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The Prognostic Value of Elevated Cardiac Troponin-I in Short-term Outcome of Acute Ischemic Stroke

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Abstract **Background:** Heart disease and stroke rank the second most common cause of mortality worldwide and the third most common in more developed countries. Elevated levels of serum troponin are found in 10-34% of patients with acute stroke. In addition, elevated cardiac troponin (cTnT) or cardiac troponin I (cTn-I) levels have been regarded as prognostic biomarkers of poor outcome and higher in-hospital mortality rates in acute ischemic stroke. **Aim of the work:** The aim of this study was to evaluate the role of cardiac troponin-I (cTn-I) as a predictive biomarker of both poor short-term outcome and in-hospital mortality in acute ischemic stroke patients. **Methods:** This prospective cohort study included 74 patients (30 males and 44 females) presented with acute ischemic stroke from March 2016 to December 2016. Data included clinical assessment involving detailed history taking, general examination, thorough neurological examination, laboratory data including measurement of serum level of (cTn-I) on admission, assessment of stroke severity using National Institute of Health Stroke Scale(NIHSS) within the first 48 hours of stroke onset. Stroke severity and functional outcome were assessed 2 months from stroke onset using (NIHSS) and modified Rankin scale (mRS). **Results:** Patients with elevated cTn-I level were older(mean age was 67.92(±12.77) Vs. 63.2(±13.24) years than in those with normal cTn-I level with no significant statistical difference, were suffering more from diabetes and TIA, having higher mean scores of NIHSS on admission (18.7±8.14 Vs 13.85±7.66 respectively, $p<0.05$). poor functional outcome as assessed by mRS was statistically significantly more in patients with elevated cTn-I level (42.9%) than in patients with normal level (38.3%), $p<0.05$.Also major neurological improvement as assessed by NIHSS was significantly less common in patients with elevated cTn-I level (7.1%) than in patients with normal cTn-I (45.0%), $p<0.05$. Mortality rate was also statistically higher in patients with elevated cTn-I than in patients with normal level (16.7%), $p<0.05$. Insular brain lesion was statistically significant more in ischemic stroke patients with elevated cTn-I level than in those with normal level(35.7% Vs 5.0 %, $p<0.05$). multivariate logistic regression analysis of factors predicting poor functional outcome including death in the study patient revealed that both gender [OR(95%CI) 0.142(0.024-0.821), P=0.029], diabetes [OR(95%CI) 0.151 (0.030-0.749), P=0.021], admission NIHSS score >12 [19.52 (9.59-39.73), p=0.0001] and elevated cTn-I level > 0.01ug/l [19.42(1.293-293.276), p=0.035] were significant predictors of poor outcome and in-hospital mortality. **Conclusion:** This study reached to a conclusion that the short-term outcome is less favorable and the stroke is more severe in ischemic stroke patients with elevated serum level of cTn-I than in those with normal level, making it a reliable prognostic predictor of both poor stroke outcome and high in-hospital mortality rates.

Keywords: *acute ischemic stroke, Cardiac troponin-I, short-term functional outcome*

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1. Introduction

Stroke is defined as rapidly developing clinical symptoms and/or signs of focal, and global loss of brain function, with symptoms lasting more than 24 hours or leading to death, with no apparent cause other than that of vascular origin [1]. It is increasingly becoming a major health problem and considered a major cause of long term disability as well [2]. Heart disease and stroke rank the second most common cause of mortality worldwide and the third most common in more developed countries. Both cerebrovascular and coronary heart diseases share common risk factors. Also cardiac mortality represents the second most common cause of death among acute stroke patients , second to neurologic deaths which occur as a direct result of the incident stroke [3]. Approximately 87% of strokes are ischemic in nature with subtypes based largely on pathophysiology. Various schemes have been developed to classify subtypes of ischemic stroke, the most important is the Trial of Org 10172 in Acute Stroke Treatment (TOAST) which classified ischemic stroke in to large artery atherothrombosis, cardioembolism, lacunar, stroke of other defined etiology and stroke of other undetermined etiology [4,5].

