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Research Article

# Prognosis of acute stroke patients monitored in neurological intensive care unit

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## Abstract

**Introduction:** Stroke is the second most common cause of death in the world. Identifying the factors that influence stroke prognosis in advance is important to take the necessary precautions and improve the patient's chances of survival.

**Aim:** We investigated the clinical and laboratory parameters that predict mortality and prognosis in patients with acute stroke in a neurological intensive care unit (ICU).

**Materials and methods:** A total of 219 adult acute stroke patients who were admitted to the neurological ICU over two years were included in the study. Each patient's coma score was determined using the Glasgow Coma Scale (GCS) from day one to day 12. The patients' clinical parameters, laboratory and brain scans results were recorded. On day 12, the characteristics of patients still alive and those who had died by that time were compared.

**Results:** One hundred and forty-three of the patients died within 12 days of the stroke. In patients who died, fasting blood glucose, hemoglobin, blood urea, blood creatinine and triglyceride levels measured on the first day were higher than in patients who survived ($p<0.05$) and GCS calculated from the moment of hospitalization to day 11 was significantly lower ($p<0.05$). There was a negative correlation between GCS and laboratory parameters including fasting blood glucose, blood urea, triglycerides, leukocytes, and fibrinogen.

**Conclusion:** We found a 65% mortality rate in patients with acute stroke. We demonstrated that the severity of the initial neurological condition is one of the most important poor prognostic factors, and abnormal laboratory parameters should be carefully monitored in patients because of their negative impact on short-term stroke prognosis.

## Keywords

Glasgow coma score, intensive care, mortality, prognoses, stroke

## Introduction

Despite advances in treatment, stroke remains the third leading cause of disability and the second leading cause of death worldwide.[1] A variety of factors influence stroke prognosis, including the mechanism of stroke, the location of the lesion on imaging at presentation, comorbid conditions, epidemiologic factors, clinical findings, and stroke-related complications, especially advanced age and severity of neurological status in the acute phase.[2]

Hemorrhagic stroke has been associated with a higher rate of morbidity and mortality than ischemic stroke.[2-5] Reports from different countries have shown that the mortality rate in the first 30 days after stroke is 16%–23% for ischemic stroke[6,7] and 32%–52% for hemorrhagic stroke[8-10]. Half of the deaths due to cerebral hemorrhage

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occur within the first two days[8], while deaths due to cerebral infarction occur mostly between the third and sixth days.

## Aim

Identifying the factors that influence stroke prognosis in advance is important to take the necessary precautions and improve the patient's chances of survival. In this study, we investigated the clinical and laboratory parameters that predict mortality and prognosis in patients with acute stroke in the neurological intensive care unit (ICU).

## Materials and methods

A total of 219 adult (≥50 years) acute stroke patients (78 men and 141 women) who were admitted to the neurological intensive care unit (ICU) over two years were included in the study. From day one to day 12, each patient's coma score was determined using the Glasgow Coma Score (GCS). At 12 days, the characteristics of the patients who were still alive were compared with those of the patients who had died by that time. The type of lesion was categorized into ischemic and hemorrhagic according to the results of the brain tomography scan at the time of admission to the hospital. Ischemic stroke was categorized according to the Trial of Org 10172 in Acute Stroke Treatment (TOAST) classification as large artery atherosclerosis, cardioembolism, small vessel occlusion, undetermined, and other known causes.[11] Vascular imaging studies used for etiologic classification consisted of carotid Doppler ultrasound or computed tomography (CT) angiography. The localization of the hemorrhage was divided into putaminal, thalamic, cerebellar, pontine, and lobar groups.

