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BRIEF COMMUNICATION

Association of early general anesthesia with outcome in adults with status epilepticus: A propensity-matched observational study

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Abstract

General anesthesia (GA) earlier than recommended (as first- or second-line treatment) was recently described to improve status epilepticus (SE) outcome. We aimed to assess the impact of early GA on outcome in matched groups. Data from a multicenter, prospective cohort of 1179 SE episodes in 1049 adults were retrospectively analyzed. Incident SE episodes were categorized as "early anesthesia" (eGA; GA as first- or second-line treatment) or "non-early anesthesia" (neGA; GA after second-line treatment or not at all). Using propensity score matching, eGA episodes were paired 1:4 with neGA episodes. We assessed survival, functional outcomes at discharge (good: modified Rankin Scale = 0–2 or no worsening),

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SE cessation rate, SE duration, and hospital stay. Among 1049 SE episodes, 55 (5.2%) received eGA, and 994 constituted the neGA group; 220 represented the matched controls. Patients receiving eGA were younger (median = 63, interquartile range [IQR] = 56–76 vs. median = 70, IQR = 54–80 years, $p = .004$), had deeper consciousness impairment (80% vs. 40% stuporous/comatose, $p < .001$), and had more severe SE forms (89% vs. 54% generalized convulsive SE/nonconvulsive SE in coma, $p < .001$). Mortality, functional outcome, SE cessation rate, and duration of SE and hospital stay were similar between the eGA group and matched controls. We conclude that early anesthesia for SE treatment did not influence prognosis.

KEYWORDS

antiseizure medication, intubation, mortality, refractory status epilepticus, seizure

1 | INTRODUCTION

Status epilepticus (SE) bears significant morbidity and mortality.[1] International treatment guidelines recommend starting with benzodiazepines, followed by intravenous antiseizure medications (ASMs)[2]; third-line treatment with general anesthesia (GA) is advocated for persisting SE, called refractory SE (RSE).[2] Beyond first- and second-line treatments, evidence is mainly based on expert opinions, and GA administration and its timing are debated[3–5]; on the other hand, insufficient or delayed treatment is linked to poor prognosis.[6,7] Furthermore, a recent study suggested that anesthesia as first- or second-line treatment (hence not complying with current recommendations[2]) correlates with better outcome compared to later anesthesia.[8] However, nonanesthetized patients and SE refractoriness were not considered.[3,9]

We aimed to compare outcomes of SE patients receiving GA as first- or second-line treatment versus controls not receiving early anesthesia.

2 | MATERIALS AND METHODS

We conducted a retrospective analysis of data from the large, multicenter, prospective, observational Sustained Effort Network for Treatment of Status Epilepticus (SENSE) cohort,[6] which received ethical approval at all centers (DRKS00000725). Informed consent was waived due to the purely observational nature and the complete anonymization of data, except for Innsbruck, which obtained consent from all patients.

The detailed SENSE study protocol and main results have been published.[6,10] In summary, data on adults experiencing SE were collected between 2011 and 2015 at nine European centers, excluding hypoxic–anoxic brain injury. SE resolution was assessed clinically, including electroencephalographic (EEG) information.[6] Semiology was characterized as nonconvulsive SE (NCSE), generalized convulsive SE (GCSE; including focal to bilateral and generalized onset), or any other, including but not restricted to focal (motor or nonmotor) SE, absence SE, and myoclonic SE. Etiology was reported according to the International League Against Epilepsy criteria, categorized as (1) acute, including purely acute and acute on remote; and (2) nonacute, including remote, progressive, and unknown/other etiologies.[1,4] Level of consciousness before treatment was dichotomized as alert/somnolent versus coma/stupor. The Status Epilepticus Severity Score was calculated on admission.[11] SE treatment protocol was defined by each center in accordance with the German, Austrian, and Swiss neurologic society recommendations at that time.[12] Treatment in accordance with guidelines was defined as at least 1 mg clonazepam, 10 mg diazepam, 4 mg lorazepam, or 10 mg midazolam as first line, and at least 400 mg lacosamide, 30 mg/kg levetiracetam, 17 mg/kg phenobarbital, 17 mg/kg phenytoin, or 20 mg/kg valproate as second line according to the ESETT trial and the recommendations of German, Austrian, and Swiss neurologic societies.[12,13]

