Zagazig University Medical Journal
www.zumj.journals.ekb.eg
https://dx.doi.org/10.21608/zumj.2021.86908.2290 Volume 29, Issue 2, Mars2023, Page (559-565)
Manuscript ID ZUMJ-2107-2290 (R1)
DOI **10.21608/zumj.2021.86908.2290**

ORIGINAL ARTICLE

Cardiac Troponin T: an Indicator of Severity in Patients with Primary Intracerebral Hemorrhage.

Arwa Magdi Ragab[1]; Mahmoud Elsaid Alebiary[2]; Wael Mahmoud Elsayed[3]; Mohammed Hanafy Aly Ghonemy[4]

Neurology Department, Faculty of Medicine, Zagazig University, Zagaig, Egypt.

Corresponding author
Arwa Magdi Ragab
E-mail:
arwamagdi@rocketmail.com

Submit Date 2021-07-19
Revise Date 2021-07-23
Accept Date 2021-07-27

ABSTRACT

Background: Primary intracerebral hemorrhage is one of the most common forms of stroke producing major disability. Cardiac troponin T is used as an indicator of severity in patients with primary intracerebral hemorrhage. The association between cardiac troponin T and severity of intracerebral hemorrhage has been infrequently studied.

Aim: to identify the relationship between cardiac troponin T and severity of the clinical condition in patients with primary intracerebral hemorrhage measured by National Institutes of Health Stroke Scale (NIHSS).

Methods: 60 patients were recruited from Neurology department Intensive care and Stroke units, Zagazig University Hospital with first time ever primary intracerebral hemorrhage during the period from October 2020 till July 2021. Cardiac troponin T was measured upon admission. Severity was assessed upon admission by NIHSS.

Results: 35 (58.3%) patients had moderate score (5-15) upon admission compared to 15 (25%) patients with extremely severe condition (21-42), whereas 7 (11.7%) patients had severe condition and 3 patients (5%) had minor condition.

Conclusions: Cardiac troponin T level is a valuable biomarker correlated with severity of intracerebral hemorrhage.

Keywords: Intracerebral hemorrhage, Cardiac troponin T, Severity, National Institute of Health and Stroke.

INTRODUCTION

Spontaneous Intracerebral Hemorrhage (SICH) is the second most common cause of stroke and accounts for 7.5 − 30% of all strokes. Hemorrhagic stroke is generally associated with higher morbidity and mortality rates than ischemic stroke **[1].**There are several biomarkers which are strongly associated with the severity of intracerebral hemorrhage such as the inflammatory biomarkers including C- reactive protein (CRP), vascular endothelial growth factor (VEGF) and tumor necrosis factor (TNF) **[2].**

Recently, elevation of cardiac troponin has been reported to occur in ICH patients along with only 1.2 % of them died of cardiac causes **[3].**

In NIHSS, Patients are classified to have minor condition when having a score from 0 to 4, moderate condition when having a score from 5 to 15, moderate to severe condition when having a score from 16 to 20 and extremely severe condition when having a score from 21 to 42 **[7].**

METHODS

After considering the ethical rules according to the Code of Ethics of the World Medical Association (Declaration of Helsinki), written informed consent was obtained from all participants in this study approved by the research ethical committee of Faculty of Medicine, Zagazig University. 60 patients were recruited from Intensive care and Stroke units during the period from October 2020 till July 2021 fulfilling the following inclusion criteria: age above 18 years, patients with CT brain showing spontaneous ICH admitted within 24 hours after the onset of the qualifying event. The following were considered as exclusion criteria: suspicion or documented history of a bleeding disorder, history of head trauma, receiving anticoagulant drugs before the onset of ICH, a documented and discovered arterio-venous (A-V) malformation; aneurysm or cerebral neoplasm as the underlying cause of intracerebral hemorrhage, hemorrhagic transformation of cerebral infarct, a recent ischemic heart diseases, defined as previous myocardial infarction (MI) within 2 weeks prior to and up to 3 days after onset of hemorrhagic stroke,

symptoms suggestive of acute MI or unstable angina before admission, previous coronary surgery, other heart diseases and debilitating diseases with the possibility of serum cardiac troponin T elevation, such as congestive heart failure, valvular heart disease (VHD) and renal impairment.