Risk factors for stroke (e.g., hypertension, diabetes mellitus, heart disease, hyperlipidemia, smoking, and alcohol consumption) were recorded. Patients with a history of heart disease were evaluated with 24-hour cardiac rhythm monitoring and transthoracic echocardiography. To investigate the relationship with prognosis, venous blood samples were taken early in the morning after 12 hours of fasting, and fasting blood glucose, leukocyte, platelet, hemoglobin, erythrocyte sedimentation rate (ESR), blood urea, blood creatinine, total cholesterol, triglyceride levels were studied three times every other day. The date and cause of death were recorded. Exclusion criteria were venous infarcts secondary to dural sinus thrombosis, border-zone infarcts, subarachnoid hemorrhages, cancer or autoimmune diseases, infections prior to hospitalization, previous stroke or other comorbid conditions (severe liver and kidney diseases) that could cause disability.

Our study was registered at the local Scientific Research Ethics Committee with decision number 09/28, and written informed consent was obtained from all participants before inclusion in the study, which was conducted in accordance with the Declaration of Helsinki.

### Statistical analysis

Results were expressed as mean ± standard deviation or number (%). The Shapiro-Wilk test was used to examine the conformity of quantitative data to normal distribution. Student t test was used to compare variables with normal distribution between groups (survived vs. dead, female vs. male, ischemic stroke vs. hemorrhagic stroke), and Mann-Whitney U test was used to compare variables with non-normal distribution. Chi-square test was used to compare categorical data between groups. Relationships between quantitative variables were examined with Pearson correlation analysis. $p<0.05$ was accepted as the statistical significance limit value. SPSS 20.0 (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.) package program was used to analyze the data.

## Results

There was no difference in age between the 78 male (mean age 65.23±12.51 years) and 141 female (mean age 67.01±11.78 years) acute stroke patients included in the study. While 76 (35%) patients were alive on day 12, 143 (65%) died within 12 days of stroke. Seventy-five (52%) of the patients who died were hemorrhagic stroke patients with a mean survival of 7.62 days. The remaining 68 (48%) ischemic stroke patients had a mean survival of 7.74 days, which was not significantly different from the hemorrhagic stroke patients.

Thirty-nine (18%) patients could not be classified because lesion location could not be clarified on acute phase CT. Of the lesions whose location could be determined, 86.7% were supratentorial and 13.3% were infratentorial. The basal ganglia was the most common site of location in fifty-six patients (25.6%). This was followed by parietal (43 patients) and thalamic (39 patients) location. Other regions are shown in **Fig. 1**.

On the CT performed within the first 24 hours, 81 patients (37%) had diffuse cortical atrophy and 124 patients (57%) had cerebral edema.

Demographic and clinical characteristics of the patients are shown in **Table 1**.

The mean age of patients who died in the first 12 days (65.67±11.58 years) was not statistically different from the mean age of patients who survived (65.84±12.44 years) ($p=0.623$). Fasting blood glucose, hemoglobin, blood urea, blood creatinine, and triglyceride levels measured on the first day were found to be higher in patients who died than in those who survived ($p=0.040$, $p=0.043$, $p=0.012$, $p=0.006$, $p=0.049$, respectively). Leukocyte, platelet, and cholesterol levels were similar between the two groups. No difference was observed between the groups of patients who died and those who survived with regard to the ar-

