-clinical and neuroimaging profile of spontaneous cardiac arrest (SCA) survivors
- One-third (11/32) of patients had epileptiform discharges, which were noted in a signature, vertex, and central region
- One-third (11/32) developed epilepsy during a mean follow-up of 28.2 (±42.3) months, including 3 patients with only chronic post-anoxic myoclonus
- Late neuroimaging abnormalities seem to be better indicator of epilepsy development than EEG findings
- Clinical follow-up with epileptologists helped in deprescribing of anti-seizure medications, when not indicated

2 | METHODS

After IRB approval, we retrospectively used our EEG database to identify adults (≥18 years) with SCA history who underwent an outpatient laboratory-based electroencephalogram (EEG) between 01/01/2011 and 12/31/2018. We reviewed the electronic medical records (EMR) of the identified patients. We excluded patients with a history of epilepsy prior to SCA. The EMR of the remaining patients was reviewed to extract demographical and clinical data, including details about the presence of acute clinical seizures, myoclonus, and anti-seizure medications (ASM) management at the time of SCA. Patients were subsequently excluded if they did not receive acute care in our institution at the time of SCA and their EMR lacked information on a majority of above acute variables. To estimate the percentage of SCA survivors during the study duration who were included in our study, we used our prospectively maintained SCA database to identify patients undergoing cEEG who survived acute hospitalization and were not discharged to hospice.

Information about acute and long-term neuroimaging findings, post-hospital discharge clinical seizures, chronic post-hypoxic myoclonus (cPHM), and ASM status was also extracted. Based on availability in the majority of patients, we chose the first CT head after admission for neuroimaging findings in acute stage and MRI brain post-hospitalization for long-term analysis. Neuroimaging was categorized as abnormal based on the original reading by the neuroradiologist. The cerebral performance category (CPC) score was calculated from the discharge note.[4] The reason for including CPC was to help contextualize the EEG and imaging findings in terms of severity of the anoxic injury during acute hospital admission. Data on the acute EEG within our health system was extracted from our cEEG database, and the raw EEG was reviewed to confirm findings. Acute cEEG is performed as part of the hypothermia protocol in most patients since June 2016 at our institution. For patients whose EEGs were outside our health system, acute EEG findings were extracted from the EMR. Acute cEEG, performed within 7 days of SCA, was classified into one of the following categories based on the predominant finding during the first 24 hours of the recording:

- Suppression pattern: background suppression (<10 μV) or burst suppression
- Epileptiform: seizures (clearly evolving EEG patterns or generalized periodic discharges (GPDs) > 2.5 Hz) or sporadic epileptiform discharges[5] (regional, eg, sharp waves, spikes; lateralized, eg, lateralized periodic discharges; generalized, eg, GPDs b/w 1.5-2.5 Hz[6])
- Continuous: non-suppressed background, non-epileptic EEG findings (excluding above; includes GPDs < 1.5 Hz with continuous background[6])

Outpatient EEG was ordered by the primary care provider or neurologist and could be either a routine (22 minutes) or a long (75 minutes) study, based on their clinical judgment. The outpatient EEG is performed as per ACNS guideline.[7] Two dual (epilepsy and neurophysiology) board-certified authors (HS, VP) independently reviewed the raw tracings of outpatient EEGs. A consensus was reached, in case of disagreements, after mutual review. They were classified into the following categories: normal, non-epileptiform, and epileptiform (based on the presence of interictal epileptiform abnormalities).[6] Functional outcome at the last follow-up was classified based on modified Rankin Scale (mRS) as good (mRS 0 to 3) and poor (4 or 5). Categorical variables are described using frequencies and percentages. Continuous variables are described using medians and quartiles (first - third quartiles) or means and standard deviations depending on the distribution. Fisher's exact test was used to assess non-random associations between categorical variables.

