RESEARCH PAPER

COMPARATIVE STUDY OF SERUM FERRITIN AND HIGH SENSITIVITY CRP (HSCRP) IN PREDICTING OUTCOMES OF ACUTE ISCHAEMIC STROKE

Dr. Shivansh Khanijo[1], Dr. Shailendra Mane[2], Dr. Sushama K Jotkar[3], Dr. Rajesh J. Khyalappa[4]

[1]Junior Resident, Department of General Medicine, D.Y. Patil Medical College, Kolhapur, Maharashtra, India. D.Y. Patil Education Society (Deemed to be University) Kolhapur

[2]Professor, Department of General Medicine, D.Y. Patil Medical College, Kolhapur, Maharashtra, India. D.Y. Patil Education Society (Deemed to be University) Kolhapur

[3]Professor and HOD, Department of General Medicine, D.Y. Patil Medical College, Kolhapur, Maharashtra. D.Y. Patil Education Society (Deemed to be University), Kolhapur

[4]Dean and Professor, Department of General Medicine, D.Y. Patil Medical College, Kolhapur, Maharashtra, India. D.Y. Patil Education Society (Deemed to be University) Kolhapur

Received: 28th Feb, 2026; Revised: 6th March 2026; Accepted: 7th April, 2026; Available Online: 20th April, 2026

ABSTRACT

Background: Stroke is a major cause of mortality and long-term disability worldwide, with ischaemic stroke accounting for approximately 80–85% of all stroke cases. Inflammation and oxidative stress play important roles in the pathophysiology of acute ischaemic stroke. High-sensitivity C-reactive protein (hs-CRP) and serum ferritin have been investigated as prognostic biomarkers; however, comparative studies evaluating their relative prognostic significance are limited.

Methodology: An observational, comparative, cross-sectional study was conducted in the Department of General Medicine, Dr. D. Y. Patil Medical College Hospital and Research Institute, Kadamwadi, Kolhapur, after Institutional Ethics Committee approval. The study included 56 patients with acute ischaemic stroke admitted to the Medicine Intensive Care Unit and Neurology Unit. Clinical evaluation, CT/MRI, serum ferritin, and hs-CRP measurements were performed at admission. Functional outcome was assessed using the Modified Rankin Scale (MRS). Statistical analysis was performed to assess the association of serum ferritin and hs-CRP with stroke outcomes.

Results: The mean age of patients was 55.52 ± 13.63 years with male predominance. A high prevalence of obesity, hypertension, diabetes mellitus, smoking, tobacco use, and alcohol consumption was observed. The overall mean serum ferritin level was 410.87 ± 159.05 ng/mL and showed no significant association with mortality or MRS score ($r = -0.004$, $p = 0.977$). In contrast, the overall mean hs-CRP level was 15.14 ± 9.08 mg/L and demonstrated a significant positive correlation with MRS score ($r = 0.487$, $p < 0.001$). Patients with poor outcomes had markedly elevated hs-CRP levels.

Conclusion: hs-CRP is a significant and reliable prognostic biomarker in acute ischaemic stroke and demonstrated superior predictive utility compared to serum ferritin. Elevated hs-CRP levels were significantly associated with increased mortality, greater neurological disability, and poorer functional outcomes, whereas serum ferritin demonstrated limited prognostic value in the present study population.

Keywords: Acute Ischaemic Stroke; High-Sensitivity C-Reactive Protein (hs-CRP); Serum Ferritin; Prognostic Biomarkers.

How to cite this article: Khanijo S, Mane S, Jotkar SK, Khyalappa RJ. Comparative Study of Serum Ferritin and High Sensitivity CRP (hs-CRP) in Predicting Outcomes of Acute Ischaemic Stroke. Int J Drug Deliv Technol. 2026;16(60s):729-737. DOI: 10.25258/ijddt.16.60s.83

Source of support: Nil.

Conflict of interest: None

INTRODUCTION

Stroke is a major cause of mortality and long-term disability worldwide and represents a significant public health challenge. According to the Global Burden of Disease (GBD) 2019 study, stroke remains the second leading cause of death globally and a leading cause of disability-adjusted life years (DALYs) lost across all age groups.[1] Ischaemic stroke accounts for approximately 80–85% of all stroke cases, making it the predominant subtype encountered in clinical practice.[2]

In India, the burden of stroke has increased substantially over the past two decades, driven by population ageing, urbanisation, and an increasing prevalence of vascular risk factors such as hypertension, diabetes mellitus, dyslipidaemia, smoking, and obesity. Community-based studies and national estimates suggest that the incidence of stroke in India ranges between 105 and 152 per 100,000 population per year, with ischaemic stroke constituting the majority of cases.[3] Stroke-related morbidity contributes significantly to functional dependence, loss of productivity, and healthcare expenditure, underscoring the need for improved prognostic assessment and outcome

***Author for Correspondence: Dr. Shailendra Mane ***

prediction.

