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The Impact of Neurological Disorders on Cardiac Functions in Non-Cardiac Children

Hebatallah Ahmed Bedair (MSc) [1]* Khaled Talaat Abu-Eala (MD) [1], Osama Abdrab Elrasol Tolba (MD) [1], Sahar Abdel-Azeem Abdel-Aziz (MD) [1], Magdy Abdel-Aziz Elmahallawy (MD) [2]

[1] Pediatric Department, Faculty of Medicine, Tanta University, Egypt.
[2] Neurosurgery Department, Faculty of Medicine, Tanta University, Egypt.
Email: hebabedair86@yahoo. com

Abstract: Background: Cardiac injury frequently complicates neurological disorders, leading to higher morbidity and mortality. Neurological disorders such as hemorrhagic and ischemic stroke, epilepsy, CNS infection and traumatic brain injury, results in dysfunctions of the autonomic nervous system, alteration of systemic circulation homeostasis mediated by neurogenic stimuli, change in serum cardiac enzyme levels, electrocardiographic, and echocardiographic abnormities. **Aim of the work:** The aim of the present study was to detect the impact of neurological disorders on cardiac functions in pediatric non-cardiac patients. **Subjects and methods:** This prospective cohort study was carried out on one hundred and fifty children (aged from 2 months to 16 years) equally divided into three groups (n=50); group I: critically ill neurological patients, group II: non critically ill neurological patients, group III: healthy children matched for age and sex as a healthy control group. Echocardiography, electrocardiography and cardiac enzymes (including cardiac troponin I and lactate dehydrogenase) were done within 12 hours of admission. **Results:** Regarding ECG abnormalities, group I showed that there were 41cases (82%) with ECG abnormalities, group II showed that there were 14 cases (28%) with ECG abnormalities. ECG abnormalities include ischemic changes, rhythm and conduction abnormalities. Regarding echocardiographic examination there was significant decrease in both left ventricular systolic and diastolic functions (measured by tissue Doppler and strain echocardiography) in patients with neurological disorders compared with the control group. The mean plasma concentration of cardiac enzymes (lactate dehydrogenase, cardiac troponin I) was statistically higher in patients with neurological disorders compared with the control group. **Conclusion:** Cardiac injury has to be anticipated as an impact of neurological disorders and cardiac evaluation should be an essential step in the assessment of neurological pediatric patients.

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Key words: arrhythmia, cardiac injury, neurological disorders, troponin.

Introduction:

There is a growing interest in understanding the interactions between the brain and heart driven by the need to identify, at an early stage, patients at risk for developing cardiovascular manifestations due to neurological diseases. (1)

Understanding of the brain-heart cross link has been greatly enhanced during the last decade by observations from a variety of clinical settings. Many CNS disorders can affect the heart including traumatic brain injury, CNS infections, degenerative brain diseases, CNS tumors, vascular disorders (hemorrhagic and ischemic stroke) and functional (as in epilepsy disorders). (2)

Affection of the heart by CNS diseases may be acute or chronic, mild or severe. Cardiac abnormalities occurring as a result of neurological disorders include arrhythmias, stress cardiomyopathy, and autonomic dysfunction. Rarely, CNS-disorders may directly induce systolic or diastolic dysfunction, heart failure, myocardial infarction (MI), arterial hypertension, or pulmonary hypertension. (3, 4)

Though, only limited data, mostly from retrospective studies or from case reports are available, there is increasing evidence that the heart cannot be neglected in the presence of a central nervous system disorder. Intensivist must be aware of the possible cardiac complications of CNS disorders and be prepared for such complications by proper monitoring and duly consultation of neurologists and cardiologists. (5)

The number of cases of neurological disorders with increased morbidity and mortality in pediatric intensive care were rising in a way that cannot be explained simply by the original disease. The involvement of other organs (including the heart) as an

etiology is rising. Therefore, the aim of this study was to evaluate the impact of neurological disorders on cardiac functions in children.

2. Patients and methods:.

This study was an observational prospective cohort study which was carried out in pediatric department (Pediatric ICU and pediatric neurology unit) and Pediatric neurosurgical department, Tanta University Hospitals upon one hundred and fifty children, aged from 1month to 216 months (18 years), 88males and 62 females from October 2017 to October 2019.

