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Study of Correlation of Serum Cortisol Levels with Severity of Stroke

Dr Ruchit Patel[1], Dr Priyanka Jadhav[2*], Dr Archana Bhate[3], Dr Sagar Sinha[4]

[1]Consultant Gastroenterologist, Genesis Gastroenterology Clinic, Vile Parle (W), Mumbai, India

[2]Associate Professor, General Medicine, DY Patil School of Medicine Nerul & DY Patil University, Navi Mumbai, India

[3]Professor, General Medicine, DY Patil School of Medicine Nerul & DY Patil University, Navi Mumbai, India

[4]Professor, Emergency Medicine, MGM Medical College Kamothe & MGM Institute of Health Sciences, Navi Mumbai, India

Corresponding Author
Dr Priyanka Jadhav
Email: drpriyankabjadhav@gmail.com
Mobile: +91-9833030030
Address: B608, Israni Tower, Sector-15, CBD Belapur, Navi Mumbai, Maharashtra, PIN 400614

Abstract

Background: Stroke is a major cause of death and disability worldwide, with a particularly high burden in India. Prognostic markers that help assess severity at presentation are needed. Cortisol, the main stress hormone, rises after acute stroke due to activation of the hypothalamic–pituitary–adrenal (HPA) axis. While elevated levels have been associated with hyperglycemia, inflammation, and poor outcomes, its role as a marker of stroke severity remains uncertain.

Aim and Objective: To evaluated the relationship between serum cortisol and stroke severity using the Scandinavian Stroke Scale (SSS).

Materials and Methods: A hospital-based observational study was conducted over one year at the Department of General Medicine, Padmashree Dr. D. Y. Patil Medical College, Navi Mumbai. Fifty patients with acute stroke within 24 hours of onset were included. Stroke type was confirmed by neuroimaging, and severity was graded by SSS. A single serum cortisol measurement was obtained at admission using a competitive immunoenzymatic colorimetric assay. Correlations between cortisol, SSS, and clinical/laboratory parameters were analyzed using Spearman's correlation and Student's t-test.

Results: Of 50 patients (mean age >60 years; 32 males, 18 females), 76% had ischemic and 24% hemorrhagic stroke. Hypertension (40%) was the most common risk factor. The mean serum cortisol was 25.04 ± 14.65 μg/dL, and mean SSS was 33.44 ± 8.6. Cortisol showed a

strong inverse correlation with SSS (r = –0.465, p = 0.001). Significant correlations were also noted with serum cholesterol (p = 0.013) and leukocyte count (p = 0.042).

Conclusion: Serum cortisol levels correlate significantly with stroke severity and may serve as a simple, cost-effective biomarker for early risk stratification in acute stroke.

Keywords: Stroke, Cortisol, Scandinavian Stroke Scale, Prognostic marker, Inflammation

INTRODUCTION

Stroke is a major cause of death and disability worldwide, with an especially heavy impact in countries like India.[1] It arises suddenly due to either ischemia or intracerebral hemorrhage and often leaves survivors with significant neurological deficits. Despite advances in acute care, outcomes remain unpredictable, creating a need for simple markers that can help gauge stroke severity and guide early management.[2] [3]

The body's stress response is thought to play an important role in this process. Acute stroke activates the hypothalamic–pituitary–adrenal (HPA) axis, leading to a rise in cortisol, the main stress hormone.[4] Elevated cortisol has been linked with hyperglycemia, inflammation, larger infarcts, and higher mortality. However, its exact role remains unclear—whether it worsens neuronal damage or merely reflects the body's natural response to stress is still debated.[5] [6] [7]

Because of these mixed findings, it is important to study cortisol levels in stroke patients more closely, especially in the Indian population where data are scarce. This study was therefore undertaken to explore whether serum cortisol levels measured within the first 24 hours of stroke onset are related to stroke severity, assessed using the Scandinavian Stroke Scale (SSS). Understanding this link could provide a simple, cost-effective tool for identifying patients at higher risk, guiding closer monitoring and early intervention, and improving insights into how stress hormones influence recovery after stroke.

