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Original Research Article

Clinical Profile and Outcomes in Patients with Acute Intracerebral Hemorrhage Presenting to Emergency Department: A Prospective Observational Study

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Abstract

Background: Intracerebral hemorrhage (ICH) is a devastating subtype of stroke with a 30-day mortality close to 50%. Incidence is disproportionately high in South and East Asian populations. Despite advances in stroke care, no intervention of proven benefit exists for ICH. This study was undertaken to delineate the clinical profile, imaging characteristics, and short-term outcomes of ICH patients at a tertiary emergency department in South India.

Methods: A prospective observational study was conducted at Kurnool Medical College / Government General Hospital, Kurnool from May 2025 to January 2026. One hundred patients (>18 years) with CT-confirmed ICH were enrolled. GCS score, ICH score, hematoma volume (ABC/2 method), intraventricular extension (IVE), midline shift, and mean arterial pressure (MAP) were documented. Primary endpoint was in-hospital outcome (death or discharge alive)

Results: Mean age was 58.37 ± 12.26 years; males predominated (M:F = 1.85:1). Hypertension was the leading risk factor (68%). Putaminal hemorrhage was most common (54%). Limb weakness occurred in 83% of patients. GCS ≤ 6 was associated with 100% mortality ($p < 0.001$). Hematoma volume > 60 ml, ICH score ≥ 4, IVE (mortality 64.2% vs 13.8%), midline shift > 5 mm (mortality 66.6% vs 15.7%), and MAP > 130 mmHg (mortality 55.5% vs 21.9%; $p = 0.004$) were significantly associated with death. Infratentorial hemorrhage carried the highest mortality (cerebellar 100%, brainstem 77.7%). Surgical vs medical management did not differ significantly ($p = 0.182$). Overall in-hospital mortality was 28%.

Conclusion: ICH predominantly affected hypertensive middle-aged males. Low GCS, high ICH score, large hematoma, IVE, midline shift > 5 mm, and elevated MAP at admission were key mortality predictors. Aggressive blood pressure control and early risk stratification using validated tools are essential in the emergency setting.

Keywords: Intracerebral Hemorrhage; Glasgow Coma Scale; ICH Score; Intraventricular Extension; Midline Shift; Hypertension; Mortality.

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Introduction

Stroke is defined by the World Health Organization as rapidly developed clinical signs of focal cerebral dysfunction lasting more than 24 hours or leading to death, with no apparent cause other than vascular origin. [1] Intracerebral hemorrhage (ICH) accounts for 10–15% of all strokes in Western populations and up to 30% in Asian series, with an overall 30-day case fatality of 40–50%. [2,3] Despite improvements in ischemic stroke management, no intervention of proven benefit exists for ICH. [4] Hypertension is the dominant modifiable risk factor, accounting for up to 74% of population-attributable risk in multicenter studies. [5] Prognostic tools such as the ICH score and Glasgow Coma Scale (GCS) have been validated internationally, yet data from South Indian

emergency settings remain limited. [6] This study was designed to describe the clinical and radiological profile of ICH patients and to identify predictors of in-hospital mortality at a tertiary care emergency department in Andhra Pradesh.

Aims and Objectives

To prospectively study the clinical presentation, CT imaging characteristics, and in-hospital outcomes of patients with acute ICH, and to identify independent predictors of mortality including demographic variables, hematoma characteristics (site, volume, intraventricular extension, midline shift), GCS score, ICH score, and mean arterial pressure at admission.

Materials and Methods

This prospective observational study was conducted at the Department of Emergency Medicine, Kurnool Medical College / Government General Hospital, Kurnool from May 2025 to January 2026. Consecutive adult patients (>18 years) diagnosed with ICH on non-contrast CT brain were enrolled. Exclusions were age <18 years and refusal of consent.

