Risk Factors for In-Hospital Seizure and New-Onset Epilepsy in Coiling and Clipping Treatment of Aneurysmal Subarachnoid Hemorrhage

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■ OBJECTIVE: **To identify risk factors associated with in-hospital seizures and new-onset epilepsy in patients with aneurysmal subarachnoid hemorrhage (SAH) who underwent coiling embolization or clipping surgery.**

■ METHODS: **This retrospective descriptive study included 195 patients diagnosed with aneurysmal SAH and treated with coiling embolization or clipping surgery between January 2018 and June 2022.**

■ RESULTS: **Among the 195 patients meeting inclusion criteria, 9 experienced an onset seizure at the time of SAH. In-hospital seizures were observed in 33 patients, of which 24 were electrographic seizures detected in 24 patients with suspected subclinical seizures. After 12 months of follow-up, 11 patients met criteria for diagnosis of epilepsy. The incidence of epilepsy after discharge at 12 months was 2.41% in the coiling group and 8.03% in the clipping group. The risk of in-hospital seizures was significantly higher in the clipping group ($P = 0.007$), although the difference was not statistically significant after 12 months of follow-up ($P = 0.121$).**

■ CONCLUSIONS: **Epilepsy following aneurysmal SAH was relatively common. Clipping surgery and brain edema emerged as independent predictive factors for in-hospital seizures, while onset seizures and in-hospital seizures were identified as independent predictors of epilepsy during follow-up. Patients presenting with these risk factors may benefit from long-term electroencephalogram monitoring and should be considered for prophylactic antiepileptic drugs. Additionally, lumbar drainage proved effective in improving both early and late epileptic outcomes in the group with Fisher grades 3 and 4.**

INTRODUCTION

Approximately 30,000 patients with subarachnoid hemorrhage (SAH) are hospitalized each year in the United States.[1] Most cases of spontaneous SAH are attributed to aneurysmal rupture. A review study reported that onset seizures occurred in 4%–26% of patients with SAH, with 1%–28% experiencing early seizures within the first 2 weeks and 1%–35% developing late seizures after 2 weeks. Patients who presented with seizures had a 27% mortality rate at 12 months following SAH.[2] Furthermore, the occurrence of epilepsy after SAH had a negative impact on patients' quality of life and mental status compared with patients in whom epilepsy did not occur.[3]

The use of antiseizure medications (ASMs) for patients with SAH lacks convincing evidence either in favor or against. However, one study showed that patients taking prophylactic ASMs experienced cognitive impairment and increased risk of complications.[4] Therefore, anticonvulsants may be selectively considered for

Key words
- Aneurysm
- Epilepsy
- Long-term electroencephalogram
- Lumbar drainage
- Subarachnoid hemorrhage

Abbreviations and Acronyms
ASM: Antiseizure medication
CI: Confidence interval
CSF: Cerebrospinal fluid
CT: Computed tomography
EEG: Electroencephalogram
LD: Lumbar drainage
MCA: Middle cerebral artery
OR: Odds ratio
SAH: Subarachnoid hemorrhage
TCD: Transcranial Doppler
WFNS: World Federation of Neurological Surgeons

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patients with known risk factors for delayed seizures following SAH, including increasing age (>65 years old), middle cerebral artery (MCA) aneurysm, amount of hemorrhage accumulation, associated intracerebral or subdural hematoma, poor neurological grade, rebleeding, cerebral infarction, vasospasm, and hydrocephalus.[5]

Research analyzing factors that increase the probability of seizures between clipping and coiling treatments is insufficient. Furthermore, limited data are available regarding the impact of long-term electroencephalogram (EEG) monitoring and lumbar drainage (LD) on patient outcomes. Therefore, we conducted a study to assess the risk factors for in-hospital seizures and new-onset epilepsy in patients undergoing coil embolization or clipping surgery for ruptured aneurysmal SAH. We aimed to identify better candidates for more comprehensive use of ASMs and to determine the potential benefits of performing long-term EEG monitoring and implementing LD to improve overall management of patients and guide decision making in clinical practice.

MATERIALS AND METHODS

A retrospective study was conducted at the University Medical Center of patients with aneurysmal SAH who underwent coiling embolization or clipping surgery between January 2018 and June 2022. Patients with a prior history of epilepsy or who underwent previous brain surgeries were excluded from the study. Data were collected from admission up to at least 1 year after discharge.

