RESEARCH ARTICLE

Correlation between Serum Troponin-I Levels and Acute Stroke in Coronary Artery Disease Patients

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ABSTRACT

Background: Studies reveal that the average age of stroke in less developed nations would be younger due to changing population structures brought on by increased mortality rates and other causes of death. The risk of death for AS patients is highest in the first few weeks and varies from 20 to 50% in the first month, depending on age, severity of stroke, other medical disorders, and treatment effectiveness for all outcomes. Furthermore, Tn subunits bind to muscle filaments, according to studies. Since it is a sensitive sign of myocardial necrosis, researchers have determined that it is widely used to diagnose acute coronary syndromes.

Objective: To assess the link between serum TnI levels and AS in CAD cases.

Methods: Based on the average patient volume and available resources at the KIMS, Karad from October 2017 to December 2019, our study comprised 57 patients. A structured and validated questionnaire was utilized to collect data. Microsoft Excel received the data. Tests were conducted using Epi-Info 7.2 and SPSS version 20. The student's t-test is used to determine correlation, whereas the chi-square test was utilized to measure association.

Result: A strong correlation was observed between mortality and elevated TnI levels in AS cases ($p < 0.001$).

Conclusion: Patients with AIS showed significant TnI levels. Serum TnI levels were higher in patients with AIS, despite these levels being thought to be more selective for myocardial damage. TnI levels and AIS prognosis were substantially associated. The results of this study will improve our understanding of cardiac enzymes in AIS patients as well as how they impact prognosis.

Keywords: AIS, CAD, Mortality, Outcome, TnI levels.

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INTRODUCTION

The "World Health Organization (WHO) has provided a definition for stroke, which is characterized as the rapid onset of clinical symptoms indicating a localized or widespread disruption of cerebral function. These symptoms must persist for more than 24 hours or result in death, with no apparent cause other than vascular origin. This encompasses the majority of instances of subarachnoid hemorrhage, intracranial hemorrhage, and cerebral infarction. However, it does not encompass cases of transient ischemic attacks. Here, term "global" disturbances of cerebral function specifically pertains to patients diagnosed with subarachnoid hemorrhage who do not exhibit any focal neurological deficits".[1] According to many studies, "it presents with a wide range of neurological signs and symptoms that vary based on the extent and location of the brain damage as well as the underlying cause". "The conditions encompassed in this category consist of coma, hemiplegia, paraplegia, monoplegia, cranial nerve palsy, speech disturbance, and sensory impairment, among others. Among these options, hemiplegia is the prevailing manifestation, observed in approximately 90% of patients".[2]

According to global data from 2013, "stroke ranked as the second leading cause of mortality, accounting for 11.8% of all deaths (95% uncertainty interval [UI]: 10.9–13.0%), following ischemic heart disease, which accounted for 14.8% of all deaths (95% UI: 13.4–15.8%). Additionally, stroke was identified as the third most prevalent cause of disability, contributing to 4.5% of disability-adjusted life years (DALYs) across all causes (95% UI: 4.1–5.2%), with ischemic heart disease ranking higher at 6.1% (95% UI: 5.5–6.8%). In general, the stroke burden estimates from the Global Burden of Disease (GBD) 2013 study validate prior findings about the notable rise in stroke burden worldwide over the last 25 years, particularly in developing nations. Additionally, there are considerable regional disparities in the prevalence of stroke".[3] Studies have concluded that the average age at which a stroke occurs in demographically developed nations is approximately 73 years, this reflects that the older age structure of these nations. Hence, "it was also concluded by my

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researchers that the probability of a first AIS is approximately 1.6 and 0.4% per 1,000 individuals, respectively".[4]