A structured proforma recorded demographic data, presenting complaints, comorbidities, GCS score, and MAP. CT scans were performed on a 128-slice MDCT (Siemens SOMATOM Definition AS+) with 6 mm reconstructed sections. Hematoma volume was calculated by the ABC/2 method. [7] ICH scores were computed using the validated five-parameter scale encompassing GCS, hematoma volume, IVE, hemorrhage location, and age. [8] Patients received medical or surgical management per institutional protocol and were followed until discharge or in-hospital death. Patients leaving against medical advice were classified as worst outcome (death). Statistical analysis used IBM SPSS v22.0; chi-square tests compared categorical outcomes; $p < 0.05$ was significant.

Results

Of 487 patients screened for cerebrovascular accident, 116 had ICH on CT; 100 met inclusion criteria and were analysed. The mean age was 58.37 ± 12.26 years; 92% were above 40 years, and 79% fell in the 41–70 year bracket. [9] Males predominated (65%). Hypertension was the most frequent risk factor (68%), followed by smoking (58%) and alcoholism (46%).

Putaminal hemorrhage was most common (54%), followed by thalamic (21%), lobar (11%), brainstem (9%), and cerebellar (5%). Limb weakness was the presenting feature in 83%; loss of consciousness in 27%. Comprehensive results are summarised in Table 1 and ICH score–mortality correlation in Table 2. A GCS score ≤ 6 at presentation carried 100% in-hospital mortality (chi-square 49.148, $p = 0.000$; Figure 1).

Hematoma volume > 40 ml was associated with mortality of 86.9%, with 100% mortality when volume exceeded 60 ml ($p = 0.000$). ICH score correlated strongly with outcome (Figure 2; chi-square 63.743, $p = 0.000$). IVE was present in 28%; mortality was 64.2% vs 13.8% without IVE ($p = 0.000$).

Midline shift > 5 mm occurred in 24%; mortality was 66.6% with shift vs 15.7% without ($p = 0.000$). Admission MAP > 130 mmHg was associated with 55.5% mortality vs 21.9% for MAP ≤ 130 mmHg ($p = 0.004$). Surgical vs medical mortality (42.8% vs 25.5%) was not significantly different ($p = 0.182$). Overall in-hospital mortality was 28%.

Table 1: Summary of Clinical, Radiological Parameters and Outcomes (n = 100)

Parameter	Category / Value	n (%) / Statistic	Outcome & P value
Age	Mean ± SD	58.37 ± 12.26 yrs	Not a significant predictor
Sex	Male : Female	65 : 35 (1.85:1)	Mortality M 37.1%, F 23.1%; NS
Hypertension	Present	68 (68%)	Major risk factor; increased mortality
Smoking / Alcohol	Present	58% / 46%	Secondary risk factors
Site: Putamen	Supratentorial	54 (54%)	Mortality 12.9%
Site: Thalamic / Lobar	Supratentorial	21% / 11%	Mortality 33.3% / 22.2%
Site: Cerebellar / Brainstem	Infratentorial	5% / 9%	Mortality 100% / 77.7%
GCS ≤ 6	On admission	13 (13%)	Mortality 100%; p = 0.000
GCS 6–10	On admission	27 (27%)	Mortality 24.5%; p = 0.000
GCS > 10	On admission	60 (60%)	Mortality 15%
Hematoma vol > 40 ml	Large bleed	15 (15%)	Mortality 86.9%; 100% if > 60 ml; p = 0.000
IVE	Present	28 (28%)	Mortality 64.2% vs 13.8%; p = 0.000
Midline shift > 5 mm	Present	24 (24%)	Mortality 66.6% vs 15.7%; p = 0.000
MAP > 130 mmHg	On admission	18 (18%)	Mortality 55.5% vs 21.9%; p = 0.004
Management	Medical / Surgical	86 / 14	Mortality 25.5% / 42.8%; p = 0.182 (NS)
Overall mortality	All patients	28 (28%)	72 discharged alive

IVE = intraventricular extension; GCS = Glasgow Coma Scale; MAP = mean arterial pressure; NS = not significant.

