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Original Research Article

COMPARISON OF TROPONIN T LEVEL IN PATIENTS WITH ACUTE ISCHEMIC STROKE AND ACUTE SPONTANEOUS INTRACRANIAL HEMORRHAGE WITH ALL-CAUSE MORTALITY

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ABSTRACT

Background
This study was conducted to investigate the association between patients' levels of troponin T and acute spontaneous intracranial hemorrhage with all-cause mortality in acute ischemic stroke patients.

Methods
After receiving approval from the institutional ethics committee and signed informed consent from the study participants, a hospital-based observational study involving 100 patients who presented with acute ischemic and acute spontaneous hemorrhagic stroke to the Department of Internal Medicine, Madras Medical College, and Rajiv Gandhi Government General Hospital, Chennai, was carried out over a 6-month period.

Results
When troponin T level was compared to 1-week mortality following a stroke, it was shown that 85.7% of those who passed away within that time had an elevated troponin T. The p-value was equal to 0.000. It matters a lot. Therefore, there is unquestionably a relationship between a rise in troponin T and stroke mortality after one week.

Conclusion
A protein molecule called troponin T has been shown to increase not only in myocardial infarction but also in a number of other disorders. Stroke is one of these significant conditions. It is high in both intracerebral hemorrhage and acute ischemic stroke for a variety

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of reasons. The 1-week all-cause mortality in our study for both acute spontaneous intracerebral hemorrhage and acute ischemic stroke was substantially correlated with troponin T increase. Thus, in the case of stroke, troponin T is an independent predictor of all-cause death.

Keywords: Troponin T Level, Acute Ischemic Stroke, Acute Spontaneous Intracranial Hemorrhage, Mortality.

INTRODUCTION

An acute cerebrovascular accident, or stroke, manifests as an acutely focused neurological impairment. Since this is the second most prevalent cause of death worldwide, it is critical to comprehend the etiology, pathophysiology, complications, and indications and markers of worsening in order to reduce mortality and morbidity in these individuals.[1] Furthermore, it puts a great deal of strain on both the national and family economies. In a nation like India where males often head the household, a stroke puts a tremendous strain on the affected man, the family, and the caregiver because the patient needs long-term care, frequent medical attention, and rehabilitation services. It has a positive effect on the nation's gross domestic product. These patients undoubtedly have significant rates of mortality and case fatalities. The WHO (World Health Organization) estimates that if the patient survives at all, their disability adjusted life years will be 28,500,000 globally[2] and 6,398,000 in India. Stroke is no longer a disease exclusive to wealthy nations. Approximately two-thirds of the world's stroke cases occur in poorer nations. The true prevalence of stroke in India is unknown due to improperly managed stroke registries, wrongly categorized causes of death, and inadequate autopsy procedures for sudden, unexplained deaths. It will take years for appropriate governmental policies and regulations to be passed and properly enforced in order to rectify all of these falsehoods, as all of these hurdles are extremely tough to remove.

Regional Incidence and Prevalence of Stroke

Hemorrhagic and ischemic strokes are the two types of strokes. The cause of an ischemic stroke is a blockage in blood flow, which can be attributed to a thrombus or emboli. These can include cardiac emboli, artery-to-artery emoli, or paradoxical emboli. Another kind is cerebral venous thrombosis, which is defined as a cerebral hemorrhage or infarct brought on by cerebral venous thrombosis.[3] Subarachnoid hemorrhage and spontaneous intracerebral hemorrhage are the two forms of bleeding. When there is intracerebral hemorrhage, the blood vessel bleeds, compressing and reducing the blood flow to the surrounding regions. The main consequence of subarachnoid hemorrhage is cerebral vasospasm, which can result in infarctions. The bleeding occurs into the subarachnoid space, compressing the internal capsule and creating symptoms.

