E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.548

Research Article

Radiological and Biochemical Predictors of Early Neurological Deterioration in Patients with Spontaneous Intracerebral Hemorrhage: A Prospective Cohort Study

S Asad ur Rehman Omer[1*], Hina Farooq[2], Tasleem Abbas[3], Muhammad Usman Khawar[4], Muhammad Ahsan Iqbal[5], Maliha Ali[6]

[1*]Consultant Neurosurgeon, Department of Neurosurgery, District Headquarters Hospital, Nankana Sahib, Punjab, Pakistan.

[2]Senior Registrar, Department of Radiology, Mayo Hospital, Lahore, Punjab, Pakistan.

[3]Senior Registrar, Department of Neurosurgery, Imran Idrees Teaching Hospital, Sialkot, Punjab, Pakistan.

[4]Postgraduate Resident, Neurosurgery Unit III, Punjab Institute of Neurosciences, Lahore General Hospital, Lahore, Punjab, Pakistan.

[5]Consultant Neurosurgeon, Department of Neurosurgery, District Headquarters Hospital, Nankana Sahib, Punjab, Pakistan.

[6]Postgraduate Resident, Department of Otorhinolaryngology, Gulab Devi Hospital, Lahore, Punjab, Pakistan.

Corresponding Author: S Asad ur Rehman Omer

Consultant Neurosurgeon, Department of Neurosurgery, District Headquarters Hospital, Nankana Sahib, Punjab, Pakistan.

Email: syedasad2011@hotmail.com

Received: 17.07.2026, Revised: 13.08.2026, Accepted: 15.09.2026

ABSTRACT

Background: Poor functional outcome and high mortality rates are associated with neurological deterioration in the early phase of spontaneous intracerebral hemorrhage (ICH). Identifying patients at high risk for rapid deterioration after admission could allow for increased surveillance, repeated reassessment, and prompt escalation of care.

Objectives: To determine the risk factors for early neurological deterioration (END) from spontaneous intracerebral hemorrhage (ICH) on radiologic and biochemical admission variables alone and to explore the discriminatory power of a model combining both radiologic and biochemical variables.

Methods: This prospective cohort study enrolled 150 adults with spontaneous ICH confirmed by computed tomography between January 2025 and January 2026 at two neurosurgical centers in Punjab, Pakistan. Clinical characteristics at presentation, non-contrast brain CT findings, and routinely obtained laboratory measurements were recorded. END was defined as an increase of ≥4 points on NIHSS and/or a decrease of≥2 points on GCS within 24 hours. Independent associations were estimated by multivariable logistic regression, and receiver operating characteristic analysis was used to compare the discriminative ability of the prediction models.

Results: 44 of 150 patients (29.3%) experienced END. Compared with neurologically stable patients, those who deteriorated had a larger hematoma burden and more frequent intraventricular extension, blend sign, midline shift, and subsequent hematoma expansion; they also showed higher NLR, CRP, glucose, and D-dimer values. Independent predictors were hematoma volume >30 mL (aOR 3.12), intraventricular hemorrhage (aOR 2.47), blend sign (aOR 2.74), NLR >7.5 (aOR 2.38), CRP >10 mg/L (aOR 2.29), and GCS ≤10 (aOR 2.56). The model integrating radiological and biochemical information achieved an AUC of 0.86, with sensitivity of 81.8% and specificity of 78.3%.

Conclusion: Early deterioration after spontaneous ICH was independently associated with routinely visible CT markers and readily available inflammatory indices. A joint radiological-biochemical approach provided stronger early risk discrimination than either domain considered alone.

Keywords: Intracerebral Bleeding, Early Neurological Decline, Hematoma Growth, Non-Contrast CT, Systemic Inflammation, Risk Prediction.

INTRODUCTION

Spontaneous intracerebral hemorrhage (ICH) represents a particularly destructive form of

stroke because neurological injury is often substantial at onset and may continue to evolve during the first hours after bleeding. Some patients are clinically stable, and others deteriorate quickly in the first 1-2 days[1,2]. This early neurological deterioration (END) has clinical significance as it often heralds poor functional recovery and death. The mechanisms responsible for such decline are not singular; progressive hematoma enlargement, ventricular extension of blood, mass effect, perihematomal edema, hydrocephalus, and secondary inflammatory injury can contribute simultaneously or sequentially[3,4].