Table 2: ICH Score Correlation with In-Hospital Mortality

ICH Score	Total (n)	Deaths (n)	Survived (n)	Mortality (%)
0	39	0	39	0%
1	23	2	21	8.6%
2	16	6	10	37.5%
3	15	13	2	86.6%
4	5	5	0	100%
5	2	2	0	100%
Total	100	28	72	28%

Chi-square = 63.743; p = 0.000

Figure 1: GCS Score at Admission vs Outcome (Chi-square = 49.148; p < 0.001)

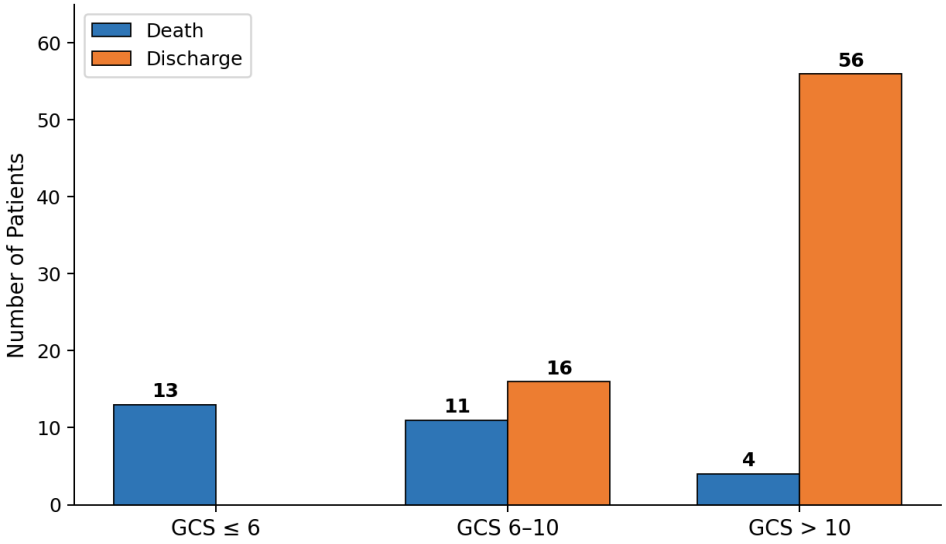

Figure 1: GCS Score at Admission vs In-Hospital Outcome (GCS = Glasgow Coma Scale; Chi-square = 49.148; p < 0.001)

Figure 2: ICH Score vs In-Hospital Mortality (%) (Chi-square = 63.743; p < 0.001)

