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Original Article

Assessment of Left Ventricle Systolic Function by 2D Speckle Tracking Echocardiography in Patients with Subarachnoid Hemorrhage

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ABSTRACT

Background: Subarachnoid hemorrhage [SAH] is a neurological emergency with high mortality rates; Cardiac dysfunction with unfavorable neurological outcomes is common association.

The Aim of The Work: Subclinical left ventricular systolic dysfunction and its association with the outcome of SAH was assessed.

Patients and Methods: 120 cases were involved in this study divided in to two groups 60 healthy control group and 60 Patients with non-traumatic aneurysmal SAH. They were scaled according to Hunt & Hess scale and analyzed for age, sex, history of hypertension, diabetes mellitus, and smoking. ECG was done for all cases, Serum cholesterol, LDLc and troponin I were measured. Conventional and 2D speckle tracking echo were done for the following parameters, ejection fraction EF [by both M mode and biplane], LVEDD, LVESD, LVFS, RWMA and LV GLS.

Results: Female gender was more affected beside, significant increases hypertension, smoking, s. cholesterol, LDLc, and troponin I. ECG showed significant ST- T wave changes and prolongation in QTc . Conventional echocardiography showed no significant changes regarding EF, FS, LVEDD and LVESD while, there were significant RWMA in the form of anteroseptal hypokinesia. Strain imaging of LV showed significant reduction of LV GLS in SAH cases which indicates subclinical dysfunction. Hunt& Hess scaling showed about 28.3% of cases had 3 grades which indicated bad prognosis of these cases. Positive correlation between Hunt & Hess scale and hypertension, troponin I and LV GLS which confirms the association between myocardial damage and bad clinical outcome of SAH.

Conclusion: Subclinical myocardial damage and dysfunction is common complication of SAH, which is associated with poor prognosis SAH.

Keywords: Subarachnoid Hemorrhage, Speckle Tracking; Echocardiography; Global Longitudinal Strain.

INTRODUCTION

Subarachnoid hemorrhage [SAH] is considered difficult disease, especially aneurysmal bleeding with high serious outcomes [1]. Multiple extracranial complications including cardiac, pulmonary, inflammatory and renal effects [2]. The mechanism of systemic manifestations in SAH is unknown. However, acute SAH triggers have been postulated like, widespread neuroendocrine reactions, inflammatory and immune-mediated mechanisms that are responsible for its systemic actions including the myocardium. The cardiac dysfunction in SAH was attributed to various neural and humoral mechanisms. There is strong activation in the regions of hypothalamus, insula and brain stem Immediately after SAH which lead to activation of sympathetic nerve endings causing release of norepinephrine [3].

THE AIM OF THE WORK

In this study we assessed the left ventricular [LV] function by both 2D and speckle tracking echocardio-graphy and the impact of presence of LV systolic dysfunction on the prognosis of SAH.

PATIENTS AND METHODS

This study included 120 patients, whom attended to Echocardiography laboratory of cardiology department in Al-Hussein University from September 2019 to July 2021. These patients were classified into Group [A]: Healthy control group [60] patients with normal neurological function, normal ventricular function and ECG, and no cardiac risk factors, Group [B]**:** Included [60] patients who presented with non-traumatic SAH at our hospital, the criteria for inclusion of cases were; above18 years old, computerized tomography showing non-traumatic SAH and Cardiology evaluation including ECG within 48 hrs. of admission, while we Excluded Patients with a previous SAH, stroke, traumatic brain injury and other intracerebral processes, patients with pacemaker, coronary artery disease**,** heart failure, previous cardiac surgery, patients with imminent clinical signs of brain death, and patients with poor-quality of echocardiographic images for strain analysis. Demographic data including age, sex, history of hypertension [4]**,** diabetes mellitus [5]**,** smoking [6]**.** Clinical parameters, scaling of SAH according to Hunt and Hess classification as follow; Grade 1 Asymptomatic, mild headache, Grade 2 Moderate to severe headache Grade 3 Mild mental status change, mild focal neurologic deficit, Grade 4 Stupor, or moderate-to-severe hemiparesis Grade 5 Comatose, or decerebrate rigidity [9]. Resting surface 12 leads ECG was done for all patients [10] and laboratory results [serum cholesterol, LDLc and high sensitive troponin I] were analyzed [7, 8 ,11]**.** Echocardiography were done for all cases, Examination were performed with a "Philips iE33 X Matrix" ultrasound machine using "S5-1" matrix array transducers [Philips Medical Systems, Andover, USA] equipped with STE technology, using a multi frequency [1- 5 MHz]. ECG-gated examination used to help us during image acquisition & later analysis. The images were digitally stored for analysis. Recordings and calculations were made according to the recommendations of the American Society of Echocardiography [12,13]**.** The following measurements were measured; LV systolic function: by calculating Ejection Fraction [EF] using both M mode and modified biplane Simpson's method, regional Wall Motion Abnormalities.

