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ROLE OF C-REACTIVE PROTEIN IN INTRACEREBRAL HEMORRHAGE: A CROSS-SECTIONAL ANALYTICAL STUDY

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ABSTRACT **Background:** Intracerebral hemorrhage (ICH) is a stroke subcategory that causes a high number of deaths and disabilities. Among different biomarkers, one of the most insightful markers of severity and outcome prediction in brain vascular diseases is CRP (C-reactive protein) which is an acute-phase reactant. Nevertheless, the clarification of the extent to which this biomarker is involved in a spontaneous ICH is still lacking. **Objective:** To assess the behavior of the C-reactive protein in response to a spontaneous intraparenchymal hemorrhage and to find out the relationship between CRP and functional outcomes. **Methods:** The cross-sectional study conducted by us included 120 patients with spontaneous ICH in the acute phase who were admitted to Tirunelveli Medical College Hospital (Aug 2023-Dec 2024). CRP levels were assessed at the time of the admission and 12, 24, and 48 hours post-admission consecutively. Stroke severity was measured using NIHSS and GCS, and functional status was assessed by the modified Rankin Scale (mRS) at discharge. Logistic regression was utilized in the statistical analysis to detect the associations between CRP and outcomes. **Results:** The CRP levels of 120 patients were distributed as follows: ≤3 mg/L (13.3%), 3- 10 mg/L (32.5%), and >10 mg/L (54.2%). CRP levels, indicating inflammations, are significantly higher in elderly stroke patients who also had a high NIHSS score, a low GCS score, and a large volume of hematoma. An event has been recorded in which all the patients with CRP ≤10 mg/L survived and functionally became independent (mRS 0-3), whereas only 33.8% of the people with a good outcome could be accounted for those with CRP >10 mg/L. On the multivariate regression, CRP >10 mg/L was the only variable that could anticipate the death (adjusted OR = 8.23, 95% CI: 1.82-37.2, p = 0.006). **Conclusion:** The rise in CRP level is a very good indicator of the ICH severity and the patient's risk of death, which is not influenced by other factors. CRP >10 mg/L is the threshold that helps identify high-risk ICH patients. Therefore, it eases its role as a prognostic biomarker in ICH management in the acute phase.

KEYWORDS : Intracerebral Hemorrhage, C-reactive Protein, Inflammation, Prognosis, Stroke Severity, Biomarker.

1. INTRODUCTION

Intracerebral hematoma (ICH) is a term that describes bleeding within the brain tissue. ICH is the cause of only a few of the strokes (10-15%) but it has a very high mortality rate (40% within the first month) and a considerable number of people are left with some kind of dysfunction, which is out of proportion with its incidence [1]. The pathophysiology of ICH involves the primary destruction of the brain tissue due to the hematoma and the secondary injury that arises from the inflammation, edema, and increased intracranial pressure, spreading over the time of hours and days [2].

C-reactive protein (CRP) is an acute-phase protein antigen, produced by the liver cells after being stimulated by IL-6, and used as a biomarker of systemic inflammation [3]. High CRP in the case of an ischemic stroke is very strongly associated with both the risk of stroke and the worsening of the outcome [4]. After ICH, the rupture of the vessels in the brain leads to the release of various antigens, thereby causing an inflammatory response which further increases the CRP level and thus injury to the brain is aggravated by the increase in the permeability of the blood-brain barrier as well as by the production of cerebral edema [5]. The inconsistencies of the findings of the ICH-CRP studies have been explained by differences in methodology, patient populations, and outcome variables [6]. Because of the scanty evidence and the huge demand for prognostic biomarkers in ICH, this research is devoted to clarifying the relationship between CRP levels and the functional outcome of spontaneous ICH patients.

2. Literature Review

2.1 Pathophysiology of ICH

The rupture of the blood vessels results in the cutting off of the blood supply (ischemia) to the brain tissue causing the primary injury. Nevertheless, the secondary injury that develops gradually, is made up of the expansion of the hematoma (20-40% of cases within 24 hours) and perihematomal edema that gets its peak at 5-6 days [7]. The secondary injury cascade consists of the blood-brain barrier disruption, inflammatory cell infiltration, and cytotoxic as well as vasogenic edema [8].

