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Prevalence of Stress Cardiomyopathy in Polytrauma Patients

Samir Mohamed Attia[1], Nader El Shahat Awad[2], Mohamed Elsaid Ahmed[3], Amany Medhat Ezzat Youssef*[4]
Departments of [1]Vascular Surgery, [2]Cardiology, [3]Critical Care Medicine and
[4]Emergency Medicine, Faculty of Medicine, Mansoura University, Egypt
***Corresponding author:** Amany Medhat Ezzat Youssef, **Mobile:** (+20) 01011271652,
E-Mail: mohamedsaeedahmed68@gmail.com

ABSTRACT

Background: Polytrauma is a leading cause of death globally and usually involving young victims. Takotsubo cardiomyopathy (TCM) is a serious clinical presentation of myocardial dysfunction characterized by acute, transient, and reversible heart failure syndrome owing to regional wall abnormalities of the ventricular myocardium associated with ECG changes and rising of myocardial biomarkers in the absence of cardiac condition causing the temporary ventricular dysfunction**. Objective:** To detect the prevalence of stress cardiomyopathy among polytrauma patients attending to Emergency Hospital Mansoura University. **Patients and methods:** The current study was a cross sectional study conducted on 500 patients who attended to Emergency Department, Emergency Hospital Mansoura University, suffering from polytrauma within the period from August 2019 to August 2020.

Results: Prevalence of cardiomyopathy among the studied trauma patients was 4.4%. Mode of trauma (either blunt or penetrating) had no effect in the incidence of cardiomyopathy. Sequential organ failure assessment (SOFA) score and revised trauma score (RTS) demonstrated positive correlation with incidence of cardiomyopathy among polytrauma cases (P<0.001). Cardiomyopathy group was associated with marked affection of all echocardiographic parameters (Stroke volume, end diastolic volume, ejection fraction and fractional shortening) except end systolic volume. Cardiomyopathy group was associated with marked increase in ICU admission, ICU length of stay, total length of stay and mortality rate.

Conclusion: Presence of stress cardiomyopathy in trauma cases may be used as an indicator of poor outcomes. Both SOFA and RTS scores were considered as reliable tools used to describe organ dysfunction/failure in polytrauma cases and have a positive correlation with stress cardiomyopathy.

Keywords: ICU, Polytrauma, RTS, SOFA, Stress Cardiomyopathy.

INTRODUCTION

Polytrauma is a leading cause of death worldwide, often involving young victims. Stress cardiomyopathy (inverted takotsubo cardiomyopathy) is a rare but potentially lethal complication in this setting, featuring transient myocardial contractile dysfunction**.** The sequelae of severe polytrauma may include myocardial dysfunction followed by acute heart failure and death **(1).** Evidence is emerging that systemic inflammation after trauma drives structural and functional impairment of cardiac function, thus worsening the outcome of poly-trauma patients **(2).**

Inverted takotsubo cardiomyopathy (ITCM) usually has benign course, however, on rare instance, it can result in life-threatening and fatal complications including acute cardiogenic shock, ventricular arrhythmias and ventricular wall rupture **(3).**

ITCM is a variation, first described by **Dote *et al.*** **(4).** It was so-named because of an unusual shape of the left ventricle that is similar to that of a Japanese octopus's trap, 'Takotsubo', when observed on left ventriculography in the systolic phase.

The inverted-type is more common in young people and maybe associated with specific clinical conditions **(5),** including trauma **(6).** It is characterized by akinesia or dyskinesia of the basal left ventricular segments and hypercontractility of the apex. Endogenous and exogenous catecholamine surges may be the mediators **(7).**

Although ITCM is a rare complication, it occurs more frequently in polytrauma patients. Moreover, it is characterized by left ventricular wall motion abnormalities in the mediobasal segments and always by normal coronary arteries **(8).**

The aim of the present study was to detect the prevalence of stress cardiomyopathy among polytrauma patients attending to Emergency Hospital Mansoura University.