Non-contrast computed tomography (CT) is still the mainstay of emergency imaging in the setting of spontaneous ICH (sICH). Early evaluation is often based on the site and amount of bleeding, but the characteristics of the clots might also provide clues to the persistence of instability[5]. The CT features that may accompany a less stable hemorrhage include heterogeneous attenuation, the blend sign, black hole sign, and ventricular extension. Identification of these characteristics at presentation might therefore help identify those patients at risk of early deterioration before a significant change in bedside neurological examination occurs[6].

Laboratory results with respect to intracerebral bleeding provide a helpful adjunctive perspective of disease severity. Local and systemic inflammatory activity, leucocyte recruitment, breakdown of the blood-brain barrier, oxidative injury, and further neuronal damage are the result of acute hemorrhage[7-9]. There are a number of tests that can be done in routine clinical practice that have been associated with hematoma growth, disability, or death following ICH, like the neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), serum glucose, and D-dimer. Their specificity for predicting END, especially in the context of structural CT data, continues to be somewhat unclear[10,11].

Finally, an imaging-based prediction strategy that integrates biochemical information might be more informative than using either type of information alone[12]. CT is able to show evidence of the anatomic burden and evidence of hemorrhage instability, while blood-based markers may show evidence of the inflammatory and physiologic response. Both types of information are usually obtainable soon after admission without specialized testing, making an integrated approach potentially useful for practical early triage and monitoring decisions[13].

Hence, this present prospective cohort investigation examined which radiological and biochemical variables were associated with END following spontaneous ICH and assessed whether combining these variables improved prediction compared with radiological or biochemical models used separately.

MATERIALS AND METHODS

Study Design and Setting:

A prospective cohort study was conducted from January 2025 to January 2026 in the Department of Neurosurgery at District Headquarters Hospital, Nankana Sahib, Punjab, Pakistan, and Neurosurgery Unit III at the Punjab Institute of Neurosciences, Lahore General Hospital, Lahore, Punjab, Pakistan. Consecutive adults presenting with spontaneous ICH were screened against the study criteria. A total of 150 eligible patients were enrolled and observed prospectively for the development of early neurological deterioration during the prespecified assessment period.

Eligibility Criteria

Patients aged 18 years or older were eligible when spontaneous ICH was demonstrated on non-contrast CT of the brain obtained within six hours of symptom onset or the last known well time. Enrollment also required an interpretable baseline neurological examination together with admission neuroimaging and the laboratory measurements specified in the study protocol.

Exclusion criteria were applied to hemorrhages with an identifiable secondary cause, including head trauma, ruptured aneurysm, arteriovenous malformation, intracranial tumor, hemorrhagic conversion of an ischemic infarct, or a recent intracranial surgical procedure. Patients were also excluded for active systemic infection, hematological malignancy, severe chronic inflammatory or autoimmune disease, terminal illness, or missing clinical, biochemical, or imaging information required for analysis.

Clinical Assessment:

At admission, the study team documented age, sex, hypertension, diabetes mellitus, current smoking, use of antiplatelet or anticoagulant therapy, and the interval between symptom onset and the first CT examination. Neurological severity was quantified at baseline with both the Glasgow Coma Scale (GCS) and National Institutes of Health Stroke Scale

(NIHSS). These assessments were repeated during the first 24 hours and were performed again whenever a clinically meaningful decline was suspected.

Definition of Early Neurological Deterioration:

END within 24 hours from admission was used as the primary outcome. A patient was classified as an END if the NIHSS score rose by 4 or more points from baseline, or the GCS score dropped by 2 or more points in that time period. Any change that was likely to be explained mainly by sedation, seizure, hypoglycemia, anesthesia, or another reversible non-neurological cause was not considered to be an END event.

