Epilepsy Research 202 (2024) 107343

Contents lists available at ScienceDirect

Epilepsy Research

journal homepage: www.elsevier.com/locate/epilepsyres

Status Epilepticus after mechanical thrombectomy: The role of early EEG assessment in Stroke Unit, clinical and radiological prognostication

Gabriele Prandin *, Giovanni Furlanis, Ilario Scali, Federica Palacino, Laura Mancinelli, Emanuele Vincis, Paola Caruso, Giulia Mazzon, Marinella Tomaselli, Marcello Naccarato, Paolo Manganotti

Clinical Unit of Neurology, Department of Medicine, Surgery and Health Sciences, University Hospital and Health Services of Trieste, ASUGI, University of Trieste, Trieste, Italy

ARTICLE INFO

Keywords:
Stroke
Status epilepticus
Endovascular thrombectomy

ABSTRACT

Background: Convulsive (CSE) and non-convulsive (NCSE) Status Epilepticus are a complication in 0.2–0.3% ischemic strokes. Large stroke and cortical involvement are the main risk factors for developing SE. This study evaluates the prevalence of SE in patients treated with endovascular thrombectomy (EVT) through EEG recording within 72- h from admission. Moreover, we compared clinical, radiological, and outcome measures in SE and no-SE patients.

Materials and Methods: We collected retrospectively demographical and clinical characteristics of acute ischemic stroke patients who underwent EVT, admitted in the Stroke Unit (SU) of the University Hospital of Trieste between January 2018 and March 2020 who underwent EEG recording within 72- h from the symptoms' onset.

Results: Out of 247 EVT patients, 138 met the inclusion criteria, of whom 9 (6.5%) showed SE with median onset time of 1 day (IQR 1–2). No difference was found between the two groups as for age, sex, risk factors, grade of recanalization, etiology of stroke, and closed vessel. The no-SE group presented higher NIHSS improvement rate ($p=0.025$) compared to the SE group. The sum of the lobes involved in the ischemic lesion was significantly higher in SE group ($p=0.048$).

Conclusion: SE after EVT in large strokes is a non-rare complication, with most being NCSE. Performing a rapid EEG assessment in a Stroke Unit setting may allow for a prompt recognition and treatment of SE in the acute/hyper-acute phase. SE may be correlated with worse clinical outcomes in patients with large vessel occlusion.

1. Introduction

Status Epilepticus (SE) may be a complication of an ischemic stroke. 0.2–0.3% of ischemic strokes are reported to possibly develop SE (Bateman et al., 2007; Sinka et al., 2023). SE is related to cortical infarction and large size stroke (Belcastro et al., 2014) and shows a complex pathophysiology involving inflammation, blood-brain barrier disruption, metabolic dysfunction and the release of excitotoxic neurotransmitters secondary to ischemic brain damage, with a prominent role of the penumbra area (Galovic et al., 2021; Heiss et al., 1992; Luhmann et al., 1995). Particularly, the effect of epileptic activity on the ischemic penumbral tissue results in a deleterious alteration of local metabolism, described in some works as "metabolic crisis" (Scoppettuolo et al., 2019). The perpetuation of the epileptiform activity and the development of Status Epilepticus may be a consequence of a failure of the seizure-terminating mechanisms (Sinka et al., 2023). Previous research focused on post-stroke SE either included patients with clinical symptoms or signs suggestive of convulsive (CSE) or non-convulsive Status Epilepticus (NCSE) (Sinka et al., 2023), or they have performed EEG monitoring within 7 days after the admission (Belcastro et al., 2014). The occurrence of NCSE after the ischemic event is reported in 3–4% of strokes (Belcastro et al., 2014). Looking at the effects of endovascular thrombectomy (EVT) in post-stroke seizures, studies and meta-analyses showed that this procedure does not increase the risk of acute symptomatic seizures (Alemany et al., 2021; Jiang et al., 2023). However, no data are available on SE prevalence after EVT. The aim of this study was

* Correspondence to: Clinical Unit of Neurology, Department of Medical Sciences, University Hospital and Health Services of Trieste, Strada di fiume, 447, Trieste 34149, Italy.
E-mail address: gprand94@gmail.com (G. Prandin).

https://doi.org/10.1016/j.eplepsyres.2024.107343
Received 8 January 2024; Received in revised form 28 February 2024; Accepted 10 March 2024
Available online 12 March 2024

to evaluate the prevalence of SE (NCSE and CSE) in large vessel occlusion (LVO) strokes treated with EVT by applying an EEG monitoring within 72- h from admission in Stroke Unit.