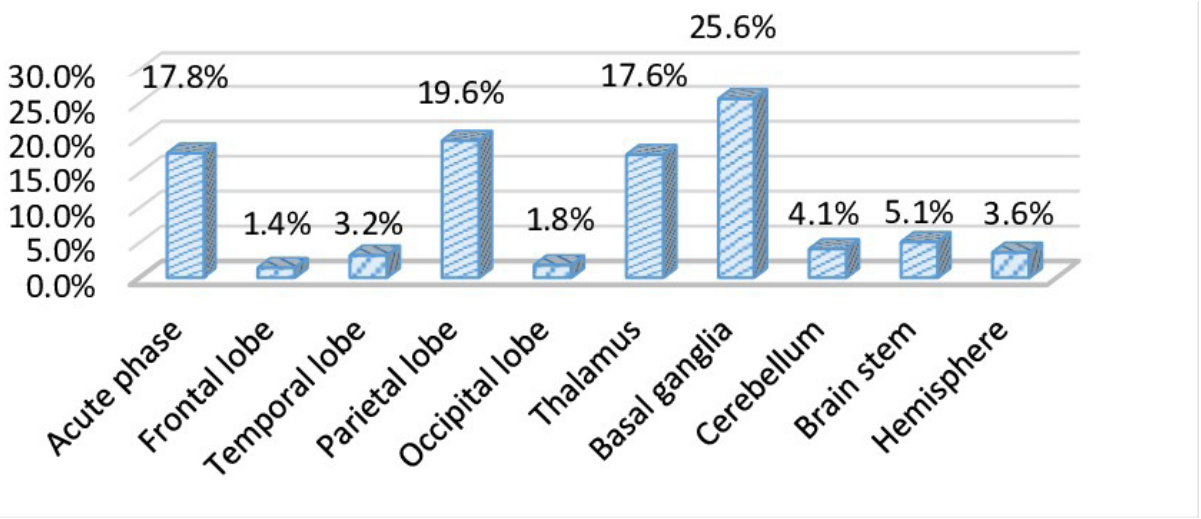


**Figure 1.** Lesion distribution.

**Table 1.** Demographic and clinical characteristics of patients

| | n=219 | % |
|---|---|---|
| Age (year) | 64.62±14.05 | - |
| Sex | | |
| Female | 141 | 64.4 |
| Men | 78 | 35.6 |
| Heart disease | | |
| Yes | 104 | 47.5 |
| No | 115 | 52.5 |
| Diabetes mellitus | | |
| Yes | 32 | 14.6 |
| No | 187 | 85.4 |
| Hypertension | | |
| Yes | 157 | 71.7 |
| No | 62 | 28.3 |
| Hyperlipidemia | | |
| Yes | 106 | 48.4 |
| No | 113 | 51.6 |
| Smoking | | |
| Yes | 121 | 55.3 |
| No | 98 | 44.7 |
| Alcohol consumption | | |
| Yes | 39 | 17.8 |
| No | 180 | 82.2 |
| Stroke type | | |
| Ischemic | 107 | 48.9 |
| Hemorrhagic | 112 | 51.1 |
| Classification for ischemic stroke (TOAST) | | |
| Large artery atherosclerosis | 26 | 24.3 |
| Cardioembolism | 64 | 59.8 |
| Small vessel occlusion | 10 | 9.3 |



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| | | |
|---|---|---|
| Other determined causes | 4 | %3.7 |
| Undetermined causes | 3 | %2.8 |
| Classification of hemorrhagic stroke | | |
| Basal ganglia | 54 | %48.2 |
| Thalamic | 29 | %25.8 |
| Cerebellum | 7 | %6.3 |
| Brain stem | 7 | %6.3 |
| Lobar | 15 | %13.4 |
| Cerebral edema | | |
| Yes | 124 | %56.6 |
| No | 95 | %43.4 |
| Cortical Atrophy | | |
| Yes | 81 | %37.0 |
| No | 138 | %63.0 |

terial blood pressure measured on the first day. When we looked at GCSs, we found that coma scores at the time of ICU admission were significantly lower in patients who died than in those who survived ($p$=0.014). The difference continued with similar significance between days 1 and 11. The mean length of hospital stay of the patients who died was significantly shorter than that of the patients who survived ($p$<0.001) **(Table 2)**.

We divided the patients monitored in the ICU into two groups according to their sex and compared their laboratory data. While there was no significant difference in ESR between the two groups on day 1, it was significantly higher in male patients than in female patients on day 3 of stroke ($p$=0.009). While fibrinogen, hematocrit, and blood urea levels were higher in male patients ($p$=0.023, $p$=0.000, $p$=0.004, respectively), platelet and cholesterol levels were higher in female patients ($p$=0.039, $p$=0.023, respectively) **(Table 3)**.