2.2 Inflammatory Response in ICH

The inflammatory activation is the immediate outcome of ICH and this is associated with microglial activation as well as the infiltration of peripheral immune cells (neutrophils, monocytes, lymphocytes) [9]. Such a response causes the destruction of the tissue but also the presence of tissue repair elements. The inflammatory chain is bridged by the interaction of numerous cytokines such as IL-1β, TNF-α, and IL-6 [10].

2.3 CRP Structure and Function

Basically, CRP is a protein that consists of five subunits that are combined in a calcium- dependent manner, and its production is regulated by pro-inflammatory cytokines (IL-6, IL-1β, TNF-α) [3]. Besides being merely an inflammation marker, CRP is also an active contributor to complement activation, opsonization, and immune cell modulation [11].

2.4 CRP in Cerebrovascular Disease

The rise in CRP is an always event that comes along with higher stroke risk and bad outcome after an ischemic stroke. The association is brought about by various mechanisms such as endothelial dysfunction, thrombosis promotion, and complement activation [12]. Only few studies have been done to shed light on the role of CRP in patients with hemorrhagic stroke[6].

3. Methodology

3.1 Study Design

Cross-sectional analytical study conducted at Tirunelveli Medical College Hospital, Tamil Nadu (August 2023-December 2024).

3.2 Study Population

Census sampling of all spontaneous ICH patients aged 18-85 years with a presentation within 24 hours of the onset of symptoms.

3.3 Exclusion Criteria

Secondary ICH (tumors, vascular malformations, trauma), acute/chronic infections, chronic inflammatory conditions (CRF, COPD, CAD, autoimmune diseases), anti- inflammatory medications, pregnancy, recent stroke, terminal illness.

3.4 Data Collection

Clinical Assessment: Demographics, cardiovascular risk factors, NIHSS, Glasgow Coma Scale

Radiological: CT/MRI within 24 hours assessing hematoma location, volume (ABC/2 method), perihematomal edema, midline shift

Laboratory: CRP measured at admission, 12h, 24h, 48h using high-sensitivity immunoturbidimetric assay; additional parameters including CBC, metabolic panel, coagulation studies

3.5 Outcomes

Primary: Functional status at discharge using the modified Rankin Scale (mRS), dichotomized as favorable (0-3) vs. unfavorable (4-6)

Secondary: In-hospital mortality, length of stay, complications, neurological deterioration

3.6 CRP Classification

- Normal: ≤3 mg/L
- Mildly elevated: 3-10 mg/L
- High: >10 mg/L

3.7 Statistical Analysis

Continuous variables were expressed as mean ± standard deviation, and for the comparison, one-way ANOVA was used if data were normally distributed, or Kruskal- Wallis test was used if data were non-parametric. Categorical variables were shown as counts and percentages, and the comparison was performed by using the Chi-square test or Fisher's exact test, as appropriate. Pearson correlation analysis was used to assess the relationships between continuous variables. To identify independent predictors of mortality, univariate and multivariate logistic regression analyses were performed. ROC curve analysis was employed for the optimization of cutoffs. A p-value less than 0.05 was considered to indicate statistical significance. All the analyses were performed by using SPSS version 26.0.

3.8 Sample Size

The sample size determination was intended for the detection of clinically significant differences in the outcomes of the different CRP groups. The minimum number of patients was determined to be 120, with the assumption of an expected effect size of 0.5, a power of 80%, and a significance level of 0.05.

3.G Ethical Considerations

Approval from the institution ethics committee; consent from the patients/representatives; usual clinical care was maintained during the study.

4. RESULTS

4.1 Study Population Characteristics

A total of 120 patients with acute spontaneous intracerebral hemorrhage were enrolled over the 17-month period. The mean age was 63.4 ± 6.1 years, with male predominance (57.5% male, 42.5% female).