All patients were subjected to the following:

(A) Clinical assessment including detailed medical history, full general examination, and thorough neurological examination.

National Institutes of Health Stroke Scale (NIHSS) for initial evaluation of intracerebral hemorrhage severity, within the first 24 hours of stroke onset (at admission). It is a 15-item neurologic examination stroke scale used to evaluate the effect of acute ischemic stroke on the levels of consciousness, language, neglect, visual-field loss, extraocular movement, motor strength, ataxia, dysarthria, and sensory loss that provides a quantitative measure of stroke related neurologic deficit**.** Patients are classified to have minor condition from 0 to 4, moderate condition from 5 to 15, moderate to severe condition from 16 to 20 and extremely severe condition with a score from 21 to 42 **[7].**

(B) Investigations:

I) Laboratory investigations:

- **Full routine laboratory investigations at admission including** complete blood count (CBC), liver function tests (LFT), kidney function tests (KFT), urine analysis, random plasma glucose level on admission followed by fasting and 2 hours post-prandial plasma glucose assessment in diabetes, lipid profile, coagulation profile.
- **Special laboratory investigations**: **Serum troponin T level** was measured within 12 - 72 h of stroke with reference values: < 100ng/l

II) Radiological investigations:

CT scan of the brain: within the first 24 hours from the onset of symptoms with stressing on the site, size of hematoma, surrounding edema and intraventricular extension. Volume of hematoma was assessed using the following equation ABC/2 where A is the maximum diameter, B maximum dimension perpendicular to A and C is the approximate number of slices with hemorrhage multiplied by slice thickness **[8].**

III) 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.

Statistical analysis

Statistical analysis was done using Statistical Package for the Social Science (SPSS) software version 25**.** Data was presented in tables and figures. Quantitative data was presented as mean, median and interquartile range. Qualitative data was presented as frequencies and proportions. Kolmogorov-Smirnov and Levene tests were used to determine the distribution characteristics of variables and variance homogeneity. Pearson's chi square (χ^2) test and fisher's exact test were used to analyze qualitative data as appropriate. Freidman test (F) was used to analyze dependent continuous data. Student t test (T) and Mann Whitney test (MW) was used to analyze continuous data between two groups as appropriate. Kruskal-Wallis H (KW) tests were used to analyze continuous data between more than two groups.

Spearman's correlation coefficient (r) was used to test correlation between neutrophil lymphocyte ratio and continuous variables. Binary logistic regression analysis of the predictors of the outcome of ischemic stroke was done to test independent variables and exclude confounders. P-value of ≤0.05 was accepted as statistically significant.

RESULTS

This prospective cohort study was conducted during the period between October 2020 and July 2021 on 60 patients with first time ever diagnosed primary intracerebral hemorrhage admitted to Neurology department intensive care unit and stroke unit within 24 hours of the onset of the symptoms with performing CT brain, cardiac troponin T level and full routine lab showing that mean age 60.1 ± 9.4 years ranging from 35 to 80 years. Males were 43 (71.7%) and 17 (28.3%) were females (Tables 1, 2) (Figure 1, 2).

It was also found that 50 of the studied patients were hypertensive either known hypertensive or recently discovered (83.3%), 28 patients were diabetic (46.7%), 22 patients were smokers (36.7%), 20 patients tested positive for HCV (33.3%), 15 patients showed dyslipidemia (25%) and 3 patients were addict (5%) (Table 2).

CT findings of the studied patients showed that basal ganglia hematoma came in first position by 25 patients (41.7%) compared to 15 patients with lobar hematoma (25%), 12 patients with thalamic hematoma (20%), 5 patients with cerebellar hematoma (8.3%) and 3 patients with brainstem hematoma (5%). We also found that mean volume of hematoma of the studied patients was 35 ± 20.6 with median 25 and range (5-150), 10 patients showed surrounding edema (16.7%) and 15 patients (25%) showed intraventricular extension (Table 3).