3 | RESULTS

A total of 603 SCA patients received cEEG monitoring during acute admission at our institution, and 294 survived hospitalization, including 33 patients discharged to hospice. The initial search of outpatient EEG revealed 58 patients fulfilling the inclusion criteria. We excluded 26 patients due to the lack of adequate data or prior history of epilepsy (n = 5). The final analysis included a total of 32 patients (16 females; 50%), with a mean age of 52.1 [standard deviation (SD) ± 13.6] years. Twenty-four patients among them received cEEG during their acute admission at our institution, accounting for 9.2% of the SCA patients who survived initial hospitalization and were not discharged to hospice.

3.1 | Acute hospitalization findings

The median return of spontaneous circulation (ROSC) was 13.5 (6.5-21.5) minutes among the 24/32 (75%) patients with available data. Eleven (34.4%) patients underwent therapeutic hypothermia (TH), another 11 did not. The status was unknown in the rest. Among the 24 patients with available acute cEEG data, 6 (25%) had suppression pattern: two background suppression and 4 burst suppression (two had non-epileptic theta frequency burst, and two had vertex region spikes), 6 (25%) had an epileptiform pattern, and the rest (50%) had continuous EEGs (Table 1). A total of 12 (37.5%) patients suffered either an acute (within 7 days of SCA) clinical seizure [8 patients—generalized tonic–clonic seizure prior to EEG recording and 4 patients had an electrographic seizure (2 GPDs >2.5 Hz and 2 lateralized pattern) on cEEG] or acute post-hypoxic myoclonus (aPHM; 4 patients; Table 2). None of the patients had myoclonic status epilepticus. Apart from 4 patients with epileptiform EEG having acute electrographic seizures, the other 2 had GPDs between 1.5 and 2.5 Hz and vertex polyspikes (Patient #16 Table 3), respectively. Acute CT head was available in 31 out of 32 patients. It was unremarkable in all except 4 patients, including one with intraparenchymal hemorrhages, one with bilateral globus pallidus edema, and two with diffuse cerebral edema. During acute hospitalization, 24 (75%) patients were initiated and subsequently discharged on ASMs. Table 3 provides information about SCA etiology, CPC, and hospital discharge

TABLE 1 Acute EEG and clinical findings in the study population

Acute EEG	Number of patients (N = 32)	Acute Clinical Seizure (10/32)	Acute Post anoxic Myoclonus (5/32)	ASM d(24/32)
Suppressed background	6 (18.7%)	3	2	5
Epileptogenic	6 (18.7%)	1	1	5
Continuous	12 (37.5%)	6	1	11
Unknown	8 (25%)	0	1	3

Abbreviation: ASM, Antiseizure medications.

TABLE 2 Acute EEG and follow-up outpatient EEG findings in the study population

Acute EEG Follow-up EEG	Suppressed background (n = 6)	Epileptiform (n = 6)	Continuous (n = 12)	Unknown (n = 8)
Epileptiform abnormality (n = 11)	4 (2)	5 (2)	0	2 (2)
Non-epileptiform abnormality (n = 8)	1	0	6 (2)	1
Normal (n = 13)	1	1	6 (1)	5 (2)

Note: Number in parenthesis signify patients with either long-term clinical seizures and/or chronic post-hypoxic myoclonus.

