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Mohammad Mahbub Ahsan
Junior Consultant (Medicine), Manikgonj Medical College Hospital, Manikgonj, Bangladesh

Md. Lokman Hossain Talukder
Assistant Professor (Medicine), Shaheed Suhrawardy Medical College, Dhaka, Bangladesh

Mohammad Niamul Kabir Khan Siddique
Assistant Professor, (Medicine), Anwer Khan Modern Medical College Hospital, Dhanmondi, Dhaka, Bangladesh

Badal Kumar Saha
Junior Consultant (Medicine), Upazilla Health Complex, Rupganj, Narayanganj, Bangladesh

Mohammad Fazlul Huq
Assistant Professor (Medicine), Faridpur Medical College, Faridpur, Bangladesh

Monira Akter
Assistant Professor, (Radiology and Imaging), Jilla Sadar Hospital, Netrokona, Bangladesh

Mussamat Rumin Tamanna
Medical Officer (Obs & Gynae), Kurmitola General Hospital, Dhaka, Bangladesh

Corresponding Author:
Mohammad Mahbub Ahsan
Junior Consultant (Medicine), Manikgonj Medical College Hospital, Manikgonj, Bangladesh

Study of inflammatory markers after acute ischemic stroke: A cross-sectional study

Mohammad Mahbub Ahsan, Md. Lokman Hossain Talukder, Mohammad Niamul Kabir Khan Siddique, Badal Kumar Saha, Mohammad Fazlul Huq, Monira Akter and Mussamat Rumin Tamanna

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Abstract
Background: Stroke remains a leading cause of mortality and long-term disability worldwide, with ischemic stroke accounting for the majority of cases. Increasing evidence suggests that inflammation plays a pivotal role in the pathogenesis and prognosis of acute ischemic stroke. Inflammatory biomarkers such as C-reactive protein (CRP), leukocyte count and erythrocyte sedimentation rate (ESR) have been linked to both the severity and outcome of ischemic injury.
Objective: To assess the levels of inflammatory markers (CRP, leukocyte count, and ESR) in patients with acute ischemic stroke and to determine their association with stroke subtype and clinical outcome.
Methods: This cross-sectional study was conducted at the Department of Medicine, Shaheed Suhrawardi Medical College & Hospital, Dhaka, over six months (December 2010–May 2011). Fifty patients aged 18 years and above with confirmed acute ischemic stroke (by CT and/or MRI within seven days of onset) were enrolled. Patients with hemorrhagic stroke or any known infective/inflammatory condition were excluded. Data on demographic, behavioral, and clinical characteristics were collected. Serum CRP, leukocyte count, and ESR were measured to evaluate the inflammatory response. Data were analyzed using SPSS version 11.5, applying descriptive statistics, Chi-square tests, ANOVA, and unpaired t-tests where appropriate, with $p < 0.05$ considered statistically significant.
Results: The mean age of the patients was 65.6 ± 10.7 years; 68% were male and 48% were smokers. Hypertension (92%) and diabetes (46%) were the most common comorbidities. Mean CRP and leukocyte counts were significantly higher in patients who died compared to survivors (CRP: 9.8 ± 2.1 vs. 8.7 ± 1.3 mg/L, p = 0.045; leukocyte count: 18.0 ± 0.1×10⁹ vs. 11.4 ± 3.4×10⁹/µL, p = 0.011). ESR was slightly lower among non-survivors (20.0 ± 0.2 vs. 24.5 ± 2.5 mm/hr, p = 0.009). Patients with cerebral infarction had significantly higher leukocyte counts compared to those with cerebellar or lacunar infarction (p = 0.039).
Conclusion: Elevated inflammatory markers, particularly CRP and leukocyte count, are associated with poor outcomes in acute ischemic stroke, highlighting their potential prognostic significance in early disease evaluation and management.