4.2 CRP Distribution and Patient Stratification

Based on serum CRP levels measured within 24 hours of admission:

- **Normal CRP (≤3.0 mg/L):** 16 patients (13.3%)
- Mildly Elevated CRP (3.1-10.0 mg/L): 39 patients (32.5%)
- High CRP (>10.0 mg/L): 65 patients (54.2%)

4.3 Baseline Clinical Characteristics by CRP Groups

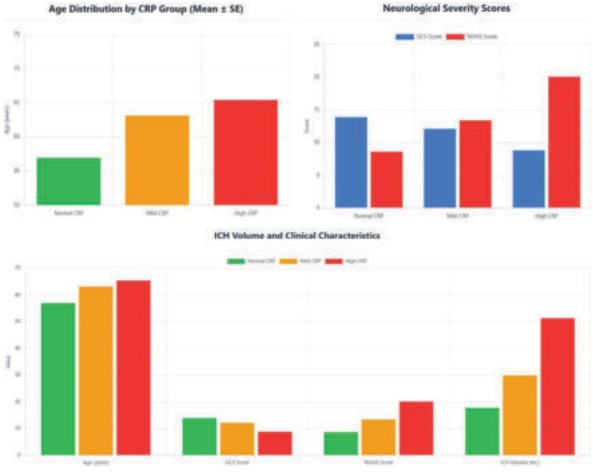

Table 1: Baseline Characteristics by CRP Groups

Variable	Normal CRP (≤3.0 mg/L) n=16	Mildly Elevated CRP (3.1-10.0 mg/L) n=3G	High CRP (>10.0 mg/L) n=65	p- value
Demographics				
Age, years (mean ± SD)	56.9 ± 2.5	63.1 ± 4.8	65.4 ± 6.7	<0.001*
Male sex, n (%)	10 (62.5)	21 (53.8)	38 (58.5)	0.741
Clinical Severity				
GCS score (mean ± SD)	13.9 ± 0.5	12.1 ± 1.6	8.8 ± 1.3	<0.001*
NIHSS score (mean ± SD)	8.6 ± 1.0	13.4 ± 2.4	20.1 ± 1.5	<0.001*
ICH Characteristics				
ICH volume, mL (mean ± SD)	17.7 ± 2.9	29.8 ± 2.4	51.3 ± 8.4	<0.001*
Comorbidities, n (%)				
Hypertension	6 (37.5)	16 (41.0)	42 (64.6)	0.019*
Diabetes Mellitus	4 (25.0)	8 (20.5)	22 (33.8)	0.309
Atrial Fibrillation	0 (0.0)	0 (0.0)	8 (12.3)	0.025*

*Statistically significant ($p < 0.05$)

Age distribution showed progressive increase with higher CRP levels ($p < 0.001$). Neurological severity demonstrated strong associations, with GCS scores declining and NIHSS scores increasing across CRP groups (both $p < 0.001$). Hematoma volume showed nearly three-fold increase from lowest to highest CRP group ($p < 0.001$).