Due to limited resources, continuous EEG was not available for all enrolled patients. Some patients in the study underwent routine and long-term EEG monitoring (580-G2CGS S32; Biologic or EEG-1200; Nihon Kohden, Japan) perioperatively as decided by physicians, particularly in suspected instances of subclinical seizure, such as in patients with decreased consciousness, comatose patients, and sedated patients. Long-term EEG monitoring was defined as lasting >1 hour, while routine EEG monitoring was defined as <1 hour. EEG recordings were carefully analyzed by an epileptologist. Any interictal epileptiform discharges, including lateralized periodic discharges (previously termed periodic lateralized epileptiform discharges), generalized periodic discharges (previously termed generalized periodic epileptiform discharges), and isolated nonperiodic spikes or sharp waves, were scored as present or absent. Electrographic seizures were defined as an abnormal paroxysmal event that was different from the background, lasting >10 seconds (or shorter if associated with a clinical change) with a temporal-spatial evolution in morphology, frequency, and amplitude and with a plausible electrographic field. Electrographic seizures were classified as nonconvulsive seizures (no clinical signs observed by bedside caregivers or on video review) or electroclinical seizures (clinical abnormal stereotypic and paroxysmal movements associated with the EEG seizure).[6] Epileptiform discharges and/or seizures were collected for further investigation.

To detect vasospasm, transcranial Doppler (TCD) (Rimed Digi-Lite; Rimed USA, Inc., Long Island City, New York, USA) ultrasound was routinely performed every 48–72 hours for all patients with SAH during the hospital stay, especially for obtunded patients or patients with clinically severe SAH with significant bleeding on computed tomography (CT) scan (Fisher grades 3 and 4). Vasospasm was diagnosed based on serial TCD ultrasound scans and/or neurological deficit with negative findings on CT scan of new hemorrhage, hydrocephalus, and infarction. Other causes of deterioration were excluded, such as hypoxia, hyponatremia, meningitis, and seizure.

Cerebral edema was diagnosed when localized brain tissue hypodensities associated with mass effect were present. Focal edema was categorized as 1) hemorrhage related if the hypodense area surrounded a thick clot in the subarachnoid space or an intraparenchymal hematoma, 2) infarction related, or (3) edema related to other causes. When diagnosing edema, infarction, or other radiographic findings, independent confirmation was obtained from the other study physicians from CT or magnetic resonance imaging scans. In addition, mass effect was defined as any radiologic evidence that the patient had compression of adjacent structures in the brain from any masses, including blood. If a radiologist observed evidence of mass effect on a brain CT or magnetic resonance imaging scan, the patient was deemed to have mass effect. All radiologic variables were collected in similar fashion.

Following admission to our center, ASMs (e.g., levetiracetam, phenytoin valproic acid) were prescribed to patients only if specific indications warranted their use. Generally, ASMs were indicated for patients with substantial intracerebral hemorrhage, high Fisher grade, or severe clinical presentation and were discontinued within 3–7 days after coiling or clipping.

Data were collected from various sources, including history and physical examination, progress notes, operative reports, radiologic reports, EEG reports, and discharge summaries. The variables analyzed included age, sex, past medical history, World Federation of Neurological Surgeons (WFNS) grade, Fisher grade, intraparenchymal hematoma, hydrocephalus, cerebral edema, treatment modality (clipping or coiling), postoperative vasospasm (based on TCD ultrasound scans and clinical examinations), and epileptiform discharge on EEG. The outcome variables examined were in-hospital seizures and epilepsy during the follow-up period.

An onset seizure was defined as a seizure occurring within 12 hours of SAH, which typically led to hospitalization.[7,8] In-hospital seizures were categorized as clinical or subclinical (electrographic) seizures. Clinical seizures were referred to as any convulsions occurring from 12 hours after admission to discharge. Subclinical seizures were electrographic seizures that occurred without subjective or objective clinical symptoms. Epilepsy at follow-up examinations was diagnosed when there were ≥2 unprovoked seizures.

After discharge, telephone interviews were conducted with patients or close family members. In cases where seizures had occurred, further details regarding seizure characteristics and any early symptoms or provocation were noted. Epilepsy was determined based on the provided history if there were 2 separate seizures without acute provocations. The follow-up period was 12 months after discharge.