Ischemic Stroke

Stroke clinical signs include a sudden start of weakness in limbs, deviation of the mouth's angle, loss of sensation, loss of vision, and many more. A stroke doesn't hurt, and the patient may think that the symptoms are just clumsiness or have anosognosia, which is the inability to recognize the symptoms.[4] For all these reasons, the patient arrives at the doctor's late. If

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the patient is brought in within 3 or 4.5 hours after the onset of symptoms, thrombolysis has emerged as the preferred course of action. Several research suggest that it can be liberalized to as much as six hours.[5] Other more recent techniques include intravenous and intraarterial thrombolysis combined with endovascular treatments employing coil and stent retrievers. However, the primary settings for these more recent modalities' utilization are clinical trials and highly skilled stroke centers.[6] In 1998, the FAST acronym gained popularity in the UK with the goal of making stroke detection simple for the general population. Facial drooping, arm weakness, speech difficulties, and time or FAST for short-all emphasize how critical it is to get to the hospital as soon as possible.[7] Mostly for ischemic strokes, people should know that the symptoms can be reversed if they are treated with thrombolysis at the appropriate time. Because ischemic stroke is more prevalent than hemorrhagic stroke, it has received media attention. The primary therapeutic approach for bleeding is blood pressure reduction. Although it has not been recommended for ischemic stroke, an acute blood pressure decrease has been shown to be beneficial for patients with spontaneous intracerebral bleeding.

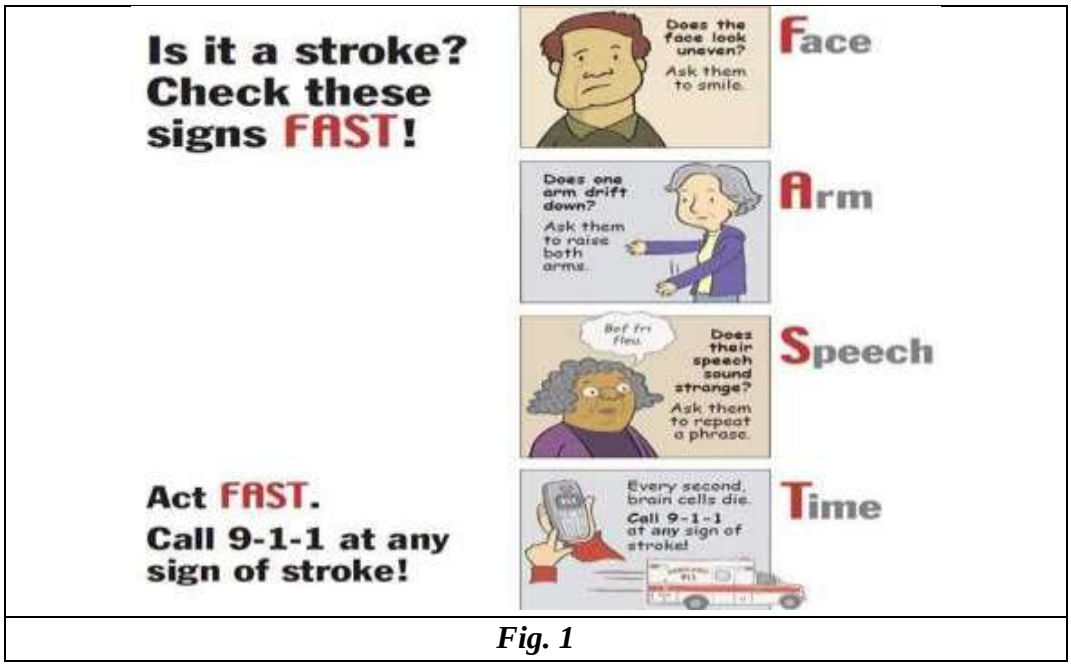

Fig. 1

Worsening cerebral edema, myocardial infarction, direct myocardial injury, arrhythmias, aspiration pneumonia, catheter-related infections, and skin infections are the leading causes of death in stroke cases. It's critical to understand which patients should be looked at for the aforementioned issues. Furthermore, numerous investigations have demonstrated an elevation in troponin T in the majority of these diseases.[8,9]

The insular cortex, which is located deep within the Sylvian fissure and is responsible for autonomic control of the body, is thought to be the cause of the increase in troponin. Damage to this insula causes an excessive release of catecholamines, which in turn causes myocytolysis, a direct injury to the cardiac myocytes. This is referred to as stroke-related sympatho-adrenal activity.

Three components make up the protein complex known as troponin, which exists in two different forms. Troponin T, I, and C are the three subunits. There are two types: one

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where they are attached to the actin protein in the muscle and another where they are free in the cytosol. If there is injury to them, they are liberated from the myocytes. It need not always be ischemia damage; mycytolysis, myocarditis, and stress brought on by a mismatch in supply and demand can all result in direct injury to the myocytes.