When comparing laboratory parameters with stroke types, fibrinogen levels were found to be lower in the hemorrhagic patient group than in the ischemic patients ($p$=0.047). Total protein was lower in ischemic than hemorrhagic groups ($p$=0.006). Similarly, albumin levels were significantly lower in patients with ischemic lesions than in patients with hemorrhagic lesions ($p$=0.001). When we evaluated the clinical characteristics, we found that the mean age of patients with ischemic lesions (69.52±10.36 years) was significantly higher than that of patients with hemorrhagic stroke (63.38±12.54 years) ($p$=0.000). While GCSs did not differ between the two groups during the first 5 days, they were significantly lower in patients with ischemic stroke on day 6 ($p$=0.031). The difference in GCSs remained similar on days 9 and 11 ($p$=0.035, $p$=0.017, $p$=0.021) **(Table 4)**.

When we examined the relationship between GCS and laboratory parameters in patients monitored in the ICU, there was an inverse relationship between fasting blood glucose measured on day one and GCS detected on day one ($r$=−189.05, $p$=0.031). The significant relationship between blood glucose and GCS remained similar until day 9 ($r$=−205.051, $p$=0.022). A positive correlation was found between the serum albumin level and the GCS on day 11 ($r$=0.305, $p$=0.044). A significant negative correlation between ESR and GCS was observed from day one ($r$=−0.278, $p$=0.012). A significant inverse relationship was found between the leukocyte and TG levels obtained on day one and the calculated GCS ($r$=−0.261, $p$=0.004; $r$=−0.211, $p$=0.004, respectively). A significant negative correlation was found between GCS and blood urea and fibrinogen levels from day 4 ($r$=−0.183, $p$=0.013; $r$=−0.372, $p$=0.013, respectively). Hematocrit, platelet count, blood pressure and age of the patients did not show any relationship with GCS ($p$>0.05) **(Table 5)**.

Neurological and systemic complications observed in our patients are shown in **Figs 2** and **3**. The most common neurological complication was brain herniation. Aspiration was identified as the most common systemic complication.

## Discussion

Stroke risk factors have been studied in detail in many studies because of their importance in stroke prevention. Despite treatments aimed at modifying or eliminating modifiable factors in light of these studies, stroke cannot be prevented in most cases, which has led researchers to investigate and evaluate prognostic factors after stroke. The first studies date back more than 65 years.[12] While patients with transient ischemic attacks and minor strokes generally have a good prognosis, mortality rates of up to 80% have been reported in patients with poor initial neurological status who require intensive care monitoring.[13] In our study, the 12-day mortality of patients we followed with an acute stroke diagnosis in the neurological ICU was 65%.

The strongest prognostic factors have been reported to be initial stroke severity and patient age. Studies have

**Table 2.** Comparison of laboratory and clinical parameters between the groups of patients who died and those who survived

| | n | Mean ± SD | *p* |
|---|---|---|---|
| Age (year) | | | |
| Survived | 76 | 65.84±12.44 | 0.623 |
| Died | 143 | 65.67±11.58 | |
| Length of hospital stay (day) | | | |
| Survived | 76 | 14.52±7.21 | <0.001 |
| Died | 143 | 7.68±5.23 | |
| Fasting blood glucose (mg/dL) | | | |
| Survived | 68 | 174.87±100.46 | 0.040 |
| Died | 84 | 211.63±115.45 | |
| ESR (mm/h) | | | |
| Survived | 42 | 25.38±15.35 | 0.056 |
| Died | 54 | 32.48±19.52 | |
| Hemoglobin (g/dl) | | | |
| Survived | 69 | 14.39±2.13 | 0.043 |
| Died | 82 | 15.13±2.31 | |
| Creatinine (mg/dL) | | | |
| Survived | 76 | 91.09±29.04 | 0.006 |
| Died | 138 | 117.29±79.58 | |
| Blood Urea (mg/dL) | | | |
| Survived | 76 | 38.25±14.11 | 0.012 |
| Died | 143 | 45.66±23.27 | |
| Leukocyte (/mL) | | | |
| Survived | 37 | 11470.21±3712.02 | 0.072 |
| Died | 80 | 13002.41±4482.56 | |
| Thrombocyte (/mL) | | | |
| Survived | 75 | 246430.54±90700.02 | 0.412 |
| Died | 139 | 256320.41±80140.22 | |
| Triglyceride (mg/dL) | | | |
| Survived | 52 | 96.59±55.98 | 0.049 |
| Died | 54 | 117.94±54.32 | |
| Cholesterol (mg/dL) | | | |
| Survived | 52 | 214.44±51.33 | 0.437 |
| Died | 54 | 222.31±52.50 | |
| Systolic BP (mm/Hg) | | | |
| Survived | 76 | 171.51±31.33 | 0.087 |
| Died | 143 | 180.94±40.78 | |
| Diastolic BP (mm/Hg) | | | |
| Survived | 76 | 96.83 ±18.73 | 0.224 |
| Died | 143 | 100.53±22.72 | |
| GCS at admission | | | |
| Survived | 76 | 11.3±2.7 | 0.014 |
| Died | 143 | 6.3±0.1 | |
| GCS Day 1 | | | |
| Survived | 76 | 8.5±2.6 | 0.000 |
| Died | 143 | 6.3±2.3 | |



