

## Epileptic Seizures as a Predictive Factor for Mortality in Patients with Aneurysmal Subarachnoid Hemorrhage: A Retrospective Cohort Study

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### Abstract

**Background:** Aneurysmal subarachnoid haemorrhage (SAH) is a prevalent condition, especially in regions such as Latin America, with a global in-hospital mortality rate of 20%. 26% of patients with SAH present with seizures, leading current guidelines to recommend the prophylactic use of antiepileptic drugs (AEDs) in certain situations.

**Objectives:** To determine the presence of epileptic seizures and the mortality rate in patients hospitalized with a diagnosis of subarachnoid haemorrhage (SAH) secondary to aneurysmal rupture.

**Methods:** A retrospective analytical observational study was carried out in a cohort of patients hospitalized in our institution with a diagnosis of SAH secondary to aneurysmal rupture who suffered epileptic seizures during hospitalization and the associated mortality rate was analysed in a 5-year period (2020-2024). IBM SPSS® (v30.0.0) was used for descriptive statistics, Chi-square test ( $\chi^2$ ), T-test and ANOVA for group comparison and logistic regression to identify predictors of mortality.

**Results:** The study included 45 patients, with a predominance of women (73%) with a mean age of 58 years. 29% of patients had epileptic seizures during hospitalization. Overall mortality was 60%, and patients who experienced seizures had a significantly higher mortality rate (85% /  $p = 0.013$ ) compared to those who did not have seizures.

**Conclusion:** The results suggest a statistically significant correlation between the presence of epilepsy and mortality in patients with aneurysmal SAH. Although the presence of epileptic seizures in these patients is relatively frequent, this finding underlines the need for further studies to identify specific factors that contribute to increased mortality in this clinical context.

**Keywords:** Aneurysmal Rupture; Epilepsy; Mortality; Subarachnoid Haemorrhage; Fisher

### Abbreviations

ACoA: Anterior Communicating Artery; AEDs: Antiepileptic Drugs; aSAH: Aneurysmal Subarachnoid Haemorrhage; Carotid-Ophthalmic Segment: Carotid-Ophthalmic Segment Artery; CNS: Central Nervous System; CTA: Computed Tomography Angiography; EEG: Electroencephalography; ES: Epileptic Seizures; GTCS: Generalized Tonic-Clonic Seizures; H&H: Hunt and Hess Scale; ICA: Internal Carotid Artery; IPH: Intraparenchymal Haemorrhage; MCA: Middle Cerebral Artery; mFisher: Modified Fisher Scale; MR: Medical Record; NCCT: Non-Contrast Computed Tomography; PComA: Posterior Communicating Artery; RR: Relative Risk; SAH: Subarachnoid Haemorrhage; UL: Unlocalized

## Introduction

Subarachnoid haemorrhage (SAH) is defined as the presence of blood within the subarachnoid space and may result from traumatic (most common) or spontaneous causes [1]. Among the latter, aneurysmal rupture represents the leading etiology and constitutes the focus of the present study. Aneurysmal subarachnoid haemorrhage (aSAH) is an acute haemorrhagic cerebrovascular event characterized by high mortality and significant morbidity among the affected population [2].

It is particularly prevalent in regions such as Latin America, Oceania, and Central Asia [3], with an estimated global incidence of 6.1 per 100,000 inhabitants per year and a worldwide prevalence of approximately 8.09 million cases [4]. The risk of occurrence increases with age, peaking between 50 and 60 years, with a specific predilection for the female sex, showing a relative risk (RR) of 1.3 compared with males [5].

Approximately 22 - 26% of patients with aSAH die before receiving medical attention, and in-hospital mortality rates range between 19% and 20% [6,7]. These figures likely underestimate the true burden of disease, as they do not account for the long-term impact on survivors' quality of life or the economic losses associated with reduced productivity [8,9].

It is estimated that approximately 26% of patients with aSAH develop epileptic seizures (ES); however, more recent studies suggest a lower incidence, ranging from 7.8% to 15.2% [10-12].

Factors such as rupture of middle cerebral artery (MCA) aneurysms, high Hunt and Hess (H&H) grades (>3), modified Fisher scale (mFisher) grades III/IV, hydrocephalus, intraparenchymal haemorrhage (IPH), and cortical infarction have been identified as predisposing factors for the development of ES. These observations have led to recommendations for the prophylactic use of antiepileptic drugs (AEDs) in current clinical guidelines; however, the utility of this strategy remains controversial [13-16].