PATIENTS AND METHODS

The current research was a cross sectional study conducted on 500 patients who attended to Emergency Department, Emergency Hospital Mansoura University, suffering from polytrauma within the period from August 2019 to August 2020.

Inclusion criteria: Polytrauma patients, injury severity score ≥ 16, admission to Emergency Hospital, all age group, and both genders.

Exclusion criteria: Previous cardiac disease, injury severity score <16, and patients who refused the participation in the study.

Methods:

1. **Primary survey and resuscitation:**

- **A = Airway opening and maintenance of airway.**
- **B = Breathing and ventilation.**
- **C = Circulation.**
- **D = Disability:** neurological status and Glasgow coma scale (GCS).

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- **E = Exposure**: segmental exposure.

1. **Secondary survey** (from head to nail examination: head, neck, chest, abdomen, pelvis, lower limb, upper limb) and Vital sign (pulse, blood pressure, respiratory rate).
2. **AMPLE History:**
   - **A**= Allergies.
   - **M**= Medication currently used.
   - **P**= Past illnesses / Pregnancy.
   - **L**= Last meal.
   - **E**= Events / Environment related to injury (Exactly what happened?).
3. **Both SOFA and Revised trauma score** were evaluated on admission.
4. **Investigations:**

   (A) Laboratory tests:
   - Complete blood count (CBC).
   - International normalization ratio (INR) with coagulations profile.
   - ABO grouping.
   - Serum Creatinine.
   - Arterial blood gases.
   - Total bilirubin.
   - Cardiac biomarkers including troponins and CK-MB.

   (B) Radiological investigations:
   - X-ray.
   - Focused assessment sonography for trauma patient (FAST) (Ultrasound, Model)
   - CT Brain if needed.
   - Echocardiography (ECHO): searching for criteria of cardiomyopathy by searching for wall motion abnormalities, ejection fraction, fractional shortening, left ventricular size.
5. **Outcome was calculated within one week in those who had cardiomyopathy and those who didn't according to:** ICU Admissions, mechanical ventilation, and length of hospital stay.
6. **Diagnosis and incidence of stress cardiomyopathy.**
7. **Mortality within one week.**

Ethical consideration:

The research approval of the study was obtained from institutional review board of Faculty of Medicine at Mansoura University before starting the study. The researcher clarified the objective and aim of the study to the patients included in the study. The researcher assured maintaining anonymity and confidentiality of patient's data. Patients were informed that they were allowed to choose to participate or not in the study and that they had the right to withdraw from the study at any time without giving any reasons. Ethics, values, culture and beliefs of patients were respected.

Statistical analysis

IBM's SPSS statistics (Statistical Package for the Social Sciences) for windows (version 25, 2017) was used for statistical analysis of the collected data. Shapiro-Wilk test was used to check the normality of the data distribution. All tests were conducted with 95% confidence interval. P (probability) value < 0.05 was considered statistically significant. Charts were generated using SPSS' chart builder and Microsoft Excel for windows 2019. Quantitative variables were expressed as mean and standard deviation while categorical variables were expressed as frequency and percentage.

RESULTS

The age and gender of the studied patients and medical history are shown in table (1).

Table (1): Demographic characteristics and medical history of the studied patients

		All patients (n= 500)
Age		41.35 ± 13.039
Gender	**Male**	361 (72.2%)
	Female	139 (27.8%)
Diabetes mellitus		68 (13.6%)
Hypertension		49 (9.8%)
Hypercholesterolemia		61 (12.2%)

There were statistically significant differences between the patients with no cardiomyopathy and those with cardiomyopathy as regards oral hypoglycemic use and insulin use (Table 2).