Acute ischaemic stroke is a clinically heterogeneous entity, with outcomes influenced by multiple factors including stroke subtype, infarct location and volume, collateral circulation, time to reperfusion, and baseline patient characteristics. Traditional prognostic indicators such as age, baseline neurological deficit, and neuroimaging findings provide valuable information but may not fully capture the biological processes driving secondary brain injury. Consequently, there has been growing interest in identifying biochemical markers that reflect underlying pathophysiological mechanisms, particularly inflammation and oxidative stress, which may contribute to neurological deterioration and poor recovery following acute ischaemic stroke.[5]

The pathophysiology of acute ischaemic stroke involves a complex cascade of biochemical and cellular events initiated by cerebral arterial occlusion. Interruption of cerebral blood flow results in energy failure, membrane depolarisation, excitotoxicity, intracellular calcium overload, and mitochondrial dysfunction, ultimately leading to neuronal injury and cell death.[6] Inflammation plays a pivotal role in the evolution of ischaemic brain injury. Following cerebral ischaemia, activation of resident microglia and astrocytes occurs within minutes to hours, followed by recruitment of circulating leukocytes into the ischaemic brain tissue. These immune responses are mediated by the release of pro-inflammatory cytokines, chemokines, and adhesion molecules, which contribute to endothelial dysfunction, blood–brain barrier disruption, and secondary neuronal damage.[7]

C-reactive protein (CRP) is an acute-phase protein synthesised by hepatocytes in response to inflammatory cytokines, particularly interleukin-6. High-sensitivity CRP (hs-CRP) assays enable detection of low-grade systemic inflammation and have been widely utilised in cardiovascular risk prediction.10 Elevated hs-CRP levels have been shown to predict the risk of first-ever ischaemic stroke and are associated with greater neurological deficit, larger infarct size, poorer functional outcomes, increased mortality, and unfavourable long-term outcomes.[11–13] However, hs-CRP is a nonspecific marker of inflammation and may be influenced by co-existing infections, chronic inflammatory conditions, and systemic comorbidities.[14,15]

Ferritin is the principal intracellular iron storage protein and serves as a marker of total body iron stores. In addition to its role in iron metabolism, ferritin is a positive acute-phase reactant, and serum levels increase in response to systemic inflammation.16 Iron-mediated oxidative stress contributes to lipid peroxidation, mitochondrial dysfunction, neuronal apoptosis, and secondary brain injury following cerebral ischaemia.[17,18] Elevated serum ferritin levels may therefore serve as a surrogate marker of both systemic inflammation and increased iron availability, linking peripheral iron metabolism with central oxidative injury. Clinical studies have demonstrated that elevated serum ferritin levels are associated with increased stroke severity, larger infarct volumes, poorer functional outcomes, and increased mortality.[19,20]

Although both hs-CRP and serum ferritin have been independently associated with stroke severity and outcomes, direct comparative studies evaluating their relative prognostic value in acute ischaemic stroke are limited, particularly in Indian tertiary-care settings. Given that hs-CRP primarily reflects systemic inflammation, while serum ferritin reflects both inflammation and oxidative stress, comparative evaluation of these biomarkers may provide valuable insights into their relative utility in predicting outcomes of acute ischaemic stroke.

Early identification of patients at high risk of poor outcomes following acute ischaemic stroke is essential for optimising management strategies and resource allocation. hs-CRP and serum ferritin are inexpensive, widely available laboratory parameters that can be measured routinely at admission. The present study aims to compare serum ferritin and high-sensitivity C-reactive protein (hs-CRP) as predictors of outcomes in patients with acute ischaemic stroke.

METHODOLOGY

An observational, comparative, cross-sectional study was conducted at Department of General Medicine, Dr. D. Y. Patil Medical College, Kolhapur. The study started after getting approval from the Institutional Ethics Committee. The sample size was 56. Patients admitted consecutively to the Medicine Intensive Care Unit and Neurology Unit with a diagnosis of acute ischemic stroke during the study period were included in the study. Assessment included complete medical history and physical examination. Investigations included complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), CT/MRI, serum ferritin, high-sensitivity C-reactive protein (hsCRP), and assessment of the association of hsCRP and serum ferritin in predicting acute ischemic stroke outcome.