Keywords: Acute Ischemic Stroke, Inflammatory Markers, C-Reactive Protein (CRP), Erythrocyte Sedimentation Rate (ESR), Leukocyte Count, Cerebral Infarction, Prognosis

Introduction
Stroke continues to have devastating impact on public health and remains the third leading cause of death worldwide. The disease has also turned out to be the most common cause of disability and dependence with majority of stroke survivors remaining vocationally impaired and requiring assistance for daily living activities. The loss of life and morbidity not only cause significant social deprivation but also lead to direct and indirect financial burdens [1]. Majority of the patients presenting with stroke will have sustained a cerebral infarction due to inadequate blood flow to part of the brain. Major causes for these reasons are intracranial thrombosis, extracranial embolism, lacunar infarction, systemic hypoperfusion, venous sinus thrombosis, vasospasm etc. As a result, the affected area of brain is unable to function leading to inability to move one or more limbs on one side of the body, inability to understand or formulate speech or an inability to see one side of the visual field [2].

Inadequate blood flow in a single brain artery can often be compensated for by an efficient collateral system, particularly between carotid and vertebral arteries via anastomoses at the circle of Willis and to a lesser extent between major arteries supplying the cerebral hemispheres. However normal variation in the circle of Willis and the calibre of various collateral vessels, atherosclerosis and other acquired arterial lesions can interfere with collateral flow, increasing the chance that blockage of artery will cause brain ischemia [3]. Focal cerebral infarction occurs via two distinct pathways; a necrotic pathway in which cellular cytoskeletal breakdown is rapid due principally to energy failure of the cell and the other one is an apoptotic pathway in which cells become programmed to die [4]. The role of Inflammation in acute ischemic stroke is considered of utmost importance. In healthy brain tissue inflammatory mediators are expressed in low levels. Cerebral ischemia induces an inflammatory response associated with the activation of release of various pro-inflammatory cytokines which are up-regulated in the brain after acute ischemic stroke and are expressed not only by cells of the immune system but also by the resident brain cells including gila and neurons and also release of additional acute phase reactants that aggravate tissue damage [5]. Inflammatory markers like CRP (C reactive protein) participate in multiple mechanism promoting a decrement in the survival of neurons subjected to ischemia. These include an intracerebral influx of leukocytes, the propagation of an intra-vascular thrombus and a reduction in blood flow as well as formation of odema in the perilesional area [6]. The damage that occurs in the early hours after a blood clot has lodged in the brain can be blocked by an enzyme on the surface of white blood cells. These white blood cells are normally protective during emergencies such as cuts and infections but their accumulation on the brain in stroke can be devastating. There is growing evidence that CRP (C-reactive protein) a peripheral marker of inflammation is also a marker of generalized atherosclerosis. This relationship between inflammation and atherosclerosis make CRP (C reactive protein), a potential marker for prognosis after vascular events and a potential predictor of future vascular events [7]. A number of authors have indicated that acute phase response is involved in ischemic brain damage (mechanisms including inflammation and activation of coagulation system). However, one study has demonstrated that higher ESR values observed in patients within 72 hours of ischemic stroke were associated with larger brain infarcts.

Materials & Methods

Study design

This was a cross-sectional study.

Place and period of study

The study was carried out in the Department of Medicine, Shaheed Suhrawardi Medical College & Hospital, Dhaka, over a six-month period from December 2010 to May 2011.

Study population

All adult patients admitted with acute ischemic stroke confirmed by CT or MRI within seven days of onset were included.

Inclusion criteria

- Adults aged 18 years or older, irrespective of sex
- Clinically and radiologically confirmed cases of acute ischemic stroke

Exclusion criteria

- Hemorrhagic stroke
- Patients with other known infective or inflammatory diseases
- Patients who refused to provide informed consent

Sample size and sampling procedure:

The sample size was calculated using the formula: $n = (Z^2 \times p \times q) / d^2$
where Z = 1.96, p = 0.52 (prevalence of stroke in Bangladesh), q = (1 – p) = 0.48, and d = 0.104 (20% of p). The estimated sample size was 69, but due to time constraints, 50 patients were enrolled consecutively.