The impact of epileptic seizures on clinical outcomes and mortality in patients with aSAH remains incompletely defined, largely due to the complex interaction of multiple prognostic factors affecting this population.

## Objectives of the Study

To determine the presence of epileptic seizures and the mortality rate in hospitalized patients diagnosed with subarachnoid haemorrhage secondary to aneurysmal rupture.

## Materials and Methods

An observational, analytical, retrospective study was conducted in a cohort of patients admitted to our institution with a diagnosis of subarachnoid hemorrhage (SAH) secondary to aneurysmal rupture over a 5-year period (January 2020 to October 2024).

### Inclusion criteria:

- Patients aged ≥18 years with a confirmed diagnosis of SAH secondary to aneurysmal rupture.
- Presence of epileptic seizures (ES) documented in the medical record (MR).
- Absence of a prior history of epilepsy or epileptic seizures before the occurrence of SAH.

### Exclusion criteria

- Patients with SAH secondary to trauma, rupture of arteriovenous malformations, vasculitis, or other structural lesions.
- Lack of documented epileptic seizures in the medical record.

The diagnosis of SAH was established by non-contrast computed tomography (NCCT) at admission, identifying the presence of blood (hyperdense lesions) within the subarachnoid space, primarily in the basal cisterns and/or the sylvian fissure [17]. In cases in which NCCT findings were inconclusive, the diagnosis was confirmed by lumbar puncture with a positive three-tube test, defined as the persistent presence of red blood cells across samples obtained from a non-traumatic puncture [18].

Demographic data (age, sex), relevant medical history (epilepsy or other seizure disorders), and clinical characteristics at admission were recorded. Initial clinical status was assessed using the Hunt and Hess (H&H) scale, with high grade defined as  $\geq 3$ , which was used to estimate mortality and neurological prognosis based on predefined clinical criteria. In parallel, admission NCCT scans were graded using the modified Fisher scale (mFisher), considering grades III and IV as high-grade tomographic severity (Table 1 and 2).

Grade	Characteristics
0	Incidental aneurysm
1	Asymptomatic
	Mild headache or minimal nuchal rigidity
2	Cranial nerve involvement
	Moderate to severe headache
	Nuchal rigidity
3	Mild focal neurological deficit
	Lethargy/confusion
4	Moderate to severe hemiparesis
	Stupor
5	Coma
	Decerebrate posturing
	Agonal state

**Table 1:** Hunt and Hess scale (H&H).

Grade	Characteristics	Intraventricular haemorrhage
0	No blood	-
I	Thin focal or diffuse SAH	Absent
II	Thin focal or diffuse SAH	Present
III	Thick focal or diffuse SAH	Absent
IV	Thick focal or diffuse SAH	Present

**Table 2:** Modified Fisher scale (mFisher).

*Note:* Modified tomographic grading scale used to predict the risk of cerebral vasospasm.

Etiological diagnosis of SAH and aneurysm localization were established using computed tomography angiography (CTA) with three-dimensional reconstruction processed with RadiAnt/Horos software, or by digital cerebral angiography.

Epileptic seizures (ES) were defined as “the transient occurrence of signs and/or symptoms resulting from abnormal excessive or synchronous neuronal activity in the brain,” according to the International League Against Epilepsy (ILAE) [19,20]. Patients with any type of ES were included in the analysis, including generalized tonic-clonic seizures (GTCS), focal seizures with impaired awareness, absence seizures, behavioral changes or alterations in the level of consciousness compared with baseline, unexplained depressed mental status, failure to respond to ventilatory weaning, and/or abnormal electrical activity detected by electroencephalography (EEG) during hospitalization.

### Statistical analysis

A descriptive analysis was performed using measures of central tendency (mean and standard deviation) and percentages for categorical variables. Outcome variables were dichotomized for analysis. Group comparisons were conducted using the chi-square ( $\chi^2$ ) test, Student's *t* test, and analysis of variance (ANOVA), as appropriate.

To identify predictors of mortality, logistic regression analysis was performed using a backward stepwise selection model. Bidirectional interactions among all variables with independent predictive value were explored. A *p* value  $<0.05$  was considered statistically significant, with a 95% confidence interval. Statistical analysis was conducted using IBM SPSS® software (version 30.0.0).