TABLE 3 Characteristics of patients with SCA survivors

Subjects	Age at outpatient EEG	ROSC (mins)	Arrest etiology	Acute EEG	Acute CT head	CPC score	Disposition	ASM on discharge	MRI brain follow-up	mRS outcome on last f/u	F/U Seizures	ASM on last f/u	F/U EEG Findings
1	42.4	5	Long QT syndrome	Continuous	WNL	2	Home/ self-care	LEV	Multiple intracranial white matter lesions	Good	No	LEV	Normal
2	40.4	40	Overdose	Not available	WNL	3	Long-term care facility	LEV	Not done	Good	SZ	VPA, OXC, GBP	Epileptiform
3	72.0	6	NSTEMI	Suppression	WNL	3	Long-term care facility	LEV	Near symmetric abnormalities in bilateral frontal, parietal, and occipital regions	Good	No	None	Epileptiform
4	38.3	15	Overdose	Continuous	Edema in bilateral globus pallidus	2	Home/ self-care	LEV	Changes compatible with anoxic brain injury	Good	SZ+PAM	None	Non-epileptiform
5	56.4	23	V. Fib	Suppression	WNL	3	Acute care facility	LEV	Mild chronic microvascular ischemic changes	Good	No	LEV	Normal
6	58.6	20	STEMI	Continuous	WNL		-	LEV	WNL	Poor	PAM	Ativan	Normal
7	69.0	10	STEMI	Continuous	WNL	3	Acute care facility	LEV	WNL	Good	No	None	Normal
8	41.0	8	V. Fib	Continuous	WNL	2	Home/ self-care	LEV	WNL	Good	No	None	Normal
9	62.2	-	V. Fib	Continuous	WNL	3	Acute care facility	LEV	WNL	Good	No	None	Non-epileptiform
10	48.0	15	-	Continuous	Not available	-	-	LEV	WNL	Good	No	None	Non-epileptiform

(Continues)

TABLE 3 (Continued)

Subjects	Age at outpatient EEG	ROSC (mins)	Arrest etiology	Acute EEG	Acute CT head	CPC score	Disposition	ASM on discharge	MRI brain follow-up	mRS outcome on last f/u	F/U Seizures	ASM on last f/u	F/U EEG Findings
11	47.8	-	-	Continuous	Not available	-	-	LEV	Mild asymmetry of the left mesial temporal lobe	Good	No	LEV	Normal
12	81.1	5	-	Suppression	WNL	-	-	LEV PB	Severe diffuse brain atrophy	Poor	SZ+PAM	LEV PB	Epileptiform
13	41.5	3	SAH	Epileptiform	Subarachnoid hemorrhage	3	Acute care facility	LEV, LCM	Encephalomalacia in the right ICA distribution.	Good	No	LEV	Epileptiform
14	19.5	15	Long QT syndrome	Continuous	WNL	3	-	LEV, PB	Symmetric increased signal in the basal ganglia	Good	SZ+PAM	LEV, ZNS,VPA, CLN	Non-epileptiform
15	57.7		V. Fib	Epileptiform	WNL	2	Home/ self-care	LEV, PHT	WNL	Good	No	LEV, PHT	Epileptiform
16	51.6	5	STEMI	Epileptiform[a]	WNL	3	Long-term care facility	LEV	Moderate to severe diffuse brain atrophy	Good	SZ+PAM	LTG, LCM, CLB	Epileptiform
17	39.1		Long QT syndrome	Epileptiform	Not available	3	Long-term care facility	LMT, LEV	Not done	Poor	SZ	LEV, LTG	Epileptiform
18	48.5		V. Fib	Epileptiform	WNL	2	Home/ self-care	LEV, ZNS	WNL	Good	No	LEV, ZNS	Normal
19	47.0		Alcohol withdrawal seizure	No info	WNL	-	-	LEV	Moderate to severe diffuse brain atrophy	Good	SZ+PAM	CLB, CLN, ZNS, LEV, OXC	Epileptiform
20	72.0	8	Flu sepsis	Suppression	WNL	-	-	None	WNL	Good	No	None	Epileptiform
21	52.3	9	asystole 2/2 Sepsis	Epileptiform	WNL	3	Acute care facility	None	WNL	Poor	No	None	Epileptiform
22	51.7		Anaphylaxis		Not available	2	Home/ self-care	None	WNL	Good	No	None	Non-epileptiform

(Continues)

TABLE 3 (Continued)

