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Role of Serum Creatine Phosphokinase in Predicting Stroke Severity and Short-Term Outcomes: A Prospective Observational Study in a Tertiary Care Hospital in Srinagar (J&K)

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Abstract

Background: Stroke is a leading cause of disability and mortality worldwide, with an increasing burden in low- and middle-income countries like India. Serum creatine phosphokinase (CPK), a widely available biomarker, may serve as an early indicator of stroke severity and prognosis. This study evaluates the predictive utility of serum CPK in stroke patients admitted within 24 hours of symptom onset.[1,2]

Methods: A prospective observational study was conducted over 24 months at a tertiary care center in Srinagar, Kashmir. Adults aged >45 years with first-ever strokes were enrolled. Stroke severity and functional outcomes were assessed using the NIH Stroke Scale (NIHSS) at admission and the Modified Rankin Scale (mRS) at 1 and 4 weeks. Serum CPK levels at admission were measured and correlated with clinical outcomes.

Results: Among 183 patients (66.7% male, mean age 72.3 ± 6.8 years), ischemic stroke was predominant (76.1%). Elevated serum CPK levels correlated strongly with stroke severity (NIHSS, $r = 0.88$, $p < 0.001$) and functional impairment (mRS at 1 week, $F = 881.57$, $p < 0.001$; mRS at 4 weeks, $F = 175.94$, $p < 0.001$). ROC analysis showed high diagnostic accuracy for mortality (mRS = 6) at 1 week (AUC = 0.984) and 4 weeks (AUC = 0.970). Optimal CPK cut-off values for mortality were 573 IU/L at 1 week and 530 IU/L at 4 weeks.

Conclusion: Serum CPK serves as an accessible and cost-effective biomarker for early risk stratification in stroke patients. Its strong correlation with stroke severity and short-term prognosis suggests that CPK testing could enhance clinical decision-making, especially in resource-limited settings.

Keywords: Creatine Phosphokinase, Prognostic, Stroke,

Introduction

Stroke remains a major global public health concern, particularly in low- and middle-income countries like India, where healthcare access is limited. Early identification of high-risk patients is crucial for optimizing management and improving outcomes. While neuroimaging plays a fundamental role in stroke evaluation, biomarkers such as creatine phosphokinase (CPK) provide a rapid, inexpensive means of assessing tissue damage and systemic stress.[3,4]

CPK is an enzyme found in muscle, cardiac tissue, and the brain, with elevated levels indicating cellular injury. Previous research has suggested that CPK levels may correlate with stroke severity and functional outcomes, but data remain inconsistent. The present study seeks to establish the prognostic value of serum CPK in predicting stroke severity and short- term recovery.[5,6]

Methods

Study Design & Setting

A prospective observational study was conducted at Government Medical College, Srinagar, over two years (January 2023–January 2025). This center serves as the primary referral hospital in Kashmir.

Inclusion Criteria

- First-ever acute stroke patients (ischemic or hemorrhagic)
- Admitted within 24 hours of symptom onset
- Age > 45 years

Exclusion Criteria

- Previous history of stroke
- Liver or renal disease, acute coronary syndrome
- Recent trauma or intramuscular injections
- Neuroleptic drug use

Assessment Tools

- NIHSS (Neurological impairment severity at admission)
- mRS (Functional recovery at 1 & 4 weeks)
- Serum CPK levels at admission (biochemical marker)

Results

Demographics & Stroke Characteristics

Among 183 patients, the majority were male (66.7%), with mean age 72.3 ± 6.8 years. Ischemic strokes were most common (76.1%), with hypertension (40.98%) and diabetes (27.32%) as prevalent comorbidities.

Serum CPK and Stroke Severity

- Higher CPK levels were strongly correlated with worse NIHSS scores.
- Patients with severe strokes (NIHSS > 20) had significantly elevated CPK levels.
- CPK had high predictive accuracy for stroke severity and short-term functional outcomes.

Serum CPK and Short-Term Outcomes

- mRS at 1 week: Elevated CPK levels were associated with higher disability and increased mortality.
- mRS at 4 weeks: CPK remained an independent predictor of poor recovery.
- ROC Analysis confirmed CPK thresholds of 573 IU/L (1 week) and 530 IU/L (4 weeks) for identifying high-risk patients.

Regression Analysis

Multivariate analysis showed that NIHSS and fasting blood sugar were independent predictors of mortality,

with CPK emerging as an additional independent predictor at 4 weeks.