Variables studied: Demographic variables included age and sex; behavioral variable was smoking. Clinical variables included diabetes, hypertension, ischemic heart disease, hyperlipidemia, and previous stroke/TIA. Inflammatory markers measured were serum CRP, leukocyte count, and ESR. CT/MRI findings were recorded to determine lesion location, and clinical outcome (death or survival) was noted.

Data collection procedure: Data were obtained through structured interviews of attendants, clinical examination, and laboratory investigations.

Data processing and statistical analysis: Data were processed and analysed using software SPSS (Statistical Package for Social Sciences) version 11.5. The test statistics used to analyse the data were descriptive statistics and Chi-square (χ^2) tests**.** For comparison of data presented on continuous scale unpaired t-Test was done. For all analytical tests, the level of significance was set at 0.05 and $p < 0.05$ was considered significant.

Results

A total of 50 patients of acute ischemic stroke confirmed by computed tomography and/or magnetic resonance imaging of brain admitted in the Medicine Department of Shaheed Suhrawardi Medical College and Hospital were included in the study to assess the inflammatory response after acute ischemic stroke. The findings of the study derived from data analysis are presented below.

Age distribution

Table 1 shows that 20% of the patients was below 60 years of age, 38% between 60 – 70 years and rest 42% 70 years or more than 70 years old. The mean age of the patients was 65.6 ± 10.7 years and the lowest and highest ages were 42 and 90 years respectively.

Table 1: Distribution of patients by age (n = 50)

Age (yrs)*	Frequency	Percentage
< 60	10	20.0
60 – 70	19	38.0
≥ 70	21	42.0

* Mean age = (65.6 ± 10.7) years; range = (42 – 90) years.

Sex distribution

Sex distribution of the patients shows that out of 50 patients 34(68%) were male and 16(32%) were female giving a male to female ratio of 2:1.

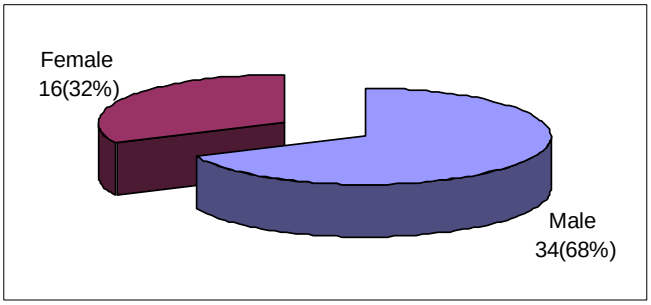

Fig 1: Distribution of patients by sex (n = 50)

Smoking habit

Figure 2 shows that nearly half (48%) of the patients was smoker and the remaining 52% non-smoker.

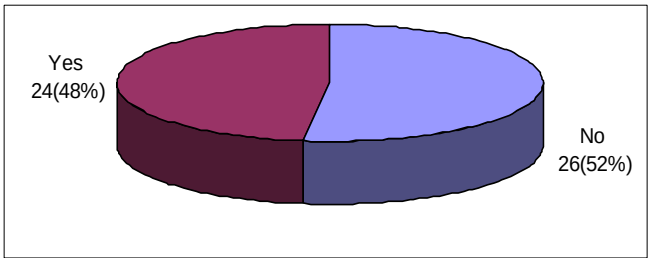

Fig 2: Distribution of patients by smoking habit (n = 50)

Clinical findings:

Clinical findings demonstrate that majority (92%) of the patients had hypertension. Nearly half (46%) of the patients had diabetes followed by 36% hyperlipidemia, 24% ischemic heart disease and only 4% had prior history of stroke/TIA (Table 2).