### Results

The study included a sample of 45 patients admitted to our institution with a diagnosis of subarachnoid hemorrhage (SAH) secondary to aneurysmal rupture over a 5-year period (January 2020 to October 2024). There was a predominance of female patients (73%), with a mean age of 58 years ( $SD \pm 17.27$ ), ranging from 19 to 95 years, reflecting a broad age distribution. Patients were subclassified into two age groups ( $> 55$  years and  $\leq 55$  years); those older than 55 years accounted for 53.3% of the sample ( $n = 24$ ).

An independent comparative analysis between sexes yielded a *t* value of 0.48 and a *p* value of 0.64. No statistically significant differences were found in age distribution according to sex (Table 3 and figure 1).

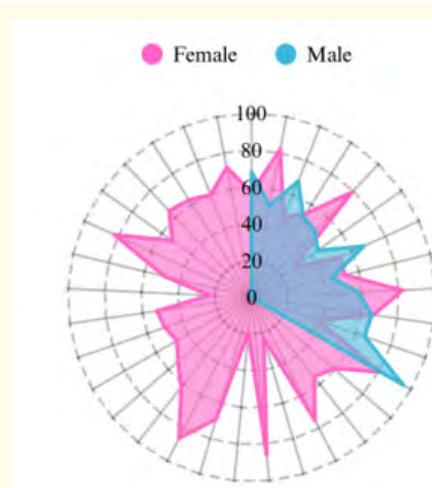
Different aneurysm locations were evaluated. The most frequent location was the posterior communicating artery (PComA), accounting for 35.6% ( $n = 16$ ), followed by the anterior communicating artery (ACoA) in 20% ( $n = 9$ ). In 15% of cases ( $n = 7$ ), aneurysm location was unlocalized due to the absence of etiological imaging studies. Middle cerebral artery (MCA) aneurysms accounted for 8.9% ( $n = 4$ ), vertebrobasilar aneurysms for 6.7% ( $n = 3$ ), internal carotid artery (ICA) aneurysms for 4.4% ( $n = 2$ ), carotid-ophthalmic aneurysms for 4.4% ( $n = 2$ ), paraclinoid aneurysms for 2.2% ( $n = 1$ ), and pericallosal aneurysms for 2.2% ( $n = 1$ ) (Table 3 and figure 2).

Variable	n (%)	p value
<b>Demographics</b>		
Total	45 (100)	
Female	33 (73)	0.48*
Age (years)	58 (DE $\pm 17.27$ ) (range 19-95)	
Age $\geq 55$ years	24 (53.3)	0.64#
<b>Epileptic seizures (ES)</b>		
	13 (29)	
<b>Mortality</b>		
	27 (60)	
<b>Treatment</b>		
Phenytoin	24 (53.3)	

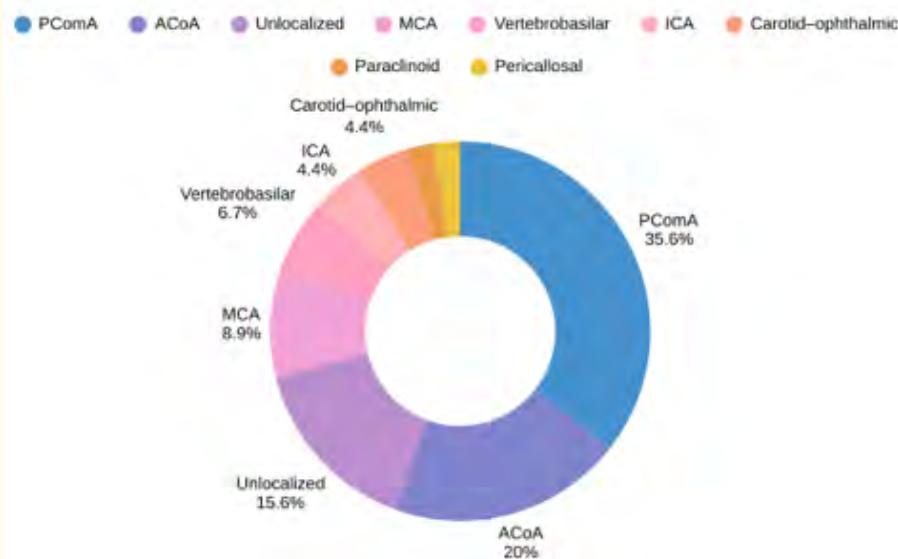
Levetiracetam	21 (46.7)	
<b>Rebleeding</b>	6 (13)	
<b>Hunt and Hess scale</b>		
0	1 (2.2)	
1	3 (6.7)	
2	9 (20)	
3	13 (28.9)	
4	7 (15.6)	
5	12 (26.7)	
<b>Modified Fisher scale</b>		
0	2 (4.4)	
I	1 (2.2)	
II	2 (4.4)	
III	13 (28.9)	
IV	27 (60)	
<b>Aneurysm location</b>		
PComA	16 (35.6)	
ACoA	9 (20)	
Unlocalized	7 (15.6)	
MCA	4 (8.9)	
Vertebrobasilar	2 (6.7)	
ICA	2 (4.4)	
Carotid-ophthalmic	2 (4.4)	
Paraclinoid	1 (2.2)	
Pericallosal	1 (2.2)	