2. Material and Methods

We retrospectively analyzed clinical data of patients with acute ischemic stroke admitted to the Stroke Unit (SU) of the University Hospital of Trieste (Italy) between January 2018 and April 2020. The study was approved by the institutional review board. The patients included in the study showed acute focal neurologic symptoms compatible with ischemic stroke and received EVT with or without bridging thrombolytic treatment (rtPA 0.9 mg/Kg, max 90 mg). The diagnosis of ischemic stroke was confirmed by a neuroradiological and clinical examination, and eligibility criteria for treatment were chosen in accordance with the ESO (European Stroke Organization) guidelines (Berge et al., 2021; Turc et al., 2019). Included patients presented at the hospital within 4.5 hours from symptoms onset or presenting with a favorable core/penumbra mismatch at CT Perfusion evaluation (Caruso et al., 2018; Furlanis et al., 2020). In particular, we also included patients with extended LVO window according to the radiological findings (i.e. CT perfusion) or patients with unknown time of symptoms onset (wake-up strokes) with a penumbra area $>$ 50% of the total hypoperfused area. We excluded hemorrhagic strokes, ASPECTs (Alberta Stroke Program Early CT Score) $\leq$ 6, patients who were eligible but refused the reperfusion treatment, patients without an EEG recording within 72- h. We exclude patients with an EEG before 2 hours since the stop of sedation.

We collected demographic data such as age, sex, comorbidities as atrial fibrillation (AF), history of diabetes mellitus, arterial hypertension, hypercholesterolemia, history of coronary artery disease, history of epilepsy, anamnestic cognitive decline. Clinical features such as NIHSS (National Institutes of Health Stroke Scale) score and mRS (modified Rankin Scale) were collected at admission and at discharge; mRS and mortality rate at 3 months from the index event were recorded, too. The mRS at admission referred to the premorbid condition of the patient. Etiology of stroke (Trial of Org 1072 in Acute Stroke Treatment: TOAST classification) was evaluated and classified as large artery atherosclerosis (LAA), cardioembolic (CE), other cause, indeterminate/cryptogenic (Adams et al., 1993). Looking at the neuroimaging assessment, we collected data on the closed vessel (Middle cerebral artery with M1 or M2 tracts, internal carotid artery (ICA), posterior circulation vessel including Basilar artery or posterior cerebral artery P1 tract), the location of site lesion in the non-contrast CT (NCCT) performed 24- h after the treatment; we considered as estimation of stroke extension the global burden of involved lobes. Concerning endovascular treatment, we discriminated between direct thrombectomy and thrombectomy with previous rtPA infusion, and we evaluated the recanalisation rate with modified Thrombolysis in Cerebral Infarction (mTICI) score (successful recanalisation with mTICI 2b-3, unsuccessful treatment with mTICI 0–2a) (Anon, 2019). For all patients, we also evaluated the presence of hemorrhagic infarctions after treatment with NCCT (performed within 1- h from the end of the endovascular procedure) and after 24–36 h from the cerebrovascular event. We divided the hemorrhagic infarctions into symptomatic and asymptomatic in case of clinical deterioration (acute NIHSS worsening $>$ 4 points). We also calculated TAGE score (Janvier et al., 2022) recently defined. It is composed by 4 different items as time-to-successful reperfusion, ASPECTs, Glucose level and Early venous filling. For each item is assigned 0–2 or 0–3 points, with a final score range from 0 to 10 points. This score is reported to be highly predictive of symptomatic intracranial hemorrhage in patients with successful recanalization (TICI$\geq$2b). Conventional EEG recordings (with median recording time of 30 min) were implemented in patients admitted to our Stroke Unit within the first 72 h after stroke onset. EEG could be performed again after the first recording, if there was a clinical or suspected seizure or SE. EEG was recorded digitally using 21 fixed electrodes placed according to the International 10–20 system. The recording was obtained in sedative-free patients after EVT. An experienced neurophysiologist then evaluated the EEGs, reporting pathological patterns following the established international glossary (Kane et al., 2017). EEGs abnormalities (slowing or epileptic activity) were categorized as unilateral or bilateral. NCSE were diagnosed following the Salzburg criteria (Beniczky et al., 2013). In case of evidence of epileptic activity, we collected time of onset from the ischemic event. Lastly, for NCSE we evaluated the performed therapy and the resolution rate of the SE after I, II, or III line therapy (according to recent evidence) (Trinka and Leitinger, 2022).

