Original Article

Determinants of Outcome in Convulsive Status Epilepticus in Adults: An Ambispective Study from Central India

Raunak Dani, Ajoy Sodani, Kapil Telang, Richa Nigam

Department of Neurology, Sri Aurobindo Medical College and P.G. Institute, Indore, Madhya Pradesh, India

Abstract

Background: The determinants of the outcome in adult convulsive status epilepticus (CSE), also the implication of the value of mean arterial blood pressure (MAP), and random blood sugar at admission on the outcome are not clear. **Objectives:** The objective of this study is to look for the determinants of unfavorable outcome in CSE. **Materials and Methods:** Ambispectively gathered data from 55 patients, treated consecutively with identical protocol during January 2010–December 2016, were analyzed. The demographic and clinical variables were identified and correlated with outcome in each individual. **Results:** There were 65.45% males and 34.55% females. Favorable outcome (conscious and discharged) was seen in 63.6%, unfavorable (death 14.5%, absent cortical functions 10.9%, and inability to wean-off anesthetic agents 10.9%). The parameters associated with unfavorable outcome were female gender (odds ratio [OR]: 1.45), MAP ≤80 mmHg (OR: 2.57), time to first medical attention >5 h (OR: 127.8), and time to control clinical seizures >3.5 h (OR: 7.87). Almost 44.2% of patients with SE severity score >2 had unfavorable outcome (sensitivity 75% and specificity 45.7%). New scoring system, the CSE outcome score (CSEOS, developed by combining the predictors associated with higher odds of poor outcome), predicted the poor outcome with the sensitivity and specificity of 90% and 54.29%, respectively. **Discussion and Conclusion:** Low MAP and delay of >3.5 h in treatment initiation or seizure control are the key determinants of poor outcome in CSE. With the incorporation of CSEOS, we believe that our findings can be helpful in the process of clinical decision-making and prognostication of patients with CSE.

Keywords: Convulsive status epilepticus, mortality, outcome, prognosis, status epilepticus severity score

Introduction

Status epilepticus (SE), lasting beyond 5 min without regaining of consciousness,[1] is a neurological emergency associated with high short- and long-term mortality and morbidity.[2] The case fatality and incidence rates differ widely among the reports emerging from the different part of the globe. Sánchez and Rincon,[3] in a recent review, reported the incidence of SE in the adult population of the US to be 28.4/100,000/y, in Asian continent 42/100,000/y, and Honduras 104/100,000/y.

The death rates associated with SE have declined from 50% to 20%–39% in last few decades.[4] Recent investigators from India have found the mortality in SE in the range from 5% to 29.3%.[5-7] Improved management strategies and availability of better antiepileptic medications might underlie the change in the outcome of SE.

While it is known that aged persons and delayed treatment initiation and control of SE are at higher risk of unfavorable outcome,[5,8] but how old is too old, and what duration is too long is not clear. Most of the studies[5,8] have provided a range of age and duration of SE, difficult to apply directly to the individual patient.[5,8] Furthermore, the reported cohorts of the patients are not uniform. Some have patients of myoclonic epilepsies and nonconvulsive SE (CSE) with different levels of sensorium ranging between awake and deep coma[9,10] and age groups ranging from infants[11] to septuagenarian.[5] To the best of our knowledge, important patient-related variables with the implication in the cerebral metabolism of a critically sick patient, such as random blood sugar (RBS) and mean arterial blood pressure (MAP), have not been examined. Clear knowing of the determinants of the prognosis is of prime importance as it helps in the planning of effective treatment and preventive strategies.[9,12] It is, therefore, not surprising that humongous work has been done in this direction, and scores have been developed to help the clinician draw a sensible management algorithm for an individual patient.[9,13,14] One of the frequently used scores, status epilepticus severity score (STESS),[9] was developed to predict the outcome of SE have had limited succe ss.[13,15,16]

We, therefore, planned an ambispective study and reviewed the medical records of the adults admitted to our neurological Intensive Care Unit (NICU) with CSE, between January 2010 and December 2014, and prospectively enrolled the patients of CSE admitted between January 2015 and December 2016. The aim was to identify the determinants of short-term outcome in

Address for correspondence: Dr. Raunak Dani, 4th Floor, Department of Neurology, Sri Aurobindo Medical College and P.G. Institute, Sanwer Road, Indore, Madhya Pradesh, India.
E-mail: raunak.dani@gmail.com

DOI: 10.4103/aian.AIAN_466_17

CSE. The objectives were to identify, if there exists, a cutoff value for age, duration of time before treatment is started and for the time elapsed before the control of the clinical seizures and also the ability of STESS to predict the poor outcome in our cohort.