Because of the altered population death structure brought on by higher mortality rates and other causes of death, the average age of stroke in less developed countries will be younger. "The risk of death for AIS patients is greatest in the first few weeks following the attack and ranges from 20 to 50% during the very first month, depending on the patient's age, the severity of the stroke, number of other medical conditions, and the efficacy of treatment for all consequences". Furthermore, according to studies, "patients who had made a full recovery might have been left with no disability at all, a light disability, a moderate disability, or a severe disability. Typically, patients have up to six months to heal on their own.[5] However, studies have also concluded that "patients with a history of stroke have a 10% chance of having another stroke within the first year, and a 5% chance every year after that. The range of patients who achieve independence in self-care within one year after a stroke varies from approximately 60 to 83%. The user's text is too short to be rewritten in a technical writing style".[5] In addition to that, researchers have also concluded that "Cardiac morbidities contribute to 20% of mortality cases following AIS and rank as the second most prevalent cause of death among individuals with AIS".[5]

"Studies have shown that Tn complex consists of three subunits: TnC, TnI, and TnT. Additionally , studies have also concluded that, it is located on the myofibrillar thin (actin) filament of striated (skeletal and cardiac) muscle. Out of which, studies revealed that TnC binds to the calcium ions to produce a change in TnI, TnT binds to the tropomyosin, locking them to form a troponin- tropomyosin complex and troponin I binds to the actin in thin myofilaments and holds the troponin- tropomyosin complex in place".[6] Since Tn estimation is a sensitive indicator of myocardial necrosis, researchers have come to the conclusion that it is widely employed for the precise diagnosis of acute coronary syndromes(ACS).[7,8] Hence, our study aimed to evaluate the relationship between serum Tn I level and AIS in unknown CAD cases.

AIM

The study aim to evaluate the relationship between serum TnI level and AIS in unknown CAD cases.

Inclusion Criteria

- Those cases with confirmed Acute cerebral infarction by CT Brain or MRI.
- Both male and female genders are involved in our study.
- Patients of above 18 years of age were included.

Exclusion Criteria

Patients who are affected by the following disease were excluded from our study are as follows:-

- Hemorrhagic stroke
- TIA
- Acute coronary syndrome
- Ischemic changes on ECG at rest
- End-stage renal disease
- Pulmonary embolism
- Recent ischemic heart disease (2 weeks prior)
- Heart failure
- Sepsis/Septic shock/systemic inflammatory response syndrome

MATERIALS AND METHOD

Our study included a minimum of 57 patients based on the average patient volume at our hospital and the available resources.

Study Type

In our study, we have done an observational cross-sectional study.

Study Area and Setting

The research will encompass the timeframe from October 2017 to December 2019 at the KIMS, Karad.

Selection of Participants

Patients who were admitted to the medicine ward of the hospital and had just had an AIS but who did not have any known history of CAD were included in the study.

Statistical Analysis

A standardized and validated questionnaire was utilized to gather the data. The collected data was input into Microsoft Excel. Data is commonly depicted using frequencies, percentages, charts, and graphs. The mean and standard deviation of the quantitative variables are displayed. The statistical tests were conducted using Epi-Info version 7.2 and SPSS software version 20 for analysis. The Chi-square test is used to assess association, while the student's t-test is employed to determine correlation when applicable.

RESULT

The current study included a cohort of 100 patients who were admitted with a diagnosis of AIS. A comprehensive analysis was conducted to examine the levels of TnI in these patients, taking into account various factors such as the patient's medical history, co-morbidities, and outcome.

In our study, the participant population consisted of 36 females, accounting for 36% of the total, and 64 males, representing 64% of the total. This resulted in a male-to-female ratio of 1.78:1 (Table 1).

According to our study, it was observed that 58 patients (58%) experienced weakness in their right limbs, while the