2.1. Statistical analysis

We performed all statistical analyses using SPSS statistical software (v.23, IBM inc.). The Kolmogorov-Smirnov test was used to evaluate the normal distribution of variables. Continuous variables with a normal distribution were presented as mean and standard deviations (SDs), those with a skewed distribution as median and interquartile ranges (IQRs) indicating the 1st and 3rd quartile, and categorical variables as counts and percentages (%). Subgroups were defined with the presence/absence of SE. Differences between groups were tested with Mann-Whitney U-test for skewed variables, and Pearson's chi square for categorical variables, or Fisher's Exact Test. A value of $p<0.05$ was considered as significant.

3. Results

As many as 247 patients underwent a EVT in the study period in our center, of which 138 (56%) underwent EEG recording within 72- h and were thus included in our analysis. Out of them, we observed 9 (6.5%) SE (all NCSE, no reported CSE) and no SE in 129 patients. EEG recordings were performed in a median time of 1 day (IQR 0–2) and SE were detected with a median time of 1 day (IQR 1–2).

Tables 1–3 summarize the general characteristics of the two groups.

Looking at the SE diagnostic criteria (Salzburg), we found 2 possible NCSE and 7 definite NCSE.

Table 1
Demographics of the two groups.

	Non SE n = 129	SE n =9	Sig.
Age (years) [median (IQR)]	77 (68–83)	79 (74–81)	p= 0.992
Females [n (%)]	74 (57%)	7 (77%)	p= 0.306
Comorbidities [n (%)]			
Atrial fibrillation	66 (51%)	4 (44%)	p=0.743
Diabetes mellitus	19 (15%)	1 (11%)	p= 1.000
Hypertension	94 (73%)	6 (66%)	p=0.706
Dyslipidemia	70 (54%)	4 (44%)	p=0.733
Ischemic cardiopathy	20 (15%)	1 (11%)	p=1.000
Previous epilepsy	2 (1%)	0 (0%)	p=1.000
Anamnestic cognitive decline	7 (6%)	1 (11%)	p=0.425
Clinical features [median (IQR)]			
NIHSS at admission	13 (8–20)	17 (12–18)	p=0.395
NIHSS at discharge	4 (1–13)	14 (4.5–17.5)	p=0.200
NIHSS significantly improved [n (%)]	84 (65%)	2 (22%)	**p=0.025**
mRS anamnestic	0 (0–0)	0 (0–0.75)	p=0.401
mRS at discharge	4 (1–5)	5 (4–5)	p=0.058
mRS after 3 months	3 (1–5)	5 (2–6)	p=0.061
Death within 3 months [n (%)]	21 (16%)	3 (33%)	p=0.190

Notes: participants' reported age (years); sex (female); history of hypertension, diabetes mellitus, dyslipidemia, ischemic cardiopathy, atrial fibrillation; NIHSS (National Institutes of Health Stroke Scale) at admission, we defined as NIHSS significantly improved patients those who had NIHSS lowering$>$5 points after treatment or reaching NIHSS of 0–1 points; anamnestic, at discharge and at 3 months modified Rankin Scale (mRS), stroke subtype classification at discharge classified by Trial of ORG 10172 in Acute Stroke Treatment(TOAST): cardioembolic, atherothrombotic, cryptogenic, other cause.