An informed written consent was obtained from the guardians of the patients included in the study. Patients were divided into three groups (I, II, III), group I:50 critically ill patients (with system failure) with acute neurological disorders were chosen from those admitted to pediatric intensive care unit and Pediatric Neurosurgical department, Tanta university hospitals, group II:50 none critically ill patients (no system failure) with neurological disorders matched for age, sex and diagnosis were chosen from those admitted at pediatric neurology unit, Tanta university hospitals taken as a diseased control group, group III:50 healthy children matched for age; sex were taken as a healthy control group.

The inclusion criteria were patients aged from 2months to 16 years with any of the following CNS disorders: intracranial hemorrhage including (Intracerebral hemorrhage, subdural hematoma, and subarachnoid hemorrhage), ischemic stroke, meningioencephalitis (non-bacterial), epilepsy and traumatic brain injury. Exclusion criteria were those with previous cardiac disease including congenital, rheumatic, etc.

All the participants included in this study were subjected to the following (within the first 12 hours of admission):

- Complete history taking and thorough clinical examination.
- PRISM III score at admission and SOFA score which was done every 24 hours in PICU,.
- Routine laboratory investigations including ABG, liver enzymes, PT, PTT, renal function tests and CBC.
- Electrocardiography using 3 channels α 1000 apparatus.
- Echocardiography including M-mode, 2D, tissue Doppler, 2D and 3D strain echocardiography using Vivid 7 ultrasound machine (GE Healthcare, Horten, Norway, with 3.5 and 4S multi-frequency transducers).
- Plasma levels of c Tn I and LDH: were measured by enzyme linked immunosorbent assay (ELISA) technique.

The collected data were tabulated and statistically analyzed using SPSS (IBM©, USA) version 25. Quantitative data were presented as range and median and were compared by Kruskal Wallis test with Post Hoc. Qualitative data were presented as number and percentage and were compared by chi-square test (X^2). The level of significance was adopted at a P value < 0.05.

3. Results:.

Clinical diagnoses of the studied patients are shown in (table 1). Regarding demographic data, there were no statistically significant differences between the studied groups (Table 2), this represents the nonbiased choice of the studied cases.

Regarding PRISM III score the present study showed that there was statistically significant increase in groups I compared with group II. This denoted the increased severity of the disease condition in the critically ill neurological patients compared with the non-critically ill neurological patients' group.

Regarding SOFA Score the present study showed that there was statistically significant increase in group I compared with groups II in the 1st, 2nd and 3rd days.

Regarding ECG abnormalities (Table 3), group I shows that there were 9 cases with normal ECG (18%), and 20 cases with sinus tachycardia (40%), 3 cases with sinus bradycardia (6%), 5 cases with supraventricular tachycardia (10%), 1 case with PACs (2%), 2 cases with PVCs (4%), 3 cases with heart block (6%), 3 cases with T wave inversion (6%) and 4 cases with ST depression (8%). group II shows that there were 36 cases with normal ECG (72%), 10 cases with sinus tachycardia (20%), 3 cases with supraventricular tachycardia (6%) and 1 case with heart block (25). group III shows that all cases have normal ECG.

Regarding plasma levels of lactate dehydrogenase and cardiac troponin I (Table 4), there was statistically significant increase in group I compared with group III, statistically significant increase in group II compared with group III, there was no statistically significant differences between group I and group II.

Regarding echocardiographic examination (Table 5), there was statistically significant decrease in LV EF in group I compared with group II and statistically significant decrease in LV EF in group I compared with group III. There was statistically significant decrease in mitral annulus systolic velocity (S) in group I compared with group II, statistically significant decrease in S in group I compared with group III. There was statistically significant decrease in E'/ A', in group I compared with group II and statistically significant decrease in E'/ A', in group I compared with group III. There was statistically

significant increase in LV MPI in group I compared with group II and statistically significant increase in LV MPI in group I compared with group III, statistically significant increase in group II compared with group III. Regarding LV 2Ds and LV 3DS, there was statistically significant decrease in group I compared with group II and statistically significant decrease in group I compared with group III, statistically significant decrease in group II compared with group III (Figure 1).

