RESEARCH PAPER

Unraveling the mystery of status epilepticus: a retrospective study at a tertiary care centre from India

[1]Dr.Shweta Mallikarjun Revannavar [2] Dr.Aralikatte Onkarappa Saroja [3] Dr.Karkal Ravishankar Naik

[1]Senior Resident, Department of Neurology, Jawaharlal Nehru Medical college and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi, Karnataka, India , Email: shweta.connect2@gmail.com

[2]Professor and Head, Department of Neurology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi, Karnataka, India, Email: sarojaao2002@yahoo.com

[3]Professor, Department of Neurology, Jawaharlal Nehru Medical College and KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belagavi, Karnataka, India

Received: 26-07-2026; Revised: 10-08-2026; Accepted: 01-09-2026; Available Online: 05-09-2026

ABSTRACT

Introduction: Status epilepticus (SE) is a neurological emergency associated with substantial morbidity and mortality. Data from tertiary care centres in low- and middle-income settings remain limited, and the heterogeneity of aetiology, presentation, treatment response, and outcome continues to obscure clear prognostic patterns.1,2 This study aimed to characterise the demographic, clinical, biochemical, electrographic, and therapeutic profile of patients admitted with SE, and to identify factors associated with poor functional outcome.

Methods: We conducted a retrospective observational study of 100 consecutive adult patients admitted with SE to a tertiary care hospital in India. Demographics, clinical features, laboratory parameters, imaging, electroencephalography (EEG), antiseizure medication (ASM) use, and Status Epilepticus Severity Score (STESS) were recorded. Outcome was assessed using the modified Rankin Scale (mRS) at discharge and dichotomised as good (mRS 0–2) or poor (mRS 3–6). Continuous variables were compared using Welch's t-test and categorical variables using the chi-square test; $p < 0.05$ was considered significant.

Results: The mean age was 48.9 ± 19.2 years; 70% were male. Generalised convulsive SE was the predominant type (65%). Acute symptomatic aetiology (34%) was the most common category, with cerebrovascular events (cerebral venous thrombosis 14%, stroke 2%) and CNS infections being frequent. The mean GCS at presentation was 7.1 ± 3.2; 60% required intubation. Mean STESS was 1.8 ± 0.8, with 84% in the favourable STESS group (≤3). Polypharmacy (>2 ASMs) was required in 80%, and 48% received midazolam infusion. Poor functional outcome (mRS 3–6) occurred in 64% of patients. Age >65 years ($p = 0.024$), absence of prior epilepsy ($p = 0.036$), need for intubation ($p = 0.017$), lower haemoglobin ($p = 0.009$), and lower serum potassium ($p = 0.035$) were significantly associated with poor outcome.

Conclusion: SE in this cohort carried a high burden of poor functional outcome, driven largely by advanced age, acute symptomatic aetiologies, depressed sensorium requiring airway support, and biochemical derangements. De novo SE without prior epilepsy was particularly ominous. Early recognition, aggressive treatment of underlying aetiology, and correction of metabolic abnormalities are essential to improving outcomes in resource-limited tertiary care settings.

Keywords: Status epilepticus; STESS; modified Rankin Scale; refractory SE; tertiary care; India.

How to cite this article: Dr.Shweta Mallikarjun Revannavar , Dr.Aralikatte Onkarappa Saroja , Dr.Karkal Ravishankar Naik. Unraveling the mystery of status epilepticus: a retrospective study at a tertiary care centre from India. Int J Drug Deliv Technol. 2026;16(80s):1044-1049. DOI: 10.25258/ijddt.16.80s.133.