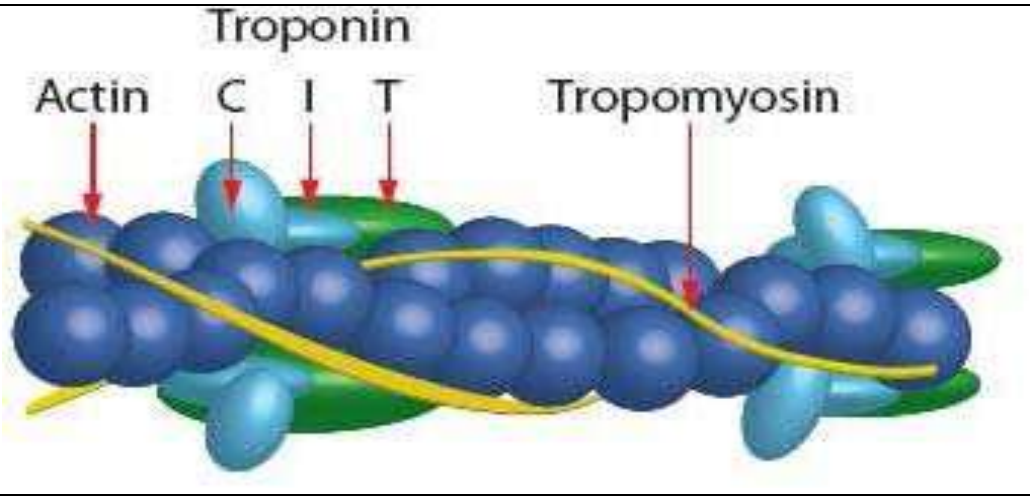

Fig. 2

AIMS AND OBJECTIVES

To study the relationship between patients' levels of troponin T and acute spontaneous intracranial hemorrhage with all-cause morality in cases of acute ischemic stroke.

MATERIALS & METHODS

After receiving approval from the institutional ethics committee and signed informed consent from the study participants, a hospital-based observational study involving 100 patients who presented with acute ischemic and acute spontaneous hemorrhagic stroke to the Department of Internal Medicine, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai, was carried out over a 6-month period.

Inclusion Criteria

- Patients with an acute ischemic stroke lasting for more than 24 hours.
- Acute spontaneous hemorrhagic patients

Exclusion Criteria

- Traumatic intracranial hemorrhage.
- Intracranial hemorrhage secondary to procedures, neoplasm, hemorrhagic transformation of massive infarct, cerebral venous thrombosis, anti-coagulation, arterio-venous malformation.
- Coronary artery disease.
- Rheumatic heart disease.
- Congestive cardiac failure.
- Post-cardiac arrest.

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- End-stage renal disease.
- Pericarditis.
- Myocarditis.
- Sepsis.
- Post-cardiac, thoracic and abdominal procedures.
- Post-envenomation.
- Connective tissue disorders.
- Under chemotherapy.
- Acute respiratory diseases.

Statistical Methods

The analysis was done using the statistical software SPSS 20.0.

RESULTS

			Troponin (pg/ml)		Total
			<14	>14	
DM	Yes	Count	28	19	47
		% within DM	59.6%	40.4%	100.0%
		% within Trop	47.5%	46.3%	47.0%
	No	Count	31	22	53
		% within DM	58.5%	41.5%	100.0%
		% within Trop	52.5%	53.7%	53.0%
Total		Count	59	41	100
		% within DM	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
Comparison of Diabetes Mellitus and Troponin T-Levels in Stroke					
			Troponin (pg/ml)		Total
			<14	>14	
SHT	Yes	Count	45	31	76
		% within SHT	59.2%	40.8%	100.0%
		% within Trop	76.3%	75.6%	76.0%
	No	Count	14	10	24
		% within SHT	58.3%	41.7%	100.0%
		% within Trop	23.7%	24.4%	24.0%
Total		Count	59	41	100
		% within SHT	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
Comparison of Systemic Hypertension and Troponin T-Level in Stroke					

Table 1

Forty-four percent of the study group's participants with diabetes mellitus had elevated troponin T levels. 46.3% of the patients with elevated troponin T levels had diabetes

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mellitus. The p-value was equal to 0.912. It is not important. Therefore, there is no relationship between diabetes mellitus and elevated troponin T in stroke cases.