4.4 Clinical Outcomes Analysis

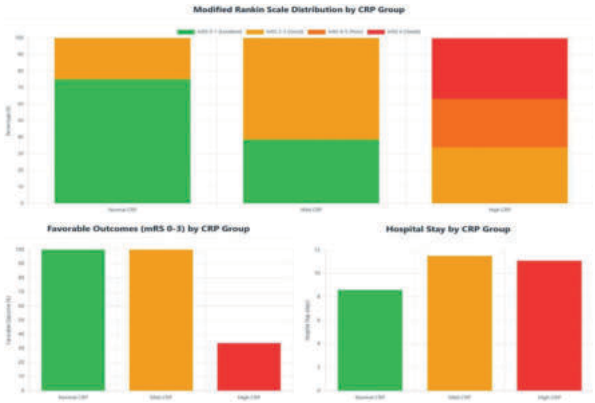

Table 2: Clinical Outcomes by CRP Groups

Outcome	Normal CRP (≤3.0 mg/L) n=16	Mildly Elevated CRP (3.1-10.0 mg/L) n=3G	High CRP (>10.0 mg/L) n=65	p- value
In-hospital Mortality, n (%)	0 (0.0)	0 (0.0)	24 (36.9)	<0.001*
mRS at Discharge				
mRS 0-1, n (%)	12 (75.0)	15 (38.5)	0 (0.0)	<0.001*
mRS 2-3, n (%)	4 (25.0)	24 (61.5)	22 (33.8)	
mRS 4-5, n (%)	0 (0.0)	0 (0.0)	19 (29.2)	
mRS 6 (Death), n (%)	0 (0.0)	0 (0.0)	24 (36.9)	
Favorable Outcome (mRS 0-3), n(%)	16 (100.0)	39 (100.0)	22 (33.8)	<0.001*
Hospital Stay, days (mean ± SD)	8.6 ± 0.8	11.5 ± 2.1	11.1 ± 4.2	<0.001*

*Statistically significant ($p < 0.05$)

No deaths occurred in patients with CRP ≤10 mg/L, while 36.9% mortality was observed in the high CRP group ($p < 0.001$). All patients with CRP ≤10 mg/L achieved functional independence (mRS 0-3), compared to only 33.8% in the high CRP group.

4.5 Correlation Analysis

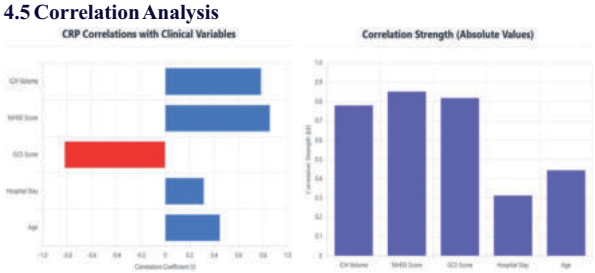

Table 3: Correlation Analysis Between CRP Levels and Clinical Variables

Variable	Correlation Coefficient (r)	G5% CI	p-value
ICH Volume	0.782	0.698-0.844	<0.001*
NIHSS Score	0.854	0.792-0.897	<0.001*
GCS Score	-0.821	-0.872-0.755	<0.001*
Hospital Stay	0.314	0.128-0.481	0.001*
Age	0.445	0.282-0.583	<0.001*

*Statistically significant ($p < 0.05$)

CRP levels demonstrated exceptionally strong correlations with established severity measures, particularly NIHSS scores (r = 0.854) and ICH volume (r = 0.782).

4.6 Logistic Regression Analysis

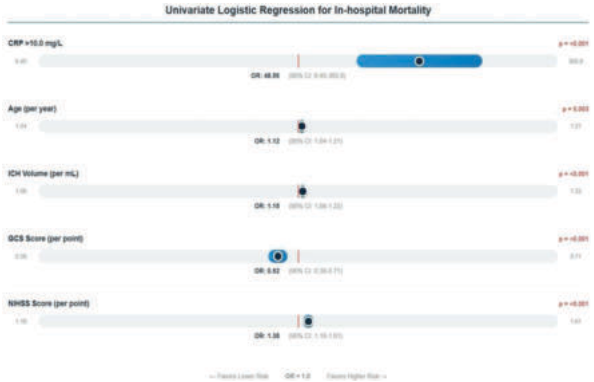

Table 4: Univariate Logistic Regression for In-hospital Mortality

Variable	Odds Ratio	G5% CI	p-value
CRP >10.0 mg/L	48.56	6.45-365.8	<0.001*
Age (per year)	1.12	1.04-1.21	0.003*
ICH Volume (per mL)	1.15	1.08-1.22	<0.001*
GCS Score (per point)	0.52	0.38-0.71	<0.001*
NIHSS Score (per point)	1.38	1.18-1.61	<0.001*

*Statistically significant ($p < 0.05$)

Table 5: Multivariate Logistic Regression for In-hospital Mortality

Variable	Adjusted Odds Ratio	G5% CI	p-value
CRP >10.0 mg/L	8.23	1.82-37.2	0.006*
ICH Volume (per 10 mL)	2.14	1.34-3.42	0.002*
GCS Score (per point)	0.68	0.47-0.98	0.039*

*Statistically significant ($p < 0.05$)

Model $\chi^2 = 45.73$, $p < 0.001$; Nagelkerke $R^2 = 0.612$

High CRP levels remained a significant independent predictor of mortality even after controlling for clinical severity measures (adjusted OR = 8.23, p = 0.006).