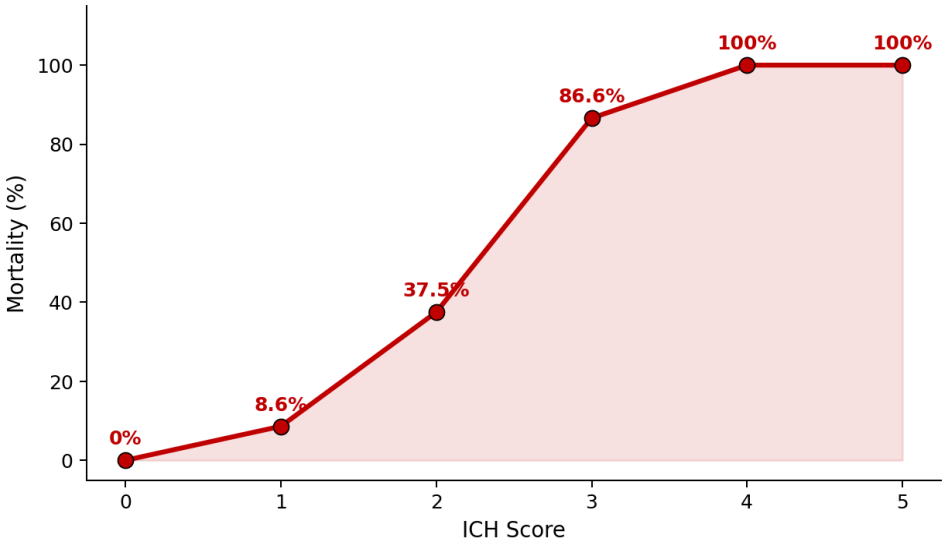

Figure 2: ICH Score vs In-Hospital Mortality (%) (ICH = intracerebral hemorrhage; Chi-square = 63.743; p < 0.001)

Discussion

The mean age of 58.37 years in our cohort aligns with South Asian reports — Mohammad Wasay et al reported 55 years and Sai Sampath et al reported 56.8 years — and reflects the well-established age-dependent rise in ICH incidence. [10,11] The pathophysiological basis for this age association is

the progressive accumulation of hypertension-driven vascular injury. Sustained hypertension causes medial hypertrophy, fibrinoid necrosis, and lipohyalinosis of small penetrating arteries — particularly the lenticulostriate, thalamoperforating, and basilar paramedian arteries — progressively weakening the vessel wall over decades. [14] Charcot-Bouchard microaneurysms develop at the bifurcations of these arteries and represent the final structural substrate for spontaneous rupture. The preponderance of males (65%) in our series is consistent with Watson et al (M:F 1.43:1) and may reflect the higher prevalence of hypertension, smoking, and alcohol use in males, all of which independently accelerate arterial wall degeneration and are compounded by androgenic effects on platelet aggregation. [12] Hypertension was the dominant risk factor (68%), concordant with the INTERSTROKE study, which attributed 74% of population-attributable risk for ICH to hypertension — the highest of any single modifiable factor. [5] Putaminal hemorrhage was the most common site (54%), consistent with Sampath et al (62.6%) and Namani et al (46%). [11,13] This predilection is explained by the anatomy of the lenticulostriate arteries — thin-walled perforators arising at right angles from the middle cerebral artery, exposed to the full force of systemic blood pressure without the protection of a gradual pressure gradient. [14] Hypertension-induced lipohyalinosis disproportionately affects these vessels, making the putamen, thalamus, and pons the classical sites of hypertensive ICH. The thalamus (21%) and brainstem (9%) share the same vulnerability through thalamoperforating and basilar paramedian arteries respectively. Infratentorial hemorrhages carried the highest mortality — cerebellar 100% and brainstem 77.7% — a direct consequence of the critically compact anatomy of the posterior fossa. [14] Brainstem hemorrhage disrupts the reticular activating system, respiratory centres, and cardiovascular regulatory nuclei within millimetres of the bleed, producing rapid herniation and cardiorespiratory failure even with small hematoma volumes. Cerebellar hemorrhage rapidly compresses the fourth ventricle, causing acute obstructive hydrocephalus and upward herniation through the tentorium, both of which are rapidly fatal. In contrast, lobar hemorrhage (22.2% mortality) occurs predominantly in cortical and subcortical white matter where mass effect can be partially accommodated by sulcal spaces before critical herniation thresholds are reached. [13] GCS $\leq$ 6 at admission was associated with 100% mortality (chi-square 49.148, $p < 0.001$), replicating findings of Togha M et al — who documented 100% mortality at GCS 3–4 — and Namani et al. [13,15] The mechanistic explanation for this powerful association lies in the relationship between level of consciousness and extent of primary and secondary brain injury. A severely depressed GCS reflects either direct destruction of the ascending reticular activating system (ARAS) by blood and mass effect, or bilateral hemisphere dysfunction from raised intracranial pressure (ICP) and midline shift compressing the diencephalon.6 Blood itself is directly neurotoxic: thrombin released during clot formation activates PAR-1 receptors on neurons and astrocytes, triggering apoptosis, mitochondrial dysfunction, and excitotoxic glutamate release within hours of hemorrhage onset. Iron from haemoglobin degradation generates reactive oxygen species via the Fenton reaction, initiating lipid peroxidation of neuronal membranes in the perihematomal zone within 24–72 hours. [16] Concurrently, clot retraction releases osmotically active proteins that drive perihematomal oedema, further compressing viable brain tissue and worsening the ICP–cerebral perfusion pressure (CPP) gradient. A GCS $\leq$ 6 therefore signals a degree of neurological devastation that exceeds the brain's capacity for auto-regulation and functional recovery, explaining its 100% mortality threshold in our series.