In the study group, 40.8% of individuals with systemic hypertension had elevated troponin T levels. A total of 75.6% of patients with increased troponin T had systemic hypertension. The P-value was equal to 0.939. It is not important. Therefore, there was no relationship between elevated troponin T in stroke and systemic hypertension.

			Troponin (pg/ml)		Total
			<14	>14	
DM and SHT	Yes	Count	15	11	26
		% within DM and SHT	57.7%	42.3%	100.0%
		% within Trop	25.4%	26.8%	26.0%
	No	Count	44	30	74
		% within DM and SHT	59.5%	40.5%	100.0%
		% within Trop	74.6%	73.2%	74.0%
Total		Count	59	41	100
		% within DM and SHT	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%

Table 2: Comparison of Diabetes Mellitus and Systemic Hypertension with Troponin T-Level in Stroke

42.3% of the study group's participants with diabetes mellitus and systemic hypertension had elevated troponin T levels. 26.8% of individuals with high troponin T also had diabetes mellitus and systemic hypertension. The p-value was equal to 0.875. It is not important. Therefore, there was no relationship between diabetes mellitus, systemic hypertension and the rise of troponin T in stroke.

			Troponin (pg/ml)		Total
			<14	>14	
Ischemic stroke	Yes	Count	44	21	65
		% within Infarct	67.7%	32.3%	100.0%
		% within Trop	74.6%	51.2%	65.0%
	No	Count	15	20	35
		% within Infarct	42.9%	57.1%	100.0%
		% within Trop	25.4%	48.8%	35.0%
Total		Count	59	41	100
		% within Infarct	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
			Troponin (pg/ml)		Total
			<14	>14	
ICH	Yes	Count	15	20	35
		% within ICH	42.9%	57.1%	100.0%
		% within Trop	25.4%	48.8%	35.0%
	No	Count	44	21	65

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		% within ICH	67.7%	32.3%	100.0%
		% within Trop	74.6%	51.2%	65.0%
Total		Count	59	41	100
		% within ICH	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
Ischemic Stroke and Troponin T-Level					
			Troponin (pg/ml)		**Total**
			<14	**>14**	
ICH	Yes	Count	15	20	35
		% within ICH	42.9%	57.1%	100.0%
		% within Trop	25.4%	48.8%	35.0%
	No	Count	44	21	65
		% within ICH	67.7%	32.3%	100.0%
		% within Trop	74.6%	51.2%	65.0%
Total		Count	59	41	100
		% within ICH	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
Spontaneous Intracerebral Hemorrhage: Spontaneous Intracerebral Hemorrhage and Troponin T-Level					
Table 3					

The study group included 32.3% of ischemic stroke patients with elevated troponin T levels. 57.1% of the research group's participants who experienced spontaneous intracerebral hemorrhage had elevated troponin T levels.

			Troponin (pg/ml)		**Total**
			<14	**>14**	
Outcome	Living	Count	55	17	72
		% within Outcome	76.4%	23.6%	100.0%
		% within Trop	93.2%	41.5%	72.0%
	Dead	Count	4	24	28
		% within Outcome	14.3%	85.7%	100.0%
		% within Trop	6.8%	58.5%	28.0%
Total		Count	59	41	100
		% within Outcome	59.0%	41.0%	100.0%
		% within Trop	100.0%	100.0%	100.0%
Table 4: Comparison between Troponin T-Level and 1-Week Mortality in Stroke					

85.7% of the research group's stroke deaths within a week had elevated troponin T. The p-value was equal to 0.000. It matters a lot. Therefore, there is unquestionably a relationship between a rise in troponin T and stroke mortality after one week.

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DISCUSSION

Research has established the elevation of a protein complex known as troponin T in myocardial infarction. Increased catecholamines in stroke are expected to occur in a number of conditions, including worsening cerebral edema, sepsis, aspiration pneumonia, pulmonary embolism, and myocytolysis, which is a direct myocardial injury caused by elevated catecholamines in stroke caused by insular damage, particularly the right insula.

In our study, 28% of stroke victims passed away within a week. Of these, 85.7% showed elevated troponin levels. T.

