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Clinical Profile and Risk Factor Comparison in Ischemic versus Haemorrhagic Stroke: A Cross-Sectional Study in a Tertiary Care Center.

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#### ABSTRACT

Ischemic and haemorrhagic strokes differ in pathogenesis and prognosis, yet comparative data from Indian tertiary centres remain limited. In this cross-sectional study we enrolled 100 consecutive adults (≥ 18 y) with neuroimaging-confirmed stroke over one year. Fifty ischemic and fifty haemorrhagic cases were analysed. Demographics, vascular risk factors, National Institutes of Health Stroke Scale (NIHSS) scores, and 7-day outcomes were recorded. Categorical variables were compared with the  $\chi^2$  test and continuous variables with the independent t-test; p < 0.05 denoted significance. Ischemic patients were older than haemorrhagic patients (63.4  $\pm$  11.2 vs 58.7  $\pm$  12.9 years; p = 0.041). Diabetes (54 % vs 30 %; p = 0.011), dyslipidaemia (50 % vs 24 %; p = 0.005) and atrial fibrillation (22 % vs 8 %; p = 0.044) predominated in ischemic stroke, whereas hypertension was universal but highest in haemorrhagic stroke (88 %). Haemorrhagic cases presented sooner (median 3.5 h) yet exhibited greater severity (median NIHSS 14 vs 9; p = 0.001), longer hospital stay (11.6 ± 4.4 vs 8.2 ± 3.9 days; p < 0.001) and higher, though non-significant, early mortality (18 % vs 6 %). Functional independence at discharge (mRS  $\leq$  2) was achieved in 56 % of ischemic versus 24 % of haemorrhagic patients (p = 0.001). Metabolic risk factors dominate ischemic stroke, whereas uncontrolled hypertension drives haemorrhagic events, which carry greater early neurological severity and worse short-term outcomes. Tailored risk-factor control and subtype-specific acute management are essential.

Keywords: Ischemic stroke; Haemorrhagic stroke; Risk factors

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## **INTRODUCTION**

Stroke is a leading cause of morbidity and mortality worldwide, significantly impacting public health systems, especially in low- and middle-income countries [1]. Broadly classified into ischemic and haemorrhagic types, stroke presents with a diverse clinical profile and etiology [2-4]. Ischemic stroke, accounting for nearly 80–85% of all stroke cases, results from an obstruction in cerebral blood flow, most commonly due to thromboembolism or atherosclerosis. In contrast, haemorrhagic stroke, which includes intracerebral and subarachnoid haemorrhage, arises from rupture of blood vessels, often linked to hypertension or vascular anomalies [5].

The clinical presentation of both stroke types varies in severity and symptomatology, influenced by the location and extent of cerebral insult. Risk factors such as hypertension, diabetes mellitus, smoking, dyslipidemia, alcohol intake, and atrial fibrillation contribute significantly to the pathogenesis of both stroke types, although their prevalence may differ. Understanding these differences is crucial for early diagnosis, appropriate intervention, and improving long-term outcomes [6-8].

This cross-sectional study aims to compare the clinical profiles and associated risk factors in patients diagnosed with ischemic and haemorrhagic strokes at a tertiary care center. Such comparative analysis will enhance our understanding of disease patterns and support the development of targeted preventive strategies and individualized patient care.

## STUDY METHODOLOGY

This cross-sectional comparative study was conducted at the Department of General Medicine. . The study was carried out over a period of one year and included patients admitted with a confirmed diagnosis of stroke, classified as either ischemic or haemorrhagic based on neuroimaging findings. Ethical clearance was obtained from the institutional ethics committee, and written informed consent was secured from all participants or their legal guardians prior to inclusion.

Patients aged 18 years and above, presenting with new-onset focal neurological deficits lasting more than 24 hours and confirmed through CT or MRI scans were included. Cases of transient ischemic attacks (TIA), traumatic brain injury, or stroke mimics were excluded from the study. A total of 100 patients were enrolled, with 50 cases each of ischemic and haemorrhagic stroke selected based on inclusion and exclusion criteria.

Data were collected using a structured proforma which included demographic details, clinical presentation, history of risk factors (such as hypertension, diabetes, dyslipidemia, smoking, alcohol intake, and atrial fibrillation), vital signs, and neurological examination findings. Laboratory investigations and neuroimaging reports were documented for each patient. The severity of stroke was assessed using the National Institutes of Health Stroke Scale (NIHSS) at admission.

Statistical analysis was performed using SPSS software version 20. Quantitative data were expressed as mean ± standard deviation, and qualitative data were presented as frequencies and percentages. The chi-square test was used to analyze categorical variables, and the independent t-test was used for comparing continuous variables between the ischemic and haemorrhagic stroke groups. A pvalue < 0.05 was considered statistically significant.