Materials and Methods[Figure 1]

This is an ambispective observational study conducted in the department of neurology attached to a teaching hospital of Central India.

The patients of CSE, i. e., prolonged or intermittent seizures lasting beyond 5 min, without full recovery of sensorium, are admitted to our well-equipped NICU and treated consecutively as per the departmental CSE treatment protocol adopted and modified from the International League against Epilepsy (ILAE).[1,17] Every patient with CSE is subjected to continuous electroencephalogram (EEG; 21 channel, RMS model-Maximus, version 4.2.54), noninvasive blood pressure (BP), and two hourly blood glucose monitoring. The MAP and blood sugar levels are actively maintained above 80 mmHg and below 140 mg%, respectively.

A total of 59 patients were admitted during the study period with ongoing convulsive seizures beyond 5 min and altered consciousness.[1] Patients with acute traumatic brain injury, myoclonic epilepsies, and psychogenic seizures (n = 4) were excluded from the study. The medical records and study pro forma (including raw EEG data) of remaining the 55 patients were reviewed between January and June 2017, and the following information was extracted: (a) age in years (y), (b) gender, (c) history of epilepsy and treatment, (d) type of seizure at the onset of SE, (e) MAP and RBS at the time of admission, (f) history of seizure/s with complete recovery of sensorium (premonitory seizure) in previous 24 h (h), (g) time of onset and etiology of SE, (h) time of the first medical attention and treatment received, (i) time of the cessation of the clinical seizure, (j) treatment received after admission, and (k) the outcome, i.e., discharge with full recovery of sensorium; ability to wean off from an induced medical coma at the time of discharge on request; and the state of the brain function at the time of discharge on request/inhospital death. The etiology and classification of SE were ascertained as per the guidelines of ILAE.[1] Type of SE as per the semiology[1] at the onset-generalized tonic/clonic/tonic–clonic (GTCS, n = 45); partial with secondarily generalized (n = 10).

Data management

The cohort

The patients were grouped according to the (1) age ≤40 year (n = 34) and >40 (n = 21); (2) gender (men n = 36; women n = 19); (3) history of epilepsy into break through seizure evolving into SE (n = 20) and *de novo* SE (n = 35); (4) etiology – acute symptomatic (n = 40) and others (remote symptomatic and unknown; n = 15); and (5) results of MAP and RBS were recorded as continuous variable and later were grouped as >80 mmHg (n = 44) and ≤80 mmHg (n = 11), and RBS was divided >140 (n = 27) and 140 mg% (n = 28).

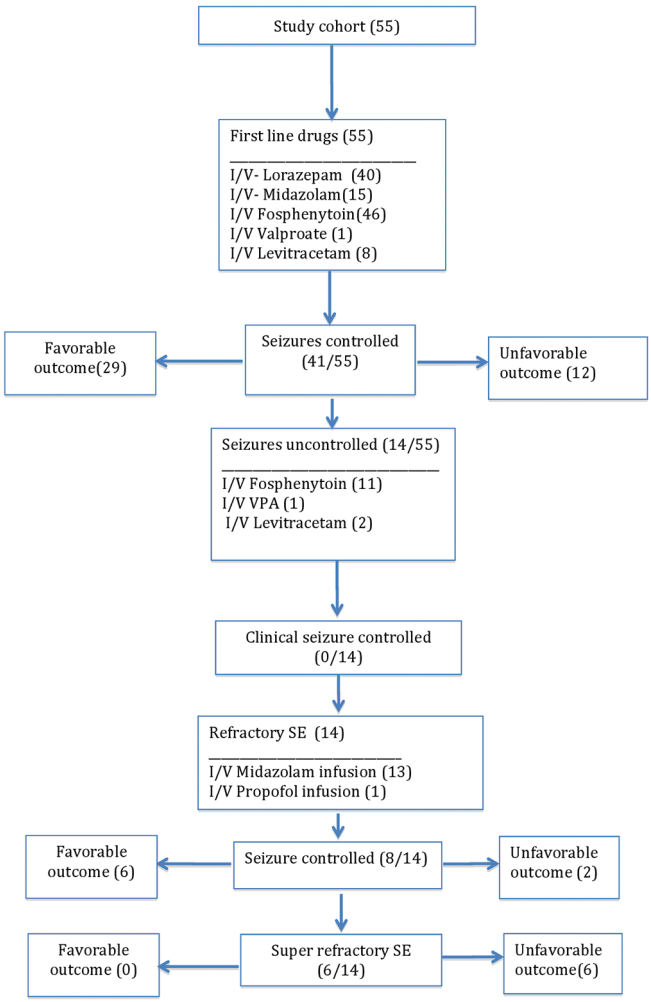


Figure 1: Summary of treatment and outcome/numbers in parenthesis (n)