Radiological Assessment:

All the patients had non-contrast CT of the brain at presentation. The location of hemorrhage was classified as lobar, deep, cerebellar, or brainstem. Baseline hematoma volume was estimated with the ABC/2 technique: A represented the largest hemorrhage diameter, B the maximum diameter perpendicular to A, and C the craniocaudal extent estimated from the number and thickness of slices containing blood; the resulting product was divided by two. Prespecified CT variables included hydrocephalus, intraventricular hemorrhage (IVH), midline shift ≥ 5 mm, hematoma volume, and blend sign and black hole sign. Hematoma volume was also separated into <<=30 mL and >30 mL for categorical analyses.

A "blend" sign was noted when a relatively hypoattenuating and a hyperattenuating component of the hematoma were seen side by side with a distinct interface recognizable between them. The black hole sign was described as a relatively low-attenuation region, surrounded by the hyperdense clot. Follow-up CT was obtained at approximately 24 hours, or sooner when neurological worsening prompted earlier imaging. Hematoma expansion was deemed present if an increase in volume > 33% or > 6 mL from the baseline scan was observed. The scans were reviewed individually by experienced radiologists blind to the biochemical finding and clinical outcome. All discrepancies in image interpretation were decided by consensus review.

Biochemical Assessment:

Venous blood was collected on admission, whenever feasible, before substantial therapeutic intervention. Testing included a complete blood count, serum glucose, C-reactive protein (CRP), renal function, serum electrolytes, and D-dimer. The NLR was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count. For categorical analyses, NLR >7.5 and CRP >10 mg/L were treated as elevated on the basis of clinical relevance and receiver operating characteristic analysis. Admission glucose >180 mg/dL was classified as significant hyperglycemia.

Outcome Assessment:

Participants were classified as END or non-END according to neurological change during the first 24 hours. Radiological and biochemical measurements were compared between these two outcome groups, after which the prognostic contribution of admission CT features and laboratory markers was examined individually and in combined models.

Statistical Analysis:

Data were analyzed with IBM SPSS Statistics version 26.0. Distributional characteristics of continuous variables were assessed before analysis; normally distributed measurements were summarized as mean ± standard deviation, whereas non-normally distributed values were presented as median and interquartile range. Categorical data are expressed as counts and percentages. Between-group comparisons used the independent-samples t-test or Mann-Whitney U test for continuous variables, as appropriate, and the chi-square test or Fisher exact test for categorical variables. Variables regarded as clinically important and variables with a univariable p-value ≤0.10 were considered for multivariable logistic regression to determine factors independently associated with END. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) are reported for the final model. Receiver operating characteristic (ROC) analysis was then used to assess discrimination of the radiological model, biochemical model, and combined model. Model performance was summarized by the area under the ROC curve (AUC), together with sensitivity and specificity at the selected cut-off. Statistical testing was two-sided, and $p<0.05$ was considered significant.

Ethical Considerations:

The protocol was conducted in accordance with the principles of the Declaration of Helsinki and received approval from the relevant institutional ethics review committee. Written informed

consent was obtained from each participant or, when the clinical condition prevented direct consent, from a legally authorized representative. Identifying information was protected, and patient confidentiality was maintained throughout data collection and analysis.

RESULTS

Study Population and Baseline Characteristics:

The current study included 150 patients for the analysis. The cohort had a mean age of 63.2 ± 12.7 years; 94 participants (62.7%) were men and 56 (37.3%) were women. END developed in 44 of the 150 patients (29.3%) during the first 24 hours, while 106 (70.7%) remained neurologically stable by the study definition. The END group comprised 29 men (65.9%) and 15 women (34.1%), whereas the non-END group included 65 men (61.3%) and 41 women (38.7%). Sex distribution was comparable between groups and did not show a statistically significant association with deterioration (p=0.598).

Neurological impairment was more severe at presentation among patients who subsequently deteriorated: their baseline GCS was lower, and their NIHSS was higher. In contrast, the groups were not significantly different with respect to age, hypertension, diabetes mellitus, current smoking, antiplatelet exposure, admission systolic blood pressure, or the time from symptom onset to CT acquisition. The complete baseline comparison is provided in Table 1.