Ethical Considerations

The study was conducted at the University Medical Center, Ho Chi Minh City, Vietnam, which is the national referral center for the patients with epilepsy and related disorders. The medical charts and electronic records of all patients with epilepsy admitted from January 2018 to June 2022 were retrospectively reviewed. The study

adhered to the ethical principles outlined in the 1964 Declaration of Helsinki.

Data Analysis

Descriptive statistics such as mean, standard deviation, median, interquartile range, frequency, and percentage were used to summarize the data. The association between patient characteristics and epileptic outcome was evaluated using χ^2 and Fisher exact tests. A 2-sided significance level of 0.05 was applied to all statistical tests. Non-normally distributed continuous data before the operation were analyzed using the Mann-Whitney U test. Data analysis was performed using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, New York, USA) software.

RESULTS

Incidence

Table 1 presents the participants' demographic, clinical, and subclinical characteristics. The study enrolled 195 eligible patients with a male-to-female ratio of 1:1.38 and a mean age of 43 ± 11.5 years. Of patients, 67.2% were hospitalized with mild to

Table 1. Characteristics of Subarachnoid Hemorrhage Patients, In-Hospital Seizure and Epilepsy Outcomes, and Factors Related to Outcome

Patients (N = 195)		In-Hospital Seizure				Epilepsy			
Characteristic	**Number (%)**	**Number**	***P* Value**	**95% CI**	**OR**	**Number**	***P* Value**	**95% CI**	**OR**
Sex, female	113 (57.9)	18	0.702	0.39–1.82	0.85	5	0.532	0.22–1.99	0.59
Age >50 years	81 (41.5)	22	0.002*	1.57–7.75	3.49	9	0.009*	3.10–33.36	7.00
WFNS grades									
4 + 5	64 (32.8)	16	0.043*	1.04–4.80	2.24	6	0.182	1.03–8.86	2.61
1–3	131 (67.2)	17		0.20–0.99	0.45	5		0.14–1.30	0.38
Fisher grades									
3 + 4	74 (37.9)	19	0.017*	1.22–5.69	2.64	7	0.107	1.27–10.82	3.06
1 + 2	121 (62.1)	14		0.17–0.83	0.38	4		0.11–1.16	0.33
Aneurysm location									
MCA	81 (41.5)	13	0.160	0.42–1.94	0.90	5	0.438	0.44–4.02	1.18
Acom	42 (21.5)	11		0.93–4.82	2.11	4		0.75–7.77	2.20
Pcom	30 (15.4)	0		—	—	0		—	—
ICA	26 (13.3)	9		1.28–8.00	3.20	3		0.78–10.22	2.63
Posterior circulation	16 (8.2)	4		0.52–5.72	1.72	1		0.15–8.99	1.13
Multiple aneurysms	5 (2.6)	1		0.13–11.41	1.23	0		—	—
ICH	82 (42.1)	20	0.021*	1.15–5.38	2.48	9	0.009*	3.03–32.61	6.84
Treatment modality									
Coiling	83 (42.6)	13	0.007*	0.40–1.84	0.85	2	0.121	0.07–1.34	0.28
Clipping	112 (57.4)	26		1.33–8.11	3.28	9		1.57–16.87	3.54
Vasospasm	62 (31.8)	18	0.003*	1.49–6.96	3.22	7	0.039*	1.70–14.53	4.10
Cerebral edema	82 (42.1)	22	0.002*	1.53–7.55	3.40	7	0.207	1.06–9.00	2.54
Hydrocephalus	57 (29.2)	16	0.011*	1.28–6.01	2.78	7	0.016*	1.95–16.61	4.69
Onset seizure	9 (4.6)	3	0.180	0.62–10.98	2.60	3	0.009*	3.29–43.31	11.13
Outcome									
In-hospital seizure	33 (16.9)	33	—	—	—	6	0.004*	2.76–23.72	6.98
Epilepsy	11 (5.6)	6	—	—	—	—	—	—	—

CI, confidence interval; OR, odds ratio; WFNS, World Federation of Neurological Surgeons; MCA, middle cerebral artery; Acom, anterior communicating artery; Pcom, posterior communicating artery; ICA, internal carotid artery; ICH, intracerebral hemorrhage.
*Significant value.

moderate clinical SAH (WFNS grades 1–3), although imaging studies showed that 37.9% of cases were Fisher grade 3 or 4. The flow of patient inclusion in the study and events of seizure and epilepsy is shown in Figure 1. The incidence of onset seizures was 4.6% (9 patients), and 33 patients experienced in-hospital seizures, including clinical and subclinical (electrographic) types. The average time of onset for these seizures was 5.4 days (range 2.1–6.3 days). Additionally, interictal epileptiform discharges (not including seizure) were detected in 28 patients, and 24 subclinical seizures were recorded. During the follow-up period, 11 patients met the criteria for an epilepsy diagnosis, including 22 patients treated with coiling and 9 patients treated with clipping.