Inclusion Criteria

Patients with clinical features suggestive of stroke, defined by the World Health Organization as a vascular lesion of the brain resulting in rapidly developing clinical signs of focal or global loss of brain function lasting at least 24 hours and documented by brain computerized tomography (CT) or magnetic resonance imaging (MRI), Patients of all genders, Patients aged above 18 years, Patients admitted in ICU/wards/casualty of the Department of Medicine, Dr. D. Y. Patil Medical College Hospital and Research Institute, Kadamwadi, Kolhapur.

Exclusion Criteria

Patients with a history of infection within the previous 2 weeks, History of transient ischemic attack, signs of acquired in-hospital infection, Surgery or trauma within the previous month, Patients with malignancy, systemic inflammatory diseases, hemochromatosis,

immunosuppressive diseases, or major cardiac, renal, hepatic, or endocrinological disorders, Patients receiving immunosuppressive therapy, Patients with low functional level for immediate rehabilitation care.

ASSESSMENTS

A complete medical history, including risk factors for cerebrovascular disease, was obtained for each patient. All patients underwent physical and neurological examinations, complete blood count, white blood cell count with differential count, erythrocyte sedimentation rate, renal and hepatic function tests, urinalysis, 12-lead electrocardiogram, chest X-ray, immunological study, and CT/MRI.

Venous blood samples for hs-CRP and ferritin measurements were obtained from the patients at admission. Functional prognosis, stroke severity, vascular risk factors, mortality, ferritin levels, and hsCRP levels were analyzed.

BLOOD SAMPLING AND LABORATORY METHODS:

The serum was separated within one hour by centrifugation at 3,000 rpm for 10 minutes. The separated sera were stored at -25°C until laboratory testing was performed. Laboratory data were obtained blinded to the patients' clinical details.

Serum hs-CRP levels were measured by an ultrasensitive latex-enhanced immunoassay method using an Abbott Architect C8000 Analyzer (Abbott Laboratories, Abbott Park, IL) according to the manufacturer's specifications. The detection limit was 0.01 mg/dL and the measuring range was 0.01–16 mg/dL. Based on serum hs-CRP levels measured within 48 hours after stroke onset, subjects were divided into two groups: elevated hs-CRP group (serum hs-CRP ≥0.5 mg/dL) and normal hs-CRP group (serum hs-CRP <0.5 mg/dL).

Serum ferritin was measured by immunoturbidimetric assay using a Hitachi Modular PP Analyzer (Roche Diagnostics GmbH, Mannheim, Germany) according to the manufacturer's specifications. The reference range for ferritin in the laboratory was 15–300 ng/mL.

PROCEDURE TO ENSURE ETHICAL CONSIDERATIONS IN RESEARCH WITH HUMAN SUBJECTS:

After obtaining informed consent, detailed history, general physical examination, and clinical examination were carried out.

No financial burden was imposed on the patients.

Required ethical clearance from the University Ethics Committee was obtained.

Privacy and confidentiality of the patients were maintained.

COLLABORATIVE WORK WITH OTHER DEPARTMENTS OR INSTITUTIONS:

Collaborative work was conducted with the Department of Biochemistry, Dr. D. Y. Patil Medical College Hospital and Research Institute, Kolhapur.

RESULTS

Demographic and Clinical Profile of Study Patients

Table 1: Demographic and Anthropometric Characteristics of Study Patients

Variable	Total (n=56)	Death (n=10)	Disabled (n=46)
Age (years), Mean ± SD			
Mean ± SD	55.52 ± 13.63	56.80 ± 16.10	55.24 ± 13.16
Range	29 – 78	35 – 78	29 – 78
Gender, n (%)			
Male	35 (62.5%)	6 (60.0%)	29 (63.0%)
Female	21 (37.5%)	4 (40.0%)	17 (37.0%)
BMI (kg/m²), Mean ± SD			
Mean ± SD	32.90 ± 7.13	31.42 ± 8.20	33.22 ± 6.89
Underweight (<18.5)	1 (1.8%)	0 (0.0%)	1 (2.2%)
Normal (18.5–25)	10 (17.9%)	2 (20.0%)	8 (17.4%)
Overweight (25–30)	5 (8.9%)	1 (10.0%)	4 (8.7%)
Obese (>30)	40 (71.4%)	7 (70.0%)	33 (71.7%)

Note: SD = Standard Deviation; NS = Not Significant; n = Number of patients.