As for NIHSS upon admission we found that most of the patients had moderate NIHSS by 58.3% followed by 25% of the patients had extremely severe NIHSS, 11.7% with severe condition and only 5 % with minor condition with median value 11(2-29). There was statistically highly significant ($p < 0.00001$) difference among patients with minor, moderate, severe, and extremely severe NIHSS regarding mean values of cardiac troponin T. Also, there was statistically significant positive correlation between them ($r = 0.6313$, $p < 0.00001$) and there was statistically significant increase in mean value of cardiac troponin T in patients with minor ($p = 0.0004$), moderate ($p < 0.0001$) and severe ($p = 0.01$) cases compared to extremely severe cases. However, there was no statistically significant ($p > 0.05$) increase in mean value of cardiac troponin T in patients with minor and severe cases compared to moderate ones (Table 4, 5). There is statistically significant correlation between cardiac troponin T and NIHSS (Table 6) (figure 3).

Table 1: Demographic criteria of the studied patients.

Variables		Studied patients (n=60)
Age (years)	Mean ± SD Range	 60.1 ± 9.4 35.0 – 80.0
Sex	Male Female	43 (71.7%) 17 (28.3%)

Table 2: Risk factors of the studied patients.

		Studied patients (n=60)	
		No.	%
Side of hematoma	**Right**	20	33.3%
	Left	40	66.7%
Site of hematoma	**Basal ganglia**	25	41.7%
	Lobar	15	25%
	Thalamic	12	20%
	Cerebellar	5	8.3%
	Brainstem	3	5%
Surrounding edema		10	16.7%
Intraventricular extension		15	25%
Hematoma volume		Mean± SD	35±20.6
		Median (Range)	25(5-150)

Table 3: CT findings of the studied patients.

Variables	Studied patients (n=60)	
	No.	**%**
Hypertension	60	100%
Diabetes mellitus	28	46.7%
Smoking	22	36.7%
HCV	20	33.3%
Dyslipidemia	15	25%
Addiction	3	5%

Table 4: Clinical scale of severity on admission by NIHSS.

Scale	Studied patients (n=60)		
	Severity	**No**	**%**
NIHSS	Minor (1-4)	3	5
	Moderate (5-15)	35	58.3
	Severe (16-20)	7	11.7
	Extremely severe (21-42)	15	25

Table 5: Relation between cardiac troponin T and scale of severity (NIHSS).

Scale		Troponin T		test	P value
		Mean ± SD	Range		
NIHSS	Minor (1- 4)	13.33±14.43	5.0-30.0	19.351	<0.00001** HS
	Moderate (5-15)	33.97±29.84a	6.0-120.0		
	Severe (16-20)	54.71±39.23bd	8.0-120.0		
	Extremely severe (21-42)	102.47±29.59cef	60.0-155.0		

**highly significant (p < 0.00001).
Ap = 0.2477, bp = 0.1225, Cp = 0.0004 compared to minor cases.
dp =0.1188, ep < 0.0001 compared to moderate cases.
fp = 0.01 compared to severe cases.

Table 6: Correlation between cardiac troponin T and NIHSS.

Variable	Troponin	
	R	P
NIHSS	0.6313	<0.00001(HS)