Acute ischemic cerebrovascular events can induce various myocardial changes with pathological proof of myocytolysis and electrophysiologic proof of cardiac conduction abnormalities [6]. Cardiac troponins are considered important risk and prognostic biomarker of cardiac diseases especially ischemic heart disease (IHD) as they are routinely assessed in the setting of IHD, moreover, it has been reported that elevated serum troponin levels are linked to poor outcome of cerebrovascular diseases like ischemic stroke and intracerebral hemorrhage [7,8]. Previous researches have revealed an increased prevalence of elevated cardiac troponin T (cTnT) or I (cTn-I) levels in acute stroke population .Elevated levels of serum troponin are found in 10-34% of patients with acute stroke .In addition, elevated cTnT or cTn-I levels have been linked to poor stroke outcome, increased cardiovascular complications and higher in-hospital mortality rates [7,9,10]. In explanation for elevated concentrations of serum(cTn -I) levels in the setting of acute phase of ischemic stroke, Etgen et al. [11] reported that the primary myocardial damage with secondary cardioembolic cerebral ischemia or primary cerebral ischemia, with secondary myocardial injury attributable to central activation of the sympatho-adrenal system are accepted reasons for such elevation. Many cardiovascular risk factors such as hypertension, diabetes mellitus, cardiac diseases and hyperlipidemia are well know co-morbidities of stroke, and if not well controlled, they are correlated with worsening of clinical severity and poor outcome of stroke, so in this study, we investigated the impact of certain clinical features and laboratory biomarkers including (cTn-I) on the clinical severity of stroke and their role as predictors of poor outcome.

2. Aim of Work

The aim of this study was to evaluate the role of cardiac troponin-I((cTn-I) as a predictive biomarker of both poor short-term outcome and in-hospital mortality in acute ischemic stroke patients.

3. Patients and Methods

This follow up prospective cohort study was conducted in intensive care and stroke units, Neurology Department, Zagazig University Hospitals, during the period from march 2016 to December 2016. Seventy four patients(30 males and 44 females) with mean age (±SD) of 64.13± (13.2) years old who fulfilled the inclusion and exclusion criteria were diagnosed as having ischemic stroke and enrolled in this follow-up cohort study. These patients were presenting with a clinical diagnosis of acute ischemic stroke with suspected symptom onset within the preceding 48 hours.

All patients have fulfilled the following inclusion criteria:

Patients who presented with acute ischemic stroke with symptom onset within preceding 48 hours, CT scan of the brain showed evidence of early imaging signs of cerebral infarct or recent infarction.

Patients who were excluded from the study those who had non-ischemic etiology such as hemorrhagic stroke (intracerebral hemorrhage or subarachnoid hemorrhage), recent ischemic heart disease, defined as acute myocardial infarction (MI) within 2 weeks prior to and up to 3 days after stroke onset, newly developed pathologic Q waves on admission ECG , previous coronary angioplasty or coronary bypass surgery, other heart diseases and debilitating diseases with the possibility of serum troponin I elevation, such as congestive heart failure, valvular heart disease and renal impairment.

All patients in the study were assessed according to the following scheme of clinical evaluation and investigations:

I- Clinical assessment: which included detailed history taking, general examination, thorough neurological examination including assessment of stroke severity using National Institute of Health Stroke Scale(NIHSS) [12] within the first 48 hours of stroke onset. clinical deterioration was defined when the patients showed increase of two or more points on NIHSS.

-Evaluation of short-term functional outcome of stroke patients after 60 days of onset using NIHSS and modified Rankin Scale(mRS). The mRS consists of 6 grades, from 0 to 5, with the best score 0 corresponding to no symptoms and the worse score 5 corresponding to severe disability. Death can be rated 6 in the mRS .A score on the mRS>2 is defined as unfavourable or poor outcome [13]. All causes of deaths during hospitalization were registered as in-hospital mortality.

II-Investigations:

A- Laboratory investigations:

(1) Full routine laboratory investigations at admission including Complete blood count (CBC), liver and kidney function tests(LFT, KFT), random plasma glucose level on admission followed by: Fasting and 2 hours post-prandial plasma glucose assessment in diabetic patients, lipid profile including total cholesterol, triglycerides, high density lipoprotein (HDL), and low density lipoprotein (LDL), coagulation profile: Prothrombin time (PT), prothrombin concentration (PC), international normalized ratio (INR) and activated partial thromboplastin time (aPTT).

(2) Quantitative measurement of Serum cardiac troponin I level within 12-72 hours of stroke onset using standard sandwich enzyme-linked immune-sorbent assay technology (ELISA) following manufacturer's instructions (ALPCO, 26G Keewaydin Drive, Salem NH03079, USA).

Blood sampling and determination of Serum troponin I level.