Comorbidities and Lifestyle Habits

Table 2: Distribution of Comorbidities and Lifestyle Habits

Characteristic	Total (n=56)	Death (n=10)	Disabled (n=46)
Comorbidities, n (%)			
Hypertension Only	17 (30.4%)	3 (30.0%)	14 (30.4%)
Diabetes Mellitus Type 2 Only	17 (30.4%)	4 (40.0%)	13 (28.3%)
Hypertension + DM Type 2	22 (39.3%)	3 (30.0%)	19 (41.3%)
Lifestyle Habits, n (%)			
None	18 (32.1%)	2 (20.0%)	16 (34.8%)
Smoking + Alcohol	18 (32.1%)	4 (40.0%)	14 (30.4%)
Tobacco	12 (21.4%)	3 (30.0%)	9 (19.6%)
Tobacco + Smoking + Alcohol	8 (14.3%)	1 (10.0%)	7 (15.2%)

Note: HTN = Hypertension; DM = Diabetes Mellitus; n = Number of patients; % = Percentage.

Infarct Territory and MRS Score Distribution

Table 3: Distribution of Infarct Territory by Outcome

Infarct Territory	Total n (%)	Death n (%)	Disabled n (%)
Left ACA	11 (19.6%)	0 (0.0%)	11 (23.9%)
Right ACA	8 (14.3%)	0 (0.0%)	8 (17.4%)
Left MCA	5 (8.9%)	2 (20.0%)	3 (6.5%)
Right MCA	8 (14.3%)	2 (20.0%)	6 (13.0%)
Left PCA	6 (10.7%)	0 (0.0%)	6 (13.0%)
Right PCA	7 (12.5%)	0 (0.0%)	7 (15.2%)
Brainstem	6 (10.7%)	4 (40.0%)	2 (4.3%)
Lacunar Infarct	5 (8.9%)	2 (20.0%)	3 (6.5%)
Total	56 (100%)	10 (100%)	46 (100%)

Note: ACA = Anterior Cerebral Artery; MCA = Middle Cerebral Artery; PCA = Posterior Cerebral Artery. Percentages within outcome groups.

Table 4: Distribution of Modified Rankin Scale (MRS) Score

MRS Score	Interpretation	n (%)
0	No symptoms	0 (0.0%)
1	No significant disability	2 (3.6%)
2	Slight disability	5 (8.9%)
3	Moderate disability	17 (30.4%)
4	Moderately severe disability	20 (35.7%)
5	Severe disability	2 (3.6%)

6	Death	10 (17.9%)

5. Serum Ferritin Levels in Acute Ischaemic Stroke

Table 5: Descriptive Statistics of Serum Ferritin Levels by Outcome

Statistical Parameter	Total (n=56)	Death (n=10)	Disabled (n=46)
Mean (ng/mL)	410.87	397.36	413.35
Standard Deviation	159.05	137.50	163.21
Median (ng/mL)	375.19	372.66	380.00
Minimum (ng/mL)	205.00	205.00	220.00
Maximum (ng/mL)	835.00	560.00	835.00
25th Percentile	292.50	266.88	302.50
75th Percentile	490.00	487.50	490.00
t-statistic	−0.286		
p-value	0.775 (NS)		

Note: NS = Not Significant. Independent samples t-test was used for comparison between Death and Disabled groups. Ferritin measured in ng/mL.

hs-CRP Levels in Acute Ischaemic Stroke

Table 6: Descriptive Statistics of hs-CRP Levels by Outcome

Statistical Parameter	Total (n=56)	Death (n=10)	Disabled (n=46)
Mean (mg/L)	15.14	27.36	12.48
Standard Deviation	9.08	4.71	7.48
Median (mg/L)	13.31	27.97	11.98
Minimum (mg/L)	3.05	17.34	3.05
Maximum (mg/L)	47.30	34.65	47.30
25th Percentile	8.00	24.12	7.72
75th Percentile	18.15	30.97	16.85
t-statistic	5.447		
p-value	< 0.001***		

*Note: *** $p < 0.001$ (Highly Significant). Independent samples t-test. hs-CRP measured in mg/L.*

