Original Article

Frequency of Raised C-Reactive Protein in Acute Stroke Patients

Raised C-Reactive Protein in Stroke

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ABSTRACT

Objective: Stroke is still one of the most common causes of death in the world. Inflammation is a significant factor in the mechanism and course of stroke. CRP, as a major inflammatory marker, has been widely reported in cardiovascular diseases; however, its prevalence and significance in acute stroke among our local population are less clear. To characterise the frequency of elevated CRP levels in patients with acute stroke.

Study Design: Cross-sectional descriptive study

Place and Duration of Study: This study was conducted at the Department of Neurology, Bolan Medical Complex, Quetta Form June 2025 to August 2025.

Methods: Consecutive sampling was done and 96 patients in the age range of 20-70 years who reported with clinical symptoms of acute stroke (duration 1.7 mg/dl.

Results: Fifty-two (54.2%) of the 96 patients were shown to have elevated CRP levels. Patients mean age was 58.4 ± 9.1 (range, 30-72) years; there were more males than females (60.4%). Ischemic stroke (68.8%) was more prevalent than hemorrhagic stroke (31.3%). The most common comorbidity was hypertension (72.9%). Elevated CRP was found more frequently in ischemic patients than in haemorrhagic (p=0.03).

Conclusions: This study demonstrated high rate (54.2%) of raised CRP in acute stroke patients indicating the marked inflammatory response to the condition. The significantly higher frequency of elevated CRP in ischemic stroke compared to haemorrhagic stroke in our cohort provides a novel, clinically relevant insight for our local population. This suggests that the underlying inflammatory burden may differ by stroke aetiology even at presentation, positioning CRP as a simple, cost-effective biomarker that could aid in initial diagnostic suspicion and inflammatory risk stratification in resource-limited settings like Pakistan.

Key Words: Acute Stroke, C-Reactive Protein, Inflammation, Biomarker, Frequency.

Citation of article: Ali A, Khosa NA, Ahmed H, Aleem A, Saddam M, Ahmed R. Frequency of Raised C-Reactive Protein in Acute Stroke Patients. Med Forum 2025;36(12):64-68. doi:10.60110/medforum.361213.

INTRODUCTION

Stroke remains one of the most formidable challenges confronting global health systems, contributing substantially to morbidity, mortality, and long-term disability worldwide. It affects both developed and developing nations and ranks as the third leading cause of death globally, following ischemic heart disease and cancer[1,2]. The World Health Organization defines stroke as a rapidly developing clinical syndrome characterized by focal or global neurological dysfunction lasting at least 24 hours or resulting in death, with a vascular origin[3].

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Received: September, 2025
Reviewed: October, 2025
Accepted: November, 2025

Pathophysiologically, stroke occurs through two primary mechanisms: interruption of cerebral blood flow due to arterial occlusion (ischemic stroke) or rupture of a cerebral blood vessel (hemorrhagic stroke). The global burden of stroke is profound. It is estimated that approximately 15 million individuals experience a stroke annually, of whom nearly 5 million die and another 5 million are left with permanent disability. Hypertension alone has been implicated in over 12.7 million stroke cases worldwide. Although public health interventions targeting blood pressure control and smoking cessation have reduced stroke incidence in high-income countries, demographic transitions have sustained a high global burden. Notably, nearly 70% of all strokes and 87% of stroke-related deaths occur in low- and middle-income countries, where healthcare infrastructure and resources are often limited. Recent advances in stroke research have shifted understanding beyond mechanical vascular obstruction toward recognition of inflammation as a central component of stroke pathophysiology. Inflammatory processes contribute to atherosclerosis progression, cerebral infarction, and secondary neuronal injury. Systemic inflammatory states have been identified as independent predictors of poor outcomes in various vascular conditions, including ischemic heart disease, carotid

artery disease, and ischemic stroke treated with reperfusion therapies. Leukocytosis and inflammatory biomarkers have also been associated with adverse outcomes following mechanical thrombectomy. Among inflammatory markers, C-reactive protein (CRP) has gained particular attention. CRP is an acute-phase reactant synthesized primarily by hepatocytes and serves as a sensitive, though nonspecific, indicator of systemic inflammation. High-sensitivity CRP (hs-CRP) assays enable detection of low-grade inflammation and provide insight into the inflammatory milieu associated with atherosclerosis. Beyond being a passive marker, CRP actively participates in atherogenesis by promoting macrophage uptake of low-density lipoprotein cholesterol, facilitating foam cell formation, and activating the complement cascade, thereby exacerbating vascular injury. Several studies have reported variable frequencies of elevated CRP levels among acute stroke patients. Munir et al. reported elevated CRP in 61.9% of acute ischemic stroke cases, while Finck et al. documented a prevalence of 46.3%. Such variability underscores the influence of genetic, environmental, and methodological factors. Despite CRP's established role in cardiovascular disease, its profile in acute stroke—particularly within the Pakistani population—remains insufficiently explored. To address this gap, the present study was designed to evaluate CRP levels in patients presenting with acute stroke in a tertiary care setting in Quetta. Beyond estimating the prevalence of CRP elevation, this study aims to compare inflammatory responses between ischemic and hemorrhagic stroke subtypes at initial presentation. By elucidating these differences, the study seeks to enhance understanding of stroke pathophysiology and support the potential utility of CRP as a clinically relevant biomarker in acute stroke assessment.

