Original article

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Can emergency medicine residents diagnose neurogenic stunned myocardium in the emergency department by Focused Assessment Diagnostic Echocardiography?

Ehsan Karimialavijeh, Aaliyeh Zarrabi, Hamideh Akbari, Mehrad Aghili, Elnaz Vahidi, Fatemeh Rasooli

Abstract

***Purpose*: To assess the ability of trained emergency medicine (EM) residents to diagnose neurogenic stunned myocardium (NSM) in patients with acute intracranial events in the emergency department (ED) using the Focused Assessment diagnostic Echocardiography (FADE).**

***Methods*: This prospective cross-sectional study was conducted between February 2018 and January 2019. First, four postgraduate year-3 (PGY-3) EM residents were trained through participation in 10 hours of theoretical FADE education and 20 hands-on FADE on ED patients. Then, adult patients (aged over 18 years old) with acute cerebrovascular accidents (CVAs) were enrolled in the study. Patients with a history of prior cardiac disease, concurrent CVA and ST-elevation myocardial infarction (STEMI), and cardiopulmonary arrest in the ED were excluded. The results of echocardiography by the cardiologists were considered as the gold standard.**

***Results*: Eighty-five cases were enrolled in the final analysis. The mean age of participants was 62.7 years, and 47 (55.3%) were male. There was a good agreement between the EM residents and cardiologists in diagnosing left ventricular (LV) dysfunction (Cohen's Kappa=0.7, 95%CI=0.65-0.8). Troponin I was positive in 3 (3.5%) patients. Based on the diagnosis of cardiologists, all three patients had heart failure and received a definitive diagnosis of NSM. Whereas, according to the FADE exam by EM residents, 2 out of 3 cases with positive enzyme had heart failure (interrater agreement=66.6%).**

***Conclusion*: Our study showed that trained EM residents could use the FADE exam to diagnose the NSM in CVA patients in the ED.**

Key words: FADE, echocardiography, neurogenic stunned myocardium, emergency medicine, stroke.

Introduction

Neurogenic stunned myocardium (NSM) is a sudden onset myocardial dysfunction, occurs after various acute intracranial events as a consequence of the imbalance in the autonomic nervous system. (1) NSM has mostly been reported in patients with subarachnoid hemorrhage (SAH), but it has also been detected in other neurologic insults including ischemic stroke, encephalitis, myelitis, Guillain-Barre syndrome, status epilepticus, head trauma, post neurosurgery, acute hydrocephalus, and other acute central nervous system (CNS) injuries. (2-12)

Clinical manifestations of NSM comprises acute left ventricular (LV) failure, hypotension, pulmonary edema, and cardiogenic shock. Electrocardiogram (ECG) may reveal arrhythmias or conduction disorders. (13) Due to the myocardial dysfunction, the plasma level of cardiac biomarkers will increase, including creatine kinase (CK) and creatine kinase-MB (CK-MB), troponin I, and B-type natriuretic peptide (BNP). (14,15) The echocardiography in NSM shows a decreased systolic and diastolic function of the left ventricle. The most common echocardiographic abnormalities include

From Tehran University of Medical Sciences, Tehran, Iran, Hospital and Prehospital Emergency Research Center, Tehran, Iran (Ehsan Karimialavijeh, Aaliyeh Zarrabi, Hamideh Akbari, Mehrad Aghili, Elnaz Vahidi, Fatemeh Rasooli).

Address for correspondence:
Fatemeh Rasooli, MD
Hospital and Prehospital Emergency Research Center, Department of Emergency Medicine, Sina Hospital
Imam Khomeini Ave, Tehran, Iran
Tel: +989125041003
Fax: +98216634 8561
Email: fa.rasooli2@gmail.com

global hypokinesis of the left ventricle, ventricular wall motion abnormality, or hypokinesis of the basal and intraventricular segments with apical sparing. (16)

NSM will increase the risk of fatal complications and poor neurologic outcome. Thus, the early diagnosis of this situation is crucial. (17) Fortunately, if the underlying neurologic condition comes under control by proper interventions, the NSM will completely resolve, and the normal left ventricular function will restore within a few days or several weeks. (18,19)

Although curable, NSM is underdiagnosed in many patients; (15) in a study of 92 ICU patients, NSM affected 28% of patients. (16) Another study of 569 patients with ischemic strokes, reported NSM in 7 patients. (20)

Focused Assessment Diagnostic Echocardiography (FADE) is a limited transthoracic echocardiography that examines the left ventricular ejection fraction (LVEF), wall motion abnormalities, right ventricle, and valvular diseases. (17,18) FADE is well known among critical care and emergency medicine (EM) specialists, but studies on the ability of FADE to diagnose the NSM are missing. (19)

In this study, we aimed to assess the ability of trained EM residents to diagnose NSM in patients with acute intracranial events in the emergency department (ED) using FADE. Besides, the short term outcome of the patients with NSM was evaluated.