Table 2
Clinical findings of the two groups.

	Non SE n = 129	SE n =9	Sig.
TOAST classification [n (%)]			
Cardioembolic (CE)	69 (53%)	3 (33%)	p=0.301
Atherothrombotic (LAA)	22 (17%)	2 (22%)	p=0.655
Cryptogenic	28 (22%)	4 (44%)	p=0.212
Other cause	10 (8%)	0 (0%)	p=1.000
Vessel closed [n (%)]			
ICA	17 (13%)	0 (0%)	p=0.600
M1	71 (55%)	4 (44%)	p=0.732
M2	34 (26%)	5 (56%)	p=0.117
Posterior circulation	7 (5%)	0 (0%)	p=1.000
Side of the ischemic lesion [n (%)]			
Rightside	59 (43%)	4 (44%)	p=1.000
Leftside	68 (53%)	5 (56%)	p=1.000
Bilateral	2 (1%)	0 (0%)	p=1.000
Lobe involved in the NCCT			
Frontal	67 (52%)	6 (67%)	p=0.500
Parietal	52 (40%)	6 (67%)	p=0.166
Temporal	52 (40%)	6 (67%)	p=0.166
Insular	41 (32%)	8 (89%)	**p=0.001**
Occipital	22 (17%)	4 (44%)	p=0.065
Basal ganglia/thalamus	57 (44%)	8 (89%)	**p=0.013**
Brainstem	6 (5%)	0 (0%)	p=1.000
Cerebellar	3 (2%)	0 (0%)	p=1.000
Sum [median (IQR)]	2 (1–3)	4 (3–6)	**p=0.048**

Notes: stroke subtype classification at discharge classified by Trial of ORG 10172 in Acute Stroke Treatment (TOAST): cardioembolic, atherothrombotic, cryptogenic, other cause; vessel closed: ICA (internal carotid artery), M1-M2 (middle cerebral artery).

Table 3
Type of Treatment and EEG characteristics of the two groups.

	Non SE n = 129	SE n =9	Sig.
Type of treatment [n (%)]			
Direct Thrombectomy	25 (19%)	1 (11%)	p=1.000
TICI [n (%)]			
2b-3	89 (69%)	7 (77%)	p=0.723
TAGE score [median (IQR)]	1 (0-4)	4 (4-5)	**p<0.001**
Hemorrhagic complication [n (%)]			
asymptomatic hemorrhage	25 (19%)	1 (11%)	p=1.000
symptomatic hemorrhage	9 (7%)	2 (22%)	p=0.158
EEG characteristics [n (%)]			
Normal	20 (16%)	0 (0%)	
Slowing abnormalities	105 (81%)	0 (0%)	
Unilateral	75 (71%)	0 (0%)	
Bilateral	30 (29%)	0 (0%)	
Epileptiform abnormalities	35 (27%)	9 (100%)	
Unilateral	21 (60%)	7 (77%)	
Bilateral	14 (40%)	2 (23%)	
SE resolutive therapy [n (%)]			
BDZ	/	2 (22%)	
AED	/	6 (66%)	
Sedative drugs	/	1 (11%)	

Notes: Treatment: number of patients that underwent direct thrombectomy treatment, successful recanalization rate through the TICI score (Thrombolysis In Cerebral Infarction score), number of patients with intracerebral hemorrhage (symptomatic or asymptomatic) after treatment. In the unilateral SE, 2 were found controlateral to ischemic lesion side. BDZ: Benzodiazepines; AED: anti-epileptic drugs.