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| | | | |
|---|---|---|---|
| GCS Day 11 | | | |
| Survived | 34 | 10.3±2.4 | 0.000 |
| Died | 30 | 7.2±2.5 | |

GCS: Glasgow coma score; BP: blood pressure; ESR: Erythrocyte sedimentation rate

**Table 3.** Comparison of laboratory parameters between male and female patient groups

| | **Female n= 141** | **Male n= 78** | ***p*** |
|---|---|---|---|
| Age (year) | 67.01±12.52 | 65.23±11.78 | NS |
| GCS at admission | 8.9±2.3 | 8.7±2.1 | NS |
| ESR | | | |
| Day 1 | 17.82±13.21 | 19.21±14,42 | NS |
| Day 3 | 31.91±21.18 | 56.18±54.45 | 0.009 |
| Fibrinogen (g/L) | 3.63±1.62 | 4.9±1.72 | 0.023 |
| Hematocrit (%) | 40.78±6.58 | 44.81±5.99 | 0.000 |
| Platelets (/mL) | 261630.24±92640.12 | 236921.23±62631.42 | 0.039 |
| Cholesterol (mg/dL) | 225.96±51.93 | 201.09±49.18 | 0.023 |
| Blood urea (mg/dL) | 61.14±29.22 | 78.02±36.64 | 0.004 |

GCS: Glasgow coma score; ESR: Erythrocyte sedimentation rate; NS: no significance

**Table 4.** Comparison of clinical and laboratory parameters between ischemic and hemorrhagic stroke patient groups

| | **Ischemic stroke n = 107** | **Hemorrhagic stroke n = 112** | ***p*** |
|---|---|---|---|
| Age (year) | 69.52±10.36 | 63.38±12.54 | 0.000 |
| Length of hospital stay (day) | 7.74±4.32 | 7.62±3.78 | NS |
| Fibrinogen (g/L) | 4.02±1.34 | 4.33±1.85 | 0.047 |
| Total protein (g/L) | 70.20±7.61 | 74.75±8.62 | 0.006 |
| Albumin (g/L) | 39.52±5.30 | 43.37±6.47 | 0.001 |
| GCS | | | |
| Day 1 | 8.9±3.4 | 9.1±2.3 | NS |
| Day 6 | 7.7±2.9 | 8.9±3.3 | 0.031 |
| Day 9 | 7.4±1.5 | 8.0±2.2 | 0.017 |
| Day 11 | 7.3±4.4 | 8.4±3.4 | 0.021 |

GCS: Glasgow coma score; NS: no significance

**Table 5.** Relationship between Glasgow Coma Score and laboratory parameters (r)

| | **FBG Day 1** | **FBG Day 9** | **Blood Urea** | **Albumin** | **ESR** | **Fibrinogen** | **Leukocytes** | **Triglycerides** |
|---|---|---|---|---|---|---|---|---|
| **GCS (n=219)** | −189.05* | −205.051* | −0.183* | 0.305* | −0.278* | *r*=−0.372* | −0.261* | −0.211* |

FBG: Fasting blood glucose; ESR: Erythrocyte sedimentation rate; * $p < 0.05$

shown that age is an independent risk factor for mortality in both minor and major stroke, and the incidence of poor prognosis increases with age.[2,14-16] This is thought to be due to general deterioration, comorbid conditions (e.g., hypertension, diabetes, hyperlipidemia, heart disease), and the increased likelihood of complications in older stroke patients.[17,18] In our study, we found that the age of the patients did not play a role in the short-term prognosis of stroke ($p$=0.623).