7. Comparison of Serum Ferritin and hs-CRP in Predicting Outcome

Table 7: Pearson Correlation Matrix – Serum Ferritin, hs-CRP, and MRS Score

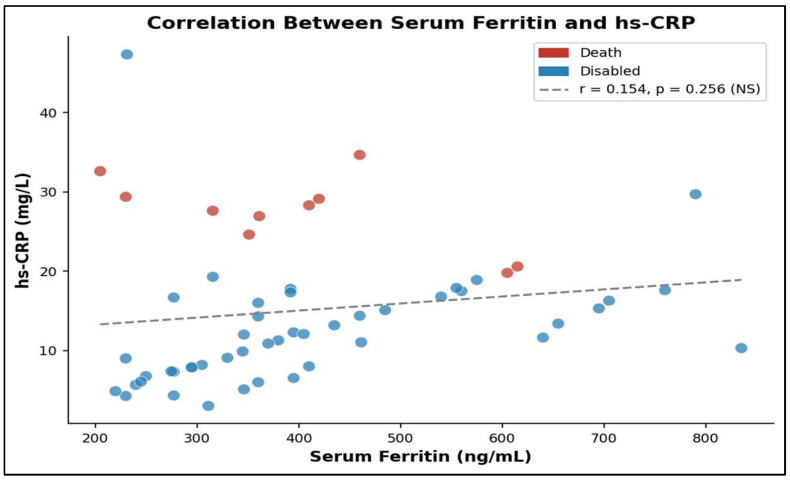
Variable	Serum Ferritin	hs-CRP	MRS Score
Serum Ferritin	1.000	r = 0.154 p = 0.256 (NS)	r = −0.004 p = 0.977 (NS)
hs-CRP	r = 0.154 p = 0.256 (NS)	1.000	r = 0.487 p < 0.001***
MRS Score	r = −0.004 p = 0.977 (NS)	r = 0.487 p < 0.001***	1.000

*Note: Pearson correlation coefficient (r) and p-values are reported. *** $p < 0.001$; NS = Not Significant.*

Table 8: Comparative Analysis of Serum Ferritin vs. hs-CRP as Predictors of Stroke Outcome

Parameter	Serum Ferritin (ng/mL)	hs-CRP (mg/L)	MRS Score
By Outcome			
Death (n=10)	397.36 ± 137.50	27.36 ± 4.71	6.00 ± 0.00
Disabled (n=46)	413.35 ± 163.21	12.48 ± 7.48	3.33 ± 0.90
p-value (t-test)	0.775 (NS)	< 0.001***	< 0.001***
Correlation with MRS Score			
Pearson r	−0.004	0.487	–
p-value	0.977	< 0.001	–
Predictive value	Poor	Strong	–

*Note: Values expressed as Mean ± SD. *** $p < 0.001$; NS = Not Significant ($p > 0.05$). Independent samples t-test used for outcome comparison.*



Correlation Between Serum Ferritin and hs-CRP Levels

DISCUSSION

The present study included 56 patients with acute ischaemic stroke, with a mean age of 55.52 ± 13.63 years. Patients in the death group had a slightly higher mean age (56.80 ± 16.10 years) compared to the disabled group (55.24 ± 13.16 years). These findings are comparable with studies by **Ranjan et al. (2023)**[117] and Mallikarjuna **Reddy et al. (2020)**[118], who reported mean ages of 58.87 ± 11.41 years and 58.58 ± 10.11 years, respectively. **Phad et al. (2025)**[119] reported that 77.5% of patients were older than 46 years, while **Thanikachalam et al.** (2020)[112] demonstrated predominance in the 51–70 years age group. Male predominance was observed in the present study, with males constituting 62.5% of the population. Similar male predominance has been reported by **Ranjan et al.** (2023)[117], **Phad et al.** (2025)[119], and **Mallikarjuna Reddy et al.** (2020)[118]. The predominance of males may be explained by the higher prevalence of smoking, alcohol consumption, hypertension, diabetes mellitus, and dyslipidaemia among males, while estrogen-mediated vascular protection may contribute to lower stroke incidence in females.

The mean BMI in the present study was 32.90 ± 7.13 kg/m², indicating a predominantly obese study population, with obesity present in 71.4% of patients. Obesity contributes to insulin resistance, chronic inflammation, endothelial dysfunction, dyslipidaemia, and accelerated atherosclerosis, thereby increasing susceptibility to acute ischaemic stroke. Overall, the demographic profile was comparable with previous Indian studies demonstrating middle-aged to elderly predominance, male predominance, and a high burden of vascular risk factors.

The present study demonstrated a high burden of vascular comorbidities. Hypertension combined with type 2 diabetes mellitus was the most common comorbidity (39.3%), followed by isolated hypertension and isolated diabetes mellitus (30.4% each). Similar findings were reported by **Ranjan et al. (2023)**[117], **Phad et al. (2025)**[119], and **Mallikarjuna Reddy et al.** (2020)[118]. Hypertension contributes to endothelial injury, accelerated atherosclerosis, vascular remodelling, and impaired

cerebral autoregulation, while diabetes mellitus aggravates vascular injury through hyperglycaemia, oxidative stress, platelet activation, and inflammation. Regarding lifestyle habits, 32.1% of patients had combined smoking and alcohol consumption, while only 32.1% had no addiction history. Similar observations were reported in previous Indian studies. Smoking and tobacco exposure promote endothelial dysfunction, platelet aggregation, oxidative stress, vasoconstriction, and accelerated atherosclerosis, whereas chronic alcohol intake contributes to hypertension, arrhythmias, endothelial injury, and metabolic disturbances.

