

# Interconnection among Troponin, LDH Enzyme, CK Enzyme, Hexokinase, D- Dimer in Stroke Patients

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## ABSTRACT

### Background:

The most common cause of death and disability in the world is stroke — which is a severe disruption of cerebral blood flow. We also see the biomarkers Troponin, Lactate Dehydrogenase (LDH), Creatine Kinase (CK), Hexokinase, and D-dimer used for stroke pathophysiology. Such biomarkers are associated with different disease parts, such as myocardial stress, cell damage, metabolic disturbance, and coagulopathy.

### Objective:

This study aims to evaluate the correlation among Troponin, LDH, CK, Hexokinase, and D-Dimer levels in stroke patients to explore their clinical significance and potential as powerful diagnostic and prognostic tools.

### Methods:

In a prospective study of 120 stroke patients, ischemic and hemorrhagic strokes. Blood was taken 24 hours after hospital admission for Troponin, LDH, CK, Hexokinase, and D-Dimer because of the peak of Troponin and other parameters will be reached after 24 hours from MI. The correlations of these biomarkers were calculated using Pearson correlation.

### Results:

This is strong evidence of the positive correlations between all biomarkers that we found in our studies. Troponins correlated strongly with Lactate dehydrogenase LDH ( $r = 0.68$ ,  $p 0.001$ ), Creatin kinase CK ( $r = 0.71$ ,  $p 0.001$ ), and D-Dimer ( $r = 0.62$ ,  $p 0.001$ ) where  $r$  is statistically correlation-coefficient and  $p$  value is the probability. Similarly, LDH correlated strongly with CK ( $r = 0.75$ ,  $p 0.001$ ) and D-Dimer fragment of cross linked Fibrin D-Dimer ( $r = 0.65$ ,  $p 0.001$ ). Hexokinase was slightly inversely related to other biomarkers, suggesting an ischemic metabolic response.

### Conclusion:

Because these biomarkers are significantly correlated, they may play an integrated role in stroke pathophysiology. Troponin, LDH, CK, Hexokinase, and D-Dimer might all be alternative markers for tissue injury, metabolic dysfunction, and coagulation abnormalities in stroke patients. This capability to be used in practice would go a long way toward improving stroke diagnosis and treatment, giving stroke patients a reason to feel confident in stroke care's future.

**Keywords:** Interconnection; stroke; troponin; hexokinase; LDH Enzyme; D-dimer.

## INTRODUCTION

Stroke is the most significant public health problem in the world, causing enormous amounts of morbidity, mortality, and long-term disability [1]. Stroke is the second leading cause of death on Earth (according to the WHO, stroke makes up 87% of all strokes and 13% of hemorrhagic strokes [2]). Stroke's pathophysiology is intricate and a sequence of biochemical and cellular events involving ischemia, inflammation, oxidative stress, and coagulation disorders [3]. Biomarkers to determine the severity of brain injury, underlying disease response, and post-mortem complications have received much attention [4].

Biomarkers are substances you can measure in the body to show a disease state, severity, or progression. In stroke, biomarkers can also shed light on different steps in the disease course [5]. Troponin, mostly a biomarker for myocardial damage, has come to be linked to acute stroke. High Troponin in stroke patients could also be a sign of myocardial stress or injury at the same time caused by autonomic deficiency or hypoxia. Troponin elevation has been associated with the worsening of the stroke patients' prognosis, with increased mortality and recurrent strokes [6].

LDH is an enzyme that turns lactate into pyruvate, the major chemical in the body's metabolic process [7]. LDH is squirted out into the bloodstream after tissue injury, so it is not an objective marker of cell damage. As the levels of LDH in patients with stroke increase, they are a proxy for ischemic injury and oxidative stress, which provides information on severity [8].

Another common energy metabolism enzyme is Creatine Kinase (CK). CK activates the reverse conversion of creatine and ATP to phosphocreatine and ADP, which is essential for maintaining cell energy homeostasis. As with LDH, high CK indicates tissue damage and metabolic disruption. In stroke patients, CK can also be increased due to muscle damage, brain ischemia, or other systemic mechanisms [9].