- Specimen Collection:

Three ml of venous blood were drawn from all ischemic stroke patients using standard venipuncture techniques, within 12-72 h of stroke onset.Blood samples were left to clot for 4 hours at room temperature, then centrifuged to obtain the serum which was stored frozen at (-20C). The levels of troponin I were measured by the use of standard sandwich enzyme-linked immune-sorbent assay technology.

- Reference values: <0.01µg/l.

B- Cardiac investigation: 12-leads electrocardiogram (ECG): was done to all recent ischemic stroke patients to determine ECG changes in patients with unstable angina or recent myocardial infarction such as newly developed pathological Q wave and ST segment elevation. Transthoracic echocardiography also was done when indicated.

C- Radiological investigations: computed tomography (CT) scan of the brain was done by using Philips (Tomoscan 350) with 4.8 scanning time and 512 x 512

matrix size. Axial scans were obtained with the patients supine, the slice thickness was 9 mm. All stroke patients enrolled in this study were subjected to CT scan of the brain within the first 48 hours after stroke onset to exclude patients with stroke mimics or intracerebral hemorrhage and to diagnose site and size of cerebral infarction. The size of the brain infarct was calculated according to the formula (0.5 x a x b x c) where (a and b are the largest perpendicular diameters measured on CT and c is the slice thickness which is 9mm) [14].

Ethical consideration: A written informed consent was obtained from every patient or his/her relative to be included in the study. This study was approved by the institute research board of Faculty Of Medicine, Zagazig University.

4. Statistical Analysis

The obtained data were tabulated and analyzed using Statistical Package of Social Science (SPSS version, 22) [15]. Continuous variables were expressed as mean ± SD (standard deviation) and median(range) .The means were compared with independent student's t-test. Categorial variables were compared using the chi-square test. Odds ratio (ORs) and confidence interval (CIs) were also calculated in a logistic regression model to assess independent factors that were significantly associated with poor outcome among ischemic stroke patients. A difference was considered statistically significant if P-value was less than <0.05.

5. Results

This follow up prospective cohort study was conducted in intensive care and stroke units, Neurology Department, Zagazig University Hospitals, during the period from march 2016 to December 2016. Seventy four patients(30 males and 44 females) with mean age (±SD) of 64.13± (13.2) years old who fulfilled the inclusion and exclusion criteria were diagnosed as having ischemic stroke and enrolled in this study . These patients were presenting with a clinical diagnosis of acute ischemic stroke with suspected symptom onset within the preceding 48 hours.

Table 1 shows the base line demographic and clinical characteristics of ischemic stroke patients regarding the mean age (±SD) of patients with elevated cTn-I level was 67.92(±12.77) Vs 63.2(±13.24) years in those with normal cTn-I level with no significant statistical difference. Regarding sex distribution between the two groups, there wasn't statistically significant difference regarding sex between patients with elevated and normal cTn-I. Also hypertension, AF and dyslipidemia and smoking showed no statistical significant difference between ischemic stroke patients with elevated and normal cTn-I level. Ischemic stroke patients with diabetes and TIA were statistically significant more in patients with elevated cTn-I than in those with normal cTn-I level (85.7% Vs 55.0% and 7.1% Vs 0.0% respectively), p<0.05). Mean WBC was also significantly higher in patients with elevated cTn-I than in those with normal level (12.36±4.34 Vs 9.26 ±4.05, p<0.05).

Table 1. Base line demographic data and clinical characteristics of ischemic stroke patients regarding cardiac Troponin I level

Data \ Cardiac troponin I	Normal cardiac Troponin I (n = 60)	Elevated cardiac Troponin I (>0.01.ug/L) (n = 14)	P
- Mean age (±SD)	63.25(±13.24)	67.92(±12.72)	1.198
- Sex			
Male n(%)	24(40.5%)	6(42.9%)	NS
Female n(%)	36(60%)	8(57.1%)	
- Hypertension n (%)	33 (55.0%)	6 (42.9%)	NS
- Diabetes mellitus n(%)	33 (55.0%)	12 (85.7%)	<0.05*
- Atrial fibrillation (AF) n(%)	10 (16.7%)	5 (35.7%)	NS
- Smoking n (%)	11 (18.3%)	5 (35.7%)	NS
- Dyslipidemia n(%)	28 (46.6%)	8 (57.1%)	NS
- TIA n(%)	0 (0.0%)	1 (7.1%)	<0.05*
- Mean WBC (±SD)	9.26(±4.05)	12.36(±4.05)	<0.05*

n: number.
TIA: Transient ischemic attack. ug/L: microgram per liter.
*Significant P<0.05 SD: Standard deviation.
WBC: White blood cell. NS: Non significant.