Among infarct territories, left ACA infarcts were most common (19.6%), followed by right ACA and right MCA infarcts (14.3% each). Brainstem infarction demonstrated disproportionately high mortality, accounting for 40.0% of deaths despite representing only 10.7% of infarcts. MCA infarcts and lacunar infarcts also contributed significantly to mortality. Brainstem infarcts are associated with poorer outcomes because of involvement of respiratory centres, cardiovascular regulatory centres, cranial nerve nuclei, and reticular activating systems. MCA infarcts are commonly associated with larger infarct volumes, cerebral edema, aphasia, hemiplegia, and impaired consciousness, all of which worsen outcomes.

The Modified Rankin Scale (MRS) distribution demonstrated that moderately severe disability (MRS 4) was the most common outcome (35.7%), followed by moderate disability (MRS 3) in 30.4% of patients. Mortality (MRS 6) accounted for 17.9% of the study population. Similar findings have been reported by **Ranjan et al.** (2023)[117], **Phad et al.** (2025)[119], and **Mallikarjuna Reddy et al.** (2020)[118]. Functional disability following stroke is influenced by infarct size, anatomical location, neuronal injury, cerebral edema, and inflammatory responses. The predominance of MRS scores 3 and 4 suggests that most patients survived with significant functional dependence rather than complete neurological recovery.

The overall mean serum ferritin level was 410.87 ± 159.05 ng/mL. Patients in the death group demonstrated mean ferritin levels of 397.36 ± 137.50 ng/mL, while disabled patients had mean levels of 413.35 ± 163.21 ng/mL. The difference was statistically not significant ($p = 0.775$), and ferritin showed no correlation with MRS score ($r = -0.004$, $p = 0.977$). These findings differ from studies by **Thanikachalam et al**. (2020)[112], **Ranjan et al**. (2023)[117], **Phad et al.** (2025)[119], and **Mallikarjuna Reddy et al**. (2020)[118], which demonstrated significant associations between elevated ferritin levels and severe stroke or poor outcomes. Ferritin is an acute phase reactant and intracellular iron storage protein that may contribute to oxidative neuronal injury through free radical generation and lipid peroxidation. However, ferritin levels are also influenced by obesity-related inflammation, chronic metabolic disorders, and systemic inflammatory conditions, which may reduce its specificity as a prognostic marker.

In contrast, hs-CRP demonstrated significant prognostic utility. The overall mean hs-CRP level was 15.14 ± 9.08 mg/L. Patients in the death group had markedly elevated hs-CRP levels (27.36 ± 4.71 mg/L) compared to disabled survivors (12.48 ± 7.48 mg/L), with statistically highly significant difference ($p < 0.001$). hs-CRP additionally demonstrated significant positive correlation with MRS score ($r = 0.487$, $p < 0.001$). Similar findings were reported by **Sanober et al. (2023)**[107], **Rather SA et al. (2024)**[121], and **Mallikarjuna Reddy et al. (2020)**[118]. hs-CRP reflects cytokine-mediated endothelial dysfunction, leukocyte activation, oxidative stress, thrombus propagation, and blood-brain barrier disruption associated with cerebral ischaemia. Elevated hs-CRP therefore reflects ongoing inflammatory injury and infarct progression more accurately than ferritin.

The present study therefore demonstrated that hs-CRP was superior to serum ferritin in predicting mortality and functional disability in acute ischaemic stroke. While serum ferritin showed no significant association with MRS score or mortality, hs-CRP demonstrated strong positive correlation with disability and poor clinical outcome. These findings suggest that hs-CRP may serve as a more reliable biomarker for prognostic assessment and risk stratification in acute ischaemic stroke, whereas serum ferritin demonstrated limited predictive utility in the present cohort.

CONCLUSION

Based on the findings of the present study, it can be concluded that high-sensitivity C-reactive protein (hs-CRP) is a significant and reliable prognostic biomarker in patients with acute ischaemic stroke, whereas serum ferritin demonstrated limited prognostic utility. Elevated hs-CRP levels were significantly associated with increased mortality, greater neurological disability, and poorer functional outcomes as assessed by the Modified Rankin Scale (MRS). The study demonstrated a statistically significant positive correlation between hs-CRP and MRS score ($r = 0.487$, $p < 0.001$), indicating that higher hs-CRP levels were associated with worsening stroke outcome.

In contrast, serum ferritin levels did not show significant association with mortality or functional disability. No significant correlation was observed between serum ferritin and MRS score ($r = -0.004$, $p = 0.977$), suggesting that serum ferritin may not serve as a reliable independent prognostic marker for acute ischaemic stroke in the present cohort.