Table 2: Distribution of patients by clinical findings (n = 50)

Clinical findings	Frequency	Percentage
Hypertension	46	92.0
Diabetes	23	46.0
Ischemic heart disease	12	24.0
Hyperlipidemia	18	36.0
Prior stroke/TIA	02	4.0

Association between inflammatory markers and type of stroke

Table 3 shows the distribution of inflammatory markers among different kinds of stroke. The mean CRP was considerably higher in lacunar stroke patients than those in cerebellar and cerebral infarction patients (p = 0.074). However, the mean leukocyte count was found significantly higher in patients with cerebral infarction than those in patients with cerebellar infarction and lacunar stroke (p = 0.039). The level of ESR was also much higher in the cerebral infarction than that found in two other types of stroke (p = 0.087).

Table 3: Association between inflammatory markers and type of stroke

Inflammatory markers	Type of stroke			p-value
	Cerebellar infarction (n = 4)	Cerebral infarction (n = 35)	Lacunar Stroke (n = 11)	
CRP level (mg/L)	10.1 ± 1.6	9.8 ± 0.9	11.0 ± 2.1	0.074
Leukocyte count (/microL)	$9.9 \pm 3.8 \times 10^9$	$12.5 \pm 3.6 \times 10^9$	$9.6 \pm 2.4 \times 10^9$	0.039
ESR (mm/hrs)	12.5 ± 2.8	25.6 ± 11.7	24.4 ± 9.5	0.087

Data were analysed using ANOVA and presented as mean ± SD.

Association between inflammatory markers and outcome

Association between outcome and inflammatory markers demonstrates that the mean CRP level and leukocyte were significantly higher in patients who died compared to those who survived of the disease (9.8 ± 2.1 vs. 8.7 ± 1.3 mg/L, p = 0.045 and $18.0 \pm 0.1 \times 10^9$ vs. $11.4 \pm 3.4 \times 10^9$ /microL, p = 0.001 respectively). However,the mean ESR was significantly lower in the former group than that in the latter group (20.0 ± 0.2 vs. 24.5 ± 2.5 mm/hrs, p = 0.009) (Table 4).

Table 4: Association between inflammatory markers and outcome

Inflammatory markers	Outcome		p-value#
	Died (n = 2)	Alive (n = 48)	
CRP level (mg/L)	9.8 ± 2.1	8.7 ± 1.3	0.045
Leukocyte count (/microL)	$18.0 \pm 0.1 \times 10^9$	$11.4 \pm 3.4 \times 10^9$	0.011
ESR (mm/hrs)	20.0 ± 0.2	24.5 ± 2.5	0.009

Data were analysed using Student's t-Test and were presented as mean ± SD.

Association between type of stroke and outcome

Table 5 shows that 2(100%) patients died of the disease were suffering from cerebral infarction. Out of 48 alive patients, 4(8.3%) had a history of cerebellar infarction, 33(68.8%) cerebral infarction and 11(22.9%) lacunar type of ischemic stroke. There was no significant difference between types of stroke and outcome as evident by p = 0.640.

Table 5: Association between type of stroke and outcome

Type of stroke	Outcome		p-value#
	Died (n = 2)	Alive (n = 48)	
Cerebellar infarction	00	4(8.3)	
Cerebral infarction	2(100.0)	33(68.8)	0.640
Lacunar	00	11(22.9)	

#Data were analysed using Chi-square (χ^2) Test.

Discussion

Stroke is the third leading cause of death and the most frequent cause of permanent disability worldwide. Inflammation appears to play an important role in the pathogenesis of ischemic stroke [8]. There is much evidence to suggest that neuro-inflammatory mechanisms play an important role in ischemic injury, and that interruption of these processes can result in improved neurological outcomes [9]. In the present study mean age of our patients was 65.6 ± 10.7 years with majority (80%) of the patients being 60 or more than 60 years old meaning that the stroke is primarily a disease of old age. A male preponderance was observed with almost half (48%) of the patients being smoker. Hypertension was identified as the predominant co-morbidity (92%) and over 46% of patients exhibited diabetes. Hyperlipidemia and ischemic heart disease were found in 36% and 24% of the cases respectively. In a Turkish study, Bircan *et al.* [10] reported a much lower mean age (47.2 years) indicating that the Turkish population acquire the disease at middle age; however, they also observed a male predominance (75.3%) in