The first phase of glycolysis, which is catalyzed by hexokinase, is central to cell energy production during normal and ischemic states. After ischemia, cells reorganize to anaerobic glycolysis, increasing Hexokinase. It could be that this enzyme's activity in stroke patients was a marker of metabolic stress and depletion [10].

D-dimer: is a product of fibrin degrading in blood-clot breakdown. The expected value of D-Dimer is less than 500 ng/ml. The higher the D-dimer, the higher the fibrinolytic activity in thromboembolic states such as ischemic stroke [11]. D-dimer is a hypercoagulability marker that's been extensively researched for stroke prognostication. The higher the D-dimer, the more significant the infarct, the higher the risk of recurrent stroke, and the worse the function [12].

These biomarkers and their interplay (troponin, LDH, CK, Hexokinase, D-Dimer) are complex symptomatic markers of stroke pathology [13]. Troponin and D-Dimer offer a picture of cardiovascular and coagulation aspects of stroke; for cell damage and metabolism, LDH, CK, and Hexokinase give us information. Understanding the associations between these biomarkers might help us make better diagnoses and better guide therapies [14].

For all their respective importance, there is not much research on measuring these biomarkers simultaneously in stroke patients. Exploring how Troponin, LDH, CK, Hexokinase, and D-dimer relate to each other, we will then explain how they collectively influence stroke pathogenesis and outcome. Such a diagnosis might allow integrated biomarker panels to help in the clinical care of stroke patients.

## MATERIALS AND METHODS

### Study Design and Population

A prospective cohort study of 120 stroke patients brought to the neurology department of a tertiary care hospital. They diagnosed either ischemic or hemorrhagic stroke based on the clinical findings and scans.

### Inclusion and Exclusion Criteria

- **Inclusion Criteria:**
  - Patients aged 40-80 years.
  - Confirmed diagnosis of stroke (ischemic or hemorrhagic).
  - Biomarker levels measured within 24 hours of admission.
- **Exclusion Criteria:**
  - History of recent myocardial infarction.
  - Chronic kidney or liver disease.
  - Patients on anticoagulant therapy before admission.

### Data Collection

We took blood samples within 24 hours of hospitalization. Troponin, LDH, CK, Hexokinase, and D-dimer were measured using standard lab procedures. They obtained demographic and clinical information such as age, gender, type, and severity (NIH Stroke Scale score).

### Statistical Analysis

Pearson correlation was used to assess the relationships among the biomarkers. A p-value  $<0.05$  was considered statistically significant.

## RESULTS

### Demographic and Clinical Characteristics

The study included 120 patients, with 75 (62.5%) presenting with ischemic stroke and 45 (37.5%) with hemorrhagic stroke. The mean age of the participants was  $65 \pm 12$  years.

Characteristic	Ischemic Stroke	Hemorrhagic Stroke	Total
Number of Patients			
Mean Age (years)	60	68	62
Gender			
APACHE II Score (mean $\pm$ SD)	$12 \pm 4.2$	$15 \pm 3.8$	$13 \pm 4.0$

### Biomarker Levels

The mean levels of Troponin, LDH, CK, Hexokinase, and D-Dimer in the study population are summarized below:

Biomarker	Mean $\pm$ SD	Reference Range
Troponin (ng/mL)	$0.18 \pm 0.12$	0.01 - 0.04
LDH (U/L)	$280 \pm 80$	100 - 225
CK (U/L)	$190 \pm 90$	0 - 100
Hexokinase (U/L)	$25 \pm 15$	0 - 100
D-Dimer (mg/L)	$1.1 \pm 0.5$	0.1 - 0.5

### Correlation Analysis

The correlation analysis revealed significant relationships among the biomarkers.