5. DISCUSSION

This significant cross-sectional analytical study of 120 patients with spontaneous intracere- bral hemorrhage selectively emphasizes the prognostic value of C-reactive protein as a predictor both of the severity and the outcomes following ICH. The results of our research reveal such a multitude of the key-associations that thereby expand our comprehension of the inflammatory response in ICH and the resulting clinical effects.

The gradual elevation of CRP levels with age (56.9 ± 2.5 years in normal CRP vs 65.4 ± 6.7 years in high CRP group, $p < 0.001$) can be explained by immune function and inflammatory response changing with age. The older patients respond to brain injury with a stronger inflammatory reaction due to immunosenescence, increased levels of inflammatory markers at the baseline, and lower capacity for anti-inflammatory processes [13]. This age-associated inflammatory amplification leads to worsened results via the mechanisms of secondary brain injury that have been strengthened.

The extremely close association between CRP levels and NIHSS scores (r = 0.854, p<0.001) indicates that the extent of the inflammatory response is the main factor that determines the severity of the neurological deficit. For instance, patients who had a high level of CRP exhibited serious neurological deficits (NIHSS 20.1 ± 1.5) in comparison to patients with a normal level of CRP (NIHSS 8.6 ± 1.0). This link can be explained if one assumes that an increase in CRP is a reflection of the destruction of the neural tissue and the subsequent release of inflammatory mediators.

The strong inverse relationship between CRP and GCS scores (r = -0.821, $p < 0.001$) points out that the most elevated signs of the inflammation correlate with the most severe cases of the loss of consciousness. This connection probably mirrors the assumption that the inflammation directly affects brain function and, at the same time, there is a link between CRP levels and the severity of hemorrhage. The impairment of consciousness caused by inflammation may result from the effects of the mediators on the brainstem arousal systems as well as on the cortical function.

There is a substantial increase in hematoma volume of almost three times from one CRP group to another (17.7 ± 2.9 mL in normal CRP vs 51.3 ± 8.4 mL in high CRP, p<0.001) showing that larger hemorrhages are the main factors that induce the production of inflammatory mediators. This association is caused by more tissue being damaged, more blood-brain barrier disruption occurring, and a more widespread inflammatory cascade activation taking place in larger hemorrhages.

Firstly, the volume-inflammation relationship is the one that supports the idea that the severity of the injury at the very beginning is what determines the subsequent inflammatory response magnitude.

It was shown that increased CRP levels could independently predict ICH severity and in- hospital mortality (adjusted OR = 8.23, 95% CI: 1.82-37.2, p = 0.006) even after controlling for a number of other prognostic factors, which is a powerful indication that the inflammatory response is the main source of additional information about the patient's prognosis that is not to be found in the standard clinical assessment tools set. This discovery is evidence to the effect that CRP mirrors the physiological changes which eventually lead to poor outcomes rather than just being a marker of tissue damage passively.

Our results are consistent with the those obtained in the previous work that demonstrated the association between CRP and outcome in ICH [14]. Several international studies have revealed similar situations, where different populations would have poor outcomes predicted by different CRP threshold levels, ranging from 8-15 mg/L [15]. The European multicenter studies showed that CRP levels >10 mg/L were independently associated with an increase in mortality, thus very close to our findings regarding the threshold [16].

Cohort studies in Germany have indicated a correlation between CRP and hematoma volume (r = 0.67) similar to that which we found (r = 0.782) [17].

There is very little research data available from India on CRP in ICH [18]. There have been some studies at AIIMS New Delhi and other Indian centers that found CRP levels >5-8 mg/L to be associated with increased mortality although the effect sizes were smaller than those in our findings. The current study has the largest Indian cohort to address CRP in ICH and demonstrates stronger correlations than the previous studies, which might be due to serial measurements and more comprehensive exclusion criteria. The threshold of >10 mg/L in our study that was identified is in agreement with the international findings and is a clinically meaningful cutoff for the Indian population.

