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Research Article

Evaluation of Neurogenic Myocardial Stunning by Two-Dimensional Speckle Tracking Echocardiography

Cigdem Ileri,[1] Emre Eynel,[2] Zekeriya Dogan,[3] Beste Ozben,[4] Nevin Pazarci[5]

[1]Department of Cardiology, Kartal Kosuyolu Training and Research Hospital, Istanbul, Türkiye
[2]Department of Cardiology, Umraniye Training and Research Hospital, Istanbul, Türkiye
[3]Department of Cardiology, Marmara University Faculty of Medicine, Istanbul, Türkiye
[4]Department of Cardiology, Marmara University Faculty of Medicine, Istanbul, Türkiye
[5]Department of Neurology, Umraniye Training and Research Hospital, Istanbul, Türkiye

Abstract

Objectives: Neurogenic stunned myocardium (NSM) is a cardiac condition resulting from acute neurological events that can lead to a reduction in myocardial strain, an important parameter used to assess cardiac function. The aim of our study was to investigate the NSM and its impact on myocardial strain.

Methods: 82 consecutive patients (mean age: 66 ±14 years, 36 male) presenting with acute ischemic stroke were consecutively included. LV functions of all patients were evaluated by two-dimensional (2D) speckle-tracking echocardiography. High-sensitive cardiac Troponin I (hs-cTnI) level>0.04ng/mL was accepted as increased.

Results: 33 patients (40.2%) had elevated hs-cTnI and 23 patients (28%) had insular cortex involvement. The stroke patients with elevated hs-cTnI had higher frequencies of coronary artery disease and insular cortex involvement, higher NT-proBNP levels, and lower left ventricular ejection fraction (LVEF), global longitudinal strain (GLS), global and regional circumferential strain compared to patients with normal troponin levels. Although the patients with insular cortex involvement had significantly higher hs-cTnI levels, there were not any significant differences in LVEF and 2D-STE parameters between the groups.

Conclusion: Elevated troponin levels may be associated with a decrease in LVEF and myocardial strain parameters, especially in acute ischemic stroke patients with insular cortex involvement; and may be considered as a sign of neurogenic myocardial stunning.

Keywords: Acute ischemic stroke; myocardial stunning; neurogenic cardiac injury; troponin

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Neurogenic stunned myocardium (NSM) is a definition of reversible myocardial dysfunction resulting from dysregulation of the autonomic nervous system after an acute neurologic event.[1] NSM incidence varies between 20%-40%, depending on the neurological disease.[2] Although it has been reported mostly in subarachnoid hemorrhage (20-30% of cases); it can be seen after various neurological events such as cerebral trauma, ischemic stroke, seizure, encephalitis, and epilepsy.[3,4] NSM is characterized by increased troponin levels, new electrocardiographic (ECG) changes, and wall motion abnormalities with decreased left ventricular ejection fraction (LVEF).[5] Studies on NSM have shown a remarkable involvement of the insular cortex, which plays a role in the regulation of both car-

Address for correspondence: Cigdem Ileri, MD. Department of Cardiology, Kartal Kosuyolu Training and Research Hospital, Istanbul, Türkiye
Phone: +90 553 010 97 96 **E-mail:** cgdmileri@gmail.com

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diovascular function and the autonomic nervous system.[3]

Myocardial strain is a measure of the deformation of the myocardium during the cardiac cycle and is commonly evaluated using advanced imaging techniques such as speckle tracking echocardiography (STE) or cardiac magnetic resonance imaging.[6] Strain imaging provides quantitative information about the myocardial function and can detect subtle changes in myocardial contractility.[7] 2D-STE is a technique that uses 2-D images to detect speckles (acoustic backscatters) in a predetermined region and tracks them frame-by-frame.[8] 2D-STE can identify subtle forms of NSM and may differentiate the pathophysiology of NSM in different types of stroke.[9]

The aim of our study was to investigate the left ventricular (LV) functions by 2D-STE and determine whether there was a correlation between neurogenic stunned myocardium and strain parameters.