Table (2): Treatment history of the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	p
Anti-Platelet	2.7% (13)	4.5% (1)	>0.05
Oral Hypoglycemic	2.1% (10)	31.8% (7)	**< 0.001**
Insulin	8.8% (42)	40.9% (9)	**< 0.001**
Anticoagulants	2.1% (10)	0.0% (0)	>0.05
Beta blocker	3.3% (16)	0.0% (0)	>0.05
angiotensin-converting enzyme inhibitors	2.5% (12)	4.5% (1)	>0.05
Ca Channel blocker	5.0% (24)	0.0% (0)	>0.05
Diuretic	3.6% (17)	0.0% (0)	>0.05
Statin	7.3% (35)	13.6% (3)	>0.05

There was no statistically significant differences between the patients with no cardiomyopathy and those with cardiomyopathy as regard the mode of trauma (Table 3)

Table (3): Mode of trauma of the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	p
Penetrating	15.1% (72)	22.7% (5)	>0.05
Blunt	84.9% (406)	77.3% (17)	

The mean SOFA score was significantly higher among patients with cardiomyopathy compared with those with no cardiomyopathy. On the contrary, the mean RTS score and the mean systolic blood pressure (SBP) were significantly higher among patients with no cardiomyopathy compared with those with cardiomyopathy (Table 4).

Table (4): SOFA, RTS scores and systolic blood pressure in the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	95% CI	p
SOFA	2.83 ± 1.685	8.00 ± 2.047	- 5.90, - 4.44	< **0.001**
RTS	3.50 ± 0.023	2.52 ± 0.698	0.91, 1.04	< **0.001**
SBP (mmHg)	125.76 ± 8.565	97.27 ± 8.827	24.82, 32.17	< **0.001**

Patients with no cardiomyopathy had a significantly higher mean Hb level than those with cardiomyopathy but they also had a significantly lower mean creatinine level than those with cardiomyopathy (Table 5).

Table (5): Laboratory investigations in the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	95% CI	p
Hb (g/dL)	11.25 ± 1.382	9.40 ± 1.490	1.25, 2.44	< **0.001**
WBC (mcL)	10.96 ± 1.880	10.65 ± 1.684	-0.49, 1.11	>0.05
PLT (mcL)	259.10 ± 46.706	247.18 ± 53.199	- 8.22, 32.05	>0.05
INR	1.30 ± 0.180	1.26 ± 0.173	-0.04, 0.12	>0.05
Creatinine (mg/dL)	1.28 ± 0.240	2.32 ± 0.297	-1.15, -0.94	< **0.001**

Patients with no cardiomyopathy had significantly higher means of stroke volume, ejection fraction and fractional shortening compared with those with cardiomyopathy but they showed a significantly lower mean end diastolic volume compared with those with cardiomyopathy (Table 6).

Table (6): Echocardiographic parameters in the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	95% CI	p
Stroke Volume	73.89 ± 10.201	62.41 ± 9.936	7.12, 15.85	< **0.001**
End systolic volume	63.04 ± 11.443	69.86 ± 17.094	- 11.85, - 1.80	>0.05
End diastolic volume	137.14 ± 15.999	163.68 ± 27.228	-33.66, -19.42	< **0.001**
Ejection fraction	54.12 ± 5.952	45.77 ± 7.880	5.75, 10.93	< **0.001**
Fractional shortening	23.77 ± 3.267	19.23 ± 3.816	3.13, 5.96	< **0.001**

As shown in table 7, there was a statistically significant difference between both groups. As regard ICU admission and length of stay, total length of stay and the overall mortality rate was significantly.

Table (7): ICU admission, length of ICU and hospital stay in the studied patients

	No Cardiomyopathy (n= 478)	Cardiomyopathy (n= 22)	95% CI	p
ICU admission	7.3% (35)	81.8% (18)	0.58, 0.91	< **0.001**
ICU Length of Stay (Days)	4.29 ± 1.487	10.00 ± 1.680	- 6.62, - 4.81	< **0.001**
Total Length of Stay (Days)	19.33 ± 9.183	27.05 ± 10.214	-11.67, -3.76	<**0.001**
Mortality rate	2.3% (11)	31.8% (7)	0.1, 0.49	< **0.001**