The electroencephalogram

As per the previously reported protocol by Kalita *et al*.,[7] the raw data of the 1st h of EEG were reviewed by one of the investigators (Ajoy Kumar Sodani [AKS], blinded for the treatment and outcome of the patients) in 42 out of 55 (76.36%) patients and were arbitrarily grouped into "Type A pattern" (focal slowing/periodic lateralized epileptiform discharges [PLEDs]/focal ictal epileptiform discharges with or without secondary generalization) and "Type B pattern" (generalized slowing, generalized low amplitude, or featureless EEG). The EEG data were not retrievable in the remaining (n = 13) because of the damaged storage media.

The time-related parameters

Time to first medical attention (t2MA) was calculated as the time lapsed between the onset of SE and reaching a medical facility. t2MA was recorded as a continuous variable and for categorical analysis was divided into two groups modified from the observation of Murthy *et al*.,[5] who reported the

good outcome in those who reach medical facility within 5 h of seizure onset.

Time to control clinical seizures (t2CS) was calculated as the time lapsed between the onset of SE and cessation of clinical seizures.

The status epilepticus severity score

STESS score was calculated as per the previously described methods,[9] which incorporates age (<65 years, $n = 53$; ≥65 years, $n = 02$), level of consciousness (alert/confused, n = 0; stuporous/comatose, $n = 55$), worst seizure type (convulsive $n = 55$), and previous history of seizures (yes $n = 20$; no $n = 35$). They predicted that higher the score, poorer is the outcome. The cutoff score for the same being 2, i.e. score >2 having poorer outcome. The same cutoff score was applied to look for favorable outcome in patients with score <2, as compared to >2.

The outcome

The cohort was divided according to the condition of the patient at the time of discharge from NICU as following:

- Unfavorable outcome – inhospital death or absence of obvious cortical functions after cessation of sedatives or inability to wean the patient off the medication used to induce a therapeutic medical coma
- Favorable outcome – SE controlled and discharged with full recovery of sensorium.

Statistical analysis

The relationship between various variables and the outcome was evaluated using multivariate binary logistic regression; sensitivity, specificity, and area under the curve (AUC) were determined by drawing receiver operating characteristic (ROC) curves using SPSS software.

For categorical analysis, odds ratio (OR) and its 95% confidence interval (CI) were calculated according to Altman 1991 using the free online software.[18]

The level of significance (two-tailed *P* value, Fisher's exact test) was calculated as and when required by drawing a 2 × 2 contingency table.[19]

The significance level was set at OR >1 and $P < 0.05$.

The Institutional Ethical Committee approved the study protocol.

Results

The results have been summarized in Table 1.

Of 55 patients, 35 (63.63%; mean age 34.7 ± 12.7y) had favorable outcome while 20 (36.37%; mean age 46.8 ± 16.8y) had the unfavorable outcome, later include death 14.5% (8 of 55); no meaningful cortical function 10.9% (6 of 55), and inability to withdraw from medical coma 10.9% (6 out of 55). In 75% (41 of 55) patients, SE got controlled with the first-line therapy, while 25% ($n = 14$) patients required induction of medical coma. None of the patients, in whom the super-refractory SE was encountered (10.9%, 6 of 55), had a favorable outcome [Figure 1]. Of the six patients who had control of SE but had unfavorable outcome, all had t2MA of >5 h and had achieved control of seizures (t2CS) in >5 h (5 h >24 h).

Demographic variables versus outcome

There were 36 (65.45%) men with mean age of the cohort being 39.09 ± 15.34 years (range: 16–70). The odds of unfavorable outcome were significantly high for the women (OR: 1.45, 95% CI: 0.4–4.5) and those aged >40 years (OR: 3.05, 95% CI: 0.9–9.6); furthermore, the cutoff age associated with the unfavorable outcome was found to be >52 years on ROC (sensitivity – 50%, specificity –91.43, and AUC –0.71) [Figure 2].