INTRODUCTION

Status epilepticus (SE) is one of the most serious neurological emergencies. The conceptual definition for status epilepticus was based on seminal work by Meldrum et al, on animal models which suggested that 82 min or longer of ongoing seizure activity in baboons can lead to irreversible neuronal injury due to excitotoxicity. But the limited knowledge of pathophysiology of status epilepticus led to the existence of a conceptual and an operational definition. The conceptual definition of convulsive status epilepticus in humans older than five years was defined as continuous seizure activity lasting more than 5 minutes or two or more discrete seizures between which there is incomplete recovery of consciousness. The International League Against Epilepsy operational definition recognises two time points: t1, beyond which seizure is considered abnormally prolonged (5 minutes for convulsive SE), and t2, beyond which long-term consequences (neuronal injury, network alteration, and death) are expected to occur (30 minutes for convulsive SE). For focal status epilepticus with impaired consciousness, t1 is 10 minutes and t2 is greater than 60 minutes and for absence status epilepticus, t1 is 10 – 15 min while t2 is undefined.[1]

The aetiological spectrum of SE is diverse — encompassing acute symptomatic causes such as stroke, central nervous system (CNS) infection, metabolic derangement, traumatic

brain injury, and drug withdrawal; remote symptomatic causes such as prior cerebrovascular accidents or posttraumatic injury; progressive symptomatic causes include neurodegenerative disorders and CNS tumours and idiopathic or cryptogenic causes. The relative frequencies of these categories differ across geographies, healthcare systems, and age groups. It is associated with significant short-term mortality (10–30%) and long-term neurological disability.[2]

Prognostic stratification in SE relies on validated tools, most notably the Status Epilepticus Severity Score (STESS), which incorporates age, level of consciousness, worst seizure type, and history of prior seizures. While STESS has been validated in several Western cohorts, its performance in South Asian populations, where the epidemiology of CNS infections, cerebral venous thrombosis, and access to critical care differs substantially warrants continued evaluation.[3]

Despite advances in pharmacotherapy and neurocritical care, refractory and super-refractory SE remain associated with high morbidity and mortality. Anaesthetic agents such as midazolam infusion are commonly required, and polypharmacy is the rule rather than the exception. The present study aims to unravel the clinical, biochemical, electrographic, therapeutic, and prognostic landscape of SE in a 100-patient population presenting to a tertiary care hospital in India and to identify factors associated with poor outcome.

METHODOLOGY

Study design and setting

This was a retrospective, single-centre, observational study conducted in the Department of Neurology of a tertiary care teaching and referral hospital in India.

Study population

One hundred consecutive adult patients (≥17 years) admitted over a period of 6.5 years (January 2019 to June 2025) to the department of Neurology with a diagnosis of SE were included. Patients with incomplete records or those who were declared brought-dead were excluded.

Data collection

Demographic data (age, sex), clinical history (duration of SE prior to presentation, history of prior epilepsy, drug withdrawal, comorbidities, addiction history), vital parameters (pulse, blood pressure, Glasgow Coma Scale [GCS], need for intubation), and laboratory investigations (haemoglobin, total leucocyte count, platelet count, blood urea, serum creatinine, sodium, potassium, total bilirubin, albumin, SGOT, SGPT, INR) were extracted from inpatient records. Electroencephalography (EEG), neuroimaging (CT/MRI), cerebrospinal fluid (CSF) analysis when performed were documented.

Treatment details included first-line, second-line, and third-line antiseizure medications (ASMs), use of short-acting benzodiazepines, polypharmacy (defined as >2 ASMs), midazolam infusion (with duration), other anaesthetic agents, complications, and total duration of hospital stay.

Severity assessment and outcome measures

The STESS score was calculated for each patient, incorporating age (≥65 years), worst seizure type (generalised convulsive vs non-convulsive vs simple partial/complex partial), level of consciousness (alert/somnolent/confused vs stuporous/comatose), and history of prior seizures. STESS ≤3 was considered favourable and ≥4 unfavourable. The primary outcome was functional status at discharge assessed using the modified Rankin Scale (mRS), dichotomised as good (mRS 0–2) and poor (mRS 3–6, with 6 denoting death).