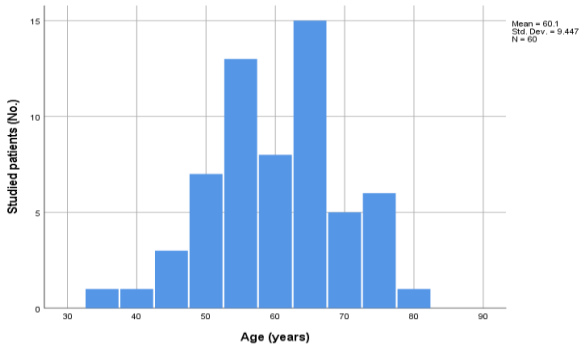

Figure 1: Showing age of the studied patients

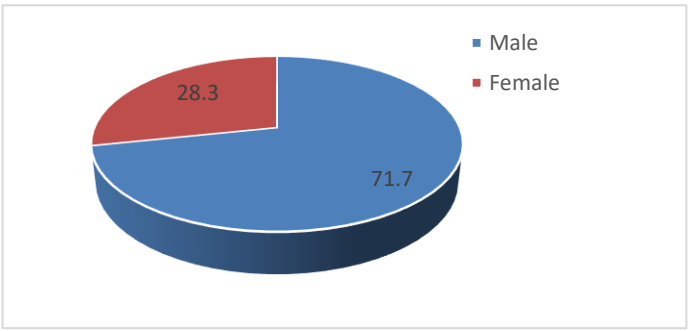

Figure 2: Sex distribution of the studied patients

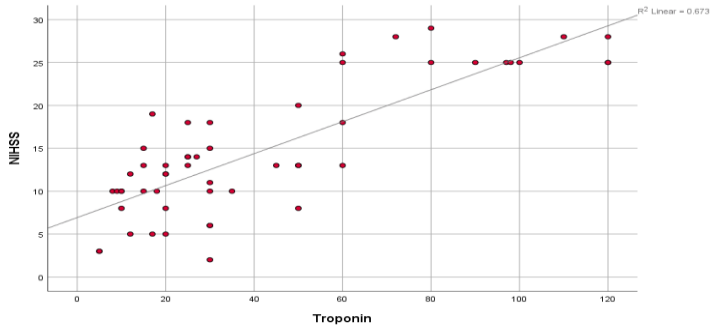

Figure 3: Correlation between cardiac troponin T and NIHSS of the studied patients

DISCUSSION

The mean age 60.1 ± 9.4 years ranging from 35 to 80 years. Males were 43 (71.7%) and 17 (28.3%) were females which was consistent with Thrift et al., Tveiten et al. as well as Rincon et al. who described that the overall incidence of primary intracerebral hemorrhage was more in men than in women **[9 – 11]**. In contrast, Nilüfer et al. and Ruiz et al. who stated that there was no difference among male and female in the incidence of primary intracerebral hemorrhage **[12,13].**

During analysis of the included patients, we found that 50 of the studied patients were hypertensive either known hypertensive or recently discovered (83.3%) which was close to the percentage proved by Brouwers et al. (82%), Safatli et al. (86.5%) and Kheder and Ahmed (80.9%) **[14 – 16].** However, the difference in these percentages could be attributed to difference in sample size, socioeconomic levels, and health education in the studied groups.

Also, 28 patients (46.7%) were diabetic which was close to the results found by Zheng et al. (44.7%) and Xu et al. (42%) [17,18] but way more than Kremer et al. (19.4%) and Melmed et al. (25%) mostly due to difference in the population and sample size **[19,20].**

22 patients (36.7%) were smokers which was consistent with Rathor et al. and Ren et al. **[21,22]**

but more than Li et al. (25.5%) and Xu et al. (26.5%) **[23,18].**

20 patients (33.3%) tested positive for hepatitis C virus and 15 patients (25%) showed dyslipidemia close to Melmed et al. (28%) and Djelilovic-Vranic et al**.** (27.38%) **[22,24]** but higher than Ji et al. (6.4%) **[25].**

3 patients (5%) were addict either IV drug addicts, pill addicts or alcohol addicts which was lower than most of studies as Li et al. (18.4%) and Ren et al. mostly due to ethical and religious factors **[23,22].**

CT findings of the studied patients showed that basal ganglia hematoma came in first position by 25 patients (41.7%) compared to 15 patients with lobar hematoma (25%), 12 patients with thalamic hematoma (20%), 5 patients with cerebellar hematoma (8.3%) and 3 patients with brainstem hematoma (5%) which is close to the findings of Nag et al. and Safatli et al. who both reported that basal ganglia hematoma was the most common among patients with primary intracerebral hemorrhage studied **[26,27]** but far more than the findings found by Attia et al. who found lobar hematoma to be the most common one of the patients. This could be attributed to difference in studied population regarding their number, age, and demographic criteria **[28].**

We also found that mean volume of hematoma of the studied patients was 35 ± 20.6 with median 25 and range (5-150) which was close to Rahmani et al. but there was a difference regarding intraventricular extension of hematoma and surrounding edema which was less in our study **[29].**

As for NIHSS upon admission we found that most of the patients had moderate NIHSS by 58.3% followed by 25% of the patients had extremely severe NIHSS, 11.7% with severe condition and only 5% with minor condition with median value 11(2-29) which is in hand with Sun et al. but less than Han et al. and Wan et al. who reported that median value of NIHSS was 14 and 15 respectively which is mostly attributed to the difference in selected patients between the studies regarding inclusion and exclusion criteria **[30 – 32].**