Clinical variables versus outcome

The premonitory seizures were seen in 27.2% (15 of 55) of patients, the mean time lapses between them, and SE was 6.48 ± 5.6 h (range: 0.5–19 h). The absence of a previous history of epilepsy (*de novo* SE) was associated with higher odds of unfavorable outcome (OR: 3.36, 95% CI: 0.9–12.1). Acute symptomatic CSE, when compared with CSE because of other etiologies (remote symptomatic, unknown cause), showed that 53.4% of patients of the latter group had poor outcome as compared to 30% in the former. The difference was not statistically significant.

The mean MAP at admission was 93 ± 21 mmHg. MAP of ≤80 mmHg was associated with significant odds of poor outcome (OR: 2.57, 95% CI: 0.66–9.8), whereas no correlation was found between the value of RBS at admission and the outcome.

The mean t2MA for the cohort was 3.19 ± 4 h. The mean t2MA in those with favorable and unfavorable outcome was 1.38 ± 1 h and 6.37 ± 5.32 h, respectively. None of the patients with t2MA >5 h ($n = 13$) had a favorable outcome. ROC curve further supports that the delay in reaching to a medical facility is a robust determinant of an unfavorable outcome (OR: 127.8, 95% CI: 6.8–2394; sensitivity –70%, specificity – 97.14%, AUC – 0.79, and cutoff >3.5 h, two-tailed $P < 0.001$). Table 2 presents the t2MA and its relationship to the outcome from the perspective of different variables. (a) There is no gender bias in seeking medical attention, (b) higher percentage of the patients without previous history of epilepsy reach a medical facility beyond 5 h as compared with those with a history of epilepsy (45.7% vs. 20%) with the resultant poor outcome, and (c) the common causes of CSE in our cohort were drug default ($n = 14$) and brain scar ($n = 11$). Other causes and outcome have been summarized in Table 2. The results suggest that the persons with an unfavorable outcome, with above-mentioned causes, had longer t2MA as compared with those admitted because of SE precipitated due to alcohol-related causes ($n = 8$) in which all the patients presented within 1.73+-1.34 h of CSE onset and all had a good outcome.

Mean t2CS of the cohort was 10.2 ± 25.46 h, which in patients with poor outcome was 20.5 ± 38.9 h as against 4.93 ± 9.72 h in participants with a favorable outcome.

Table 1: Clinical characteristic of cohort (*n*=55) versus outcome

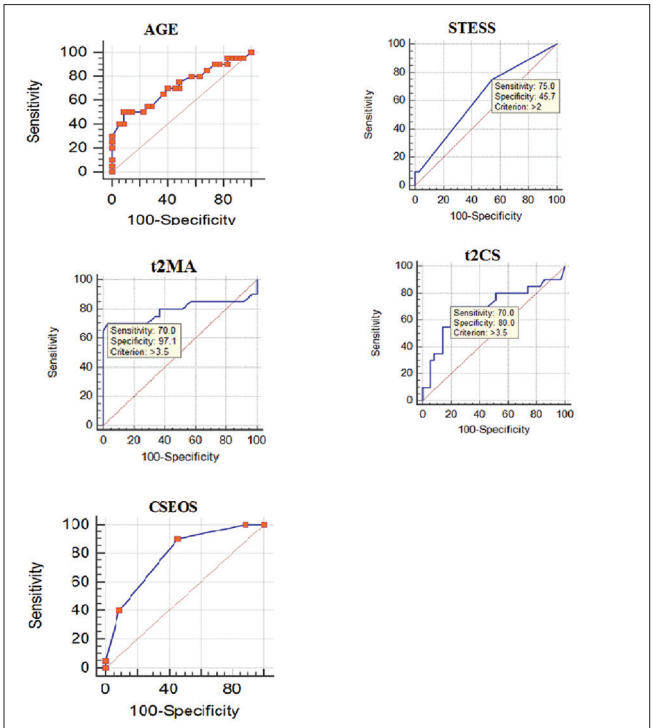
Variable	Division	Favorable outcome (35), *n* (%)	Unfavorable outcome (20), *n* (%)	Statistical significance OR (95% CI)
Age (years)	≤40 (*n*=34)	25 (73.5)	9 (26.5)	3.05 (0.9-9.6) (S)
	>40 (*n*=21)	10 (47.7)	11 (52.3)	
Gender	Male (*n*=36)	24 (66.7)	12 (33.3)	1.45 (0.4-4.5) (S)
	Female (*n*=19)	11 (57.9)	8 (42.10)	
Premonitory seizures	Yes (*n*=15)	11 (73.3)	4 (26.7)	0.82 (0.25-2.6) (NS)
	No (*n*=40)	24 (60)	16 (40)	
Breakthrough seizures	Yes (*n*=20)	16 (80)	4 (20)	3.36 (0.9-12.1) (S)
	No (*n*=35)	19 (54.3)	16 (45.7)	
Etiology of SE	Acute symptomatic (*n*=40)	28 (70)	12 (30)	0.37 (0.11-1.26) (NS)
	Others (*n*=15)	7 (46.6)	8 (53.4)	
Type of SE as per onset	GTCS (*n*=45)	27 (60)	18 (40)	2.66 (0.5-14.02) (S)
	Partial with secondary gen (*n*=10)	8 (80)	2 (20)	
MAP at admission (mmHg)	≤80 (*n*=11)	5 (45.4)	6 (54.6)	2.57 (0.66-9.8) (S)
	>80 (*n*=44)	30 (75)	14 (25)	
RBS at admission (mg/dl)	≤140 (*n*=28)	18 (64.2)	10 (35.8)	1.05 (0.35-3.17) (NS)
	>140 (*n*=27)	17 (62.9)	10 (37.1)	
t2MA (h)	≤5 (*n*=42)	35 (83.3)	7 (16.7)	127.8 (6.8-2394) (S)
	>5 (*n*=13)	0	13 (100)	
t2CS (h)	≤3.5 (*n*=33)	27 (81.2)	6 (18.18)	7.87 (2.2-27.2) (S)
	>3.5 (*n*=22)	8 (36.4)	14 (63.6)	
EEG patterns (*n*=42)	Type A (*n*=26)	16 (61.5)	10 (38.5)	1.87 (0.47-7.45) (S)
	Type B (*n*=16)	12 (75)	4 (25)	
STESS	≤2 (*n*=21)	16 (76.1)	5 (23.9)	2.52 (0.75-8.4) (S)
	>2 (*n*=34)	19 (55.8)	15 (44.2)	
CSEOS	≤1 (*n*=26)	23 (88.4)	3 (11.6)	10.86 (2.6-44.6) (S)