In evaluating the relationship between the initial neuro-

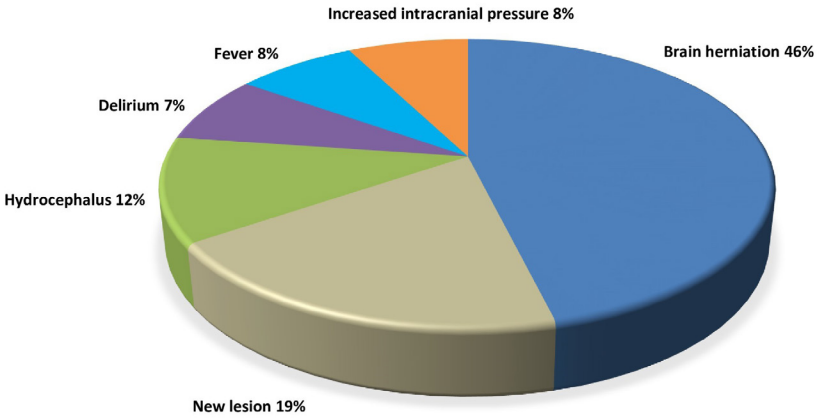


**Figure 2.** Neurological complications.

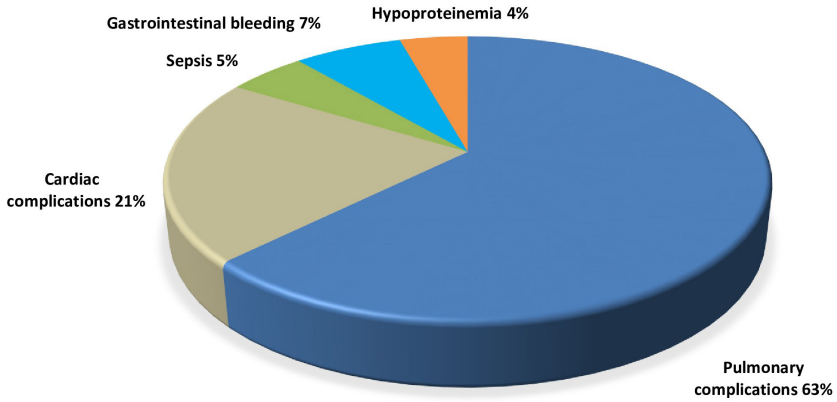


**Figure 3.** Systemic complications.

logical status of our stroke patients and prognosis, we observed that coma scores were low in our patients who died from the first day. Despite studies mentioning the effect of infarct volume on stroke prognosis[19,20], we know that two infarcts of the same size but in different locations may cause very different functional expression. Therefore, we agree that lesion location attenuated the strength of infarct volume as a predictor of stroke prognosis. In 18% of our patients, the lesion could not be classified because its location could not be clarified on brain CT taken during the acute phase. Of the localized lesions, 27% were infratentorial and 55% were supratentorial. The basal ganglia (26%) was the most common location. Studies evaluating the relationship between lesion type and prognosis have reported that hemorrhages carry a higher risk of mortality than cerebral infarctions.[2-4] In the ischemic stroke groups, cardioembolic strokes had a worse prognosis.[21] In our study, coma scores did not differ between hemorrhagic and ischemic stroke groups in the first days, but were significantly lower in ischemic stroke patients after day 6. When we examined the differences in prognostic factors according to stroke type, we found that patients with ischemic lesions had a higher mean age.