2-D Speckle tracking echocardiography study was done to measure LV global longitudinal strain [GLS] [14]. This study was approved by the Ethical Committee of Al Azhar University Hospitals, and an informed consent was obtained from all enrolled participants. The study protocol was designed in accordance with The Code of Ethics of the World Medical Association [Declaration of Helsinki] for experiments involving humans WMA General Assembly, 2018. Statistical analysis of data was done using the Statistical Package for Social Sciences [version 21; SPSS Inc., Chicago, IL, USA]. Data were expressed as mean ± SD and percentages. Independent t student test, chi-square test, and ANOVA test were used for comparison of groups and Pearson coefficients test for correlation correlations between parameters. P-values <0.05 were considered significant

RESULTS

The age of study whole participants were 47.7±7.6, with 57.5% female and 42.5% males, 13.3% smokers and 86.7% nonsmokers, 7.5% diabetic and 92.5 % non-diabetic, 34.2% have high blood pressure,65.8% have normal blood pressure, and with serum cholesterol and LDL levels of 189.1 ± 3.4 mg/dl and 89.7 ± 4.5 mg/dl respectively as shown in table [1].

No significant difference in demographic data between cases and control regarding age and sex except for 65% of cases was females. 26.7% of cases were smokers with significant increase [P<0.0001] in smoking among cases. No significant changes regarding diabetes, serum cholesterol and LDL between cases and control groups [P: 0.729, 0.122 and 0.408 respectively]. Otherwise, there was significant increase in incidence of hypertension among cases compared to control group [P<0.0001] as shown in tables [2]. In addition to, significant increase [P<0.0001] in serum HsTn I in SAH cases compared to control group as shown in table [3].

There was significant increase in Hunt Hess scale in SAH cases [P value < 0.0001] in the form of 31.7% for grade 1, 40% for grade 2 and 28.3% for grade 3. In addition to, significant ECG changes were observed in the form of nonspecific ST-T wave changes and prolongation in corrected QT interval [P value < 0.0001 in both parameters] as shown in table [3].

In the current work, there were no significant changes between cases and control regarding the following parameters ejection fraction by both biplane and M mode, LVEDD, LVESD and FS [P value: 0.206, 0.139, 0.112,

0.079 and 0.246 respectively]. While, there were significant RWMA in the form of anteroseptal hypokinesia. Speckle tracking echo showed significant decrease in left ventricle global longitudinal strain [LVGLS] in SAH cases compared to control group [P<0.001] as shown in table [3].

Positive correlations were observed between Hunt Hess grade and hypertension, Hs Tn I and LVGLS. While, there were negative correlation between Hunt Hess grade and diabetes, serum cholesterol, LDLc, EF [by both M mode and biplane], LVEDD and LVESD as shown in table [4].

Table [1]: Characteristics of the whole participants in the study [N = 120]:

The variable		*Statistical Measures*
Age: [year]	Mean ± SD; [Range]	47.7 ± 7.6 ; [39 – 57]
Sex [n, %]	Male Female	51[42.5%] 69 [57.5%]
Smoking [n, %]		16 [13.3%]
Diabetes [n, %]		9 [7.5%]
Hypertension [n, %]		41 [34.2%]
Lipid profile[mean±SD; range]	Cholesterol [mg/dl] Low density lipoproteins [mg/dl]	189.1 ± 3.4; [178 – 196] 89.7 ± 4.5; [79 – 100]

Table [2]: Frequency distribution of different demographic characteristics among the cases and control groups

The variable		Cases [60]	Control [60]	P value
Age: [year]	Mean ± SD; [Range]	47.8 ± 7.2; [29 – 46]	47.7 ± 6.4; [29 – 45]	0.953
Sex [n, %]	Male Female	21 [35.0%] 39 [65.0%]	30 [50.0%] 30 [50.0%]	 0.097
Smoking [n, %]		16 [26.7%]	0 [0.0%]	<0.001*
Diabetes [n, %]		5[8.3%]	4[6.7%]	0.729
Hypertension [n, %]		37[61.7%]	4[6.7%]	<0.001*
Lipid profile[mean±SD; range]	Cholesterol [mg/dl] Low density lipoproteins [mg/dl]	189.6 ± 3 [180-196] 89.4±5.8 [79-99]	188±6 [178-194] 90.1±2.6 [87-100]	0.122 0.408

SD: Standard Deviation, %: Percentage, *: significant.