Table (1): Patients Clinical diagnoses

Diagnosis Group I	**Number of Patients**	**%**
Status epilepticus	34	68
Traumatic brain injury	7	14
CNS infection	5	10
Stroke	4	8
Group II	**Number of Patients**	**%**
Idiopathic epilepsy	12	24
Symptomatic epilepsy:	38	76%
Cerebral palsy. degenerative brain disease. post-meningitic. post-encephalitic. stroke. posttraumatic brain injury. Hydrocephalus	12. 6. 6. 6. 3. 3. 2	24. 12. 12. 12. 6. 6. 4

Table (2): Demographic Data of the Studied Groups

	Group I (n= 50)		**Group II (n= 50)**		**Group III (n= 50)**		**Test of sig.**	**p**
	No.	**%**	**No.**	**%**	**No.**	**%**		
Sex								
Male	29	58.0	28	56.0	31	62.0	χ^2=. 0.385	0.825
Female	21	42.0	22	44.0	19	38.0		
Age (months)								
Min. – Max.	2.0 – 192.0		1.0 – 216.0		6.0 – 144.0		H=. 1.092	0.579
Mean ± SD	**45.23 ± 52.19**		**45.68 ± 49.82**		**40.60 ± 34.46**			
Median	25.0		19.0		36.0			
Weight (Kilogram)								
Min. – Max.	4.0 – 52.0		3.0 – 40.0		6.0 – 30.0		H=. 3.895	0.143
Mean ± SD	**14.40 ± 13.09**		**12.96 ± 7.73**		**13.40 ± 5.71**			
Median	10.0		11.50		13.0			

χ^2: **Chi square test** H: H for **Kruskal Wallis test.**
p: p value for comparing between the different studied groups *: Statistically significant at p ≤ 0.05 Group I: With system failure .
Group II: No system failure with CNS disorders Group III: control

Table (3): Electrocardiography of the Studied Groups

Electrocardiographic finding	**Group I (n= 50)**		**Group II (n= 50)**		**Group III (n= 50)**		χ^2	**p**
	No.	**%**	**No.**	**%**	**No.**	**%**		
EGG								
Normal ECG	9	18.0	36	72.0	50	100.0	74.813*	<0.001*
-Sinus tachycardia	20	40.0	10	20.0	0	0.0	25.0*	<0.001*
-Sinus bradycardia	3	6.0	0	0.0	0	0.0	4.137	MCp=0.107
-Supraventricular tachycardia	5	10.0	3	6.0	0	0.0	5.253	MCp=0.100
-PACs	1	2.0	0	0.0	0	0.0	1.831	MCp=1.000
-PVCs	2	4.0	0	0.0	0	0.0	2.682	MCp=0.333
-Heart block (prolonged PR interval)	3	6.0	1	2.0	0	0.0	2.959	MCp=0.322
-T wave inversion	3	6.0	0	0.0	0	0.0	4.137	MCp=0.104
-ST segment depression	4	8.0	0	0.0	0	0.0	5.856*	MCp=0.034*
Total number of cases with ECG abnormalities	**41/50**	**82.0**	**14/50**	**28.0**	**0/50**	**0.0**	85.857*	MCp<0.001*

χ^2: **Chi square test MC: Monte Carlo** p: p value for comparing between the different studied groups *: Statistically significant at p ≤ 0.05.
Group I: With system failure Group II: No system failure with CNS disorders Group III: control **ECG:** Electrocardiography.
PACs: Premature atrial contractions. **PVCs:** Premature ventricular contractions.

Table (4): Comparison of different echocardiographic parameters between the different studied groups

	Group I (n= 50)	Group II (n= 50)	Group III (n= 50)	F	p
LV EF (%)					
Min. – Max.	40.0 – 72.0	50.0 – 75.0	50.0 – 75.0	38.471*	<0.001*
Mean ± SD	**50.52 ± 6.69**	**62.62 ± 8.59**	**62.90 ± 8.73**		
Median	50.0	65.0	65.0		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3=0.983				
S (cm/s)					
Min. – Max.	.0 – 7.0	5.0 – 8.0	6.0 – 8.0	43.761*	<0.001*
Mean ± SD	**5.02 ± 1.62**	**6.66 ± 0.63**	**6.84 ± 0.65**		
Median	5.0	7.0	7.0		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3=0.679				
E'/A'					
Min. – Max.	0.50 – 1.90	1.10 – 1.90	1.10 – 1.90	33.489*	<0.001*
Mean ± SD	**1.14 ± 0.37**	**1.51 ± 0.19**	**1.53 ± 0.19**		
Median	1.15	1.50	1.50		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3=0.951				
LV MPI					
Min. – Max.	0.30 – 1.90	0.30 – 1.20	0.30 – 0.50	16.943*	<0.001*
Mean ± SD	**0.72 ± 0.43**	**0.55± 0.28**	**0.38 ± 0.07**		
Median	0.50	0.40	0.40		
Sig. bet. grps.	p_1=0.009*, p_2<0.001*, p_3=0.014*				
LV 2DLS (%)					
Min. – Max.	-20.0 – -7.0	-23.0 – -13.0	-29.0 – -17.0	103.995*	<0.001*
Mean ± SD	**-14.26 ± 3.05**	**-17.45 ± 2.63**	**-22.32 ± 2.74**		
Median	-15.0	-17.0	-22.0		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3<0.001*				
LV 3DLS (%)					
Min. – Max.	-18.0 – -7.0	-20.0 – -10.0	-25.0 – -17.0	139.691*	<0.001*
Mean ± SD	**-11.68 ± 2.49**	**-14.72 ± 3.02**	**-20.20 ± 2.18**		
Median	-11.0	-15.0	-20.0		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3<0.001*				