Early GA (eGA) was defined as GA prescribed as first or second line, regardless of indication (SE treatment, airway protection[4,14]), whereas patients never receiving GA or getting GA after the second line were defined as non-eGA (neGA). eGA was administered at clinicians' discretion. Mortality and functional outcome were prospectively assessed at discharge. The modified Rankin Scale (mRS) before SE was prospectively estimated; mRS = 0–2 or lack of mRS worsening were considered to be good functional outcomes.[14]

Comparison of categorical data was performed through chi-squared or Fisher exact tests, and ordinal or

continuous data were analyzed using Mann–Whitney or *t*-tests, as appropriate. To assess the eGA impact on outcome, we performed a propensity score matching using a logistic regression based on sex, age, acute etiology, level of consciousness before treatment dichotomized as stuporous/comatose versus alert/somnolent, SE semiology classified as GCSE/NCSE in coma versus any other, and previous seizure occurrence. Size effect was assessed using Cramér's phi. The association strength was interpreted based on standard thresholds: negligible ($\varphi < .1$), moderate ($.1 \leq \varphi < .3$), strong ($.3 \leq \varphi < .5$), and very strong ($\geq .5$). eGA patients were matched 1:4 with neGA patients with the nearest neighbor method within caliper bounds of ±.2. Logistic regression regarding mortality and functional outcome were subsequently conducted as sensitivity (exploratory) analyses. Statistics were performed using IBM SPSS software (v27) and R (v4.0.0, R Foundation).

3 | RESULTS

SENSE comprised 1049 patients experiencing 1179 SE episodes. For the present analysis, only the first 1049 SE episodes were considered. Treatment was initiated outside the hospital in 371 (35.4%), and 55 (5.2%) patients received eGA. Recruitment was inhomogeneous among centers; however, similar eGA rates ($p = .7$) were observed (Marburg 13/172 [7.6%], Kiel 2/81 [2.5%], Osnabrück 9/217 [4.1%], Basel 0/7 [0%], Regensburg 0/7 [0%], Lausanne 18/351 [5.1%], Salzburg 12/186 [6.5%], Innsbruck 1/28 [3.6%]). The seven patients from Basel were not included in a later study.[8]

Demographics and outcome characteristics of eGA versus neGA are presented in Table 1. eGA patients had deeper consciousness impairment and more severe SE semiology. Age decades were comparable. Latency to first SE treatment (GA in the eGA group or ASM/benzodiazepine in the neGA group) was similar. Although SE refractoriness was, by definition, impossible to formally assess in the eGA group, 545 of 994 (54.7%) neGA patients developed RSE, of whom 57 (19.9%) received GA (all were intubated). Mortality was similar between eGA (12.7%, 95% confidence interval [CI] = .05–.24) and neGA patients (15.1%, 95% CI = .13–.17; $p = .8$). Among RSE patients, mortality increased to 19.2% (95% CI = .15–.24) for those treated without GA, and 27.3% (95% CI = .19–.37) for those receiving GA after the second line.

Propensity matching achieved a good balance between eGA and control groups, with all standardized mean differences less than .1 (Figure S1, Table 2). Additional clinical variables that could influence outcomes but were not part of the propensity matching score, such as baseline mRS or delay before SE treatment, were similarly distributed across groups (Table 2). Deviations from guidelines occurred in matched controls; the majority (89%) received benzodiazepines as first treatment, but only 20 patients (9.2%) at proper doses. SE ceased after the first treatment in 39 (17.7%) of the controls; among the 181 patients who received a second SE treatment, 68.5% received intravenous ASM, but only 26.5% had it administered at recommended doses.

All measured outcomes were comparable between the eGA group and matched controls (Table 2). Exploratory logistic regression confirmed that mortality and functional outcome were not related to GA timing (Table S1).

4 | DISCUSSION

This study analyzed, in a large, multicenter prospective cohort, the impact of GA timing on SE prognosis. All outcome measures were similar between patients receiving eGA and controls.