Methods

Ethical Approval

The investigation conformed to the principles outlined in the Declaration of Helsinki. All participants gave written informed consent. The study was approved by the ethics committee of the University of Health Sciences, Umraniye Education and Research Hospital.

Study Population

Eighty-two consecutive patients admitted to the neurology clinics with the diagnosis of acute ischemic stroke were included in the study. The Baseline National Institutes of Health Stroke Scale (NIHSS) of patients were noted at admission. Cranial images of the patients were evaluated by an experienced neurologist who was blind to patients' characteristics to determine whether there was an insular cortex involvement. Transient ischemic attacks (TIA) were not included in our study population.

All patients had daily standard 12-lead electrocardiography (ECG). The presence of comorbidities, such as hypertension, hyperlipidemia, and diabetes was assessed. Blood samples for high sensitive C-reactive protein (hs-CRP), hs-cTnI, and NT-proBNP levels were noted. Serum troponin I levels were analyzed by the Siemens ADVIA Centaur hs-cTnI assay (Siemens Healthcare Diagnostics, Deerfield, IL, USA), and a hs-cTnI> 0.04 ng/mL was accepted as elevated in our laboratory.

All patients underwent a complete transthoracic echocardiographic study by a Philips Epic echocardiography device (Philips Medical Systems, Andover, MA, USA) by an experienced cardiologist within the first three days following acute ischemic stroke. Echocardiographic parameters were measured according to the recent guidelines of the American Society of Echocardiography, and LVEF was calculated using the biplane Simpson method.[10] Standard 2-D images were obtained during breath-hold, and stored in cine-loop format from 3 consecutive beats. Three standard apical views (4-chamber, 2-chamber, and apical long axis) and three parasternal short axis views (basal, mid, and apical views) were obtained. QLAB workstation was used for further offline analysis (QLAB cardiac 3DQ, Philips Medical Systems). Multidirectional analysis of the LV [in the circumferential (GCS), and longitudinal (GLS) directions] was performed using 2-D STE imaging. GCS was assessed by applying 2-D STE imaging to the three parasternal short-axis views. The values of GCS were derived from the average peak systolic strain values of 17 segments. The longitudinal peak systolic strain was measured as 2-D STE imaging of the apical views of the LV (in six segments in each of the three apical views) and averaged.

Statistical Analysis

All statistical tests were performed by a statistical analysis program (SPSS 21.0 for Windows, Chicago, IL). The distribution of data was tested using a one-sample Kolmogorov–Smirnov test. Categorical variables were defined as a percentage, and comparisons were made using the Chi-square test. Continuous data were expressed as mean±SD and Student's t-test was used to compare the normally distributed continuous variables while the Mann-Whitney U test was used to compare the nonparametric continuous variables. A significance level was set at $p<0.05$.

Results

Eighty-two consecutive acute ischemic stroke patients (mean age: 66±14 years, 36 male) were included in the study. Thirty-three patients (40.2%) had elevated troponin. The general characteristics and laboratory parameters of the patients according to troponin levels are shown in Table 1. Although there were not any significant differences in the general characteristics of the patients, the stroke patients with elevated hs-cTnI had significantly higher frequencies of coronary artery disease (CAD) and insular cortex involvement. They also had higher NT-proBNP and hs-CRP levels. There were no significant differences in the NIHSS scores and ST segment and T wave changes among patients with different troponin levels.

The conventional echocardiographic parameters and 2D-STE indices of the patients are listed in Table 2. The troponin-positive group had significantly larger left atriums and lower LVEF values compared to patients with normal troponin levels. GLS and GCS values and also regional circumferential strain parameters were significantly lower in stroke patients with elevated hs-cTnI.