The demographic and clinical profile of the study population demonstrated that acute ischaemic stroke was more common among middle-aged and elderly individuals, with clear male predominance and a high prevalence of obesity, hypertension, diabetes mellitus, smoking, tobacco use, and alcohol consumption. These findings reinforce the important contribution of metabolic and vascular risk factors in the development and

progression of cerebrovascular disease.

The significantly elevated hs-CRP levels observed among patients with poor outcomes support the central role of inflammation in the pathophysiology of acute ischaemic stroke. Inflammatory mechanisms involving endothelial dysfunction, cytokine release, oxidative stress, leukocyte activation, and blood-brain barrier disruption likely contribute to secondary neuronal injury and infarct progression, thereby worsening neurological recovery and increasing disability.

Although serum ferritin has been proposed as an inflammatory and oxidative stress-related biomarker in previous studies, the present study failed to demonstrate statistically significant prognostic value. This discrepancy may be related to variations in sample size, timing of biomarker assessment, heterogeneity of infarct distribution, obesity-related inflammation, and the nonspecific acute phase behaviour of ferritin.

REFERENCES

1. Feigin VL, Roth GA, Naghavi M, Parmar P, Krishnamurthi R, Chugh S, Mensah GA, Norrving B, Shiue I, Ng M, Estep K, Cercy K, Murray CJL, Forouzanfar MH; Global Burden of Diseases, Injuries and Risk Factors Study 2013 and Stroke Experts Writing Group. Global burden of stroke and risk factors in 188 countries, during 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. Lancet Neurol. 2016 Aug;15(9):913-924. doi: 10.1016/S1474-4422(16)30073-4. Epub 2016 Jun 9. PMID: 27291521.
2. Donnan GA, Fisher M, Macleod M, Davis SM. Stroke. Lancet. 2008 May 10;371(9624):1612-23. doi: 10.1016/S0140-6736(08)60694-7. PMID: 18468545.
3. Kamalakannan S, Gudlavalleti ASV, Gudlavalleti VSM, Goenka S, Kuper H. Incidence & prevalence of stroke in India: A systematic review. Indian J Med Res. 2017 Aug;146(2):175-185. doi: 10.4103/ijmr.IJMR_516_15. PMID: 29265018; PMCID: PMC5761027.
4. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, Grubb RL, Higashida RT, Jauch EC, Kidwell C, Lyden PD, Morgenstern LB, Qureshi AI, Rosenwasser RH, Scott PA, Wijdicks EF; American Heart Association; American Stroke Association Stroke Council; Clinical Cardiology Council; Cardiovascular Radiology and Intervention Council; Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke. 2007 May;38(5):1655-711. doi: 10.1161/STROKEAHA.107.181486. Epub 2007 Apr 12. Erratum in: Stroke. 2007 Jun;38(6):e38. Erratum in: Stroke. 2007 Sep;38(9):e96. PMID: 17431204.
5. Whiteley WN, Gupta AK, Godec T, Rostamian S, Whitehouse A, Mackay J, Sever PS. Long-Term Incidence of Stroke and Dementia in ASCOT. Stroke. 2021 Oct;52(10):3088-3096. doi: 10.1161/STROKEAHA.120.033489. Epub 2021 Jul 1. PMID: 34192893; PMCID: PMC8478091.
6. Dirnagl U, Iadecola C, Moskowitz MA. Pathobiology of ischaemic stroke: an integrated view. Trends Neurosci. 1999 Sep;22(9):391-7. doi: 10.1016/s0166-2236(99)01401-0. PMID: 10441299.
7. Iadecola C, Anrather J. The immunology of stroke: from mechanisms to translation. Nat Med. 2011 Jul 7;17(7):796-808. doi: 10.1038/nm.2399. PMID: 21738161; PMCID: PMC3137275.
8. Libby P. Inflammation in atherosclerosis. Nature. 2002 Dec 19-26;420(6917):868-74. doi: 10.1038/nature01323. PMID: 12490960.
9. Liesz A, Bauer A, Hoheisel JD, Veltkamp R. Intracerebral interleukin-10 injection modulates post-ischemic neuroinflammation: an experimental microarray study. Neurosci Lett. 2014 Sep 5;579:18-23. doi: 10.1016/j.neulet.2014.07.003. Epub 2014 Jul 11. PMID: 25019688.
10. Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation. 2003 Jan 28;107(3):363-9. doi: 10.1161/01.cir.0000053730.47739.3c. PMID: 12551853.
11. Rost NS, Wolf PA, Kase CS, Kelly-Hayes M, Silbershatz H, Massaro JM, D'Agostino RB, Franzblau C, Wilson PW. Plasma concentration of C-reactive protein and risk of ischemic stroke and transient ischemic attack: the Framingham study. Stroke. 2001 Nov;32(11):2575-9. doi: 10.1161/hs1101.098151. PMID: 11692019.
12. Weiss A, Ding Y. Inflammatory Markers and Stroke Outcomes: Lessons From Neutrophil Counts and Antiplatelet Therapy. J Am Heart Assoc. 2025 Jul 15;14(14):e043158. doi: 10.1161/JAHA.125.043158. Epub 2025 Jul 14. PMID: 40654234; PMCID: PMC12533669.
13. Winbeck K, Poppert H, Etgen T, Conrad B, Sander D. Prognostic relevance of early serial C-reactive protein measurements after first ischemic stroke. Stroke. 2002 Oct;33(10):2459-64. doi: 10.1161/01.str.0000029828.51413.82. PMID: 12364738.