**Table 3:** General characteristics of the studied population.

\*Paired t test. #Chi-square ( $\chi^2$ ) test.



**Figure 1:** Age distribution by sex. Trends or potential differences between males and females across age ranges can be observed.



**Figure 2:** Aneurysm locations. Frequencies are expressed as percentages. PComA: posterior communicating artery. ACoA: anterior communicating artery. Unlocalized: without identified location. MCA: Middle Cerebral Artery; ICA: Internal Carotid Artery; Carotid-Ophthalmic: Carotid-Ophthalmic Segment.

Twenty-nine percent of patients ( $n = 13$ ) experienced epileptic seizures (ES) during hospitalization. Overall mortality among patients with aSAH was 60%. Within the subgroup of patients who developed ES, mortality increased to 92%, with a  $p$  value of 0.013, demonstrating a statistically significant difference compared with patients without seizures (Table 4 and 5).

All patients received antiepileptic drugs (AEDs) for at least 7 days. Phenytoin was administered to 53.3% of patients ( $n = 24$ ), with an initial loading dose of 17 mg/kg at admission followed by a maintenance dose of 100 mg every 8 hours starting 24 hours later. Levetiracetam was administered to 46.7% of patients ( $n = 21$ ), with a loading dose of 3000 mg diluted in 500 mL of normal saline infused over 30 minutes, followed by 1500 mg every 12 hours. A statistically significant difference in mortality rates was observed between patients who received phenytoin and those who did not.

The most frequent Hunt and Hess (H&H) grades were 3 and 5, with the following distribution: grade 0, 2.2% ( $n = 1$ ); grade 1, 6.7% ( $n = 3$ ); grade 2, 20% ( $n = 9$ ); grade 3, 28.9% ( $n = 13$ ); grade 4, 15.6% ( $n = 7$ ); and grade 5, 26.7% ( $n = 12$ ). For further analysis, patients were grouped into low grade (grades 0-2) and high grade (grades 3-5). Seventy-one percent of patients presented with high-grade SAH, and mortality in this group reached 78.1%, with a  $p$  value  $<0.001$ , demonstrating a statistically significant correlation between high H&H grade and mortality. High-grade H&H was also evaluated as a predictor of epileptic seizures, with no statistically significant differences observed.

The majority of patients were classified as high grade on the modified Fisher scale (grades III and IV), representing 88.9% of cases. A statistically significant difference between groups was observed ( $p < 0.001$ ), with higher modified Fisher grades associated with both increased mortality and a greater predisposition to the development of epileptic seizures (Table 4 and 5; Figure 3).

Additionally, patients who experienced rebleeding were found to have a higher likelihood of developing epileptic seizures.

Characteristics	Epileptic seizures (ES)				p value	
	Yes		No			
	n (%)	n (%)	n (%)	n (%)		
	13 (29)	32 (71)		45		
<b>Hunt and Hess scale</b>						
Low grade	2 (15.4)	11 (34.4)	13 (29)		0.36 <sup>a</sup>	
High grade	11 (84.6)	21 (65.6)	32 (71)			
<b>Modified Fisher scale</b>						
Low grade	0 (0)	5 (11.1)	5 (11.1)		0.015 <sup>a</sup>	
High grade	13 (28.9)	27 (60)	40 (88.9)			
<b>Aneurysm location</b>						
PCoM A	3 (6.7)	13 (28.9)	16 (35.6)		0.19 <sup>#</sup>	
ACoA	4 (8.9)	5 (11.1)	9 (20)			
Unlocalized	4 (8.9)	3 (6.7)	7 (15.6)			
MCA	2 (4.4)	2 (4.4)	4 (8.9)			
Vertebrobasilar	0 (0)	3 (6.7)	3 (6.7)			
ICA	0 (0)	2 (4.4)	2 (4.4)			
Carotid-ophthalmic	0 (0)	2 (4.4)	2 (4.4)			
Paraclinoid	0 (0)	1 (2.2)	1 (2.2)			
Pericallosal	0 (0)	1 (2.2)	1 (2.2)			
<b>Rebleeding</b>						
No	7 (15.6)	32 (71.1)	39 (86.7)		<0.001 <sup>#</sup>	
Yes	6 (13.3)	0 (0)	6 (13.3)			
<b>Age (years)</b>						
≥55	6 (13.3)	18 (40)	24 (53.3)		0.77*	
≤55	7 (15.6)	14 (31.1)	21 (46.7)			

