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Risk Factors for Mortality in the Acute Phase of Hemorrhagic Stroke at Befelatanana University Hospital, Antananarivo, Madagascar

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Abstract

Background: Hemorrhagic stroke is characterized by the sudden onset of a neurological deficit of presumed vascular origin, resulting from blood extravasation into the cerebral parenchyma. This study aimed to identify clinical and paraclinical risk factors associated with mortality during the acute phase of hemorrhagic stroke at Befelatanana University Hospital. **Methods:** We conducted a retrospective case-control study, over a 30-month period. All hospitalized patients diagnosed with hemorrhagic stroke confirmed by neuroimaging were included. Cases were defined as patients who died within the first 7 days of admission due to neurological causes; controls were patients discharged alive from the neurology department. Independent predictors of mortality were subsequently identified using multivariate binary logistic regression. **Results:** A total of 313 patients were included, with a slight male predominance (50.8%) and a mean age of 55.5 ± 11.3 years. Factors significantly associated with mortality on bivariate analysis included: mean arterial pressure ≥ 130 mmHg ($p < 0.0001$), hyperthermia ≥ 38˚C ($p < 0.0001$), hyperglycemia ≥ 2 g/L (OR, 17.35; $p < 0.001$), Glasgow Coma Scale (GCS) score ≤ 13 (OR, 39.94 for 8 - 13, and non-estimable for ≤8), National Institutes of Health Stroke Scale (NIHSS) score ≥ 15 (OR, 32.66), modified Rankin Scale (mRS) score ≥ 3 ($p < 0.0001$), clinical and radiologic signs of cerebral herniation (OR, 97.52 and 19.47, respectively), infratentorial hematoma ($p = 0.044$), Intracerebral Hemorrhage (ICH) score between 3 and 5 (OR, 112.21), acute hydrocephalus (OR, 41.51), and neurosurgical intervention performed for severe presentations

(OR, 110). On multivariate binary logistic regression only four variables remained independently associated with in-hospital mortality: GCS < 14 (adjusted OR, 35.47), clinical signs of cerebral herniation (adjusted OR, 20.43), acute hydrocephalus (adjusted OR, 9.69), and mean arterial pressure ≥ 130 mmHg (adjusted OR, 3.53). **Conclusions:** Identification of these risk factors may improve early management strategies during the acute phase of stroke. Multivariate analysis confirms altered consciousness, cerebral herniation, hydrocephalus, and elevated mean arterial pressure as independent early predictors, which should guide risk stratification and clinical decision-making from admission.

Keywords

Hemorrhagic Stroke, Risk Factors, Mortality, Madagascar

1. Introduction

Hemorrhagic stroke is defined as a sudden loss of neurological function caused by extravasation of blood into the brain parenchyma in the absence of trauma or surgical intervention [1]. It is associated with high morbidity and mortality rates. The 30-day mortality rate ranges from 30% to 55% [2] with approximately half of deaths occurring during the acute phase, particularly within the first 48 hours [1].

The objective of this study was to determine the risk factors associated with mortality in patients with hemorrhagic stroke during the acute in-hospital phase and to describe their epidemiologic, clinical, and radiologic profiles.

2. Methods

This retrospective case-control study was conducted in the Medical Intensive Care Unit (MICU) and the Neurology Department of the Centre Hospitalier Universitaire Joseph Raseta Befelatanana (CHU JRB) in Antananarivo, Madagascar, over a 30-month period (January 1, 2022, to June 30, 2024).

All hospitalized patients diagnosed with hemorrhagic stroke confirmed by computed tomography (CT) or magnetic resonance imaging (MRI) were included. The following patients were excluded: those with traumatic intracranial hemorrhage, subarachnoid hemorrhage, hemorrhagic transformation of ischemic stroke, postoperative bleeding, or incomplete medical records. No matching was performed. Recruitment was exhaustive for all eligible patients.

Cases were defined as patients who died within the first 7 days of hemorrhagic stroke due to neurological causes during admission to the MICU. Controls were defined as patients discharged alive from the neurology department.