4. Discussion

In this study, we found a higher proportion of SE in patients treated with mechanical thrombectomy (6.5%) than ischemic stroke in general (0.2–0.3%). SE is known to be a possible complication following ischemic strokes. An early EEG assessment (within 72- h from onset of symptoms) in the Stroke Unit setting is useful to diagnose subtle epileptic activity, in particular NCSE. A rapid recognition of NCSE neurological emergency may allow a prompt treatment by the neurologist, thus reducing mortality and morbidity after the ischemic event.

Most of the SE we found were unilateral (hemispheric), although in the opposite side of the ischemic lesion in 2 cases. We may suggest a possible role of diaschisis or increase of excitability of the non-affected hemisphere, as documented in some TMS study (Kubis, 2016). All SE were responsive to a I-II line therapy, generalized SE were successfully treated with II-III line therapy.

No-SE and SE groups had similar baseline characteristics (comorbidities, NIHSS and mRS at admission, TOAST etiologies, type of vessel closed). Yet, SE cohort had a worse outcome than the other group, in particular lower improvement of NIHSS at discharge; however can be noted a higher mRS at discharge and at 3 months, and higher mortality rate (although not statistically significant). These data are in line with previous publications on this topic (Velioglu et al., 2001). The fact that we did not find any CSE may be explained by the prompt recognition and treatment of SE before it becomes clinically evident. Based on these data, we cannot clearly establish a causality role of NCSE and outcomes (as mRS), but we can suggest a possible correlation between the sustained epileptic activity and patient recovery after EVT.

The location and size of the ischemic lesion have a significant role in the development of SE. In some pieces of research, lobar involvement (including insular one) has proved to be a risk factor for SE in both ischemic and hemorrhagic events (Zhang et al., 2022; Matsubara et al., 2018), while in others no clear localization has been identified as risk factor for early SE (Afsar et al., 2003). In our sample, the SE group had significantly higher involvement of insular cortex and basal ganglia than the no-SE group. Other lobes were generally less involved in the no-SE group. To the best of our knowledge, such findings (insular and basal ganglia more involved in SE group) have not been described in other works. The higher insular infarction in SE group may explain the higher morbidity and mortality rate, in particular a possible autonomic dysfunction could lead to a worse prognosis. The higher prevalence of deep brain grey matter infarctions in SE may be linked to the embolic passage through the first tract of middle cerebral artery, which induces ischemia in areas without collateral circulation that could supply them until the reperfusion therapy. The fact that there is a basal ganglia involvement, could be further marker of stroke extension as a consequence of proximal middle cerebral artery occlusion. Our data show a trend related to the occipital cortex involvement, which could be a consequence of an infarction in middle cerebral artery territory (thus including a small piece of the occipital lobe (González Delgado and Bogousslavsky, 2012) larger than the lower size of necrosis in the no-SE cohort.

The role of reperfusion therapy success shows no difference between the two groups. In general, SE group has a greater lesion but similar location in vessel occlusion than the non-SE group. A possible explanation is that a weak collateral circulation cannot save enough tissue up to the thrombectomy, and when this procedure succeeds in re-opening the vessel, a reperfusion-effect in the ongoing dying tissue can promote an epileptic activity. However, this hypothesis requires further investigation with CT perfusion imaging to be confirmed. Yet, according to recent meta-analysis performed by Bentes et al. (Bentes et al., 2020) and a study of Brigo et al. (Brigo et al., 2020), the association of reperfusion therapies with early post-stroke seizure is still unclear. A possible marginal role may be played by the symptomatic hemorrhages, as they may worsen the underlying bioelectrical instability. This issue can be sustained by the TAGE score, a predictor or symptomatic hemorrhage after MT. In fact, we found a higher TAGE score in the SE group compared to the non-SE group.

This study has several limitations, such as the small sample of patients (which did not let us perform a logistic regression), the non-standardized timing of the EEG recording, and the retrospective design of the data collection from a single center. As a consequence, we do not have blood pressure values at admission and after EVT. In fact, hyperemia is thought to be a significant contributor to seizure risk post-

EVT. We did not have timely EEG (or EEG record at all) in patients admitted to ICU or in patients who died before undergoing EEG recording. We did not calculate an infarct lesion volume but only a description of the infarction extension looking at the lobes involved. On the other hand, the study was conducted in a real setting, namely the clinical practice of a Stroke Unit, a detailed neuroimaging data has been conducted for each patient, as well as a rapid EEG assessment after admission. Further studies with higher number of cases and stronger statistical power are needed to better investigate NCSE rate after mechanical thrombectomy, independent factors associated with SE, and to develop a protocol in SU for EEG recording after stroke.