Hematoma volume > 40 ml was associated with 86.9% mortality, rising to 100% when volume exceeded 60 ml ($p < 0.001$). This volume–mortality relationship has a clear mechanistic basis rooted in the Monroe-Kellie doctrine: the skull is a rigid container with fixed total volume; any space-occupying hematoma must displace CSF and venous blood, and once these compensatory mechanisms are exhausted, ICP rises exponentially. [17] Beyond the mechanical mass effect, hematoma volume determines the extent of the perihematomal inflammatory cascade. Clot-derived thrombin and complement activation products (C3a, C5a) recruit peripheral neutrophils and monocytes across a disrupted blood-brain barrier within 4–6 hours; these release matrix metalloproteinases (MMPs), particularly MMP-9, which degrade the basal lamina of cerebral microvessels, compounding oedema formation and extending the injury zone. [18] The ICH score — integrating GCS, volume, IVE, infratentorial location, and age $\geq$ 80 — demonstrated near-linear mortality escalation from 0% (score 0) to 100% (scores 4–5; chi-square 63.743, $p < 0.001$), consistent with Hemphill et al's original validation and Cheung et al's external replication. [6,16] Each additional ICH score unit compounds independent pathophysiological insults: larger volume worsens mass effect; IVE adds CSF pathway obstruction and iron-mediated ependymal injury; infratentorial location adds posterior fossa herniation risk; and older age reduces neuronal plasticity and cerebrovascular reserve, as well as increasing the likelihood of pre-existing cerebral amyloid angiopathy. Intraventricular extension (IVE) was present in 28% of patients and was associated with 64.2% mortality vs 13.8% without

IVE ($p < 0.001$), nearly quintupling mortality — corroborated by Bhatia et al (OR 2.66, $p = 0.007$) and Daverat et al (OR 5.3). [17,18] The pathophysiological mechanisms by which IVE worsens outcome are multiple and additive. First, blood in the ventricular system obstructs CSF flow through the foramen of Monro and aqueduct of Sylvius, causing acute non-communicating hydrocephalus with rapid ICP surge. Second, haemoglobin breakdown products — particularly bilirubin oxidation products and haem iron — directly injure the ependymal lining and periventricular white matter, impairing CSF absorption at the arachnoid granulations and producing a secondary communicating hydrocephalus. [19] Third, blood in the cisterns and ventricular system activates complement and neutrophil-mediated inflammation far beyond the primary hematoma, amplifying cerebral oedema globally. Midline shift > 5 mm carried 66.6% mortality vs 15.7% ($p < 0.001$), consistent with Bhatia et al's 40% threshold. [17] Midline shift is the radiological surrogate of subfalcine herniation, wherein the cingulate gyrus displaces beneath the falx cerebri, compressing the contralateral anterior cerebral artery and causing parasagittal infarction. Progressive shift above 7–8 mm leads to transtentorial herniation — the uncus of the temporal lobe compresses the ipsilateral oculomotor nerve (producing the classic blown pupil), the posterior cerebral artery (causing occipital infarction), and ultimately the brainstem (Duret haemorrhages), all of which are mechanistically fatal. [20] Admission MAP > 130 mmHg was associated with 55.5% mortality vs 21.9% for MAP ≤ 130 mmHg ($p = 0.004$), supporting Dhandapani et al's MAP > 145 mmHg threshold and Fogelholm et al's first-day blood pressure data. [19,20]