Statistical analysis

Continuous variables are reported as mean ± standard deviation or median (interquartile range) and were compared between good and poor outcome groups using Welch's t-test. Categorical variables are reported as frequencies (percentages) and compared using the chi-square test. A two-sided p-value < 0.05 was considered statistically significant. Analyses were performed using standard statistical methods

RESULTS

Baseline demographic and clinical characteristics

A total of 100 patients with status epilepticus were analysed. The mean age was 48.9 ± 19.2 years (range 17–93 years; median 49 years). Majority of the population was male (70%). Twenty-four percent of patients were aged ≥65 years. According to onset of status epilepticus, generalised convulsive (65%) was the most common semiology, followed by focal onset while one had myoclonic status epilepticus. A history of prior seizures was present in 34% of patients, whereas 66% presented with de novo status epilepticus.

The sensorium at admission was assessed by Glasgow Coma Scale (mean 7.1 ± 3.2), indicating severe encephalopathy in the majority at presentation to the hospital. Sixty percent of the study population required endotracheal intubation and mechanical ventilation. The mean hospital stay was 12.4 ± 10.9 days (median 8.5 days).

Demographic data	N = 100	Percentage
Age Mean ± SD (in years) Median (range)	 48.9 ± 19.2 49 (17 – 93 years)	
Sex Male Female	 70 30	 70 % 30 %

Aetiology of SE		
Acute symptomatic	34	34 %
Progressive symptomatic	9	9 %
Remote symptomatic	26	26 %
Idiopathic/ Cryptogenic	24	24 %
Epilepsy syndrome	7	7 %
Type of SE at onset		
Generalised	64	64 %
Focal	35	35 %
Myoclonic	1	1 %
Sensorium (GCS) at admission		
Mild (13-15)	9	9 %
Moderate (9-12)	16	16 %
Severe (3-8)	75	75 %
Past history of seizures		
Yes	34	34 %
No	66	66 %
Triggering factor for SE		
None	53	53 %
Febrile illness	9	9 %
Drug default	12	12 %
Alcohol binge/withdrawal	2	2 %
Acute symptomatic		
Cerebral venous thrombosis	14	14 %
Stroke (hemorrhagic)	2	2 %
CNS infection	9	9 %
Metabolic encephalopathy	2	2 %
Autoimmune	3	3 %
Toxin	1	1 %
Intubataion required		
Yes	60	60 %
No	40	40 %
Duration of hospital stay		
Mean	8.5 days	

Table 1. Baseline demographic and clinical characteristics.

Aetiological spectrum

On classifying according to aetiology of status epilepticus, acute symptomatic causes were most frequent (34%), followed by remote symptomatic (26%), cryptogenic or unknown (24%), progressive symptomatic (9%), and defined epilepsy syndromes (7%). Among symptomatic SE (acute or remote), cerebral venous thrombosis (14%) and ischaemic/haemorrhagic stroke (14%) together accounted for over a quarter of all cases. CNS infections including viral encephalitis (9%) and prion disease (1%) constituted a minority of the cases. Autoimmune encephalitis (LGI-1 positive) made up 3% of population, post-traumatic (3%), brain tumors (4%) and metabolic causes (uraemic, hepatic, hypoglycemic) made up even smaller fractions. Among the patients with history of prior epilepsy (mean age 42.65 years), missing the drugs or drug withdrawal accounted for the majority of cases.

Biochemical and electrographic profile

Mean haemoglobin was 12.7 ± 2.3 g/dL, total leucocyte count 13.6 ± 4.8 × 10^3/µL, blood urea 29.4 ± 19.2 mg/dL, serum creatinine 1.0 ± 0.7 mg/dL, sodium 137.6 ± 6.5 mEq/L, and potassium 4.1 ± 0.6 mEq/L. Hepatic enzymes showed substantial variability, with a small number of patients (7%) demonstrating markedly elevated transaminases attributable to underlying hepatic dysfunction or hypoxic injury. Electroencephalography was done in 77% of the study population and it ranged from normal background to frank electrographic seizures. It was categorised into benign EEG (having normal findings or generalised or focal slowing) and malignant EEG (having spikes or sharp waves or periodic discharges or interictal discharges or electrographic

seizures). Among our study population, benign EEG was seen in 25 out of 77 cases (32.4 %) and malignant EEG pattern was seen in the remaining (67.5%). Neuroimaging (CT/MRI) was normal in 26% of cases while it was abnormal in the majority (74%), demonstrating findings consistent with the underlying aetiology.