Table 2. Comparison between ischemic stroke patients with elevated and those with normal cardiac Troponin I regarding mean scores of admission NIHSS

NIHSS \ Ischemic stroke patients	Elevated cardiac Troponin I (n = 14)	Normal cardiac Troponin I (n = 60)	P
- Admission mean score of NIHSS (±SD)	18.71(±8.14)	13.85(±7.66)	NS

n: number. NS: Non significant.
NIHSS: National Institute of Health Stroke Scale.

Table 2 shows comparison between ischemic stroke patients with elevated cTn-I level and those with normal cTn-I level regarding the mean score of admission NIHSS where mean score of admission NIHSS were statistically significant higher in patients with elevated cTn-I level than in those with normal level (18.7±8.14 Vs 13.85±7.66 respectively, p<0.05).

Table 3 shows functional outcome of ischemic stroke patients with elevated and normal cardiac troponin- I after two months of stroke onset in which poor functional outcome as assessed by mRS (unfavorable outcome was defined as mRS>2) was statistically significantly more in patients with elevated cTn- I level (42.9%) than in patients with normal level (38.3%), p<0.05. Also major neurological improvement as assessed using NIHSS (major neurological improvement was determined to be 8-points improvement in NIHSS or NIHSS score of 0 or 1) was significantly less common in patients with elevated cTn-I level (7.1%)than in patients with normal cTn-I (45.0%), p<0.05. Mortality rate was also statistically higher in patients with elevated cTn-I (50%) than in patients with normal level (16.7%), p<0.05.

Table 4 shows the relationship between serum cTn- I level and anatomical brain regions involved in which insular lesion was statistically significant more in ischemic stroke patients with elevated cTn- I level than in those with normal level (35.7% Vs 5.0 %, p<0.05), while other regions of the brain showed no statistically significant difference between the two groups.

Table 5 shows the relation between anatomical brain regions involved and functional outcome among the ischemic stroke patients after 2 months of the stroke onset in which unfavorable outcome as assessed by mRS was significantly more in insular region than in other anatomical brain regions (22.7% Vs. 33.3% respectively, p<0.05), also major neurological improvement on NIHSS was significantly less in insular lesion than in other brain regions (9.1% Vs. 42.9% respectively, p<0.05).

Table 6 shows multivariate regression analysis of factors predicting poor functional outcome including death in the study patients which revealed that both gender [OR (95% CI) 0.142 (0.024-0.821), P=0.029], diabetes [OR (95%CI) 0.151 (0.030-0.749), P=0.021], admission NIHSS score >12 [19.52 (9.59-39.73), p=0.0001] and elevated cTn-I level > 0.01ug/l [19.42 (1.293-293.276), p=0.035] were significant predictors of poor outcome and in-hospital mortality.

Table 3. Functional outcome of ischemic stroke patients with elevated and normal cardiac Troponin I after 2 months of stroke onset

Cardiac troponin level / Functional outcome	Elevated cardiac Troponin I (n = 14)	Normal cardiac Troponin I (n = 60)	P
- Major neurological improvement on (NIHSS) n(%)	1 (7.1%)	27 (45.0%)	
- Unfavorable outcome (mRS>2) n(%)	6 (42.9%)	23 (38.3%)	<0.05*
- Mortality n(%)	7 (50%)	10 (16.7%)	

n: number.
NIHSS: National Institute of Health Stroke Scale.
mRS: Modified Rankin scale.
*: Significant P<0.05.

Table 4. Relation between serum level of cardiac Troponin I and anatomical brain regions involved among ischemic stroke patients

Cardiac troponin I / Brain region	Elevated cardiac Troponin I (n = 14)	Normal cardiac Troponin I (n = 60)	P
- Basal ganglia n(%)	3 (21.4%)	16 (26.7%)	NS
- Brain stem n(%)	0 (0.0%)	3 (5.0%)	NS
- Frontal n(%)	0 (0.0%)	1 (1.7%)	NS
- Parietal n(%)	3 (21.4%)	21 (35.0%)	NS
- Insular n(%)	5 (35.7%)	6(10.0%)	<0.05*
- Temporal n(%)	0 (0.0%)	3 (5.0%)	NS

n: number.
*: Significant P<0.05
NS: Non significant

Table 5. Relation between anatomical brain regions involved and functional outcome in acute ischemic stroke patients after 2 months of stroke onset

Brain region / Functional outcome	Insular (n = 11)	Other brain regions (n=63)	P
- Neurological improvement (NIHSS) n(%)	1 (9.1%)	27 (42.9%)	
- Unfavorable outcome (mRS) n(%)	8 (72.7%)	21 (33.3%)	<0.05*
- Mortality n(%)	2 (18.2%)	15 (23.9%)	

n: number.
NIHSS: National Institute of Health Stroke Scale.
mRS: Modified Rankin scale.
*: Significant P<0.05.