Table [3]: Comparison between cases and controls, regarding Echocardiographic data

Items		cases	control	Test	P .value
EF [bi plane]	Mean±SD	58.5 ± 5.5	59.5 ± 2.9	1.016	0.206
RWMA [n, %]	Present Absent	8 [13.3%] 52 [86.7%]	0 [0.0%] 60 [100.0%]	8.571	0.003*
EF [By M Mode]	Mean±SD	58.2 ± 6.4	59.5 ± 2.9	1.488	0.139
LVEDD	Mean ± SD	4.9 ± 0.3	4.8 ± 0.4	1.600	0.112
LVESD	Mean ± SD	3.2 ± 0.2	3.1 ± 0.18	1.771	0.079
LV FS [%]	Mean ± SD	32.4 ± 2.4	31.9 ± 0.8	1.166	0.246
QTc	Mean ± SD	433.6 ± 41.4	382.7 ± 21.5	8.441	<0.001*
Nonspecific ST-T wave changes [n,%]	Present Absent	46[67.7%] 14 [23.3%]	0 [0.0%] 60 [100.0%]	74.59	<0.001*
LVGLS	Mean ± SD	20.8 ± 4.3	22.8 ± 1.9	3.451	0.001*
Hs tn I	Mean ± SD	0.04 ± 0.009	0.02 ± 0.004	5.439	<0.001*
Hunt Hess grade [n,%]	Normal Grade 1 Grade 2 Grade 3	0[0.0%] 19[31.7%] 24[40.0%] 17[28.3%]	60[100.0%] 0 [0.0%] 0 [0.0%] 0 [0.0%]	120.0	<0.001*

SD: Standard Deviation, %: Percentage, *: significant.

Table [4]: Correlation between the Hunt Hess grade and the different parameters among the SAH cases

	Hunt Hess grade	
The correlation variables	Correlation coefficient [r]	P value
DM	-0.009	0.113
HTN	0.617	<0.001*
S. Cholesterol	-0.196	0.161
LDLc	-0.037	0.688
Hs Tn I	0.519	<0.001*
LVEDD	0.126	0.179
LVESD	-0.0079	0.090
EF [MM]	-0.155	0.067
EF [biplane]	-0.179	0.052
LVGLS	-0.481	<0.001*

DM: Diabetes mellitus; HTN: Hypertension; LDLc: Low Density lipoprotein cholesterol: HsTnI: High sensitivity troponin-1; LVEDD: left ventricle end diastolic dimension; LVESD: left ventricle end systolic dimension; EF: Ejection fraction; LVGLS: left ventricle global longitudinal strain.

DISCUSSION

Aneurysmal subarachnoid hemorrhage [SAH] is considered a neurologic emergency with stable incidence over the past 30 years **[15],** the worldwide incidence of about 10.5 per 100,000 person-years **[16].** Frequent occurrence of cardiac dysfunction was observed after acute SAH [17]. Patients with SAH often develop complications from bleeding that lead to the high mortality rate of this disease. Complications in the form of hydrocephalus, seizures, cerebral ischemia, tissue shifts and herniation, hyponatremia, cardiac anomalies, and respiratory depression are can result [18].

Large proportion of ICU admitted SAH patients showed echocardiographic and biological signs of cardiac injury, the severity of cardiac injury is associated with the degree of catecholamine release in the plasma [19].

In the current work the mean age of patient were 47±18.5 years and females significantly, more affected than males which agreed with the results of Van Rooij *et al*. [20]. Another study showed that 69% of patients with SAH [195 of 284], was due to ruptured aneurysm [15].

Subarachnoid hemorrhage [SAH] from a ruptured constitutes 5% of all strokes which carries high disease-specific burden as half of patients are younger than 55 years, in addition to, one-third die within the initial days to weeks after the hemorrhage, and most survivors have long-term disability or cognitive impairment **[21].**

Vergouwen *et al*. showed that the age of SAH incidence was decreased by 24% between 1998 and 2012 **[22].** Other study found that the mean age was 57.1 ± 12.8 years, and 56.1% were females, and aneurysmal rupture was the most common cause of SAH in China [77.4%] **[23].**

In the current work there were no significant difference between cases and control regarding diabetes, serum cholesterol and LDLc while there were significant increase in smoking and hypertension in SAH which agreed with the results of many researches [22, 24, 25].