LV EF: left ventricular ejection fraction**, S:** systolic mitral annulus velocity, **LV MPI:** left ventricular myocardial performance index**, LV 2DLS:** left ventricular two dimentional global longitudinal strain **LV 32DLS:** left ventricular three dimentional global longitudinal strain.
F: F for **ANOVA test**, Pairwise comparison bet. each 2 groups was done using **Post Hoc Test (Tukey).**
p: p value for comparing between the different studied groups.
p_1: p value for **group I** and **group II** p_2: p value for **group I** and **group III** p_3: p value for **group II** and **group III**.
*: Statistically significant at $p \leq 0.05$ Group I: With system failure .
Group II: No system failure with CNS disorders Group III: control

Table (5): Comparison of Cardiac Enzymes between the Studied Groups

	Group I (n= 50)	Group II (n= 50)	Group III (n= 50)	H	p
LDH (U/d L)					
Min. – Max.	40.0 – 10080.0	30.0 – 1966.0	30.0 – 110.0	48.863*	<0.001*
Mean ± SD	**504.5 ± 1413.8**	**243.8 ± 313.8**	**63.08 ± 22.68**		
Median	225.0	189.5	60.0		
Sig. bet. grps.	p_1=0.243, p_2<0.001*, p_3<0.001*				
Troponin I					
Min. – Max.	0.11 – 0.41	0.02 – 0.36	0.01 – 0.03	113.313*	<0.001*
Mean ± SD	**0.30 ± 0.08**	**0.17 ± 0.10**	**0.02 ± 0.01**		
Median	0.30	0.17	0.02		
Sig. bet. grps.	p_1<0.001*, p_2<0.001*, p_3<0.001*				

LDH: lactate dehydrogenase enzyme.
H: H for **Kruskal Wallis test,** Pairwise comparison bet. each 2 groups was done using **Post Hoc Test (Dunn's for multiple comparisons test)** p: p value for comparing between the different studied groups p_1: p value for **group I** and **group II**.
p_2: p value for **group I** and **group III** p_3: p value for **group II** and **group III** *: Statistically significant at $p \leq 0.05$.
Group I: With system failure Group II: No system failure with CNS disorders .
group III: control

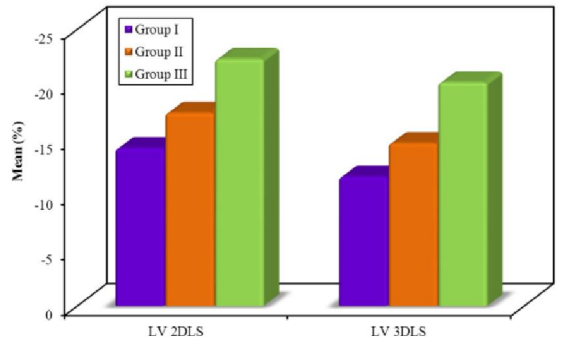

Figure (1): Comparison of Left Ventricular Global Longitudinal Systolic Two Dimentional and Three Dimentional Strain between the Studied Groups.

4. Discussion:

It is common knowledge that decreases in the cardiac output below a critical level, such as in heart failure, myocardial infarction and dysrhythmia, may lead to the development of cerebral hemodynamic and metabolic abnormalities. However, the impact of the brain on cardiac functions is less widely recognized and appreciated. [6]

The primary goal of the present study was to evaluate the impact of neurological disorders on cardiac functions in pediatrics and to highlight the role of strain echocardiography for early detection of cardiac function affection in these patients.