Table 1: Gender distribution

Gender	*n*	*Percent*
Female	36	36
Male	64	64
Total	100	100

Table 2: Weakness of limbs

Weakness of limbs	*n*	*Percent*
Left	42	42
Right	58	58
Total	100	100

Table 3: History of chest pain

History of chest pain	*n*	*Percent*
No	86	86
Yes	14	14
Total	100	100

Table 4: History of tabacco

History of tobacco chewing	*n*	*Percent*
No	36	36
Yes	64	64
Total	100	100

Table 5: History of alcoholism

History of alcoholism	*n*	*Percent*
No	80	80
Yes	20	20
Total	100	100

Table 6: Diabetes mellitus type 2

Diabetes mellitus type 2	*n*	*Percent*
No	65	65
Yes	35	35
Total	100	100

Table 7: Hypertension distribution

Hypertension	*n*	*Percent*
No	49	49
Yes	51	51
Total	100	100

Table 8: Dyslipidemia distribution

Dyslipidemia	*n*	*Percent*
No	22	22
Yes	78	78
Total	100	100

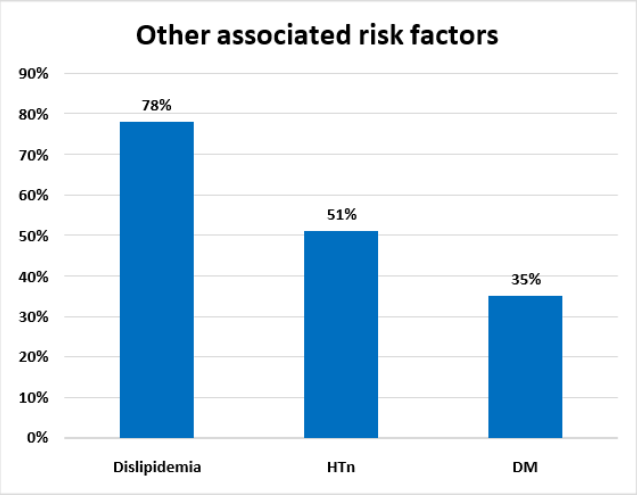

Figure 1: Associated risk factor (RF)

Table 9 : Troponin-I distribution

Troponin I	*n*	*Percent*
Normal	82	82
Raised	18	18
Total	100	100

Table 10: Outcome distribution

Outcome	*n*	*Percent*
Expired	23	23
Recovered	77	77
Total	100	100

Table 11: Descriptive statistics

	Minimum	*Maximum*	*Mean*	*Standard deviation*
Age	34	95	64.01	14.43
TC	100	297	175.99	48.69
Triglycerides	60	607	141.03	69.42
HDL	13	75	42.89	11.67
LDL	23	254	119.48	52.23
TROPONIN-I	0	4	0.14	0.57
SerumCreatinine	0	7	1.26	0.76
HEMOGLOBIN	5	19	12.76	2.48

remaining 42 patients (42%) exhibited weakness in their left limbs (Table 2).

Among a sample of 100 patients, it was found that 14 patients, accounting for 14% of the total, had a documented history of chest pain (Table 3).

Out of the 100 patients surveyed, 64 individuals, accounting for 64% of the sample, reported a history of tobacco consumption (Table 4).

In our study, a total of 100 patients were assessed, with 20 patients (20%) identified as having alcoholism (Table 5).

A study revealed that 78% of individuals exhibited dyslipidemia, 51% had hypertension, and 35% had DM type 2 as additional RF (Figure 1).

Our study found that among a sample of 100 patients, 35 individuals were undergoing treatment for type 2 DM (Table 6).

In our study we found that, 51 patients had hypertension (51%) (Table 7).

Our study observed that 78 patients out of 100 (78%) had dyslipidemia (Table 8).

A total of 18 patients (18%) exhibited elevated levels of troponin I, while the remaining 82 patients (82%) demonstrated normal levels (Table 9).

The study observed a mortality rate of 23%, with 23 patients experiencing fatalities while the remaining 77 patients achieved recovery (Table 10).

The average age of patients included in the study was 64.01 ± 14.43 years, ranging from 34 to 95 years. The average total cholesterol level was 175.99 ± 48.69, ranging from 100

to 297. The average triglyceride level was 141.03 ± 69.42, ranging from 60 to 607. The average high-density lipoprotein (HDL) level was 42.89 ± 11.67, with a range of 13 to 75. The average serum TnI level was 0.14 ± 0.57, ranging from 0 to 4. The average hemoglobin level was 12.76 ± 2.48, ranging from 5 to 19. Our study observed a mortality rate of 23%, with 23 patients experiencing fatalities while the remaining 77 patients achieved recovery (Table 11).

Increased Tn I Analysis

The increase in tnI levels did not have a significant association with gender. The probability value is 0.42 (Table 12).

In our study, we found no significant correlation between the elevation of TnI levels and the presence of chest pain. The value of p is 0.72 (Table 13).