The most common type of aneurysm observed in the study was MCA aneurysms, followed by anterior communicating artery and posterior communicating artery aneurysms. Multiple aneurysms were present in 2.6% of patients, all of which were located in the anterior circulation. Aneurysm ligation was performed in 112 patients, and the remaining 83 patients underwent coiling. Of the patients who underwent clipping, 26 experienced in-hospital seizures, whereas only 7 patients who underwent coiling experienced in-hospital seizures. Among the 11 patients diagnosed with new-onset epilepsy, 9 patients previously underwent surgery, and 2 patients received endovascular intervention.

Vasospasm detected either clinically or on TCD ultrasound was seen in 62 cases. Among patients with vasospasm, 18 experienced in-hospital seizures, and 7 developed epilepsy during the follow-up period. Intracerebral hematoma was observed in 82 cases, primarily associated with MCA aneurysms. Cerebral edema occurred in 82 patients due to cerebral ischemia and infarction. Hydrocephalus was present in 57 patients, of whom 16 experienced in-hospital seizures and 7 developed late epilepsy.

Long-term EEG monitoring was not conducted in all patients but was mainly used for assessment of patients with severe clinical conditions and impaired consciousness. Epileptiform discharges and electrographic seizures were captured and analyzed by epileptologists. Of 155 patients who underwent long-term EEG monitoring, epileptiform discharges (without subclinical seizures) were recorded in 28 cases, and subclinical seizure was shown in 24 patients (Figure 2). In a subgroup of 64 patients with WFNS grade 4 or 5, all underwent long-term EEG monitoring, which revealed epileptiform discharges in 13 (20.3%) patients. In the short-term follow-up, epilepsy was diagnosed in 4 of 13 patients (Table 2).

At our center, as a general practice, LD was indicated for most patients with Fisher grade 3 or 4, regardless of whether the patient received vascular or surgical intervention. In contrast, LD was rarely used in patients with Fisher grade 1 or 2 unless there was an absolute indication (hydrocephalus). About 100–300 mL of cerebrospinal fluid (CSF) was removed each day until the red hue of the fluid turned transparent (Figure 3). In this study, of 74 patients with Fisher grade 3 or 4, 68 (91.9%) received LD. Among the patients with Fisher grade 3 or 4, up to 98.2% of patients without an in-hospital seizure and 95.5% of those without epilepsy received LD, while the percentage of patients with LD in the in-hospital seizure and epilepsy groups was only 73.7% and 57.1%, respectively (Table 2).

In-Hospital Seizure

Table 1 lists several factors that were correlated with in-hospital seizures, including age >50 (odds ratio [OR] 3.49, 95%