Mortality, functional outcome, and length of SE or hospital stay were comparable across groups, contradicting a recent study reporting better functional outcome, shorter SE duration, and fewer infections in patients with eGA.[8] A selection bias might explain this discrepancy, as in the aforementioned study[8] eGA patients were only compared with those receiving GA later (fulfilling RSE diagnosis, a known factor for poor outcome[3,14,15]), and unlike the present study, leaving out patients not treated with GA. A significant proportion of patients may receive eGA after a single seizure in the postictal phase for nonepileptic events or for treatment-responsive SE.[16] The hypothesis that several eGA patients might have less aggressive SE (which would have likely ceased after early nonsedating treatments according to current recommendations[2]) seems supported by the lower mortality rate (12.7%) among eGA patients compared to the ones with RSE (21.5% in the SENSE cohort).[4] Remarkably, our mortality rates (12%–15%) were strikingly similar to the previously mentioned cohort,[8] supporting the need for adequately controlling potential confounders. In investigating the efficacy of a treatment, the choice of the control group is of prime importance. As far as eGA is concerned, three control groups might have been considered: (1) patients receiving GA later as done previously,[17] introducing a potential bias toward favorable outcomes in the eGA group; (2) patients not treated with GA, which we felt could have introduced a bias toward more severe outcome in the eGA group; and (3) patients receiving later GA or never receiving GA, which we chose to mitigate these biases. Deviation from guidelines, mainly consisting of underdosing, occurred frequently in matched controls, which may have

TABLE 1 Demographic characteristics and outcomes in patients treated with eGA and in patients receiving general anesthesia after the second treatment steps or not anesthetized (neGA).

Characteristic	eGA	neGA	OR (95% CI)	*p*
n (%)	55 (5.2)	994 (94.8)		
Age, years, median (IQR)	66.0 (56–76)	66.5 (53–80)		**.037**
Age > 65 years, *n* (%)	32 (58.2)	606 (61.0)	.9 (0.5–1.5)	.68
Female sex, *n* (%)	23 (41.8)	514 (51.7)	.7 (.4–1.2)	.167
Good [0–2] baseline mRS, *n* (%)	20 (36.6)	303 (30.5)	1.3 (.7–2.4)	.34
Seizure semiology, *n* (%)				
GCSE	28 (50.9)	314 (31.6)		**<.001**
NCSE in coma	21 (38.2)	224 (22.5)		
Other	6 (10.9)	456 (45.9)		
Level of consciousness before treatment, *n* (%)				
Alert/somnolent	11 (20.0)	599 (60.3)	6.1 (3.0–13.2)	**<.001**
Stuporous/comatose	44 (80.0)	395 (39.7)		
Previous seizures, *n* (%)	25 (45.5)	491 (49.4)	.9 (9.5–1.5)	.583
Known epilepsy, *n* (%)	21 (28.2)	464 (46.7)	.7 (.4–1.3)	.27
Acute etiology, *n* (%)	26 (47.3)	404 (40.6)	1.3 (.7–2.3)	.329
Latency SE onset to 1st treatment, min, median (IQR)	42.5 (20–121)[a]	70 (25–250)[b]		.35
Mortality, *n* (%)	7 (12.7)	150 (15.1)	.8 (.3–1.9)	.846
Mortality in SE, *n* (%)	3 (5.5)	56 (5.6)	1.4 (.03–9.5)	.537
Good functional outcome: mRS = 0–2 or delta mRS = 0 at discharge, *n* (%)	30 (54.6)	626 (63.0)	.7 (.4–1.3)	.252
SE termination, *n* (%)	52.0 (94.6)	927.0 (93.3)	1.3 (.4–6.4)	.925
SE duration, h, median (IQR)	7.0 (1–119)[c]	8.0 (2–48)[d]		.961
Hospitalization duration, days, median (IQR)	14.0 (5–25)	9.5 (4–18)[e]		.117

Note: Number of missing values: [a]7, [b]131, [c]9, [d]183, [e]12. Bold indicates statistical significance.

Abbreviations: CI, confidence interval; eGA, early general anesthesia; GCSE, generalized convulsive SE; IQR, interquartile range; mRS, modified Rankin Scale; NCSE, nonconvulsive SE; neGA, non-eGA; OR, odds ratio; SE, status epilepticus.

altered their outcome. However, some data suggest that lower doses might not automatically influence SE prognosis,[18] and in the SENSE cohort, inadequate first-line treatment was associated with SE refractoriness but not mortality.[4]

In our patients, eGA was more frequent in patients presenting with GCSE and NCSE in coma, in line with a previous report.[7] Clinicians might be more reluctant to expose patients without significant consciousness impairment to the risk of GA.

Overall, our findings suggest that eGA might not be beneficial in terms of duration of SE or hospital stay (which was nonsignificantly longer in the eGA group), mortality, and functional outcome. However, corroborating a previous study,[7] we did not find evidence regarding a potential harm of GA earlier than recommended.