DISCUSSION

In our study, regarding demographic data, the mean age of the studied cases was 41.35 and the majority of them were male with no statistically significant difference among stress and non-stress cardiomyopathy groups as regards both parameters. This came in agreement with many previous researches who demonstrated that blunt trauma was more common in male sex in comparison with female one [9, 10]. While, **Naganathar *et al.*** [11] performed their study at level I trauma center in London from September 2010 to October 2012. All patients were above the age of 16 and met the criteria for trauma team activation. The study population had a median age of 38 years and 73% were males. They demonstrated that there were statistically significant differences among stress and non-stress group as regards all age groups (from more than 16 years old, 30-45, 46-60, 60-80 and more than 81 years old)

(P<0.05) while there was no statistically significant difference as regards the sex (P=0.07).

The current study demonstrated that the prevalence of stress cardiomyopathy among polytrauma cases was 4.4%. In the same line, **Seguin *et al.*** [12] revealed in their study that, the prevalence of stress cardiomyopathy and atrial fibrillation (AF) in polytrauma cases was 16/293 (5.5%) especially in cases who were older, had a higher number of regions traumatized, and received more fluid therapy, transfusion products and catecholamine.

Moreover, **Naganathar *et al.*** [11] evaluated the prevalence of cardiac events according to the area of trauma. They demonstrated that direct thoracic injury was shown to be associated with stress cardiomyopathy and a rise in cardiac biomarkers. In addition the patients who developed stress cardiomyopathy had more significant thoracic Abbreviated Injury Scale (AIS score 3 vs 1) and head injuries (AIS 0 vs 3). Patients with severe thoracic injury were more likely to develop an adverse cardiac event (42% vs 18%, p=0.01). However, severe head injury (AIS>3) was not associated with a higher prevalence of cardiac events.

Regarding head trauma studies, there was higher incidence of cardiac injuries and stress cardiomyopathy in comparison with polytrauma as evaluated by **Prathep *et al.*** [13] who demonstrated that the incidence of cardiac dysfunction in patients with head trauma was about 32%. They reported abnormal echocardiographic examination in 22.3 % of patients and elevated serum troponin in 24 % of patients, with no data about patients' ECG abnormalities. In addition, **Hasanin *et al.*** [14] reported in their study on head trauma cases that half of the patients developed cardiac injury as documented by elevated troponin I in 54 % of patients, abnormal ECG in 62 % of patients, and echocardiographic examination in 42 % of patients.

The discrepancy among the current study and their studies is mainly due to the fact that, the present study was performed on polytrauma cases, while their studies were performed on head trauma only and it is well known that there is a positive correlation between stress cardiomyopathy and acute mental or physical stress and major head trauma, as a result of exogenous catecholamine administration which increase the possibility of development of stress cardiomyopathy in head trauma than in polytrauma [15]**.** This can be explained by that in polytrauma patients, high circulating levels of adrenaline have been shown to be associated with endothelial dysfunction, coagulopathy and increased mortality [16, 17]**.**

As regards, echocardiographic parameters, there were highly statistically significant differences between stress and non-stress cardiomyopathy groups regarding stroke volume, end diastolic volume (EDV), ejection fraction (EF) and fractional shortening. Supporting to this theory **Seguin *et al.*** [18] demonstrated a greater incidence of AF associated with catecholamine use.

As regards, drug history of the studied cases, there were no statistically significant differences among both groups except in oral hypoglycemic and insulin, which showed higher utilization among stress cardiomyopathy group with highly statistically significant differences in comparison with non-stress group. While, **Naganathar *et al.*** [11] revealed that there were no statistically significant differences among both groups except in insulin, antiplatelet, calcium channel blockers (CCBs) and statin only.

As regards mode of trauma, most of the patients were affected by blunt trauma (84.6%), while the remaining ones were penetrating (15.4%). This came in accordance with **Naganathar *et al.*** [11] who demonstrated that the majority of patients had suffered blunt injury (82%) while only 12% had suffered from other injuries with higher incidence of stress cardiomyopathy among blunt injuries.