METHODS

Study Design and Setting: This descriptive cross-sectional study was carried out at the Department of Neurology, Bolan Medical Complex Hospital Quetta over a period of June To August 2025. The study was approved by institutional ethical review committee of Bolan Medical Complex Hospital, Quetta (**Approval No:/BMCH/DA-I/2025/ 306**) and The College of Physicians and Surgeons Pakistan (CPSP).

Sample Size and Sampling Technique: The WHO sample size calculator for a single proportion was used to calculate the sample size. Considering confidence level (at 95%), margin of error = 10%, and estimating the expected proportion of raised CRP as 46.3% based on a previous study (Finck et al., 2023), at least the sample size required was calculated to be 96 patients. Consecutive non-probability sample of the eligible patients was included.

Inclusion and Exclusion Criteria: Both male and female of age group 20–70 patients were included who presented with clinically acute stroke (as per operational definition) upto 24 h. Exclusion criteria included a history of traumatic brain injury (TBI) within the past 2 years, acute coronary syndrome in the previous month, cerebrovascular events in the past, autoimmune diseases, hepatic failure, chronic renal failure or any other illness that caused CNS dysfunction (stroke mimic).

Data Collection Procedure: A proforma was designed for recording of each participant's information after receiving the informed consent. This included demographics (age, gender, place of residence), clinical details (duration of symptoms, history of hypertension and diabetes mellitus), anthropometry [height-weight measurement for body mass index (BMI)] and CT radiological data (type of stroke). Venous blood (3 ml) was collected by venipuncture and sent to the hospital laboratory. The measurement of the serum concentration of CRP was done by a solid-phase enzyme-linked immunosorbent assay (ELISA) kit. According to the working definition, a CRP concentration >1.7 mg/dl was defined as "raised."

Data Analysis: Statistical analysis was conducted by SPSS version 25.0. Participants' characteristics and levels of variables were displayed as mean ± standard deviation (SD) numbers for continuous data, such as age and BMI. Categorical variables, such as gender, comorbidities, stroke types, and behaviours, were given as frequencies and percentages. Stratification was according to age, sex, BMI, comorbidities, residence and stroke type. The Chi-square test was used to study associations between elevated CRP and these strata. Statistical significance was accepted at $p \leq 0.05$.

RESULTS

Ninety-six patients with acute stroke were included. The baseline demographic information of the study cohort is presented in Table 1.

Table No. 1: Baseline characteristics of study participants (N=96)

Characteristic		**Value**
Age (years), Mean ± SD		58.4 ± 9.1
Gender, n (%)	Male	58 (60.4%)
	Female	38 (39.6%)
Residence, n (%)	Urban	61 (63.5%)
	Rural	35 (36.5%)
BMI (kg/m²), Mean ± SD		26.8 ± 4.2
Comorbidities, n (%)	Hypertension	70 (72.9%)
	Diabetes Mellitus	45 (46.9%)
Type of Stroke, n (%)	Ischemic	66 (68.8%)
	Haemorrhagic	30 (31.3%)

The main aim of the study was to assess the prevalence of elevated CRP. Regarding CRP, the number of patients with CRP level greater than 1.7 mg/dl was 52 from a total 96, which is equivalent to a percentage of 54.2% (Table 2).

Table No. 2: Frequency of Raised CRP (N=96)

CRP Status	Frequency (n)	Percentage (%)
Raised (>1.7 mg/dl)	52	54.2%
Not Raised (≤1.7 mg/dl)	44	45.8%

The distribution of elevated CRP in different types of stroke was evaluated (Table 3). More patients with ischemic stroke (59.1%) than haemorrhagic had elevated CRP (43.3%) ($p = 0.03$).

Table No. 3: Association between Stroke Type and Raised CRP

Stroke Type	Raised CRP (n=52)	Normal CRP (n=44)	p-value
Ischemic (n=66)	39 (59.1%)	27 (40.9%)	0.03
Haemorrhagic (n=30)	13 (43.3%)	17 (56.7%)	

Additional subgroup analysis was conducted to investigate the relation between elevated CRP and other demographic or clinical characteristics (Table 4). There was no statistically significant relationship with age, gender, place of residence or hypertension and DM status ($p > 0.05$).