The main point of our study was to assess the relation between cardiac troponin T and NIHSS as a scale of severity in patients with primary intracerebral hemorrhage. We found that regarding NIHSS, there was statistically highly significant ($p < 0.00001$) difference among patients with minor, moderate, severe, and extremely severe NIHSS regarding mean values of cardiac troponin T. Also, there was statistically significant positive correlation between them ($r = 0.6313$, $p < 0.00001$) which is consistent with Hjalmarsson et al. ($p = 0.002$) and Shi et al. ($p = 0.005$) **[33,34].**

Also, there was statistically significant increase in mean value of cardiac troponin T in patients with minor ($p = 0.0004$), moderate ($p < 0.0001$) and severe ($p = 0.01$) cases compared to extremely severe cases. However, there was no statistically significant ($p > 0.05$) increase in mean value of cardiac troponin T in patients with minor and severe cases compared to moderate ones.

The mechanisms of elevated cTnI in some of the patients with ICH was complicated and incompletely understood. Cardiac troponins are considered the most sensitive and specific biochemical markers for cardiac injury **[35].** Thus, elevation and dynamic changes of serum cTnI levels are indicators of cardiac injury after ICH. The excess of catecholamines was found in patients with ICH, which peaked on the first week and then declined **[36].** Given that catecholamine could produce myocardial necrosis, even in the non-ischemic heart, the catecholamine surge theory has been reasonable linked to cardiac injury after ICH **[37].**

In addition, systemic inflammatory responses are activated after ICH and might contribute to myocyte injury and cell death **[38].** Several other putative causes were also responsible for the observed elevated cTnI levels, including cardiopulmonary disease, renal insufficiency, and ICH patients with neurological deterioration **[39].**

CONCLUSIONS

High levels of cardiac troponin T upon admission are associated with more severe condition of intracerebral hemorrhage assessed by NIHSS.

REFERENCES

1- Cadilhac DA, Dewey HM, Vos T, Carter R, Thrift AG. The health loss from ischemic stroke and intracerebral hemorrhage: evidence from the North-East Melbourne Stroke Incidence Study (NEMESIS). *Health Qual Life Outcomes.* (2010); *8*(1): 1-8.

2- Bernstein JE, Savla P, Dong F, Zampella B, Wiginton JG, Miulli DE, et al. Wacker MR, & Menoni R. Inflammatory Markers and Severity of Intracerebral Hemorrhage. *Cureus.* (2018); *10*(10): e3529.

3- He Y, Liu Q, Wang J, Wang DW, Ding H,Wang W. Prognostic value of elevated cardiac troponin I in patients with intracerebral hemorrhage. *Clin cardiol.* (2020); *43*(4): 338–345.

4- Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. *Lancet.* 1974;2(7872): 81-4.

5- Hemphill JC, Bonovich DC, Besmertis L, Manley GT, Johnston SC. The ICH score: a simple, reliable grading scale for intracerebral hemorrhage. *Stroke.* 2001; 32(4): 891-7.

6- Fonarow GC, Saver JL, Smith EE, Broderick JP, Kleindorfer DO, Sacco RL, et al. Relationship of national institutes of health stroke scale to 30-day mortality in medicare beneficiaries with acute ischemic stroke. *J Am Heart Assoc*. 2012; 1(1): 42-50.

7- Finocchi C, Balestrino M, Malfatto L, Mancardi G, Serrati C, Gandolfo C. National Institutes of Health Stroke Scale in patients with primary intracerebral hemorrhage. *Neurol Sci*. 2018; 39(10): 1751-1755.

8- Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, et al. The ABCs of measuring intracerebral hemorrhage volumes. *Stroke. 1996; 27*(8): 1304-1305.

9- Thrift AG, Dewey HM, Sturm JW, Srikanth VK, Gilligan AK, Gall SL, et al. Incidence of stroke subtypes in the North- East Melbourne Stroke Incidence Study (NEMESIS): differences between men and women. *Neuroepidemiology*. 2009; 32(1): 11-8.

10- Tveiten A, Ljøstad U, Mygland Å, Thomassen L, Pripp AH, Naess H. Intracerebral Hemorrhage in Southern Norway – A Hospital-Based Incidence Study. *Eur Neurol*. 2012; 67: 240–5.