We evaluated associations between mortality and the following variables: demographic characteristics (age, sex), clinical parameters (measured at admission: symptom onset, medical history, vital signs, neurological status assessed using the

Glasgow Coma Scale (GCS), the National Institutes of Health Stroke Scale (NIHSS), and the modified Rankin Scale (mRS), as well as the presence of clinical signs of cerebral herniation), radiologic findings (assessed on admission CT scan: hematoma location, volume, ICH score, radiologic herniation signs, and presence of acute hydrocephalus), and therapeutic factors (including neurosurgical intervention status).

Bivariate associations between mortality and each candidate variable were assessed using the chi-square test (or Fisher's exact test when expected cell counts were <5), with calculation of the crude odds ratio (OR) and its 95% confidence interval by Woolf's method, applying the Haldane-Anscombe continuity correction for null cells; the comparison of age between groups used the non-parametric Mann-Whitney U test, as the distribution was non-Gaussian. Independent predictors of in-hospital mortality were then identified using multivariate binary logistic regression (maximum-likelihood estimation, Newton-Raphson algorithm), with mortality as the dependent variable. Candidate variables were selected on the basis of 1) their significance on bivariate analysis ($p < 0.20$), 2) their recognized clinical relevance in the hemorrhagic stroke prognosis literature, and 3) the absence of major collinearity with other retained covariates. The composite ICH score was not entered into the multivariate model alongside its own components (Glasgow score, hematoma location) to avoid statistical redundancy and circularity; similarly, therapeutic variables applied in direct response to clinical severity (neurosurgery, resuscitation, osmotherapy) were excluded from the adjusted model owing to a likely reverse-causation bias, and are presented and discussed only as bivariate findings. Model discrimination was assessed using the area under the receiver operating characteristic curve (AUC) and the McFadden pseudo-R^2. All analyses were performed using Python (pandas, NumPy, SciPy), superseding the online calculator used in earlier drafts of this analysis. Statistical significance was defined as a p value ≤ 0.05.

The study protocol was reviewed and approved by the heads of the departments of the two study sites before data collection commenced. Given the retrospective nature of the chart review, individual informed consent was waived. All patient data were anonymized and handled in accordance with applicable data protection regulations.

3. Results

Among 7113 medical records screened, 313 patients were included for analysis, including 78 deaths (24.9%) and 235 survivors (75.1%). The mean age was 55.5 ± 11.3 years (range, 24 - 86), with a male predominance (50.8%; sex ratio, 1.03).

Mortality risk was significantly increased in patients presenting with:

- Older age (mean 58.1 ± 11.6 years among deaths vs. 54.7 ± 11.0 years among survivors; $p = 0.026$, Mann-Whitney U test), this bivariate association did not persist as an independent predictor on multivariate analysis (adjusted OR, 0.98 per year; $p = 0.50$).

- Mean arterial pressure (MAP) ≥ 130 mmHg at admission (crude OR, 2.38; 95% CI, 1.15 - 4.91), isolated systolic (≥180 mmHg) or diastolic (≥110 mmHg) blood pressure thresholds were not, on their own, significantly associated with mortality when analyzed by pressure category; MAP ≥ 130 mmHg remained an independent predictor on multivariate analysis (adjusted OR, 3.53; 95% CI, 1.16 - 10.75; $p = 0.027$).
- Hyperthermia at admission (crude OR, 21.6 for 38˚C - 40˚C and 40.5 for ≥40˚C, relative to 36˚C - 38˚C; both $p < 0.0001$). This was not included in the multivariate model.
- Capillary blood glucose ≥ 2 g/L (crude OR, 17.35; 95% CI, 6.00 - 50.18; $p < 0.001$). This was not included in the multivariate model.

Neurological severity was strongly associated with mortality:

- GCS score 8 - 13 (OR, 39.94). The multivariate model, using GCS < 14 vs 14 - 15, confirms altered consciousness as the strongest independent predictor of mortality (adjusted OR, 35.47; 95% CI, 10.70 - 117.53).
- NIHSS score 15 - 20 (OR, 32.66), NIHSS was not included in the multivariate model.
- mRS score 3 - 5 (crude OR, 28.72; 95% CI, 1.74 - 473.77; $p < 0.0001$, vs. 0 - 2), no deaths occurred among the 36 patients with baseline mRS 0 - 2 and this was not included in the multivariate model.
- Clinical signs of cerebral herniation (crude OR, 97.52; 95% CI, 35.68 - 266.57). This was confirmed on multivariate analysis as an independent predictor (adjusted OR, 20.43; 95% CI, 5.53 - 75.53; $p < 0.0001$).