Regarding ECG abnormalities, group I showed that there were 9 cases with normal ECG (18%), and 20 cases with sinus tachycardia (40%), 3 cases with sinus bradycardia (6%), 5 cases with supraventricular tachycardia (10%), 1 case with PACs (2%), 2 cases with PVCs (4%), 3 cases with heart block (6%), 3 cases with T wave inversion (6%) and 4 cases with ST depression (8%). group II showed that there were 36 cases with normal ECG (72%), 10 cases with sinus tachycardia (20%), 3 cases with supraventricular tachycardia (6%) and 1 case with heart block (2%). group III showed that all cases have normal ECG.

This was in accordance with Zhili Chen et al. who reported ECG abnormalities in the form of supraventricular tachycardia in patients with hemorrhagic and ischemic stroke. [7].

Also, MM Manea, et al. [8], reported that electrocardiographic alterations in stroke can appear in the context of a preexistent cardiac disease, but also in its absence, in which case they have a neurologic origin. The incidence of these manifestations is higher in subarachnoid hemorrhage (SAH) (60-70% of patients) and its complications, than in ischemic stroke (15-20%). Some of the most frequent electrocardiographic abnormalities in stroke are: ST segment abnormalities, negative T waves, U waves, left axis deviation, prolonged QT, atrial fibrillation (AF)/ atrial flutter, sinus tachycardia, ventricular tachycardia, atrial and ventricular premature complexes and bradyarrhythmias (sinus-node dysfunction, 2nd and 3rd degree heart block).

This was also in accordance with El Amrousy D, et al. [9] who carried out a study on 60 pediatric patients presented with CSE. ECG changes were present in 55% of patients with CSE in the form of conduction abnormalities, ischemic changes, and arrhythmias.

The present study showed that regarding LV EF (estimated by conventional echocardiographic imaging), there was statistically significant decrease in group I compared with group II and statistically significant decrease in group I compared with group III with no statistically significant difference between group II and group III.

In the clinical practice, ejection fraction (EF) is the most used parameter to judge LV systolic function. Unfortunately, EF is limited by several technical and hemodynamic factors. Quantitative approaches are dependent on geometrical assumptions and on endocardial border definition. EF only reflects global LV function and does not take into consideration that a hyperkinetic segment may compensate a hypokinetic one leading to a false “normal” result. [10]

Also, EF is a load-dependent parameter. In addition, cardiac mechanics is a complex process and EF can only roughly describe it. [11]

The present study showed that regarding tissue Doppler echocardiography, there was statistically significant decrease in mitral annulus systolic velocity (S) in group I compared with Group II and statistically significant decrease in S in group I compared with group III, and no statistically significant difference between group II and group III. There was statistically significant decrease in E'/ A’ in group I compared with group II and statistically significant decrease in E'/ A’ in group I compared with group III, and no statistically significant difference between group II and group III. There was statistically significant increase in LV Myocardial performance index (MPI) in group I compared with group II and statistically significant increase in LV MPI in group I compared with group III, and statistically significant increase in LV MPI in group II compared with group III. These results denote impaired both systolic and diastolic functions of the left ventricle in group I and group II.

MPI is a complex parameter, capable of estimating combined systolic and diastolic performance and could be more advantageous than the isolated measurement of either systolic or diastolic parameters in the evaluation of the global LV function and RV function. [12]

This was in accordance with Putaala J et al. [13] who reported that ICH may induce systolic dysfunction without overt heart failure.

In a study of 88 children with enterovirus-71 encephalitis carried out by Griffiths MJ et al. [14] 11 developed cardiorespiratory compromise, such as systolic dysfunction or sudden cardiac death. Higher concentrations of cytokines were observed in the cerebrospinal fluid (CSF) than in the serum suggesting that the CNS infection was responsible for the cardiac manifestations.

This was in accordance with Hong-Kyun Park et al. [15] who reported that in a study carried out on ischemic stroke population, more than half of the cases exhibited LV diastolic dysfunction, and approximately 10% of the patients exhibited profound dysfunction. LV diastolic dysfunction deteriorated the functional status of the patient over the course of stroke recovery and may have increased the risk of recurrent vascular events in ischemic stroke survivors.

Diastolic dysfunction is much more common than previously believed and, in one study, was identified in 71% of patients after SAH and associated with a higher incidence of pulmonary edema. [16].