Subjects	Age at outpatient EEG	ROSC (mins)	Arrest etiology	Acute EEG	Acute CT head	CPC score	Disposition	ASM on discharge	MRI brain follow-up	mRS outcome on last f/u	F/U Seizures	ASM on last f/u	F/U EEG Findings
23	35.2	60	Surgery (C- section)	Not available	WNL			None	WNL	Good	No	None	Normal
24	31.5	45		Not available	WNL	2	Home/ self-care	None	WNL	Good	No	None	Normal
25	48.4	40	H1N1 sepsis	-	Not available	3	Long-term care facility	None	WNL	Good	No	None	Normal
26	50.6	12	MI	-	Not available	2	Home/ self-care	None	Moderate to severe diffuse brain atrophy	Good	SZ	LEV, LTG	Normal
27	67.0	0.5	PEA	Continuous	WNL	2	Home/ self-care	None	WNL	Good	No	None	Normal
28	56.9		V Fib	Continuous	Not available	2	Home/ self-care	LEV	WNL	Good	No	None	Non-epileptiform
29	32.7	10	Asthma exacerbation	Not available	Not available	2	Home/ self-care	PHT	Moderate diffuse brain atrophy	Poor	PAM	PHT	Normal
30	62.2	15	-	Suppression	Diffuse cerebral edema	2	Home/ self-care	PHT	Not done	Poor	PAM	LEV	Epileptiform
31	31.5	16	-	Continuous	Diffuse cerebral edema	2	Home/ self-care	LEV, CLN	WNL	Poor	No	None	Non-epileptiform
32	70.0	22	Respiratory failure	Suppression	WNL	3	Home	VPA	WNL	Good	No	None	Non-epileptiform

Abbreviations: ASM, Antiseizure medications; BS, Background slow; CLB, Clobazam; CLN, clonazepam; CPC, Cerebral performance category; CS, Continuous slow; Flu, Influenza; GBP, Gabapentin; H1N1, swine flu; LCM, Lacosamide; LEV, Levetiracetam; LMT, Lamotrigine; MI, Myocardial Infarction; NSTEMI, non-ST-segment elevation myocardial infarction; OXC, Oxcarbazepine; PAM, Post-anoxic myoclonus; PB, Phenobarbital; PEA, Pulseless electrical activity; PHT, Phenytoin; ROSC, Return of spontaneous circulation; SAH, subarachnoid hemorrhage; STEMI, ST-elevation myocardial infarction; SZ, Seizure; V. fib, Ventricular fibrillation; VPA, Valproic Acid; WNL, within normal limits; ZNS, Zonisamide.

[a]Vertex polyspikes, mRS 0-3 = Good Outcome, mRS ≥ 4 = Poor outcome.