Table 1. Baseline Clinical Characteristics According to Early Neurological Deterioration

Variable	No END (n=106)	END (n=44)	p-value
Age, years	61.9 ± 12.5	66.2 ± 12.8	0.058
Male, n (%)	65 (61.3)	29 (65.9)	0.598
Female, n (%)	41 (38.7)	15 (34.1)	—
Hypertension, n (%)	83 (78.3)	37 (84.1)	0.421
Diabetes mellitus, n (%)	29 (27.4)	16 (36.4)	0.275
Current smoking, n (%)	31 (29.2)	14 (31.8)	0.754
Antiplatelet use, n (%)	21 (19.8)	12 (27.3)	0.310
Admission SBP, mmHg	177 ± 25	185 ± 28	0.087
GCS score, median (IQR)	13 (11–15)	10 (8–13)	<0.001
NIHSS score, median (IQR)	11 (7–16)	17 (12–23)	<0.001
Onset-to-CT time, hours	3.2 ± 1.4	2.9 ± 1.3	0.226

Radiological and Biochemical Findings:

A substantially greater baseline hematoma burden was observed among patients who developed END. Median hematoma volume in the END group was 31.8 mL (IQR 21.4-45.6), compared with 17.2 mL (IQR 10.6-27.1) among patients without deterioration (p<0.001). A volume exceeding 30 mL was present in 25 of 44 END patients (56.8%) but in only 21 of 106 non-END patients (19.8%), demonstrating a marked difference in the distribution of larger hemorrhages.

Intraventricular extension was also more frequent in the END group, occurring in 54.5% compared with 25.5% of patients who remained stable (p=0.001). Other CT indicators of greater mass effect or hematoma instability, including midline shift ≥5 mm, blend sign, black hole sign, and subsequent hematoma expansion, were each significantly more common among patients with END.

Biochemical profiles differed in the same direction. Patients who deteriorated had higher median NLR, CRP, serum glucose, and D-dimer values than those in the non-END group. An NLR >7.5 was found in 68.2% of patients with END versus 29.2% without END. Likewise, CRP >10 mg/L was present in 70.5% of the END group compared with 38.7% of neurologically stable patients. Numerical results for all radiological and biochemical variables are summarized in Table 2.

Table 2. Radiological And Biochemical Characteristics According to Early Neurological Deterioration

Variable	No END (n=106)	END (n=44)	p-value
Hematoma volume, mL	17.2 (10.6–27.1)	31.8 (21.4–45.6)	<0.001
Hematoma volume >30 mL, n (%)	21 (19.8)	25 (56.8)	<0.001
Intraventricular hemorrhage, n (%)	27 (25.5)	24 (54.5)	0.001
Midline shift ≥5 mm, n (%)	13 (12.3)	16 (36.4)	0.001

Blunt sign, n (%)	13 (12.3)	17 (38.6)	<0.001
Black hole sign, n (%)	9 (8.5)	11 (25.0)	0.006
Hematoma expansion, n (%)	10 (9.4)	17 (38.6)	<0.001
NLR	6.1 (4.5–7.8)	9.4 (7.1–12.2)	<0.001
NLR >7.5, n (%)	31 (29.2)	30 (68.2)	<0.001
CRP, mg/L	8.2 (4.7–12.6)	15.6 (9.8–24.3)	<0.001
CRP >10 mg/L, n (%)	41 (38.7)	31 (70.5)	<0.001
Serum glucose, mg/dL	155 (128–181)	188 (151–224)	0.002
D-dimer, mg/L	0.78 (0.48–1.16)	1.22 (0.71–1.89)	0.004

Independent Predictors of Early Neurological Deterioration:

Variables with clinical relevance or evidence of association in univariable testing were entered into the multivariable logistic model. After adjustment for the other covariates, hematoma volume >30 mL showed the largest radiological effect estimate and was associated with more than a threefold increase in the odds of END (aOR 3.12; 95% CI 1.37-7.12; p=0.007).

Intraventricular hemorrhage retained an independent relationship with END (aOR 2.47; 95% CI 1.07-5.70; p=0.034), and the blend sign also remained significant after adjustment (aOR 2.74; 95% CI 1.09-6.91; p=0.032). Among the biochemical variables, NLR >7.5 was independently associated with deterioration (aOR 2.38; 95% CI 1.05-5.40; p=0.038), as was CRP >10 mg/L (aOR 2.29; 95% CI 1.04-5.02; p=0.039). A baseline GCS ≤10 was another independent predictor. Although glucose >180 mg/dL was associated with a higher estimated odds of END, the adjusted result did not reach statistical significance (aOR 1.83; 95% CI 0.81-4.15; p=0.147). The full multivariable estimates are displayed in Table 3.