Some limitations must be acknowledged. The numbers of included patients varied between centers, potentially affecting homogeneity. Controls often received inadequate first-line and second-line treatment, mainly with underdosing (additional inadequate treatment in eGA was not possible to assess, given the major deviation of the protocol through eGA). eGA was administered on clinicians' judgment; compounds, dosages, titration, and intubation times were neither standardized nor recorded. We can therefore not exclude a dose-dependent medication effect, with possibly different doses used for SE treatment or airway protection. Due to the post hoc design of this analysis, and the frequent occurrence of intubation outside the hospital, before EEG or neurological evaluation, detailed information for GA, such as airway protection, agitation, or RSE treatment, could not be sorted out reliably (as it was not documented). We therefore chose not to consider this aspect.[14] Of note, however, it has recently been shown that as few as 16% of patients receiving GA for GSE were actually in RSE.[16] Due to the design of the study, we cannot rule out that some factors, not accounted for in the propensity matching score, could have influenced outcome; however, major confounders were symmetrically distributed

TABLE 2 Comparison of patients receiving eGA versus matched controls (receiving general anesthesia after the second treatment steps or not anesthetized).

Characteristic	eGA	Matched controls	OR (95% CI)	*p*
Total *n*	55	220		
Matching criteria				
Age, years, median (IQR)	66.0 (56–76)	64.2 (18.7)		.790
Female sex, *n* (%)	23 (41.8)	97 (44.1)	.9 (.5–1.7)	.879
Level of consciousness before treatment, *n* (%)				
Alert/somnolent	11 (20.0)	44 (20.0)	1 (.5–2.3)	1.000
Stuporous/comatose	44 (80.0)	176 (80.0)		
Seizure semiology, *n* (%)				
GCSE	28 (50.9)	122 (55.4)		.780
NCSE in coma	21 (38.2)	74 (34.0)		
Other	6 (11.0)	24 (10.9)		
Previous seizures, *n* (%)	26 (47.3)	108 (49.0)	.9 (.5–1.8)	.880
Acute etiology, *n* (%)	25 (46.0)	102 (46.8)	.9 (.5–1.8)	.881
Clinical variables, *n* (%)				
Good [0–2] baseline mRS	20 (36.6)	56 (25.5)	1.7 (.8–3.3)	.129
Known epilepsy	21 (38.2)	96 (43.6)	.8 (.4–1.5)	.54
Latency SE onset to first treatment (min), median (IQR)	42.5 (20–121)[a]	50.0 (20–150)[b]		.66
Received GA for SE treatment, *n* (%)	55 (100.0)	38 (17.3)		
Outcomes, *n* (%)				
Mortality	7 (12.7)	27 (12.3)	1 (.4–2.6)	1.00
Mortality in SE	3 (5.5)	15 (6.8)	1 (.02–7.9)	.884
Good functional outcome: mRS = 0–2 or delta mRS = 0 at discharge	30 (54.6)	139 (63.2)	.7 (.4–1.4)	.355
SE termination, *n* (%)	52 (94.6)	201 (91.4)	1.7 (.5–9.4)	.596
SE duration, h, median (IQR)	7 (1–119)[c]	6 (1–40)[d]		.128
Hospitalization duration, days, median (IQR)	14 (5–25)	8 (5–18)[e]		.151

Note: Number of missing values: [a]7, [b]18, [c]9, [d]33, [e]1.

Abbreviations: CI, confidence interval; eGA, early GA; GA, general anesthesia; GCSE, generalized convulsive SE; IQR, interquartile range; mRS, modified Rankin Scale; NCSE, nonconvulsive SE; OR, odds ratio; SE, status epilepticus.

across groups or were considered for matching. Some additional variables with potential impact on outcome (EEG, infections, previous SE, refractory epilepsy, or presence of advanced directives) were not available. Acute etiology covers a wide spectrum of causes, including relatively benign such as ASM withdrawal or metabolic disturbances, and more severe ones. Etiology is known to represent a main predictor of SE outcome, and adjustment/matching for "potentially fatal" etiology might have been relevant. Unfortunately, this information was not available. Finally, we considered the length of hospital stay, which admittedly can be influenced by social factors, but not intensive care unit (ICU) stay (as this cohort was not ICU limited).

In conclusion, early anesthetic treatment does not seem to exert a meaningful impact on SE prognosis after adequately controlling for potential confounders, therefore not supporting a routine use of anesthetics earlier than currently recommended.

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The authors have nothing to report.

CONFLICT OF INTEREST STATEMENT

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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