Radiologic predictors included:

- Infratentorial hematoma location (crude OR, 2.78; 95% CI, 1.22 - 6.32). This was not remained significant on multivariate analysis, although a strong trend persisted (adjusted OR, 3.86; 95% CI, 0.88 - 16.99; $p = 0.074$).
- ICH score 3 - 5 (OR, 112.21; 95% CI, 46.43 - 271.17). This was not included in the multivariate model, to avoid circularity with its own components (Glasgow score, hematoma location).
- Radiologic signs of herniation (OR, 19.47; 95% CI, 9.68 - 39.61).
- Acute hydrocephalus (crude OR, 41.51; 95% CI, 15.41 - 111.86). This was confirmed on multivariate analysis as an independent predictor (adjusted OR, 9.69; 95% CI, 2.19 - 42.92; $p = 0.003$).

Additionally, neurosurgical intervention was strongly associated with mortality in bivariate analysis (OR, 110.67; 95% CI, 25.68 - 477.02, recalculated from OR 111.15). This association, together with that of osmotherapy (mannitol; crude OR, 14.68) and resuscitation/intubation (crude OR, 42.23), most likely reflects reverse causation: these interventions were undertaken in response to clinical severity and deterioration rather than being themselves a cause of death. For this reason, and unlike in the original interpretation of these results, this cannot be read as evidence that withholding neurosurgery (for instance owing to family refusal) directly worsens prognosis, and these variables were excluded from the multivariate model.

The clinico-radiologic characteristics of our patients are presented in Table 1. The independent predictors of mortality identified by multivariate logistic regression are presented in Table 2 and Figure 1 and Figure 2.