Methods

Study design and setting

This prospective cross-sectional study was conducted between February 2018 and January 2019 at the ED of a university-affiliated urban hospital of Tehran University of Medical Sciences (TUMS) in Iran.

The Ethics Committee of TUMS approved the conduct of the study (reference number: 42851300298), and informed consent was obtained from the participants or their legal guardians. Before embarking on the study, four postgraduate year-three (PGY-3) EM residents were trained through participation in 10 hours of theoretical FADE education and 20 hands-on FADE on ED patients under the supervision of two board-certified emergency medicine faculty member that expert in the FADE. The results of echocardiography by cardiologists were considered as the gold standard.

EM residents had prior experience in cardiac ultrasound. They had basic knowledge of the echo machine and performed bedside echocardiography to measure LVEF or detect cardiac tamponade through the years of their practice in the ED.

To diagnose the NSM, EM residents assessed the LV systolic and diastolic function, as well as wall motion abnormalities (hypokinesis, dyskinesis, or akinesis of a segment compared to the other contracting segments of the myocardium).

LV systolic function was measured using the Simpson method (LV cavity was traced at the end of diastole and systole in the four-chamber and two-chamber views, then the machine's software calculated the LVEF).

LV diastolic function was assessed using the pulse wave (PW) and tissue doppler imaging (TDI). Mitral inflow velocity was evaluated by placing the PW doppler at the tip of the mitral valve. Mitral annular velocity was measured by placing the TDI at the medial (septal) mitral annulus. An E/E¢ ratio >15 was considered as LV diastolic dysfunction.

Wall motion abnormalities were assessed visually on the parasternal long-axis and short-axis, apical four-chamber, and apical two-chamber views. Wall motion abnormalities were described as hypokinesis, dyskinesis, or akinesis of a segment compared to the other contracting segments of the myocardium.

Patients were followed up for the outcomes of the mortality and cardiac events within a month of the ED admission by phone calls.

We used strict criteria for diagnosing NSM to increase the specificity of the diagnostic criteria. Thus, the criteria of NSM were defined as a cerebrovascular accidents (CVA) patient that had three conditions: 1. LV dysfunction (systolic dysfunction, diastolic dysfunction, wall motion abnormality); 2. Ischemic changes on ECG; 3. Rising serum troponin levels.

Troponin was measured in a 2-hour interval using the VIDAS® high-sensitivity troponin I, in which over 10 ng/dl difference between values at 0 and 2 hours considered as a positive test.

Ischemic ECG changes included inverted, flat, or hyperacute T-wave and ST-segment depression.

Two research nurses recorded the following data and transferred data into an Excel worksheet (Microsoft® Office 365): gender, age, type of CVA, ECG changes, troponin results, findings of FADE, and findings of cardiologist echocardiography.

Participants

Participants were enrolled in the study by convenience sampling. Adult patients (aged over 18 years old) with acute CVAs, including ischemic infarction, intracranial hemorrhage, and subarachnoid

hemorrhage, were enrolled in the study. Patients with a history of prior cardiac disease (systolic or diastolic heart failure, valvular heart disease, congenital heart diseases, coronary arterial diseases, atrial fibrillation, or other arrhythmias that were under controlled by antiarrhythmic agents), concurrent CVA and ST-elevation myocardial infarction (STEMI), and cardiopulmonary arrest in the ED were excluded.

Sample size

The sample size of 81 participants was calculated based on the LVEF agreement estimation in the prior reports, (21,22) the accuracy of 0.2, and confidence interval (CI) of 95%.

Data analysis

SPSS (version 22, Chicago, IL, USA) was used to analyze the data. Independent t-test performed for comparison of the continues quantitative variables where p-value <0.05 was considered statistically significant. Interclass correlation coefficient (ICC) with a two-way mixed model and 95% CI was used for comparing LVEF measurements, and weighted kappa coefficient used for evaluating the agreement between EM residents and cardiologists in the diagnosing LV failure.