Table 1. The general characteristics and laboratory parameters of the patients according to high sensitive cardiac troponin I levels

	Stroke patients with elevated troponin (n=33)	Stroke patients with normal troponin (n=49)	p
Age (years)	69.12±13.2	63.9±13.6	0.093
Male sex (n – %)	15 (45.5%)	21 (42.9%)	0.825
Body mass index (kg/m^2)	27.4±4.7	27±4.2	0.936
Hypertension (n – %)	36 (78.8%)	31 (63.3%)	0.151
Diabetes (n – %)	14 (42.4%)	15 (30.6%)	0.348
Hyperlipidemia (n – %)	8 (24.2%)	12 (24.5%)	1
Coronary artery disease (n – %)	15 (45.5%)	7 (14.3%)	0.002
Insular cortex involvement (n – %)	14 (42.4%)	9 (18.4%)	0.024
NIHSS	6.3±5.4	4.6±3.7	0.224
Glucose (mg/dL)	131.8±49.5	121.6±46.9	0.263
Creatinine (mg/dL)	1.1±0.56	0.84±0.35	0.006
Total cholesterol (mg/dL)	182±52	205±46	0.052
LDL cholesterol (mg/dL)	116±45	131±40	0.111
hs-cTnI (ng/mL)	0.26±0.53	0.014±0.009	<0.001
hs-CRP (mg/L)	23.7±29.4	13.5±27.1	0.001
NT-proBNP (pg/mL)	6377±11166	467±797	<0.001
ST segment/T wave changes (n–%)	18 (54.5%)	24 (49%)	0.658

NIHSS: National Institutes of Health Stroke Scale; LDL: Low-density lipoprotein; hs-cTnI: high sensitive cardiac Troponin I; hs-CRP: High sensitive C reactive protein; NT-proBNP: N terminal pro-brain natriuretic peptide.

Table 2. Conventional transthoracic echocardiographic measures and 2D speckle tracking echocardiography indices of the patients according to hs-cTnI levels

	Stroke patients with elevated troponin (n=33)	Stroke patients with normal troponin (n=49)	p
Left atrium (mm)	42.2±8.3	36.3±5.3	<0.001
LAVI (mL/m^2)	30.3±13.3	21.3±8.9	<0.001
LVEDD (mm)	47.3±7.3	46.1±5.4	0.400
LVESD (mm)	28.6±7.4	27.3±5.6	0.566
IVS (mm)	12.8±2.2	12.7±2	0.970
PW (mm)	10.9±1.6	10.2±1.4	0.115
LVEF (%)	51.4±10.3	59.2±6.6	<0.001
GLS (%)	14.6±4.3	17.5±4.0	0.003
GCS (%)	17.6±5.1	21.6±5.4	0.002
BCS (%)	16.1±5.2	19.7±6.3	0.010
MCS (%)	18.3±6.6	22±6.4	0.017
ACS (%)	18.9±6.5	23.5±6.4	0.003

hs-cTnI: high sensitive cardiac Troponin I; LAVI: Left atrial volume index; LVEDD: Left ventricular end-diastolic diameter; LVESD: Left ventricular end-systolic diameter; IVS: interventricular septum thickness; PW: posterior wall thickness; LVEF: left ventricular ejection fraction; GLS: Global longitudinal strain; GCS: Global circumferential strain, BCS: Basal circumferential strain; MCS: Mid circumferential strain; ACS: Apical circumferential strain.

Twenty-three of the patients (28%) had insular cortex involvement. The general characteristics, laboratory parameters, conventional transthoracic echocardiographic measures, and 2D speckle tracking echocardiography indices of the patients according to insular cortex involvement are shown in Table 3. The patients with insular cortex involvement had significantly higher NIHSS scores, hs-cTnI, and hs-CRP levels, and higher LAVI values compared to those without involvement. There were not any significant differences in LVEF and 2D-STE parameters between the groups.

Table 3. The general characteristics, laboratory parameters, conventional transthoracic echocardiographic measures, and 2D speckle tracking echocardiography indices of the patients according to acute ischemic stroke with insular cortex involvement