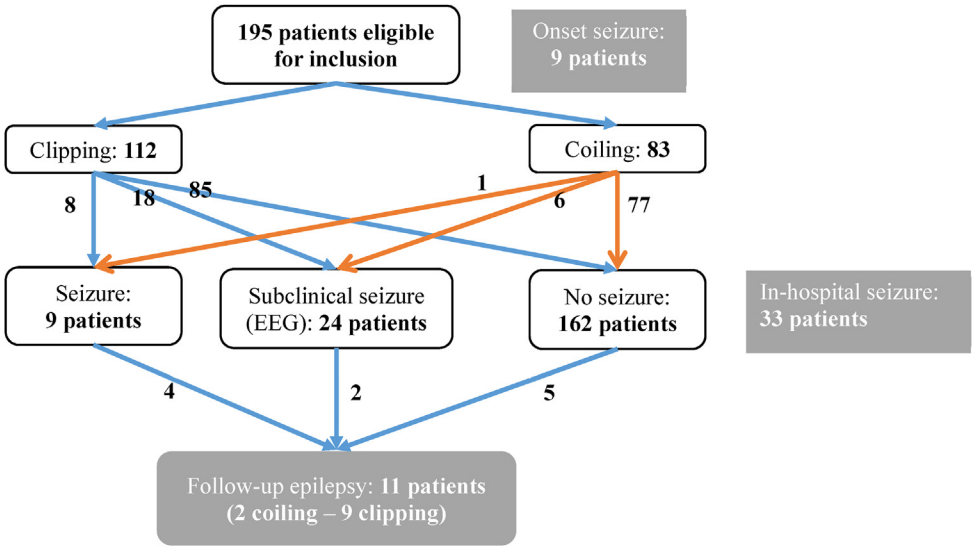

Figure 1. Schematic flow of patients enrolled in this study. Patients with previous brain surgeries and a history of epilepsy were excluded. Onset seizures occurred in 9 patients. Following either clipping or coiling procedures, 33 patients experienced in-hospital seizures, of which 24 were classified as subclinical seizures. On subsequent follow-up examinations, 11 patients developed epilepsy. EEG, electroencephalogram.

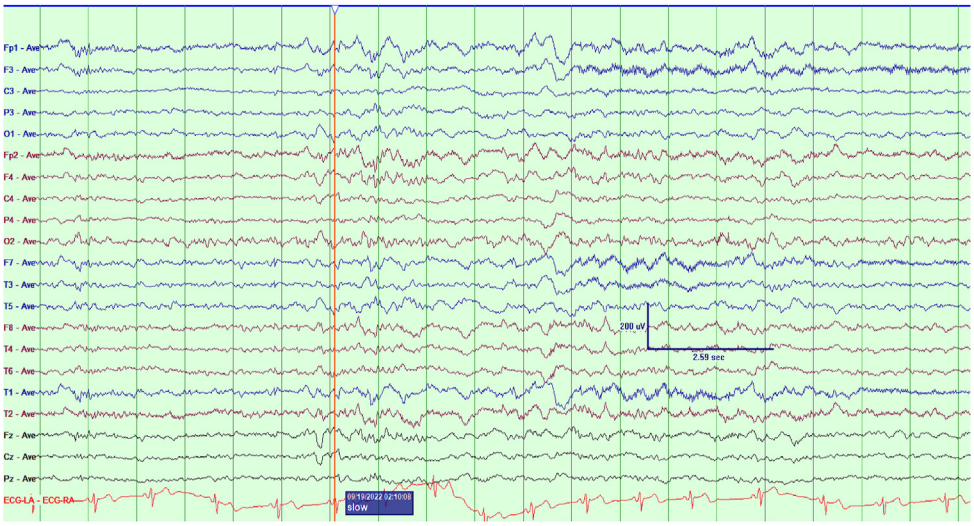

Figure 2. Abnormal electroencephalogram. This 24-hour electroencephalogram demonstrated a normal background with interictal spike-slow waves/polyspikes/continuous spikes in left frontotemporal polar (Fp1>F7T1), spread to right frontotemporal regions. EEG, electroencephalogram.

confidence interval [CI] 1.57–7.75), WFNS grade 4 or 5 (OR 2.24, 95% CI 1.04–4.80), Fisher grade 3 or 4 (OR 2.64, 95% CI 1.22–5.69), intracerebral hemorrhage (OR 2.48, 95% CI 1.15–5.38), clipping surgery (OR 3.28, 95% CI 1.33–8.11), vasospasm (OR 3.22, 95% CI 1.49–6.96), cerebral edema (OR 3.40, 95% CI 1.53–7.55), and hydrocephalus (OR 2.78, 95% CI 1.28–6.01). Multivariate analysis revealed that clipping surgery (95% CI 1.06–7.42) and brain edema (95% CI 1.07–6.20) were independent risk factors for in-hospital seizures.

Epilepsy During 12-Month Follow-Up

Table 1 lists factors that were related to epilepsy outcomes in follow-up, including age >50 (OR 7.0, 95% CI 3.10–33.36), intracerebral hemorrhage (OR 6.94, 95% CI 3.03–32.61), vasospasm (OR 4.10, 95% CI 1.70–14.53), hydrocephalus (OR 4.69, 95% CI 1.95–16.61), onset seizure (OR 11.13, 95% CI 3.29–43.31), and in-hospital seizure (OR 6.98, 95% CI 2.76–23.72). In multivariate analysis, onset seizure (95% CI 1.47–50.67) and in-hospital seizure (95% CI 1.04–19.15) remained independent predictors for epilepsy during follow-up.