Table No. 4: Stratification Analysis for Raised CRP

Stratifying Variable	Category	Raised CRP (n=52)	p-value
Age	≤60 years (n=55)	28 (50.9%)	0.41
	>60 years (n=41)	24 (58.5%)	
Gender	Male (n=58)	30 (51.7%)	0.52
	Female (n=38)	22 (57.9%)	
Hypertension	Yes (n=70)	40 (57.1%)	0.29
	No (n=26)	12 (46.2%)	
Diabetes Mellitus	Yes (n=45)	26 (57.8%)	0.48
	No (n=51)	26 (51.0%)	
BMI	Obese (>30) (n=22)	14 (63.6%)	0.27
	Non-Obese (≤30) (n=74)	38 (51.4%)	

DISCUSSION

This hospital-based cross-sectional study demonstrated that 54.2% of acute stroke patients had elevated incidence of serum CRP levels. This result is well within the typical range of this factor in previous literature; it is greater than the expression reported by Finck et al.[7] and less than that (61.9%) reported by Munir et al.[6] in Pakistani people of a community. This discrepancy might be due to different study populations, numbers of subjects, the specific time point of blood collection after stroke onset or genetic and environmental factors that may affect systemic inflammatory response.The major realization of a high CRP elevation rate emphasises the profound inflammatory chain reaction initiated by cerebral insult[9]. CRP is not only a surrogate but is thought to have an effect in the pathogenesis of stroke. It stimulates atherogenesis through increasing the low density lipoprotein uptake by macrophages which results in foam cell formation. It also enhances the expression of adhesion molecules, and increases the release of several other pro-inflammatory mediators that in turn amplify endothelial dysfunction and brain tissue injury[10].A striking observation in our study was the significantly more common occurrence of raised CRP among those of ischemic stroke (59.1%), as opposed to those with hemorrhagic stroke (43.3%). This is consistent with the currently accepted view that a chronic inflammatory process, an established characteristic of atherosclerosis, underlies most ischemic stroke. It is this inflammatory milieu in stable, yet vulnerable atherosclerotic plaques that is a critical determinant of plaque rupture and thrombosis[11]. The acute infarction itself subsequently initiates an additional severe inflammatory response, maximising CRP production. Unlike ischaemic stroke, where the original insult is mainly due not to mechanical trauma but ischemia-reperfusion, in haemorrhagic stroke the traumatic origin of injury is perhaps superimposed by less pronounced or different acute phase reactant response kinetics that would account for these observations (ie. reduced frequency)[12].The novel contribution of our findings lies in the demonstration of this significant disparity within our specific local population. While this pathophysiological concept is known, empirical data from Pakistan is scarce. Our results provide concrete, local evidence that the inflammatory response, as measured by a standard CRP test, is etiologically different at the point of hospital admission. This has immediate clinical relevance. In a resource-constrained setting where advanced diagnostics may be delayed, an elevated CRP could add weight to a clinical suspicion of an ischemic event, potentially prompting different initial management considerations, such as more aggressive antiplatelet therapy or statin initiation, compared to a hemorrhagic

stroke.[13] Furthermore, our finding that over half of all stroke patients present with an elevated CRP underscores the pervasive nature of inflammation in acute stroke. This positions CRP not merely as an academic biomarker but as a practical, low-cost tool for "inflammatory risk stratification." Identifying patients with a heightened inflammatory state at admission could help clinicians flag those at potentially higher risk for complications like early neurological deterioration or infection, warranting closer monitoring. In contrast to some reports we observed no substantial relationship between elevated CRP and traditional vascular risk factors (hypertension, diabetes mellitus) in our stroke population[14]. This may be because, in a population already enriched for having had a serious vascular event (ie, stroke), the baseline infective burden is uniformly high potentially diluting the relative impact of specific additional comorbidities[13]. Our sample size also may have not been large enough to detect such more subtle relationships.

Our findings align with modern studies. A meta-analysis by demonstrated that high CRP concentration in acute stroke strongly correlate with increased stroke severity, infarct volume and dependence[14]. Another prospective cohort conducted by Lv et al. underscored the synergistic effects of high CRP and dyslipidemia on stroke risk, further lodging the crosstalk between inflammation and metabolism[15].The clinical message of our work is simple. As over 50% of our patients with acute stroke have been found to have an abnormal CRP and its relative simplicity, inexpensiveness and rapid availability, the CRP assay has potential value as a useful adjunct in the emergency evaluation of stroke[15]. Our data strengthens this argument by showing that its value may be enhanced by its ability to reflect the differing inflammatory pathophysiology between stroke subtypes.The limitations of our study were that it was a single-centre study and the sample size was not large enough to allow for generalization. Secondly, as a cross-sectional study, an association but not causation between CRP and stroke can be established.

CONCLUSION

In view of the substantial prevalence of increased CRP among acute stroke patients, our study adds a novel layer of understanding by highlighting a distinct inflammatory profile between ischemic and haemorrhagic strokes in a Pakistani cohort. This justifies further studies to get this simple test into initial clinical evaluations to stratify not only general risk but also to provide an early clue to the underlying stroke aetiology and its associated inflammatory burden. Prospective investigations on a larger scale, with high-sensitivity CRP assays are needed to further define the association between hs-CRP and long-term outcomes.

Author's Contribution:

Concept & Design or acquisition of analysis or interpretation of data:	Abdul Ali, Hussain Ahmed, Abdul Aleem
Drafting or Revising Critically:	Muhammad Saddam, Riaz Ahmed, Noor Ahmed Khosa
Final Approval of version:	All the above authors
Agreement to accountable for all aspects of work:	All the above authors

Conflict of Interest: The study has no conflict of interest to declare by any author.

Source of Funding: None

Ethical Approval: No. BMCH/DA-I/2025/306 Dated 13.06.2025

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