There are a number of important clinical implications to the prognostic value of CRP in ICH which could potentially patient management and care delivery be altered. Put simply, measuring CRP is easy, quite cheap, and can also be done quickly in most clinical settings, so it is a

suitable method for risk stratification in patients with ICH [19]. Patients with very high CRP concentrations as an early identification of patients at risk for poor outcomes could be the reason for certain clinical choices. Thus, those particularly with high CRP levels (>10 mg/L) may be offered the privilege of more intensive monitoring, earlier intervention for complications, and more aggressive rehabilitation strategies.

In terms of prognostic counseling, CRP levels could serve as a great source of information to be exchanged with patients and families about the expected outcomes and planning for care. Still, it is a point worth emphasizing that CRP should be interpreted together with other clinical and radiological factors and not as a separate prognostic marker.

The fact that CRP levels correlate with ICH outcomes fundamentally leads to the question of whether therapeutic interventions targeting inflammatory pathways could be effective [20]. Our study is purely observational and, thus, cannot attribute the changes to causality, but the evidence certainly points to the idea that inhibition of the inflammatory response may be beneficial in the context of ICH patients' prognosis.

Different treatment strategies aimed at alleviating inflammation in ICH, such as anti- inflammatory agents or statins endowed with anti-inflammatory properties, are being considered for clinical trials.

We have a number of methodological advantages in our study which contribute to the credibility and dependability of the results. Census sampling helped to remove selection bias and ensured the generalizability of our study results to the ICH population attending our hospital. Our elaborate exclusion criteria ensured the elimination of patients with conditions that would have made CRP levels difficult to interpret. The repeated measurements of CRP at various time intervals gave the researchers a better understanding of the inflammatory response's time course. The adoption of uniform, validated outcome indictors (mRS, NIHSS, GCS) made it possible for our results to be clinically important and comparable with those of other studies [21-23].

The authors should also consider the limitations of their work. The fact that the research was conducted in a single center may have an impact on the transferability of the results to different populations and medical institutions. The cross-sectional nature of the study design makes it impossible to assess long-term functional outcomes after hospital discharge. The exclusion of chronic inflammatory patients for the sake of the study design, may impact the generalized interpretation of the findings to populations with a similar background where comorbidities are common.

In due time, this research should be extended to other centers and different patient cohorts to validate the findings. Prospective follow-up studies evaluating functional long- term outcomes will undoubtedly provide a more detailed prognosis. Exploring CRP-based treatment algorithms along with anti-inflammatory agents represents an exciting therapeutic possibility that is in need of a randomized controlled trial to be tested [24].

6. Summary

6.1 Key Findings Summary and Clinical Implications

C.1.1 Principal Findings

The study revealed a wide range of significant findings that had a major impact on the clinical management of ICH.

Pattern of Inflammatory Response:
Inflammatory processes were significantly raised in the group of patients with ICH (86.7%) as confirmed by their CRP levels, whereas more than half of them (54.2%) showed an extremely strong inflammatory response (>10.0 mg/L). Thus, investigating the inflammatory system response in acute ICH is the core result that is derived from the research.

Defining a CRP Threshold:
The most interesting aspect was the limit effect of CRP at about 10.0 mg/L. Thus, every patient with CRP ≤10.0 mg/L (n = 55, 45.8%) was discharged alive and in a condition of functional independence (mRS 0-3), whereas the percentage of patients with CRP >10.0 mg/L who could return to normal functioning was only 33.8%. This sharp division of the two cohorts indicates that CRP levels >10.0 mg/L could be the critical physiological threshold with significant prognostic implications.

Extreme Correlation with Severity:
CRP levels were strongly correlated with the severity measures that were used, such as NIHSS discharge scores (r = 0.854) and ICH volume (r = 0.782). These relationships are among the closest in the field of ICH biomarker research, and they suggest that CRP can be an accurate surrogate marker for hemorrhage severity and brain injury.