5. Conclusions

SE after large strokes treated with EVT is a non-rare complication, with most of them being NCSE. SE after EVT may be correlated to worse outcome and higher final infarct lesion. Performing a rapid EEG assessment in a SU setting may allow for a prompt recognition and treatment of NCSE in the acute/hyper-acute phase.

CRediT authorship contribution statement

Emanuele Vincis: Conceptualization. **Laura Mancinelli:** Conceptualization. **Paola Caruso:** Supervision, Conceptualization. **Giovanni Furlanis:** Writing – review & editing, Project administration, Methodology, Conceptualization. **Gabriele Prandin:** Writing – original draft, Investigation, Formal analysis, Conceptualization. **Federica Palacino:** Conceptualization. **Ilario Arturo Scali:** Conceptualization. **Marinella Tomaselli:** Supervision, Conceptualization. **Giulia Mazzon:** Supervision, Conceptualization. **Paolo Manganotti:** Writing – review & editing, Supervision, Project administration, Methodology, Conceptualization. **Marcello Naccarato:** Supervision, Project administration, Conceptualization.

Declaration of Copmeting Interest

None.

References

Adams, H.P.J., Bendixen, B.H., Kappelle, L.J., et al., 1993. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke 24 (1), 35–41.

Afsar, N., Kaya, D., Aktan, S., Sykut-Bingol, C., 2003. Stroke and status epilepticus: stroke type, type of status epilepticus, and prognosis. Seizure 12 (1), 23–27. https://doi.org/10.1016/s1059131102001437. PMID: 12495645.

Alemany, M., Nuñez, A., Falip, M., Lara, B., Paipa, A., Quesada, H., Mora, P., De Miquel, M.A., Barranco, R., Pedro, J., Cardona, P., 2021. Acute symptomatic seizures and epilepsy after mechanical thrombectomy. A prospective long-term follow-up study. Seizure 89, 5–9. https://doi.org/10.1016/j.seizure.2021.04.011. Epub 2021 Apr 20. PMID: 33933947.

Anon, 2019. Correction to: Guidelines for the Early Management of Patients With Acute Ischemic Stroke: 2019 Update to the 2018 Guidelines for the Early Management of Acute Ischemic Stroke: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association. Stroke 50 (12), e440–e441. https://doi.org/10.1161/STR.0000000000000215. Epub 2019 Nov 25. Erratum for: Stroke. 2019 Oct 30;:STR0000000000000211. PMID: 31765293.

Bateman, B.T., Claassen, J., Willey, J.Z., Hirsch, L.J., Mayer, S.A., Sacco, R.L., Schumacher, H.C., 2007. Convulsive status epilepticus after ischemic stroke and intracerebral hemorrhage: frequency, predictors, and impact on outcome in a large administrative dataset. Neurocrit. Care 7 (3), 187–193. https://doi.org/10.1007/s12028-007-0056-2. PMID: 17503112.

Belcastro, V., Vidale, S., Gorgone, G., Pisani, L.R., Sironi, L., Arnaboldi, M., Pisani, F., 2014. Non-convulsive status epilepticus after ischemic stroke: a hospital-based stroke cohort study. J. Neurol. 261 (11), 2136–2142. https://doi.org/10.1007/s00415-014-7471-z. Epub 2014 Aug 20. PMID: 25138478.

Beniczky, S., Hirsch, L.J., Kaplan, P.W., Pressler, R., Bauer, G., Aurlien, H., Brøgger, J.C., Trinka, E., 2013. Unified EEG terminology and criteria for nonconvulsive status epilepticus. Epilepsia *54*, 28–29.