Discussion

The results of this study strongly reinforce the prognostic utility of serum creatine phosphokinase (CPK) in acute stroke patients, highlighting its correlation with disease severity and short-term outcomes. The findings demonstrated a significant positive relationship between elevated serum CPK levels and higher National Institutes of Health Stroke Scale (NIHSS) scores at admission, suggesting that CPK levels reflect the extent of neurological impairment. Patients presenting with higher CPK concentrations had markedly worse functional outcomes as measured by the Modified Rankin Scale (mRS) at one and four weeks post-stroke, further supporting its role as a biomarker for predicting early recovery. The Receiver Operating Characteristic (ROC) analysis revealed an exceptional diagnostic accuracy of CPK in predicting mortality, with optimal cut-off values determined at 573 IU/L for one-week mortality prediction and 530 IU/L for four-week prognosis. These cut-off values exhibited high sensitivity and specificity, implying that CPK could serve as a reliable marker for identifying high-risk patients requiring intensive care and early interventions. Additionally, multivariate regression analysis confirmed that NIHSS and fasting blood sugar remained independent predictors of stroke mortality, but serum CPK emerged as an additional predictor specifically at four weeks. This finding suggests that while traditional clinical measures like NIHSS are critical for early stroke severity assessment, CPK can enhance predictive accuracy over time, allowing clinicians to refine their risk stratification approaches. The gender distribution in the study showed that stroke incidence was higher in males, and hypertension and diabetes mellitus were prevalent among participants, indicating a well-established association between vascular risk factors and stroke burden. The predominance of ischemic strokes in the study aligns with global epidemiological patterns, reinforcing that ischemic strokes account for the majority of cerebrovascular events worldwide. Furthermore, the data on hypertension grades showed that most hemorrhagic stroke patients had significantly higher blood pressure levels, reaffirming hypertension's role as a major contributing factor to hemorrhagic stroke events. The lipid profile analysis indicated moderate dyslipidemia among participants, underscoring the importance of lipid control in stroke prevention. The mean duration of hospital stay among stroke patients was approximately 19.4 days, suggesting prolonged recovery periods and the need for sustained rehabilitation efforts. Importantly, smoking history among male participants was significantly higher than among females, which may explain the greater stroke incidence among men and aligns with prior research demonstrating a strong link between smoking and cerebrovascular diseases. The structured assessment through NIHSS and mRS enabled robust statistical validation of CPK's predictive value, with Pearson correlation analysis showing a strong association between NIHSS scores and CPK levels. Given the practical advantages of CPK— its affordability, accessibility, and ease of measurement—it holds promise as a biomarker for stroke severity assessment, especially in resource-constrained healthcare environments where neuroimaging may not be immediately available. Despite its potential, clinicians must interpret elevated CPK levels cautiously, considering confounding factors such as muscle breakdown, myocardial infarction, and systemic stress responses that could influence CPK readings independent of stroke severity. Future research should focus on validating CPK's prognostic role in larger, multi-center

studies and explore whether serial CPK measurements over the course of stroke recovery could further enhance predictive accuracy. Overall, this study underscores the growing relevance of biochemical markers in stroke management, advocating for the integration of serum CPK testing into routine clinical protocols to facilitate timely risk stratification and personalized treatment strategies.

Clinical Implications

- CPK is an effective early biomarker for assessing stroke severity.
- Elevated CPK levels correlate with higher mortality risk.
- CPK testing could aid early risk stratification, particularly in settings with limited access to advanced neuroimaging.

Study Strengths

- Prospective design enhances data validity.
- Robust statistical analyses confirm CPK's predictive power.
- Large sample size (n = 183) strengthens conclusions.

Limitations

- Single-center study limits generalizability.
- CPK elevations may result from non-stroke causes, requiring clinical context for accurate interpretation.

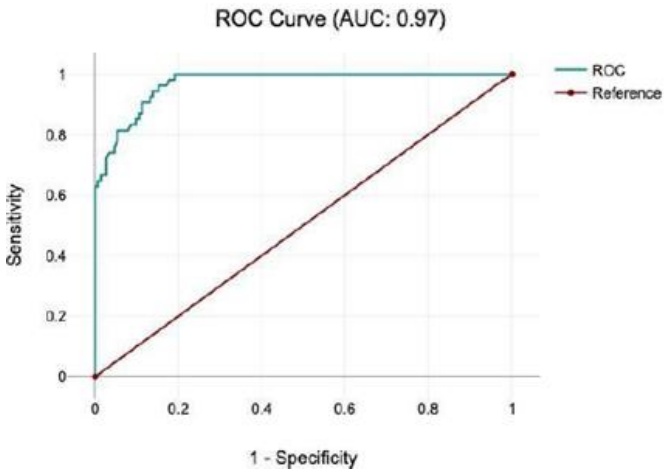
Conclusion

This study confirms that serum CPK is a valuable biomarker for predicting stroke severity and short-term recovery. Given its cost-effectiveness and accessibility, incorporating CPK measurements into routine stroke assessment protocols could enhance early intervention strategies—particularly in resource-limited settings.

Table 1: Multivariate logistic regression, Dependent variable is MRS Score of 6 at 1 week

	Coefficient B	Standard error	z	p	Odds Ratio	95% conf. interval
Constant	-158.08	110.55	1.43	.153	0	0 - 2
Gender Male	-9.52	7.97	1.19	.232	0	0 - 446.73
Systolic blood pressure	0.17	0.17	0.97	.33	1.18	0.85 - 1.65
Diastolic blood pressure	-0.15	0.14	1.13	.257	0.86	0.66 - 1.12
Hypertension yes	-2.51	1.99	1.26	.207	0.08	0 - 4.01
Diabetes yes	-9.01	6.65	1.36	.175	0	0 - 55.59
Dyslipedemia Yes	-3.91	3.38	1.16	.247	0.02	0 - 14.95
Stroke type 2	-7.52	5.73	1.31	.189	0	0 - 40.73
age	-0.23	0.2	1.17	.243	0.79	0.53 - 1.17
Lipid (TG)	0.01	0.07	0.1	.92	1.01	0.87 - 1.16
Total Cholesterol	0.06	0.06	0.91	.364	1.06	0.94 - 1.19
Serum CPK	0.16	0.11	1.49	.137	1.17	0.95 - 1.44
NIHSS at Admission	**0.58**	**0.28**	**2.07**	**.039**	**1.79**	**1.03 - 3.09**
Fasting Blood Sugar (mg/dL)	0.26	0.21	1.23	.217	1.3	0.86 - 1.97
BMI	1.04	0.94	1.11	.267	2.84	0.45 - 17.93
Smoking History Yes	0.66	3.1	0.21	.833	1.93	0 - 845.64

Figure 1:



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