*CI=95% confidence interval, STESS=Status epilepticus severity score, t2MA=Time to first medical attention, t2CS=Time to control clinical seizures, MAP=Mean arterial blood pressure, RBS=Random blood sugar, OR=Odds ratio, S=Significant, NS=Not significant, STESS=Status epilepticus severity score, SEOS=Proposed status epilepticus outcome score, GTCS=Generalized tonic-clonic seizure, SE=Status epilepticus, EEG=Electroencephalogram

Table 2: Time to medical attention versus outcome

Variables	Good outcome		Bad outcome	
	n (%)	t2MA (h, mean±SD)	*n* (%)	t2MA (h, mean±SD)
Demographic variables (years)				
Age ≤40 (34)	25 (73.5)	1.30±1.04	9 (26.5)	7.15±3.69
Age >40 (21)	10 (47.7)	1.56±1.11	11 (52.3)	5.74±6.46
Male (36)	24 (66.7)	1.5±1.07	12 (33.3)	6.82±6.47
Female (19)	11 (57.9)	1.09±1	8 (42.10)	5.71±3.15
Clinical variables				
Known case of epilepsy (20)	16 (80)	1.33±0.93	4 (20)	8±3.55
Not a known case of epilepsy (35)	19 (54.3)	1.41±1.16	16 (45.7)	6.22±5.77
MAP ≤80 (11)	5 (45.4)	0.9±0.75	6 (54.6)	5.51±4.06
MAP >80 (44)	30 (75)	1.46±1.08	14 (25)	6.03±5.37
Etiology				
Drug default (14)	11 (78.5)	1.57±0.92	3 (21.5)	9±3.60
CNS infection (9)	3 (33.3)	0.31±0.14	6 (66.7)	8.6±6.52
Stroke (8)	5 (62.5)	1.11±1.22	3 (37.5)	0.82±1.02
Alcohol related (8)	8 (100)	1.73±1.34	0	-
Metabolic (1)	1 (100)	1	-	-
Brain scar (11)	6 (54.5)	1.52±0.89	5 (45.5)	7.22±5.07
Genetic (1)	0	-	1 (100)	0.08
Unknown etiology (3)	1 (33.3)	0.4	2 (66.7)	5±1.41

CNS=Central nervous system, MAP=Mean arterial blood pressure, SD=Standard deviation