Table 6. Logistic regression model of factors predicting poor functional outcome including death in our ischemic stroke patients

Clinical factors	Poor outcome (mRS>2) and death	
	OR (95% CI)	P-value
- Gender	0.142 (0.024-0.821)	0.029*
- Hypertension	3.708 (0.791-17.932)	0.080
- Diabetes	0.151 (0.030-0.749)	0.021*
- AF	1.232 (0.235-6.454)	0.805
- Dyslipidemia	0.908 (0.257-3.471)	0.889
- NIHSS (admission score ≥12)	19.52 (9.59-39.73)	<0.0001*
- Cardiac troponin I (>0.01 ug/L)	19.421 (1.293-293.276)	0.035*
- Insular region	6.172 (0.417-91.397)	0.186

mRS: Modified Rankin scale.
NIHSS: National Institute of Health Stroke Scale.
AF: Atrial fibrillation, *: Significant P<0.05
OR: Odds ratio, CI: Confidence interval.

6. Discussion

One of the most frequent etiologies of Stroke is increasingly becoming a major health problem and considered a major cause of long term disability as well [2]. Heart disease and stroke rank the second most common cause of mortality worldwide and the third most common in more developed countries [3]. Cardiac troponin I being highly sensitive and specific marker for detection of myocardial necrosis may be also elevated in patients with ischemic stroke [16]. Elevated cTn-I levels have been linked to poor stroke outcome, increased cardiovascular complications and higher in-hospital mortality rates [9,10]. This study was conducted to assess the prognostic role of cTn-I in acute ischemic stroke. In our study, elevated serum level of cTn-I>0.01ug/L was observed in 18.91% of recent ischemic stroke patients who do not have evidence of myocardial infarction, valvular heart diseases or other cause of elevated troponin. Our results were in accordance to those of Di Angelantonio et al. [6] and Amin et al. [7] who reported nearly Similar percentages of elevated cardiac troponin-I level in acute ischemic stroke patients (16-20%). In our study, patients with elevated cTn-I were older than those with normal level but without statistically significant difference. In contrary to our results, several studies found that elevated serum troponin level was significantly more prevalent among older age groups [8,17,18]. The results of our study also showed that there were no statistically significant difference between ischemic stroke patients with elevated cardiac troponin I and those with normal level regarding sex, This was in agreement with Di Angelantonio et al. [7], Scheitz et al. [8] and Kral et al. [18] who reported that gender had no effect on serum troponin I level. In the current study, we found no significant association between elevated cardiac troponin I and hypertension, a finding which was in accordance with results of previous studies done by Di Angelantonio et al. [7], Jensen et al. [8], Scheitz et al. [17] and Kral et al. [18] who found no significant association between elevated cTn-I and hypertension. On the other hand, Amin et al. [6] reported highly significant relation between elevated cardiac troponin and hypertension. Regarding the relationship between dyslipidemia and elevated cardiac troponin I level, we found no statistical significant difference between ischemic stroke patients with elevated cardiac troponin I (57.1%) and patients with normal level (43.3%). In contrary to our finding, Amin et al. [6] found highly significant association between elevated cardiac troponin I and dyslipidemia. However, Scheitz et al. [8] revealed that dyslipidemia was found to be negatively associated with troponin elevation. Possible causes of this observation are that patients with known dyslipidemia are likely to receive statins, which have shown to exert protective effects on arteriosclerotic plaques This might reduce the vulnerability of pre-existing coronary plaques as indicated by elevated cardiac troponin [19].

The results of our study showed non-significant relation between atrial fibrillation and elevated cardiac troponin I. This finding was similar to results of Di Angelantonio et al [7]. On the other hand, Scheitz et al. [8] and Kral et al. [18] found that patients with elevated troponin show a high prevalence of AF of highly significant value. This may be explained by strain exerted on the myocardium as a result of AF.