Treatment and complications

All patients received short-acting benzodiazepines on arrival to emergency room (figure 1). Midazolam (90%) followed by lorazepam (9%) was the most frequently used benzodiazepine. Levetiracetam (83%), phenytoin (9%) and sodium valproate (7%) were the principal first-line antiseizure medications. Polypharmacy (greater than two ASMs) was required in 80 patients (80%) to control status epilepticus. Around 48 patients progressed to refractory status epilepticus. Midazolam infusion was used in all patients with refractory SE. In addition, anaesthetic agents (propofol and thiopentone in two each) were used in selected patients with super-refractory SE (4%). Non-pharmacological treatment included ketogenic diet, magnesium and high dose pyridoxine infusion. Among those on mechanical ventilation, the common complications noted were aspiration pneumonia (7%), ventilator-associated pneumonia (3%), sepsis (8%), myocardial infarction (1%) and hypotension (1%). The rate of mortality was 13% (mean age 53 years).

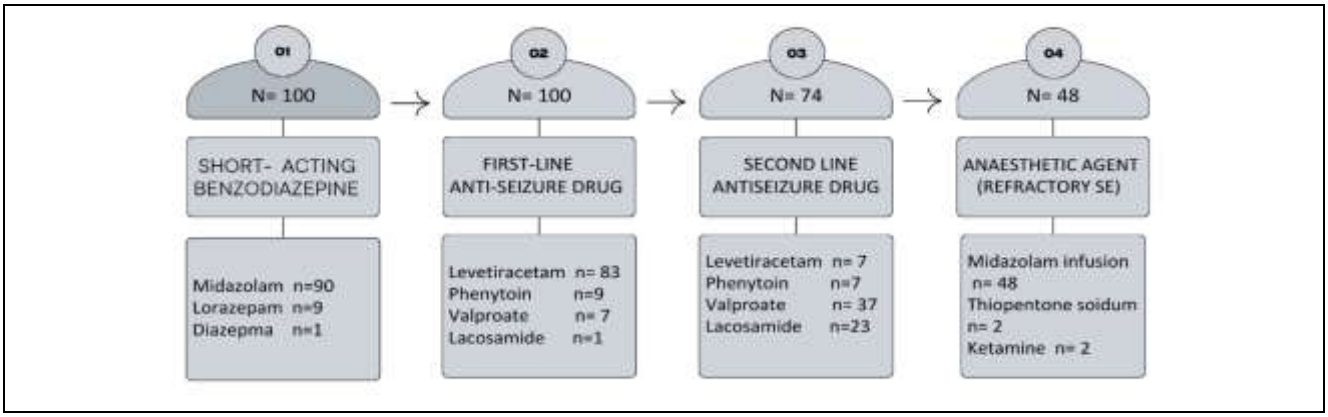

Fig 1: Treatment of status epilepticus

Outcome and predictors

At discharge, 36 patients (36%) had a good functional outcome (mRS 0–2) and 64 patients (64%) had a poor outcome (mRS 3–6, including in-hospital mortality). Table 2 compares baseline parameters between the good and poor outcome groups.