11- Rincon F, Lyden P, Mayer SA. Relationship between temperature, hematoma growth, and functional outcome after intracerebral hemorrhage. *Neurocrit Care*. 2013; 18(1): 45-53.

12- Nilüfer t Y, Koyuncu BA, Çoban O, Tuncay R, Bahar SZ. Gender differences in acute stroke: Istanbul medical school stroke registry. *Neurol India*. 2011; 59 (2): 174-179.

13- Ruiz-Sandoval JL, Chiquete E, Gárate-Carrillo A, Ochoa-Guzmán A, Arauz A, León-Jiménez C, et al. Spontaneous intracerebral hemorrhage in Mexico: results from a Multicenter Nationwide Hospital-based Registry on Cerebrovascular Disease (RENAMEVASC). *Rev Neurol*. 2011; 53(12): 705-12.

14- Brouwers HB, Chang Y, Falcone GJ, Cai X, Ayres AM, Battey TW, et al. Predicting hematoma expansion after primary intracerebral hemorrhage. *JAMA Neurol*. 2014; 71(2): 158-64.

15- Safatli DA, Günther A, Schlattmann P, Schwarz F, Kalff R, Ewald C. Predictors of 30-day mortality in patients with spontaneous primary intracerebral hemorrhage. *Surg Neurol Int*. 2016; 7(Suppl 18): S510-7.

16- Kheder AH, Ahmed AF. Prognosis of patients with primary intracerebral haemorrhage in Erbil city: evaluating presentation, location, and outcome. *J Kurdistan B Med Spec*. 2019; 5 (2).

17- Zheng Y, Hu Q, Manaenko A, Zhang Y, Peng Y, Xu L, et al. 17β-Estradiol attenuates hematoma expansion through estrogen receptor α/silent information regulator 1/nuclear factor-kappa b pathway in hyperglycemic intracerebral hemorrhage mice. *Stroke*. 2015; *46*(2): 485–491.

18- Xu XH, Ye XH, Li JW, Cai JS, Gao T, Zhang WJ, et al. Association between remote diffusion-weighted imaging lesions and cerebral small vessel disease in primary intracerebral hemorrhage. *Eur J Neurol*. 2019; 26(7): 961-968.

19- Kremer PH, Jolink WM, Kappelle LJ, Algra A, Klijn CJ; SMART and ESPRIT Study Groups. Risk Factors for Lobar and Non-Lobar Intracerebral Hemorrhage in Patients with Vascular Disease. *PLoS One*. 2015; 10(11): e0142338.

20- Melmed KR, Cao M, Dogra S, Zhang R, Yaghi S, Lewis A, et al. Risk factors for intracerebral hemorrhage in patients with COVID-19. *J Thromb Thrombolysis.* 2021; 51(4): 953-960.

21- Rathor MY, Rani MF, Jamalludin AR, Amran M, Shahrin TC, Shah A. Prediction of functional outcome in patients with primary intracerebral hemorrhage by clinical-computed tomographic correlations. *J Res Med Sci.* 2012; *17*(11): 1056–1062.

22- Ren H, Kong Y, Liu Z, Zang D, Yang X, Wood K, et al. Selective NLRP3 (Pyrin Domain-Containing Protein 3) Inflammasome Inhibitor Reduces Brain Injury After Intracerebral Hemorrhage. *Stroke.* 2018; *49*(1): 184–192.

23- Li QQ, Li LJ, Wang XY, Sun YY, Wu J. Research Progress in Understanding the Relationship Between Heme Oxygenase-1 and Intracerebral Hemorrhage. *Front Neuro*. (2018); *9*: 682.

24- Djelilovic-Vranic J, Alajbegovic A, Zelija-Asimi V, Niksic M, Tiric-Campara M, Salcic S, et al. Predilection role diabetes mellitus and dyslipidemia in the onset of ischemic stroke. *Med Arch*. 2013; 67(2): 120-3.

25- Ji R, Shen H, Pan Y, Du W, Wang P, Liu G, et al. Risk score to predict hospital-acquired pneumonia after spontaneous intracerebral hemorrhage. *Stroke*. 2014; 45(9): 2620-8.