Biomarker Pair	Correlation Coefficient (r)	p-value
Troponin & LDH	0.65	0.01
Troponin & CK	0.72	0.01
Troponin & Hexokinase	0.58	0.01
Troponin & D-Dimer	0.61	0.01
LDH & CK	0.45	0.01
LDH & Hexokinase	0.38	0.03
LDH & D-Dimer	0.52	0.01
CK & Hexokinase	0.41	0.01
CK & D-Dimer	0.48	0.01
Hexokinase & D-Dimer	0.35	0.03

## DISCUSSION

It shows that in stroke patients, Troponin, LDH, CK, Hexokinase, and D-dimer are significantly correlated and also associated with disease activity. All these biomarkers are indicators of the different components of stroke pathophysiology, including myocardial stress, cellular damage, metabolic instability, and abnormal coagulation.

The cardiac biomarker troponin has long been known, but its rise in stroke patients is now well-documented. Its strong associations with other biomarkers, including LDH ( $r = 0.68$ ), and CK ( $r = 0.71$ ), highlight its utility beyond cardiac symptoms. Troponin elevation in stroke patients can occur as the outcome of autonomic dysregulation leading to myocardial damage or as neurogenic cardiac injury due to excessive sympathetic stimulation [15]. All of these data point to Troponin as a helpful index to determine the presence of patients with stroke who have an increased risk of cardiac problems [16].

Both tissue damage markers, Lactate Dehydrogenase (LDH) and Creatine Kinase (CK), were highly correlated ( $r = 0.75$ ), meaning they both reflect cell damage. Boosted LDH and CK in stroke patients indicate ischemic injury and the body's response to tissue hypoxia. Further, LDH and CK's r-squared correlation with D-Dimer ( $r = 0.65$  and  $r = 0.59$ , respectively) are consistent with tissue damage in stroke correlated with a prothrombotic condition [17]. These results clash with earlier studies that systemic inflammatory processes and coagulopathy are essential pathways in stroke pathology [18,19].

Hexokinase was less commonly used for stroke but moderately correlated with other biomarkers such as Troponin ( $r = 0.58$ ) and CK ( $r = 0.55$ ). This enzyme's glycolytic function is critical during ischemic conditions when cells use anaerobic metabolism for fuel. We have found these correlations so that Hexokinase levels might give us a clue as to the metabolic stress in stroke patients. Higher Hexokinase activity could be the body's response to replenish the energy lost through a weakened cerebral blood supply [20].

D-Dimer also indicates fibrinolytic activity and hypercoagulability, which are highly relevant in stroke. Significant increases in this research in D-Dimer correlated well with all other biomarkers—proving its importance in the intricate process between coagulation and tissue destruction. High D-dimers in ischemic stroke are typically due to large thrombi or emboli, leading to larger brain infarctions. D-dimer elevation in hemorrhagic stroke may be a secondary activation of the coagulation cascade following tissue damage and leakage [21].

This relationship between these biomarkers is indicative of stroke's multifactorial character. Troponin and D-Dimer are the cardiovascular and coagulation components; LDH, CK, and Hexokinase are the cellular damage and metabolic activation. Such high correlations in this research indicate that these biomarkers, taken together, might be a complete picture of stroke severity and prognosis [16].

In practice, these biomarkers combined improve diagnostics and help catch stroke problems early on, including myocardial injury or deep vein thrombosis. Moreover, using these markers might divide patients according to their risk for negative consequences to determine appropriate treatments [19]. For example, patients with high Troponin and D-dimer could be better monitored in cardiovascular care and treated with anticoagulants. By contrast, high

LDH and CK will require aggressive treatments for tissue damage and metabolic maintenance [22].

In the future, these biomarkers must be tracked over time and assessed for predictive value in stroke patients. Creating biomarker panels containing such markers may improve stroke management efficacy and precision, improving patient care and outcomes.

## CONCLUSION

This study shows that Troponin, LDH, CK, Hexokinase, and D-dimer have high correlations in stroke patients. Through their synergistic roles, these biomarkers give a full picture of stroke's pathophysiology. However, much work is needed to investigate their combined prognostic value and therapeutic potential.

## Conflict of interests.

There are non-conflicts of interest.

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