VARIABLES	AUC	SENSITIVITY (%)	SPECIFICITY(%)	CRITERION
Age	0.71	50	91.4	> 52 y
t2MA	0.79	70	97.14	>3.5 h
t2CS	0.69	70	80	>3.5 h
STESS	0.62	75	45.7	>2
CSEOS	0.78	90	54.2	>1

Figure 2: Receiver operating characteristic curves for age, t2MA, t2CS, status epilepticus severity score, and convulsive status epilepticus outcome score

The critical time for control of clinical seizures was 3.5 h (AUC – 0.6, sensitivity – 70, and specificity – 80), [Figure 2]. We, accordingly, divided the cohort into those requiring more ($n = 22$) or less ($n = 33$) than 3.5 h for the control of their clinical seizures. The group with t2CS >3.5 h had significantly higher odds of unfavorable outcome (OR – 7.87, CI – 2.2–27.2, two-tailed $P = 0.001$) [Table 1].

Severity scales versus outcome

Although the STESS score of >2 was found to be associated with higher odds of poor outcome (OR – 2.52, CI – 0.75–8.4) [Table 1]. With 55.8% of the patients with STESS >2 getting discharged and 23.9% of those with a score of ≤2 experiencing poor outcome, its ability to identify the unfavorable outcome correctly was poor (AUC – 0.62, sensitivity – 75%, and specificity – 45.7%); [Figure 2]. Logistic regression analysis done did not show any significance [Table 3].

We, therefore, integrated the STESS score with other parameters which were associated with higher odds of unfavorable outcome, namely, gender, MAP, t2MA, and t2CS. One point was assigned to the presence of each of following: female gender, MAP <80 mmHg, t2MA >3.5 h, t2CS >3.5 h, and STESS > 2. The absence of them was scored as zero. This new six-point CSE outcome score (CSEOS) was applied to every patient, and the net score was summed up which ranged between 0 and 5 such that higher the factors relating to an unfavorable outcome, higher the score [Table 4]. A ROC curve was drawn which showed AUC of 0.82, sensitivity of 85%, specificity of 65.7%, and cutoff value of >1. The cohort was divided accordingly, and the score of >1 found to be strongly associated with unfavorable outcome in CSE (OR – 10.86, 95% CI – 2.6–44.6, two-tailed $P = 0.0006$). Although we found that Type A EEG pattern, which included focal slowing/PLEDs/focal ictal epileptiform discharges with or without secondary generalization, to be associated with unfavorable outcome, but as the data in 13 patients were missing, we did not use this parameter in CSEOS scoring.

Table 3: Binary logistic regression of outcome with other parameters

Parameters	*Z*	*P*
Age	1.53	0.125 (NS)
STESS	1.52	0.128 (NS)
t2MA	2.66	0.008*
t2CS	−0.87	0.384 (NS)
MAP	−0.86	0.391 (NS)
RBS	−0.48	0.633 (NS)
History of epilepsy	0.41	0.679 (NS)
Cause of status epilepticus (yes)	1.04	0.297 (NS)

STESS=Status epilepticus severity score, t2MA=Time to first medical attention, t2CS=Time to control clinical seizures, MAP=Mean arterial blood pressure, RBS=Random blood sugar, NS=Not significant

Discussion

Our ambispective study shows that age >40 years, female gender, *de novo* SE, and type of SE (GTCS) are the chief clinical determinants associated with the unfavorable outcome of CSE. Association of older age[5,8,9] and female gender,[5] *de novo* SE,[9,15,20-24] and GTCS[7,9,25] with poor outcome has been reported previously.

The mortality of patients with our cohort was 14.5%. The mortality in SE has been reported to vary between 5% and 29.2%.[5-7,11,22]

The variability in the reported mortality rates could be due to the differences in the cohort characteristics. While our cohort consists solely of CSE while those reporting higher mortality[7] than us have included non-CSE, which is known to have poorer outcome.[9] In contrast, the low fatality rates of 5% as reported by Bhalla *et al*.[6] could be explained by the inclusion of higher percentage of patients of SE related to alcohol and drug default known to have better outcome.[26] Our patients with CSE related to drug default and alcohol had lowest incidences of unfavorable outcomes, a finding in line with Towne *et al*.[26]

To the best of our knowledge, no previous researchers have compared MAP and RBS of the patients with the outcome. We found that a MAP of ≤80 at presentation was associated with poor outcome of CSE and could be an important factor in prognostication of the patient. In the patients of