Subgroup Analysis

In an analysis examining subgroups with Fisher grades 3 and 4 and focusing on how effectively CSF was cleared using LD, placement of LD was found to have a correlation with reduction of in-hospital seizure and epilepsy rates. The statistical significance

Table 2. Factors Related to Outcome in Subgroup Analysis of Fisher Grades 3 and 4 and World Federation of Neurological Surgeons Grades 4 and 5

		In-Hospital Seizure				Epilepsy			
	Number (%)	Number	*P* Value	95% CI	OR	Number	*P* Value	95% CI	OR
Fisher 3 and 4 ($n = 74$)									
Lumbar drainage (+)	68 (91.9)	14	0.004*	2.08–178.65	19.3	4	0.009*	2.41–106.17	16.0
Lumbar drainage (–)	6 (8.1)	54				3			
WFNS 4 and 5 ($n = 64$)									
Epileptiform discharge	13 (20.3)	—	—	—	—	4	0.013*	1.73–68.58	10.89
Normal EEG	51 (79.7)	—		—	—	2			

CI, confidence interval; OR, odds ratio; WFNS, World Federation of Neurological Surgeons; EEG, electroencephalogram.
*Significant value.

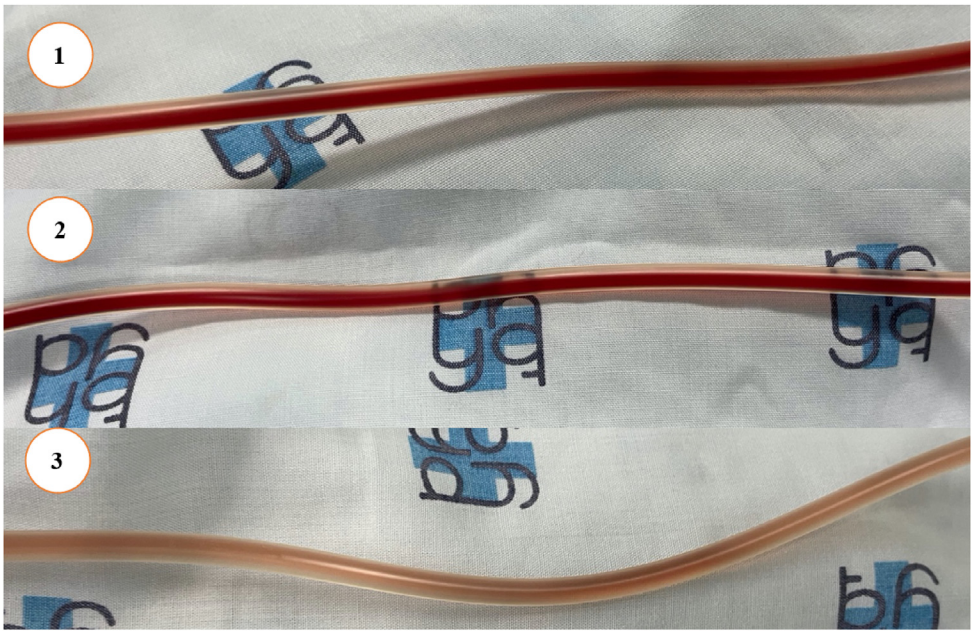

Figure 3. Demonstration of the clearance of subarachnoid hemorrhage through lumbar drainage. On the first day (*1*) and third day (*2*), cerebrospinal fluid appears red with significant blood clots. On the fifth day (*3*), cerebrospinal fluid becomes transparent, and lumbar drainage can be removed to minimize infection rate.

was notable, with P values of 0.004 and 0.009, respectively, showing a meaningful relationship between using LD to clear CSF and the occurrence rates of seizures and epilepsy during the hospital stay. In contrast, the incidence of epilepsy in the subgroups with WFNS grades 4 and 5 was correlated with epileptiform discharge recorded on long-term EEG monitoring ($P = 0.013$) (Table 2).