**Table 4:** Predictors of epilepsy in patients with aSAH.

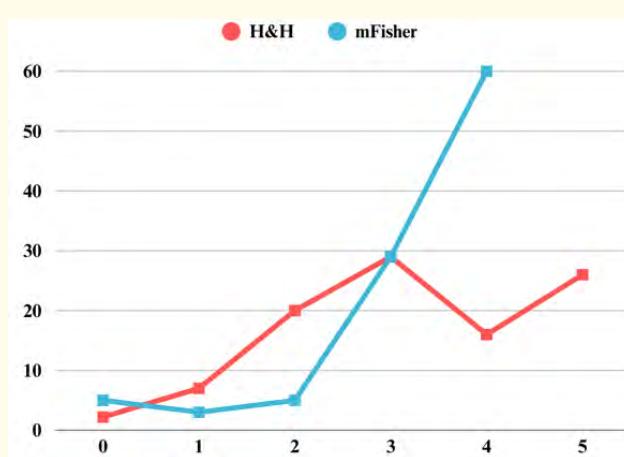
\*: Paired t test. #: Chi-square ( $\chi^2$ ) test.

Characteristics	Mortality				p value	
	Yes		No			
	n (%)	n (%)	n (%)	n (%)		
	27 (60)	18 (40)	45			
<b>Epileptic seizures (ES)</b>					0.013 <sup>a</sup>	
Yes	12 (92.3)	1 (7.7)	13 (29)			
No	15 (46.9)	17 (53.1)	32 (71)			
<b>Treatment</b>						
Phenytoin	23 (85.2)	1 (5.6)	24 (53.3)		<0.001 <sup>a</sup>	
Levetiracetam	4 (14.8)	17 (94.4)	21 (46.7)			

<b>Hunt and Hess scale</b>				
Low grade	2 (15.4)	11 (84.6)	13 (29)	<0.001 <sup>a</sup>
High grade	25 (78.1)	7 (21.9)	32 (71)	
<b>Modified Fisher scale</b>				
Low grade	0 (0)	5 (11.2)	5 (11.2)	0.015 <sup>a</sup>
High grade	27 (67.5)	13 (32.5)	40 (88.8)	
<b>Aneurysm location</b>				
PCoA	10 (22.2)	6 (13.3)	16 (35.6)	0.3 <sup>#</sup>
ACoA	6 (13.3)	3 (6.7)	9 (20)	
Unlocalized	4 (8.9)	3 (6.7)	7 (15.6)	
MCA	3 (6.7)	1 (2.2)	4 (8.9)	
Vertebrobasilar	0 (0)	3 (6.7)	3 (6.7)	
ICA	2 (4.4)	0 (0)	2 (4.4)	
Carotid-ophthalmic	1 (2.2)	1 (2.2)	2 (4.4)	
Paracclinoid	1 (2.2)	0 (0)	1 (2.2)	
Pericallosal	0 (0)	1 (2.2)	1 (2.2)	
<b>Rebleeding</b>				
No	22 (48.9)	17 (37.8)	39 (86.7)	0.42 <sup>#</sup>
Yes	5 (11.1)	1 (2.2)	6 (13.3)	
<b>Age (years)</b>				
≥55	16 (35.6)	8 (17.8)	24 (53.3)	0.5 <sup>#</sup>
≤55	11 (24.4)	10 (22.2)	21 (46.7)	

**Table 5:** Predictors of mortality in patients with aSAH.

#: Chi-square ( $\chi^2$ ) test.  $\alpha$ : One-way ANOVA.



**Figure 3:** Modified Fisher scale (mFisher) and Hunt and Hess (H&H) grades in our study population. Frequencies are expressed as percentages.

## Discussion

In this study, epileptic seizures (ES) were observed in 29% of patients diagnosed with aneurysmal subarachnoid hemorrhage (aSAH) and were independently associated with a significant increase in mortality, as evidenced by the fact that 92.3% of patients who died had experienced ES.