	With insular cortex involvement (n=23)	Without insular cortex involvement (n=59)	p
Age (years)	70.3±15.5	64.4±12.6	0.086
Male sex (n – %)	6 (26.1%)	30 (50.8%)	0.051
Body mass index (kg/m^2)	26.3±4.6	27.5±4.3	0.185
Hypertension (n – %)	17 (73.9%)	40 (67.8%)	0.790
Diabetes (n – %)	5 (21.7%)	24 (40.7%)	0.129
Hyperlipidemia (n – %)	5 (21.7%)	15 (25.4%)	1
Coronary artery disease (n–%)	7 (30.54%)	15 (25.4%)	0.782
NIHSS	8.2±5.9	4.1±3.3	0.006
Glucose (mg/dL)	117.8±43.2	128.5±49.5	0.488
Creatinine (mg/dL)	0.93±0.48	0.93±0.45	0.859
Total cholesterol (mg/dL)	203±59	194±46	0.494
LDL cholesterol (mg/dL)	134±51	122±39	0.289
hs-cTnI (ng/mL)	0.22±0.614	0.07±0.162	0.026
Elevated hs-cTnI (n-%)	14 (60.9%)	19 (32.2%)	0.024
hs-CRP (mg/L)	28.7±36	13.2±23.7	0.012
NT-proBNP (pg/mL)	3984±9274	2430±6950	0.622
ST segment/T wave changes (n–%)	10 (43.5%)	32 (54.2%)	0.464
Left atrium (mm)	41.1±8.5	37.7±6.6	0.069
LAVI (mL/m^2)	31.1±12.8	22.5±10.3	0.003
LVEDD (mm)	45.9±5.1	46.8±6.6	0.565
LVESD (mm)	27.3±7	28±6.1	0.651
IVS (mm)	12.9±2.1	12.7±2.1	0.777
PW (mm)	10.7±1.6	10.4±1.4	0.545
LVEF (%)	54.5±10.9	56.7±8.3	0.316
GLS (%)	16.1±4.5	16.4±4.3	0.790
GCS (%)	19.7±6	20.1±5.5	0.800
BCS (%)	18.7±5.5	17.9±6.4	0.639
MCS (%)	19.9±7.1	20.6±6.6	0.677
ACS (%)	20.5±8	22.0±6.2	0.365

NIHSS: National Institutes of Health Stroke Scale; LDL: Low-density lipoprotein; hs-cTnI: high sensitive cardiac Troponin I; hs-CRP: High sensitive C reactive protein; NT-proBNP: N terminal pro-brain natriuretic peptide; LAVI: Left atrial volume index; LVEDD: Left ventricular end-diastolic diameter; LVESD: Left ventricular end-systolic diameter; IVS: interventricular septum thickness; PW: posterior wall thickness; LVEF: left ventricular ejection fraction; GLS: Global longitudinal strain; GCS: Global circumferential strain; BCS: Basal circumferential strain; MCS: Mid circumferential strain; ACS: Apical circumferential strain.

Discussion

In our study, we investigated neurogenic stunned myocardium and its effect on LV functions by 2D-STE. The most important result of our study was that we found significantly low GLS and GCS values in ischemic stroke patients with high troponin levels. Studies have shown that patients with NSM may exhibit reduced GLS, which reflects myocardial deformation along the long axis of the LV.[9] Reduced GLS indicates impaired systolic function and can be an early marker of myocardial dysfunction in NSM.[11] Data on the effects of NSM on myocardial strain assessment were mostly obtained from hemorrhagic stroke studies. Unfortunately, there are not many studies on the use of 2D-STE in NSM in acute ischemic stroke.

There are conflicting reports on whether there is a correlation between cardiac biomarker levels and the extent of LV dysfunction. Although there are several publications stating that the increase in troponin I and LV dysfunction are correlated, some studies have also reported only moderate troponin increase despite marked contractility abnormalities.[12,13] On the other hand, increased BNP levels correlate with LV dysfunction, reduced EF, pulmonary edema, and death.[14]

In NSM, regional wall motion abnormalities extend beyond a single epicardial vascular distribution, especially in the basal sections with intense sympathetic innervation, giving rise to the designation of inverted Takotsubo cardiomyop-

athy.[1,15,16]. Histological changes, such as contraction band necroses, are most intense in the subendocardial regions of the heart that correspond to the areas of sympathetic innervation.[3] In our study, all regional circumferential strain values were significantly lower in the high troponin group, and therefore we could not reveal a segmental strain difference. However, a recent study showed a lower longitudinal strain value in the basal segment in ischemic stroke patients, which is suggested to be even able to distinguish between stroke types (ischemic or hemorrhagic).[9]