As regards SOFA and RTS scores, the current study revealed that stress group developed more SOFA and RTS score in comparison with non-stress group with highly statistically significant differences among both group. In contrary, **Muratsu *et al.*** [19] reported in their study on ICU patients that, the septic patients with takotsubo cardiomyopathy (TCM) had lower SOFA scores than the patients without TCM. They explained that such action may be due to catecholamine-induced cardiomyopathy. They also recommended that further studies are needed to understand the relationship between the degree of physical stress experienced and the development of TCM.

The present study demonstrated that systolic blood pressure (SBP) among TCM group was reduced in comparison with no stress group with highly statistically significant difference. In the same line, **Naganathar *et al.*** [11] revealed that, adverse cardiac events (ACE) group was more hypotensive than non-ACE group with a median SBP of 100 vs 130 and more shocked with a lactate of 3.5 vs 2.0 respectively.

As regards medical history, the present study revealed that there was statistically significant difference among both groups as regards diabetes mellitus and hypertension, while there were no statistically significant difference as regards hypercholesterolemia. This was in agreement with **De'Ath *et al.*** [20] who demonstrated that there was no difference in pre-existing diseases such as hypercholesterolaemia and ischemic heart disease among stress and non-stress traumatized groups, while they were in contrast to the present result as regards diabetes mellitus and hypertension, which demonstrated no statistically significant differences in their study. This raised the possibility of further underlying mechanisms for secondary cardiac injury in trauma patients.

As regards ICU admission, the current study demonstrated that there were highly statistically significant differences as regards ICU admission (81.8% vs 7.3%), ICU length of stay, total length of stay and mortality rate (31.8% vs 2.3%) among stress and non-stress cardiomyopathy groups. In accordance, **Naganathar *et al.*** [11] reported that, patients who developed adverse cardiac events (ACEs) were twice as

more likely to require admission to ICU (95% vs 47%, p<0.01) and on average required 13 days longer in ICU when compared to those who did not develop ACE. In addition, those that developed ACEs spent 21 days longer in hospital (28 vs 7 days, p<0.01). Cardiac events were also associated with four-fold increase in mortality (24% vs 6%, p<0.01).

Moreover, **Brinjikji *et al.* [21]** demonstrated that, a total of 24,701 patients with TCM were identified in their study. In-hospital mortality rate was 4.2%. A total of 21,994 patients (89.0%) were female. Male patients had a higher mortality rate than females (8.4% vs 3.6%, P < 0.0001). In the same line, **Singh *et al.* [18]** reported in their study that, in-hospital mortality rate among patients with TCM was 4.5% (95% CI 3.1 to 6.2, I2 = 60.8%). Among all deaths, 38% were directly related to TCM complications and rest to underlying non-cardiac conditions.

CONCLUSION

Polytrauma cases had positive correlation with stress cardiomyopathy and were more liable for more adverse cardiac events, which were associated with prolonged hospital and ICU admission and were also associated with high morbidity and mortality rates. Thus, presence of stress cardiomyopathy in trauma cases may be used as an indicator of poor outcomes. Both SOFA and RTS scores were considered as reliable tools used to describe organ dysfunction/failure in polytrauma cases and have a positive correlation with stress cardiomyopathy.