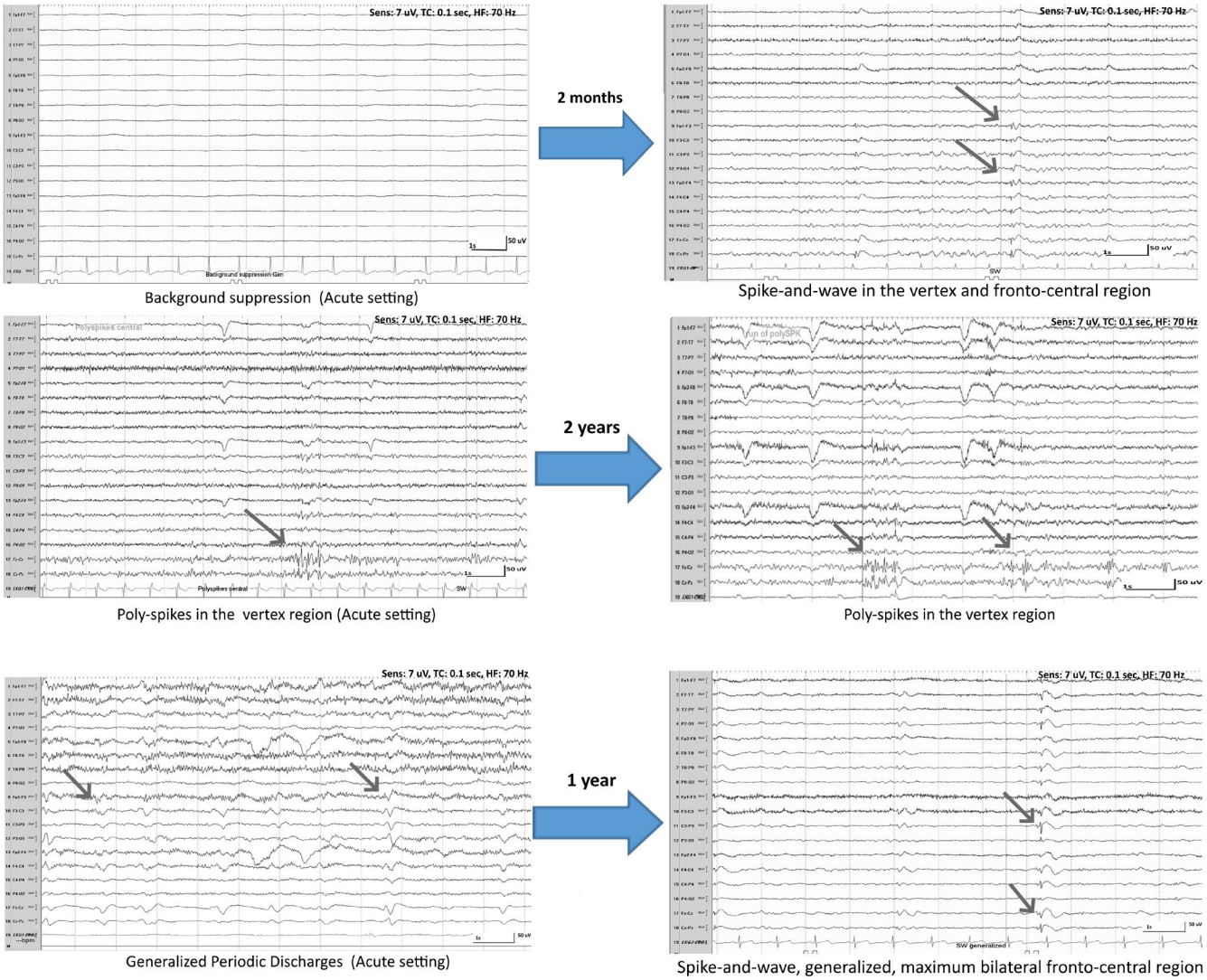

FIGURE 1 EEGs on the right side show outpatient EEGs with IEDs in the study population. The EEGs on the left side capture the prominent acute EEG finding for each patient and the duration between the two tests is reflected between the corresponding EEGs

disposition of individual patients included in the study. In addition, Table 3 also provides details of individual patient's post-discharge electro-clinical, neuroimaging, and functional outcome data.

3.2 | Long-term electrographic (EEG) profile

The median duration between the SCA and follow-up outpatient EEG was 13.2 (4.9-26.2) months. Twelve patients (37.5%) underwent a routine EEG, and 20 (62.5%) underwent a long EEG. The follow-up EEGs were performed as part of a clinical evaluation of events concerning for seizures in 21 (65.6%) patients and for guiding ASM management in the rest. Follow-up EEG showed interictal epileptiform discharges (IEDs) in 11(34.4%) patients, non-epileptiform abnormality (slowing) in 8 (25%), and was within normal limits in the rest (Table 2). Table 2 shows the correlation of long-term EEG findings with available acute EEG data. None of the patients with continuous cEEG pattern (ie, lacking suppression or epileptiform findings) had IEDs on their follow-up EEG. Among the follow-up EEGs with IEDs, 4 (36.4%) patients had suppression pattern, 5 (45.5%) had an epileptiform pattern on acute cEEGs, and data were not available for 2 patients. The review of raw follow-up EEG showed that 10 out 11 (90.9%) had IEDs in the vertex and frontocentral regions (Figure 1), and only one patient with a history of SAH, had left temporal spikes.