Table 4: Convulsive status epilepticus outcome score versus outcome

Score	Good outcome, *n* (%)	Bad outcome, *n* (%)
0 (*n*=9)	9 (100)	0
1 (*n*=17)	14 (82.3)	3 (17.7)
2 (*n*=11)	6 (54.54)	5 (45.45)
3 (*n*=10)	5 (50)	5 (50)
4 (*n*=7)	1 (14.2)	6 (85.8)
5 (*n*=1)	0	1 (100)

CSE, remaining uncontrolled beyond 30 min, the cerebral autoregulation fails, and the cerebral blood flow becomes dependent on systemic BP.[27]

Our results show that delay of beyond 3.5 h in reaching to a medical facility (t2MA) is a robust predictor of unfavorable outcome (either death, inability to withdraw anesthetic drugs, or absence of cortical functions) in CSE [Figure 2]. t2MA has been shown to be an important factor related the outcome in SE in previous works. Murthy *et al*.[5] and Towne *et al*.[26] also found the time lapse of more than 5 h and 1 h to be linked with poor outcome.

The previous researchers have found that survival was greater with shorter time to control seizures.[24,28,29] We also found that majority of the patients with t2CS >3.5 h had unfavorable outcome.

The proposer of STESS score, Rossetti *et al*.,[9] have commented that their score has a negative predictive value and thus it reliably identifies the SE patients who would survive. We found that the ability of STESS score of >2 to correctly identify the unfavorable outcome was poor (AUC –0.62, sensitivity –75%, and specificity – 45.7%) [Figure 2]. Atmaca *et al*.[15] also did not find STESS >2 to be a dependable factor for predicting poor outcome. The STESS scoring grid takes into consideration the age, the semiology of SE, the level of consciousness at admission, and history of previous seizures and summarily ignores the delay in starting treatment; time is taken in control of SE. The more robust EMSE (epidemiology-based mortality score in SE)[10] also does not make the use of t2MA and semiology in the score. Gender, a proven risk factor for SE, and MAP have not been used in any of the above-mentioned scoring systems to predict outcome of SE. A wide range of clinical situations, ranging from awake to comatose pretreatment consciousness level, mild (simple partial/absence/myoclonic) to severe (generalized tonic–clonic seizure/nonconvulsive) seizure types, have been clubbed in the above-mentioned scoring methods.

Our cohort is more uniform in the sense that it is exclusively composed of CSE (GTCS or secondarily generalized CSE) who reached to the research site in stuporous or comatose condition. We, therefore, developed a score by utilizing variables associated with higher odds of unfavorable outcome (viz., gender, MAP, STESS, t2MA, and t2CS). The new score for CSEOS correctly identified the outcome with a sensitivity and specificity of 90% and 54.29%, respectively. The drawback of our study is nonavailability of the EEG data in 23.6% of patients and that of comorbidities.

The CSEOS score needs to be validated by a prospective study in future.

Conclusion

Our study identifies that low MAP and delay of >3.5 h in treatment initiation or seizure control are the determinants of poor outcome in CSE. With incorporation of CSEOS, we believe that our findings can be helpful in the process of clinical decision-making and prognostication of patients with CSE.