A relevant finding was that 50% of patients with aneurysms located in the middle cerebral artery (MCA) developed ES, in agreement with existing literature, which describes a similar predisposition in this subgroup.

Regarding antiepileptic drug (AED) therapy, all patients received prophylaxis with either phenytoin or levetiracetam during the first 7 days of hospitalization. A statistically significant correlation was identified between AED use and mortality. In particular, patients treated with phenytoin exhibited higher mortality rates, supporting previous reports suggesting a potential negative impact of this drug in comparable clinical settings.

Additionally, patients with higher grades on the Hunt and Hess clinical scale exhibited both a higher incidence of ES and increased mortality compared with those with lower grades, underscoring the importance of initial clinical status as a crucial prognostic factor [21-23].

Our findings reinforce the hypothesis that the development of ES during hospitalization for aSAH has a significant impact on patient mortality, positioning seizures as a relevant marker in the clinical course of this condition. Compared with previous studies, both the frequency of ES and the mortality rate observed in our cohort were higher, which may be related to specific characteristics of the population studied [24-28].

Furthermore, hemorrhage severity-reflected by the extent of blood on initial computed tomography (CT) scans and by clinical grade at admission-was identified as an important risk factor for both the development of ES and increased associated mortality [3,29-31].

Several mechanisms may explain the relationship between epileptic seizures and increased mortality in these patients. Seizures themselves may exacerbate secondary brain injury following aSAH [32,33], additionally, AED treatment-particularly phenytoin-has been associated with worse motor outcomes, possibly due to interference with alternative neuronal networks required for neurological recovery [11,34-36].

It should be emphasized that patients with MCA aneurysms and those presenting with high clinical and radiological grades have a greater predisposition to develop ES, findings that are consistent with current literature. Clinical guidelines recommend prophylactic AED use in patients with specific characteristics, such as high clinical grade (Hunt and Hess >3), high radiological grade (modified Fisher >3), ruptured MCA aneurysms, intraparenchymal hemorrhage (IPH), hydrocephalus, and cortical infarction, for at least 7 days to prevent early seizures [12,37]. Conversely, in patients without these features, prophylactic treatment does not appear to confer significant benefit [34,35,38].

Several limitations must be considered when interpreting our results. First, mortality in patients with aneurysmal SAH is multifactorial and influenced by numerous variables, including central nervous system infections, complications related to prolonged mechanical ventilation, intracranial hypertension secondary to acute hydrocephalus, systemic infections, multiorgan failure, cerebral infarction secondary to vasospasm, and hydroelectrolytic disturbances, among others.

Another important limitation is that the diagnosis of epileptic seizures was primarily based on clinical observations-such as changes in neurological examination or lack of neurological responsiveness-without the support of continuous electroencephalographic (EEG)

monitoring, a common limitation in public healthcare settings due to restricted resource availability. This may have led to underestimation or overestimation of ES cases. Additionally, in some medical records, the precise timing of seizure onset relative to aneurysmal rupture could not be determined, limiting our ability to analyze early versus late seizures.

Finally, this study was conducted at a single center, which may limit the generalizability of the findings to other populations or clinical settings.

## **Conclusion**

The results of this study highlight a statistically significant correlation between the presence of epileptic seizures and mortality in patients with aneurysmal subarachnoid hemorrhage. This finding reinforces the importance of identifying and actively monitoring the occurrence of epileptic seizures in this patient population as a potential marker of poor prognosis.

The relatively high frequency of epileptic seizures observed in our cohort, together with their adverse impact on survival, raises important questions regarding the underlying mechanisms that may mediate this association, including factors such as aneurysm location, initial clinical status, and pre-existing comorbidities.

Furthermore, the efficacy and safety of antiepileptic drug (AED) therapy in this clinical context remain controversial. These findings suggest the need for a critical re-evaluation of current recommendations for seizure prophylaxis and management in patients with aSAH, taking into account potential differences in AED response among patient subgroups, as well as the criteria used for seizure detection in resource-limited settings.

Finally, these results underscore the importance of future multicenter studies with larger sample sizes aimed at identifying specific predictive factors and optimized therapeutic strategies for this high-risk population. A better understanding of the determinants of mortality in patients with aSAH and epileptic seizures could not only improve clinical management but also reduce the mortality rates associated with this highly lethal condition.

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## **Conflict of Interest**

The author declares no financial interests or conflicts of interest.

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