Kerr et al. conducted a comprehensive review of 15 studies comparing high troponin in stroke patients and found that 18.1% of patients had elevated troponin, independently linked to an increased risk of death.[10]

In their retrospective investigation, Chung et al. separated intracerebral hemorrhage patients into two groups: those with normal troponin levels and those with high troponin levels. Group 1 mortality was 7.6%, whereas group 2 mortality was 28.6%. They came to the conclusion that elevated troponin was linked to an increase in in-hospital mortality and ought to be taken into consideration as a standard test in stroke patients who have been hospitalized.[11]

Twenty percent of the 222 ischemic stroke patients in the research by Barber et al. had increased troponin levels. Furthermore, due to its correlation with elevated creatinine, adrenaline, and age, high troponin was not a reliable indicator of death or dependence.[12] However, Hays et al. retrospective study of 235 ischemic stroke patients showed that troponin levels were a reliable indicator of in-hospital mortality in stroke, with death rates being higher in the 18% of these patients who had higher troponin levels.[13] In a study including 244 ischemic stroke patients, Jensen et al. reported that 10% of the patients had high troponin T, which indicated that troponin predicted the patient's probability of dying within two years.[14] In a prospective research conducted by Faiz et al., the level of high sensitivity troponin T was compared in 347 individuals who had an ischemic stroke. Researchers found that patients who did not survive had higher levels of it compared to those who did. Additionally, it had a separate correlation with death from all causes.[15]

In a five-year retrospective analysis involving 871 ischemic stroke patients, YC Su et al. examined the relationship between troponin level, stroke outcome and mortality. Using the NIHSS, modified Rankin scale, and Barthel index, it was shown that increased troponin was independently linked to death as well as a bad result..[16]

CONCLUSION

Studies have shown that a protein molecule known as troponin T increases not only in myocardial infarction but also in a number of other disorders. A stroke is one of these significant conditions. It is high in both intracerebral hemorrhage and acute ischemic stroke for a variety of reasons. Our study found a strong correlation between troponin T elevation and 1-week all-cause mortality in cases of acute spontaneous intracerebral hemorrhage and acute ischemic stroke. As a result, troponin T independently predicts all-cause death in stroke patients.

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REFERENCES

[1] Sacco RL, Kasner SE, Broderick JP, Caplan LR, Connors JJ, Culebras A, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2013;44(7):2064-89.

[2] Finger S, Boller F, Tyler KL. Handbook of clinical neurology. North-Holland Publishing Company 2010: p. 401

[3] https://en.wikipedia.org/wiki/Stroke

[4] http://www.who.int/mediacentre/factsheets/fs310/en/

[5] WHO World Health Statistics, 2012

[6] Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. J Stroke 2013;15(3):128.

[7] Feigin VL, Forouzanfar MH, Krishnamurthi R, Mensah GA, Connor M, Bennett DA, et al. Global and regional burden of stroke during 1990-2010: findings from the Global Burden of Disease Study 2010. Lancet 2014;383(9913):245–54.

[8] http://www.cdc.gov/stroke/facts.htm

[9] Brown RD, Whisnant JP, Sicks JD, O'Fallon WM, Wiebers DO. Stroke incidence, prevalence, and survival: secular trends in Rochester, Minnesota, through 1989. Stroke 1996;27(3):373–80.

[10] Kerr G, Ray G, Wu O, Stott DJ, Langhorne P. Elevated troponin after stroke: a systematic review. Cerebrovasc Dis 2009;28(3):220-6.

[11] Chung PW, Won YS, Kwon YJ, Choi CS, Kim BM. Initial troponin level as a predictor of prognosis in patients with intracerebral hemorrhage. J Korean Neurosurg Soc 2009;45(6):355-9.

[12] Barber M, Morton JJ, Macfarlane PW, Barlow N, Roditi G, Stott DJ. Elevated troponin levels are associated with sympathoadrenal activation in acute ischaemic stroke. Cerebrovasc Dis 2006;23(4):260-6.

[13] Hays A, Diringer MN. Elevated troponin levels are associated with higher mortality following intracerebral hemorrhage. Neurology 2006;66(9):1330-4.

[14] Jensen JK, Kristensen SR, Bak S, Atar D, Høilund-Carlsen PF, Mickley H. Frequency and significance of troponin T elevation in acute ischemic stroke. Am J Cardiol 2007;99(1):108-12.

[15] Faiz KW, Thommessen B, Einvik G, Omland T, Rønning OM. Prognostic value of high-sensitivity cardiac troponin T in acute ischemic stroke. J Stroke Cerebrovasc Dis 2014;23(2):241-8.

[16] Su Y, Huang K, Yang F, Lin S. Elevation of troponin I in acute ischemic stroke. Peer J 2016;4:e1866.