Studies on the relationship between sex and stroke prognosis have reached different conclusions. In addition to studies indicating that sex has no effect on clinical course and prognosis, there are also studies finding the mortality rate higher in male or higher in female. This difference was mostly associated with advanced age, stroke severity, time to reach the hospital, the degree of dependence of the person in daily living activities before the stroke, and accompanying comorbid conditions.[22-26] Although we found differences in prognostic parameters such as fibrinogen, platelets, hematocrit, cholesterol, blood urea and ESR between sexes (fibrinogen, hematocrit, blood urea levels, and ESR were higher in male patients than in female patients), we did not find differences in coma scores. In addition, we believe that the similar mortality rates in both sexes may be explained by the fact that our male and female patients did not differ in age.

The relationships between some laboratory parameters and prognosis in acute stroke patients have been reported in studies. Among these parameters, blood glucose has been shown to be one of the most important prognostic



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factors. The association between high fasting blood glucose at the time of hospital admission and poor prognosis in ischemic stroke patients has been demonstrated in many studies, but its effect on early outcome in spontaneous intracranial hemorrhage has been less studied and no similar association has been found.[27] Hyperglycemia (≥110 mg/dL) at the time of hospital admission has been reported in 40%–60% of stroke patients[27,28], due to both pre-existing diabetes/impaired glucose tolerance and the sympathoadrenal stress response[29,30]. It has been shown that in hyperglycemic patients, the infarct area expands due to lactate accumulation[27,30,31] and hemorrhagic transformation occurs more easily due to disruption of the blood-brain barrier[32,33]. It has been reported that the risk of mortality increases threefold in these patients.[27,29] 14.6% of our patients participating in the study were diabetic. The fasting blood glucose levels measured on the first day were significantly higher in patients who died than in those who survived. We observed a significant inverse relationship between GCS and fasting blood glucose in patients who died, which lasted from day one to day 9.

The prognostic value of renal dysfunction has been demonstrated in many stroke studies. High blood urea and creatinine levels have been associated with higher mortality rates in both ischemic and hemorrhagic acute stroke patients and have been reported to be independent predictors of short- and long-term survival after stroke.[34] Although none of our patients had symptomatic renal disease or signs of dehydration, we observed that blood urea and creatinine levels measured on day one were significantly higher in patients who died than in those who survived, and that there was a significant negative correlation between coma score and blood urea from day four onward. In our patients monitored in the ICU, diuretics were used only for heart failure. Therefore, it is unlikely that patients with elevated blood urea and creatinine levels received treatment that increased their mortality. We thought that elevated blood urea and creatinine might reflect widespread vascular atherosclerotic damage.

The relationship between stroke and serum lipid levels is still unclear. Some studies have reported that high triglyceride levels are associated with a similar risk for ischemic heart disease as well as ischemic stroke[35-37], while others have reported that low triglyceride levels worsen the clinical course of acute ischemic stroke and increase in-hospital mortality[38]. There have also been studies that have found no association.[39] An inverse relationship is mentioned between triglyceride levels and the incidence of cerebral hemorrhage.[40] Hypotriglyceridemia has been shown to increase the risk of hemorrhagic stroke and worsen neurological status.[40,41] Hyperlipidemia has been reported to protect against cerebral hemorrhage. Serum lipids are thought to protect against rupture by ensuring vascular strength and integrity and may be important and necessary for intravascular fluidity.[42] In our study, higher triglyceride levels were observed in patients who died, and there was a significant inverse relationship between the triglyceride levels obtained on the first day and the calculated GCS.