3.3 | Long-term clinical profile

During a mean clinical follow-up period of 28.2 (±42.3) months, a total of 11 (34.4%) patients suffered seizures. The semiology varied, with unknown-onset tonic–clonic seizures in 5 patients; in which 2 of them had additional cPHM. The

following 3 patients had unknown-onset behavioral arrest, and the remaining 3 had an exclusive cPHM.

Of note, the semiology remained unchanged during the follow-up period. Six of these 11 patients had IEDs on EEG. Conversely, 5 patients (15.6% of the study population) with IEDs on outpatient EEGs did not have clinical seizures or cPHM during the follow-up period. There was no significant correlation between the presence of IEDs and seizures during the follow-up in the study cohort [Odds Ratio (OR) = 3.8 (95% confidence interval (CI) 0.8-18.2, $P = .12$)]. No electro-clinical correlate between IEDs and cPHM could be established on outpatient EEGs.

All patients underwent neuroimaging tests during the follow-up. Twenty-nine (90.6%) patients had a brain MRI, and the rest had a CT head, a mean of 10.3 (±19.9) months after SCA. Neuroimaging was unremarkable in 17 (53.1%) patients and abnormal in the rest (Table 3). Ten out of 11 patients with seizures during the follow-up had abnormal neuroimaging findings, most commonly described as moderate to severe brain atrophy for age (Table 3). Patients with seizures were significantly more likely to have abnormal neuroimaging findings during the follow-up compared to patients who did not develop seizures [OR = 25 (95% CI 2.6->100, $P = .002$)].

Of the 24 patients discharged on ASMs (21/24 on Levetiracetam), 16 (50% of the study population) were taking them at the time of the last clinical follow-up (Table 3). The latter included 5 (15.6%) patients with no seizures (or cPHM) since hospital discharge. Out of 8 patients weaned off of ASMs, 7 were seen by epileptologists. A good functional outcome at last follow-up was achieved by 25 (78.1%) patients, including 6 (24%) patients suffering seizures. In contrast, 5 out of 7 (71.4%) patients with poor outcomes were having seizures at the last follow-up, including the 3 patients with exclusive cPHM [OR = 7.9 (95% CI 1.2-51.8, $P = .03$)].

4 | DISCUSSION

In our study of 32 SCA survivors with post-hospitalization electro-clinical data, one-third (34.4%) developed new-onset epilepsy[8] after the hospital discharge. Additionally, one-third of the study population had IEDs on their outpatient EEGs, including 5 patients without seizures during an average follow-up period of more than 2 years. Combined, 16 patients (50% of the study population) had developed epilepsy or showed markers of epileptogenicity during the follow-up. This is likely an over-estimation of the overall epilepsy risk in SCA survivors because our inclusion criteria required patients with outpatient EEG. A recent study analyzed data from 5% of the elderly (≥66 years old) Medicare beneficiaries from 2008 to 2015 who survived to hospital discharge after SCA and did not suffer an acute seizure. They did not find an increase in the long-term risk of epilepsy development after adjusting for demographics and comorbidities.[9] However, this was a much older patient population than our cohort and did not have acute seizures. Around 10%–35% of patients undergoing cEEG after SCA have NCS and NCSE,[10] which is consistent with the acute findings in our cohort.