DM has negative associated with the risk of saccular intracranial aneurysm development and rupture, which may cause aSAH **[26]**. However, results are inconclusive regarding the relationship between DM and aSAH **[27].**

Diabetes increases the incidence of cerebrovascular events by 1.7 times higher than non-diabetic persons. Which was restricted to atherosclerotic disease and cerebral infarction, and the frequency of cerebral infarction is higher than expected in diabetic patients. However, a low occurrence of DM in patients with aSAH were reported by the Harvard Cooperative Stroke Registry [24].

Inagawa suggested that an inverse relationship between diabetes mellitus and aneurysm rupture, which might be attributed to the atherosclerotic wall because atherosclerotic aneurysms are less likely to rupture [28], which also explain why we have decreased association in older than younger diabetic patients.

Studies regarding the role of total cholesterol [TC] in risk for SAH are conflicting, which showed both high **[29]** and low [30] TC to raise risk. One meta-analyses suggest no association between TC and SAH, whereas they reported that high HDL has a protective effect against SAH [31].

Recently, it was concluded that hyper-cholesterolemia is associated with paradoxical 40% reduction in the risk of a SAH [32]. Hypertension is established as a major risk factor for the development and rupture of cerebral aneurysms [33].

Although it was suggested that systolic BP in SAH should remain below 180 mm Hg to reduce the risk of rebleeding till the coiling or clipping of ruptured aneurysms [34]. It was reported that high levels of blood pressure were linearly and positively related to higher incidence of SAH in addition to, 10 mm Hg higher usual level of SBP, or a 5 mm Hg higher level of DBP, was associated with about 20% higher risk of SAH [35]. In 2017, Xu *et al*. analyze the relationship between blood pressure variability [BPV] and prognosis. He found that systolic BPV is an independent predictor of functional recovery in patients with SAH [36].

In subclinical studies, experimental induction of aneurysm formation in 3 weeks were done by injection elastase to degrade the internal and external elastic lamina of cerebral vessels **[37]** combination of hypertension, hemodynamic stress, and elastase triad, lead to the first mouse model with intracranial aneurysm formation [38]. In this method, mice were injected with elastase and continuously infused with angiotensin-II to produce the desired hypertension and hemodynamic stress [39].

This, lead to intracranial aneurysms of 500 μm size with a dose-dependent relationship between aneurysm incidence and concentrations of both elastase and angiotensin-II [38].

The use of angiotensin-converting enzyme inhibitors attenuated aneurysm rupture [40]. The mechanism by which angiotensin II promotes aneurysm rupture were attributed to its involvement in systemic inflammation and generation of the reactive oxygen species in the vessel wall [41].

Cigarette smoking is one of the most important preventable risk factor of subarachnoid hemorrhage [SAH], with a strong dose-response relationship which has been shown in many studies **[22].** Cigarette smoking increase the risk of SAH by 5 times compared with nonsmokers in addition to, about one third of all cases of SAH are current smokers **[42]**.

In the current work there were about 15% of SAH cases showed impairment of fraction of shortening and systolic function by M mode and biplane methods, 9% of

cases showed RWMA in the form of anteroseptal hypokinesia by speckle tracking echo LV GLS were impaired in about 25% of cases in addition to, significant increase in cardiac biomarker troponin I in about 9% of cases. There were ST T wave changes in about 80% of cases in addition to, significant prolongation in QTc compared to control group. Hunt and Hess scale were done for cases as prognostic scale to classify cases which was above 3 in 17% of cases. These results were agreed with the results of different studies [3, 43-49].

Systemic manifestations of SAH are due to wide spread neuroendocrine responses, inflammatory and immune-mediated mechanisms that occur in acute phase of SAH. These mechanisms are responsible for the systemic actions of SAH including the myocardium. Intense activation of hypothalamus, and brain stem lead to activation of sympathetic nerve endings causing release of norepinephrine with reciprocal decrease in para-sympathetic outflow [50].

Reduction in cardiac injury was noticed after blocking of sympathetic outflow to the heart. High levels of circulating catecholamine were found in the blood of patients with cardiac damage indicating the stimulation of adrenal medulla [51].

The myocardial necrosis in the form of coagulative myocytolysis was which was characterized by an excessive calcium influx and early myocyte calcification. Besides, invasion of the myocardium of patients who died after subarachnoid hemorrhage [SAH] with neutrophil granulocytes, lymphocytes and macrophages [52].