Results

During the study period, the EM residents examined a total of 107 patients with CVAs and performed the FADE exams, and 85 cases were enrolled in the final analysis (**Figure 1**).

The mean age of participants was 62.7 years, and 47 (55.3%) were male. In the majority of the patients (87.1%), the brain injury was ischemic, two (2.4%) patients had concurrent ischemia and hemorrhage in the brain, and 9 (10.6%) patients had hemorrhagic strokes (**Table 1**).

According to the American Heart Association classification, (23) ventricular ejection fraction (EF) was divided into four categories: hyperdynamic=LVEF greater than 70%, normal=LVEF 50% to 70%, mild dysfunction=LVEF 40% to 49%, moderate dysfunction=LVEF 30% to 39%, and severe dysfunction=LVEF less than 30%. But, in order to simplify the diagnosis for the residents, we used the following categorization: normal=LVEF 50% to 70%, mild dysfunction=LVEF 40% to 49%, and moderate to severe dysfunction=LVEF<40%. (23)

There was a good agreement between the EM residents and cardiologists in measuring the LVEF (ICC of 0.9, 95% CI=0.94 to 0.86). The mean difference in the estimate of LVEF by the EM residents and the cardiologists was not statistically significant (p-value=0.160) (**Figure 2**).

Based on this classification and the LVEF assessment by the cardiologists, 50 (58.8%) patients had normal LV function, 9 (10.58%) had mild LV systolic dysfunction, and 26 (30.58%) had moderate to severe LV dysfunction. According to the EM residents' measurements, these numbers were 54 (63.52%), 5 (5.88%), and 26 (30.58%), respectively. The agreement between the EM residents and the cardiologists in the classification of the cardiac dysfunction was good (weighted kappa coefficient =0.7) (**Table 2**).

Of patients with LV dysfunction (based on the cardiologist echocardiography), three cases (3.5%) had rising troponin levels. All of them had ischemic ECG changes and received a definitive diagnosis of NSM. However, according to the diagnosis of trained EM residents, 2 out of 3 cases with rising troponin levels had LV dysfunction (**Table 3**). The percentage of agreement in the definite diagnosis of NSM between the two groups was 66.6%.

Discussion

Current data indicates that the culprit mechanism of NSM is supraphysiologic catecholamine levels. (13,24,25) The diagnosis of NSM is made on the ischemic T wave and ST-segment changes, positive plasma troponin assay, and echocardiography. (26,27)

Early diagnosis of the NSM is of undisputed importance. NSM can cause hypotension and interrupt blood flow to the ischemic penumbra. Also, NSM can be the underlying cause of the LV thrombosis formation. (28)

The aim of the present study was to compare the agreement between the FADE exam performed by the trained EM residents and echocardiography performed by cardiologists in the detection of cardiac dysfunction in patients with acute brain insult to realize how much trained EM residents can diagnose NSM in the ED.

Based on our results, the EM residents had a good agreement with cardiologists in identifying the patients with heart failure (Cohen’s kappa=0.7). Nevertheless, based on the findings of the cardiac ultrasound, ECG, and cardiac troponin, EM residents diagnosed two cases of NSM and cardiologists diagnosed three cases of NSM among 85 patients with acute CVAs (interrater agreement=66.6%). This low agreement can be a result of the limited sample size of the present study.

To our best knowledge, there is no prior study to evaluate the ability of EM residents in diagnosing NSM by the FADE exam in the ED. In one study, Townsend, et al reported that surgical residents are

able to perform the FADE exam after participation in 4 hours of simulation. (29)

Although there are more accurate methods for the diagnosis of cardiac dysfunction such as speckle tracking echocardiography (STE), it requires higher levels of education and expertise and is beyond the current curriculum of EM residency training. (27)

NSM in the ED is underreported, and EM physicians should consider this entity in the patients with acute brain injury to expedite the diagnosis and improve team work with other specialists to minimize the damage of the NSM in the patients with brain injury. (28)