There was no statistically significant association between the increase in TnI levels and a history of smoking. (p = 0.06) (Table 14).

No statistically significant correlation observed between the elevation of TnI levels and a history of alcoholism. The probability value is 0.29 (Table 15).

Table 12: Comparison between Tn-I level and gender

Troponin I	*Gender*		*Total*
	F	*M*	
Normal	31	51	82
Raised	5	13	18
Total	36	64	100

$X^2 = 0.64$, p = 0.42, Not significant

Table 13: Comparison between Tn-I and chest pain

Troponin I	*Chest pain*		*Total*
	No	*Yes*	
Normal	71	11	82
Raised	15	3	18
Total	86	14	100

$X^2 = 0.13$, p = 0.72, Not significant

Table 14: Comparison between Tn-I and tobacco use

Troponin-I	*Tobacco*		*Total*
	No	*Yes*	
Normal	33	49	82
Raised	3	15	18
Total	36	64	100

$X^2 = 3.56$, p = 0.06, not significant

Table 15: Comparison between TnI and alcoholism

Troponin I	*Alcoholism*		*Total*
	No	*Yes*	
Normal	64	18	82
Raised	16	2	18
Total	80	20	100

$X^2 = 1.08$, p = 0.29, Not significant.

Table 16: Comparison between troponin-I and DM-2

Troponin I	*DM-2*		*Total*
	No	*Yes*	
Normal	57	25	82
Raised	8	10	18
Total	65	35	100

$X^2 = 4.08$, p = 0.043, Significant

Table 17: Comparison between Tn-I and hypertension

Troponin I	*Hypertension*		*Total*
	No	*Yes*	
Normal	43	39	82
Raised	6	12	18
Total	49	51	100

$X^2 = 2.16$, p = 0.14, Not Significant

Table 18: Comparison between TnI and dyslipidemia

Troponin I	*Dyslipidemia*		*Total*
	No	*Yes*	
Normal	17	65	82
Raised	5	13	18
Total	22	78	100

$X^2 = 0.43$, p = 0.52, Not Significant

Table 19: Comparison between TnI and outcome

Troponin I	*Outcome*		*Total*
	Expired	*Recovered*	
Normal	10	72	82
Raised	13	5	18
Total	23	77	100

$X^2 = 30.03$, $p < 0.001$, Significant

A significant correlation was observed between the occurrence of DM type 2 in AIS patients and the elevated levels of TnI (p = 0.043) (Table 16).

No statistically significant correlation was observed between the elevation of TnI levels and hypertension. The probability value is 0.42 (Table 17).

No significant association was observed between the elevation of TnI levels and dyslipidemia. The probability value, denoted as p, is equal to 0.52 (Table 18).

A strong correlation has been observed between mortality and elevated levels of TnI in AIS cases ($p < 0.001$) (Table 19).

In our study we found that , significant correlation was seen between the TnI levels and serum creatinine and Haemoglobin levels. The mean age of patients with normal TnI was 63.07 ± 14.58 years while those with raised TnI was 68.28 ± 13.25 years. There was no any correlation between age and rise in

TnI (p = 0.17). The mean total cholesterol (TC) of patients with normal TnI was 176.32 ± 49.50, while those with raised Trop I was 174.5 ± 46.13. There was no any correlation between the total cholesterol and rise in TnI (p = 0.88). Mean triglycerides of patients with normal TnI was 142.94 ± 70.49, while those with raised TnI was 132.33 ± 65.51. There was no any correlation between the triglycerides and rise in TnI (p = 0.56). Mean HDL of patients with normal TnI was 43.18 ± 11.06 while those with raised TnI was 41.56 ± 14.44. There was no any correlation between the HDL and rise in Trop I (p = 0.59). The mean LDL of patients with normal TnI was 119.37 ± 48.98, while those with raised TnI was 120.00 ± 66.73. There was no any correlation between the LDL and rise in Trop I (p = 0.96). The mean serum creatinine of patients with normal TnI was 1.18 ± 0.45 while for those with raised TnI was 1.61 ± 1.5. There was a significant correlation between the Serum creatinine and rise in TnI (p = 0.03). The mean hemoglobin of patients with normal TnI was 13.00 ± 2.54 while those with raised TnI was 11.67 ± 1.88. There was a significant correlation between the Serum creatinine and rise in TnI (p=0.04) (Table 20).