MATERIAL AND METHODS

This hospital-based observational study was carried out in the Department of General Medicine, Padmashree Dr. D. Y. Patil Medical College, Hospital & Research Center, Navi Mumbai, Maharashtra, for the period of 1 year. Patients admitted with acute stroke within 24 hours of onset were screened for eligibility. After explaining the study purpose and procedures, written informed consent was obtained from either the patient or their legal guardian. Ethical

clearance was granted by the Institutional Ethics Committee of the college, and all procedures adhered to the principles of the Declaration of Helsinki. A total of 56 consecutive patients were initially enrolled, of which six were excluded as per criteria, leaving 50 patients for final analysis. Each patient underwent detailed clinical evaluation, neuroimaging to confirm stroke type, and routine laboratory investigations. Stroke severity was assessed on admission using the Scandinavian Stroke Scale (SSS), and a single serum sample for cortisol estimation was collected at the same time.

Eligible patients were adults aged 18 years or older with a confirmed diagnosis of either ischemic infarct or intracerebral hemorrhage on CT brain. Exclusion criteria included pregnancy, pre-existing liver disease, or history of treatment with drugs known to alter cortisol metabolism such as phenytoin, rifampicin, ketoconazole, or corticosteroids. Patients with incomplete clinical records or inconclusive neuroimaging findings were also excluded to ensure accuracy of correlation.

All data were entered into a structured proforma and analyzed using appropriate statistical methods. Serum cortisol levels were measured by competitive immunoenzymatic colorimetric assay (DiaMetra kit) in laboratories blinded to clinical details. Correlations between serum cortisol, SSS scores, and other paraclinical parameters (blood glucose, blood pressure, total leukocyte count, cholesterol, and pulse rate) were assessed. Normality of data was tested using the Kolmogorov–Smirnov test. Correlation analyses were performed using Spearman's correlation coefficient, while Student's t-test was used for comparing means. A p-value <0.05 was considered statistically significant.

RESULTS

Out of 56 patients initially enrolled, 50 met the inclusion criteria and were analyzed. The study population consisted of 32 males (64%) and 18 females (36%). The majority of patients (60%) were aged above 60 years, while only 4% were younger than 30 years. Among the 50 patients, 38 (76%) presented with ischemic stroke and 12 (24%) with hemorrhagic stroke.

Regarding comorbidities, 20 patients (40%) were hypertensive, 10 (20%) were diabetic, 14 (28%) reported chronic alcohol consumption, and 11 (22%) were smokers. Hypertension was the most common risk factor observed, followed by alcohol use and diabetes.

Table 1 below summarizes the baseline profile of patients at admission, showing that most presented with elevated blood pressure and hyperglycemia, with mean serum cortisol levels of 25.04 µg/dL and mean SSS score of 33.44, indicating moderate stroke severity.

Table 1: Patient Profile on Admission				
Parameter	**Mean**	**Standard Deviation**	**Maximum**	**Minimum**
Systolic Blood Pressure (mmHg)	158.32	27.04	240	110
Diastolic Blood Pressure (mmHg)	92.24	9.12	120	70
Random Blood Sugar (mg/dL)	176.8	55.22	384	83
Serum Cholesterol (mg/dL)	197.42	23.51	312	164
Total WBC Count (cells/cu.mm)	9947.7	2679.56	14340	4500
Serum Cortisol (Âµg/dL)	25.04	14.65	70.12	6.55
Scandinavian Stroke Scale (SSS)	33.44	8.6	53	20
Pulse Rate (beats/min)	80.42	6.57	92	68

Table 2 demonstrates that serum cortisol levels had a significant positive correlation with serum cholesterol (p=0.013) and total leukocyte count (p=0.042), while showing a strong inverse correlation with SSS scores (p=0.001), highlighting its association with stroke severity.

Table 2: Correlation of Serum Cortisol with Clinical/Lab Parameters		
Parameter	**Correlation with Serum Cortisol**	**p-value**
Systolic Blood Pressure (mmHg)	0.277	0.051
Diastolic Blood Pressure (mmHg)	0.167	0.247
Random Blood Sugar (mg/dL)	-0.081	0.575
Serum Cholesterol (mg/dL)	0.348	0.013*
Total WBC Count (cells/cu.mm)	0.082	0.042*
Scandinavian Stroke Scale (SSS)	-0.465	0.001*
Pulse Rate (beats/min)	0.006	0.966

[Correlation by Spearman's correlation coefficient, P value by Univariate regression,* indicates significant results]

Table 3 further supports these findings, showing that lower SSS scores (greater severity) were significantly related to higher cortisol levels (p=0.001), elevated leukocyte count (p=0.037), and higher pulse rate (p=0.002), suggesting that both endocrine and inflammatory responses play an important role in determining stroke severity.