NSM is considered to be one of the stress-related cardiomyopathies, including Takotsubo cardiomyopathy, and cardiomyopathies associated with pheochromocytoma, exogenous catecholamine intake, and critical conditions such as sepsis.[12] The exact pathophysiological mechanisms underlying NSM are still not fully understood and the observed reduction in myocardial strain in NSM patients can be attributed to several factors. Firstly, the sympathetic hyperactivity and catecholamine surge commonly seen in acute neurologic events can lead to myocardial stunning and dysfunction.[17] Catecholamine discharge is usually an increase in local catecholamine release from nerve endings in the myocardium rather than a significant increase in systemic catecholamine levels.[3,18] Sympathetic overactivity is also another important factor causing NSM.[5] Since vagal stimulation inhibits inflammation, parasympathetic system dysfunction and unopposed sympathetic overactivation lead to uncontrolled myocardial inflammation and damage.[1,19,20] Additionally, the neurohormonal activation and dysregulation of the autonomic nervous system seen in NSM may contribute to abnormal myocardial strain.[21]

We showed significantly high troponin levels in stroke patients with insular cortex involvement. The insular cortex is one of the most important regulatory centers of cardiovascular functions, and its damage results in complex events such as autonomic dysfunction, catecholamine discharge, and neuroinflammation.[3,20] Although troponin elevation has been reported more frequently in strokes with insular cortex involvement, there are conflicting findings as to which of the right and left hemisphere involvements causes NSM more frequently.[5,22,23] Due to the small number of patients, we could not show the difference in involvement between the right and left insula.

Although there are studies reporting a correlation between troponin levels and stroke severity,[24] we did not find any significant difference in NIHSS between the groups according to troponin levels. On the other hand, we found significantly elevated NIHSS scores in stroke patients with insular cortex involvement. Unlike our finding, Liesirova K et al. found an association between troponin elevation and stroke severity but not with stroke localization.[25] Although NIHSS scores are affected by both infarct size and location, infarct location may be more important than infarct size for NSM development.[25]

Myocardial strain assessment has the potential to aid in risk stratification, prognostication, and treatment decisions in patients with acute neurologic events.[26] Additionally, monitoring changes in myocardial strain over time may help guide therapeutic interventions and evaluate the effectiveness of interventions aimed at improving cardiac function.[27] Further research is needed to unravel the underlying mechanisms and optimize the clinical utility of myocardial strain assessment in NSM in ischemic stroke.

Study Limitations

The main limitation of our study was the lack of a consensus definition of NSM. In our study, we used elevated troponin levels as a sign of NSM as most studies defined NSM based on data from troponin, ECG, and TTE. The small sample size and being a single-center study were also the limitations of our study. We did not evaluate the reversibility of LV dysfunction with repeating transthoracic echocardiography; which was another limitation of the study.

Conclusion

In acute ischemic stroke, troponin levels may be elevated, which may be accompanied by a decrease in GLS and GCS. These may be a sign of neurogenic stunned myocardium. Although troponin levels are elevated more in patients with insular cortex involvement, no significant differences in strain values were observed. Myocardial strain assessment offers a promising tool for evaluating myocardial dysfunction in NSM and has the potential to enhance risk stratification and clinical management strategies in this population. Further research is needed to better understand the pathophysiology of NSM and its impact on various strain parameters and optimize the clinical utility of myocardial strain assessment in NSM patients.

Disclosures

Ethics Committee Approval: The study was approved by the ethics committee of the University of Health Sciences, Umraniye Education and Research Hospital on 11.02.2021 with decision number 07.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept – C.I.; Design C.I.; Supervision – B.O., N.P.; Materials – C.I., Z.D., E.E; Data collection &/or processing – C.I., E.E.; Analysis and/or interpretation –C.I., Z.D., B.O.; Literature search – C.I.; Writing – C.I., B.O.; Critical review – N.P., B.O.

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