Variable	Good (n=36)	Poor (n=64)	p-value
Age, mean ± SD (years)	45.4 ± 15.4	50.9 ± 20.8	0.128
Age ≥ 65 years, n (%)	4 (11.1)	20 (31.3)	0.024*
Male sex, n (%)	26 (72.2)	44 (68.8)	0.716
Aetiology of SE Acute symptomatic Progressive symptomatic Remote symptomatic Idiopathic/ Cryptogenic Epilepsy syndrome	 25 4 14 18 3	 9 5 12 6 4	0.242
Past history of seizure De novo SE	17 47	17 19	0.036*
Sensorium Alert/ Drowsy (GCS 9-15) Stupor/Coma (GCS 3-8)	 48 16	 27 9	1.0
Intubation, n (%)	16 (44.4)	44 (68.8)	0.017*
STESS Favourable Unfavourable	 55 9	 29 7	0.481
Haemoglobin (g/dL)	13.4 ± 2.1	12.2 ± 2.3	0.090
Total leucocyte count (×10³/µL)	13.0 ± 4.2	13.9 ± 5.0	0.372

Variable	Good (n=36)	Poor (n=64)	p-value
Blood urea (mg/dL)	25.6 ± 14.6	31.6 ± 21.0	0.093
Serum creatinine (mg/dL)	0.87 ± 0.39	1.07 ± 0.83	0.092
Sodium (mEq/L)	138.2 ± 4.9	137.3 ± 7.3	0.426
Potassium (mEq/L)	4.25 ± 0.57	3.99 ± 0.62	0.059
EEG pattern Benign Malignant	 14 37	 11 15	0.188
Number of ASMs required to control SE 1 2 3 or more	 11 12 36	 11 13 17	0.385
Refractory SE, n (%)	14 (38.9)	34 (54.7)	0.129
Hospital stay (days)	12.5 ± 911.8	12.1 ± 9.4	0.85

Table 2. Comparison of baseline variables between good (mRS 0–2) and poor (mRS 4–6) outcome groups. *p < 0.05.

On univariate analysis, advanced age (≥65 years), de novo SE and requirement for intubation were significantly associated with poor functional outcome. Although mean GCS, STESS, and refractory status epilepticus were numerically worse in the poor outcome group, these did not reach statistical significance in our study.

DISCUSSION

This 100-patient retrospective cohort from a tertiary care hospital in South India provides a detailed snapshot of the clinical, aetiological, and prognostic landscape of status epilepticus. The outcome of SE reported all over the world is highly variable with a reported mortality rate of 5% to 56%.[4,5] The variability noted in demographic factors, aetiologies and treatment factors are responsible for the diversity noted in the outcome.

The study group was distinctly male-predominant (70%) and middle-aged (mean age 48.9 ± 19.2 years), with nearly one in four patients aged ≥65 years. Generalised convulsive status epilepticus (65%) remained the dominant semiology, consistent with previous reports from both Indian and international cohort.[2,4] The proportion of patients presenting with de novo status epilepticus (66%) was high, reflecting the referral nature of the centre and indicating that SE often heralds previously unrecognised neurological disease.

Among the acute symptomatic cases, cerebral venous thrombosis (14%) and acute stroke (2%) made a significant contribution which is generally a common cause in the developing world.[6-8] The high prevalence of cerebral venous thrombosis is noteworthy and aligns with regional epidemiological patterns in South Asia, where dehydration, peripartum states, prothrombotic conditions, and infections contribute disproportionately to its burden.[9] In contrast, cerebrovascular aetiology was predominantly seen in developed nations.[10] Infectious aetiology (viral encephalitis, tubercular meningitis) was seen in a minor population(9%) which remains an important contributor to SE in tropical settings.[11,12] Our study had a higher contribution from cryptogenic causes (24%).[9] The most common trigger factor for SE in our study was drug default (12%) considering the large burden of elderly population in our study.[13]

On presentation to the hospital, the mean GCS was 7.1 with 60% of the study population requiring intubation and mechanical ventilation. Among all, 64% showed a poor functional outcome (mRS 3-6) at discharge and a mortality rate of 13%.[14] Despite this severity, the mean STESS was modest (1.8) and 84% fell into the favourable STESS category. STESS did not show a significant discriminative value as an outcome measure in our study (p = 0.887).[15] This dissociation between STESS and outcome echoes prior observations in lower-middle-income settings, where the contribution of acute symptomatic aetiologies, delayed presentation, and limited pre-hospital care may outweigh the predictors captured by STESS.