The mechanistic link between elevated MAP and ICH mortality operates through three converging pathways. First, acute hypertension in the setting of an active hematoma raises intraluminal pressure in the perilesional microvasculature, promoting hematoma expansion — a phenomenon occurring in up to 38% of ICH patients within the first 24 hours of onset, each 1 ml of expansion associated with measurable neurological deterioration. [21] Second, elevated MAP raises cerebral perfusion pressure, which — in the context of absent autoregulation in the perihematomal zone — directly increases microvascular hydrostatic pressure and drives oedema formation through the damaged blood-brain barrier. Third, sustained hypertension activates the sympatho-adrenal axis, releasing catecholamines that produce neurogenic cardiac dysfunction (Takotsubo-like cardiomyopathy and ECG changes), arrhythmias, and aspiration risk from impaired airway reflexes — all contributing to systemic organ failure in ICH.22 The INTERACT and ATACH-2 trials evaluated intensive BP lowering (target SBP < 140 mmHg within 1 hour) and found modest attenuation of hematoma growth without significant improvement in functional outcomes at 90 days, suggesting that while acute hypertension drives expansion, other factors — including perihematomal inflammation and clot-mediated neurotoxicity — independently determine mortality beyond the acute BP phase. [21]

Surgical intervention did not significantly reduce mortality compared to medical management (42.8% vs 25.5%, $p = 0.182$), mirroring the landmark STICH and STICH-II randomised controlled trials. [22,23] The pathophysiological explanation for the failure of surgery to reduce mortality lies in the dual nature of ICH injury. While surgical evacuation removes the physical mass and reduces mechanical compression, it cannot reverse the biochemical cascade already triggered: thrombin-driven neuronal apoptosis, iron-mediated oxidative injury, and complement-activated perihematomal inflammation persist and progress regardless of hematoma removal. [24]

Furthermore, the surgical corridor through functional brain tissue introduces additional injury, and the inability to achieve haemostasis in friable, coagulopathy-affected tissue carries risk of re-bleeding. The MISTIE-III trial of minimally invasive surgery with tPA-assisted clot lysis similarly showed no 90-day functional outcome benefit, reinforcing that the secondary injury cascade is the principal determinant of outcome. [23] The overall mortality of 28% in our series compares favourably with Chiranjib Nag et al (29.3%), Bhatia et al (32.7%), and the classic Broderick series (44%), potentially reflecting the benefit of a structured emergency triage pathway, rapid CT acquisition, and evidence-based ICU management at our tertiary centre. [24,17,25]

The progressive reduction in ICH mortality over decades reflects incremental improvements in airway management, ICP monitoring, fever control, glycaemic management, and avoidance of routine steroid use — all of which attenuate secondary injury cascades — rather than any single therapeutic breakthrough. [6] These findings collectively underscore that ICH management must be fundamentally neuroprotective — targeting the molecular cascades of thrombin toxicity, iron-mediated oxidative injury, neuroinflammation, and blood-brain barrier disruption — alongside rigorous blood pressure control and early risk stratification using validated scoring tools.

Conclusion

ICH in this South Indian cohort predominantly affected hypertensive males aged 41–70 years, with putaminal hemorrhage as the commonest site. Low GCS score, high ICH score, hematoma volume > 40

ml, intraventricular extension, midline shift > 5 mm, and admission MAP > 130 mmHg were robust predictors of in-hospital mortality. Infratentorial hemorrhage — particularly cerebellar and brainstem — carried the highest mortality through posterior fossa herniation and compression of vital centres. Surgical intervention conferred no additional survival benefit over medical management, consistent with the STICH trial evidence. The overall in-hospital mortality of 28% underscores the urgent need for early risk stratification using validated tools and aggressive blood pressure management targeting hematoma expansion prevention in the emergency setting.

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