14. Pepys MB, Hirschfield GM. C-reactive protein: a critical update. J Clin Invest. 2003 Jun;111(12):1805-12. doi: 10.1172/JCI18921. Erratum in: J Clin Invest. 2003 Jul;112(2):299. PMID: 12813013; PMCID: PMC161431.
15. Di Napoli M. Early inflammatory response in ischemic stroke. Thromb Res. 2001 Aug 1;103(3):261-4. doi: 10.1016/s0049-3848(01)00290-0. PMID: 11680414.
16. Kell DB, Pretorius E. Serum ferritin is an important inflammatory disease marker, as it is mainly a leakage product from damaged cells. Metallomics. 2014 Apr;6(4):748-73. doi: 10.1039/c3mt00347g. PMID: 24549403.
17. Zecca, Luigi & Youdim, Moussa & Riederer, Peter & Connor, James & Crichton, Robert. (2004). Iron, brain ageing and neurodegenerative disorders. Nature reviews. Neuroscience. 5. 863-73. 10.1038/nrn1537.
18. Selim M. Deferoxamine mesylate: a new hope for intracerebral hemorrhage: from bench to clinical trials. Stroke. 2009 Mar;40(3 Suppl):S90-1. doi: 10.1161/STROKEAHA.108.533125. Epub 2008 Dec 8. PMID: 19064798.
19. Castellanos M, Leira R, Tejada J, Gil-Peralta A, Dávalos A, Castillo J; Stroke Project, Cerebrovascular Diseases Group of the Spanish Neurological Society. Predictors of good outcome in medium to large spontaneous supratentorial intracerebral haemorrhages. J Neurol Neurosurg Psychiatry. 2005 May;76(5):691-5. doi: 10.1136/jnnp.2004.044347. PMID: 15834028; PMCID: PMC1739633.
20. Garg R, Aravind S, Kaur S, Singh Chawla SP, Aggarwal S, Goyal G. Role of serum ferritin as a prognostic marker in acute ischemic stroke: A preliminary observation. Ann Afr Med. 2020 Apr-Jun;19(2):95-102. doi: 10.4103/aam.aam_35_19. PMID: 32499465; PMCID: PMC7453951.
21. Ranjan U, Panneerselvam K. Association of serum ferritin in patients with acute ischemic stroke in tertiary care hospital. Int J Adv Med. 2023;10(7):518-522. doi:10.18203/2349-3933.ijam20231870.
22. Reddy MM, Senthilnathan P, Dhivya E, Rajasekaran D, Mohan Rao VR. Study of acute phase reactants (ESR, CRP, serum ferritin) and their relationship to the severity and outcome of acute stroke. Mallikarjuna Reddy et al Acute Phase Reactants in Acute Stroke. 2020;23(20).
23. Phad R, Farooqui S, Kusalkar S, Ghanekar J. Prognostic value of serum ferritin in acute ischemic stroke assessed by modified Rankin Scale: a prospective study. Eur J Cardiovasc Med. 2025;15(8):199–203.
24. Zhao Y, Rempe DA. Targeting astrocytes for stroke therapy. Neurotherapeutics. 2010 Oct;7(4):439-51. doi: 10.1016/j.nurt.2010.07.004. PMID: 20880507; PMCID: PMC5084305.
25. Sanober A, Khan SA, Afshan J, et al. N-terminal pro-B-type natriuretic peptide, high-sensitivity C-reactive protein and ferritin in predicting the functional outcome in stroke patients. The Journal of Clinical and Scientific Research 2023; 12: 279–284.
26. Rather SA, Farooq O, Sobia, Chacho J, Shah I. Role of serum hsCRP in predicting outcome after one week in ischemic stroke patients. Int J Med Sci Innov Res. 2024;9(5):136-143.