DISCUSSION

Overview

Numerous studies have reported the incidence of onset seizures in SAH. In our study, the in-hospital seizure rate was 16.9%, with a follow-up epilepsy rate of 5.6%. These figures closely match previous research, which found onset seizure rates of 5.3%–14.7% and epilepsy rates of 5.3%–15.2% among patients with SAH.[9]

Factors Related to In-Hospital Seizure and New-Onset Epilepsy

Choi et al.[8] reported a lower incidence of in-hospital seizure and new-onset epilepsy in patients >40 years old. However, in other studies, age >65 was considered a risk factor for seizure in patients with SAH.[5] Our study shared similar results that patients >50 years old were at higher risk than younger patients. Generally, not exclusively in SAH, older patients have greater risk of epilepsy because many cerebrovascular events and dementing disorders in old age are common causes of seizures.[2,10]

We noted that patients with severe clinical presentation (WFNS grade 4 or 5) were at substantial risk for in-hospital seizure. Nathan et al.[11] showed similar risk in patients who had Hunt and Hess grade 5. Considering the amount of SAH using Fisher grade, patients who had a higher grade (Fisher grade 3 or 4) also had a higher risk of seizure (OR 2.64, 95% CI 1.22–5.69), which was comparable to studies by Lin et al.,[7] Choi et al.[8] (OR 5.1, 95% CI 2.6–10.4), and Molyneux et al.[12] from the International Subarachnoid Aneurysm Trial (Fisher grade ≥2: hazard ratio 1.34, 95% CI 0.62–2.87). In our study, high grades (Fisher 3 and 4) had a stronger correlation with late epilepsy than lower grades (Fisher 1 and 2), with OR of 3.06 and 95% CI of 1.27–10.82. A large amount of SAH increases the intracranial pressure abruptly, negatively affects cerebral perfusion, and reduces venous blood flow. The accumulation of subarachnoid clot interferes with the circulation and absorption of CSF and causes disturbance in the microcirculation.[13] This consequently results in brain tissue injuries, formation of epileptogenic foci, and seizure susceptibility.

In our study, MCA aneurysms were not significantly linked to seizures, but other studies suggest that ruptured MCA aneurysms may increase epilepsy risk.[11] A common hypothesis is that when an MCA aneurysm ruptures, it often causes hematoma, compressing brain tissue and vessels. Accompanying cerebral edema also generates a compressing effect. As there are many lesions associated with ruptured MCA aneurysms, the independent predictive ability of MCA aneurysms must be carefully investigated. In contrast, unruptured aneurysms rarely pose seizure risks, typically only in larger sizes, with thrombus, microbleeds, or gliosis.[14]

The occurrence of seizures following craniotomy can be influenced by several contributing factors, including postoperative hematoma, metabolic and structural disturbances, inadequate seizure prophylaxis, and severity of the physical injury. Our understanding suggests that craniotomy often relates to neuronal injury and ischemia, promoting epileptic focus formation. In contrast, coiling procedures cause less surgical trauma and stress. At our center, treatment modalities are guided by surgical and interventional vascular specialists. They assess clinical features and aneurysm characteristics (morphology, location, accessibility) and discuss treatment options with the patient and their family. Among the 195 patients studied, surgery was preferred over endovascular intervention. This preference can be attributed to the high proportion of MCA aneurysms, which are not always amenable to coiling, and the relatively early age of the study population, who stand to benefit more from surgery in terms of recurrence rates. However, it is worth noting that surgery was associated with a less favorable seizure outcome compared with endovascular intervention (OR 3.28, 95% CI 1.33–8.11). This is similar to the study by Byrne et al.[15]: despite an 11% incidence of onset seizures, 233 patients who received only coil embolization experienced no seizures during the period after the procedure. Only 0.85% of patients experienced late epilepsy across a mean of 1.9 months of follow-up. Several authors indicate higher epilepsy risks in patients who undergo surgery compared with patients who undergo vascular intervention.[16] We observed a significant correlation between treatment modalities (e.g., coiling or clipping surgery) and the likelihood of epilepsy occurrence.

There is considerable evidence regarding the correlation between hydrocephalus and seizures. The occurrence of epileptic seizures among all causes of hydrocephalus ranges from 4.5% to 9.2%[17,18] and among SAH-induced hydrocephalus is 2.01%.[8] This is more significant in a substantial buildup of SAH (Fisher grades 3 and 4). Our study identified SAH-induced hydrocephalus as a predictive risk factor for in-hospital seizures and subsequent development of epilepsy, similar to the findings reported by Choi et al.[8] (OR 4.5, 95% CI 1.9–9.7; P = 0.003).