Bentes, C., Brigo, F., Zelano, J., Ferro, J.M., 2020. Reperfusion therapies and poststroke seizures. Epilepsy Behav. 104 (Pt B), 106524 https://doi.org/10.1016/j.yebeh.2019.106524. Epub 2019 Nov 11. PMID: 31727547.

Berge, E., Whiteley, W., Audebert, H., et al., 2021. European Stroke Organisation (ESO) guidelines on intravenous thrombolysis for acute ischaemic stroke. Eur. Stroke J. 6 (1), I–LXII. https://doi.org/10.1177/2396987321989865.

Brigo, F., Schneider, M., Wagenpfeil, G., Unger, M.M., Holzhoffer, C., Walter, S., Faßbender, K., Lochner, P., 2020. Early poststroke seizures following thrombolysis and/or thrombectomy for acute stroke: Clinical and stroke characteristics. Epilepsy Behav. 104 (Pt B), 106353 https://doi.org/10.1016/j.yebeh.2019.05.048. Epub 2019 Jun 21. PMID: 31231037.

Caruso, P., Naccarato, M., Furlanis, G., Ajčević, M., Stragapede, L., Ridolfi, M., Polverino, P., Ukmar, M., Manganotti, P., 2018. Wake-up stroke and CT perfusion: effectiveness and safety of reperfusion therapy. Neurol. Sci. 39 (10), 1705–1712. https://doi.org/10.1007/s10072-018-3486-z. Epub 2018 Jul 10. PMID: 29987433.

Furlanis, G., Ajčević, M., Buoite Stella, A., Cillotto, T., Caruso, P., Ridolfi, M., Cova, M.A., Naccarato, M., Manganotti, P., 2020. Wake-up stroke: thrombolysisreducesischemiclesion volume and neurological deficit. J. Neurol. 267 (3), 666–673. https://doi.org/10.1007/s00415-019-09603-7. Epub 2019 Nov 12. PMID: 31720820.

Galovic, M., Ferreira-Atuesta, C., Abraira, L., Döhler, N., Sinka, L., Brigo, F., Bentes, C., Zelano, J., Koepp, M.J., 2021. Seizures and Epilepsy After Stroke: Epidemiology, Biomarkers and Management. Drugs Aging 38 (4), 285–299. https://doi.org/10.1007/s40266-021-00837-7. Epub 2021 Feb 23. PMID: 33619704; PMCID: PMC8007525.

González Delgado, M., Bogousslavsky, J., 2012. Superficial middle cerebral artery territory infarction. Front Neurol. Neurosci. 30, 111–114. https://doi.org/10.1159/000333604. Epub 2012 Feb 14. PMID: 22377875.

Heiss, W.D., Huber, M., Fink, G.R., et al., 1992. Progressive derangement of periinfarct viable tissue in ischemic stroke. J. Cereb. Blood Flow Metab. 12, 193–203.

Janvier, P., Kerleroux, B., Turc, G., Pasi, M., Farhat, W., Bricout, N., Benzakoun, J., Legrand, L., Clarençon, F., Bracard, S., Oppenheim, C., Boulouis, G., Henon, H., Naggara, O., Ben Hassen, W., 2022. TAGE score for symptomatic intracranial hemorrhage prediction after successful endovascular treatment in acute ischemic stroke. Stroke 53 (9), 2809–2817. https://doi.org/10.1161/STROKEAHA.121.038088. Epub 2022 Jun 14. PMID: 35698971.

Jiang, W., Zhu, X., Lei, C., Jiang, G., Zhang, L., Mei, S., Zhong, L., 2023. Risk assessment of mechanic thrombectomy on post-stroke seizures: a systematical review and meta-analysis. J. Stroke Cereb. Dis. 32 (8), 107155 https://doi.org/10.1016/j.jstrokecerebrovasdis.2023.107155. Epub ahead of print. PMID: 37172469.