Table 3. Multivariable Logistic Regression Analysis of Predictors of Early Neurological Deterioration

Predictor	Adjusted OR	95% CI	p-value
GCS ≤10	2.56	1.12–5.83	0.026
Hematoma volume >30 mL	3.12	1.37–7.12	0.007
Intraventricular hemorrhage	2.47	1.07–5.70	0.034
Blend sign	2.74	1.09–6.91	0.032
NLR >7.5	2.38	1.05–5.40	0.038
CRP >10 mg/L	2.29	1.04–5.02	0.039
Glucose >180 mg/dL	1.83	0.81–4.15	0.147

Predictive Performance:

The radiological prediction model, comprising hematoma volume, intraventricular hemorrhage, midline shift, and blend sign, produced an AUC of 0.80 (95% CI 0.73-0.87). The biochemical model based on NLR, CRP, serum glucose, and D-dimer showed lower discrimination, with an AUC of 0.75 (95% CI 0.67-0.83).

When the radiological and biochemical predictors were combined, discrimination increased to an AUC of 0.86 (95% CI 0.80-0.92). At the selected operating point, the integrated model identified END with 81.8% sensitivity and 78.3% specificity. Thus, using structural CT information together with biochemical measurements yielded the strongest predictive performance among the three approaches evaluated, as illustrated in Figure 1.

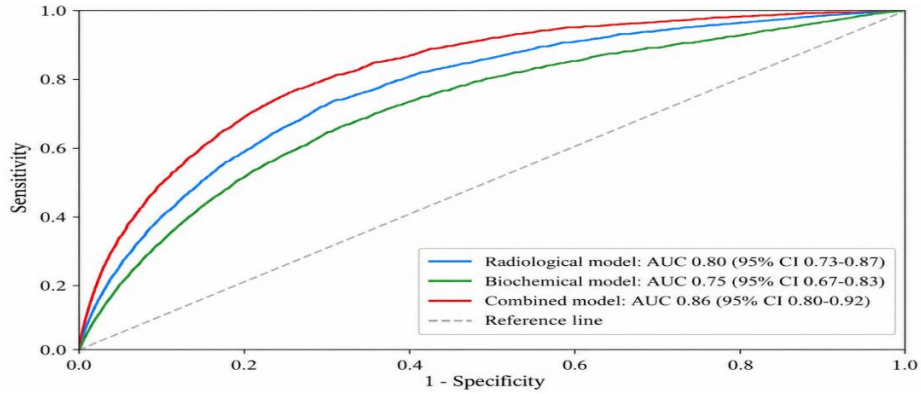

Figure 1. Receiver Operating Characteristic Curves for Early Neurological Deterioration.

The integrated radiological-biochemical model demonstrated the greatest discrimination (AUC 0.86; 95% CI 0.80-0.92), compared with the radiological model (AUC 0.80) and the biochemical model (AUC 0.75).

Overall, END was present in close to one-third of all participants and was associated with more severe initial neurological deficits, higher hematoma burden, intraventricular extension, evidence of hematoma instability on CT, and increased systemic inflammatory activity. The main independent variables in the adjusted analysis were hematoma volume >30 mL, intraventricular hemorrhage, blend sign, NLR >7.5, CRP >10 mg/L, and GCS ≤10, which means both the anatomic and the systemic parameters were useful for predicting early clinical deterioration.

DISCUSSION

In this prospective cohort, END occurred in 29.3% of patients with spontaneous ICH and was characterized by a combination of greater structural brain injury and a stronger inflammatory response at admission[1]. Patients who deteriorated had higher inflammatory markers and a greater likelihood of intraventricular blood, CT findings indicative of hematoma instability, and worse baseline neurologic status. Hematoma volume >30 mL, intraventricular hemorrhage, blend sign, NLR >7.5, and CRP >10 mg/L remained independent factors associated with END after adjustment. The results here serve as an argument in favour of understanding early worsening following ICH based on imaging severity and systemic biologic response, instead of a single measure[2,3].