their study. In another study Yudkin and associates [11] found hypertension to be the common comorbidity which goes in favour of our study. In a survey of Iranian population conducted by Ahmad *et al.* [12] hypertension and diabetes mellitus were more frequent occurrence (64% and 36% respectively) than the average global findings. They found a male to female ratio of almost 1: 1, which contrasts with the findings of the present study. The mean age was 68 ± 13.8 years which is quite consistent with our findings. Hypertension was found in 64% of patients, followed by diabetes mellitus (36%), heart disease (34%). In this study mean CRP was higher in lacunar stroke patients than those in cerebellar and cerebral infarction stroke patients. However, mean leukocyte count and ESR were found higher in patients with cerebral infarction than those in patients with cerebellar infarction and lacunar stroke and these were in accordance with the findings of some other previous studies [13, 14]. On the other hand, DiNapoli *et al.* [15] in an attempt to find the relationship between inflammatory markers and different kinds of ischemic stroke, concluded that the CRP level, leukocyte count and ESR were significantly higher in cerebral infarction patients compared to two other types of ischemic stroke. Contrary to these findings, Sesso *et al.* [16] reported that the average CRP and ESR both were significantly higher in cerebellar infarction patients. This study revealed that mean CRP level and leukocyte were significantly higher in patients who died of the disease compared to those who escaped. However, the mean ESR was significantly higher in patients who remained alive than those who died of the disease. Emsley & Hopkins [17] in a recent study reported that mean levels of CRP (11.2 ± 1.4 mg/L), leukocyte count (19.8 ± 9.5 x 10(3)/microL) and ESR (27.2 ± 6.8 mm/hour) all were statistically significantly higher in patients who died than the mean levels of CRP (9.2 ± 2.8 mg/dL), leukocyte count (14.6 ± 5.4 x 10(9)/microL) and ESR (23.1 ± 5.9 mm/hour) of those who remained alive ($p = 0.000$, $p = 0.001$, $p = 0.012$, respectively). But very few studies [18, 19] found association of inflammatory markers with types of ischemic stroke. In our study, two patients died who were suffering from cerebral infarction. Out of 48 patients survived of the disease, 4(8.3%) had a history of cerebellar infarction, 33(68.8%) cerebral infarction and 11(22.9%) lacunar type of ischemic stroke. These data were in agreement with the findings of other investigators [20]. Moreover, Mizrahi *et al.* [21] results reached a significant value as regards to stroke severity. Thus, the findings of the study suggest that inflammatory markers like CRP, leucocyte count and ESR all increase in ischemic stroke. However, the increases are significantly higher in the cerebral infarction than those observed in cerebellar and lacunar type of ischemic stroke. Although these findings are consistent with most of the study findings conducted around the world, some investigators reported that inflammatory markers are more pronounced in the cerebellar type of stroke than the cerebral and lacunar types.

Conclusion

The inflammatory response after ischemic stroke involves the changes in several inflammatory markers, all connected with each other and connected to other pathways of the ischemic cascade. The level of CRP, leukocyte count and ESR all increase in ischemic stroke. However, increase in leukocyte count is significantly pronounced in cerebral infarction than those observed in cerebellar and lucunar type of ischemic stroke, while CRP and ESR tend to increase with lacunar infarction than with cerebral and cerebellar infarction. The detection of inflammatory markers especially CRP and leukocyte count are very important in predicting the fate of patients with ischemic stroke as higher CRP and leukocytes count are associated with increased in-hospital mortality.

Recommendations

From the findings of the study, the following recommendations are put forward:

1. As the serum levels of inflammatory markers like CRP, leukocyte count and ESR increase differently according to site ischemic stroke, they should be investigated to predict the location of ischemic stroke.
2. If ischemic stroke patients are admitted in the hospital, inflammatory markers like CRP, leukocyte count and ESR should be done to predict the fate of the patients.

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Author's Contribution

Not available

Conflict of Interest

Not available

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