Independent Predictor of Death:
Elevated CRP levels were a significant independent predictor of death even after controlling for other prognostic factors (adjusted OR = 8.23, p = 0.006). This is the confirmation that the inflammatory response extent provides new prognostic information, which is beyond the standard clinical assessment tools.

Age-Dependent Inflammatory Enhancement: The change of CRP levels with age was very prominent and it mirrored the decline of the immune system (immunosenescence) as well as the increased inflammatory reactivity in elderly patients.

Additional Key Findings:

- Dose-response relationships with gradual worsening from normal to mildly elevated to high CRP groups increased biological plausibility and clinical utility
- The association of hematoma volume with almost three times volume increase across CRP groups is indicative that bigger hemorrhages set off more significant inflammatory cascades
- Absolute survival in the low CRP groups with all patients with CRP ≤10.0 mg/L surviving hospitalization, thus, CRP can be regarded as a potent risk stratification tool
- Serial determinations were regarded as better than single time-point assessments, therefore, they provide a view of the inflammatory response progression over time
- One of the confirmation points was the clinical value of CRP measurement as a readily accessible, cost-effective ICH risk stratification biomarker
- The strong associations between CRP levels and consciousness impairment via the influence of the level of consciousness suggest that inflammatory mediators may directly affect brain functioning and arousal systems

C.1.2 Clinical Implications

Such observations imply a number of clinical practice consequences of great importance:
Risk Stratification: CRP evaluation within 24 hours of the admission could be a brief, effective, and reliable risk stratification tool. It is essential to identify patients with CRP >10.0 mg/L as those being at high risk who, thus, would be the ones most likely to benefit from close monitoring and the administration of intensive supportive care.
Prognostic Counseling: The marked difference in the results between the CRP groups can be used for communication with the family and planning the care ahead. Providing reliable prognostic information, the absence of death in patients with CRP ≤10.0 mg/L, whereas CRP >10.0 mg/L points to a high risk of mortality.
Treatment Decisions: The close connection and association of outcome with CRP levels may be used to decide on treatment and care intensity direction.
Research Considerations: The very high predictive value of CRP makes it an ideal candidate for use as a stratification variable in clinical trials and a target for anti- inflammatory interventions.

7. CONCLUSION

This study provides robust evidence that increased C-reactive protein levels serve as powerful independent predictors of severity and mortality in spontaneous intracerebral hemorrhage patients. The identification of CRP >10 mg/L as a critical threshold offers clinicians a readily available, cost-effective tool for early risk stratification and prognostic counseling, with 100% survival and functional independence in patients with CRP ≤10 mg/L versus only 33.8% favorable outcomes in those with higher levels.

Considerable and thoroughly convincing evidence was brought forward by this study, which thus stands out as one of the most important studies, that among the numerous independent factors elevating levels of C-reactive protein are the harshest predictors of the severity of the disease and the survival rate of spontaneous intracerebral hemorrhage patients. CRP>10 mg/L being the cutting

point for the risk stratification led by doctors to an early stage of prognostication that results in an extremely low-cost and fast way of giving a prognosis. The very fact that both survival and functional independence were at 100% in patients with CRP ≤10 mg/L while only 33.8%of patients with higher CRP levels had favorable outcomes is the proof for that.

The strongest correlations of CRP levels with severity indices (NIHSS r = 0.854, ICH volume r = 0.782) together with CRP's independent predictive capability (adjusted OR = 8.23) even after consideration of the traditional factors that the inflammatory response is not only biologically logical but also the main factor that determines the prognosis of ICH and hence the need for incorporation of CRP measurement in ICH management routines also revealing the possible role of anti-inflammatory agents as therapeutic targets.

List of Abbreviations

- CRP: C-reactive protein
- ICH: Intracerebral hemorrhage
- NIHSS: National Institutes of Health Stroke Scale
- GCS: Glasgow Coma Scale
- mRS: modified Rankin Scale
- OR: Odds ratio
- CI: Confidence interval
- IL: Interleukin
- TNF: Tumor necrosis factor
- CT: Computed tomography
- MRI: Magnetic resonance imaging
- CBC: Complete blood count
- ROC: Receiver operating characteristic

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