The identified predictors of poor outcome such as advanced age, de novo status epilepticus and need for intubation and mechanical ventilation are biologically coherent. Lower haemoglobin showed a trend towards poor outcome however it was not statistically significant (p=0.090). Older patients have less cerebral reserve and higher comorbidity. De novo SE is more often acute

symptomatic, with the underlying aetiology (stroke, encephalitis, autoimmune) itself driving prognosis. Endotracheal intubation reflects depressed sensorium and poor airway protection which could contribute to higher rate of complications such as aspiration pneumonia and sepsis seen in 15% of our total study population. Lower haemoglobin may reduce cerebral oxygen delivery during prolonged seizure activity, may reflect acute systemic stress, gastrointestinal losses, or contribute to cardiac and neuromuscular instability. These findings support aggressive correction of anaemia as part of comprehensive SE management.

Out of the 77% study participants who underwent EEG, 52% had malignant pattern such as periodic discharges or electrographic seizures demonstrating the need for continuous

EEG monitoring in SE.[16] However the presence or absence of malignant EEG patterns did not significantly affect the outcome.[17,18]

All patient were treated with short acting benzodiazepines on arrival to emergency services to abort the on-going seizure followed by first line anti-seizure medication (ASM). The most commonly used first line ASM was levetiracetam and second line ASM was sodium valproate with a response rate of 26% each. The possible reason to initiate second line agent could be a delay in reaching the health care or inadequacy of available resources at home.[19] A large portion (48%) of study participants landed in refractory status epilepticus requiring polypharmacy and midazolam infusion.[20] Despite intensive pharmacotherapy, almost two-thirds of them had poor functional outcome, highlighting the limits of pharmacological control when the underlying aetiology is severe. This reinforces the principle that aetiology-directed treatment such as thrombolysis or anticoagulation for cerebrovascular causes, immunotherapy for autoimmune encephalitis, antimicrobials for CNS infections are at least as important as antiseizure pharmacotherapy.

Limitations of this study include its retrospective single-centre design, lack of long-term follow-up beyond discharge, absence of continuous EEG in many patients which may have underestimated non-convulsive SE, and the relatively modest sample for multivariable modelling. Despite these constraints, the study offers valuable real-world data from a setting under-represented in the global SE literature.

CONCLUSION

SE in this 100-patient tertiary care cohort from India was characterised by male predominance, a high burden of acute symptomatic aetiologies (cerebral venous thrombosis, stroke, CNS infections), severe encephalopathy at presentation, and a striking 64% rate of poor functional outcome at discharge. Advanced age, de novo presentation without prior epilepsy, need for intubation and lower haemoglobin emerged as significant predictors of poor outcome, whereas the conventional STESS did not discriminate prognosis in this cohort. The findings argue for early aetiological diagnosis, aggressive correction of metabolic and haematological derangements, and the development of context-adapted prognostic scores for SE in South Asian tertiary settings. Prospective multicentre studies with continuous EEG monitoring and longer-term follow-up are warranted to refine risk stratification and optimise outcomes.