The myocardial necrosis provokes the systemic inflammatory reaction, which lead to increase in release of cytokines, complement system [50]. Electrocardio-graphic changes after SAH are well recognized and studied. It is seen in 25%–90% of SAH cases usually in acute phase and resolve within 6 weeks [53].

ECG changes usually in the form of repolarization abnormalities, ischemic changes, and rhythm or conduction disturbances [50]. QT interval prolongation, ST segment and T wave changes are common abnormalities seen, because of sympathetic activation due to hypo-thalamic stimulation [54].

Increased heart rate in addition to, QTc prolongation in 91% SAH patients was noted. Global LV dysfunction was associated with ischemic changes, whereas patients with re-polarization changes had normal LV function. Poor case prognosis were noted in patients with ischemic changes while, not in repolarization abnormalities [55].

SAH may have features of global or regional systolic dysfunction, diastolic dysfunction. Stroke volume and cardiac output may fall. Global or regional systolic dysfunctions have significant incidence ranging from 8% to 50% Within first two days of SAH RWMA can be seen followed by gradual improvement over time, majority of these patients have normal coronaries [56].

Poor Hunt-Hess classification and elevated cTnI >1.0 µg/L, were found to be strong predictor of RWMA while, comorbidities such as hypertension, old age, diabetes mellitus and hyper-lipidaemia are not [50]. Some studies showed that RWMA was seen in the apical segments whereas the basal segments were hyperkinetic. Others showed that it may be in the anterior or anteroseptal area but both of them do not correspond to a particular coronary artery territory. Presence of RWMA has been associated with poor prognosis which may be explained by an impaired cardiac output in addition to, disturbed cerebral autoregulation after aneurysmal SAH [57]. Left ventricle dysfunction decreases cerebral blood flow in comparison with those without LV dysfunction [58].

Stunned myocardium [SM] in which extensive myocardial injury following SAH can occur which is presented with severely depressed global cardiac, especially LV function. SM is considered as fully reversible, occurring within 24 h of SAH and gradual recovery as early as 48 h to 7 days [59]. The cTnI increased within 12 h, peaked within 48 h and returned to normal in 7–10 days which is considered a reliable marker of myocardial injury leading to LV dysfunction. Increase in cTnI concentration is a marker of poor prognosis in patients with SAH [60].

In the current work there were positive correlations between Hunt Hess grade and hypertension, Hs Tn I and LVGLS. While, there were negative correlation between Hunt Hess grade and diabetes, serum cholesterol, LDLc, EF [by both M mode and biplane], LVEDD and LVESD which agreed with the results of [61-63].

Previous studies have identified severity of neuro-logical injury as a major predictor of cardiac injury after a SAH, with patients presenting as Hunt-Hess > 3 having higher risk. This association supports the hypothesis that cardiac injury after subarachnoid hemorrhage is a neurally mediated process [64]. Hypertension is widely known to be a major risk factor for the development and rupture of cerebral aneurysms [65-66].

Lin *et al*. reported abnormal troponin level was associated with future major adverse cardiac events and unfavorable long-term outcomes. In addition to, increased cardiac TnI was related to an increased risk of future major adverse cardiac events and deaths after a SAH [67].

Acute brain injury from SAH is associated with sudden increases in intracranial pressure which triggers a strong autonomic stimulation. In addition to, local and systemic release of catecholamine, especially at the level of adrenergic receptors in the myocytes leading to cell injury and death. In addition, catecholamine toxicity may result in cellular functional changes including conduction or contractile impairment or death. The pattern of cellular injury due to intense sympathetic stimulation follows the distribution of the sympathetic nerve terminal endings, rather than following the distribution of the coronary arteries. It has been noted that the myocardial cellular damage associated with neurocardiac injury has been previously observed as cardiac troponin release in

approximately 20 to 35% of patients [68].

Strain imaging of myocardium can be used to quantify neurocardiac injury associated with poor clinical outcomes in patients with aneurysmal SAH. Decrease in GLS was significantly associated with in-hospital mortality and was considered as routine clinical markers for prognosis of SAH. Specifically, abnormal LV GLS and RV strains in acute SAH patients were observed to be commonly occur even in the setting of preserved or hyperdynamic LVEF. Furthermore, abnormal LV GLS remained significantly associated with in-hospital mortality even after adjusting for known factors associated with outcome after SAH, including Hunt-Hess grade [69].

Conclusion

Subclinical LV systolic dysfunction is common complication of SAH, which may be associated with poor case prognosis and bad clinical outcome, so we recommend routine assessment of systolic function for all admitted cases of SAH.

Conflict of interest

The authors declare that there is no conflicts of interest.

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