In the present study, diabetis mellitus was significantly found to be more prevalent in ischemic stroke patients with elevated cTn-I than in those with normal level (85.7% Vs 55% respectively, p <05). This was in agreement with Amin et al [6] who found that diabetes was more prevalent among patients with elevated cTn-I than in those with normal level which may probably reflect the high cardiovascular burden of patients with diabetes. On contrary to our results, Amin et al. [6], Di Angelantonio et al. [7], Jensen et al. [17], Kral et al. [18] found no statistically significant relation between diabetes and troponin elevation.

In the current study, a statistically significant high mean white blood cells (WBCs) count was reported in stroke patients with elevated cardiac troponin I level, a finding which was compatible with the results of Di Angelantonio et al. [7] who found significant association between WBCs count and elevated troponin. Many studies found that elevated WBCs count in acute stroke was reported to be a significant predictor of initial stroke severity, poor outcome and disability as well [21,22]. On the other hand, Farhard et al. [23] found no significant association between WBCs count and in-hospital mortality in patients with ischemic stroke. In explanation for that significant association, Agwell et al. [24] in their study reported high prevalence of elevated serum cardiac troponin in 58% of

stroke patients treated in intensive care unit for sepsis and who are most likely to have hypotension, septic shock, respiratory failure, and left ventricular systolic dysfunction.

On assessing stroke severity on admission using NIHSS, no statistically significant difference in mean scores of NIHSS between patients with elevated cTn-I level and those with normal level. When functional outcome was assessed in ischemic stroke patients, major neurological improvement on NIHSS was reported in 7.1% of patients with elevated cTn- I level versus 45% in patients with normal cTn- I level and unfavorable outcome using mRS was significantly more in patients with elevated cTn- I level than in those with normal cTn- I level [42.9% Vs. 38.3%, $p<0.05$], also mortality was significantly found to be more in patients with elevated cTn- I level than in those with normal cTn- I level [50% Vs 16.7%, respectively, $p<0.05$]. These findings were in agreement with results of studies done by Di Angelantonio et al. [7], James et al. [25] and Barber et al. [26] who found significant association between elevated troponin-I and poor functional outcome. The same result was documented by Faiz et al. [16] who found significant association between cardiac troponin elevation and increased long-term mortality in ischemic stroke patients. On the other hand, Trooyen et al. [27] in their study did not find significant association between both stroke severity and mortality and elevated cTn-I level in ischemic stroke patients.

Insular cortex is regarded to play an important role in controlling the autonomic nervous system particularly right-sided insular lesions which have been linked to disturbance of autonomic balance [28]. In the present study, on analyzing the relationship between cTn-I and anatomical brain region involved, the insular cortex was reported to be significantly involved in patients with elevated cTn-I level than in those with normal level [35.7% Vs. 10% respectively, $p<0.05$]. Also, patients with insular infarction showed less favorable outcome and more disability. Using NIHSS, major neurological improvement was significantly lower (9.1%) among patients with insular infarction than in those with infarction involving other brain areas (72.7%), $p <0.05$. Also poor outcome as assessed by mRS, was significantly worse in patients with insular infarction (72.7%) than in those with infarction of other brain regions (33.3%), $p<0.05$. Our results were in agreement with those of Song et al. [20] and Ghali et al. [29] who found that infarction of insular cortex was significantly more in patients with elevated serum cTn-I and those patients had more severe stroke and worse outcome**.** In our study, on doing logistic regression analysis to study factors that are independently associated with poor functional outcome including mortality, our results showed that both sex, diabetes, elevated caradiac troponin and admission mean NIHSS score>12 were independently associated with poor functional outcomeand mortality .so they were considered as markers of poor outcome. These findings were in accordance to results of several studies which demonstrated that Elevated cTn-I and NIHSS score were independently predicting poor outcome in ischemic stroke patients [7,16,17]. On contrary to our findings, Barber et al. [26] in their study in ischemic stroke patients, showed that elevated cTn -I level was not an independent predictor of neither death or dependency at 30 days.

7. Conclusion

The short-term outcome is less favorable and the stroke is more severe in stroke patients with elevated serum cTn-I than in those with normal serum cTn-I levels, making cTn-I a strong independent prognostic biomarker of both poor short-term functional outcome and high in-hospital mortality .Carful cardiac evaluation in acute ischemic stroke is warranted for early detection of possible cardiac disorders with abnormal troponin level for better secondary prevention and improving functional outcome after stroke.

Study limitations

The study had some limitations as small sample size.

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