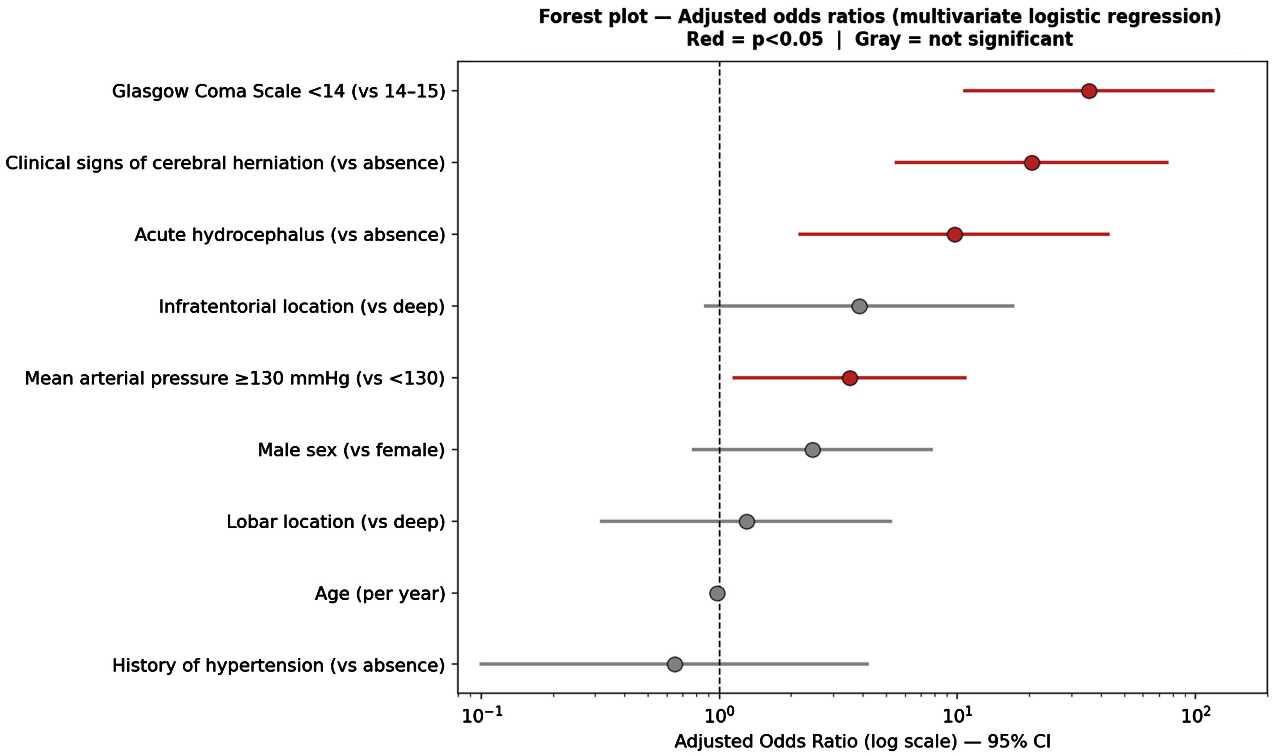

Figure 1. Forest plot of adjusted odds ratios from the multivariate logistic regression model (in-hospital mortality of hemorrhagic stroke, n = 313). Bars represent the 95% CI; red markers denote statistically significant associations ($p < 0.05$).

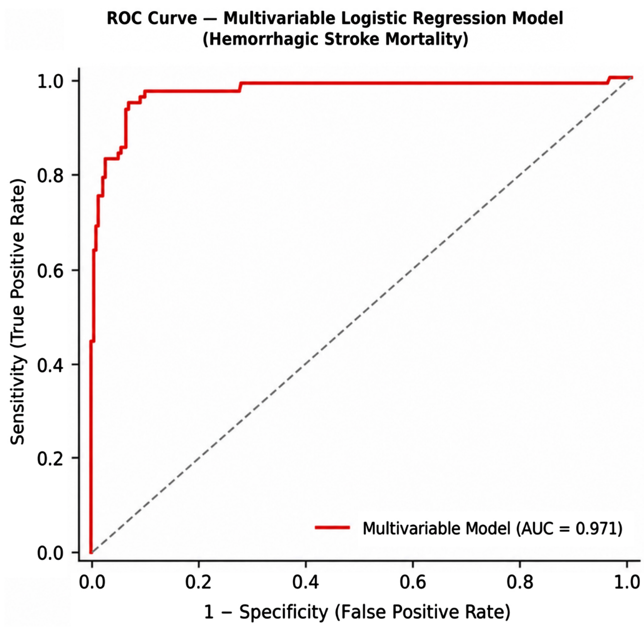

Figure 2. Receiver operating characteristic (ROC) curve of the multivariate logistic regression model predicting mortality (AUC = 0.971), indicating excellent model discrimination.

Table 1. Mortality risk factors according to clinico-radiologic characteristics of patients with hemorrhagic stroke seen at CHU JRB from January 1, 2022, to June 30, 2024 (N = 313).

Parameters	Cases n = 78	Controls n = 235	OR [95% CI]	p value
Age				
18 - 65 years	53	195	2.243 [1.253 - 4.018]	0.003
≥65 years	25	41	Ref	
Sex				
Male	45	114	1.459 [0.870 - 2.446]	0.075
Female	33	122	Ref	
Residence				
Rural	29	69	1.432 [0.836 - 2.453]	0.095
Urban	49	167	Ref	
Presenting symptoms				
Neurologic deficit	44	223	-	0.016
Intracranial hypertension	12	1	0.473 [0.159 - 1.412]	0.089
Seizures	5	12	-	
Symptom onset				
≤6 h	21	44	0.622 [0.342 - 1.131]	0.059
6 - 48 h	57	192	Ref	
Medical history				
None	3	2	-	
Hypertension	69	215	0.75 [0.33 - 1.72]	0.649
Dyslipidemia	13	27	0.320 [0.048 - 2.162]	0.121
Previous stroke	5	25	0.133 [0.018 - 1.016]	0.025
Diabetes mellitus	9	17	0.352 [0.050 - 2.514]	0.149
Smoking	19	47	0.269 [0.042 - 1.743]	0.08
Alcohol use	29	80	0.241 [0.038 - 1.520]	0.065
Admission GCS				
≤8	46	0	Not estimable (complete separation: 46/46 deaths, 0 survivors)	<0.001
8 - 13	26	23	39.94 [14.89 - 107.11]	<0.001
14 - 15 (reference)	6	212	Ref	
Admission NIHSS				
5 - 14 (reference)	4	182	Ref	
15 - 20	28	39	19.48 [1.11 - 341.37]	<0.001
>20	46	1	837.00 [32.21 - 21749.48]	<0.001