The lack of seizures in 1 out of 6 patients (15.6% of the study population) with IEDs is a notable finding. It is in sharp contrast to the previous studies in the healthy adult population that found only 2%–3% of individuals with IEDs without clinical seizures.[3] This electro-clinical disconnect in a substantial number of the long-term SCA survivors is either a phenomenon unique to this population or suggestive of an enduring epileptogenicity with epilepsy onset much later than our current follow-up period, akin to patients with TBI.[11] The possibility of missed clinical seizures or ongoing NCS cannot be ruled out based on our data. Another critical finding of our study, unlikely to be influenced by patient selection, is the unique location of IEDs in SCA survivors. Almost uniformly (91% patients), they were noted in the vertex and frontocentral regions. This specificity in the location of the IEDs is quite unique and a curious finding. One potential possibility is damage to the vulnerable watershed areas, in this case, anterior cerebral artery-middle cerebral artery "ACA-MCA" territory, which are affected first in the setting of reduced cerebral blood flow during SCA. However, such specific changes were not seen in the available CT scans during the acute stage. Recent studies analyzing the electro-clinical significance of aPHM after SCA have reported the presence of midline/vertex spikes over a continuous background as a favorable prognostic marker.[10,12] It is unclear if the vertex and frontocentral location of IEDs in our cohort is an electrographic vestige from the acute stage. On the other hand, it could represent a signature, long-term epileptogenic marker in the SCA survivors. However, long-term follow-up studies are needed to confirm this observation. In contrast to the lack of IEDs and their association with post-discharge seizures, the neuroimaging abnormalities and functional outcomes were highly associated with epilepsy development in the study cohort. Lacking discrete lesions, the moderate-to-severe atrophy disproportionate to the patient's age noted in these patients suggests SCA-related epilepsy may emerge from diffuse neuronal loss incurred as a sequela to the anoxic injury.

One out of 4 patients in the study cohort suffered cPHM. The incidence of cPHM after SCA is unclear. A recent literature review found 77 case reports or case series (most extensive with 14 patients) of cPHM (also known as Lance-Adams syndrome).[13] Of note, unlike 5 patients in our cohort, the review did not find patients with co-existing clinical seizures and cPHM. It is likely due to under-reporting or may actually be due to the rarity of this

phenomenon. The review found that 29 out of the 74 cases of cPHM with EEG had interictal epileptiform discharges, which is not unexpected as it is considered a cortical form of myoclonus.[13] Similarly, in our cohort, 4 out of 8 patients with cPHM patient had IEDs.

The acute ASM management of patients with SCA on cEEG remains controversial. Depending on the acute SCA patients analyzed, 20%-75% of them are on ASMs.[12,14] More than two-thirds of our cohort was on ASMs at the time of hospital discharge, and half of them were still on ASMs at the time of the last follow-up. While these figures may be higher than in general SCA survivors, our study highlights that SCA survivors who are treated with ASMs acutely may end up staying on ASMs for a prolonged period. The indication for an outpatient EEG in one-thirds of the study population was to guide ASM management. Of note, 7 out of 8 patients who successfully discontinued ASMs were under the care of epileptologists, highlighting the importance of models of outpatient care like post-acute symptomatic seizure (PASS) clinic reported by us recently.[15]

While our study comprehensively analyzes the long-term electrographic, clinical, neuroimaging findings, functional outcomes and ASM management practices in SCA survivors in light of the acute care and findings, it is limited by a small study cohort and its retrospective study design. Additionally, we did not have uniform neuroimaging data available for all patients due to retrospective nature. The most significant limitation of our study is that we only included SCA survivors who had a follow-up EEG after SCA-related hospitalization. This selection skews the study population toward SCA survivors, more likely to have epilepsy-related concerns. However, this limitation is secondary to the primary aim of the study—to analyze the long-term electro-clinical correlates after surviving SCA. Additionally, we did not have access to acute EEG information of all patients.

In conclusion, using a small cohort, we present the first long-term electro-clinical profile of SCA survivors. We found that as high as 34% of survivors could develop epilepsy, and 15.6% show markers of potential epileptogenicity on their EEGs after hospital discharge. There seems to be a potential signature localization of IEDs among SCA survivors in the vertex and frontocentral regions, noted in 10 out of 11 patients. We found co-existence of typical clinical seizures and cPHM in SCA survivors and a wide variability in outpatient ASM management among them. Despite its limitations, our study warrants a multi-center collaborative effort to investigate epilepsy development and its management among SCA survivors.

CONFLICT OF INTEREST

None of the authors has any conflict of interest to disclose. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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