REFERENCES

1. **Bonacchia M, Vanninia A, Harmelina G *et al.* (2015):** Inverted-Takotsubo cardiomyopathy: severe refractory heart failure in poly-trauma patients saved by emergency extracorporeal life support. Interactive CardioVascular and Thoracic Surgery, 20: 365–371.
2. **Braun C, Kalbitz M, Halbgebauer R *et al.* (2018):** Early structural changes of the heart after experimental polytrauma and hemorrhagic Shock. PLoS ONE 12(10): 0187327.
3. **Iskander M, Abugroun A, Shehata K *et al.* (2018):** Takotsubo cardiomyopathy-induced cardiac free wall rupture: A case report and review of literature. Cardiol Res, 9(4): 244–249.
4. **Dote K, Sato H, Tateishi H *et al.* (1991):** Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases. J Cardiol., 21:203–14.
5. **Ennezat P, Pesenti-Rossi D, Aubert J *et al.* (2005):** Transient left ventricular basal dysfunction without coronary stenosis in acute cerebral disorders: a novel heart syndrome (inverted Takotsubo): Echocardiography, 22:599–602.
6. **Samol A, Grude M, Stypmann J *et al.* (2011):** Acute global cardiac decompensation due to inverted Takotsubo cardiomyopathy after skull–brain trauma—a case report. Injury Extra, 42: 54–7.
7. **D'Errico S, Neri M, Nieddu A *et al.* (2011):** Cardiac β1-adrenoceptor expression in two stress-induced cardiomyopathyrelated deaths. Forensic Sci Int., 207:8–11.
8. **Song B, Chun W, Park Y *et al.* (2011):** The clinical characteristics, laboratory parameters, electrocardiographic, and echocardiographic findings of reverse or inverted Takotsubo cardiomyopathy: comparison with mid or apicale variant. Clin Cardiol., 34: 693–9.
9. **El-Din M, Nashy M, Meshhal M (2018):** Blunt vs. penetrating chest trauma in terms of the outcome in Menoufia University Hospital. Menoufia Medical Journal, 31(1): 133-36.
10. **Naeem B, Perveen S, Naeem N *et al.* (2018):** Visceral injuries in patients with blunt and penetrating abdominal trauma presenting to a tertiary care facility in Karachi, Pakistan. Cureus, 10(11): e3604.
11. **Naganathar S, Wall J, Praditsuktavorn B *et al.* (2019):** Modeling Cardiac Dysfunction Following Traumatic Hemorrhage Injury: Impact on Myocardial Integrity. Frontiers in Immunology, 10:2774.
12. **Seguin P, Laviolle B, Maurice A *et al.* (2006):** Atrial fibrillation in trauma patients requiring intensive care. Intensive Care Medicine, 32(3): 398-404.
13. **Prathep, S, Sharma, D, Hallman, M, *et al.* (2014):** Preliminary report on cardiac dysfunction after isolated traumatic brain injury. Critical Care Medicine, 42(1): 142-7.
14. **Hasanin A, Kamal A, Amin S *et al.* (2016):** Incidence and outcome of cardiac injury in patients with severe head trauma. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine, 24(1): 1-6.
15. **Bybee K, Prasad A (2008):** Stress-related cardiomyopathy syndromes. Circulation, 118(4): 397-409.
16. **Johansson P, Stensballe J, Rasmussen L *et al.* (2012):** High circulating adrenaline levels at admission predict increased mortality after trauma. Journal of Trauma and Acute Care Surgery, 72(2): 428-436.
17. **Ostrowski S, Henriksen H, Stensballe J *et al.* (2017):** Sympathoadrenal activation and endotheliopathy are drivers of hypocoagulability and hyperfibrinolysis in trauma: a prospective observational study of 404 severely injured patients. Journal of Trauma and Acute Care Surgery, 82(2): 293-301.
18. **Singh K, Carson K, Shah R *et al.* (2014):** Meta-analysis of clinical correlates of acute mortality in takotsubo cardiomyopathy. The American Journal of Cardiology, 113(8): 1420-1428.
19. **Muratsu A, Muroya T, Kuwagata Y (2019):** Takotsubo cardiomyopathy in the intensive care unit. Acute Medicine & Surgery, 6(2): 152-157.
20. **De'Ath H, Rourke C, Davenport R *et al.* (2012):** Clinical and biomarker profile of trauma-induced secondary cardiac injury. British Journal of Surgery, 99(6): 789-97.
21. **Brinjikji W, El-Sayed A, Salka S (2012):** In-hospital mortality among patients with takotsubo cardiomyopathy: a study of the National Inpatient Sample 2008 to 2009. American Heart Journal, 164(2): 215-221.