Continued

Admission mRS				
0 - 2 (reference)	0	36	Ref	
3 - 5	78	199	28.72 [1.74 - 473.77]	0.004
Clinical signs of cerebral herniation				
Yes	53	5	97.52 [35.68 - 266.57]	<0.001
No (reference)	25	231	Ref	
Hematoma Location				
Supratentorial (reference)	53	195	Ref	
Infratentorial	25	41	2.78 [1.22 - 6.32]	0.044
Number of hematomas				
1 (reference)	76	236	Ref	
≥2	2	0	6.210 [0.555 - 69.44]	0.065
Radiologic signs of cerebral herniation				
Yes	67	56	19.47 [9.68 - 39.61]	<0.001
No (reference)	11	180	Ref	
ICH Score				
0 - 2 (reference)	8	219	Ref	
3 - 5	70	17	112.21 [46.43 - 271.17]	<0.001
Acute Hydrocephalus				
Yes	37	5	41.51 [15.41 - 111.86]	<0.001
No (reference)	41	231	Ref	

ICH = Intracerebral Hemorrhage; GCS = Glasgow Coma Scale; NIHSS = National Institutes of Health Stroke Scale; mRS = modified Rankin Scale; OR = Odds Ratio; CI = Confidence Interval; Ref = Reference category.

Table 2. Independent predictors of in-hospital mortality—multivariate binary logistic regression (n = 313; 9 covariates; likelihood-ratio $p < 0.0001$; McFadden pseudo-R2 = 70.8%; AUC = 0.971). Bold rows: $p < 0.05$.

Variable (vs reference)	Adjusted OR	95% CI	p value
Age (per year)	0.98	0.94 - 1.03	0.500
Male gender (vs female)	2.45	0.78 - 7.73	0.127
Glasgow Coma Scale < 14 (vs 14 - 15)	**35.47**	**10.70 - 117.53**	**<0.0001**
Infratentorial location (vs deep)	3.86	0.88 - 16.99	0.074
Lobar location (vs deep)	1.30	0.32 - 5.23	0.715
Acute hydrocephalus (vs absence)	**9.69**	**2.19 - 42.92**	**0.003**
Clinical signs of cerebral herniation (vs absence)	**20.43**	**5.53 - 75.53**	**<0.0001**
Mean arterial pressure ≥ 130 mmHg (vs < 130)	**3.53**	**1.16 - 10.75**	**0.027**
History of hypertension (vs. absence)	0.65	0.10 - 4.16	0.646

A multivariate binary logistic regression model was subsequently constructed, including nine variables selected on the basis of their bivariate significance ($p < 0.20$), clinical relevance, and absence of major collinearity: age, gender, Glasgow Coma Scale < 14 vs 14 - 15, hematoma location (lobar and infratentorial vs deep), acute hydrocephalus, clinical signs of cerebral herniation, mean arterial pressure ≥ 130 mmHg, and history of hypertension. The overall model was highly significant (likelihood-ratio test, $p < 0.0001$), with a McFadden pseudo-R^2 of 70.8% and excellent discrimination on the receiver operating characteristic curve (area under the curve [AUC], 0.971; **Figure 2**). Four variables remained independently associated with in-hospital mortality after adjustment (**Table 2**, **Figure 1**): Glasgow Coma Scale < 14 (adjusted OR, 35.47; 95% CI, 10.70 - 117.53; $p < 0.0001$), clinical signs of cerebral herniation (adjusted OR, 20.43; 95% CI, 5.53 - 75.53; $p < 0.0001$), acute hydrocephalus (adjusted OR, 9.69; 95% CI, 2.19 - 42.92; $p = 0.003$), and mean arterial pressure ≥ 130 mmHg (adjusted OR, 3.53; 95% CI, 1.16 - 10.75; $p = 0.027$). Age, male gender, infratentorial location, and history of hypertension all associated with mortality on bivariate analysis did not remain statistically significant after adjustment, although infratentorial location showed a strong trend (adjusted OR, 3.86; 95% CI, 0.88 - 16.99; $p = 0.074$) that may reflect limited statistical power in this subgroup (only 27 patients) rather than a true absence of effect. Notably, all 46 patients (100%) with a Glasgow Coma Scale ≤ 8 died, a complete separation of the data that precluded estimation of a finite odds ratio for this subgroup by conventional logistic regression, but which is itself a major clinical finding.