Hematoma volume was the most significant radiographic variable in the multivariate analysis. Patients with a volume > 30 mL were about three times as likely as the rest to deteriorate early, after adjusting for other factors[4]. This is clinically plausible because a larger volume of blood takes up more room in the intracranial compartment, causes more local compression, further mass effect, more perihematomal edema, and decreases the physiologic reserve for more swelling. A bigger hemorrhage is also more likely to give the bleeding time to spread or be quantifiable, both of which can result in a rapid deterioration of neurologic function in the first few hours of admission[5,6].

Ventricular extension of hemorrhage offered extra information for prognosis, apart from hematoma size. Blood entering the ventricular system can disrupt the circulation of CSF and can help to cause acute hydrocephalus, increased ICP, and additional inflammatory exposure of periventricular structures[7]. However, the much greater prevalence of IVH in patients with END in this cohort implies that involvement of ventricles is not merely a radiological term but a useful indicator of early physiological instability, which should increase the level of clinical monitoring[8].

After controlling for other factors, the blend sign was also a significant CT feature that was significant. A hematoma with contiguous areas of markedly different attenuation can be due to non-uniform clot formation or to blood of varying ages, which is consistent with ongoing or recurrent hemorrhage[9,10]. This could account for the ability of the blend sign to identify patients at higher risk for later neurological deterioration. Additionally, the black hole sign was more frequently identified in patients with END in unadjusted comparisons, but was not included in the final group of independent predictors, which may reflect the overlap in information gained from its presence with hematoma volume and/or heterogeneity and/or other markers of hemorrhage instability[11,12].

Biochemical results indicate inflammatory activation occurs in conjunction with the structural determinants of END. Patients who deteriorated had significantly higher levels of

NLR as well as CRP at admission. An elevated NLR may indicate a relative neutrophilic predominance and suppression of the lymphocytes in response to acute physiological stressors[13]. Inflammatory cell recruitment, oxidative injury, disturbance of the blood-brain barrier, and perihematomal edema can all exacerbate secondary brain injury in the context of ICH, over and above the damage caused by the primary bleeding. The independent association between NLR >7.5 and END thus indicates that a simple blood-count derived index may provide additional information beyond CT severity alone[14,15].

No different independent association with early worsening was found in those with CRP >10 mg/L. CRP is a routinely measured acute-phase reactant and may be used as an easily measured marker of the intensity of the systemic inflammatory response to cerebral tissue injury. When modeled in the adjusted model, its persistence, along with NLR, suggests that inflammatory activity provides information in addition to anatomical hemorrhage features. Combined, NLR and CRP could provide a cost-effective biochemical adjunct to the admission CT and may be useful in patients in whom apparently static structural findings are matched by active secondary injury response[16].

The between-group analysis also showed that patients with END were more likely to have elevated glucose and D-dimer on admission, reflecting a general stress and coagulation response to more severe disease. However, a glucose level >180 mg/dL was not statistically significant after adjustment for the other variables, and D-dimer did not appear in the final independent predictor in the reported regression model. The elevations might therefore be in part due to the physiological load from the initial hemorrhage, and not due to a separate pathway to neurological deterioration[17].

One of the most important findings was that discrimination was enhanced when both radiologic and biochemical data were used. The radiologic and biochemical model has an AUC of 0.80 and 0.75, respectively, while the combined model has an AUC of 0.86 and has a sensitivity of 81.8% and specificity of 78.3%[18]. This pattern leads to a multimodal approach for early risk assessment. Blood markers indicate a systemic inflammatory and physiological response, which cannot be directly observed on imaging, whereas imaging attributes detail the location, size, mass effect, and apparent instability of the hemorrhage. The two domains thus give similar but not identical information concerning the patient's changing status[19].