This study had several limitations. In comparison with the hemorrhagic strokes and SAH, NSM is less severe in ischemic strokes. (3) Based on Wira, et al (30) study, National Institutes of Health Stroke Scale (NIHSS)>10 is correlated with higher in-hospital mortality of NSM patients. In the present study, we did not evaluate the correlation of the NIHSS and diagnosis of NSM. In addition, the effects of thrombolysis by tissue plasminogen activator in the treatment of ischemic strokes may affect the incidence of NSM, (27) but we did not assess the type of treatments in CVA patients. Also, the cardiac effects of the NSM change over time, but in this study, the FADE exam was performed during the 48 hours after ED admission, and we did not repeat the exam after the transfer of the patients to the neurology ward or intensive care unit (ICU). Last but not least, coronary arterial diseases are common among patients with CVA, and many of them have underlying cardiac problems such as heart failure and arrhythmias. Although in the present study we excluded all patients with prior cardiac diseases and used rising high-sensitive troponin levels at 0 and 2 hours intervals as a criterion to diagnose new-onset cardiac events, it is still possible that underlying cardiac diseases have impacted our findings.

Conclusion

Our study showed that EM residents could use the FADE exam to diagnose NSM in patents with brain insults in ED. Further researches are needed to find simple, fast, and reliable approaches for detecting NSM in ED.

Table 1. Demography of the participants

Age (year, mean±SD)	62.7±15.7
Gender (n, %) - Male - Female	 47 (55.3) 38 (44.7)
Type of the stroke (n, %) - Ischemic - Hemorrhagic - Ischemic and hemorrhagic	 74 (87.1) 9 (10.6) 2 (2.3)
ECG findings - T-wave inversion - Flat T-wave - Hyperacute T-wave - ST-segment depression	 54 (63.5) 15 (17.6) 8 (9.4) 65 (76.5)
Difference in troponin levels at 0 and 2 hours (n, %) - ≥10 - <10	 3 (3.5) 82 (96.5)

Legend: ECG=electrocardiogram.

Table 2. Type of cardiac dysfunction in CVA patients diagnosed by the EM residents and cardiologists

Findings	EM residents (n, %)	Cardiologists (n, %)	Cohen's kappa (95%CI)
LV function (n, %) - Normal - Systolic dysfunction - Diastolic dysfunction - Systolic and diastolic dysfunction	 54 (63.5) 24 (28.2) 2 (2.4) 5 (5.9)	 50 (58.8) 20 (23.5) 3 (3.6) 12 (14.1)	0.7 (0.65-0.8)
Wall motion abnormality (n, %)	36 (42.4)	42 (49.4)	0.8 (0.72-0.86)

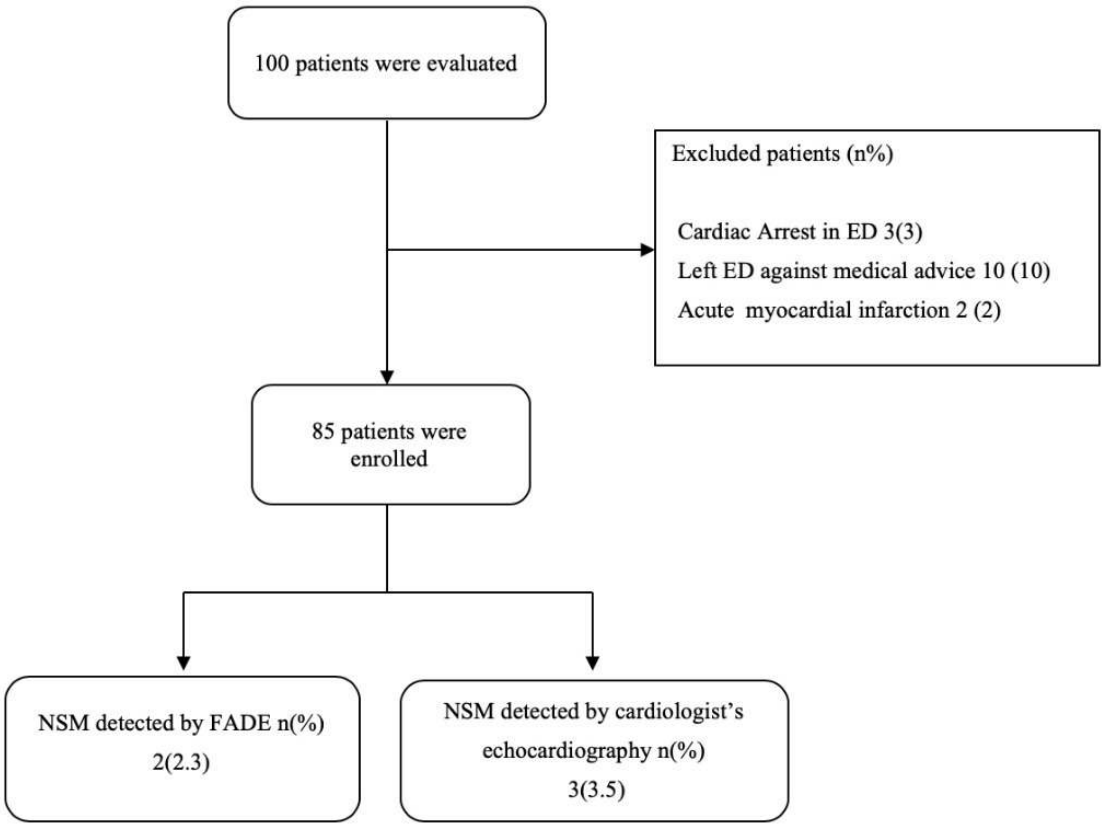
Legend: CVA=cerebrovascular accidents; EM=emergency medicine; LV=left ventricular; CI=confidence interval.