Kane, N., Acharya, J., Benickzy, S., Caboclo, L., Finnigan, S., Kaplan, P.W., Shibasaki, H., Pressler, R., van Putten, M.J.A.M., 2017. A Revised Glossary of Terms Most Commonly Used by Clinical Electroencephalographers and Updated Proposal for the Report Format of the EEG Findings. Revision 2017. Clin. Neurophysiol. Pract. 2, 170–185. https://doi.org/10.1016/J.CNP.2017.07.002.

Kubis, N., 2016. Non-Invasive Brain Stimulation to Enhance Post-Stroke Recovery. Front Neural 10, 56. https://doi.org/10.3389/fncir.2016.00056. PMID: 27512367; PMCID: PMC4961863.

Luhmann, H.J., Mudrick-Donnon, L.A., Mittmann, T., Heinemann, U., 1995. Ischemia-induced long-term hyperexcitability in rat neocortex. Eur. J. Neurosci. 7, 180–191.

Matsubara, S., Sato, S., Kodama, T., Egawa, S., Nakamoto, H., Toyoda, K., Kubota, Y., 2018. Nonconvulsive status epilepticus in acute intracerebral hemorrhage. Stroke 49 (7), 1759–1761.

Scoppettuolo, P., Gaspard, N., Depondt, C., Legros, B., Ligot, N., Naeije, G., 2019. Epileptic activity in neurological deterioration after ischemic stroke, a continuous EEG study. ClinNeurophysiol 130 (12), 2282–2286. https://doi.org/10.1016/j.clinph.2019.09.005. Epub 2019 Sep 21. PMID: 31594733.

Sinka, L., Abraira, L., Imbach, L.L., Zieglgänsberger, D., Santamarina, E., Álvarez-Sabín, J., Ferreira-Atuesta, C., Katan, M., Scherrer, N., Bicciato, G., Terziev, R., Simmen, C., Schubert, K.M., Elshahabi, A., Baumann, C.R., Döhler, N., Erdélyi-Canavese, B., Felbecker, A., Siebel, P., Winklehner, M., von Oertzen, T.J., Wagner, J. N., Gigli, G.L., Serafini, A., Nilo, A., Janes, F., Merlino, G., Valente, M., Zafra-Sierra, M.P., Bayona-Ortiz, H., Conrad, J., Evers, S., Lochner, P., Roell, F., Brigo, F., Bentes, C., Peralta, A.R., Pinho, E., Melo, T., Keezer, M.R., Duncan, J.S., Sander, J. W., Tettenborn, B., Koepp, M.J., Galovic, M., 2023. Association of Mortality and Risk of Epilepsy With Type of Acute SymptomaticSeizure After Ischemic Stroke and an UpdatedPrognostic Model. JAMA Neurol. 80 (6), 605–613. https://doi.org/10.1001/jamaneurol.2023.0611. PMID: 37036702; PMCID: PMC10087089.

Trinka, E., Leitinger, M., 2022. Management of Status Epilepticus, Refractory Status Epilepticus, and Super-refractory Status Epilepticus. Contin. (Minne 28 (2), 559–602. https://doi.org/10.1212/CON.0000000000001103. PMID: 35393970.

Turc, G., Bhogal, P., Fischer, U., et al., 2019. European stroke organisation (ESO) – European Society for minimally invasive neurological therapy (ESMINT) guidelines on mechanical thrombectomy in acute ischaemicstrokeendorsed by stroke alliance for Europe (SAFE). Eur. Stroke J. 4 (1), 6–12. https://doi.org/10.1177/2396987319832140.

Velioglu, S.K., Özmenoğlu, M., Boz, C., Alioglu, Z., 2001. Status epilepticus after stroke. Stroke 32 (5), 1169–1172.

Zhang, L., Zheng, W., Chen, F., Bai, X., Xue, L., Liang, M., Geng, Z., 2022. Associated factors and prognostic implications of non-convulsive status epilepticus in ischemic stroke patients with impaired consciousness. Front. Neurol. 12, 795076 https://doi.org/10.3389/fneur.2021.795076. PMID: 35069425; PMCID: PMC8777101.