Clinically, a combination of large hematoma, intraventricular extension, a mixture sign, and raised inflammatory markers should be considered as a subgroup that deserves special care during the first day after ICH. In these patients, more frequent neurological evaluations, early consultation with neurosurgical or neurocritical care services if the clinical course is concerning, and careful blood-pressure control may be useful[20]. While the proposed model is not a substitute for bedside judgment, it provides a structured approach to utilize routinely available information at a time when deterioration may be thought of, but not necessarily recognized, as it has already happened[5].

These results have several limitations that should be considered. The moderate size of the cohort and the absence of external validation of the prediction model reduce confidence in its applicability outside the hospital or with other patient cohorts. Advanced imaging parameters like the CT angiography spot sign and radiomic features were not assessed[7-10]. Despite excluding patients with overt infection and major inflammatory disorders, unrecognized or subclinical systemic disorders might affect the results of NLR and CRP. Furthermore, the outcome window was mainly defined by the first 24 hours, and neurological deterioration that occurred later in the hospitalization was not recorded. The cut-offs and multivariable model identified should thus be tested in larger multicenter cohorts and then independently validated before inclusion in a formal bedside prediction tool[15-20].

CONCLUSION

Nearly one-third of this spontaneous ICH cohort experienced early neurological deterioration. Larger hematoma volume (>30 mL), intraventricular hemorrhage, blend sign, NLR >7.5, CRP >10 mg/L, and GCS ≤10 independently identified patients at greater risk of worsening during the first 24 hours. The radiological-biochemical model was more effective at discriminating END than imaging or lab models alone. The availability of the required CT features and inflammatory measurements in many acute-care facilities may be a feasible way to provide early risk stratification, neurologic monitoring, timely reimaging, and timely escalation of care when deterioration is suspected.

Funding: No external funding was received.

Conflict of Interest: The authors declare no conflicts of interest.

Data Availability: Data are available from the corresponding author on reasonable request, subject to institutional and ethical requirements.

Authors' Contributions: S.A.R.O. conceived and supervised the study. H.F. contributed to radiological assessment and interpretation. T.A. and M.U.K. contributed to patient recruitment and clinical data collection. M.A.I. contributed to data analysis and interpretation. M.A. contributed to literature review and manuscript revision. All authors reviewed and approved the final manuscript.

Acknowledgments: The authors acknowledge the clinical, radiological, laboratory, and nursing teams involved in patient care and data collection.