Table 3. Characteristics of the NSM patients

Case no	Gender	Age	Type of CVA	Anatomical location	Type of cardiac dysfunction	LVEF	In-hospital mortality
1	Male	85	Ischemic	Left MCA	Systolic	20	No
2	Male	86	Ischemic	Left temporoparietal	Systolic	35	No
3	Male	62	Ischemic	Right partial	Systolic and diastolic	20	Yes

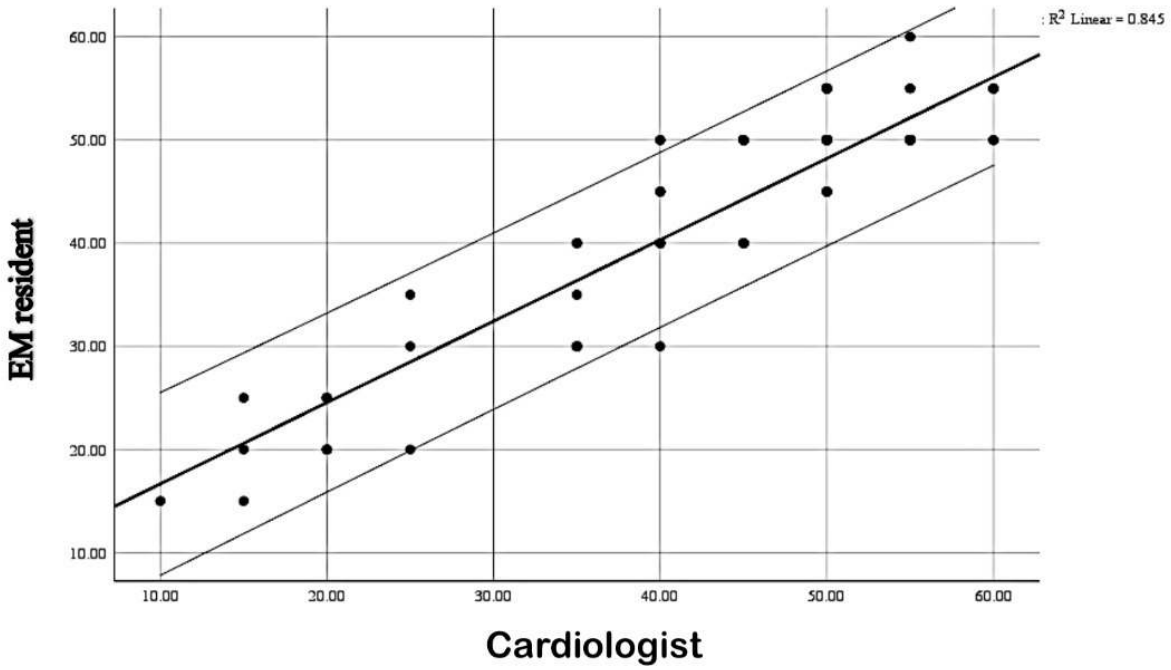
Legend: NSM=neurogenic stunned myocardium; CVA=cerebrovascular accidents; MCA=middle cerebral artery; LVEF=left ventricular ejection fraction.

Figure 1. Flow chart of the participants



Legend: ED=emergency department; NSM=neurogenic stunned myocardium; FADE=Focused Assessment diagnostic Echocardiography.

Figure 2. Dispersion plot between EM residents and cardiologists for the LVEF



Legend: EM=emergency medicine; LVEF=left ventricular ejection fraction.

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