4. Discussion

This study was conducted to identify the risk factors for mortality related to hemorrhagic stroke at the CHU JRB, which includes both intensive care and neurology departments in Madagascar. This setting enabled us to perform a retrospective study and to analyze 313 specialized medical records available for investigation, collected over a 30-month period. In addition to the bivariate comparisons, a multivariate binary logistic regression model was subsequently constructed to identify independent predictors of mortality and to limit the confounding inherent to intercorrelated clinical and radiologic variables; model discrimination was excellent (AUC, 0.971).

On multivariate analysis, four variables remained independently associated with in-hospital mortality: an admission GCS below 14, clinical signs of cerebral herniation, acute hydrocephalus, and a mean arterial pressure ≥ 130 mmHg at admission.

Altered consciousness was, by a wide margin, the strongest and most robust independent predictor of mortality (adjusted OR, 35.47; 95% CI, 10.70 - 117.53). This finding is highly consistent with the literature, in which the admission GCS is repeatedly identified as one of the most powerful early prognostic markers of hemorrhagic stroke [3], reflecting the severity of the underlying brain injury and of any associated herniation. In our study, this effect was so pronounced that all 46 patients with a GCS ≤ 8 died, precluding a finite odds ratio estimate for this subgroup and underscoring the near uniform lethality of profound coma in this setting.

Clinical signs of cerebral herniation (anisocoria, bilateral Babinski sign, Cheyne-Stokes respiration) were independently associated with a markedly increased risk of death (adjusted OR, 20.43; 95% CI, 5.53 - 75.53). These signs reflect brainstem compromise from herniation, generally the terminal and often irreversible stage of intracranial hypertension. This condition results from brain herniation occurring in response to elevated intracranial pressure, causing displacement of the brain laterally and downward through normal openings within the rigid intracranial structures that divide the brain into compartments [4].

Acute hydrocephalus also remained an independent predictor (adjusted OR, 9.69; 95% CI, 2.19 - 42.92), consistent with previous reports [2] [5] [6]. Hydrocephalus complicates intracerebral hemorrhage through obstruction of cerebrospinal fluid outflow most often related to intraventricular extension or compression of the ventricular system. This situation thereby worsening intracranial hypertension and the risk of herniation.

A mean arterial pressure ≥ 130 mmHg at admission was independently associated with mortality (adjusted OR, 3.53; 95% CI, 1.16 - 10.75; p = 0.027), whereas isolated systolic or diastolic blood pressure thresholds (≥180 or ≥110 mmHg, respectively) were not significantly associated with mortality once analyzed by pressure category. A markedly elevated mean arterial pressure at admission may reflect a Cushing response to raised intracranial pressure and/or promote hematoma expansion, a recognized mechanism of early neurologic deterioration and excess mortality [1] [7] [8].

Conversely, several variables that were significantly associated with mortality on bivariate analysis like an older age, a male gender, an infratentorial hematoma location, and a history of hypertension did not remain statistically significant after multivariate adjustment. Although infratentorial location showed a strong non-significant trend (adjusted OR, 3.86; 95% CI, 0.88 - 16.99; p = 0.074), likely reflecting limited statistical power in this subgroup (n = 27) rather than a true absence of effect. This illustrates a well-recognized epidemiologic phenomenon whereby the apparent effect of such variables is, in part, "absorbed" by more directly causal predictors principally the level of consciousness and signs of herniation with which they are correlated (residual collinearity). Age was previously reported as a mortality risk factor in similar African cohorts, with an OR of 3.2 (p = 0.16) among patients older than 60 years reported by Mahoungou-Guimbi KC *et al.* [9], and this excess risk persisted at 30 days after stroke onset in patients older than 70 years (OR, 2.64; p = 0.011) [3]. Our data, based on 313 patients, corroborate a significant unadjusted age effect (mean age 58.1 ± 11.6 years among deaths vs 54.7 ± 11.0 years among survivors; p = 0.026) but suggest that once the severity of the neurological presentation is accounted forage itself does not act as an independent driver of early mortality in this population.