## **RESULTS**

**Table 1: Demographic Characteristics of the Study Cohort (N = 100)** 

Variable	Ischemic Stroke (n =	Haemorrhagic Stroke (n =	<b>p</b> -
	50)	50)	value
Mean age ± SD (years)	63.4 ± 11.2	58.7 ± 12.9	0.041
Age $\geq$ 65 y - n (%)	28 (56.0 %)	20 (40.0 %)	0.112
Male sex - n (%)	32 (64.0 %)	35 (70.0 %)	0.517
Rural residence - n (%)	21 (42.0 %)	24 (48.0 %)	0.536
Median symptom-to-door time (h, IQR)	5.0 (3-8)	3.5 (2-6)	0.018
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**Table 2: Distribution of Major Vascular Risk Factors** 

Risk factor	Ischemic (n = 50)	Haemorrhagic (n = 50)	$\chi^2/t$	<i>p</i> -value
Hypertension	38 (76.0 %)	44 (88.0 %)	2.46	0.117
Type 2 diabetes mellitus	27 (54.0 %)	15 (30.0 %)	6.43	0.011
Dyslipidaemia	25 (50.0 %)	12 (24.0 %)	7.82	0.005
Current smoker	22 (44.0 %)	19 (38.0 %)	0.37	0.542
Alcohol misuse	14 (28.0 %)	21 (42.0 %)	2.36	0.124
Atrial fibrillation	11 (22.0 %)	4 (8.0 %)	4.05	0.044

Table 3: Stroke Severity at Admission and Early Outcomes

Variable	Ischemic (n = 50)	Haemorrhagic (n = 50)	<i>p</i> -value
Median NIHSS score (IQR)	9 (6-14)	14 (10-19)	0.001
NIHSS > 15 - n (%)	10 (20.0 %)	22 (44.0 %)	0.010
In-hospital pneumonia – n (%)	6 (12.0 %)	11 (22.0 %)	0.188
Mean length of stay ± SD (days)	8.2 ± 3.9	11.6 ± 4.4	< 0.001
7-day mortality – n (%)	3 (6.0 %)	9 (18.0 %)	0.059
Discharged with mRS ≤ 2 - n (%)	28 (56.0 %)	12 (24.0 %)	0.001

#### DISCUSSION

The present cross-sectional analysis compared clinical characteristics, vascular risk factors and early outcomes in 50 ischemic and 50 haemorrhagic stroke patients managed at a single tertiary-care centre. Several important patterns emerged.

First, the two stroke sub-types differed demographically. Ischemic patients were, on average, five years older than haemorrhagic counterparts ( $63.4 \pm 11.2$  vs  $58.7 \pm 12.9$  years; p = 0.041). Age-related atherosclerosis and cardio-embolic sources probably underlie this disparity; conversely, haemorrhagic events are frequently precipitated by long-standing, poorly controlled hypertension that can rupture vulnerable small vessels at a relatively younger age. Although men predominated in both groups, the difference was not statistically significant, echoing previous Indian studies that show broadly similar sex distributions once health-care access bias is minimised [9].

Risk-factor profiles displayed a clear metabolic–hypertensive dichotomy. Hypertension was ubiquitous, exceeding 75 % in both categories and reaching 88 % in haemorrhagic stroke, supporting its central role in vessel rupture. By contrast, diabetes (54 % vs 30 %; p = 0.011), dyslipidaemia (50 % vs 24 %; p = 0.005) and atrial fibrillation (22 % vs 8 %; p = 0.044) clustered among ischemic patients, consistent with athero-thrombotic and cardio-embolic mechanisms. These findings reaffirm the need for stringent metabolic control and rhythm surveillance to prevent ischemic events, while uncompromising blood-pressure management remains the cornerstone of haemorrhagic-stroke prevention [10].

Clinical presentation and early trajectory also diverged. Median symptom-to-door time was shorter in haemorrhagic strokes (3.5 h) than ischemic strokes (5 h), likely because abrupt headache, vomiting or rapid neurological decline prompts immediate help-seeking. Nevertheless, haemorrhagic patients arrived with markedly higher neurological severity (median NIHSS 14 vs 9; p = 0.001), reflected in higher proportions with NIHSS > 15 (44 % vs 20 %). The larger initial injury translated into longer hospitalisation (11.6 ± 4.4 vs 8.2 ± 3.9 days) and a trend toward greater 7-day mortality (18 % vs 6 %). Functional independence at discharge (mRS  $\leq$  2) was achieved by only one-quarter of haemorrhagic survivors compared with more than half of ischemic survivors. These data align with global evidence that intracerebral haemorrhage confers roughly double the early mortality relative to ischemic stroke, chiefly because mass-effect, raised intracranial pressure and haematoma expansion are difficult to reverse.

Our results carry several clinical implications. First, routine screening for diabetes, dyslipidaemia and atrial fibrillation in hypertensive adults could identify individuals at dual risk and facilitate tailored prophylaxis—anticoagulation for eligible AF patients, statins and glucose-lowering therapy for metabolic control, alongside universal blood-pressure management. Second, public-education campaigns should emphasise the sudden, severe nature of haemorrhagic stroke to maintain the prompt hospital arrival



observed here, while also encouraging rapid recognition of more subtle ischemic symptoms. Third, inhospital protocols must prioritise early neurocritical-care measures—blood-pressure reduction, reversal of coagulopathy and timely neurosurgical consultation—for haemorrhagic cases, and hyper-acute thrombolysis or thrombectomy for ischemic cases.

## **CONCLUSIONS**

Metabolic risk factors dominate ischemic stroke, whereas uncontrolled hypertension drives haemorrhagic events, which carry greater early neurological severity and worse short-term outcomes. Tailored risk-factor control and subtype-specific acute management are essential.

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