Acknowledgment

The authors acknowledge Dr. Richa Nigam, Professor Department of Preventive and Social Medicine, and Ms. Jyoti Patil, technologist, for their contributions to this work.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Trinka E, Kälviäinen R. 25 years of advances in the definition, classification and treatment of status epilepticus. Seizure 2017;44:65-73.
2. Nair PP, Kalita J, Misra UK. Status epilepticus: Why, what, and how. J Postgrad Med 2011;57:242-52.
3. Sánchez S, Rincon F. Status epilepticus: Epidemiology and public health needs. J Clin Med 2016;5. pii: E71.
4. Kalita J, Nair PP, Misra UK. A clinical, radiological and outcome study of status epilepticus from India. J Neurol 2010;257:224-9.
5. Murthy JM, Jayalaxmi SS, Kanikannan MA. Convulsive status epilepticus: Clinical profile in a developing country. Epilepsia 2007;48:2217-23.
6. Bhalla A, Das B, Som R, Prabhakar S, Kharbanda PS. Status epilepticus: Our experience in a tertiary care centre in Northwestern India. J Emerg Trauma Shock 2014;7:9-13.
7. Kalita J, Misra UK, Patel R. Initial EEG in status epilepticus is helpful in predicting seizure recurrence. Electromyogr Clin Neurophysiol 2006;46:139-44.
8. Rossetti AO, Hurwitz S, Logroscino G, Bromfield EB. Prognosis of status epilepticus: Role of aetiology, age, and consciousness impairment at presentation. J Neurol Neurosurg Psychiatry 2006;77:611-5.
9. Rossetti AO, Logroscino G, Milligan TA, Michaelides C, Ruffieux C, Bromfield EB, *et al.* Status epilepticus severity score (STESS): A tool to orient early treatment strategy. J Neurol 2008;255:1561-6.
10. Leitinger M, Höller Y, Kalss G, Rohracher A, Novak HF, Höfler J, *et al.* Epidemiology-based mortality score in status epilepticus (EMSE). Neurocrit Care 2015;22:273-82.
11. DeLorenzo RJ, Pellock JM, Towne AR, Boggs JG. Epidemiology of status epilepticus. J Clin Neurophysiol 1995;12:316-25.
12. Yeung M, Fung F, Wong YK, Chang R, Mak W, Hon S, *et al*. Prognosis after status epilepticus (P07.175). Neurology 2013;12:80.
13. Leitinger M, Kalss G, Rohracher A, Pilz G, Novak H, Höfler J, *et al.* Predicting outcome of status epilepticus. Epilepsy Behav 2015;49:126-30.
14. Gao Q, Ou-Yang TP, Sun XL, Yang F, Wu C, Kang T, *et al.* Prediction of functional outcome in patients with convulsive status epilepticus: The END-IT score. Crit Care 2016;20:46.
15. Atmaca MM, Bebek N, Baykan B, Gökyiğit A, Gürses C. Predictors of

outcomes and refractoriness in status epilepticus: A prospective study. Epilepsy Behav 2017;75:158-64.
16. Pacha MS, Orellana L, Silva E, Ernst G, Pantiu F, Quiroga Narvaez J, *et al.* Role of EMSE and STESS scores in the outcome evaluation of status epilepticus. Epilepsy Behav 2016;64:140-2.
17. Mundlamuri RC, Sinha S, Subbakrishna DK, Prathyusha PV, Nagappa M, Bindu PS, *et al.* Management of generalised convulsive status epilepticus (SE): A prospective randomised controlled study of combined treatment with intravenous lorazepam with either phenytoin, sodium valproate or levetiracetam – Pilot study. Epilepsy Res 2015;114:52-8.
18. Medcalc. Available from: https://www.medcalc.org/calc/comparison_of_means.php. [Last accessed on 2017 Dec 05].
19. Available from: http://www.graphpad.com/quickcalcs/ttest1/. [Last accessed on 2017 Dec 05].
20. Power KN, Gramstad A, Gilhus NE, Engelsen BA. Prognostic factors of status epilepticus in adults. Epileptic Disord 2016;18:297-304.
21. Tsai MH, Chuang YC, Chang HW, Chang WN, Lai SL, Huang CR, *et al.* Factors predictive of outcome in patients with *de novo* status epilepticus. QJM 2009;102:57-62.
22. DeLorenzo RJ, Hauser WA, Towne AR, Boggs JG, Pellock JM, Penberthy L, *et al.* A prospective, population-based epidemiologic study of status epilepticus in Richmond, Virginia. Neurology 1996;46:1029-35.
23. Novy J, Logroscino G, Rossetti AO. Refractory status epilepticus: A prospective observational study. Epilepsia 2010;51:251-6.
24. Drislane FW, Blum AS, Lopez MR, Gautam S, Schomer DL. Duration of refractory status epilepticus and outcome: Loss of prognostic utility after several hours. Epilepsia 2009;50:1566-71.
25. Hassan H, Rajiv KR, Menon R, Menon D, Nair M, Radhakrishnan A, *et al.* An audit of the predictors of outcome in status epilepticus from a resource-poor country: A comparison with developed countries. Epileptic Disord 2016;18:163-72.
26. Towne AR, Pellock JM, Ko D, DeLorenzo RJ. Determinants of mortality in status epilepticus. Epilepsia 1994;35:27-34.
27. Shrovon S. Status Epilepticus: Its Clinical Features and Treatment in children and Adults. Cambridge University Press; 2006. p. 55.
28. Neligan A, Shorvon SD. Prognostic factors, morbidity and mortality in tonic-clonic status epilepticus: A review. Epilepsy Res 2011;93:1-0.
29. Mazarati AM, Baldwin RA, Sankar R, Wasterlain CG. Time-dependent decrease in the effectiveness of antiepileptic drugs during the course of self-sustaining status epilepticus. Brain Res 1998;814:179-85.