REFERENCE

1. Trinka E, Cock H, Hesdorffer D, Rossetti AO, Scheffer IE, Shinnar S, et al. A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus. Epilepsia. 2015 Oct;56(10):1515–23. doi:10.1111/epi.13121
2. Trinka E, Kwan P, Lee B, Dash A. Epilepsy in Asia: Disease burden, management barriers, and challenges. Epilepsia. 2019 Mar;60(S1):7–21. doi:10.1111/epi.14458
3. Rossetti AO, Logroscino G, Milligan TA, Michaelides C, Ruffieux C, Bromfield EB. Status Epilepticus Severity Score (STESS): A tool to orient early treatment strategy. J Neurol. 2008 Oct;255(10):1561–6. doi:10.1007/s00415-008-0989-1
4. Cook AM, Castle A, Green A, Lesch C, Morrison C, Rhoney D, et al. Practice Variations in the Management of Status Epilepticus. Neurocrit Care. 2012 Aug;17(1):24–30. doi:10.1007/s12028-012-9711-3
5. 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 Jan 1;102(1):57–62. doi:10.1093/qjmed/hcn149
6. Holtkamp M. Predictors and prognosis of refractory status epilepticus treated in a neurological intensive care unit. Journal of Neurology, Neurosurgery & Psychiatry. 2005 Apr 1;76(4):534–9. doi:10.1136/jnnp.2004.041947
7. Wu YW, Shek DW, Garcia PA, Zhao S, Johnston SC. Incidence and mortality of generalized convulsive status epilepticus in California. Neurology. 2002 Apr 9;58(7):1070–6. doi:10.1212/WNL.58.7.1070
8. Chen L, Zhou B, Li JM, Zhu Y, Wang JH, Sander JW, et al. Clinical features of convulsive status epilepticus: a study of 220 cases in western China. Euro J of Neurology. 2009 Apr;16(4):444–9. doi:10.1111/j.1468-1331.2008.02404.x
9. Murthy JMK, Jayalaxmi SS, Kanikannan MA. Convulsive Status Epilepticus: Clinical Profile in a Developing Country. Epilepsia. 2007 Dec;48(12):2217–23. doi:10.1111/j.1528-1167.2007.01214.x
10. Vignatelli L, Rinaldi R, Galeotti M, De Carolis P, D'Alessandro R. Epidemiology of status epilepticus in a rural area of northern Italy: a 2-year population-based study. Euro J of Neurology. 2005 Nov;12(11):897–902. doi:10.1111/j.1468-1331.2005.01073.x
11. Hassan H, Rajiv KR, Menon R, Menon D, Nair M, Radhakrishnan A. An audit of the predictors of outcome in status epilepticus from a resource-poor country: a comparison with developed countries. Epileptic Disorders. 2016 Jun;18(2):163–72. doi:10.1684/epd.2016.0832
12. Koubeissi M, Alshekhlee A. In-hospital mortality of generalized convulsive status epilepticus: A large US sample. Neurology. 2007 Aug 28;69(9):886–93. doi:10.1212/01.wnl.0000269791.96189.70
13. Scott RA, Lhatoo SD. The treatment of epilepsy in developing countries: where do we go from here? Policy and Practice.
14. Chin RFM, Neville BGR, Scott RC. A systematic review of the epidemiology of status epilepticus. Euro J of Neurology. 2004 Dec;11(12):800–10. doi:10.1111/j.1468-1331.2004.00943.x
15. Rossetti AO, Logroscino G, Bromfield EB. A clinical score for prognosis of status epilepticus in adults. Neurology. 2006 Jun 13;66(11):1736–8. doi:10.1212/01.wnl.0000223352.71621.97
16. Jaitly R, Sgro JA, Towne AR, Ko D, DeLorenzo RJ. Prognostic Value of EEG Monitoring After Status Epilepticus: A Prospective Adult Study: Journal of Clinical Neurophysiology. 1997 Jul;14(4):326–34. doi:10.1097/00004691-199707000-00005
17. Neligan A, Shorvon SD. Prognostic factors, morbidity and mortality in tonic–clonic status epilepticus: A review. Epilepsy Research. 2011 Jan;93(1):1–10. doi:10.1016/j.eplepsyres.2010.09.003
18. Firosh Khan S, Ashalatha R, Thomas SV, Sarma PS. Emergent EEG is helpful in neurology critical care practice. Clinical Neurophysiology. 2005 Oct;116(10):2454–9. doi:10.1016/j.clinph.2005.06.024
19. Caraballo R, Fejerman N. Management of epilepsy in resource-limited settings. Epileptic Disorders. 2015 Mar;17(1):13–8. doi:10.1684/epd.2014.0721
20. Jagoda A, Riggio S. Refractory status epilepticus in adults. Annals of Emergency Medicine. 1993 Aug;22(8):1337–48. doi:10.1016/S0196-0644(05)80120-9