Table 3: Correlation of SSS with Clinical/Lab Parameters

Parameter	Correlation with SSS	p-value
Systolic Blood Pressure (mmHg)	-0.082	0.57
Diastolic Blood Pressure (mmHg)	-0.022	0.881
Random Blood Sugar (mg/dL)	0.163	0.257
Serum Cholesterol (mg/dL)	-0.138	0.341
Total WBC Count (cells/cu.mm)	0.066	0.037*
Serum Cortisol (Âµg/dL)	-0.465	0.001*
Pulse Rate (beats/min)	0.435	0.002*

[Correlation by Spearman's correlation coefficient, P value by Univariate regression,* indicates significant results]

DISCUSSION

In this hospital-based study, we observed that serum cortisol levels measured within 24 hours of stroke onset were significantly correlated with stroke severity assessed by the Scandinavian Stroke Scale (SSS). Patients with higher cortisol values tended to have lower SSS scores, indicating more severe neurological deficits. This finding is consistent with earlier reports that have highlighted cortisol as a reliable indicator of stress response in acute stroke and its potential role as a prognostic marker.

Our results align closely with the work of Christensen et al. (2004)[8], who demonstrated that serum cortisol was significantly associated with stroke severity, lesion volume, and early mortality in a cohort of 172 patients. Similarly, Neidert et al. (2009)[9] reported that elevated cortisol levels on admission predicted poor functional outcome and higher mortality in

ischemic stroke, with predictive accuracy comparable to the NIHSS score. Zi and Shuai (2013)[10] also confirmed these observations in a larger prospective cohort of 226 patients, where cortisol levels independently predicted poor outcomes and mortality within 90 days, even after adjustment for confounding clinical variables. Together, these studies establish cortisol as a consistent marker of stroke severity and prognosis.

Additional evidence from Slowik and colleagues (2002)[11] further supports this relationship, showing that hypercortisolemia after acute stroke was strongly linked to markers of inflammation such as fever, fibrinogen, and leukocyte count. Our findings mirror this association, as cortisol demonstrated significant correlations with both serum cholesterol and total leukocyte count, while stroke severity itself correlated with leukocytosis and pulse rate. These results reinforce the hypothesis that endocrine and inflammatory pathways interact during acute stroke, potentially aggravating neuronal damage.

Johansson et al. (2000)[12] reported that cytokines such as IL-6 modulate cortisol secretion, and observed significant correlations between IL-6 and cortisol during the first days after stroke. Similarly, Marlund et al. (2004)[13] showed that both excessively high and low cortisol levels early after stroke were associated with increased mortality and cognitive dysfunction. These observations highlight the dual nature of cortisol—while necessary for maintaining physiological homeostasis, both extremes of cortisol secretion may have deleterious consequences. In our study, although cytokine levels and long-term outcomes were not evaluated, the strong inverse relationship between cortisol and SSS suggests that HPA axis dysregulation plays an important role in the early phase of stroke in Indian patients.

Overall, our findings corroborate existing international literature and extend it to an Indian hospital setting where such data are limited. Elevated serum cortisol appears to be a useful biomarker of stroke severity and may serve as a cost-effective adjunct for early risk stratification in resource-limited settings. Future multicenter studies with larger cohorts and longitudinal follow-up are warranted to determine whether cortisol can reliably predict functional outcomes and mortality, and whether therapeutic modulation of the stress response could improve recovery.

CONCLUSION

This study found a significant inverse correlation between serum cortisol levels and stroke severity, with higher cortisol levels associated with poorer Scandinavian Stroke Scale scores. Cortisol also showed significant associations with leukocyte count and cholesterol, reflecting

the interplay of endocrine, inflammatory, and metabolic responses in acute stroke. These findings are in line with previous studies that identified cortisol as a marker of severity and adverse outcomes, underscoring its potential role as a simple, cost-effective biomarker for early risk stratification in stroke patients. Further large-scale studies are needed to validate its prognostic utility and explore its integration into routine clinical practice.

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