Neuroinflammation is a critical process that begins after acute ischemic and hemorrhagic stroke. In patients with severe stroke, high leukocyte counts have been shown in the first 72 hours.[43,44] In ischemic stroke, neuroinflammation begins within minutes of the onset of ischemia and continues for several days, whereas in hemorrhagic stroke, it is initiated by blood byproducts that rapidly accumulate in brain tissue. In both cases, neuroinflammation is characterized by activation of resident immune cells such as microglia and astrocytes and infiltration of peripheral immune cells, leading to the release of proinflammatory cytokines, chemokines, and reactive oxygen species. These inflammatory mediators lead to disruption of the blood-brain barrier, increased neuronal damage, and the development of cerebral edema, which in turn increases neuronal apoptosis, impairs neuroplasticity, and ultimately worsens the neurological status.[45,46] We think this would be the most plausible explanation for our findings showing an association between higher white blood cells and worse outcomes after acute stroke. We did not find a significant difference between the leukocyte counts of patients who died and those who survived, but we did find a significant inverse relationship between the leukocyte counts obtained in the first days with GCS. There was a correlation between the neurological deterioration of the patients and their leukocyte levels.

Fibrinogen is a plasma glycoprotein that has the greatest effect on erythrocyte aggregation as measured by the ESR.[47] It has been shown that with the onset of the inflammatory process during stroke, with an elevation of fibrinogen cause an increase in ESR and plasma viscosity, thereby reducing microcirculatory blood flow and promoting vascular damage.[48] In addition to studies showing that high ESR is associated with early clinical deterioration in stroke and poor short-term prognosis[49], there are also studies suggesting that it is not associated with prognosis[50]. In our study, where we considered recent and active infections as exclusion criteria, we found that the mean ESR was higher in deceased patients than in living patients, but this difference was not significant. Subsequent complications, including infection, are more likely in patients with poor initial neurological status. For this reason, we think that we see significantly higher fibrinogen and ESR values in hemorrhagic stroke patients with lower GCS and in male patients from the third day onwards. However, we believe that our patients' advanced age, history of smoking and alcohol use, comorbid diseases associated with high inflammatory markers (hypertension, diabetes, hyperlipidemia, heart failure), ordered medications (e.g., steroids, salicylates, statins, non-steroidal anti-inflammatory drugs), and factors such as the ambient temperature of the ICU may have limited our ability to determine the relationship between ESR and prognosis.

Our study had some limitations. Patients did not undergo Magnetic Resonance Imaging (MRI), so there is a possibility that small/lacunar infarcts may have been missed.

There are no data on infarct volume to assess the relationship between tissue damage and acute phase reactants. Stroke patients with comorbidities (e.g., hypertension, diabetes, hyperlipidemia) associated with high inflammatory markers were also included in the study. In the absence of creatinine clearance data, we could not draw any conclusions about the renal function of our patients.

## Conclusion

We found a 65% mortality rate among patients we monitored diagnosed as having acute stroke in the neurological ICU. We showed that GCSs, calculated from the time of admission to the ICU until day 11, were significantly lower in patients who died and that the severity of the initial neurological status was one of the most important poor prognostic factors. We also considered that high blood glucose, high blood urea, blood creatinine, triglyceride and hemoglobin levels are not only known risk factors for stroke, but also parameters that negatively affect short-term (first 12 days) stroke prognosis and therefore need to be monitored more carefully during patient follow-up. We found that the most common neurological complication was brain herniation, and the most common systemic complication was aspiration pneumonia.

## Ethical approval

This study was conducted in accordance with the Declaration of Helsinki and approved by the local Ethics Committee of the Faculty of Medicine in Trakya University, Türkiye (protocol No. 09/28).

## Ethical statements

The authors declared that no clinical trials were used in the present study.

The authors declared that no experiments on humans or human tissues were performed for the present study.

The authors declared that written informed consent was obtained from all participants before their inclusion into the study.

The authors declared that no experiments on animals were performed for the present study.

The authors declared that no commercially available immortalized human and animal cell lines were used in the present study.

## Funding

The authors have no funding to report.

## Conflict of interest

The authors have declared that no competing interests exist.

## Data availability

All data used are referenced or included in the article.

## Use of AI

No use of AI was reported.

## Author contributions

Conceptualization: HO and SO; design: HO and SO; data collection or processing: HO and SO; analysis or interpretation: SO; literature search: HO and SO; writing: HO. Both authors have approved the contents of this paper and have agreed to the Folia Medica's submission policies.

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