Vasospasm occurred in 62 of 195 patients and was associated with an increased risk of seizure and epilepsy. This is comparably close to the finding of Nathan et al.[11] (OR 6.88, 95% CI 1.81–26.25). Vasospasm results in reduced perfusion in the arterial territories. The ischemic effect from vasospasm may increase the excitability of the neurons due to the impaired function of ion pumps, thus predisposing them to epileptic activity. The consequence of vasospasm could also explain why there is a link between seizure and infarction. Early clot evacuation (within 48 hours) has been proposed to reduce the occurrence of vasospasm.[16]

A significant correlation between in-hospital seizures and cerebral edema is reported in many studies.[19,20] In hemorrhagic and ischemic strokes, studies also note a connection between seizures and cerebral edema.[20] Cerebral edema causes displacement and compression of brain structures and nerve pathways. The compressed blood vessels result in reduced perfusion. Cytotoxic edema alters cell membrane permeability, affects neuronal conducting function, and reduces the threshold for seizure onset.

To our knowledge, no practical guideline exists for EEG monitoring after SAH. Nevertheless, it is evident that long-term EEG monitoring is beneficial for patients with SAH, particularly patients in whom neurological examination is unreliable, and should be performed as soon as possible within 14 days after SAH.[21,22] EEG monitoring enables the identification of nonconvulsive seizures and the anticipation of delayed cerebral ischemia due to vasospasm before clinical signs appear, typically within 48 hours.[21,22] The presence of seizures and epileptiform discharges indicates underlying epileptogenic injuries, which increase the likelihood of subsequent seizures and necessitate a more rigorous management approach in the follow-up period (OR 3.14, P = 0.012).[23] At our center, long-term EEG monitoring was performed at the physician's discretion, predominantly for severe cases or patients exhibiting decreased levels of consciousness. Though our analysis focused on EEG-related electroconvulsive activity and epileptic outcomes in patients with WFNS grade 4 or 5, there is a reasonable expectation of a comparable correlation across all patients during long-term EEG monitoring. We advocate for broader implementation of continuous EEG, as we believe they could yield more compelling and conclusive results.

Multiple studies have consistently shown the beneficial impact of LD on post-SAH outcomes in terms of Glasgow Outcome Scale and modified Rankin Scale scores. LD effectively manages intracranial pressure and diverts CSF in hydrocephalus cases. Its use for 3–7 days removes subarachnoid blood, reducing reliance on external ventricular drainage and decreasing vasospasm and delayed cerebral ischemia.[16,24-27] Until now, there has been little evidence concerning the impact of LD on seizure outcomes. Our analysis shows a preliminary association between using LD and a potential reduction in the incidence of in-hospital seizures and subsequent epilepsy during follow-up.

Limitations

While our study has identified numerous factors associated with in-hospital seizures and epilepsy rates during follow-up, we acknowledge certain limitations. First, the observations were based on a small sample size and a relatively short follow-up period. Second, the study does not go into depth analyzing the relationship between epilepsy and comorbidities. Third, there was a lack of consensus regarding the indications for conducting long-term EEG monitoring. Due to limited resources at our center, continuous EEG was not available for all cases. Fourth, the routine prescription of antiepileptic drugs without guidelines impeded the evaluation of the effectiveness of prophylactic ASMs on late-onset epilepsy. Last but not least, the retrospective study design introduces potential biases and errors. Therefore, prospective, multicenter, randomized studies should be performed in the future.

CONCLUSIONS

Our analysis revealed that clipping surgery and brain edema were identified as independent predictive factors for in-hospital seizures. Additionally, both onset seizures and in-hospital seizures were found to be independent predictors of epilepsy during the follow-up period. Patients presenting with these risk factors may benefit from continuous EEG monitoring and should be

considered for prophylactic ASM treatment. Furthermore, LD appeared to improve both early and late epileptic outcomes in the patients with Fisher grade 3 or 4.

More detailed studies are needed to track seizure timing and frequency and their link to neurological outcomes. To enhance accuracy, a comprehensive method involving various assessments including neurological examinations, continuous EEG, and LD is crucial. This approach aims to identify reliable indicators for predicting recovery from postanoxic status epilepticus and prevent premature withdrawal of care based on just one predictive method.

CRediT AUTHORSHIP CONTRIBUTION STATEMENT

Viet-Thang Le: Conceptualization, Formal analysis, Investigation, Methodology, Supervision, Validation, Writing — review & editing. **Anh Minh Nguyen:** Conceptualization, Formal analysis, Investigation, Methodology, Project administration, Software, Supervision, Visualization, Writing — review & editing. **Phuc Long Nguyen:** Conceptualization, Data curation, Formal analysis, Methodology, Writing — original draft, Writing — review & editing.

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