Several additional bivariate predictors, although not retained in the final multivariate model. Either for reasons of statistical parsimony or because of methodological redundancy with variables already in the model remain powerful and clinically relevant prognostic markers. The Intracerebral Hemorrhage (ICH) score, a validated composite prognostic tool incorporating GCS, age, hematoma volume, intraventricular extension, and infratentorial origin, was strongly associated with mortality (crude OR, 112.21; 95% CI, 46.43 - 271.17). It was deliberately not entered into the multivariate model alongside its own individual components (GCS, hematoma location) to avoid statistical redundancy and circularity, and is instead presented separately as a validated global prognostic score. NIHSS score and radiologic signs of herniation were also strongly associated with mortality (both $p < 0.0001$), consistent with the initial clinico-radiologic severity of these patients. Hyperthermia (≥38˚C) and hyperglycemia (≥2 g/L) at admission were significantly associated with excess mortality, in keeping with pathophysiological data on the aggravation of secondary brain injury by fever and stress hyperglycemia [10]-[12].

Recourse to neurosurgery and osmotherapy (mannitol) was strongly associated with mortality in crude bivariate analysis (OR, 110.67 and 14.68, respectively). However, this association most likely reflects reverse-causation bias. These interventions are undertaken in response to initial severity and clinical deterioration rather than themselves being a cause of death and for this reason these variables were not included in the final adjusted model. This important nuance should be distinguished in which the absence of neurosurgical intervention (most often related to family refusal) was described as an independent mortality risk factor. While a strong bivariate association was indeed observed, this cannot be interpreted causally in the absence of adjustment for baseline severity, and early surgical decompression is nonetheless supported by other series as improving survival and functional outcomes when performed for an appropriate indication [13]-[15].

This study has several limitations that should be considered when interpreting the results. The retrospective single-center design limits the generalizability of the findings to other settings. The definition of cases and controls based on clinical outcomes rather than prospective enrollment introduces a risk of selection bias. The number of deaths (n = 78) also limits the statistical power of the multivariate model: the number of events per variable (EPV ≈ 8 - 9) remains below the classically recommended threshold of 10, warranting cautious interpretation of the widest confidence intervals. Several severe clinical and radiologic variables (e.g., GCS ≤ 8, resuscitation/intubation) exhibited near-complete separation of the data, precluding stable estimation by conventional logistic regression for these subgroups. Although this constitutes a statistical limitation, it is itself a striking clinical finding and has been discussed as such. Precise hematoma volume (in cm^3) and quantified intraventricular extension were not available, which would have allowed more refined modelling of the ICH score and more complete adjustment. Finally, mortality follow-up was limited to the early in-hospital phase (up to day 7), precluding conclusions regarding medium- or long-term mortality (30 or 90 days), as typically reported in the reference literature.

5. Conclusion

This study enabled us to identify the risk factors associated with increased mortality during the acute phase of hemorrhagic stroke in our patients while simultaneously describing their clinico-radiologic profile. In this cohort of 313 patients managed for hemorrhagic stroke, early in-hospital mortality reached 24.9%. Multivariate analysis identified four independent risk factors for mortality: altered consciousness (GCS < 14, and a fortiori ≤ 8), the presence of clinical signs of cerebral herniation, acute hydrocephalus, and a mean arterial pressure ≥ 130 mmHg at admission, with excellent model discrimination (AUC, 0.971). These findings, consistent with the pathophysiology and international literature, highlight the importance of rigorous neurologic assessment at admission (GCS, screening for herniation signs), early brain imaging to detect hydrocephalus, and adequate blood pressure control as priority axes for prognostic stratification and management of patients with hemorrhagic stroke. Age, gender, infratentorial location, hyperthermia, hyperglycemia, NIHSS, mRS, and the ICH score remain clinically relevant markers on unadjusted analysis but were not confirmed as independent predictors once adjusted for neurological severity. A prospective multicenter cohort study conducted at a national level would be important in order to obtain data that are representative of the epidemiology of hemorrhagic stroke in Madagascar.

Conflicts of Interest

The authors declare no competing interests.

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