Table 20: "t" Test for correlation

	Troponin-I	*"n"*	*Mean*	*Standard deviation*	*p-value*
Age	Normal	82	63.07	14.58	0.17
	Raised	18	68.28	13.25	
TC	Normal	82	176.32	49.50	0.88
	Raised	18	174.50	46.13	
Triglycerides	Normal	82	142.94	70.49	0.56
	Raised	18	132.33	65.51	
HDL	Normal	82	43.18	11.06	0.59
	Raised	18	41.56	14.44	
LDL	Normal	82	119.37	48.98	0.96
	Raised	18	120.00	66.73	
S. Creatinine	Normal	82	1.18	0.45	0.03
	Raised	18	1.61	1.50	
Haemoglobin	Normal	82	13.00	2.54	0.04
	Raised	18	11.67	1.88	

Table 21: Comaprison with various other studies

Study	*Year*	*Trop I elevated (%)*	*Mortality (%)*	*Trop I – Mortality / morbidity outcome association*
Sandhu R et al[12]	2008	28	40	Significant $p < 0.001$
Scheitz JF et al[13]	2012	14	-	Significant (Morbidity) p = 0.04
B Hasirci et al [14]	2013	18.8	-	Significant (Morbidity) p = 0.001
Maliszewska Met al [15]	2013	16.5	25.7	Significant, p = 0.002
Thålin C et al [16]	2015	16	-	Higher 5 year mortality hazard Ratio = 1.90
Abdi S et al [17]	2015	17.6	-	Significant (Morbidity), p< 0.001
Ahn SH et al [18]	2017	9.8	21	Significant $p < 0.001$
VanHouten J etal [19]	2018	20.6	-	-
Current study	2019	18	23	Significant ($p < 0.001$)

DISCUSSION

In the present study, out of all total patients, about 64% were male by gender and 36% were female. This showed the male's predominance in having a stroke. Similar results were shown**,** where about 61.1% of study patients were male by gender.[9] While another study showed the opposite results, where 45% of study patients were male.[10] We also observed that about 58% of the study patients had right-sided weakness and 42% had left-sided weakness, indicating the involvement of the left side of the brain in AIS. Thus, in my present study, patients had more left-side brain involvement. Also, about 14% of study patients had associated chest pain as their complaint. In a similar study showed that "comparison of 3 groups of patients, cTnI levels were higher in ischemic stroke patients, especially with anterior circulation involvement, and the difference was significant (p = 0.04)".[7] Furthermore , we observed that TnI levels were elevated in AIS patients with anterior circulation compared to those with posterior circulation. This finding matches the results of another study that found a possible independent link between a high level of Tn after an ischemic stroke and a source of cardiac embolism. Serum troponin and hemoglobin levels were found to be statistically significant, with *p-values* of 0.03 and 0.04 for the normal and raised TnI levels, respectively. Age and lipid profile were not found to have a statistically significant relationship. As a result, it was suggested that patients with a raised TnI range may have low creatinine and hemoglobin levels. A study showed that age, NIHSS score, smoking history, history of heart failure or peripheral vascular disease, and serum creatinine were independent predictors of higher TnI (p 0.01),[11] which was consistent with our current study (Table 21).

CONCLUSION

Patients with acute ischemic stroke showed significant TnI levels, according to the current study's findings. Serum TnI levels were also greater in patients with AIS age, despite these levels being thought to be more selective for myocardial injury. TnI levels and the prognosis of patients with AIS were significantly correlated. Elevated concentrations of TnI in individuals experiencing acute stroke have been found to be correlated with unfavorable outcomes. The clinical impact of elevated serum TnI levels in stroke patients may be better understood through long-term follow-up and serial evaluation of serum TnI in a larger prospective study. As a consequence, the results of this study will enhance our knowledge of the connection between cardiac enzymes in patients with AIS as well as how they affect prognosis.

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