Tissue Doppler imaging is a useful echocardiographic technique employing the Doppler principle to measure the velocity of myocardial segments and other cardiac structures which is load independent. However, TDI has some limitations, like angle dependency of the ultrasound beam, the complex rotational and translational movements of the heart [17].

The present study showed that regarding left ventricular global longitudinal strain measurements, there was statistically significant decrease in LV 2DLS and LV 3DLS in group I compared with group II, statistically significant decrease in group I compared with group III, statistically significant decrease in group II compared with group III, this result denotes impaired left ventricular global systolic function in both group I critically ill neurological patients and group II non critically ill neurological patients.

These results are in accordance to Kraigher-Krainer E., et al. [18] who stated that strain imaging has the ability to detect impaired systolic function even if global LVEF is preserved.

2D-STE imaging uses standard B-mode images for speckle tracking analysis. The speckle patterns are the result of acoustic backscatter generated by the reflected ultrasound beam. Speckles represent fixed tissue markers, or 'natural acoustic markers', that are randomly distributed throughout the myocardium and have their own unique signature or 'fingerprint'. The movement of this speckled pattern follows myocardial tissue motion as it tracks the defined region of speckles, frame by frame and eventually over the entire heart cycle, and extracts the displacement (the movement of those speckles), velocity (the speed at which this movement occurs), strain (the relative change in distance between those speckles. [19]. So, strain echocardiography is angle independent.

Although the utility of two- dimensional (2D) (STE) to quantify left ventricular deformation has been demonstrated, this methodology is limited by foreshortened views, geometric modeling, and the assumption that speckles can be tracked from frame to frame, despite their out of plane motion. To circumvent these limitations, a 3D speckle tracking algorithm was recently developed. 3D strain provides rapid image acquisition and does not require high level of operator skills, and with a shorter scan time, the technique has the potential to increase the efficiency in the echo laboratory. [20] So 3D strain is plane independent and time saving.

Current echocardiographic guidelines recommend the use of 3D echocardiography. 3D STE allows calculation of LVEF, volumetric analysis, and simultaneous measurement of multidirectional components of strain in a single 3D data set. This method may overcome the potential limitations of 2D STE, such as out of plane motion tracking of speckles, which can increase noise and reduce accuracy. [21].

Regarding serum lactate dehydrogenase, the present study showed that there was statistically significant increase in group I compared with group III, statistically significant increase in group II compared with group III.

This was in accordance with Wong, K. C. [22] who reported that patients with an acute illness, who had seizure as one of their clinical manifestations, had an elevated serum LDH above the upper limit, before, during, or after an episode of seizure. The variations of elevation of serum LDH were large depending on the acute illness and the extent of tissue injury involved.

The present study showed that regarding serum cardiac Troponin I, there is statistically significant increase in group I compared with group III, statistically significant increase in group II compared with group III.

This result is in agreement with Sanjay Dixit et al. [23], who conducted a blinded cohort study on 104 patients with acute neurologic events to assess the incidence of cardiac injury determined by elevations of cardiac troponin I (c Tn I) in patients presenting within 24 hours of a neurologic event and to determine their short- and long-term prognostic effect, this study showed that peak levels of c Tn I were elevated (≥0.4 µg/L) in (19%) of cases (mean + SD, 2.5 + 2.7 µg/L) and in 50% of patients who developed seizures during the course of hospitalization or who are admitted with an initial diagnosis of seizures. However, their observations were made only in patients with

hemorrhagic strokes, head injuries, and intracranial space–occupying lesions. All patients with elevated c Tn I levels had clinical, electrocardiographic, or echocardiographic evidence of cardiac injury except those with minor elevations. One-year mortality was 29%. Early death (≤30 days) accounted for 44% of total mortality and was significantly higher in patients with elevated c Tn I levels. They concluded that there is a substantial prevalence of myocardial injury in patients with acute neurologic illness. Cardiac injury in this population, as in others, seems to adversely affect prognosis. This is also similar to the results of Horowitz et al. [24] However; their observations were made only in patients with subarachnoid hemorrhage.

Conclusion:

Cardiac injury as a complication of neurological disorders have to be well recognized and anticipated. Early introduction of echocardiography and ECG for neurological patients helps early detection and proper management of cardiac complications of these disorders thus improving morbidity and mortality. Left ventricular strain obtained by four-dimensional echocardiography (3DS) can be a novel and promising technique for early detection of left ventricular deformation in patients with neurological diseases.

Conflicts of interest: Nil.

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