26- Nag C, Das K, Ghosh M, Khandakar MR. Prediction of Clinical Outcome in Acute Hemorrhagic Stroke from a Single CT scan on Admission. *N Am J Med Sci*. 2012; 4(10): 463-7.

27- Safatli DA, Günther A, Schlattmann P, Schwarz F, Kalff R, Ewald C. Predictors of 30-day mortality in patients with spontaneous primary intracerebral hemorrhage. *Surg Neurol Int*. 2016; 7(Suppl 18): S510-7.

28- Attia S, EL Khatib MG, Bilal M, Nassar H. Spontaneous intracerebral hematoma in young people: Clinical and radiological magnetic resonance imaging features by diffusion-weighted images. *Egyptian J Neurol. Psychiatry Neurosurg.* 2007*;* 44**:** 561-576.

29- Rahmani F, Rikhtegar R, Ala A, Farkhad-Rasooli A, Ebrahimi-Bakhtavar H. Predicting 30-day mortality in patients with primary intracerebral hemorrhage: Evaluation of the value of intracerebral hemorrhage and modified new intracerebral hemorrhage scores. *Iran J Neurol*. (2018); *17*(1): 47–52.

30- Sun Y, You S, Zhong C, Huang Z, Hu L, Zhang X, et al. Neutrophil to lymphocyte ratio and the hematoma volume and stroke severity in acute intracerebral hemorrhage patients. *Am J Emerg Med*. 2017; 35(3): 429-433.

31- Han JH, Lee JM, Koh EJ, Choi HY. The spot sign predicts hematoma expansion, outcome, and mortality in patients with primary intracerebral hemorrhage. *J Korean Neurosurg Soc*. (2014); *56*(4): 303–309.

32- Wan J, Ren H, Wang J. Iron toxicity, lipid peroxidation and ferroptosis after intracerebral hemorrhage. *Stroke Vasc Neurol*. 2019; 4(2): 93-95.

33- Hjalmarsson C, Bergfeldt L, Bokemark L, Manhem K, Andersson B. Electrocardiographic Abnormalities and Elevated cTNT at Admission for Intracerebral Hemorrhage: Predictors for Survival. *Ann Noninvasive Electrocardiol*. 2013; 18(5): 441-449.

34- Shi L, Xu S, Zheng J, Xu J, Zhang J. Blood Pressure Management for Acute Intracerebral Hemorrhage: A Meta-Analysis. *Sci Rep* 7. 2017; 7(1): 1-8.

35- Hamm CW, Bassand JP, Agewall S, Bax J, Boersma E, Bueno H, et al. ESC guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation. The Task Force for the management of acute coronary syndromes (ACS) in patients presenting without persistent ST-segment elevation of the European Society of Cardiology (ESC). *G Ital Cardiol (Rome)*. 2012; 13(3): 171-228.

36- Leow MK, Loh KC, Kiat Kwek T, Ng PY. Catecholamine and metanephrine excess in intracerebral hemorrhage: revisiting an obscure yet common "pseudo phaeochromocytoma". *J Clin Pathol*. 2007; 60(5): 583-584.

37- Kuo LT, Chen CM, Li CH, Tsai JC, Chiu HC, Liu LC, et al. Early endoscope-assisted hematoma evacuation in patients with supratentorial intracerebral hemorrhage: case selection, surgical technique, and long-term results. *Neurosurg Focus*. 2011; 30(4): E9.

38- Yang Y, Lv J, Jiang S, Wang D, Hu W, Deng C, et al. The emerging role of Toll-like receptor 4 in myocardial inflammation. *Cell Death Dis* 7. 2016; e2234-e2234.

39- Lord AS, Gilmore E, Choi HA, Mayer SA; VISTA-ICH Collaboration. Time course and predictors of neurological deterioration after intracerebral hemorrhage. *Stroke*. 2015; 46(3): 647-52

How to cite

Ragab, A., Alebiary, M., Elsayed, W., Aly Ghonemy, M. CARDIAC TROPONIN T: AN INDICATOR OF SEVERITY IN PATIENTS WITH PRIMARY INTRACERBERAL HEMORRHAGE. Zagazig University Medical Journal, 2023; (559-565): -. doi: 10.21608/zumj.2021.86908.2290