REFERENCES

1. Zhou H, Dai D, Xie K, Li A. Risk factors and model construction for early neurological deterioration in patients with intracerebral hemorrhage. Front Neurol. 2025;16:1663347. doi:10.3389/fneur.2025.1663347.
2. Hu Z, Zhu W, Fan C, Jiang Y. Neutrophil-to-lymphocyte ratio and systemic inflammation response index as biomarkers for the clinical outcomes of intracerebral hemorrhagic stroke patients: a prospective cohort study. Front Neurol. 2025;16:1616128. doi:10.3389/fneur.2025.1616128.
3. Guo L, Cao X, Chang L, Liu H. Neutrophil-to-lymphocyte ratio as a predictor of prognosis in patients with spontaneous intracerebral hemorrhage: a systematic review and meta-analysis. Front Neurol. 2025;16:1553263. doi:10.3389/fneur.2025.1553263.
4. Du Y, Liu L, Kang K, Lin Y, Gu H, Bian L, et al. Association of C-reactive protein with short-term outcomes in spontaneous intracerebral hemorrhage patients with or without infection: from a large-scale nationwide longitudinal registry. Clin Interv Aging. 2025;20:83-91. doi:10.2147/CIA.S489083.
5. Deng R, Wu C, Zhang L, Wang J, Guo J, Yang Z, et al. Early NCCT imaging signs for prognostication in intracerebral hemorrhage: a retrospective cohort study with long follow-up results. BMC Neurol. 2025;25:91. doi:10.1186/s12883-025-04100-z.
6. Cindea C, Saceleanu V, Canning P, Roman-Filip C, Mihaila R. Non-contrast CT hemorrhage markers and outcomes in intracerebral hemorrhage: a large single-center cohort from Romania. Reports. 2025;8(3):159. doi:10.3390/reports8030159.
7. Pensato U, Dowlatshahi D, Rodriguez-Luna D, Ospel JM, Morotti A, Tanaka K, et al. Spot sign in intracerebral hemorrhage: critical reappraisal and future clinical implications. Stroke. 2025;56(6):1612-1624. doi:10.1161/STROKEAHA.125.050637.
8. Xu Y, Zhu H, Su Y, Chen Z, Wang C, Yang M, et al. Serum neuritin as a predictive biomarker of early neurological deterioration and poor prognosis after spontaneous intracerebral hemorrhage: a prospective cohort study. Front Neurol. 2025;15:1490023. doi:10.3389/fneur.2024.1490023.
9. Sasongko AB, Perdana Wahjoepramono PO, Halim D, Aviani JK, Adam A, Tsai YT, et al. Potential blood biomarkers that can be used as prognosticators of spontaneous intracerebral hemorrhage: a systematic review and meta-analysis. PLoS One. 2025;20(2):e0315333. doi:10.1371/journal.pone.0315333.
10. Hu Z, Chen S, Zhang E, Wei L, Wang J, Shang Q, et al. Novel inflammatory markers in intracerebral hemorrhage: results from Olink proteomics analysis. FASEB J. 2025;39(2):e70341. doi:10.1096/fj.202402183RR.
11. Wu R, Hong T, Li Y. Systematic evaluation of hematoma expansion models in spontaneous intracerebral hemorrhage: a meta-analysis and meta-regression approach. Cerebrovasc Dis. 2025;54(3):333-343. doi:10.1159/000540223.
12. Guo P, Zou W. Neutrophil-to-lymphocyte ratio, white blood cell, and C-reactive protein predicts poor outcome and increased mortality in intracerebral hemorrhage patients: a meta-analysis. Front Neurol. 2024;14:1288377. doi:10.3389/fneur.2023.1288377.
13. Song L, Qiu X, Zhang C, Zhou H, Guo W, Ye Y, et al. Combining non-contrast CT signs with onset-to-imaging time to predict the evolution of intracerebral hemorrhage. Korean J Radiol. 2024;25(2):166-178. doi:10.3348/kjr.2023.0591.
14. Kölbl K, Hock SW, Xu M, Sembill JA, Mrochen A, Balk S, et al. Association of

non-contrast CT markers with long-term functional outcome in deep intracerebral hemorrhage. Front Neurol. 2024;14:1268839. doi:10.3389/fneur.2023.1268839.

15. Geng Z, Yang C, Zhao Z, Yan Y, Guo T, Liu C, et al. Development and validation of a machine learning-based predictive model for assessing the 90-day prognostic outcome of patients with spontaneous intracerebral hemorrhage. J Transl Med. 2024;22:236. doi:10.1186/s12967-024-04896-3.
16. Liang S, Tian X, Gao F, Man M, Wang Q, Li J, et al. Prognostic significance of the stress hyperglycemia ratio and admission blood glucose in diabetic and nondiabetic patients with spontaneous intracerebral hemorrhage. Diabetol Metab Syndr. 2024;16:58. doi:10.1186/s13098-024-01293-0.
17. Zhang J, Zhang Q, Gu H, Zhou Q, Li Z, Zhao X. Comparison of stress hyperglycemia ratio and glycemic gap on acute intracerebral hemorrhage in-hospital outcomes. Ann Clin Transl Neurol. 2024;11(6):1492-1501. doi:10.1002/acn3.52063.
18. Haupenthal D, Schwab S, Kuramatsu JB. Hematoma expansion in intracerebral hemorrhage—the right target? Neurol Res Pract. 2023;5:36. doi:10.1186/s42466-023-00256-6.
19. Gong Y, Wang Y, Chen D, Teng Y, Xu F, Yang P. Predictive value of hyperglycemia on prognosis in spontaneous intracerebral hemorrhage patients. Heliyon. 2023;9(3):e14290. doi:10.1016/j.heliyon.2023.e14290.
20. Greenberg SM, Ziai WC, Cordonnier C, Dowlatshahi D, Francis B, Goldstein JN, et al. 2022 guideline for the management of patients with spontaneous intracerebral hemorrhage: a guideline from the American Heart Association/American Stroke Association. Stroke. 2022;53(7):e282-e361. doi:10.1161/STR.0000000000000407.